Abstracts

Platform Presentations

Session I

1. 10 U/KG/HR OF HIGH DOSE INSULIN IS SUPERIOR TO 1 U/KG/HR IN A BLINDED, RANDOMIZED, CONTROLLED TRIAL IN POISON-INDUCED CARDIODEMIC SHOCK

Jon B. Cole, Kristin M. Engelbreten, Samuel J. Stellpflug, Heather Ellisworth, Lisa M. Havenka, Alex B. Adams, Christopher P. Anderson, Joel S. Holger

Regions Hospital, Saint Paul MN USA

Background: High dose insulin (HDI) has proven superior to glucagon and catecholamines in the treatment of Poison-Induced Cardiogenic Shock (PICS) in previous studies. Recommendations for dosing of HDI vary, from 0.5 U/kg/hr to 10 U/kg/hr. The optimal dose of HDI in PICS has not been previously studied or established. We hypothesized a dose of 10 U/kg/hr of HDI would be superior to 1 U/kg/hr in the treatment of pigs with PICS from propanolol (P) with cardiac output (CO) as our primary outcome measure.

Methods: This was a blinded, randomized and controlled trial with 4 arms consisting of 4 pigs in each arm. The arms were as follows: saline control (C), or HDI at 1 U/kg/hr (1), 5 U/kg/hr (5), and 10 U/kg/hr (10). Pigs were anesthetized and underwent placement of a tracheostomy, Swan-Ganz catheter and arterial line. All pigs received a P bolus of 0.5 mg/kg followed by an infusion of 0.25 mg/kg/min until the point of toxicity was reached, defined as a 25% reduction in baseline heart rate (HR) x mean arterial pressure (MAP). At this point a 20 mL/kg bolus of saline (NS) was administered. The P infusion was reduced to 0.125 mg/kg/min and continued throughout the protocol. After the NS bolus the blinded infusion of C, 1, 5 or 10 was started and pigs were resuscitated for 6 hours or until death. CO, HR, MAP, arterial blood gas, systemic vascular resistance (SVR) and serum glucose were recorded every 10 minutes. Serum potassium (K) and lactate were recorded hourly.

Results: Survival: 2 pigs died in the C arm, 1 pig died in each of the 1 and 5 arms, and no pigs died in the 10 arm. There was a statistically significant increase in CO of 1.13 L/min in the 10 arm compared to the 1 arm over the 6 hour study period (p = 0.009). Given the average nadir CO of 2.0 L/min, this represents a 57% increase in CO. A linear mixed-effects regression found a statistically significant increase in dose by time interaction, in which CO improves an average of 1.13 L/min in the 10 arm compared to the 1 arm over the 6 hour study period. There was also a statistically significant difference in dose by time interaction on MAP, HR, SVR and base excess, but not lactate or K. No differences in glucose utilization were found between the three insulin arms.

Conclusion: HDI was statistically and clinically significantly superior to placebo in this pranproanolol model. 10 U/kg/hr was statistically and clinically significantly superior to 5U/kg/hr, which was statistically and clinically significantly superior to 1 U/kg/hr. It may be appropriate to begin HDI therapy for Poison-Induced Cardiogenic Shock at 10 U/kg/hr.

2. CROTALINE FAB ANTIVENOM REVERSES PLATELET DYSFUNCTION INDUCED BY C. SCUTULATUS VENOM: AN IN VITRO STUDY

Shaun D. Carstairs1, Allyson A. Kreshak2, David A. Tanen1
1Naval Medical Center, San Diego CA USA; 2Brown University, Providence RI USA

Background: Patients sustaining rattlesnake envenomation often develop thrombocytopenia, the etiology of which is not clear. Laboratory studies have demonstrated that venom from several species, including the Mojave rattlesnake (Crotalus scutulatus scutulatus), can inhibit platelet aggregation. In humans, administration of crotaline Fab antivenom (CroFab®) has been shown to result in transient improvement of platelet levels; however, it is not known whether platelet aggregation also improves after antivenom administration.

Objectives: To determine the effect of C. scutulatus venom on platelet aggregation in vitro in the presence and absence of crotaline Fab antivenom.

Methods: Blood was obtained from 4 healthy male adult volunteers not currently using aspirin, NSAIDs, or other platelet-inhibiting agents. C. scutulatus venom from a single snake with known type B (hemorrhagic) activity was obtained from the National Natural Toxins Research Center. Measurement of platelet aggregation by an aggregometer was performed using 5 standard concentrations of epinephrine (a known platelet aggregator) on platelet-rich plasma over time, and a mean area under the curve (AUC) was calculated. 5 different sample groups were measured: 1) blood alone; 2) blood + C. scutulatus venom (0.3 mg/mL); 3) blood + crotaline Fab antivenom (100 mg/mL); 4) blood + venom + antivenom (100 mg/mL); 5) blood + venom + antivenom (4 mg/mL). Standard errors of the mean (SEM) were calculated for each group, and paired t-tests were used to measure differences between groups.

Results: Antivenom administration by itself (group 2) did not significantly affect platelet aggregation compared to baseline (103.8 ± 3.4%, p = 0.47). Administration of venom (group 3) decreased platelet aggregation (72.0 ± 8.5%, p < 0.05). Concentrated antivenom administration in the presence of venom (group 4) normalized platelet aggregation (101.4 ± 6.8%) and in the presence of diluted antivenom (group 5) significantly increased aggregation (133.9 ± 9.0%); p < 0.05 for both groups when compared to the venom-only group. To control for the effects of the venom and antivenom, each was run independently in platelet-rich plasma without epinephrine; neither was found to significantly alter platelet aggregation in the absence of epinephrine.

Conclusions: Crotaline Fab antivenom improved platelet aggregation in an in vitro model of platelet dysfunction induced by venom from C. scutulatus. The mechanism of action remains unclear but may involve inhibition of venom binding to platelets or a direct action of the antivenom on platelets.

3. LONG-TERM STABILITY OF ACTIVE INGREDIENTS IN EXPIRED PRESCRIPTION MEDICATIONS

Lee Cantrell1, Jeffrey Suchard2, Alan Wu1, Roy Gerona3
1California Poison Control System- San Diego Division, CA USA; 2Department of Emergency Medicine, University of California, Irvine Medical Center, CA; 3Department of Laboratory Medicine, San Francisco General Hospital/U.C. San Francisco, CA

Background: Debate exists regarding the relative potency of medications beyond their labeled expiration dates. Marketed prescription drug products in the US typically have expiration dating ranging from 12-60 months. However, federal programs (Shelf-Life Extension Program [SLEP]) have demonstrated that multiple medications can safely have their shelf-lives lengthened 10 or more years. Situations may arise where expired medications may be ingested or may be the only therapeutic option available. Our goal was to characterize the stability of some long-expired prescription medications.

Methods: Eight different prescription medications containing 13 active ingredients which had been stored in a retail pharmacy for a minimum of 25 years (range 25-40 years) in their original, unopened containers were analyzed. For each pill/capsule, three samples were analyzed. Each pill was dissolved and sonicated in methanol (20-30 min), reconstituted in analysis buffer (10% methanol) and analyzed with Liquid Chromatograph- Time-of-Flight Mass Spectrometer (TOF LC-MS, Agilent) using electrospray ionization in negative or positive polarity. The chromatography was run with gradient elution using Eclipse Plus C18 column (Agilent). Data analysis was done using Mass Hunter Qualitative and Quantitative Analysis (Agilent). Each sample was tested three times.

Results: Two drugs (ASA, amphetamine) were present in amounts <90% of their labeled content; eight drugs (acetaminophen, hydrocodone, codeine,...
butalbital, pentobarbital, secobarbital, methaqualone, phenacetin) were present in amounts between 90-110% of that listed; and 3 drugs (caffeine, meprobamate, chlorpheniramine) were found in amounts more than 110% of labeled content. **Conclusions:** The measured concentrations for 11/13 (85%) drug samples contained the generally recognized minimum acceptable potency (90% of the labeled potency) at least 2 decades from time of manufacture. The amounts of ASA detected were far below (<2%) the labeled amounts, consistent with the known poor long-term stability of this drug. The Results of this study, in conjunction with the data reported in the SLEP program, suggest that the current practices of drug expiration dating and re-stocking should be revisited. Also, healthcare providers should be aware that if long-expired medications are taken either in therapeutic or supratherapeutic amounts, they are likely to have retained their potency.

4. **USE OF COST EFFECTIVENESS STUDY TO ESTABLISH A PARTNERSHIP BETWEEN A POISON CONTROL CENTER AND A PRIVATE INSURANCE COMPANY**

Linda Kalin, Edward Bottei
Iowa Statewide Poison Control Center, Sioux City IA USA

**Background:** Poison control centers (PCC) provide tremendous savings to the health care system through cost avoidance. Beneficiaries of this cost avoidance include state and federal government health insurance programs, private insurance companies and the general public.

**Methods:** To quantify financial savings to private insurance companies, 648 callers to our PCC were surveyed to determine what alternative action they would have taken had the PCC not been available. Callers were also asked what type of health care insurance they had. Overall, 66% of callers were covered by private insurance, and PCC services generated an estimated savings of over $4.8 million per year to the private insurers. Armed with this compelling data, we approached a private insurance company in the hope of forming a partnership between the company and the PCC. Several meetings were held with the PCC’s Directors, members of the PCC’s governance board and the key leaders of the insurance company. An overview of PCC operations, core functions, and funding structure was provided. Data from the cost effectiveness study was presented in detail. An estimated annual cost savings provided to the private insurance company by the PCC was included.

**Results:** Representatives of the private insurance company acknowledged the magnitude of cost avoidance achieved by the PCC and clearly saw the direct value to their company and to the state. Executives from the private insurance company agreed to partner with the PCC and made an initial two year funding commitment to the PCC, with the potential for long-term involvement. An executive of the insurance company joined the PCC’s governance board to provide company representation and participate in the planning and decision-making of the organization.

**Conclusion:** The viability of the PCC is dependent on the support of organizations that see the most direct financial benefit. The economic value of a PCC for third party payers is clear and compelling. Our unique partnership is of mutual benefit to the private insurance company and the PCC, enhancing both mission and business goals. It is also the first step in providing other private insurance carriers in our state an increased awareness and understanding of the value of the PCC.

5. **THE EFFECT OF HOSPITAL BILLING ON POISON CENTER CALL VOLUME**

Claudia L. Barthold1, Kathy Jacobitz1, Jiangtao Luo2, Marcia Rasmussen1, Ronald Kirschen3
1Nebraska Regional Poison Center, Omaha NE USA; 2University of Nebraska Medical Center, Omaha NE USA

**Background:** A U.S. poison center (PC) serving multiple regions initiated a billing process on July 1, 2009 for calls from all acute care hospitals (ACHs) located in one state (Area A). This was done to supplement the PC’s budgetary shortfall. The ACHs in Area A were invoiced for ACH-initiated contacts on a per-case basis. ACHs were also given the option of arranging an individualized contract that provided unlimited calls for a monthly fee. Physician (MD) offices were not billed. No financial changes were made for the other regions serviced by the PC (Area B). The purpose of this study is to evaluate the effect that the initiation of ACH billing and the type of billing had on aspects of PC call volume (CV).

**Methods:** A retrospective examination of the PC database (Toxicall®) was performed after receiving IRB approval. Data was examined from the calendar year before the billing began (2008) to the calendar year after billing began (2010). A subset of the CV data was analyzed using the Spearman correlation. The Poison process was used to model the CV from each ACH. **Results:** When examining site of caller, ACH calls from Area A declined when comparing the year before and after billing began (~25% change). This decrease was not seen in Area B (~8.3% change). Total call volume before billing began was perfectly positively correlated for Area A and Area B. Total call volume after billing was perfectly negatively correlated for Area A and Area B. There was a statistically significant increase in expected calls between 2008 and 2010 when examining calls from HCFs with a PC contract (p < 0.03). There was a statistically significant decrease in expected calls between 2008 and 2010 when examining calls from HCFs without a PC contract (p = 0). All calls from MD offices declined in Area A when compared to Area B.

**Conclusions:** There was a decline in ACH calls for Area A after billing began. This decline was not seen in Area B. While Area A and Area B were positively correlated before billing, their call volumes negatively correlated the year billing began. Contracted ACHs had a statistically significant increase in CV after billing while non-contracted hospitals had a statistically significant decrease in CV after billing. There was a decline in MD office calls from Area A that was not seen in Area B during the study time. The effect on CV volume should be considered when initiating a poison center billing program.

6. **THE EFFECT OF ACETAMINOPHEN ON SERUM ALANINE AMINOTRANSFERASE ACTIVITY IN SUBJECTS WHO CONSUME ALCOHOL: A META-ANALYSIS OF PUBLISHED RANDOMIZED, CONTROLLED TRIALS**

A. B. Myracle1, B. H. Rumack1, K. Heard1, J. L. Green1, D. Albert1, R. C. Durt1, M. Bodmer2, M. L. A. Sivilotti3, B. Bucher-Bartelson1
1Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA; 2Emergency Department, University Hospital, Basel Switzerland; 3Department of Emergency Medicine, Queen’s University, Kingston ON Canada

**Background:** The current label for non-prescription acetaminophen products in the United States indicates that consumers who drink three or more alcoholic drinks every day should discuss the use of acetaminophen with their doctor as acetaminophen may cause liver damage. This recommendation is based primarily on anecdotal evidence and theoretical concerns. We sought to describe the effects of therapeutic doses of acetaminophen on serum aminotransferase activity in subjects who drink alcohol.

**Methods:** We conducted a systematic review and meta-analysis of randomized-placebo controlled trials (RCTs) involving acetaminophen that measured serum alanine aminotransferase (ALT) activity in subjects who consume alcohol. Studies were identified in a search of Medline (1950-2010), EMBASE (1980-2010), and International Pharmaceutical Abstracts (1967-2009). RCTs were included if subjects had consumed alcohol immediately prior to or during the study, were administered acetaminophen in therapeutic doses (up to 4 g daily), and compared ALT levels between placebo and treatment groups. Data were abstracted by two independent reviewers. The primary outcome was the change in ALT activity from baseline as compared to placebo using a fixed effects model. Secondary outcomes were reports of liver failure or death.

**Results:** The search resulted in 199 articles; including eight unique RCTs. Two RCTs did not compare ALT between placebo and treatment groups. Since the effects of acetaminophen on ALT are time-dependent, we compared ALT levels between placebo and treatment groups immediately prior to or during the study, were administered acetaminophen in therapeutic doses (up to 4 g daily), and compared ALT levels between placebo and treatment groups. Data were abstracted by two independent reviewers. The primary outcome was the change in ALT activity from baseline as compared to placebo using a fixed effects model. Secondary outcomes were reports of liver failure or death.

**Results:** The search resulted in 199 articles; including eight unique RCTs. Two RCTs did not compare ALT between placebo and treatment groups. Since the effects of acetaminophen on ALT are time-dependent, we compared ALT levels between placebo and treatment groups. Data were abstracted by two independent reviewers. The primary outcome was the change in ALT activity from baseline as compared to placebo using a fixed effects model. Secondary outcomes were reports of liver failure or death.

**Results:** The search resulted in 199 articles; including eight unique RCTs. Two RCTs did not compare ALT between placebo and treatment groups. Since the effects of acetaminophen on ALT are time-dependent, we compared ALT levels between placebo and treatment groups. Data were abstracted by two independent reviewers. The primary outcome was the change in ALT activity from baseline as compared to placebo using a fixed effects model. Secondary outcomes were reports of liver failure or death.

**Results:** The search resulted in 199 articles; including eight unique RCTs. Two RCTs did not compare ALT between placebo and treatment groups. Since the effects of acetaminophen on ALT are time-dependent, we compared ALT levels between placebo and treatment groups. Data were abstracted by two independent reviewers. The primary outcome was the change in ALT activity from baseline as compared to placebo using a fixed effects model. Secondary outcomes were reports of liver failure or death.

**Conclusion:** The available published RCTs suggest that acetaminophen at or near maximum recommended doses do not change serum ALT activity more
than placebo alone after four days in subjects who also consume alcohol. As our analysis was limited to study day 4, we cannot exclude that acetaminophen may affect the ALT activity of subjects who consume ethanol at other time points.

7. INTRASOSSEOUS HYDROXOCOBALAMIN VERSUS INTRAMUSCULAR HYDROXYLAMINE IN A VALIDATED SWINE MODEL OF ACUTE CYANIDE TOXICITY AND SHOCK

Vikkyat S. Beharta1, Julio Lairiet1, Patricia Dixon1, Susan Bourdreau1, Toni Vargas1, David Tanen2
1Clinical Research Division, Wilford Hall Medical Center, San Antonio TX USA; 2Naval Medical Center-San Diego, San Diego CA USA; 3Department of Emergency Medicine, Wilford Hall Medical Center, San Antonio TX USA

Background: Non-intravenous routes of cyanide (CN) antidotes are needed as an easily administered antidote for first responders and military troops. Our group has previously demonstrated intravenous (IV) hydroxocobalamin (HOC) is effective for acute CN induced shock, and intrasosseous (IO) HOC is as effective as IV HOC. Intramuscular (IM) hydroxylamine (HAX) has been used for CN induced respiratory failure, but its effectiveness for cyanide induced shock has not been reported.

Objective: To compare the return to baseline of mean arterial blood pressure (MAP) between 2 groups of swine in acute CN toxicity and treated with IO HOC or IM HAM. Secondary parameters studied included blood cyanide levels, lactate, pH, nitrotyrosine (nitric oxide marker) levels, and cerebral near infrared spectrometry (NIRS) oxygenation.

Methods: 24 swine (48-52 kg) were intubated, anesthetized, and instrumented (continuous MAP and cardiac output (CO) monitoring). CN was continuously infused until severe hypotension (50% of baseline MAP). Animals were randomly assigned to IO HOC (150 mg/kg) or IM HAM (50 mg/kg) and monitored for 60 min after return to baseline. A sample size of 12 animals per group was determined by group size analysis based on power of 80% to detect an effect size of a 0.54 difference (approx 1 stdev) of the mean MAP between the groups and an alpha of 0.05. MANCOVA was used to determine statistically significant changes between groups over time.

Results: Baseline mean weights (49, 50 kg), time to hypotension (29, 31 min), and CN dose at hypotension (5, 5 mg/kg) were similar between groups. At hypotension mean MAP (42, 42 mg Hg), blood CN (3.2, 2.9 mcg/ml) and lactate levels (7.4, 7.8 mmol/L) were similar. 12/12 animals in the IO HOC group and 9/12 in IM HAM group survived (p = 0.11). IO HOC resulted in a faster return to baseline (p<0.001). SVO2 and SVR were greater in the IO HOC group (p<0.002). CO was greater in the IM HAM group (p<0.003). Blood lactate, pH, and lactate, levels were similar. Methemoglobin (1.2% IO HOC, 12.8% IM HAM) and CN levels (0 in IO HOC, 15.5 mcg/ml in IM HAM) were greater in the IM HAM group at 60 min (p<0.001). Cerebral NIRS oxygenation decreased in parallel to MAP during CN infusion and was similar in both groups after antidote (p=0.78). Serum nitrotyrosine rose during CN infusion in all animals, but was lower in the IO HOC group at 60 min (p=0.03). TUNA, IL-1b, and IL-10 were similar.

Conclusions: Intrasosseous hydroxocobalamin led to a faster return to baseline mean arterial blood pressure compared to intramuscular hydroxylamine. Mortality with the intramuscular hydroxylamine group was greater but did not reach statistical significance.

8. CIRCULATING MICRORNA AS A NEW MARKER OF ACETAMINOPHEN POISONING IN HUMANS

James W. Dear1, Phillip Starkey Lewis2, Vivien Platt3, Kenneth J. Simpson3, Darren G. Craig1, Hugh Laverty2, Jonathan Mogg3, Chris Goldring2, Kevin Park2
1NPIS Edinburgh, Edinburgh UK; 2Liverpool University; 3Scottish Liver Transplant Unit, Edinburgh; 4Novartis

Background: New biomarkers of liver injury are required to refine the management of acetaminophen poisoning. MicroRNAs (miRNAs) are short non-coding RNA molecules that repress their own set of specific target messenger RNAs and regulate cellular protein output at the translational level. They potentially represent a new class of biomarkers which are stable and sensitive, and some of these molecules display a high degree of tissue-selective expression. Previous studies have demonstrated that two liver-enriched miRNAs (miR-122 and miR-192) are promising serum biomarkers of acetaminophen-induced liver injury in a mouse model. Here, we have examined these molecules in humans with acetaminophen poisoning.

Methods: In a cross-sectional study we measured a panel of miRNA species in serum from patients with acetaminophen-induced liver injury and compared the Results with established liver injury markers and miRNA from a control cohort, a non-acetaminophen liver injury cohort, a pancreatitis cohort and a chronic kidney disease (CKD) cohort.

Results: Levels of liver-enriched miRNAs were significantly higher (miR-122: 252-fold, P<0.0001, miR-192: 65-fold, P<0.0001) in the serum of patients with acetaminophen-induced liver injury (n=53) compared to a control cohort (n=25). Non-hepatic serum miRNA, miR-1 (enriched in heart tissue) showed no difference between the overdose and control cohorts whereas miR-218 (enriched in brain tissue) was slightly higher (3.5-fold, P=0.01). In a cohort of pancreatitis patients (n=25) miR-218 was the only miRNA to exhibit an increase (4.84-fold, P=0.0002). In a cohort of CKD patients (n=27), liver-enriched miRNAs showed only modest increases compared to healthy controls (miR-122: 1.73-fold, P=0.006, miR-192: 2.93-fold, P=0.005). miR-122 was significantly increased in non-acetaminophen induced liver injury patients (26.4-fold, P=0.0005).

In acetaminophen-induced liver injury our data show that serum miR-122 significantly correlates with peak ALT levels (r2=0.47; P<0.01) but does not correlate with prothrombin time or serum creatinine. Serum miR-122 levels were 96.3% higher in patients who satisfied King’s College Criteria but this was not statistically significant (P=0.15).

Conclusion: This work provides the first evidence for the use of miRNAs as novel biomarkers of acetaminophen poisoning in humans and provides a platform for further clinical development.

SESSION II

83. CLINICAL AND ANCILLARY DATA IN CHILDREN WITH BROWN RECLUSE SPIDER ENVENOMATION

Jennifer Lowry1, D. Adam Algren1, Heather Walters2, Gary Wasserman1
1Children’s Mercy Hospital, Kansas City MO USA; 2University of Missouri-School of Medicine, Kansas City MO USA

Background: Loxosceles spiders are known to have morbidity and occasionally mortality associated with their envenomations. Envenomations can result in both local and systemic findings, however this is largely based on case reports. We present the largest case series to date of brown recluse spider envenomation (BRS) in children.

Methods: IRB approval was obtained for this study. A retrospective chart review of patients hospitalized at a tertiary children’s hospital for a brown recluse spider envenomation between 2003 and 2007 was performed. Only cases confirmed by a medical toxicologist were included. Demographic, historical, physical exam and laboratory data were assessed.

Results: 64 children with brown recluse envenomations were identified (32 males; Age 7 mo ~ 18 years, mean 9.1 years; 31 Caucasian, 27 African American). The bite location was most commonly on the leg (24) and arm (15). Patients presented 1-10 days (mean 3.3, median 2.5) after the bite with the majority (54) not seeing the spider. Hemolysin (39 patients) occurred from 1-12 days post-bite (mean 4.6 days). The hemoglobin nadir in those who hemolyzed ranged from 4.6-12.9 grams.
Clinical Toxicology Downloaded from informahealthcare.com by University of Zuerich on 09/15/11

84. COMPARISON OF CITALOPRAM AND OTHER SELECTIVE SEROTONIN REUPTAKE INHIBITORS (SSRI) OVERDOSES IN CHILDREN

Wendy Klein-Schwartz1, Blaine Benson2, Toby Litovitz1, Samantha Lee3
1Maryland Poison Center, Baltimore MD USA; 2New Mexico Poison & Drug Information Center, Albuquerque NM USA; 3National Capital Poison Center, Washington DC USA; 4PharmD from the University of Maryland

Background: In adults, citalopram is more likely to cause seizures and ECG changes than other SSRIs. Data in children are lacking, yet the AAPCC out-of-hospital consensus guideline for citalopram is the same as for other SSRIs. The Objective of this study was to compare the clinical effects and hazard index of citalopram with other SSRIs to determine if children with citalopram overdoses experience more serious toxicity.

Methods: An 11 year retrospective review of AAPCC NPDS data was conducted. Inclusion criteria were age < 6 years with acute exposure to an SSRI followed to a known outcome. Cases were excluded for suicidal reason, chronicity other than acute, and unknown SSRI type. Clinical effects and hazard index (number of major or fatal outcomes per 1000 reported acute pediatric SSRI ingestions) were compared. Citalopram’s clinical effects were compared to non-citalopram SSRIs using Chi Square analyses.

Results: There were 35,297 cases that met inclusion criteria. Breakdown by SSRI was citalopram, 3,748; escitalopram, 4,815; fluoxetine, 5946; fluvoxamine, 273; paroxetine, 7,157; sertraline, 3,358. The hazard index for citalopram was approximately 6-fold higher than for non-citalopram SSRIs. Comparing clinical effects of citalopram with the other SSRIs, children with citalopram ingestions were more likely to develop conduction disturbances (p = 0.0182), other ECG changes (p = 0.0128), single seizures (p = 0.0240) and multiple, discrete seizures (p = 0.0001). These clinical effects occurred in children exposed to citalopram (N = 3,748) compared to other SSRIs (N = 31,549) as follows: conduction disturbances (5 vs 10), ECG change other (5 vs 9), single seizures (4 vs 7) and multiple, discrete seizures (5 vs 2). Clinical effects occurring more frequently with other SSRIs included tachycardia (p = 0.0174), oral irritation (p = 0.0243), vomiting (p = 0.0028), agitation/irritability (p = 0.0076), and hyperthermia (p = 0.0485).

Conclusion: Mirroring the data in suicidal adults, the rate of life-threatening or lethal outcomes was higher in pediatric citalopram ingestions compared to other SSRIs. Seizures and ECG changes, while uncommon, occur more frequently in children who ingest citalopram compared to other SSRIs.

86. ADVERSE EVENTS ASSOCIATED WITH THE USE OF UNAPPROVED PRESCRIPTION COUGH/COLD PRODUCTS IN CHILDREN

L. Carr1, T. H. Schaeffer, A. Bui, J. L. Green, R. C. Dart, Pediatric Cough/Cold Medication Surveillance Team
Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA

Background: The United States Food & Drug Administration (FDA) recently mandated that unapproved prescription cough/cold products be removed from the market. Though the active ingredients of some of these drugs are approved for use, some formulations have not been evaluated for safety, efficacy or quality and are illegally marketed. Despite this, health care professionals continue to prescribe them, likely unaware that they are not approved. We sought to describe adverse events (AEs) associated with exposures to unapproved products.

Methods: Active surveillance sponsored by the Consumer Healthcare Products Association captures AEs associated with over the counter (OTC) and prescription cough/cold medicine in children < 12 years of age. Cases are obtained from the FDA Adverse Event Reporting System, manufacturer reports, National Poison Data System, medical literature and news/media reports. A multi-disciplinary Pediatric Cough/Cold Medication Safety Surveillance Team reviews each case report to evaluate causality, determine dose, intent of administration, as well as a root cause analysis of adverse events. The database was searched for exposure to prescription cough/cold products from 01/01/08 to 09/30/10. These were cross referenced by product name/formulation to the list of unapproved products. Patient demographics, AEs, relatedness, indication for use and person administering the product were summarized.

Results: 46 (8%) of the 591 cases of AEs associated with prescription cough/cold products occurred after exposure to an unapproved product. No deaths
were reported. 33 (72%) of the 46 cases involved children<4 years of age. While 21 (46%) cases did not report an indication for use, 14 (30%) reported use for a cough/cold complaint; 11 (24%) accidentally ingested the drug. The drug was most commonly administered by the parent (n = 16, 35%); self-administration by the child was also reported (n = 13, 28%). There were 157 AE mentions, but many cases had more than 1 AE following product exposure. The most frequent AEs were ataxia (n = 14, %), somnolence (n = 13, 8%), and tachycardia (n = 13, 8%). The panel determined that 38 cases (83%) had AEs related to the product and 1 case (2%) where the AE was unrelated. Relatedness could not be determined in 7 cases (15%).

Conclusion: Though most marketed prescription cough/cold products are FDA approved, unapproved prescription products are utilized in the pediatric population and are associated with AEs. It is unlikely that these unapproved products would have been prescribed had the health care provider known the regulatory status. The reported adverse events are similar to expected events with approved cough/cold products.

SESSION III

161. USE OF A HEALTH INFORMATION DATA WAREHOUSE TO CONDUCT ACTIVE SURVEILLANCE AND TARGETED PROVIDER EDUCATION FOR AGRANULOCYTOSIS CAUSED BY COCAINE CONTAMINATED WITH LEVAMISOLE

J. A. Buchanan1, T. D Mackenzie2, A. Sabel3, E. J. Lavonas1
1Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA; 2Department of Patient Safety and Quality-Denver Health, Denver CO USA

Background: In December 2008, the first human cases of agranulocytosis caused by levamisole contamination of illicit cocaine were reported. Despite broad-based physician education, we observed a persistent problem with failure to correctly diagnose cases of this novel clinical entity in our hospital system. Therefore, we used our data warehouse to conduct active case surveillance and provide targeted education to providers in near-real time.

Methods: In phase I, we designed three search strategies for identifying levamisole-cocaine cases based on specific combinations of data elements in the inpatient or outpatient electronic medical record: neutropenia (<1,000 cells/mcl), total leukopenia (<3,000 cells/mcl), urine positive for cocaine metabolites within the previous 1095 days, and CDC-9 codes associated with cocaine or other illicit drug use within the previous 1095 days. These search strategies were applied retrospectively to our data warehouse. Potential cases were validated by expert clinician chart review using explicit definitions for confirmed, probable, possible, and unlikely levamisole-cocaine cases.

During phase II, we applied the search strategies to our data warehouse to prospectively identify newly-occurring cases in near real time. A toxicologist reviewed each case identified by the search and notified treating clinicians about the association between levamisole, cocaine, and agranulocytosis. After the levamisole-vasculitis syndrome was identified, a fourth search strategy was added to the protocol.

Results: In phase I, the three search strategies identified 153 records, of which 56 (36%) represented confirmed (n = 1), probable (n = 15), or possible (n = 40) levamisole-cocaine syndrome cases. In phase II, the search strategy identified 76 potential incident cases over 43 weeks, of which 26 (34%) were found to represent possible (n = 14) or probable levamisole toxicity (n = 12). In 11 (42%) of these cases, treating physicians had not considered the possibility of levamisole poisoning.

Conclusion: Active syndrome-specific surveillance of a hospital system data warehouse can be used to identify cases of a novel disease entity and direct targeted education to physicians in near-real time.

162. ADVERSE EFFECTS AND CONFIRMATORY TESTING OF JWH-018 AND JWH-073 FOLLOWING SYNTHETIC CANNABINOID USE

Keith R. McCain1, Jeffery H. Morlan1, Laura P. James2, Howell R. Foster1
1Public Health Laboratory, Arkansas Department of Health, Little Rock AR USA; 2Department of Pediatrics, University of Arkansas for Medical Sciences, Little Rock AR USA;

Background: Synthetic Cannabinoid (SC) products, marketed as “herbal incense”, are a mixture of organic plant matter laced with one or more synthetic compounds that mimic the psychoactive effects of tetrahydrocannabinol found in marijuana. Use of these “legal high” products markedly increased in 2010 with US poison centers recording over 4000 calls since early 2010. Very little is known about the pharmacology of these substances in man; however, a small number of case studies describe clinical effects atypical of marijuana, including agitation, hallucinations, and hypokalemia.

We report the clinical effects and medical treatment following use of SC products accompanied by confirmatory analytical determination of SC compounds and/or metabolites.

Methods: The Arkansas Poison and Drug Information Center (APDIC) utilized standard case management practice in combination with a SC template to record specific physical exam and laboratory findings. Additionally, APDIC provided information and follow-up to enhance proper storage and transport of urine specimens from health care facilities to the Arkansas Department of Health (ADH). ADH developed and validated a urine analytical assay to detect and quantify two common synthetic cannabinoids (JWH-018 and JWH-073) and corresponding metabolites.

Results: Between 03/2010 and 08/2010 nine exposures were documented with corresponding SC urine analysis performed. Urine assays were positive for either the glucuronide or non-conjugated metabolites of JWH-018 or JWH-073 for 6 of 9 submitted samples. Of the 6 patients with positive urine assays, the following clinical symptoms were recorded: tachycardia (6/6, 100%), hypokalemia, <3.5 mEq/L (4/4, 100%, not obtained on 2), confusion (4/6, 66.7%), agitation/irritability (3/6, 50%), hallucinations (2/6, 33.3%), pallor (2/6, 33.3%), nausea (2/6, 33.3%), vomiting (2/6, 33.3%), and mydriasis (2/6, 33.3%). Treatment included: benzodiazepines 4/6 (66.7%), intravenous fluids 2/6 (33.3%), antiepileptics 1/6 (17.7%), and potassium supplementation 1/6 (17.7%). One patient (17%) was admitted to a non-critical care unit and the remaining patients, 5/6 (83.3%), were treated, evaluated, and released from the emergency department. The duration of effects were > 8 but < 24 hours for 3/6 (50%), > 2 but < 8 hours for 2/6 (33.3%), and < 2 hours for 1/6 (17.7%). Moderate effect was coded for 5/6 (83.3%) and minor for one (17%).

Conclusions: SC products produce a wide range of adverse clinical effects. Confirmatory testing for JWH-018 and JWH-073 can facilitate our understanding of the adverse side effect profile of these illicit drugs of abuse.

163. FROM CONVENIENCE STORES TO SCHEDULE I IN LESS THAN 100 DAYS

Mark L. Ryan1, Thomas C. Arnold2
1Louisiana Poison Center, Shreveport LA USA; 2LSU Health Sciences Center, Shreveport LA USA

Background: Products containing various synthetic cathinones appeared in European countries in the mid 2000’s. These products were labeled as bath salts, plant food, stain remover, and pond scour remover. Abuse of these products eventually led to bans in several European countries. In 2010 the first cases associated with these products were reported to U.S. Poison Centers.

Discussion: The first case of exposure to a bath salts cathinone containing product in Louisiana was reported in September 2010. Additional cases were reported in November and the Louisiana Department of Health and Hospitals was notified at that time of this emerging threat to public health.

Methods: The anomaly alert capabilities of the National Poison Data System (NPDS) were used to provide notification to health officials as new bath salts cases were received. A tremendous spike in the number of cases occurred in December and the severity of the effects associated with exposure to these substances was, in many cases, alarming.

Results: After consultation with Poison Center toxicologists state health officials put in place an emergency order on January 6, 2011 placing six synthetic cathinones into Schedule I. The substances included in the ban are 3,4-methylenedioxymethylcathinone (methylenedioxymethylcathinone), 3,4-methylenedioxymethylcathinone (MDPV), 4-methylmethcathinone (mephedrone), 4-methoxymethcathinone, 3-fluoromethcathinone, and 4-fluoromethcathinone.

Conclusion: Close collaboration between the Louisiana Poison Center and the Louisiana Department of Health and Hospitals resulted in notification of this
emerging threat shortly after the initial cases were reported. Frequent interaction kept health officials aware of the scope and severity of the problem. The information provided by the Poison Center helped officials in their decision to move quickly to ban these substances.

164. OUTPATIENT CLINIC EXPERIENCE OF SYMPTOMATIC PATIENTS WITH METAL-ON-METAL HIP IMPLANTS

Harry C. Karydes1, Patrick M. Whiteley1, Brandon K. Wills2, Kirk L. Cumpston2, Jerrold B. Leikin1
1Cook County Hospital (Struger), Chicago IL USA; 2Virginia Commonwealth University Medical Center, Richmond VA USA; 3NorthShore University HealthSystem-OMEGA, Glenview IL USA

Background: The increasing number of early failure of metal-on-metal (MoM) hip arthroplasties and possible exposure to orthopedic metal alloys has caused concern for many patients (and toxicologists) in regards to short and long term biologic effects. We sought to characterize our outpatient clinical experience of symptomatic patients with MoM implants and associated serum/blood chromium and cobalt levels.

Methods: This was a consecutive series of patients presenting to two outpatient medical toxicology clinics from January 1, 2010-March 31, 2011 with history of MoM hip implants. Presenting signs, symptoms, and interventions were reviewed. Available cobalt and chromium levels were summarized as median concentration with interquartile range (IQR).

Results: A total of 20 patients were analyzed. The median cobalt concentration (normal <0.9 ng/mL) was 5 ng/mL (IQR 2.2-34) and median chromium concentration (normal <0.3 ng/mL) was 4 ng/mL (IQR 1.18-28) in 19 patients. Seventeen (85%) had pain described as hip or thigh pain, clicking sound with ambulation and/or grinding with activity. Two (10%) patients had ataxia and later diagnosed with demyelination neuropathy with one patient demonstrating marked improvement after revision. Finally, 9 (45%) patients had replacement or revision of their hip implant. Of these patients, 6 (30%) had pre- and post-procedure cobalt and chromium concentrations. The median pre- and post-procedure cobalt concentrations were 68.6 ng/mL (IQR 3.82-235) and 6.55 ng/mL (IQR 1.2-7) respectively. The median pre- and post-procedure chromium concentrations were 22.5 ng/mL (IQR 3.8-91) and 17.5 ng/mL (IQR 1.29-1) respectively.

Discussion: There are limited data on the systemic effects of metal alloys found in these implants. Patients with MoM implants frequently have localized signs and symptoms but may have other organ systems affected. The potential human risk and/or toxicity that may be associated with MoM implants should be considered. The decision for hip revision solely for toxicologic reasons is rare and usually involves a multidisciplinary approach.

Conclusion: This series suggests that MoM arthroplasty can result in elevated cobalt and chromium concentrations. Patients frequently have local signs and symptoms, however, systemic toxicity, possibly neurologic, should be considered. Future studies will be needed to determine whether a causal relationship exists between MoM arthroplasty and chronic systemic toxicity.

SESSION IV

165. PROSPECTIVE ASSESSMENT OF THE HEALTH EFFECTS OF DIETHYLENE GLYCOL EXPOSURE AMONG PERSONS IN PANAMA (2007-2008)

Laura Conklin1, James J. Sejvar1, Raquel Sabogal1, Carlos A. Sanchez1, Stephanie Kieszak1, Bertram Ackermann1, Rosemarie Jovane2, Gerardo Victoria2, Felicia Tulloch2, Joshua G. Schier1
1CDC, Atlanta GA USA; 2Santo Tomas Hospital, Panama City Panama

Background: In 2006, a cough medication unintentionally contaminated with diethylene glycol (DEG) was distributed to more than 30,000 people in Panama causing more than 100 deaths. DEG is an industrial chemical that can cause neurological and renal toxicity when ingested. The Centers for Disease Control and Prevention (CDC) assisted the Panama Ministry of Health in a prospective assessment of health effects among persons who ingested the syrup and survived. The primary Objective was to characterize the long term health effects and symptom evolution after acute DEG poisoning.

Methods: Inclusion criteria required a history of syrup exposure and elevated serum creatinine (SCR) concentration (>1.5 mg/dL) after July 1, 2006 and hospitalization due to DEG poisoning. We documented health effects at six month intervals from January 2007 through July 2008. At each evaluation, participants underwent SCr testing, neurological examination, and nerve conduction study (NCS) testing. Median SCr concentration was determined for non-dialysis-dependent patients. Neurological function and NCS results were classified into one of four categories at each assessment and for the overall study period (summary determination of all 4 assessments combined) as: normal, slightly abnormal, moderately abnormal, or moderately-severe to severely abnormal.

Results: Thirty-three persons met inclusion criteria. Median SCr concentrations of non-dialysis dependent patients decreased slightly from 1.8 (range, 0.9-5.9 mg/dL) to 1.6 (range, 0.7-5.2 mg/dL). The proportion of patients with elevated SCr concentrations or dialysis dependence decreased from 27/32 (84.4%) in January, 2007 to 20/27 (74.1%) in July, 2008. The most common neurological finding at all four assessments was abnormal lower extremity reflexes. Overall study period determinations for neurological function were as follows: 7/33 participants (21.2%), normal; 5/33 (15.1%), slightly abnormal; 11/33 (33.3%), moderately abnormal; and 10/33 (30.3%) as moderately severe or severely abnormal. Overall study period NCS outcome determinations were as follows: 4/33 (12.1%), normal; 5/33 (15.2%), slightly abnormal; 10/33 (30.3%), moderately abnormal; and 14/33, (42.4%) were moderately-severe to severely abnormal. DEG poisoning was not associated with delayed-onset renal or neurological illness.

Conclusions: Renal function among patients showed some improvement over the study period. The majority of patients had evidence of at least moderately abnormal neurological function and NCS findings for the study period. This represents the largest and most extensive investigation of survivors of a DEG mass poisoning.

166. ANALYSIS OF DIETHYLENE GLYCOL (DEG) AND SUSPECTED METABOLITES IN PATIENTS FROM THE 2006 PANAMA DEG MASS POISONING

Joshua G. Schier1, Danielle R. Hunt1, Mark J. Filary1, Adam W. Peralta1, Kenneth E. McMartin1, Michael J. Bartels1, Dana Flanders1
1CDC, Atlanta GA USA; 2Dow Chemical Company, Midland MI USA; 3Louisiana State University Health Sciences Center, Shreveport LA USA

Introduction: In 2006, an outbreak of renal and neurological illness occurred in Panama. The Centers for Disease Control and Prevention (CDC) assisted the Panama Ministry of Health in a case-control study to identify the cause of illness: diethylene glycol (DEG) contaminated cough syrup. As part of the investigation, serum and urine specimens were collected, then shipped to and stored at CDC. The primary Objectives of this study were to determine the detection frequency of specific analytes and the association between case status and presence of DEG and suspected metabolites, including ethylene glycol (EG), glycolic acid (GA), 2-hydroxyethoxyacetic acid (HEA), oxalic acid (OA), and diglycolic acid (DGA), in serum and urine samples.

Methods: Serum and urine samples previously collected and stored were analyzed. Cases were persons with acute renal failure of unknown etiology and serum creatinine concentration >2.0 mg/dL (normal, <1.5 mg/dL). Controls were randomly selected from patients hospitalized for causes other than renal failure and were matched to cases based on age (± 5 years), sex, and admission date (<2 days before case). The ratio of controls to cases for this study was 1:1 for serum and 2:1 for urine (due to limited specimen). All analytes were measured using GC/MS. Detection frequencies were determined for all analytes. Conditional logistic regression and Fisher’s exact test in SAS v9.2 was used to calculate the odds of analyte detection in cases as compared to controls. Analyte concentrations below the lowest level of quantitation were coded as non-detectable.

Results: Twenty case and 20 control serum samples were analyzed. Eleven case and 22 control urine samples were analyzed. Urinary GA, HEAA and DGA concentrations were not measurable for one case. Urinary OA concentrations were not measurable. Detection frequencies for cases and controls respectively in serum were: DEG, 11/20 (55%) and 6/20 (30%); EG, 1/20 (5%) and 0/20; GA, both 20/20 (100%); OA, both 20/20 (100%); HEAA, 20/20 (100%) and 18/20 (90%); and DGA, 20/20 (100%) and 0/20. Detection frequencies for cases and controls respectively in urine were: DEG, 1/11 and 2/22 (9%); EG,
Clinical Toxicology Downloaded from informahealthcare.com by University of Zuerich on 09/15/11
For personal use only.

176. DIGLYCOLIC ACID IS THE NEPHROTOXIC METABOLITE OF DIETHYLENE GLYCOL

Greg M. Landry1, Lauren M. Besenhofer1, Michael Bartels2, Adam Peralta2, Mark Filary2, Kenneth E. McMartin1
1LSU Health Sciences Center, Shreveport LA USA; 2 Dow Chemical Company, Midland MI USA

Introduction: The hallmark of diethylene glycol (DEG) poisoning is an acute renal failure that recent studies have convincing shown must result from a metabolite of DEG. Which of the two primary DEG metabolites, hydroxyethoxyacetic acid (HEAA) and diglycolic acid (DGA), is the responsible toxicant has yet to be determined. Therefore in vivo and in vitro studies have been conducted to assess the respective roles of the two metabolites.

Methods: Wistar rats were treated in four groups: control, low dose DEG (2 g/kg), high dose DEG (10 g/kg), or high dose DEG + fomepizole. Blood and tissue samples were collected at times up to 48 h and analyzed for the concentration of the various DEG metabolites, as well as markers of kidney toxicity. Also, studies were conducted in cultured human proximal tubule cells that were incubated with DEG, HEAA or DGA for up to 48 h. Cytoxicity was assessed using multiple measures of necrosis and of apoptosis.

Results: In the high dose animals, blood HEAA levels peaked at 8 h at 4.2 mmol/L, while blood DGA levels at 24 h were 0.04 mmol/L. However in the same animals, the kidney concentrations of DGA (4-5 mmol/L) were 100-fold higher than blood concentrations, indicating a substantial concentrated uptake of DGA by kidney tissue. Kidney HEAA levels were the same as in the blood, ~4 mmol/L. Kidney levels of both HEAA and DGA correlated strongly with increases in BUN and plasma creatinine. In the in vitro studies, neither DEG nor HEAA at up to 100 mmol/L induced any necrosis or apoptosis, while DGA at >25 mmol/L induced a marked degree of necrotic cell death, which was preceded by a depletion of ATP. DGA did not appear to produce apoptosis (only late apoptosis/necrosis) at up to 100 mmol/L. Co-treatment with combined metabolites or with DEG did not exacerbate the toxicity produced by DGA. Co-treatment with dicarboxylic transporter inhibitors reduced the toxicity of DGA, suggesting that kidney cell uptake of DGA was necessary for its toxicity.

Conclusions: These studies indicate that DGA is the likely toxic metabolite that produces the widespread, time and dose dependent cortical necrosis observed in DEG poisonings. This project was supported by the American Chemistry Council.

168. ALUMINUM CITRATE DECREASES THE RENAL INJURY INDUCED BY ETHYLENE GLYCOL POISONING

Lauren M. Besenhofer, Marie C. McLaren, Kenneth E. McMartin
LSU Health Science Center, Shreveport LA USA

Introduction: Ethylene glycol (EG) poisoning can produce an acute renal failure that, in many cases, is treatable only with long-term hemodialysis. Renal accumulation of calcium oxalate monohydrate (COM) crystals is responsible for the kidney injury associated with EG exposures. In cases where anti-metabolite therapy is delayed (due to late admissions or diagnostic problems), long-term kidney injury is common. COM accumulation is also responsible for the renal and systemic damage associated with primary hyperoxaluria and is also a major contributor to the development of kidney stones. Current treatments for these conditions do not directly address the mechanism of toxicity (accumulation of COM). Our in vitro studies have shown that aluminum citrate blocks the toxicity in human proximal tubule cells by preventing COM attachment and internalization. This effect represents a unique molecular target, not reproduced by citrate salts already used for stone therapy. Thus, this study was designed to evaluate the efficacy and mechanism of action of aluminum citrate in a rat model of EG poisoning.

Methods: Wistar rats were treated with either control (water at time 0), EG (6 g/kg, gavaged at time 0), or EG + aluminum citrate (0.2 mmol/kg, given IV at 2, 6, 12, 18, 24, 30, 36, 42, 48, 54, 60 and 66 h, with saline for controls at the same times). Urines (every 12 h up to 72 h) and plasmas (every 24 h) were analyzed for markers of kidney injury and levels of oxalate and calcium. Kidney tissue was obtained at 72 h for analysis of damage and of COM retention.

Results: BUN and plasma creatinine levels were increased by EG treatment and this effect was blocked by the co-treatment with aluminum citrate. Urine samples were centrifuged to separate soluble components (ionic calcium and oxalate) from insoluble oxalates like COM. Urinary excretion of insoluble oxalate and insoluble calcium was significantly increased by aluminum citrate, while soluble calcium and oxalate were reduced by aluminum citrate, indicating enhanced excretion of calcium oxalate as crystalline material. The amount of COM crystals in kidney tissues was markedly reduced by aluminum citrate.

Conclusions: These results suggest that aluminum citrate could decrease COM-induced renal injury by decreasing COM retention in the kidney, thus enhancing the excretion of both calcium and oxalate. This study also shows that a compound that works by the same mechanism as aluminum citrate, i.e. blocking COM retention by kidney cells, would be a useful treatment for the kidney damage produced by EG and might be useful in other diseases involving high levels of oxalate. This project was supported by the American Chemistry Council.

Table 1.

<table>
<thead>
<tr>
<th>Product name</th>
<th>Drug Found</th>
<th>Labeled “use”</th>
<th>Physical Appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>White Lightening</td>
<td>methedrone</td>
<td>Insect repellent</td>
<td>White dry powder</td>
</tr>
<tr>
<td>Zoom</td>
<td>MDPV</td>
<td>Natural stain remover</td>
<td>White dry powder</td>
</tr>
<tr>
<td>Bayou Ivory</td>
<td>Methylene and MDPV</td>
<td>Potpourri</td>
<td>Beige powder</td>
</tr>
<tr>
<td>Cloud 9</td>
<td>Methylene and MDPV</td>
<td>Bath Salt</td>
<td>Beige Powder</td>
</tr>
<tr>
<td>Cloud 10</td>
<td>MDPV</td>
<td>Bath Salt</td>
<td>Beige Powder</td>
</tr>
<tr>
<td>White China</td>
<td>Methylene and MDPV</td>
<td>Bath Salt</td>
<td>Beige Powder</td>
</tr>
</tbody>
</table>

9. WHAT IS IN “BATH SALTS”?

Henry A. Spiller1, Robert G. Weston2, Mark L. Ryan3
1Kentucky Regional Poison Center, Louisville KY USA; 2Oklahoma State Bureau of Investigation, Edmond OK USA; 3Louisiana Poison Center, Shreveport LA USA

Background: In 2010 a new group of illicit substances of abuse began to be sold in a number of locations, including gas stations, smoke shops, “head shops” and internet sites. The products were labeled as bath salts, insect repellants, stain removers and plant food. However the users of these products openly spoke of...
using these products as “legal methamphetamine” or “legal cocaine”. The product labels did not provide any indication of the true active ingredients. More than 600 cases of patients using these products have been reported to poison centers and emergency departments.

**Method:** we obtained 14 different products from multiple stores in Louisiana and Kentucky and analyzed them for content. Analysis was by GC/MS.

**Results:** See Table 1 for Results. The products contained either 4-methylmethcathinone (Mephedrone), Methylenedioxypyrovalerone (MDPV) or, 4-methylenedioxy-N-methylcathinone (methyleone). Additional substances found included: caffeine and an unidentified substance.

**Discussion:** all products contained b-keto compound analogs of phenylalkylamines. This is the first report to document the contents of this new group of illicit products.

**Conclusions:** Products that are being marketed as “bath salts” contain compounds that have the potential to cause significant toxicity. Ingredients are not listed on the package and can vary even in products with the same name.

## 10. SURREPTITIOUS FOOD POISONING WITH PHENCYCLIDINE

Cyrus Rangan

Los Angeles County Department of Public Health, Los Angeles CA USA

**Background:** PCP is a well known drug of abuse. A cluster of patients with neurological symptoms after a meal prompted evaluation of a chemical etiology.

**Case Report:** 3 family members ate a meal of meatloaf, vegetables, and beverages. An 81-year-old female (Pt #1), 55-year-old male (Pt #2), and 62-year-old male (Pt #3) were transported to 3 different hospitals. All 3 had nausea/vomiting within one hour after eating meatloaf, followed by dizziness, confusion, ataxia, diaphoresis, and slurred speech, and were observed by other members of the family. Symptoms were most severe in Pt #1. No headache, neck pain, chest pain, dyspnea, diarrhea, or ethanol consumption were noted. Head CT were normal. Naloxone was administered in all 3 cases without change in status. CXR, CBC, electrolytes, troponin, lactic acid, APAP, ASA, ammonia were normal or negative. Pt #1 was noted to have somnolence, speech “minimal”, and slurred, with “pinpoint” pupils and hypoactive bowel sounds. She answered questions poorly, with declining mental status during ED course; hyporeflexia, with flaccid tone. She progressed to somnolence and respiratory depression, requiring intubation. Given activated charcoal 1 gm/kg via NG for suspected toxic exposure. She remained hospitalized for several days, and exubated successfully after day 2. Pt #2: had lethargy/somnolence, slurred speech, “small” pupils, and was oriented to name, place, but not time. Follows commands, but somewhat sluggish and delayed secondary to somnolence. No focal deficits. Mental status improved throughout ED course, and was discharged after overnight observation. Pt #3 presented tired, but answering questions readily with small and reactive pupils. The well known Type I (distal) renal tubular acidosis leads to this hypokalemia, hypomagnesemia, hypocalcemia, and hypophosphatemia. A 50 year review of the English medical literature identified 16 publications involving toluene abuse and hypokalemia, though only three had associated respiratory failure. The fact that this patient developed respiratory failure after the initiation of potassium replacement is crucial knowledge for the physicians caring for this type of patient. The concomitant hypophosphatemia likely contributed significantly to her muscle weakness.

**Conclusions:** This case of chronic toluene abuse resulting in delayed respiratory failure requiring mechanical ventilation identifies an area to improve patient outcome. It is recommended that aggressive replacement of potassium and phosphorous occurs in these patients in a critical care setting given this risk of delayed respiratory failure requiring mechanical ventilation.

## 12. CONTROL OF MEPHEDRONE (4-METHYLMETHCATHINONE) IN THE UK APPEARS EFFECTIVE IN REDUCING PRESENTATIONS TO THE EMERGENCY DEPARTMENT WITH ACUTE TOXICITY RELATED TO ITS USE

David M. Wood, Shaun L. Greene, Paul I. Dargan

Guy’s and St Thomas’ NHS Foundation Trust, London UK

**Background:** There has been increasing evidence of the acute harms associated with the recreational use of synthetic cathinones, such as mephedrone (4-methylmethcathinone). In the UK there was evidence of increasing numbers of individuals presenting to the ED with acute toxicity related to the self-reported use of mephedrone. As a result of this, mephedrone and the other cathinones were controlled under the UK Misuse of Drugs Act, 1971 on the 16th April 2010. We report the frequency of presentations to our ED with toxicity associated with the self-reported use of mephedrone pre- and post-the UK control of the cathinones.

**Methods:** Data on all presentations with acute recreational drug toxicity is related to its use.

**Table 1.**

<table>
<thead>
<tr>
<th>Dates</th>
<th>Pre/Post Control</th>
<th>Number of ED presentations</th>
</tr>
</thead>
<tbody>
<tr>
<td>16th Aug 2009 to 15th Oct 2009</td>
<td>Pre</td>
<td>7</td>
</tr>
<tr>
<td>16th Oct 2009 to 15th Dec 2009</td>
<td>Pre</td>
<td>5</td>
</tr>
<tr>
<td>16th Dec 2009 to 15th Feb 2010</td>
<td>Pre</td>
<td>14</td>
</tr>
<tr>
<td>16th Feb 2010 to 15th Apr 2010</td>
<td>Pre</td>
<td>31</td>
</tr>
<tr>
<td>16th Apr 2010 to 15th Jun 2010</td>
<td>Post</td>
<td>23</td>
</tr>
<tr>
<td>16th Jun 2010 to 15th Aug 2010</td>
<td>Post</td>
<td>14</td>
</tr>
<tr>
<td>16th Aug 2010 to 15th Oct 2010</td>
<td>Post</td>
<td>6</td>
</tr>
<tr>
<td>16th Oct 2010 to 15th Dec 2010</td>
<td>Post</td>
<td>4</td>
</tr>
</tbody>
</table>
toxicology database. We retrospectively interrogated this database to identify all presentations relating to self-reported mephedrone use in the 8 months prior to (16th August 2009 to 15th April 2010) and 8 months post (16th April to 15th December 2010) the control of mephedrone under the UK Misuse of Drugs Act, 1971.

Results: There were 57 presentations to our ED in the eight months prior to and 47 presentations in the 8 months after control of mephedrone in the UK. The frequency of presentations in two month intervals prior to and post control is shown in the Table 1, showing that the presentations peaked in the two months prior to control and then fell significantly in the months after control.

Conclusions: This study appears to suggest that the control of mephedrone was associated with a reduction in ED presentations with acute toxicity related to its use. There are potentially multiple reasons behind this reduction in ED presentations. These include an overall reduction in its use through personal choice or reduction in availability due to the change in its legal status, leading to use of other classical or novel psychoactive substances. Additionally, as others have reported since its control the purity of mephedrone has decreased, this reduction in ED presentations may reflect that individuals are being exposed less mephedrone. Further work is needed to clearly understand the effects, and potential risks, of control of novel psychoactive substances to ensure that simple legislative changes do not potentially increase the risk of harm.

13. CASE SERIES OF BATH SALT EXPOSURES WITH BLOOD AND URINE QUANTIFICATION

Henry A. Spiller1, Mark L. Ryan2, Robert G. Weston3, Joanne Jansen2, Thomas Arnold2

1Kentucky Regional Poison Control Center, Louisville KY USA;
2Louisiana Poison Center, Shreveport LA USA; 3Okahoma State Bureau of Investigation, Edmond OK USA; 4Sullivan University College of Pharmacy, Louisville KY USA

Background: rapid emergence of a new drug of abuse referred to as bath salts recently occurred. Analysis of powder samples from a number of these products detected B-keto analogs of phenylalkylamines: methylene, mephedrone and/or methylenedioxyprovalerone (MDPV).

Method: retrospective chart review of all cases reported to 2 poison centers involving bath salts over a 6 month period.

Results: There were 236 patients of which 184 (78%) were male. Age range was 16 to 64 years (Mean 29 years, SD 9.4). All cases were intentional abuse. There were 37 separate “brand” names identified. Clinical effects were primarily neurological and cardiovascular and included: agitation (n = 194), combative behavior (n = 134), tachycardia (n = 132), delusional hallucinations (n = 94), paranoia (n = 86), confusion (n = 83), chest pain (n = 40), myoclonus (n = 45), hypertension (n = 41), mydriasis (n = 31), CPK elevations (n = 18), hypokalemia (n = 10), blurred vision (n = 7). Severe medical outcomes included: death (n = 1), Major (n = 8) and moderate (n = 130). Therapies included bezodiazepines (n = 125), antipsychotics (n = 47) and propofol (n = 10). Primary dispositions of patients were: 116 (49%) treated and released from ED, 50 (21%) admitted to critical care, 29 (12%) admitted to psych and 28 (12%) lost to FU. Eighteen patients had blood and/or urine analyzed using GC/MS. MDPV was detected in 13 of 17 patients (range 24 to 241 ng/ml, mean 58 ng/ml). The 4 samples with no drug detected reported last use of bath salts > 20 hours prior to presentation. 3 of 5 patients had MDPV detected in urine (range 34 to 1386 ng/ml, mean 856 ng/ml). No mephedrone or methylone was detected in any sample.

Discussion: This is the first report of MDPV exposures with quantitative blood and urine quantification. Prominent clinical effects from the cathinone analogs included: psychotic episodes often requiring sedation, myoclonus and tachycardia. The single fatality was from a self-inflicted gunshot during an active delusional episode. Limited information is available on the cathinone analogs suggesting: 1) increased central dopamine levels without 5HT involvement and 2) direct inhibition of dopamine and norepinephrine transporters. Reported effects in humans of new onset psychosis, myoclonus and tachycardia support a dopaminergic mechanism of toxicity in humans.

Conclusion: Use of designer drugs continues to pose serious risk with previously unreported effects. We report the first quantitative levels after MDPV abuse.

14. SELF-ADMINISTERED ALCOHOL ENEMA CAUSING DEATH: A CASE REPORT

Landen L. Rentmeester, Stephen D. Cohle, Bryan S. Judge, Jeffrey S. Jones

GRM/PSU Program in Emergency Medicine, Grand Rapids MI USA

Background: The term klismaphilia refers to the receiving of sexual arousal from introducing liquids into the rectum and colon via the anus. This case describes an individual with klismaphilia whose death resulted from acute alcohol intoxication by rectal absorption of a wine enema. To our knowledge, only one other fatality due to the rectal absorption of alcohol has been reported in the English literature.

Case Report: A 52-year-old man with a history of hypertension was found unresponsive in his home by a coworker, when he did not show up for work. EMS was summoned to the scene and found the patient in full rigor with fixed livor mortis. Assystole was documented and the patient was pronounced dead. An enema bag was hanging from a coat rack next to the bed and contained a yellow colored fluid. A tube was connected to the enema bag and the other end was inserted into the patient’s rectum. The patient was wearing a condom, women’s underwear, and numerous erotic materials were found throughout the room. Several empty boxes of white wine were also noted during the scene investigation. An autopsy demonstrated fatty liver disease consistent with chronic alcoholism. Significant pulmonary congestion and edema were also described and consistent with ethanol induced respiratory depression. Approximately 500 mL of wine was found in the colon. Toxicology showed an ethanol concentration of 350 mg/dL in the blood and 410 mg/dL in the vitreous fluid. The cause of death was respiratory depression due to acute alcohol intoxication.

Discussion: Forensic pathologists and toxicologists are very familiar with deaths due to alcohol intoxication. The overwhelming majority of these deaths are a result of the oral ingestion of ethanol. Rectal absorption of alcohol bypasses the first pass metabolic effect, allowing for a higher concentration of blood alcohol to occur for a given volume of solution, and consequently, greater potential for central nervous system depression. However, fatalities are extremely rare. Eight previous reports of illness following self-administration of alcohol enemas describe symptoms of severe clinical colitis (e.g., abdominal pain and rectal bleeding). Despite the severity of injury and the clinical features, all reported patients recovered completely in 7 to 10 days with conservative management.

Conclusions: The use of enemas as a source of sexual gratification (klismaphilia) was first described in the medical literature in 1973. Despite the large variety of introduced substances, alcohol enemas have not been previously described in many of these cases. Most patients present with symptoms of severe colitis, however rapid absorption of alcohol through the colonic mucosa can lead to CNS depression and death.

15. COMPARISON OF TOXICITY OF NONMEDICAL USE OF BUPRENORPHINE AND METHADONE

Samantha Lee, Suzanne Doyon, Wendy Klein-Schwartz

Maryland Poison Center, Baltimore MD, USA

Background: Buprenorphine and methadone are used as substitution therapy for opioid dependence and in the management of pain. Patients treated with these medications are at high risk for nonmedical use such as self-medication, misuse and abuse. Studies comparing buprenorphine and methadone toxicity generally include intentional suicidal overdoses, contain reported co-ingestants or focus on fatalities but none focus on nonmedical use. The Objective of this study is to identify and compare the clinical severity of buprenorphine and methadone toxicity resulting from nonmedical use as well as treatment and medical outcomes.

Methods: Retrospective analysis of buprenorphine and methadone cases reported to a poison center from November 2002 to August 2010. Inclusion criteria were age ≥ 18 years, nonmedical use of either buprenorphine or methadone as single substance exposure, and followed to known outcome. Nonmedical use is defined as intentional misuse or abuse.

Results: Thirty-two buprenorphine and 98 methadone cases met inclusion criteria. Frequently reported clinical effects were lethargy 37.4% vs. 64.5%,...
coma 6.3% vs. 49.0%, respiratory depression 9.4% vs. 39.8%, vomiting 25% vs. 71.2% and agitation/irritation 21.9% vs. 7.1% for buprenorphine and methadone, respectively. One methadone patient died. There were significant differences between buprenorphine and methadone cases in distribution of clinical effects, level of care and medical outcomes as well as use of naloxone, antiemetics and benzodiazepines (p < 0.001). Of the patients who received naloxone, 2 (6.3%) patients in the buprenorphine group and 34 (34.7%) patients in the methadone group received multiple doses of naloxone or naloxone infusions. The Table 1 compares levels of care, outcomes and specific treatments.

Conclusions: Nonmedical use of buprenorphine has a better safety profile in adults resulting in no major outcomes or death, no intubation/ventilation, no admission to critical care units and less naloxone use when compared to methadone. Based on clinical effects and treatments the primary toxicity with nonmedical use of buprenorphine is precipitation of opioid withdrawal compared to overdose related toxicity with methadone.

16. A CHARACTERIZATION OF SPICE EXPOSURES REPORTED TO THE NPDS IN 2010

C. Hoyte, J. Jacob, A. A. Monte, M. Al Jumaan, A. C. Bronstein, K. J. Heard
Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA

Introduction: THC homologs commonly referred to as “spice” have been increasingly abused since their introduction in 2004. In 2010, the products were packaged and sold as incense in the US and used as a “legal high” for those wishing to experience cannabinoid effects while evading basic drugs-of-abuse testing. Several cases have been reported in the literature though no systematic study has yet been performed. Our Objective is to describe the cohort of spice exposures reported to the National Poison Data System (NPDS) in order to better characterize the clinical effects in these patients.

Methods: All THC homolog exposures reported to the National Poison Data System (NPDS) between January 1st, 2010 and October 1st, 2010 were extracted using NPDS generic codes and product codes for THC homologs and Spice products. Only cases involving a single agent exposure with THC homologs as the major category were analyzed. Descriptive statistics were generated for demographic data, management site, products involved, symptoms, duration of effects, treatments, and medical outcomes, as defined by the NPDS.

Results: Over the 9-month study period there were 1,898 exposures to THC homologs. 1,353 of these cases were single agent exposures. The mean age was 22.5 years (SD 8.86). Most cases were reported in males (n = 1,005, 74.3%). The majority were coded as THC homologs (60.0%, n = 816) and the most common specific product was K2 herbal blend (33.4%, n = 452). The vast majority of exposures were acute (88.2%, n = 1,193) with the intent for intentional abuse (93.3%, n = 1,262). 1,207 of the cases were managed at a health care facility (89.2%). The most common clinical effects were tachycardia (40.0%, n = 541), agitation/irritability (23.4%, n = 317), and vomiting (15.3%, n = 207). Seizures occurred in 54 patients (4.0%). The majority of clinical effects lasted for less than 8 hours (n = 711, 78.4%). 246 (18.2%) received IV fluids, 217 (16%) received benzodiazepines, 65 (4.8%) received oxygen, and 48 (3.5%) received antiemetics. 1,088 of the 1,353 cases were followed to completion and a final medical outcome was available. Of these there were 42 with no effects (3.9%), 467 minor outcomes (42.9%), 544 moderate outcomes (50.0%), 34 major outcomes (3.1%), and 1 death (0.1%). The clinical effects were coded as “unknown if related” in the one death.

Conclusions: Many exposures to THC homologs were reported to the NPDS throughout 2010. The majority of cases were in young males intentionally abusing the agent. Most exposures resulted in minor or moderate outcomes not requiring treatment. These findings may not be generalizable to cohorts not reported to the NPDS.

18. SKIN NECROSIS ASSOCIATED WITH EXPOSURE TO COCAINE CONTAMINATED WITH LEVAMISOLE: AN EMERGING PHENOMENON

Lydia Baltarowich, Adam Schlichting, Charles Leder, Adrian Ormsby
Henry Ford Hospital, Detroit MI USA

Background: Purpura and skin necrosis have been reported in users of cocaine adulterated with levamisole (LV), a veterinary antihelminthic, which has been found in 69% of cocaine seized coming into the US. We describe a rapidly progressing skin necrosis in three patients associated with levamisole levels, histology findings, and autoimmune serology.

Table 1.

<table>
<thead>
<tr>
<th></th>
<th>Buprenorphine (n = 32) No. (%)</th>
<th>Methadone (n = 98) No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Admission for</td>
<td>4 (12.5)</td>
<td>60 (61.2)</td>
</tr>
<tr>
<td>medical care</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Admission to critical</td>
<td>0 (0)</td>
<td>37 (37.8)</td>
</tr>
<tr>
<td>care (ICU)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No effect/minor effect</td>
<td>21 (65.6)</td>
<td>25 (25.5)</td>
</tr>
<tr>
<td>Moderate effect</td>
<td>11 (34.3)</td>
<td>61 (62.2)</td>
</tr>
<tr>
<td>Major effect/Death</td>
<td>0 (0)</td>
<td>12 (12.2)</td>
</tr>
<tr>
<td>Intubation/ventilation</td>
<td>0 (0)</td>
<td>10 (10.2)</td>
</tr>
<tr>
<td>Naloxone</td>
<td>4 (12.5)</td>
<td>78 (79.6)</td>
</tr>
<tr>
<td>Antiemetics</td>
<td>7 (21.9)</td>
<td>2 (2.0)</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>10 (31.3)</td>
<td>4 (4.1)</td>
</tr>
</tbody>
</table>


20. TOXICOKINETICS OF ETHANOL IN AN INFANT

Laura Tormoehlen1, James Mowry1
1Indiana Poison Center, Indianapolis IN USA; 2Indiana University School of Medicine, Indianapolis IN USA

Background: Nearly 1.3 million poisoning exposures occur annually in children under the age of 6 years, and about 10% of these occur in children under the age of 12 months. Previous cases of infantile ethanol exposure have been reported, but none with toxicokinetic data.

Case Report: A 3 month-old boy was in his usual state of good health when he was given his afternoon bottle of formula prior to a nap. His parents found him to be excessively somnolent after his usual period of sleep, and it was then discovered that the formula had been mixed with Patrôn® tequila that was stored in a water bottle in the refrigerator. The volume of tequila used to mix the formula was estimated to be 1-3 ounces with additional bottled water to complete the volume. His initial ethanol level was 338 mg/dL at about 2 hours post-ingestion, and he was transferred to a tertiary pediatric hospital. He was mildly agitated with subcostal retractions on arrival and was placed on supplemental high-flow oxygen that was weaned to room air over several hours. Serial ethanol levels were followed as below.

Table 1.

<table>
<thead>
<tr>
<th>Time since ingestion in hours</th>
<th>Blood ethanol level in mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.10</td>
<td>338</td>
</tr>
<tr>
<td>5.53</td>
<td>411</td>
</tr>
<tr>
<td>11.00</td>
<td>253</td>
</tr>
<tr>
<td>16.25</td>
<td>144</td>
</tr>
<tr>
<td>24.10</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>

Case Discussion: As there is no toxicokinetic data in the literature on ethanol elimination in a child as young as 3 months of age, the three concentrations from 5.53 to 16.25 hours post ingestion were plotted and subjected to linear and non-linear regression analysis. The concentration vs. time data seemed to best fit a first-order elimination pattern over the concentration range observed \((r^2 = 0.9970)\) compared to non-linear regression. First-order elimination had marginally better correlation than for a zero-order elimination \((r^2 = 0.9912)\). The first-order elimination constant was 0.0978 hr\(^{-1}\) with a corresponding elimination half life of 7.1 hours. Based on these calculations, it was expected that the level at 24 hours after ingestion would have been approximately 68 mg/dL. However, the level at 24 hours was reported at <10 mg/dL. If one replots the data including the 24 hour data point at a conservative level of 10 mg/dL, the plot then seemed to fit that of zero-order kinetics \((r^2 = 0.9844)\) compared to first-order elimination \((r^2 = 0.8955)\). The elimination rate of ethanol seemed to be approximately 21 mg/dL/hr in this 3 month-old child.

Conclusion: Care must be taken in interpreting kinetics data in the overdose setting as basing clinical judgements on insufficient data may lead to erroneous decisions. The elimination rate of ethanol in this 3 month-old was similar to that reported for older children.

19. COMPLETE NEUROLOGIC RECOVERY AFTER CARDIAC ARREST IN A COCAINE STUFFER

Jonathan Gochmour1, Heath Jolliff2
1Kirkville College of Osteopathic Medicine, Kirksville MO USA; 2Central Ohio Poison Center, Columbus OH USA

Background: We present the first documented case of cocaine-induced cardiac arrest in a cocaine stuffer, who then made a complete neurological recovery.

Case report: A 34 y/o male inmate was witnessed to have swallowed an unknown white powder and within a few minutes had a generalized seizure.

On EMS arrival, he was bradycardic with a rate of 56 beats/minute. His bradycardia worsened and he went into cardiac arrest with asystole. On arrival to the Emergency Department, he was intubated and given three rounds of ACLS with atropine, epinephrine, and sodium bicarbonate. He had a return of a pulse and blood pressure (58/33) after 20 minutes. His post-arrest EKG demonstrated a sinus bradycardia with a 2:1 block and a wide QRS of 188 msec.

Physical exam, he had dilated non-reactive pupils and active hematemesis. Following resuscitation, the patient had an elevated troponin of 5.32 ng/mL within 30 minutes of arrest the patient had a return of a pulse and blood pressure (58/33) after 20 minutes. His post-arrest EKG demonstrated a sinus bradycardia with a 2:1 block and a wide QRS of 188 msec.

On physical exam, he had dilated non-reactive pupils and active hematemesis. Following resuscitation, the patient had an elevated troponin of 5.32 ng/mL at about 2 hours post-arrest. The patient was admitted to the ICU and the next day received ACLS with atropine, epinephrine, and sodium bicarbonate. He had a return of a pulse and blood pressure (58/33) after 20 minutes. His post-arrest EKG demonstrated a sinus bradycardia with a 2:1 block and a wide QRS of 188 msec. On physical exam, he had dilated non-reactive pupils and active hematemesis. Following resuscitation, the patient had an elevated troponin of 5.32 ng/mL at about 2 hours post-arrest. The patient was admitted to the ICU and the next day received ACLS with atropine, epinephrine, and sodium bicarbonate. He had a return of a pulse and blood pressure (58/33) after 20 minutes. His post-arrest EKG demonstrated a sinus bradycardia with a 2:1 block and a wide QRS of 188 msec.

Conclusions: Clinicians should be aware of emerging complications of cocaine abuse, such as vasculopathy with purpura and skin necrosis, neutropenia, and abnormal immune serology, suggestive of a non-vasculitic autoimmune process. Factors which predispose individual cocaine users to these complications need to be identified.

Reference

23. CONVULSIONS AS A COMPLICATION OF SYNTHETIC CANNABINOID USE

Aaron Schnee, Todd Baumbacker
University of California, San Diego Medical Center, San Diego CA USA

Background: Clinical presentations following the use of various “spice” or synthetic cannabinoids have included agitation, anxiety, hallucinations, psychosis, vomiting, and tachycardia. Complications led the DEA to make certain synthetic cannabinoids including JWH-018 schedule I. That we are aware of only one previously published report describes a convulsion as a possible complication of use. In that report, there was no laboratory confirmation for the presence of synthetic cannabinoids. We describe a patient who had two witnessed generalized convulsions soon after smoking a “spice” product that we later confirmed to have four different synthetic cannabinoids.

Case Report: A 19-year-old male without a history of prior convulsions, ethanol use, nor on any medications, had a witnessed generalized 1-2 minute convulsion while smoking a product “Happy Tiger Incense”. The product label described it as being “JWH-018 free”. Enroute to the hospital he vomited and had a second generalized convulsion that was treated with 5 mgs of intranasal midazolam. The patient had a short postictal period and arrived to the ED slightly sedate and confused, both of which rapidly resolved. Initial vitals were normal except for hypertension (177/82 mmHg) which quickly normalized without intervention. Physical exam, complete blood count, chemistries (including sodium and calcium), and TSH were normal. A urine drugs of abuse screen was positive for benzodiazepines as a class (presumably from the administered midazolam), and negative for amphetamines as a class, barbiturates, opiates, and benzoylcegonine. A CT scan of the brain was normal as were csf studies. The patient remained asymptomatic, with normal vital signs and was discharged after a short observation. The remains of the product smoked were sent to NMS labs (Willow Grove, PA) for analysis. Four synthetic cannabinoids (JWH-018, JWH-011, JWH-250, and AM-2201) were identified.

Case Discussion: We describe a patient who had two witnessed generalized convulsions soon after smoking a “spice” product that we later confirmed to have four different synthetic cannabinoids. Although the product label described it as being “JWH-018 free” we confirmed that this synthetic cannabinoid was in fact present. It remains unclear which specific synthetic cannabinoids are associated with which specific complications. Although four different synthetic cannabinoids were found in the product described we are unable to exclude the possibility that another compound in the product may have been responsible.

Conclusion: Convolusions appear to be another complication from the use of “spice” products.
including resting tremor and bradykinesia as well as ECG changes. Both had positive urine MDPV concentrations of 0.1 mcg/mL and 0.52 mcg/mL respectively. The third patient is a 35 year old man who presented to the emergency department complaining of a rapid heart rate and shortness of breath after snorting bath salts. His urine was positive for MDPV with a level of less than 0.05 mcg/mL. The last and most severe case involved a 30 year old man who spent the day snorting bath salts in a hotel room. He became so agitated that police were called. When they arrived he jumped from a second story window, and was later found dead in a nearby creek. His blood [MDPV] was 0.33 mcg/mL.

Discussion: MDPV has become a popular drug of abuse. Poison control centers across the US are seeing a dramatic increase in calls regarding MDPV use. The Missouri Poison Center has received 116 calls regarding bath salts in the first four months of 2011 compared to 22 total calls in all of 2010.

Conclusion: This case series highlights some of the concerning neurologic, cardiopulmonary and psychiatric side effects associated with MDPV use, and the rapid increase in its abuse.

25. HEROIN ADDICTS AND OLDER AGE; DOES MATURITY MATTER?

Gerald E. Maloney2, Sean M. Bryant1
1Toxikon Consortium, Cook County Hospital (Stroger), Illinois Poison Center, Chicago IL; 2Case Western Reserve University/MetroHealth Medical Center, Cleveland OH

Background: There is a paucity of data on the issue of heroin abuse among older patients (age ≥60). We sought to review cases from a single poison center to determine the characteristics and outcomes of heroin exposures reported in adults ≥60 years of age.

Methods: A retrospective review of poison center charts from 1/1/2002 through 12/31/2010 was performed. Charts where heroin was coded as one of the involved substances were reviewed, and those charts in which the patient was 60 years of age or older were abstracted. Information recorded included gender, age, intent (including details surrounding the exposure), disposition, and clinical outcome.

Results: There were a total of 1,312 heroin cases reported to the regional poison center (RPC) during the 9 year period. A total of 8 patients (0.006%) met inclusion criteria and were included in the study. Seven of the 8 patients were male, and the average overall age was 64.4 years (range: 60-71). Two patients were noted to be in withdrawal and 1 was admitted to the ICU for dehydration and blood pressure management. The other six were reported as intentional ingestions. One was lost to follow-up after the initial call to the poison center for ingestion of several bags of heroin. The remaining 5 patients were admitted (four to the ICU) and there was one death resulting from multi-organ failure and significant acidemia of undetermined etiology. Naloxone was administered in 3 of the patients preventing 2 intubations. One of these patients was found down on the stairs in front of a church and the other was suspected of abusing fentanyl in addition to heroin. The use of heroin in these patients was determined based on a history of exposure, however 4 patients had positive qualitative urine opiate screens while 1 was also positive for methadone.

Conclusions: Older adults accounted for a very small number of this RPC’s calls for heroin exposure during the 9 year period. However, the majority was admitted to a critical care setting with one death reported, suggesting a possible greater morbidity in this age group. Further prospective study of heroin abuse in older patients may be warranted.

26. COCAINE ABUSE IN OLDER ADULTS; BABY BOOMERS & BLOW IN THE NURSING HOME

Gerald E. Maloney2, Sean M. Bryant1
1Toxikon Consortium, Cook County Hospital (Stroger), Illinois Poison Center, Chicago IL; 2Case Western Reserve University/MetroHealth Medical Center, Cleveland OH

Background: There are a paucity of data on cocaine abuse in older adults (age ≥60 years old). We sought to review our regional poison center’s (RPCs) experience with calls regarding cocaine abuse in older adults.

Methods: We reviewed all charts from 1/1/2002 through 12/31/2010 from our RPC with cocaine recorded as one of the involved substances. From this group, all charts of patients age 60 years or older were then reviewed and abstracted. Age, gender, events associated with the exposure, and clinical outcome were recorded.

Results: A total of 2,500 calls to our RPC included cocaine as a substance over the 9 year period. Only 11 patients (0.004%) met inclusion criteria. The average age was 65 years (range, 62-77) and 6 of the 11 patients were male. A qualitative urine toxicology screen was positive for cocaine in 7 patients with 1 of these simultaneously positive for opiates. The setting for 4 of 11 reported exposures occurred in an extended-care facility (ECF). Two of the ECF patients swallowed baggies of cocaine and one admitted to also smoking crack cocaine. The other 2 patients reportedly injected cocaine and swallowed several rocks of unwrapped crack cocaine. The route of exposure was unclear in 4 cases. ICU admission was necessary in 4 patients with one patient experiencing ventricular tachycardia and another patient who required intubation for 24 hours. Polysubstance ingestions were present in 5 of the 11 patients with one patient treated with NAC for concurrent acetaminophen poisoning. Only 1 patient required administration of benzodiazepines for significant hypertension (systolic BP 223 mmHg, pulse 120) and delirium. Hyperthermia and/or death were not reported in this series of patients.

Conclusions: Older adults accounted for a small fraction of the calls made to this RPC for cocaine abuse during the study period. Interestingly, an alarming proportion of the exposures occurred while the patients were at an extended-care facility. Additionally, cocaine body stuffing was reported in this population of patients. While outcome was generally benign, prospective evaluation of cocaine abuse in geriatric patients is warranted.

27. PREVALENCE OF HYPONATREMIA OR NEUTROPENIA ASSOCIATED WITH LEVAMISOLE-ADULTERATED COCAINE IN ED PATIENTS

Kerri Friend, Michael Milone, Keri Donaldson, Jeanmarie Perrone
University of Pennsylvania, Philadelphia PA USA

Background: Most cocaine is adulterated with other pharmacological agents to either enhance or diminish the drug effects. Recently, the Drug Enforcement Agency and the Department of Health and Human Services warned that up to 80% of cocaine is cut with levamisole, an immunomodulator. Additionally, they warned of unexplained agranulocytosis in previously healthy cocaine users that has been attributed to levamisole. We have also observed unexplained hyponatremia in the setting of cocaine use. We confirmed a high co-prevalence of levamisole-adulterated cocaine by gas chromatography-mass spectroscopy in the urine of ED patients confirmed to be using cocaine in samples from 2007-2009; prior to 2007 levamisole was not detected. We sought to determine whether leukopenia or hyponatremia have been increasing in the period 2008-09 (post levamisole) compared to 2005-06 (pre-levamisole) in ED patients using cocaine. We hypothesized that these complications would increase with the increasing prevalence of levamisole-adulterated cocaine during this period.

Methods: Setting: A tertiary care urban university ED with 57,000 annual visits.

We searched our ED database for the period 2000-2010 for all patients who had a urine drug screen which revealed cocaine metabolites. This set of confirmed cocaine users was then used to identify two cohorts of patients, a pre-levamisole cohort (mean age 42 ± 11 years; 52% male) and a post-levamisole cohort (mean age of 45 ± 11 years; 62% male) that presented to our ED during the periods of 2005-2006 or in 2009, respectively.

We examined ED lab values in each cohort to determine the prevalence of hyponatremia (sodium < 130 mEq/L) and leukopenia (WBC < 4/mm3) during the pre-levamisole and post-levamisole period.

Results: 440 patients had a cocaine positive urine drug screen in the pre-levamisole period and 1235 patients in the post-levamisole period. Hyponatremia was found in 10/440, 2.3 % of patients in the earlier cohort and 27/1235, 2.2 % (p = 0.853) in the later period. Leukopenia was found in 15/440, 3.4 % of patients in the earlier cohort and 42/1235, 3.4 %, p = 1.00 ) in the later period.

Conclusions: Levamisole adulterated cocaine is currently prevalent in our region. Adverse effects previously associated with levamisole, hyponatremia and leukopenia, were not more prevalent as levamisole-adulterated cocaine use increased in a cohort of ED patients using cocaine.
28. K2 & SPICE ABUSERS: A CASE SERIES OF CLINICAL AND LABORATORY FINDINGS

Christopher D. Rosenbaum1, Anthony J. Scalzo1, Christopher Long3, Julie A. Weber1, Amanda Jenkins4, Gaylord P. Lopez5, Sean Ragone6
1Saint Louis University Division of Toxicology and Missouri Poison Center, Saint Louis MO USA; 2University of Massachusetts Department of Emergency Medicine, Division of Toxicology, Worcester MA USA; 3Saint Louis University Forensic & Environmental Toxicology Laboratory, Saint Louis MO USA; 4University of Massachusetts Toxicology Laboratory, Worcester MA USA; 5Georgia Poison Center, Atlanta GA USA

Background: Herbal marijuana alternatives (HMAs) are plant products intentionally adulterated with synthetic cannabinoid receptor agonists (synCBs), e.g. JWH-018. Although sold as incense not intended for human consumption, HMAs have gained popularity as drugs of abuse both in Europe and the U.S. Many EU member countries formally banned HMAs in 2009, and the DEA scheduled a select number of synCBs in March 2011. Little information describes clinical toxicity with HMA use or the (in)ability to detect synCBs in human serum or urine. We describe an outbreak of HMA abuse along with clinical presentation and lab Methods.

Case reports: During ongoing surveillance for emerging drugs of abuse between 11/09 and 8/10, we identified a convenience sample comprising 78 cases of abuse of HMAs known as K2, K2 Summit and Spice. Cases came from regional PCCs and toxidology consultations in 4 states. After IRB approval, clinical and lab data were reviewed and urine tested via gas chromatography mass spectroscopy (GCMS). Samples of HMAs and pure synCBs were also analyzed by GCMS.

Case Discussion: Users were predominantly young (range 12-46 years) males (79%) who smoked HMAs. Median HR was 122 (range 49-180), median mean arterial pressure (MAP) was 100 (range 57-140); max systolic BP was 200 mm Hg. Nausea or vomiting was reported in 14 cases (18%); agitation or anxiety was reported in 37 cases (47%); and hallucinations were reported in 8 patients (10%). One suicide (18 years old) was associated with K2 use 1 hour prior, and 1 case (22 years old) met DSM-IV criteria for K2 addiction. GCMS forensic analysis of 4 pure synCBs established a reference library against which analysis of HMAs showed presence of synCBs (JWH-018, JWH-073, JWH-081). Of 22 urine specimens obtained from self-identified HMA abusers, 1 sample demonstrated the presence of JWH-018. Metabolites were not measured but such analysis by arduous tandem LC/MS/MS has been described by Sobolevsky et al, 2010.

Conclusion: HMAs are currently available via the Internet under the brand names K2 and Spice. We have confirmed the presence of synCBs in samples of these HMAs. The hypertension, agitation, anxiety, and vomiting observed in this convenience sample are not fully explained by presence of cannabinoid receptor (CB1 & CB2) agonists. The ability to detect JWH-018 in only 1 of 22 samples illustrates the difficulty with drug testing in this population. Explanations may be multifactorial: synCB metabolites may be better suited for detection, synCBs may not be stable in urine, or synCBs may have been absent in the HMAs used by our sample of patients. Further research on HMAs and abusers is warranted and other detection Methods (i.e., immunoassays) should be explored.

29. BATH SALTS & SYNTHETIC CANNABINOIDS - INITIAL EXPERIENCE & COMPARISONS

Kurt Kleinschmidt1, Mathias B. Forrester2
1University of Texas Southwestern Medical Center, Dallas TX USA; 2Texas Department of State Health Services, Austin TX USA

Background: Bath Salts refer to a new group of stimulant drugs whose primary active agents are Mephedrone (4-methylmethcathinone) and Methylenedioxypyrovalerone (MDPV); both being designer substances of the phenylethylamine class and cathinone derivatives from the khat plant (Catha edulis). Another new agent group is the Synthetic Cannabinoids (SC). The SC agent group was originally derived to study the potential clinical benefits of cannabinoid receptor agonism. Both agent groups are abused for their stimulatory and hallucinogenic effects. The purpose of this study is to compare certain attributes of these agents.

Methods: Data from the Texas poison network was evaluated. Bath salt data were from Jan 1, 2010 – April 1, 2011; SC data were from calendar year 2010. Multiple search terms within the data base were used to maximize the capture of exposures. Cases were excluded if coingestants were present.

Results: The use of both agents has risen rapidly. For bath salts, there were no exposures from Jan – April 2010, 11 from May – Nov 2010, & 71 from Dec 2010- March 2011; 82 exposures total. For SC, Jan 2010 had 5 reports and the numbers steadily increased to 60 +/-month at the end of the year; 464 reports total. User age was ≥20 years old in 87% of salts users and 57% with SC users. Males were the users in 72% of the salt and in 74% of the SC exposures. Route of exposure for salts was 60% inhaled & 28% ingested (versus SC - 80% & 11%, respectively). Both agents were primarily used at a residence; 77% for SCs and 74% with the salts. The reason for use was intentional abuse in 78% for both agent groups. The clinical outcomes for the two agent groups were very similar (Table 1). In addition, the adverse events for the two agent groups were fairly similar (Table 1). The most common adverse events for each agent group are the same. Among these most common adverse events, the bath salts were associated more with hypertension, agitation, and confusion. Conversely, the SCs were associated more with vomiting and drowsiness.

Table 1.

<table>
<thead>
<tr>
<th></th>
<th>Bath Salts (%)</th>
<th>Synth Cannabinoids (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Effect</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Minor Effect</td>
<td>16</td>
<td>26</td>
</tr>
<tr>
<td>Moderate Effect</td>
<td>44</td>
<td>42</td>
</tr>
<tr>
<td>Major Effect</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Not followed; Likely Minor</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Not followed; Potentially Toxic</td>
<td>23</td>
<td>14</td>
</tr>
<tr>
<td>Chest Pain</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Hypotension</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>43</td>
<td>37</td>
</tr>
<tr>
<td>Vomiting</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>Agitation</td>
<td>29</td>
<td>19</td>
</tr>
<tr>
<td>Confusion</td>
<td>16</td>
<td>9</td>
</tr>
<tr>
<td>Drowsy</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>Hallucinations</td>
<td>15</td>
<td>11</td>
</tr>
</tbody>
</table>

Conclusions: The use of these two new stimulant drugs of abuse has risen rapidly. Bath salts users are older than SC users but both are mostly used by males. The routes of exposure and reason for using these agents are similar for the two agent groups. The clinical outcomes are similar as are the most common adverse events; however, the bath salts are more associated with hypertension, agitation, and confusion.

30. DROWNING IN BATH SALTS: A CASE OF ABUSE WITH FATAL OUTCOME

Matthew Valento1, Benjamin Feinzimer2, Sean M. Bryant1
1Toxikon Consortium, Cook County Hospital (Stroger), Chicago IL USA; 2University of Illinois-Chicago, Chicago IL USA

Background: Abuse of ‘bath salts’, often containing synthetic amphetamines such as mephedrone and mephedronedioxyprovalerone (MDPV), is a topic of increasing publicity and public health concern. Use of these substances can produce a sympathomimetic toxidrome similar to that from cocaine or methamphetamine. We present a case of bath salts abuse resulting in severe toxicity and death.

Case Report: A 55 year-old male with a history of polysubstance abuse presented to an emergency department by EMS complaining of tremor and agitation. He reported snorting bath salts prior to his arrival. Crystals were noted around his nares and upper lip. Family members later retrieved two small silver bags labeled ‘invigorating bath salts’ (containing identical crystals) from the patient’s home.
On arrival, vital signs were blood pressure of 140/56 mmHg, pulse 160, respiratory rate 30, oxygen saturation 91% on room air, and core temperature 103 F. Physical exam was remarkable for agitation, diaphoresis, and tremor. Laboratory assessment was significant for: acute renal failure with BUN of 62 mg/dL and creatinine of 6.6 mg/dL; elevated liver enzymes with AST and ALT of 1257 U/L & 292 U/L respectively; elevated serum troponin of 7 ng/mL; and rhabdomyolysis with CPK >14,000 U/L. A qualitative urinary toxicology screen was negative for standard substances. The regional poison center was contacted and recommended intravenous fluids, cooling measures, and liberal use of benzodiazepines. The patient was admitted to the intensive care unit (ICU) for further management. Shortly after arrival in the ICU, the patient developed respiratory failure and was emergently intubated. The subsequent hospital course was complicated by persistent hypotension, rhabdomyolysis and worsening renal failure, rectal bledding, and disseminated intravascular coagulation. Aggressive management with vasopressors, transfusion of blood products, parenteral steroids, broad spectrum antibiotics, and multiple courses of hemodialysis were initiated. The patient remained unresponsive to all stimuli despite weaning and removal of sedative medications. Approximately 48 hours after presentation to the hospital, the patient’s family elected to withhold care and the patient expired.

Case Discussion: We present a case of bath salt abuse presenting with severe sympatheticomimetic toxicity and ultimately proving to be fatal. We believe this is one of the first fatalities reported after abuse of these illicit synthetic amphetamine. Physicians and public health officials dealing with this burgeoning epidemic must be aware of its potentially devastating consequences.

Conclusion: Abuse of ‘bath salts’ can lead to severe sympathomimetic toxicity and death.

31. 1,4-BUTANEDIOL TOXICITY WITHOUT GAMMA-HYDROXYBUTYRATE TOXICITY

Kirk L. Cumpston, Carl E. Wolf, Brandon K. Wills, Rutherford Rose
Virginia Commonwealth University Medical Center, Richmond VA USA; Virginia Commonwealth University Medical Center, Virginia Poison Center, Richmond VA USA

Background:1,4-Butanediol (BD) is an industrial chemical, used illicitly for recreation due to its metabolism to gammahydroxybutyrate (GHB). This conversion occurs via alcohol dehydrogenase (ADH) thus interactions can occur when ethanol is a co-ingestant leading to prolonged or biphasic toxicity. We present a case of an individual who developed toxicity following ingestion of BD but without subsequent GHB formation.

Case Report: A 20 year-old male admitted to taking two spoonsfuls of liquid BD along with ethanol over an 8 hour period. Approximately 11 hours after his last ingestion he was found lethargic on the bathroom floor at his workplace. He arrived at the hospital unresponsive and apneic and was intubated after failing to respond to naloxone. He required large doses of propofol,to manage severe agitation. Vital signs post intubation were T 36.5°C, HR 88, BP 122/73 mm Hg, RR 16, SpO2 98%. The serum ethanol concentration was negative, as was analysis for OTC meds and drugs of abuse in serum and urine. He gradually awoke and was extubated the next day without complications. Analysis of the liquid contents of the patient’s bottle revealed the substance to be >95% BD. The patient’s blood was also tested for concentrations of both BD and GHB. The BD serum concentration was determined by gas chromatography with a flame ionization detection, using 1,3 butanediol as the internal standard. The GHB was extracted with ethyl acetate, dried with a nitrogen bath and analyzed by GC/MS (See Table 1).

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>BD mg/L</th>
<th>GHB mg/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>7/5</td>
<td>1510</td>
<td>58</td>
<td>5.8</td>
</tr>
<tr>
<td>7/5</td>
<td>2300</td>
<td>&lt;5</td>
<td>3.0</td>
</tr>
<tr>
<td>7/6</td>
<td>0255</td>
<td>ND</td>
<td>6.3</td>
</tr>
</tbody>
</table>

Table 1. BD and GHB Serum Concentrations

1,4-Butanediol toxicity without gammahydroxybutyrate toxicity.

Case discussion: This patient’s clinical course was typical for paradoxical agitation and coma seen after GHB overdose. Laboratory analysis of blood obtained approximately 11 hours after last ingestion revealed elevated BD serum concentrations and GHB serum concentrations consistent with endogenous levels. A study in volunteers reported an elimination half-life of BD of 40 minutes, but longer time in patients with a variant allele for ADH. It is unclear in this case why GHB serum levels did not increase as the BD serum concentration fell, particularly in the absence of ethanol or fomepizole. Therefore it appears that the toxicodynamic effects in this case were from BD with little or no contribution from GHB.

Conclusion: BD is normally rapidly metabolized via ADH to GHB which is thought to cause clinical toxicity. In some individuals this may not occur and toxicity is due to BD. This would make the decision to inhibit ADH with fomepizole potentially detrimental, and reinforce the street reasoning for co-ingestion with ethanol.

32. FACIAL FROSTBITE ASSOCIATED WITH INTENTIONAL INHALATION OF A COMMERCIAL DUSTING PRODUCT

Jennifer Plumb1, Richard G. Thomas2, Howard M. Cornelli3
1University of Utah Division of Pediatric Emergency Medicine, SLC UT USA; 2Primary Children’s Medical Center, SLC UT USA

Background: Halogenated hydrocarbon inhalation abuse has been associated with serious but infrequent complications; this case describes the first cryogenic injury from a commercial dust removal product.

Case Report: A 13 year-old male presented after developing sudden severe swelling of his lips. He described that he was lying in the grass drinking green tea when the swelling started. He denied any hoarseness, difficulty swallowing, difficulty breathing, wheezing, shortness of breath, or pruritis. He had no known envenomation and denied any medications or other new exposures. The swelling was very rapid, developed within 30 minutes, and did not change significantly after that. On initial evaluation: T 36.8, P 88, RR 22, BP 127/70, Sats 96%. He was alert, appropriate, in no distress. Exam was normal with exception of significant edema of the lips and perioral area and some swelling of the anterior tip of tongue. There was no blistering or other intraoral trauma, posterior pharynx was normal. Neck without stridor or crepitus, lungs clear, CV exam normal. Skin had 2 small reddened areas on left index finger and thumb. He was given IV epinephrine, diphenhydramine, famotidine, and solumedrol. He was monitored for 1.5 hours and did not show any change in clinical symptoms. After transfer to a pediatric facility, he did admit that he had been huffing a commercial air duster product immediately prior to the development of the swelling. He was admitted and observed overnight with no change, and was discharged home the next day. Follow up three days later showed blistering of the skin lesions on the hand with large bullae and burns of the lips consistent with cryogenic exposure.

Case Discussion: This case reports significant frostbite associated with huffing an aerosolized propellant. The product that he had been exposed to contains the halogenated hydrocarbon 1,1-difluoroethane which with inhalation has been associated with respiratory irritation, cardiac sensitization, and CNS depression, and after direct skin or eye contact with frostbite. Although rare case reports of significant oral frostbite have been reported from accidental intraoral exposures to other cryogenic halogenated hydrocarbons, this is the first case from a commercial dusting product used to clean computer keyboards. These products are frequently abused by adolescents but rarely produce this effect. Evaluation of patients presenting with unusual lip swelling or evidence suggestive of cryogenic burns should include the possibility of abuse of halogenated hydrocarbons by inhalation.

Conclusions: This case demonstrates an unusual but significant clinical effect associated with inhalational abuse of halogenated hydrocarbons.

33. CHARACTERISTICS OF “BATH SALTS” EXPOSURES REPORTED TO A REGIONAL POISON CENTER

Anna Rouse Dulaney
Carolinas Poison Center, Charlotte NC USA
Background: Use of the substances marketed as “bath salts”, which are thought to contain synthetic sympathomimetics, has skyrocketed since late 2010. There is little cumulative clinical data regarding bath salt intoxication. We sought to characterize exposures reported to a regional poison center (PC).

Case Series: A PC serving 9.3 million people and receiving over 100,000 calls yearly logged 9 bath salts exposure cases in 2010. Over 97 days in 2011, it received 127 bath salts exposure calls (1.3 cases/day). Eighty-eight (69%) were male; of the 118 cases in which age was captured, the mean age was 29 years (range 16-68). Interestingly, the two counties with the highest number of cases reported (n = 16 and n = 12) are home to military installations. Routes of exposure included insufflation, parenteral, and ingestion.

The most common clinical effects reported were tachycardia (50%), agitation/irritable (44%), hypertension (28%), and hallucinations/delusions (26%). Elevated CPK was reported in 20%, confusion in 14%, and drowsiness/lethargy in 13% of patients. Review of cases with “Other” coded as a clinical effect described abnormal movements (n = 9), paranoia (n = 6), and insomnia (n = 5). Forty-five percent (n = 58) of the patients had moderate outcomes, 18% had minor outcomes, and 11% were considered major outcomes. There were no deaths. Most patients were treated with hydration and benzodiazepines. Serious complications included one patient with multiple seizures and one patient with acute renal failure: a 50 y.o. male presented 2 days after ingesting bath salts. His initial BUN was 83 mg/dL, creatinine (Cr), 6.0 mg/dL; potassium, 5.7 mEq/L. He had not voided in over 48 hrs. His Cr peaked at 15.07 mg/dL; his CPK peaked at 123,880 IU/L. He received intermittent hemodialysis for 2 weeks. Additionally, one case of syncope, lethargy, hypotension, and bradycardia presented after persistent bath salt use and may represent a washout syndrome.

Discussion: When new substances of abuse emerge, clinical recommendations are difficult to make when there is little or nothing in the medical literature. We sought to characterize presenting symptoms in patients abusing bath salts. Our review found that most cases present with symptoms similar to those of amphetamine intoxication. Limitations include lack of confirmatory lab studies to determine exactly what compounds were used.

Conclusion: Patients presenting with bath salts exposures are likely to experience tachycardia, agitation, hypertension, and hallucinations/delusions. Rhabdomyolysis may be a severe complication. Most cases appear to be sufficiently managed with hydration and benzodiazepines.

34. SURREPTITIOUS STEROID INGESTION RESULTING IN FACTITIOUS CUSHING’S SYNDROME: SOMETIMES SENDOUTS MAKE THE DIAGNOSIS

Travis D. Olives1, Heather Ellsworth1, Samuel J. Stellpflug2, Jon B. Cole1
1Hennepin Regional Poison Center, Minneapolis MN USA; 2Regions Hospital Clinical Toxicology Service, St. Paul MN USA

Background: Cushing’s syndrome is a constellation of signs and symptoms commonly characterized by weight gain, round (moon) facies, truncal obesity, hypertension, hyperglycemia, and abdominal striae. Causes include pituitary and adrenal tumors, ectopic foci of ACTH or cortisol production, and exogenous glucocorticoid administration. We present the case of a patient referred for evaluation of Cushingoid body habitus who was found to have Cushing’s syndrome from exogenous glucocorticoids.

Case Report: A 40 year-old woman with a past medical history significant for refractory hypertension, mild asthma, dyslipidemia, and many self-reported allergies was referred to an endocrinologist for evaluation of hypertension, central obesity, a dorsal hump, and hyperglycemia noted during a recent Emergency Department visit. She expressed frustration at being obese, stating that it had developed over many years and prohibited her from working in her chosen vocation as a choreographer. A chart review revealed an extensive past lab workup notable for normal urine and serum catecholamines, normal thyroid function tests, and persistently low cortisol levels. Physical examination revealed lipodystrophy at C7, vague abdominal striae, and round facies. Her hemoglobin A1c returned elevated at 6.3%. ACTH stimulation was normal; salivary and urine cortisol levels were low. The patient denied taking exogenous steroids. In light of a workup inconsistent with the patient’s history and exam, serum prednisone, dexamethasone, and prednisolone concentrations were sent. Her serum prednisolone returned at 3.4 mcg/dL (normal range undetectable); 2 weeks later a follow-up concentration was also elevated at 2.2 mcg/dL. The patient insisted on further workup of her Cushingoid body habitus, but vehemently denied any knowledge of exogenous steroid exposure. Further chart review revealed no documented use of steroid therapy for treatment of her asthma in the preceding 10 years.

Discussion: Glucocorticoids, a common therapy for a variety of illnesses, are a well-known etiology of Cushing’s syndrome. Factitious Cushing’s syndrome is less common, however case reports support its existence. Additional case reports underscore its gravity, with glucocorticoid withdrawal and deaths secondary to opportunistic infections both reported. This case highlights the importance of a careful review of available data – in this case across multiple institutions – in uncovering this often difficult to diagnose disorder.

Conclusion: Toxicologists and endocrinologists should be aware of surreptitious administration of glucocorticoids as a hidden cause of Cushing’s syndrome.

35. A COMPARISON OF INGESTED VERSUS INHALED SYNTHETIC CANNABINOIDS

Kurt Kleinschmidt1, Mathias B. Forrester2
1University of Texas Southwestern Medical Center, Dallas TX USA; 2Texas Department of State Health Services, Austin TX USA

Background: Synthetic cannabinoids (SC) affect the cannabinoid receptors. They were created to be potential pharmaceutical agents, attempting to take advantage of the potentially useful clinical attributes of marijuana and THC. They have recently gained popularity as a recreational drug because they are believed to result in a marijuana-like high. Their use has risen rapidly over the last year. Our understanding of the clinical effects of these agents is limited. Synthetic cannabinoids are used by various routes. It is unclear if the clinical outcomes and adverse events with SCs are affected by the route of exposure. The purpose of this study is to compare these effects based upon the route of exposure.

Methods: The Texas poison center database for the calendar year 2010 was queried for all synthetic cannabinoid exposures. Many codes/terms were used to yield a complete list of exposures. Cases were excluded if coingestants were present. Data were sorted by route of exposure. Only inhalational and ingested exposures were analyzed; those who used SCs by more than one route were excluded.

Results: There were 405 SC exposures that met study criteria; 365 (90%) inhaled and 40 (10%) ingested. The age of the exposed patients was ≥20 years in 50% of the ingestions and 60% in the inhalations and the users were male in 73% and 75%, respectively. The reason for exposure was unintentional in 25% of the ingestions and in 8% of the inhalations; conversely, it was for intentional abuse in 75% and 92%, respectively. This difference may be consistent with the younger age of ingestion exposure population (younger children) may be more likely to accidentally eat the agent. The clinical outcomes and adverse events for the two routes of exposure are fairly similar (Table 1). Specifically, for the addervasive events, there was more abdominal pain with the ingestion route; albeit no difference in nausea and vomiting. There was no diarrhea with either route. No patients who ingested SC developed dyspnea while 5% of the inhalation users did experience it.

Table 1.

<table>
<thead>
<tr>
<th>Ingested (%)</th>
<th>Inhaled (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Effect</td>
<td>8</td>
</tr>
<tr>
<td>Minor Effect</td>
<td>30</td>
</tr>
<tr>
<td>Moderate Effect</td>
<td>40</td>
</tr>
<tr>
<td>Major Effect</td>
<td>3</td>
</tr>
<tr>
<td>No F/U; Likley minor</td>
<td>8</td>
</tr>
<tr>
<td>No F/U; Potentially Toxic</td>
<td>13</td>
</tr>
<tr>
<td>Chest Pain</td>
<td>3</td>
</tr>
<tr>
<td>Hypertension</td>
<td>3</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>33</td>
</tr>
<tr>
<td>Nausea</td>
<td>13</td>
</tr>
<tr>
<td>Vomiting</td>
<td>10</td>
</tr>
<tr>
<td>Abdominal Pain</td>
<td>5</td>
</tr>
<tr>
<td>Agitation</td>
<td>23</td>
</tr>
<tr>
<td>Drowsiness</td>
<td>15</td>
</tr>
<tr>
<td>Hallucinations</td>
<td>15</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>0</td>
</tr>
</tbody>
</table>
Conclusions: Most synthetic cannabinoid users inhale the product. Twenty-five percent of the ingestions were unintentional, a higher frequency than with the inhalation route. There was more abdominal pain with the ingestion route and more dyspnea with the inhalation route. Both routes of exposure resulted in similar degrees of neurologic changes including agitation, drowsiness, and hallucinations.

36. A RETROSPECTIVE REVIEW OF POISON CENTER CALLS WITH PATIENTS EXPOSED TO 'SPICE' PRODUCTS

Laura Cookman, Carl G. Skinner, Katie Von Derau, Michael Miller

Background: Spice is a mixture of herbs and spices that contains a synthetic compound which acts as a cannabinoid receptor agonist. Until recently, spice was sold as a legal alternative to marijuana in the United States. It has become an increasingly common presentation to emergency departments and phone calls to poison centers. Despite its frequent use little is known about the toxidrome and clinical presentation of patients exposed to spice.

Methods: We retrospectively reviewed a large poison center data base for all cases of spice exposure from January 1, 2009 through January 31, 2011. Variables collected and analyzed included age, sex, symptoms, and treatments. Information calls and cases with positive drug screens or history of co-ingestions were excluded from the final analysis.

Results: Of 83 cases reviewed, 22 cases were excluded (14 information calls and 8 cases with a history of either positive urine drug screen or co-ingestion); 60 of the total cases were analyzed. The average age was 22 years old (range 10-55 years old) with 80% of patients being males. 48% of the calls to the poison center were hospital calls. Treatment consisted of intravenous fluids (38%), benzodiazepines (15%), and antiemetics (8%). Most patients had effects less than 8 hours. See Table 1 for the ten most common clinical findings reported.

Table 1. Clinical Presentation of Patients Exposed to ‘Spice’ Products.

<table>
<thead>
<tr>
<th>Clinical Sign or Symptom</th>
<th>Number of Patients</th>
<th>Percent of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia</td>
<td>30</td>
<td>50</td>
</tr>
<tr>
<td>Altered mental status</td>
<td>25</td>
<td>42</td>
</tr>
<tr>
<td>Agitation</td>
<td>20</td>
<td>33</td>
</tr>
<tr>
<td>Vomiting</td>
<td>18</td>
<td>30</td>
</tr>
<tr>
<td>Dizziness</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td>Mydriasis</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td>Syncope</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Hallucinating</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Possible seizure activity</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Other</td>
<td>19</td>
<td>32</td>
</tr>
</tbody>
</table>

Conclusion: The data suggests tachycardia predominates. Based on this retrospective review, patients exposed to spice will most likely present with clinical symptoms including tachycardia, decreased consciousness or agitation, mydriasis and gastrointestinal upset. As more patients call poison centers about spice products, the toxidrome of this substance may become clearer. Medical toxicologists and poison specialists should be aware of the presenting symptoms to better identify patients exposed to spice in order to help guide management.

38. ALCOHOL INTOXICATION, CO-INGESTION AND WITHDRAWAL IN MEDICAL TOXICOLOGY CONSULTATIONS: A REVIEW OF THE TOXIC CASE REGISTRY

Timothy Wiegand, Michael Kamali, Norma Barton, Paul Wax

Objective: Very few studies describe the type of patients seen by Medical Toxicology Consultation Services (MTCS). The Toxic Investigators Consortium (ToxIC) was developed by the American College of Medical Toxicology in January, 2010 to track patients seen by MTCS and to provide an infrastructure for research. In order to obtain information for further study, and for use in QA/QI projects involving ToxIC data entry, we reviewed and analyzed registry data regarding alcohol intoxication, co-ingestion and alcohol-withdrawal syndrome (AWS).

Methods: The ToxIC registry was queried from its inception to 4/10/2011 using the search terms -Alcohols (ETOH) and -Withdrawal (WD). Cases of WD not related to alcohol were excluded. Cases involving toxic alcohols (Methanol /EG) were also excluded. Analysis using WD and ETOH separately as well as concomitantly were performed to confirm data. Descriptive statistics were used to compare and report data. Data were not reviewed for outcome information.

Results: Nearly 6,000 patients were registered in the ToxIC database from its initiation to 4/10/2011. Alcohol was involved in 574 consults, 10% of all consults registered. Alcohol-related consults were more common in men than woman (60%/40%) and occurred most frequently as part of polydrug overdoses (62%). ETOH as a sole intoxicant occurred only 4% of the time. Frequent co-ingestants included analgesics (14%), sedative-hypnotics (16%), antidepressants (12%), antipsychotics (6%), opioids (9%), and sympathomimetics (6%). 192 of the 574 consults (33%) involved AWS. AWS was separately reviewed. Most consults for AWS occurred in males (76%/24%), 91% of AWS consults occurred in patients age 19-65, 5% in age > 65 and 4% in ages 13-18. 24% of patients with AWS had contributing substances coded for, the most common being sedative-hypnotics (8%). Other common substances in patients with AWS included: opioids, antihistamines and antidepresants. AWS represented approximately 3% of consults performed by MTCS involved.
in ToxIC (192 out of nearly 6,000 cases) Less than 0.5% ToxIC cases (23) were for alcohol intoxication alone (24 out of nearly 6,000 cases).

**Conclusion:** Medical Toxicologists are rarely consulted for poisoning solely related to ethanol. Most alcohol-related consultations occur in polydrug poisonings. AWS represents 33% of alcohol-related consultations but only 3% of all consultations performed by Medical Toxicologists. Although alcohol is reported to be involved in 20-70% of all poisonings admitted to hospitals it was less frequently (10%) involved in poisonings in the ToxIC Registry.

### 39. TOXIC HEPATITIS AFTER KRATOM (MITRAGYNA SP.) CONSUMPTION

**Hugo Kupferschmidt,** Colette Degrandi, Mirjam Gessler, Rahel Zahnd

**Background:** Kratom is made from the leaves of the Malaysian tree Mitragyna speciosa. It contains more than 20 alkaloids (incl. mitragynine, mitraphylline, speciogynine, speciociliatine, mitraciliatine, paynantheine, ajmalicine, and 7-hydroxymitragynine) and is used as a stimulant, as an analggesic and antitussive, or as a substitute in opiate addiction due to its opiative- and cocaine-like effects. While moderate doses have a stimulant effect, high doses lead to sedation. Adverse effects include nausea, vomiting, diarrhea, nystagmus, tremors, CNS depression, and seizures. Hepatotoxicity has not yet been reported in humans.

**Case Report:** A 30-ye female developed jaundice, pruritus and right-sided epigastric pain 5 and 7 days after having consumed 5 g of Kratom powder with alcohol on two occasions, purchased via the internet. One day after the second intake, she developed fever (38.5°C, 101.3°F) and musculoskeletal pain which resolved spontaneously the following day. Modification history was unremarkable. The patient denied any illicit drug intake. Laboratory tests revealed elevated transaminases (maximum AST 271 U/L (normal 1-31), ALT 482 U/L (234), bilirubin 160 μmol/L (<22), alkaline phosphatase 174 U/L (98). Further diagnostic work-up included abdominal ultrasonography, MR cholangiography, serum electrophoresis, coeruloplasmin, iron, ferritin, lipase, amylase, autoimmune antibodies, antibodies against hepatitis A, B, C, EBV, CMV, thus excluding alternative etiologies such as obstructive cholangiopathies, pancreatitis, viral hepatitis, autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis, Wilson’s disease, hemochromatosis, and alpha-1-antitrypsin deficiency. No attempt was made to analytically confirm Mitragyna metabolites or to exclude other drugs. Initial therapy consisted of an oral antihistaminic and corticosteroids to treat itching interpreted as allergic reaction. The patient was discharged home after four days, and liver injury resolved spontaneously within 35 days when transaminases and bilirubin normalized.

**Case Discussion:** The intake of Mitragyna sp. plant material (Kratom) is the likely cause of hepatotoxicity in this patient, as other causes of hepatitis were excluded, and the formal criteria of causality as used to assess adverse drug reactions are met. Hepatotoxicity has been described in the literature in rats involved in poisonings in the ToxIC Registry. Although Kratom is used since a long time, this is the first report of hepatotoxicity in humans.

### 40. CO-INGESTION OF OPIOIDS WITH ACETAMINOPHEN DOES NOT APPEAR TO INCREASE RISK OF ADVERSE OUTCOME FOLLOWING ACETAMINOPHEN OVERDOSE

**David M. Wood,** Shaun L. Greene, Paul I. Dargan

**Guy’s and St Thomas’ NHS Foundation Trust, London UK**

**Background:** A recent study suggested that co-ingestion of opioids with acetaminophen may delay acetaminophen absorption in those with acetaminophen overdose, leading to a lower 4 hour acetaminophen concentration [Reddick AD 2010]. The authors of this study recommended “repeated measurement of acetaminophen concentration” in patients with acetaminophen overdose who co-ingested opioids. However, there is no evidence that those with mixed acetaminophen and opioid overdose are at greater risk of acetaminophen-related toxicity.

**Methods:** Acute acetaminophen overdoses were identified retrospectively from our purpose-designed clinical toxicology database. Exclusion criteria: no data on the amount/time of acetaminophen ingested or co-ingestion of other drugs (e.g. anticholinergics) that delay gastric emptying. Data extracted included case demographics, dose of acetaminophen ingested, ethanol intake, treatment with N-acetylcyesteine, peak acetaminophen concentration, evidence of acetaminophen toxicity.

**Results:** 1035 cases met the study criteria; 851 lone acetaminophen, 184 combined acetaminophen-opioid overdose. The median (IQR) age was lower in the lone acetaminophen than the combined acetaminophen-opioid group (28 (20-40) -vs- 32 (24-44) years; p < 0.001). There was no difference in the proportion of females (50% -vs- 62%; p = 0.09), chronic ethanol use (31% -vs- 24%, p = 0.27) or those requiring NAC treatment (27% -vs- 29%, p = 0.75) between the acetaminophen-opioid and lone acetaminophen groups. The median (IQR) acetaminophen dose (10g (6.5-16g) -vs- 10g (5-15g), p = 0.023) and the peak acetaminophen concentration (22.5mg/L (0-66.3mg/L) -vs- 43mg/L (0-104mg/L), p < 0.001) was statistically but not clinically significantly lower in the acetaminophen-opioid compared to the lone acetaminophen group. The proportion of individuals in each group with acetaminophen-related toxicity is shown in the Table 1.

**Conclusions:** This study did not show an increased risk of adverse outcome in those who co-ingested opioids with acetaminophen in overdose compared to those with lone acetaminophen overdose, there was a trend to poorer outcome in the lone acetaminophen group. We feel that currently there is insufficient evidence to suggest that those with acetaminophen overdose who have co-ingested an opioid should have repeated acetaminophen concentration measurements.

#### Table 1.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Acetaminophen-Opioid</th>
<th>Lone Acetaminophen</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT &gt;1000iU/L</td>
<td>1.1%</td>
<td>1.52%</td>
<td>0.55</td>
</tr>
<tr>
<td>INR &gt;1.5</td>
<td>0%</td>
<td>1.52%</td>
<td>0.02</td>
</tr>
<tr>
<td>Creatinine &gt;1.35 mg/dL (&gt;120 micromol/L)</td>
<td>1.1%</td>
<td>1.18%</td>
<td>0.86</td>
</tr>
<tr>
<td>Regional liver unit transfer</td>
<td>0%</td>
<td>0.12%</td>
<td>0.53</td>
</tr>
<tr>
<td>Death</td>
<td>0%</td>
<td>0.12%</td>
<td>0.53</td>
</tr>
</tbody>
</table>

### 41. PHYSICOCHEMICAL STABILITY OF INTRAVENOUS FAT EMULSION IN COMBINATION WITH MEDICATIONS USED DURING RESUSCITATION

**Craig Cocchio**¹, Ann-Jeannette Geib², Kevin O. Ryan³

¹Rutgers University, New Brunswick NJ USA; ²Robert Wood Johnson Medical School, New Brunswick NJ USA; ³Rosalind Franklin University of Medicine and Sciences, North Chicago IL USA

**Background:** Intravenous fat emulsion 20% (IFE) is a potential therapy for lipophilic drug overdose. Data exists regarding IFE stability with medications in concentrations used for total parenteral nutrition [1]. However, IFE stability with drugs commonly used in resuscitation has not been assessed. This study’s purpose is to examine the in vitro physicochemical stability of various combinations of IFE and medications commonly used in resuscitation by measuring the proportion of enlarged droplets greater than 5 micrometers (PFAT5) in IFE after mixture.

**Methods:** We compared, in vitro, PFAT5 in various IFE-medication mixtures and a control. In order to assess the degree of coalescence, globule-size distribution was assessed according to method II of United States Pharmacopeia (USP) chapter 729 [2]. The volume-weighted PFAT5 values were obtained with a Beckman-Coulter Multisizer 3 automated particle counter. IFE was mixed in a 1:1 ratio with standard concentrations of medications in Table 1. IFE-medication mixtures with PFAT5 > 0.05% at time 0 and 1 hour were considered coalesced per USP chapter 729. If any results of method II exceeded PFAT5 of the control, analysis of the globule-size distribution data was conducted using a one tailed Student’s T test with a level of significance set at p < 0.05.

**Results:** All samples tested including the normal saline control, exceeded the PFAT5 upper limit set forth by the USP chapter 729 (Table 1). Visual coalescence was observed with the glucagon sample at time 0 and 1 hour, and vasopressin at time 1 hour.
Conclusion: No IFE-medication mixture, including the saline control, met the USP chapter 729 PFAT5 standard. When compared to the control, standard concentrations and doses of calcium chloride, D50W, glucagon, insulin, lidocaine, magnesium sulfate, and sodium bicarbonate created a significantly higher PFAT5 immediately after mixing. IFE should be administered in a separate administration line when given with these medications.

References
1. Trissel, L. HANDBOOK ON INJECTABLE DRUGS (13th); 2005.

42. MULTIPLE LEAD APPENDOLITHS FOLLOWING INGESTION OF LEAD SHOT: TIME COURSE AND REMOVAL BY LAPAROSCOPIC APPENDECTOMY

Randy Badillo1, William Bannerman1, Beth Conley1, Scott Schaeffer2
1Integris Baptist Medical Center, Oklahoma City OK USA; 2Oklahoma Poison Control Center, University of Oklahoma College of Pharmacy, Oklahoma City OK USA

Background: Systemic lead poisoning may occur from ingested lead foreign bodies. Rarely, these may cause a retained appendolith. The risks of lead toxicity from these appendoliths, the time course of removal remain controversial. We describe the time course of an acute ingestion of shotgun pellets with serial x-rays, blood lead, and free erythrocyte protoporphyrin (FEP) monitoring, that was managed by laparoscopic removal.

Case Report: A 15 year old male ingested a handful of lead shotgun pellets in a suicide attempt. Four days after ingestion an abdominal x-ray showed lead pellets throughout the small bowel. No interventions were initiated at this time. Four days later a repeat film showed migration of the pellets distally but a significant number were still present within the small and large bowel. A blood lead level 8 days post ingestion was 54 mcg/dL. The mother called the poison center 14 days post ingestion; the child was subsequently admitted for treatment. An x-ray on admission showed a well-organized linear and persistent collection of pellets in the RLQ. The only complaint noted by the patient was generalized abdominal pain which may have been consistent with lead colic. Whole bowel irrigation (WBI) therapy with PEG solution was begun and continued to a clear effluent. A repeat x-ray 12 hours later showed no movement of the pellets. A CT scan of the abdomen suggested the pellets were lodged in the appendix. A laparoscopic appendectomy revealed greater than 50 pellets lodged in the appendix; a repeat abdominal film post surgical procedure showed interval removal of all but 3 of the pellets.

Results: from admission blood work found a blood lead level of 41 mcg/dL and FEP level of 114 μmol/mol heme (reference normal < 70). After a short recovery the patient was discharged on succimer chelation therapy.

Discussion: This case illustrates several points:
1. Lead from shotgun pellets may produce rapid rises in blood lead level in as quickly as 8 days.
2. FEP may rise within 2 weeks following ingestion
3. Accumulation of lead pellets in the appendix may take up to 8 days supporting the advisability of early initiation of WBI in this patient population
4. Laparoscopic removal of the appendix successfully removed a large quantity of lead with short recovery

Conclusion: The ingestion of foreign bodies, including lead pellets, may be trapped in the appendix thus requiring surgical removal to decrease the potential for systemic absorption and toxicity. Early decontamination may be warranted and laparoscopic intervention may need to be considered.

43. GASTRIC PHARMACOBEZOARS IN QUETIAPINE OVERDOSE: A CASE SERIES

Alessandro Ceschi1, Christine Rauber-Luthy1, Michael Bodmer2, Hugo Kupferschmidt1, Katharina E. Hofer1
1Swiss Toxicological Information Centre, Zurich, Switzerland
2Emergency Department, University Hospital Basel, Switzerland

Background: Quetiapine is a widely used atypical antipsychotic drug, available as immediate- (IR) or extended-release (XR) tabs. Both formulations contain the hydroxypropyl methylcellulose (HPMC) matrix. Severe quetiapine poisonings involve doses ≥ 2.5 g, according to our data. Unexpected persistence of clinical effects and non-linear pharmacokinetics have been described in large quetiapine dose.

Table 1.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Quetiapine dose, galenic formulation, No of tabs</th>
<th>Coingestants</th>
<th>Endoscopic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>50 y F</td>
<td>18 g, XR, 60</td>
<td>Lorazepam</td>
<td>Large conglomerate of agglutinated tabs material of sticky consistency in the stomach</td>
</tr>
<tr>
<td>41 y M</td>
<td>15 g, XR, 50</td>
<td>Ethanol</td>
<td>Sticky mass consisting of chunks of expanded aggregated tabs in the stomach</td>
</tr>
<tr>
<td>21 y F</td>
<td>18 g, XR, 60</td>
<td>Pantoprazole</td>
<td>Mucilaginous mass of tabs all over the esophagus and in the stomach</td>
</tr>
<tr>
<td>20 y F</td>
<td>≥2.5 g, XR</td>
<td>Chloralhydrate</td>
<td>11 sticky quetiapine tabs</td>
</tr>
<tr>
<td>20 y F</td>
<td>10 g, IR, 100</td>
<td>-</td>
<td>Many tabs in clusters (white gelatinous mass) in the stomach</td>
</tr>
<tr>
<td>49 y F</td>
<td>6 g, IR, 30</td>
<td>Bupropion XR</td>
<td>Many white and white tabs in the stomach. Whole aggregated conglomerate extending up to the antrum. 15 tabs sticking to the mucosal membrane</td>
</tr>
</tbody>
</table>

References:
1. Trissel, L. HANDBOOK ON INJECTABLE DRUGS (13th); 2005.
5. NF; 2007.
overdose, attributed to either redistribution from tissues into the blood compartment or pharmacobezoar formation. We report the first case series of gastric pharmacobezoars documented by endoscopy in quetiapine overdose.

**Case Series:** A search of our database identified 457 calls related to quetiapine overdose between Jan and Dec 2010. In 61 cases the ingested dose was ≥2.5 g; XR tabs were involved in 30 cases, IR tabs in another 30, and in 1 the formulation was unknown. In 7 cases gastroscopy was performed, and in 6 of these gastric pharmacobezoars were detected (Table 1). In 3 cases the conglomerate could be completely removed, and in 3 some tabs remained sticking to the gastric mucosa.

**Discussion:** Pharmacobezoar formation after ingestion of XR tabs in overdose has been described in the literature as a rare complication. Two findings in our case series should be emphasized: first, the incidence of pharmacobezoar formation seems to be relatively high (6 out of 61 cases) in patients who ingested ≥2.5 g, and second, in 2 cases IR tabs were involved. It remains to be elucidated whether HPMC – which shows marked mucoadhesive properties and has been demonstrated to undergo a glassy-rubbery transition – may favour pharmacobezoar formation.

**Conclusions:** The formation of pharmacobezoars should be considered in the management of quetiapine overdose, with both XR and IR tabs. Further studies are required to investigate the potential role of delivery systems based on HPMC matrices in the pharmacobezoar formation.

### 44. ATROPINE EYE DROP INGESTION TREATED WITH HIGH-DOSE PHYSOSTIGMINE

**Samuel J. Stelpling**1, Jon B. Cole1, Brain A. Isaacson1, Christian P. Lintner1, Elisabeth F. Belden1

1Hennepin Regional Poison Center, Minneapolis MN USA; 2Fairview Ridge Medical Center, Burnsville MN USA

**Background:** Atropine is a competitive acetylcholine antagonist at muscarinic receptors. In overdose it can cause tachycardia and altered mental status (AMS). Physostigmine (physo) is a carbamate that reversibly inhibits acetylcholinesterase. The usual dose is 0.5-2 mg slow intravenous (IV) push, repeated every 15-40 minutes as needed. It is unusual for doses in the emergency department (ED) to exceed 4 mg. We describe a patient with a massive atropine eye drop ingestion treated with 14 mg physo, resulting in improvement of AMS and avoidance of intubation and mechanical ventilation and the potential associated morbidity with these invasive procedures.

**Case Report:** A 34 yo man presented to an ED after ingesting 150 mg of eye drop atropine. He had AMS, heart rate (HR) of 150 beats/min, flushed skin, dry mucosa, normal glucose, and temp of 100.3°F. The remainder of his exam was unremarkable. ECG showed sinus rhythm and no interval/segment abnormalities. Sedation and intubation were eminent, and in attempt to avoid the morbidity of this procedure, physo was used. Over the next 75 minutes, physo was given in 1 mg doses to a total of 11 mg. There was minimal change with the initial doses; after the 4th dose each subsequent 1 mg improved the AMS and HR, which would worsen again over the next 10 minutes, necessitating another dose. At completion of ED treatment, HR was 90, and AMS resolved. Lab studies included a negative ethanol, acetaldehyde, and urine drug screen, normal Chem 7, and serum atropine level of 240 ng/mL. He was admitted to the ICU for further care, where he required 3 additional 1 mg doses of physo for agitation. Intubation was never needed. The remainder of his hospital stay was uneventful.

**Discussion:** There is a paucity of documented atropine eye drop ingestions. Atropine toxicity is not predictable by dose. Fatalities have been reported with less than 100 mg; survival with more than 1 g. Following a 1980 report of 2 patients with cardiac arrests after cyclic antidepressant overdose where treatment included physo, its use declined. Recent literature has tempered some concern about its deleterious effects; its use has again become more common. A large total dose of physo was needed in this case, with doses repeated in rapid succession. This was likely due to the massive atropine ingestion, confirmed by the symptoms and serum atropine concentration. This case illustrates the most beneficial aspect of physo use: the ability to control agitation and reverse delirium thereby reducing the need for invasive interventions.

**Conclusion:** High doses of physostigmine may be considered in severe pure antimuscarinic toxicity to prevent the morbidity associated with the invasive interventions of intubation and mechanical ventilation.

### 45. USE OF IV. PAMIDRONATE TO MANAGE SEVERE, NONACCIDENTAL LEAD POISONING IN A CHILD WITH CHARGE SYNDROME

**Lawrence Quang, Sumana Narasimhan**

Rainbow Babies & Children’s Hospital, Cleveland OH USA

**Background:** We describe a case of severe, non-accidental childhood lead poisoning that responded to the bone antiresorptive agent pamidronate.

**Case Report:** A 22 m.o. medically complex, technology-dependent child with CHARGE Syndrome is referred to us for evaluation and treatment of lead poisoning of indeterminate etiology since 5 months of age. The child had an elevated blood lead level (BLL) of 68 mcg/dL. Response to chelation was modest after identification and removal of the perpetrator. We hypothesized significant lead redistribution from the bone compartment. Bisphosphonate antiresorptive agents that reduce demineralization in the bone remodeling process, specifically alendronate, have been effective in reducing BLLs by 20% in perinominal females. While not FDA-approved for children, pediatric academic centers have safely and effectively used bisphosphonates to treat childhood bone and mineral diseases. Specifically, cycllical I.V. pamidronate is the standard of care for children with moderate-severe osteogenesis imperfecta (OI). In this case, I.V. pamidronate was administered in the 3-day pulse described in established OI treatment protocols. There was a reduction in the pre-pamidronate median BLL of 42.1 μg/dL to 35.6 μg/dL. Four months later, a second 3-day pulse of I.V. pamidronate was administered, which decreased the median BLL to 26.5 μg/dL.

**Case Discussion:** In the conventional 3-compartment pharmacokinetic model of lead distribution, lead in the soft tissue compartment more rapidly exchanges with the blood compartment than lead in the bone compartment. However, lead isotope studies in humans have shown that lead, like calcium, is mobilized and released from the skeleton by bone resorption during pregnancy, lactation, and postmenopausal osteoporosis. Increased bone resorption also occurs during disuse osteoporosis, which occurs during prolonged reduction in mechanical loading on bone, weight bearing, and physical activity against gravity. Lack of mechanical stress on bone accelerates osteoclast-mediated bone resorption and inhibits osteoblast-mediated bone formation. This child was nonmobile for the first 26 months of life, predisposing her to increased bone resorption and associated mobilization of lead. In this case, the response to pamidronate suggests that the severe, chronic nature of this child’s lead poisoning resulted in a large lead burden in her bone compartment. This bone lead compartment subsequently redistributed more lead into the blood than the soft tissue compartment.

**Conclusions:** I.V. pamidronate may be a therapeutic option for lead poisoned children with an unusually high lead burden in the bone compartment.

### 46. INTRAVENOUS (IV) AND ORAL FORMS OF N-ACETYLCYSTEINE (NAC) HAVE SIMILAR RATES OF HEPATOTOXICITY WHEN USED TO TREAT ACETAMINOPHEN POISONING

**K. M. Reynolds, K. J. Heard, D. Albert, J. L. Green, V. K. Khatri**

Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA

**Background:** NAC is administered by both the oral and IV routes to treat acetaminophen poisoning. The Objective of this study was to determine the percentage of oral or IV NAC-treated patients who develop hepatotoxicity following acetaminophen poisoning.

**Methods:** Meta-analysis was used to determine the percentage of patients who developed hepatotoxicity (AST/ALT > 1000 IU/L) stratified by route of administration. A literature search for articles published through March 2010 was performed using Medline, EMBASE, and International Pharmaceutical Abstracts and a cited reference search. Inclusion criteria were: use of NAC in patients with a 4-24 hour acetaminophen level above the Rumack-Mathews nomogram, reported NAC route, a reported post-NAC AST/ALT level and ≥30 patients. Because time from overdose to treatment is associated with outcome, patients were stratified as early treatment (NAC ≤10 hours) or late treatment (NAC >10 hours), or as defined by the authors. Time to NAC, route, and post-NAC peak AST/ALT level were extracted from all relevant articles. To test the relationship between route, time and the interaction of route by time, a general linear mixed model was applied.
Table 1.

<table>
<thead>
<tr>
<th></th>
<th># of Patients</th>
<th>Hepatotoxicity Rate (%)</th>
<th>95 CI (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall†</td>
<td>7547</td>
<td>12.9</td>
<td>10.1–16.3</td>
</tr>
<tr>
<td>IV†</td>
<td>3594</td>
<td>13.5</td>
<td>9.8–18.4</td>
</tr>
<tr>
<td>Oral†</td>
<td>3953</td>
<td>12.0</td>
<td>8.2–17.3</td>
</tr>
<tr>
<td>Early‡</td>
<td>904</td>
<td>5.7</td>
<td>4.3–7.4</td>
</tr>
<tr>
<td>IV-Early‡</td>
<td>320</td>
<td>5.3</td>
<td>3.2–8.5</td>
</tr>
<tr>
<td>Oral-Early‡</td>
<td>584</td>
<td>5.9</td>
<td>4.2–8.1</td>
</tr>
<tr>
<td>Late‡</td>
<td>1293</td>
<td>26.2</td>
<td>23.6–29.1</td>
</tr>
<tr>
<td>IV-Late‡</td>
<td>270</td>
<td>23.3</td>
<td>11.7–41.1</td>
</tr>
<tr>
<td>Oral-Late‡</td>
<td>1023</td>
<td>26.3</td>
<td>23.7–29.1</td>
</tr>
</tbody>
</table>

†Data from all articles meeting inclusion criteria (n = 19). ‡Data from articles with IV vs. oral and early vs. late data (n = 14).

Results: 4416 citations were reviewed and data was extracted from 334 articles. 19 articles met final inclusion criteria, of which 14 contained data that could be stratified by oral vs. IV and early vs. late. The proportion of patients developing hepatotoxicity by analysis group is presented in the Table 1. There was no significant interaction and no significant effect due to route; however there was a significant effect in favor of early administration of NAC compared to late administration.

Conclusions: The rates of hepatotoxicity for oral and IV NAC are similar. Both forms are highly effective when administered within 10 hours of acetaminophen overdose. However, late administration of NAC (>10 hours post overdose) results in approximately a 4-fold increase in hepatotoxicity.

47. A RETROSPECTIVE REVIEW OF WHOLE BOWEL IRRIGATION IN PEDIATRIC PATIENTS

Jean C. Lo, Lee F. Cantrell, Cathy Ubaldo
Veterans Affairs, San Diego CA USA

Objective: Traditionally whole bowel irrigation (WBI) has been advocated for ingestions involving substances not bound with activated charcoal as well as extended release and enteric coated medications. Other than isolated case reports, little exists in the literature regarding the use of WBI in poisoned pediatric patients. The purpose of this study is to better understand the use of WBI in pediatric patients.

Methods: For this study, we queried the electronic database of a statewide poison system for cases from January 2000–December 2010, for patients 12 years old and under, with WBI coded as an utilized treatment. An Excel spreadsheet was used to record the following variables: age, sex, substances ingested; dose of exposure; time of ingestion to time of WBI initiation; initial dose of polyethylene glycol (PEG); duration of PEG; total dose of PEG; adverse drug reactions (ADR); medication level; KUB utilization; reason for exposure; admit vs discharge; length of stay if admitted; severity of medical outcome.

Results: WBI was administered in 176 patients. The top three substances ingested were calcium channel blocker (CCB)-57 (32.4%), iron-44 (25%), and antidepressants-21 (11.9%). 17 of the 21 antidepressants were sustained/extended release formulations. The number of pills ingested ranged from 0.5 to 207. WBI was administered through NG-86, PO-16, unknown-74. 18 patients had an ADR after WBI: vomiting-16, diarrhea-1, abdominal pain-1. The number of patients with emesis was NG-11, PO-1, unknown-4. The time of ingestion to time of WBI initiation ranged 2 - 74 hours. The number of patients with the following outcome per AAPCC criteria were: no effect-99, minor effect-31, moderate effect-24, major effect-9, not followed-12, unrelated effect-1. No deaths were reported. The number admitted to a HCF was 118. The reason for exposure was unintentional in 165 patients (age 1-12), and intentional in 11 patients (age 8-12). In 36 cases, KUBs were performed in which 16 were positive and in 4 cases repeat KUBs demonstrated a decrease in opacities. The number of patients with documented toxins in their effluent was 12 patients (6.8%).

Conclusion: WBI is being utilized in at least an average of 16 pediatric poisonings per year in our state. The most common toxins in which WBI was used were CCB and iron. Although ADRs occurred in 10% of patients, they were limited to transient gastrointestinal symptoms. Additional studies are required to determine the relative efficacy and preferred treatment regimen for WBI in pediatric patients.

48. CONTINUOUS RENAL REPLACEMENT THERAPY TO OVERCOME HYDROXOCOBALMIN-INDUCED PLASMA DISCOLORATION

Stephen L. Thornton, Joseph Abdelmalek, Nizar John, Amber Sanchez, Aaron Schneir

Background: Hydroxocobalamin administration is well known to cause a red-dis coloration of body fluids. One report describes the challenges in performing hemodialysis (HD) after administration due to this effect. We describe the use of continuous renal replacement therapy (CRRT) to overcome this hydroxocobalamin-related interference.

Case Report: A 33-year-old man presented comatose for which he was rapidly intubated. An initial blood gas, ordered, and reported as venous (later determined arterial) revealed: pH of 6.92, pCO2 41 mmHg, pO2 198mmHg, COHb 0.5%. Further lab tests revealed: HCO3 8 mmol/L, mild cr elevation, nl Ca, lactate 58.8 mg/dl (nl 4.3 – 19.8 mg/dl), osmolar gap 14, and urine without crystals. There was no detectable APAP, salicylate, or ETOH. For suspected toxic alcohol poisoning the patient received fomepizole with plans for emergent HD. One hour prior to HD, 5 grams of hydroxocobalamin (Cyankit®) were administered IV for potential cyanide (CN) poisoning. Within minutes of HD initiation, the blood leak detector was triggered, and internal pre-set alarms prevented further HD despite attempts at disabling and recalibrating the alarm. CRRT was initiated with a machine that allowed recalibration of the blood leak detector. The hydroxocobalamin-induced reddish plasma effluent was monitored at regular intervals to assure blood cells were not present. CRRT was performed for 5 days until intermittent HD could be performed. Initial EG concentration was later confirmed at 28 mg/dL. Renal recovery occurred after 3 weeks of intermittent HD.

Case Discussion: The patient experienced the typical plasma discoloration described with hydroxocobalamin that in this case prevented the optimal management of his ethylene glycol poisoning. In a prior case of CN poisoning in which the patient underwent HD the same challenge was encountered which was temporarily addressed by turning off the internal alarms. In our case CRRT, an alternative, but not optimal method for EG poisoning was used in which recalibration allowed for dialysis to proceed.

Conclusion: Hydroxocobalamin administration causes plasma discoloration that can be of significant consequence if HD is to be performed. We report one method of overcoming this challenge.

49. CHARACTERISTICS OF HEMODIALYSIS USE IN PEDIATRIC AND ADOLESCENT POISONINGS

Michael A. Darracq1, F. L. Cantrell2, Patrick Sweet1, John Chen1
1University of California, San Diego, San Diego CA; 2California Poison Control System, San Diego Division, San Diego CA USA

Objective: To describe the characteristics of hemodialysis use in the treatment of pediatric and adolescent poisonings.

Methods: A state poison system’s database was queried for cases in which hemodialysis (HD) was coded as performed between 2000 and 2010 for patients between 0 and 19 years of age. 100 cases were included for analysis. Cases were reviewed and abstracted for age, gender, toxin, circumstances surrounding exposure, method and indication for HD, and outcome. Outcome was defined as good- complete or anticipated resolution of all number of patients with documented toxins in their effluent was 12 patients (6.8%).

Conclusion: WBI is being utilized in at least an average of 16 pediatric poisonings per year in our state. The most common toxins in which WBI was used were CCB and iron. Although ADRs occurred in 10% of patients, they were limited to transient gastrointestinal symptoms. Additional studies are required to determine the relative efficacy and preferred treatment regimen for WBI in pediatric patients.

Background: Hydroxocobalamin administration is well known to cause a red-dis coloration of body fluids. One report describes the challenges in performing hemodialysis (HD) after administration due to this effect. We describe the use of continuous renal replacement therapy (CRRT) to overcome this hydroxocobalamin-related interference.

Case Report: A 33-year-old man presented comatose for which he was rapidly intubated. An initial blood gas, ordered, and reported as venous (later determined arterial) revealed: pH of 6.92, pCO2 41 mmHg, pO2 198mmHg, COHb 0.5%. Further lab tests revealed: HCO3 8 mmol/L, mild cr elevation, nl Ca, lactate 58.8 mg/dl (nl 4.3 – 19.8 mg/dl), osmolar gap 14, and urine without crystals. There was no detectable APAP, salicylate, or ETOH. For suspected toxic alcohol poisoning the patient received fomepizole with plans for emergent HD. One hour prior to HD, 5 grams of hydroxocobalamin (Cyankit®) were administered IV for potential cyanide (CN) poisoning. Within minutes of HD initiation, the blood leak detector was triggered, and internal pre-set alarms prevented further HD despite attempts at disabling and recalibrating the alarm. CRRT was initiated with a machine that allowed recalibration of the blood leak detector. The hydroxocobalamin-induced reddish plasma effluent was monitored at regular intervals to assure blood cells were not present. CRRT was performed for 5 days until intermittent HD could be performed. Initial EG concentration was later confirmed at 28 mg/dL. Renal recovery occurred after 3 weeks of intermittent HD.

Case Discussion: The patient experienced the typical plasma discoloration described with hydroxocobalamin that in this case prevented the optimal management of his ethylene glycol poisoning. In a prior case of CN poisoning in which the patient underwent HD the same challenge was encountered which was temporarily addressed by turning off the internal alarms. In our case CRRT, an alternative, but not optimal method for EG poisoning was used in which recalibration allowed for dialysis to proceed.

Conclusion: Hydroxocobalamin administration causes plasma discoloration that can be of significant consequence if HD is to be performed. We report one method of overcoming this challenge.

49. CHARACTERISTICS OF HEMODIALYSIS USE IN PEDIATRIC AND ADOLESCENT POISONINGS

Michael A. Darracq1, F. L. Cantrell2, Patrick Sweet1, John Chen1
1University of California, San Diego, San Diego CA; 2California Poison Control System, San Diego Division, San Diego CA USA

Objective: To describe the characteristics of hemodialysis use in the treatment of pediatric and adolescent poisonings.

Methods: A state poison system’s database was queried for cases in which hemodialysis (HD) was coded as performed between 2000 and 2010 for patients between 0 and 19 years of age. 100 cases were included for analysis. Cases were reviewed and abstracted for age, gender, toxin, circumstances surrounding exposure, method and indication for HD, and outcome. Outcome was defined as good- complete or anticipated resolution of all
toxin-related symptoms; moderate/severe permanent or anticipated long-term effects; death.

Results: 100 cases of pediatric and adolescent hemodialysis occurred during the study period. 91 patients (91%) were between 12 and 19, while 9 (9%) were <12 years of age. Males represented patients less than 12 years of age (55%) while females represented patients older than 11 (60%). The most common reason for exposure was unintentional in patients <12 (100%) and suicide in 96% of cases >12. The remaining 4 patients (4%) required HD due to adverse drug reaction. HD was performed in 22 patients (22%) for toxin-induced renal failure rather than enhanced elimination of the toxin. The most common agents involved were salicylates (29 cases, 29%) and toxic alcohols (29 cases, 29%) (ethylene glycol -23, methanol -4, polyalkylene glycol ethers -1, and brake fluid -1). Other agents included lithium (7 cases, 7%), acetaminophen (6 cases, 6%), and valproic acid (6 cases, 6%). An elevated level or rising level was the most common trigger for initiation of HD in salicylate exposures. For ethylene glycol, the most common trigger was a history of ingestion with altered mental status, osmolar gap, or acidosis. Outcomes were good in 79 cases, moderate in 10 cases with death occurring in 6 patients (3 unknown ingestions with multiorgan failure, 2 salicylate ingestions with levels >90mg/dl from time of ingestion, and 1 ethylene glycol ingestion with profound refractory hypotension during ICU stay). 5 patients were lost to follow-up.

Conclusions: In pediatric and adolescent poisonings, HD is being utilized an average of 10 times per year in our state. It is being used for a variety of intoxications, but most frequently with salicylates and ethylene glycol. It is also used for enhanced toxin elimination and renal support secondary to toxin effects as indicated by history in conjunction with clinical status and lab data. While outcomes were generally favorable, deaths did occur despite HD. Additional studies are warranted to determine the exact role of HD in pediatric and adolescent poisonings.

50. ACTIVATED CHARCOAL DOES NOT REDUCE DURATION OF PHENYTOIN TOXICITY IN HOSPITALIZED PATIENTS

Jessica Pettrey1, Ruddy Rose2, Brandon Wills2, Kirk Cumpston2
1VCU School of Pharmacy, Richmond VA USA; 2Virginia Poison Center, Richmond VA USA

Background: Phenytoin toxicity is rarely fatal but is a common cause of hospitalization. Due to its non-linear elimination, patients often have prolonged hospital stays. Currently there is no antidote for phenytoin toxicity and the mainstay of therapy is supportive care. Several studies suggest phenytoin elimination can be enhanced by multidose activated charcoal (MDAC) however, its clinical utility is controversial. The aim of this investigation was to evaluate the efficacy of activated charcoal on time to resolution of phenytoin toxicity.

Methods: A retrospective review of poison center cases between January 1, 2002 and December 31, 2009 was performed. Hospitalized patients with phenytoin concentrations >20 mg/L were included. Patients with co-ingestants or <6 hours of observation were excluded. Patients were grouped by use of single dose activated charcoal (SDAC), MDAC, and no charcoal (NoAC). Clinical data included acuity, route of administration, dose (if known), serum phenytoin levels, clinical effects and number of charcoal doses. The composite endpoints were time to resolution of symptoms, hospital discharge or the case was closed from a toxicology management standpoint.

Results: 355 cases were identified, 198 of which were phenytoin-only. An additional 85 cases were excluded for not meeting other inclusion criteria leaving 113 cases included for analysis. There were 69 NoAC, 13 SDAC, and 31 MDAC cases. The groups were similar in age and chronicity of exposure. Mean (+ SD) peak phenytoin concentrations were 42 ± 12 mg/L, 41 ± 11 mg/L and 42 ± 11 mg/L for NoAC, SDAC and MDAC, respectively. Clinical effects for each treatment group are listed in Table 1. Mean time to reach a study endpoint was 39 hours (95% CI 31-48), 52 hours (95% CI 36-68), and 60 hours (95% CI 45-75) for NoAC, SDAC and MDAC, respectively. The treatment groups were similar with respect to peak phenytoin concentrations and prevalence of clinical effects. In this observational series, use of activated charcoal was associated with increased time to reach the composite endpoint. The potential for incomplete data and heterogeneous treatment groups may confound the validity of these findings.

Conclusion: This retrospective case series failed to demonstrate a beneficial effect of activated charcoal on time to resolution of phenytoin toxicity.

51. USE OF INTRAVENOUS SILIBININ (LEGALON SIL) FOR TREATMENT OF AMATOXIC MUSHROOM POISONING IN NORTHERN CALIFORNIA

Steven R. Offerman1, Timur Durrani1, Ben Tsutaoka2, Brian M. Kaiser2
1California Poison Control System, San Francisco CA USA; 2University of California San Francisco Medical Center.

San Francisco CA USA; 3Kaiser Permanente Northern California, Sacramento CA USA

Background: Amatoxic mushroom poisoning is a rare but potentially deadly occurrence in Northern California. When poisoning occurs it frequently involves multiple victims. Few treatment options exist and most of those are unproven. Intravenous (IV) Silibinin has been approved for use in Europe since 1984, but is not FDA approved in the U.S. Silibinin is the only treatment associated with lower mortality following amatoxic mushroom poisoning.

Case Reports: We report the case of an Asian family who presented with amatoxic mushroom poisoning in Northern California. Two family members (grandparents) foraged for the mushrooms and prepared them in a stew. 4 family members complained of symptoms (abdominal pain, vomiting, diarrhea) after eating the stew and 3 developed hepatotoxicity. The 73 year-old grandfather and 6 year-old grandson were the most severely affected, presenting >12 hours after ingestion. All three patients who developed hepatotoxicity were treated with aggressive fluid hydration and IV silibinin (Legalon SIL; Madaus Pharmaceuticals). The 6 year-old patient was transferred to the nearest liver transplantation center where he developed encephalopathy and was listed for transplant. He was given high-dose IV penicillin in addition to IV silibinin. By day 5, hepatotoxicity in all patients began to improve and by day 7 all had been discharged. Long term follow-up all affected patients have fully recovered and have no further symptoms. Laboratory assay confirmed the presence of amatoxin in a mushroom sample and urine of the two most severely affected individuals.

Case Discussion: We used IV silibinin to treat three cases of confirmed amatoxic mushroom poisoning with good outcomes. We believe that the use of early, aggressive IV fluid hydration and IV silibinin contributed to the excellent outcomes we observed. Silibinin appeared to be safe and was well tolerated. At this time silibinin is not FDA approved and can only be given under emergency investigational use conditions in the U.S.

Conclusion: IV silibinin should be considered for use in cases of suspected amatoxic mushroom poisoning. The FDA should consider orphan drug approval of this medication.

52. BRONCHOALVEOLAR LAVAGE AND EXOGENOUS SURFACTANT TO TREAT CHARCOAL ASPIRATION

Vanessa Madrigal, Adam Himebauch, John McCloskey, Fred Henretig, Kevin C Osterhoudt
The Children’s Hospital of Philadelphia, Philadelphia PA USA

Background: Aspiration pneumonitis may complicate drug overdose. Aspiration of antidotal charcoal has been associated with respiratory failure,
Clinical Toxicology Downloaded from informahealthcare.com by University of Zuerich on 09/15/11

**Objections:** To determine how US PCC have incorporated IFE as a treatment strategy for poisoning.

**Methods:** A closed-format multiple-choice survey instrument was developed, piloted, revised, and then sent electronically to every medical director of an accredited US PCC using SurveyMonkey in March 2011; addresses were obtained from the AAPCC listser, participation was voluntary and remained anonymous; 3 reminder invitations were sent during the study period. Data were analyzed using descriptive statistics.

**Results:** 45 of 57 (79%) PCC medical directors completed the survey. 100% of respondents felt that IFE therapy played a role in the acute overdose setting. 30 (67%) PCC have a protocol for IFE therapy. 29 (97%) recommend an initial bolus of 1.5mL/kg of a 20% lipid emulsion. 28 (95%) PCC recommend an infusion of lipids, and 27/28 PCC recommend an initial infusion rate of 0.25mL/kg of a 20% lipid emulsion. 33 (73%) felt that IFE had no clinically significant side effects at a bolus dose of 1.5mL/kg (20% emulsion). 44 (98%) stated that IFE had no clinically significant side effects at a bolus dose of 1.5mL/kg (20% emulsion). 33 (73%) felt that IFE had no clinically significant side effects at a bolus dose of 1.5mL/kg (20% emulsion). 28 (93%) PCC recommend titrating. 30 (67%) PCC have a protocol for IFE therapy. 29 (97%) recommend an initial bolus of 1.5mL/kg of a 20% lipid emulsion. 28 (95%) PCC recommend an infusion of lipids, and 27/28 PCC recommend an initial infusion rate of 0.25mL/kg of a 20% lipid emulsion. 33 (73%) felt that IFE had no clinically significant side effects at a bolus dose of 1.5mL/kg (20% emulsion). 44 (98%) of directors felt that the “lipid sink” mechanism contributed to the clinical effects of IFE therapy, but 26 (58%) felt that there was a yet undiscovered mechanism that likely contributed as well. In a scenario with cardiac arrest due to a single xenobiotic, directors stated that their center would always or often recommend IFE after overdose of bupivacaine (43; 96%), verapamil (36; 80%), amitriptyline (31; 69%), or an unknown xenobiotic (12; 27%). In a scenario with significant hemodynamic instability due to a single xenobiotic, directors stated that their PCC would always or often recommend IFE after overdose of bupivacaine (40; 89%), verapamil (28; 62%), amitriptyline (25; 56%), or an unknown xenobiotic (8; 18%). Directors stated that medical conferences (28; 64%) and published human case reports (25; 56%) had the most influence on their understanding of IFE.

**Conclusions:** IFE therapy is being recommended by US PCC. Protocols and dosing regimens are nearly uniform. Most directors feel that IFE is safe but are more likely to recommend IFE in patients with cardiac arrest than in patients with severe hemodynamic compromise. Medical conferences and published human case reports have the largest influence on directors regarding IFE therapy. Further research is warranted.

54. **RESOLUTION OF ANTICHOLINERGIC INDUCED ILEUS AFTER NEOSTIGMINE ADMINISTRATION**

J. Michael Kowalski, Serge-Emile Simpson
Albert Einstein Medical Center, Philadelphia PA USA

**Background:** Multi-dose activated charcoal (MDAC) is indicated for treatment of serious carbamazepine (CBZ) overdose. CBZ and quetiapine can cause impaired gastrointestinal (GI) motility via peripheral antimuscarinic action. A peripheral cholinergic agonist like neostigmine may have an adjunctive role in MDAC therapy for CBZ overdose.

**Case Report:** A 22-year-old male with bipolar disorder was found unresponsive in bed by his family. Empty bottles of CBZ and quetiapine were discovered near the patient. The patient was transported to the emergency department via EMS and required only supplemental O2 en route. Initial vital signs were 112 bpm, 112/60 mm Hg, and 38.0°C. Physical exam was significant for dry mucosal membranes, decreased level of consciousness and agitation with physical stimulation. The serum CBZ level was 18.9 mg/L (4-10 mg/L). The patient was intubated and given 50g of activated charcoal via nasogastric tube. Intermittent self-limited tonic-clonic seizure activity was observed in the ICU. MDAC was administered every four hours for 24 hours. CBZ levels increased to 46.5 mg/L. On day two, MDAC was discontinued and a total of 4 L of polyethylene glycol were administered. By day three, the patient had no bowel movement and a distended abdomen without bowel sounds. X-ray revealed an adynamic ileus. Two milligrams of intravenous neostigmine were given at 48 hours of care and the patient had a substantial bowel movement within ten minutes. Over the next 48 hours, the patient demonstrated clinical improvement and CBZ levels declined.

**Discussion:** Neostigmine is a peripherally acting acetylcholinesterase inhibitor used to reverse antimuscarinic effects. Decreased bowel activity, and ileus, may occur with CBZ and quetiapine overdoses. MDAC has been recommended for a minority of overdose situations including CBZ, theophylline, phenobarbital, danopansone or quinine. Delayed gut emptying, from muscarinic receptor inhibition retardation elimation. Concretion formation from MDAC or large medication ingestions may also prolong drug elimination. Additionally, ingestion of xenobiotics with anticholinergic properties may contribute to bowel obstruction and eventual perforation. Anticholinergic symptoms were present throughout the patient’s course. There was immediate resolution of the ileus and rapid decline in serum CBZ levels following neostigmine administration. Given the toxicodynamics of quetiapine and CBZ, antimuscarinic receptor activity was likely the etiology for the ileus.

**Conclusion:** This case demonstrates that neostigmine may be considered as an adjunct to improve the effectiveness of MDAC therapy in carefully selected patients demonstrating anticholinergic toxicity.

55. **FAILURE OF HIGH DOSE INSULIN AND INTRAVENOUS FAT EMULSION IN 2 PATIENTS WITH POISON-INDUCED CARDIOGENIC SHOCK**

Jon B. Cole, Heather Ellsworth, Kristin M. Engelbrecht, Samuel J. Stellpflug
Regions Hospital, Saint Paul MN USA

**Background:** High Dose Insulin (HDI) and Intravenous Fat Emulsion (IFE) are promising therapies for the treatment of Poison-Induced Cardiogenic Shock (PICS). The American College of Medical Toxicology recently
published a position that IFE is "a reasonable consideration for therapy, even if the patient is not in cardiac arrest." We describe 2 cases of PICS that died despite early aggressive therapy with IFE and HDI. In both scenarios, IFE was given while the patient still had a pulse and immediate cardiac arrest ensued.

**Case 1:** A 50 year old woman presented after an overdose (OD) of 80 total tabs of metoprolol 50 mg and bupropion XL 150 mg. She had profound bradycardia and hypotension refractory to IV fluids, 41 mEq of Ca\(^{2+}\), 10 mg of glucagon, and a HDI infusion of 10 U/kg/hr. With a pulse of 40 bpm and mean arterial pressure (MAP) of 30 mmHg, a bolus of 1.5 mL/kg of 20% IFE was given. Within 30 seconds she had a bradysystolic arrest. Pulses returned after 4 min of CPR. Despite catecholamines, a transvenous pacemaker (TP), and an intra-aortic balloon pump (IABP), the patient died on hospital day (HD) 4 of multi-system organ failure (MSOF). Post-mortem drug levels were: bupropion 130 ng/ml, hydroxybupropion 480 ng/ml.

**Case 2:** A 53 yo man presented after an OD of a one-month supply of diltiazem SR 120 mg and propranolol 20 mg. He had profound hypotension and bradycardia that was refractory to IV fluids, 32 mEq of Ca\(^{2+}\), a HDI infusion of 10 U/kg/hr, 2 mg each of epinephrine and atropine, and a dopamine drip. With a pulse of 30 bpm and a MAP of 40 mmHg a bolus of 1.5 mL/kg of 20% IFE was given. Within 1 minute he had a bradysystolic arrest. Pulses returned after 5 min of CPR. Despite 4 vasopressors, TP and IABP, the patient died on HD 7 of MSOF. A propranolol level on HD 2 was 53 ng/ml (therapeutic range 100-200); a diltiazem level on HD 5 was 100 ng/ml (therapeutic range 100-200).

**Discussion:** In severe cases of PICS clinicians may consider the use of both HDI and IFE. IFE has been described to work by the "lipid sink" mechanism. The relationship between IFE and HDI is unclear, but it is possible our patients were dependent upon HDI for inotropic support and IFE rendered HDI ineffectual. It is also possible the brief lack of oxygen in the lipid-laden blood circulating in the coronary vessels contributed to the arrests. The arrests also could have occurred without influence from a HDI/IFE interaction or the IFE alone. However, the temporal relationship of the arrests following IFE in these cases is difficult to ignore.

**Conclusion:** We report 2 cardiac arrests after use of HDI followed by IFE. An interaction between IFE and HDI must be contemplated. Additionally, these cases should be considered with caution when using IFE in pre-arrest scenarios.

### 56. FOMEPIZOLE TO PREVENT WORSENING LACTATE PRODUCTION IN A PATIENT WITH DISULFIRAM RELATED KETOACIDOSIS

**Rama Rao**\(^1\), Rana Biary\(^1\), Rafaele Milizia\(^1\), Colleen Birmingham\(^2\), Jane M. Prosser\(^1\), Brenna M. Farmer\(^1\), Nicole Bouchard\(^1\), Lewis Nelson\(^2\), Robert S. Hoffman\(^2\), Mary Ann Howland\(^1\)

\(^1\)New York Presbyterian Hospital, New York NY USA; \(^2\)New York City Poison Control Center, New York NY USA; \(^3\)St John's College of Pharmacy, Queens NY USA

**Background:** Alcoholic ketoacidosis occurs during the metabolism of ethanol in the setting of thiamine deficiency and poor caloric intake. Concomitant lactic acidosis may occur as thiamine is required to facilitate conversion of lactate to pyruvate. Metabolism of ethanol in the setting of thiamine deficiency drives acidosis may occur as thiamine is required to facilitate conversion of lactate to pyruvate. The patient denied ethylene glycol ingestion, a metabolite of which may cause lactate artifact. An ethylene glycol concentration was negative. Serum lipase was also negative.

**Case Discussion:** We describe a unique use of fomepizole in the setting of disulfiram related alcoholic ketoacidosis. Disulfiram inhibits acetaldheyde dehydrogenase which is associated with adverse reactions in the presence of ethanol. The accumulation of acetaldheyde induced vomiting and poor nutritional intake leading to ketosis. Fomepizole, an inhibitor of alcohol dehydrogenase, prevented further ethanol metabolism.

**Conclusions:** We propose that the reduction in ethanol metabolism was associated with a more favorable NAD+/NADH ratio which, when combined with thiamine and dextrose, allowed the conversion of lactate to pyruvate as well as the correction of ketosis.

### 57. SEVERE ETHYLENE GLYCOL TOXICITY TREATED WITH SINGLE-DOSE FOMEPIZOLE AND HEMODIALYSIS

**Steven J. Walsh**, Michael G. Abesamis, Michael J. Lynch, Anthony F. Pison

University of Pittsburgh Medical Center, Pittsburgh PA USA

**Background:** Ethylene glycol (EG) toxicity causes metabolic acidosis, renal failure, and death. Standard treatment of severely-poisoned patients includes fomepizole (4MP) and adjunctive hemodialysis (HD). 4MP's manufacturer recommends dosing every four hours during HD. We feel this adds little benefit given that HD removes EG and its metabolites in real time. We describe a case of severe EG toxicity treated with a sole initial dose of 4MP followed by a 19½-hour course of HD without untoward effect.

**Case:** Our patient was a 63 year-old man suspected of ingesting a large amount of EG. He was found obtunded with a mug adjacent to an empty antifreeze bottle. He was intubated at the scene by EMS due to lethargy. Vital signs were normal at presentation. Arterial blood gas analysis demonstrated pH 7.04, CO\(_2\) 13, and base deficit 17. Renal function was normal. EG concentration was 2 650 mg/dL two hours post-ingestion. Ethanol, methanol, isopropanol, and propylene glycol were undetectable. No coingestants were detected by gas chromatography/mass spectroscopy. The patient was aggressively hydrated and administered 50 mEq sodium bicarbonate with prompt correction of acidemia (pH\(_7\), 7.33, HCO\(_3\)\(^{-}\), 21). 15 mg/kg of 4MP was concurrently dosed. Thiamine and pyridoxine were also given. He underwent emergent HD for 19½ consecutive hours, during which no additional 4MP was given. pH and renal function were monitored every 4 hours during HD. In anticipation of abnormalities, 4MP was immediately available; none was needed. EG concentration was 9 mg/dL upon cessation of HD.

The patient never developed renal injury or other significant laboratory abnormality. His mental status rapidly improved, and he was extubated after completion of HD. The remainder of his hospital course was complicated only by mild aspiration pneumonitis treated with pulmonary toilet and a short course of oral antibiotics. He was transferred to inpatient psychiatry.

**Conclusion:** We report a case of severe EG toxicity treated with a lone loading dose of 4MP followed by HD with favorable outcome. EG and its metabolites are eliminated by HD in real time; therefore, we suspect that 4MP has little added benefit once HD has been instituted. Ours is not a mild case of EG toxicity; the patient had the highest reported concentration in the English literature with survival. Yet, only a single dose of 4MP was provided during his course. Further investigation into eliminating 4MP from the treatment algorithm once HD has begun may be warranted.

### 58. FAILURE OF LIPID EMULSION THERAPY TO TREAT A METFORMIN OVERDOSE

**Susan N. Miller**, Michael I. Greenberg

Drexel College of Medicine, Philadelphia PA USA

Background: Metformin overdose is a frequent but often underdiagnosed scenario. The incidence of metformin overdose related mortality is high. Metformin overdose can result in a number of serious and life-threatening effects, including acute renal failure, hypoglycemia, and electrocardiographic changes. In severe cases of PICS clinicians may consider the use of both HDI and IFE. IFE has been described to work by the "lipid sink" mechanism. The relationship between IFE and HDI is unclear, but it is possible our patients were dependent upon HDI for inotropic support and IFE rendered HDI ineffectual. It is also possible the brief lack of oxygen in the lipid-laden blood circulating in the coronary vessels contributed to the arrests. The arrests also could have occurred without influence from a HDI/IFE interaction or the IFE alone. However, the temporal relationship of the arrests following IFE in these cases is difficult to ignore.

**Conclusion:** We report 2 cardiac arrests after use of HDI followed by IFE. An interaction between IFE and HDI must be contemplated. Additionally, these cases should be considered with caution when using IFE in pre-arrest scenarios.
Background: Lipid emulsion therapy is the currently used as a last resort in treatment of severe toxicities. It is not known for which drug overdoses lipid emulsion therapy would work best and the mechanism of action of lipid emulsion therapy is not completely understood. 

Case Report: A 39-year-old man presented approximately 4 hours after ingesting up to 90 grams of metformin in a suicidal attempt. Bicarbonate therapy was begun 14 hours after ingestion. Starting approximately 24 hours after ingestion, he received continuous dialysis for 12 hours, followed by continuous veno-venous hemodialysis. Despite maximal therapy with bicarbonate, THAM, norepinephrine, vasopressin, epinephrine, and dialysis, the patient continued to have profound lactic acidosis and hemodynamic shock. On hospital day 2, 20% lipid emulsion (Intralipid) at 1.5 mL/kg bolus followed by 0.25 mL/kg/min for 60 minutes was infused. The decision to use lipid emulsion therapy was based on the knowledge that metformin has a high volume of distribution and the patient’s clinical status was deteriorating despite maximal therapy. The octanol-water partition coefficient for metformin was not known at the time of the infusion. The lipid emulsion therapy had no effect on hemodynamic status or lactic acidosis. The patient expired the next day.

Case Discussion: We propose that the failure of the lipid emulsion therapy to treat a metformin overdose is because metformin, despite its high volume of distribution, has a low octanol-water partition coefficient and is too hydrophilic to be treated with lipid emulsion therapy. For metformin, the volume of distribution is reported to be greater than 3 L/kg and octanol-water partition coefficient is 0.05. Metformin is also excretion unchanged by the kidneys and is hemodialyzable.

Conclusions: Lipid emulsion therapy should be considered in severe toxicities, however the drug profile should be carefully considered. The octanol-water partition coefficient, hydrophilicity, and whether a drug is hemodialyzable may indicate that an overdose with that drug may not be amenable to lipid emulsion therapy.

59. SERUM DIGOXIN LEVEL SHOULD NOT GUIDE THERAPY
Michael J. Lynch1, Robert D. Cannon1
1Lehigh Valley Health Network, Bethlehem PA USA; 2University of Pittsburgh, Pittsburgh PA USA

Background: Interpreting serum digoxin levels, particularly those drawn within 6 hours of ingestion, in order to predict clinical toxicity or determine need for administration of digoxin-specific antibody fragments (Dig Fab) can be difficult. However, treatment with Dig Fab based on serum digoxin level is recommended for a level >15 ng/mL at any time or >10 ng/mL more than 6 hours from the time of ingestion. We report a case of acute-on-chronic digoxin toxicity in a woman including a two hour digoxin level of 17.4 ng/mL after ingestion showed digoxin level 17.4 ng/mL, potassium 4.7 mMol/L, creatinine 1.9 mg/dL. Intravenous fluids and ondansetron were given and the patient was transferred to a regional toxicology treatment center. Vital signs were temperature 36.5°C, pulse 66 beats per minute, blood pressure 134/68 mmHg, respirations 18 breaths per minute, pulse oximetry 98% on room air. Serum digoxin level at 9.5 hours was 7.6 ng/mL with potassium 4.2 mMol/L. Electrocardiogram showed atrial fibrillation with ventricular pacemaker, rate of 72, and frequent premature ventricular complexes. The patient was treated symptomatically and observed overnight in the intensive care unit. Serial digoxin levels decreased over the following 26 hours to 4.0 with resolution of symptoms. The patient was discharged fewer than 48 hours from the time of ingestion.

Discussion: Current recommendations for the use of Dig Fab include a serum digoxin level greater than 15 ng/mL at any time following ingestion. By that criterion, treatment would have been indicated in this patient, however antidotal therapy was not required and hospitalization was not prolonged. The potential for adverse effects of Dig Fab treatment such as exacerbation of underlying heart failure or atrial tachydysrhythmias in addition to the cost of administration must be considered when determining the need for treatment. The decision to initiate Dig Fab therapy is more appropriately based upon physiologic parameters and serum potassium measurement than specific levels. 

Conclusion: Despite current recommendations, serum digoxin level, particularly pre-distribution, should not be used as an indication for Digoxin-specific antibody fragment administration.

60. EXTENDED RELEASE VENLAFAXINE INGESTION RESULTING IN GASTRIC BEZOAR
Nancy G. Murphy, Thelma Sonier, Kimberley Sheppard, Ruth Bona
IWK Regional Poison Centre, Halifax NS Canada

Background: Medications with sustained release formulations contribute to delayed and prolonged toxicity. Pharmacocoezors, though uncommonly reported, may also contribute to the duration of toxicity; it is unknown to what extent pharmaceutical excipients predispose to this phenomenon. We present a case of a large Effexor XR™ ingestion resulting in a gastric bezoar requiring endoscopic removal.

Case Summary: 47 year old female with a history of alcohol abuse and prior suicide attempts presented approximately 4 hours after reportedly ingesting 20 grams of Effexor XR™ with alcohol. Her medications on admission included Effexor XR™ 225 mg od, quetiapine 25 mg qHS, and diazepam 10 mg qid prn. In the emergency department, she was confused, tachycardic, and hallucinating with an EIOH level of 57 mmol/L. Activated charcoal, diazepam and cyproheptadine were administered. Whole bowel irrigation with 2 liters of PEG-lyte solution were given via NG tube but was not continued. In ICU, large amounts of IV diazepam were required for agitation and persistent sinus tachycardia, with daily doses of up to 265 mg. Rhabdomyolysis was diagnosed with a peak creatine kinase of 10400; no renal complications ensued. On day 3 post-ingestion, acute respiratory distress developed and endotracheal intubation was performed. Bilateral infiltrates on chest Xray were noted, as well as possible free air under the left hemidiaphragm. A CT of the abdomen was done to rule out intraabdominal pathology and a bezoar in the stomach was seen. Gastroenterology was consulted and an endoscopy was performed on day 4 post ingestion: intact pills in casings in the esophagus and a huge bezoar in the stomach were noted. The bezoar was broken up with forceps and a power wash. Over 90 minutes, 1700 mL of pill material were successfully extracted. The patient improved and was extubated 3 days later.

Case Discussion: Alcohol withdrawal may have been a factor in this patient’s ongoing delirium, tachycardia, and requirement of high doses of diazepam. In addition, it is unknown whether quetiapine was ingested. However, the endoscopic findings of a large bezoar support that venlafaxine toxicity contributed to the clinical course in ICU. It is unknown whether decontamination procedures influence the formation of bezoars. However, in selected patients with large ingestions of extended release products, consideration of early, aggressive decontamination and imaging to diagnose pharmacobezoars may be warranted.

Conclusion: Large ingestions of extended release venlafaxine can result in the formation of gastric bezoars.

61. NAC AD NAUSEUM: A CASE OF REPETITIVE POTENTIALLY TOXIC ACETAMINOPHEN INGESTIONS.
Matthew Valento1, Michelle Zell-Kanter1, Tony Burda2, Jerrold B. Leikin3
1Toxikon Consortium, Cook County Hospital (Stroger), Chicago IL USA; 47 year old female with a history of alcohol abuse and prior suicide attempts presented approximately 4 hours after reportedly ingesting 20 grams of Effexor XR™ with alcohol. Her medications on admission included Effexor XR™ 225 mg od, quetiapine 25 mg qHS, and diazepam 10 mg qid prn. In the emergency department, she was confused, tachycardic, and hallucinating with an EIOH level of 57 mmol/L. Activated charcoal, diazepam and cyproheptadine were administered. Whole bowel irrigation with 2 liters of PEG-lyte solution were given via NG tube but was not continued. In ICU, large amounts of IV diazepam were required for agitation and persistent sinus tachycardia, with daily doses of up to 265 mg. Rhabdomyolysis was diagnosed with a peak creatine kinase of 10400; no renal complications ensued. On day 3 post-ingestion, acute respiratory distress developed and endotracheal intubation was performed. Bilateral infiltrates on chest Xray were noted, as well as possible free air under the left hemidiaphragm. A CT of the abdomen was done to rule out intraabdominal pathology and a bezoar in the stomach was seen. Gastroenterology was consulted and an endoscopy was performed on day 4 post ingestion: intact pills in casings in the esophagus and a huge bezoar in the stomach were noted. The bezoar was broken up with forceps and a power wash. Over 90 minutes, 1700 mL of pill material were successfully extracted. The patient improved and was extubated 3 days later.

Case Discussion: Alcohol withdrawal may have been a factor in this patient’s ongoing delirium, tachycardia, and requirement of high doses of diazepam. In addition, it is unknown whether quetiapine was ingested. However, the endoscopic findings of a large bezoar support that venlafaxine toxicity contributed to the clinical course in ICU. It is unknown whether decontamination procedures influence the formation of bezoars. However, in selected patients with large ingestions of extended release products, consideration of early, aggressive decontamination and imaging to diagnose pharmacobezoars may be warranted.

Conclusion: Large ingestions of extended release venlafaxine can result in the formation of gastric bezoars.

61. NAC AD NAUSEUM: A CASE OF REPETITIVE POTENTIALLY TOXIC ACETAMINOPHEN INGESTIONS.
Matthew Valento1, Michelle Zell-Kanter1, Tony Burda2, Jerrold B. Leikin3
1Toxikon Consortium, Cook County Hospital (Stroger), Chicago IL USA; 2University of Pittsburgh, Pittsburgh PA USA; 3University of Pittsburgh, Pittsburgh PA USA
She had previously presented for self-poisoning, but without ingestion of APAP-containing products. Over the next 39 months, she presented to emergency departments in the same region on 35 separate visits reporting overdose of APAP with diphenhydramine. At presentation for these visits, she reported taking between 50 and 200 tablets, often over several hours and not as a single ingestion. Coingestions were uncommon (10/35 visits) and included olanzapine, lorazepam, levitiracetam, tramadol, and trazodone. Serum ethanol was positive on only 3 visits, at low concentrations (5-46 ug/dl). She usually presented with drowsiness and tachycardia (pulse on arrival 102-157 bpm, mean 133), likely from diphenhydramine. Average initial serum APAP concentration was 141.2 ug/ml (range 24-327 ug/ml), though time of ingestion was frequently unclear. The patient rarely showed Objective signs of acute hepatotoxicity, with an average initial SGOT of 45 U/L (range 19-172 U/L), though she did complain of nausea and vomiting in 67% (23/35) of presentations.

The patient was treated with IV NAC (21-hour protocol) shortly after arrival on each presentation. She was occasionally offered oral NAC but refused this. She did not experience adverse effects during any administration of this antidote, and never developed clinically worrisome hepatotoxicity. She was routinely cleared for psychiatric assessment after a single course of IV NAC.

Case Discussion: Repetitive NAC therapy was effective and well tolerated in this patient. There was no long-term liver toxicity from multiple ingestions of potentially toxic amounts of APAP. To our knowledge, no other patient has received more courses of IV NAC. There was no long-term liver toxicity from multiple ingestions of APAP with diphenhydramine.

Conclusion: IV NAC is safe and effective when used multiple times in the same patient.

62. RETROSPECTIVE REVIEW OF ECONOMIC IMPACT WITH CROTALIDAE POLYVALENT IMMUNE FAB (FABAV) ANTIVENOM IN PEDIATRIC TRANSFER PATIENTS

Anh-Vuong Ly1, James Rhee2, Tam Nguyen1, Susan Smith1, Eun Taik Ha1, Sean Bush1
1Loma Linda University, School of Pharmacy 2Loma Linda University, School of Medicine; 3Loma Linda University, Medical Center

Background: Currently, Crotalidae Polyvalent Immune Fab (FABAV) is the only available agent used for snakebite envenomations in North America. Although FabAV has been shown to be safe and effective in the pediatric population, there is limited post-market evidence on the economic impact of FabAV in pediatric patients. One vial of FabAV is associated with an average wholesale price of approximately $2,400. Due to its high cost and rarity of use, other institutions may not always stock an adequate supply of FabAV. An inadequate loading dose of FabAV may lead to insufficient initial control of symptoms and result in adverse patient consequences which may translate to a significant financial burden for both the hospital and the patient. The purpose of this study is to evaluate the economic impact of FabAV in pediatric snakebite transfer patients compared to the patients treated at the authors’ institution.

Methods: This single-centered, retrospective chart review included all snakebite patients less than 13 years of age treated with FabAV in the emergency department from 2001 to 2010. Additional FabAV therapy, adverse events and complications associated with lack of initial control were used to calculate the economic impact of initially uncontrolled patients. Data analysis will include descriptive statistics, proportions, means, and ranges.

Results: A total of 34 patients were included in the analysis, including 22 transfer patients and 12 non-transfer patients. The patients were similar in age and weight, and the majority of patients were male. On average, fewer transfer patients (36%) received initial control than did non-transfer patients (42%). Among the transfer patients, there was also a corresponding higher incidence of complications, including more severe hematologic abnormalities. Approximately 222 more vials of FabAV were used to treat the transfer patients, which is an average of 4 vials per patient. Among the transfer patients, a total of 170 more vials of FabAV were used to treat patients who presented without initial control, which is an average of 6 vials per patient. There was also a higher incidence of complications among the transfer patients who presented without initial control as compared to patients who received initial control.

Conclusions: Patients who do not receive initial control before transfer are more likely to require additional FabAV therapy and treatment of complications, which results in a significant economic burden to both the hospital and the patient.

63. CONTINUOUS VENO-VENOUS HEMODIALYSIS (CVVHD) AFTER ACUTE-ON-CHRONIC LITHIUM OVERDOSE

Michael R. Christian1, Trevonne M. Thompson2
1The Toxikon Consortium, Chicago IL USA 2University of Illinois at Chicago, Chicago IL USA

Background: Lithium overdose is commonly treated with intermittent hemodialysis (IHD). However, serum lithium levels often rebound to near pre-dialysis levels after a conventional IHD session (rebound phenomenon). With advances in medical technology, new forms of enhanced elimination have been developed. Experience with continuous veno-venous hemodialysis (CVVHD) in lithium overdose is limited.

Case Report: 33 year-old female physician presented to ED 1.5 hours after ingestion of 10 tablets of extended release lithium 450mg. Patient also admitted to taking 1800mg BID (twice her daily dose) for four days prior to overdose. Patient was lethargic but arousable with initial BP of 100/46 and HR of 47 bpm. Exam was notable for mild tremor and brisk reflexes. Initial labs revealed: Na = 135, K = 3.8, Cl = 102, HCO3 = 29, BUN = 12, Cr = 0.9, and lithium level of 3.8 mmol/L. Patient was treated with normal saline. After two hours, a repeat lithium level was 4.5, and the patient’s mental status worsened. Nephrology was consulted and CVVHD was initiated.

Time post-ingestion (hrs)

<table>
<thead>
<tr>
<th>Serum lithium (mmol/L)</th>
<th>1.9</th>
<th>3.8</th>
<th>4.5</th>
<th>4.9</th>
<th>2.7</th>
<th>2.1</th>
<th>1.5</th>
<th>0.7</th>
<th>0.6</th>
<th>0.5</th>
<th>&lt;0.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVVHD initiated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVVHD stopped</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = CVVHD initiated ** = CVVHD stopped.

Patient’s lithium levels declined during the 29-hour CVVHD session and no rebound phenomenon occurred. Patient fully recovered and was discharged to Psychiatry on hospital day #4.

Case Discussion: Diffusion of lithium out of the CNS may take longer than a conventional four-hour session of IHD. We report a case in which a 29-hour session of CVVHD was successfully used to treat an acute-on-chronic lithium overdose and no rebound phenomenon occurred.

Conclusions: The enhanced elimination of lithium by CVVHD requires further study.

64. SUCCESSFUL CONSERVATIVE TREATMENT OF FOUR CASES OF INTERNATIONAL SMUGGLING OF ILLICIT DRUGS

Nasrin Pakravan1, Mahmoud Omidi2
1Imam Teaching Hospital-Mazandaran University of Medical Science, Sari Iran 2School of Pharmacy-Mazandaran University of Medical Science, Sari Iran.

Background: International smuggling of illicit drugs by internal concealment is a serious and growing problem. People who engage in this practice are commonly referred to as body packers. The most serious risks associated with body packing include intestinal obstruction and death from drug intoxication. Surgical retrieval is the common method for the management of these patients. However, surgical procedures are associated with significant complications and mortality due to rupture of poorly constructed packages. Conservative management using whole bowel irrigation with polyethylene-glycol (PEG) has been shown to be a safer treatment for most patients.

Case Report: We report 4 cases of international smuggling of heroin in the border of Iran and Afghanistan. 4 patients aged 15, 16, 30 and 70 years old were brought by police to the emergency departments due to ingestions of pellets of heroin for transport. In their initial medical examination none of them had signs and symptoms of heroin intoxication. All of them were alert, oriented, normotensive with normal respiratory rate, pupillary, and abdominal examination. Upright plain abdominal radiography revealed numerous cylindrical
masses in the whole abdomen. They confessed that they swallowed 145 pellets of heroin in total. Patient aged 30 years old took 40 pellets and other ingested 35 pellets each. Patients were admitted in the surgical ward under care of a clinical toxicologist and a surgeon. PEG solution (70 g/L h) were administered orally for whole bowel irrigation. In three cases (70, 15 and 16 years old) all pellets were evacuated after ingestion of 6, 4, and 5 liter of PEG solution. They were discharged from hospital the day after. In the forth patient 38 pellets were evacuated within 24h after initiating therapy but the remaining two pellets required another 3 days. The last patient received 14 L of PEG solution and 40 g caster oil orally and also enema with 40 g caster oil within 4 days of conservative treatment. The patient was discharge in the day 5. All patients were monitored for signs and symptoms of toxicity, abdominal pain and obstruction. Serial abdominal radiography was performed during treatment. None of the pellets was ruptured and no one developed heroin toxicity. Serum electrolytes were in normal range. Patients were taken to the prison for further legal actions. Police agents collected 145 pellets that each contains 25 g heroin.

**Conclusion:** Whole bowel irrigation with PEG solution is a safe conservative treatment and is recommended in body packers who are conscious, have no signs and symptoms of intoxication or bowel obstruction. This will reduce the risk of complications and length of hospital stay.

### 65. ELEVATED COMPARTMENT PRESSURES IN TWO PEDIATRIC RATTLESNAKE ENVENOMATIONS

**Karen C. Thomas, Barbara I. Crouch, E. Martin Caravati**

**Utah Poison Control Center, Salt Lake City UT USA**

**Background:** Treatment of symptomatic rattlesnake envenomation patients with cattleine antivenom is the standard of care. Swelling is often controlled by antivenom alone, making compartment syndrome rare and surgical intervention even less common. We report two pediatric rattlesnake envenomations evaluated for compartment syndrome with documented compartment pressures. One child underwent a fasciotomy. Both children had complete resolution of symptoms with no recurrent coagulopathies.

**Case Reports:**

**Case 1:** An 8-year-old girl was bitten on her left ankle by a Great Basin rattlesnake. During the hospital course, she developed swelling of the foot and ankle, bruising of lower leg, with redness and severe pain in her upper leg. She was evaluated for compartment syndrome due to the severe leg pain. The compartment pressure in her lower leg was 65-68 mm Hg (normal: ≤16 mm Hg for children) and in her upper leg was 28 mm Hg. She wasited with antivenom only (32-38 vitals total), and her signs and symptoms resolved. She did not develop any coagulopathy. Her creatinine kinase peaked at 3,512 IU/L (normal: ≤296 IU/L). She was discharged home on hospital day 6.

**Case 2:** A 2-year-old boy was bitten on his right index finger by a Great Basin rattlesnake. Within 8 hours of the bite he had been treated with 4 vials of antivenom and was evaluated for surgical intervention because his arm was swollen and cyanotic. The child was taken the operating room emergently. A compartment pressure of 60 mm Hg was measured between the child’s first and second metacarpals and fasciotomy was performed. During his hospital stay, the child’s INR peaked at 1.6 (normal: 0.9 - 1.2), PT at 18.8 s (normal: 11.9-14.4 s), PTT at 52 s (normal: 24-37 s). His fibrinogen decreased to 159 mg/dL (normal: 200-430 mg/dL). The child received approximately 15 vials of cattleine antivenom total. He was discharged home on hospital day 5.

**Discussion:** In both cases, compartment pressures were significantly elevated in the affected limb. In Case 1, the child’s elevated compartment pressure and associated symptoms resolved with additional antivenom treatment, precluding the need for surgery. In Case 2, emergent surgical intervention was used to relieve elevated compartment pressures.

**Conclusion:** Elevated compartment pressures occurred in two pediatric rattlesnake envenomations that resulted in different therapeutic interventions.

### 66. THE USE OF INTRAVENOUS FAT EMULSION AS AN ADJUNCT TO STANDARD ACLS IN THE RESUSCITATION OF A PATIENT IN CARDIOPULMONARY ARREST AFTER 2C-E USE


1Hennepin Regional Poison Center, Minneapolis MN USA; 2Unity Hospital, Fridley MN USA

**Background:** Exposures to designer drugs have increased significantly over the last decade. 2C-E, a synthetic hallucinogenic phenethyamine, is an emerging designer drug that is available on the Internet. We report the first case, to our knowledge, of a fatal 2C-E exposure that had sustained return of spontaneous circulation (ROSC) after intravenous fat emulsion (IFE) therapy.

**Case Report:** A 19-year old man developed agitation and violent behavior one hour after inhaling 2C-E at a party. He subsequently collapsed and became unresponsive. His friends attempted CPR for approximately 30 min before driving him to a local hospital. The patient was found to be in cardiac arrest upon arrival with a rectal temperature of 104.7°F. Standard ACLS measures were performed; 1.6 mg naloxone, 4 mg atropine, 5 mg epinephrine, 3 g CaCl₂ + , and 250 mEq sodium bicarbonate (bicarb) were administered. An IFE bolus of 90 mL of 20% solution was administered 46 minutes after ED arrival and sustained ROSC occurred simultaneously. He was given an additional 150 mEq of bicarb and infusions of dopamine, norepinephrine and IFE at 900 mL/hr were started. External cooling with the Arctic Sun™ device was initiated and acetaminophen administered. Initial labs revealed the following: sodium 150 mEq/L, CO₂ <4 mEq/L, glucose 282 mg/dL, creatinine 2.1 mg/dL, lactate 32.6 mEq/L, troponin <0.01 ng/mL, acetaminophen <5 mcg/mL, salicylate <2.5 mg/dL, ethanol 0.06 mg/dL, and INR 2.1. The comprehensive urine drug screen was positive only for facility administered medications. He died several hours later due to severe physiologic derangements resulting from his prolonged arrest. The Bureau of Criminal Apprehension confirmed the substance as 2C-E.

**Discussion:** There are no documented cases of severe injury or death related to the use of 2C-E. The mainstay of treatment, similar to that of other hallucinogenic amphetamines, is hydration, benzodiazepines, and supportive care. IFE was used in this case due to cardiac arrest refractory to standard ACLS measures. Sustained ROSC correlated with the timing of IFE bolus administration. The use of IFE as a treatment is an expanding area of interest in toxicology. The literature is replete with reports of its successful use in the management of lethal overdoses, particularly those due to anesthetics and lipophilic drugs. This is the first reported use of IFE for the treatment of cardiac arrest due to phenethyamines.

**Conclusion:** Intravenous fat emulsion should be considered in patients with a deteriorating clinical condition or cardiac arrest due to phenethyamines refractory to conventional therapies.

### 67. CONTINUOUS RENAL REPLACEMENT THERAPY FOR SALICYLATE OVERDOSE IN A PATIENT WITH MULTI-SYSTEM TRAUMA

**Loren K. French**, Nathanael J. McKeown, Robert G. Hendrickson

1Oregon Health and Sciences University, Portland OR USA; 2Oregon Poison Center, Portland OR USA

**Background:** Managing a patient with concomitant poisoning and multisystem trauma can be complex. Standard treatment modalities for either process may not be feasible under certain situations. While hemodialysis (HD) is generally the preferred extracorporeal elimination technique for salicylate intoxication, the potential for large volume shifts and hypotension may worsen traumatic brain injury. We describe a case of multi-system trauma and salicylate poisoning treated with continuous renal replacement therapy (CRRT).

**Case Report:** An 18 YO, depressed, woman jumped in front of traffic in a self-harm attempt. She was intubated by paramedics and transferred to a level 1 trauma center for evaluation. Her injuries included subdural hematoma, renal laceration, multiple rib fractures, pulmonary contusions, and multiple long bone fractures. While in the trauma ICU, it was postulated that she may have overdosed and comprehensive drug testing was undertaken. Three hours after her ICU course a salicylate level was noted to be 24.7 mg/dL (therapeutic range 10-25 mg/dL). Her ABG at the same time was pH: 7.08, pCO2 71, pO2 421 and HC03 19. Serum creatinine was elevated at 1.52 mg/dL. A sodium bicarbonate drip was initiated and she received 2 doses of activated charcoal. Though serum bicarbonate levels normalized, the patient had refractory respiratory acidosis despite aggressive ventilator management. Over the next 8 hours her serum salicylate concentration rose to 45.2 mg/dL. Though her salicylate toxicity would generally not prompt dialysis, rising levels and persistent acidosis were concerning. Nephrology was consulted and due to the patient’s head injury and
hypotension requiring norepinephrine, they decided to use continuous venovenous hemodialfiltration (CVVHDF), a form of CRRT, rather than HD. Salicylate clearance rate at a blood flow rate of 150 ml/min was 7.5 ml/min with an extraction ratio of 5. Salicylate was completely cleared from her serum within 48 hours. She died as a result of her severe injuries two weeks after admission.

**Case Discussion:** Owing to much slower clearance, CRRT is a controversial and less commonly used modality to eliminate xenobiotics. In this case, the decision to initiate dialysis was based on rising salicylate concentrations and refractory respiratory acidosis, rather than profound salicylate intoxication. In cases where HD may be harmful and in situations where time allows, CRRT may have a niche in the management of xenobiotic overdoses.

**Conclusion:** We describe a case of multi-system trauma and salicylate overdose complicated by refractory respiratory acidosis utilizing CRRT as the modality for extracorporeal elimination.

### 68. CLINICAL AND ECONOMIC IMPACT: GUIDELINE-DRIVEN EMERGENCY DEPARTMENT OBSERVATION PATHWAY FOR CROTALID ENVENOMATIONS

**Anh-Vuong Ly**1,2, Jamie Rhee1, Susan Smith3, Tam Nguyen2, Eun Taik Ha1, Sean Bush1

1Loma Linda University, School of Medicine, Loma Linda CA USA; 2Loma Linda University, School of Pharmacy, Loma Linda CA USA; 3Loma Linda University, Medical Center, Loma Linda CA USA

**Background:** Factors that complicate the management of crotalid envenomations are the severity of the envenomation, clinical course, and response to treatment. Given the complexity involved in the care of snakebite patients, a guideline-driven observation pathway was developed at our institution. Guideline centered on ensuring initial control with the administration of Crotalidae Polyvalent Immune Fab (ovine) antivenom (FabAV), systematic reassessments, administration of maintenance doses of FabAV for symptom progressions, prolonged emergency department (ED) observation, and multiple follow-ups (F/U) over two weeks. Study’s purpose was to evaluate patients who follow the guideline against those who did not follow the guideline with regard to measures associated with increased economic burden: amount of FabAV administered, length of stay (LOS), complications, and need for additional therapy or medications to treat complications.

**Methods:** Retrospective chart review (N = 237) of adult (14 to 65 yrs) patients (transfers and non-transfers) who were treated with FabAV and with or without ED observation. Demographics, dose/administration to obtain maintenance control, complications types, additional therapy or medications to treat the complications, LOS and F/U values were collected per cohort.

<table>
<thead>
<tr>
<th>Table 1.</th>
</tr>
</thead>
<tbody>
<tr>
<td>NON-TRANSFER PTS (N=73) ED OBS (n=57) Non-ED OBS (n=16) DIFFERENCE</td>
</tr>
</tbody>
</table>

| Age (yrs) | 39 | 39 | 0 |
| Male (%) | 89 | 75 | 14 |
| Weight (kg) | 77 | 81 | –4 |
| Vials/Pt | 14 | 16 | –2 |
| Complications/Pt (%) | 28 | 110 | –82 |
| Additional Medications/Pt | 4 | 7 | –3 |
| LOS/Pt | 1.9 | 3.2 | –1.3 |
| F/U/Pt | 1.5 | 0.5 | 1 |

| TRANSFER PTS (N=164) ED OBS (n=126) Non-ED OBS (n=38) DIFFERENCE |

| Age (yrs) | 38 | 41 | –3 |
| Male (%) | 78 | 89 | –11 |
| Weight (kg) | 78 | 79 | –1 |
| Vials/Pt | 16 | 21 | –5 |
| Complications/Pt (%) | 17 | 100 | –83 |
| Additional Tx/Pt | 2 | 19 | –17 |
| Additional Med/Pt | 4 | 9 | –5 |
| LOS/Pt | 1.7 | 4.1 | –2.4 |
| F/U/Pt | 1.2 | 0.5 | 0.7 |

*Average, unless indicated.

**Results:** (See Table 1): Non-transferred patients with ED observation versus non-ED observation required less average number of vials/patient to establish initial and maintenance control. Also required less average rate of occurrence/patient in complications, additional medications or therapy, and LOS. The follow-up appointments to potential prevent reoccurrence complications. Larger impact was observed in transferred patients with ED observation versus non-ED observation in number of vials, complications, additional medications and therapy, and LOS. Transferred patients, non-ED observation cohort required additional dialysis and/or surgery to treat complications.

**Conclusions:** The implementation of an institutional guideline with emphasis of extended ED observation and F/U appointments was associated with clinical and economical benefits.
Conclusions: Transferred patients who do not receive initial control prior to transfer are more likely to incur costs including additional vials of Crofab\textsuperscript{®}, additional treatment of complications for all types, and result in significant economic burden to the hospital and the patient.

70. DEXMEDETOMIDINE AS EFFECTIVE ADJUNCTIVE AGENT IN THE TREATMENT OF A PATIENT WITH SEVERE ALCOHOL AND BENZODIAZEPINE WITHDRAWAL

Lars-Kristoffer Peterson, Timothy Wiegand
University of Rochester Medical Center, Rochester NY USA

Background: Dexmedetomidine is a centrally acting highly selective alpha-2 adrenergic agonist that has sedative, analgesic, anxiolytic and sympatholytic properties. It has a predictable hemodynamic profile, doesn’t induce a significant degree of respiratory depression and its short half life of 2-3 hours makes it a relatively easy medication to titrate via IV infusion. Dexmedetomidine has been shown to attenuate alcohol and opiate withdrawal in animals and has been used clinically in cases of ethanol, sedative-hypnotic and opiate withdrawal, in buprenorphine-pump failure, as well as in the treatment of agitated delirium due to cocaine. We report a case in which dexmedetomidine was used adjunctively along with benzodiazepines and barbiturates for the treatment of severe alcohol and sedative-hypnotic withdrawal.

Case Report: A 37 year-old male was hospitalized after combined alcohol and drug overdose. He developed alcohol withdrawal shortly after admission and suffered respiratory failure due to aspiration. His ICU course was complicated and he required extremely high-doses of midazolam, ultimately up to 60 mg/hour, although this eventually became ineffective in attenuating his withdrawal and providing adequate sedation. Phenobarbital was then administered IV in escalating doses over several hours until approximately 1.5 mg/kg had been given. Although the patient’s tachycardia and hypertension improved he had paroxysms of agitation and diaphoresis with a waxing and waning level of consciousness. A dexmedetomidine infusion was initiated at this point and the patient had dramatic improvement. His bouts of agitation and diaphoresis ended. Dexmedetomidine was titrated down from 0.8 mcg/kg/hour. Once he was no longer unstable with regard to his withdrawal weaning of his GABAergic medications became possible.

Case Discussion: Alpha-2 adrenergic agonists reduce the amplitude of withdrawal through their modulation of sympathetic and noradrenergic outflow primarily via action at alpha-2A receptors of the locus ceruleus, Tachycardia, hypertension, anxiety, agitation, and fever are attenuated. Alpha-2 agonists thus do not replace the drug being withdrawn but buffer the withdrawal. Although GABAergic agents remain the primary treatment modality for sedative-hypnotic withdrawal, and should be used to treat and prevent seizures, dexmedetomidine may be particularly useful for refractory withdrawal or in cases where delirium is precipitated by GABAergic agents.

Conclusions: Dexmedetomidine appears to be an effective agent for adjunctive use with benzodiazepines and for treatment of severe alcohol and sedative-hypnotic withdrawal.

71. IN UTERO EXPOSURE TO FLUOXETINE RESULTS: IN THE ANXIETY PHENOTYPE IN EPET-EYFP TRANSGENIC MICE

Lawrence Quang, Najam Kazmi
Case Western Reserve University School of Medicine, Cleveland OH USA

Background: Selective serotonin reuptake inhibitors (SSRIs) are often used to treat maternal intrapartum depression, which affects up to 15% of pregnancies. Recently, the effects of fetal exposure to SSRIs have been investigated in mice during the early postnatal period, which corresponds to the brain maturation events that occur during the 3\textsuperscript{rd} trimester of human pregnancy. In this model, early life blockade of the serotonin transporter (5-HTT) with fluoxetine during postnatal days 4 – 21 produced an anxiety phenotype in adulthood. Although SSRIs cross the placenta in mice, in utero studies haven’t been done.

Objectives: We investigated the effects of SSRIs on emotion and learning in adult mice after in utero exposure to fluoxetine on embryonic days 9 – 14 (E9 – E14). At 12 weeks of age, we evaluated the progeny from both groups for disorders of emotion and aggression with the elevated plus maze, open field test, forced swim test, acoustic startle and pre-pulse inhibition, resident intruder test, and marble burying test. We also evaluated the progeny from both groups for disorders of learning with the object recognition task, conditioned place preference, and fear conditioning.

Results: Adult mice from the fluoxetine in utero exposure group (N = 24) exhibited an anxiety phenotype when compared to their controls (N = 25) for the elevated plus maze test and the acoustic startle response. There were significant differences (P < 0.05) between the fluoxetine in utero exposure and control groups, respectively, in the elevated plus maze for: total time in seconds in the open arm (61.4 ± 4.5 vs. 90.9 ± 9.3); total time in seconds in the closed arm (185.5 ± 8.9 vs. 167.8 ± 9.9); % total time spent in the open arm (20.5 ± 1.5 vs. 30.3 ± 3.1); and % of total entries into the open arm (33.1 ± 1.3 vs. 42.9 ± 2.4). There was a significant difference (P < 0.05) between the fluoxetine in utero exposure and control groups, respectively, for the acoustic initial startle response in dB (207.38 ± 42.30 vs. 355.62 ± 75.62). Tests for depression (forced swim test), aggression (resident intruder test), obsession-compulsive behavior (marble burying test), and learning (conditioned place preference, object recognition task, and fear conditioning) showed no differences between the fluoxetine in utero exposure and control groups.

Conclusions: In utero exposure of EPET-EYFP mice to SSRIs resulted in an anxiety phenotype in adulthood. This is consistent with the previous finding of an adult anxiety phenotype in the rodent model of SSRI fetal exposure by postnatal fluoxetine.

72. DETERMINATION OF PLASMA DMPS CONCENTRATION AND URINE MERCURY EXCRETION AFTER DERMAL APPLICATION OF “TD-DMPS”

Jennifer Smith\textsuperscript{1}, Anne-Michelle Ruha\textsuperscript{1}, Steven C. Curry\textsuperscript{1}, Kalloit Biswas\textsuperscript{2}, Samia Nasr\textsuperscript{2}, Wei Ye\textsuperscript{2}, Kathleen Caldwell\textsuperscript{2}, Frank LoVecchio\textsuperscript{1}, Keith Burkhart\textsuperscript{2}, Benjamin Westenberger\textsuperscript{2}
\textsuperscript{1}Banner Good Samaritan Medical Center, Phoenix AZ USA; \textsuperscript{2}Division of Laboratory Sciences, National Center for Environmental Health, CDC

Background: 2,3-dimercaptopropanoic acid (DMPS) is a metal chelator that is approved in Europe for oral or intravenous use to treat mercury (Hg) and arsenic poisoning. DMPS is not an FDA-approved medication in the US, but is legally available as a medicinal agent from compounding pharmacies. Despite absence of any scientific data demonstrating transdermal absorption of DMPS, a transdermal formulation (TD-DMPS) is prescribed by some practitioners to treat autistic children. The Objectives of this study were to determine 1) if DMPS is detected in blood or urine following dermal application of TD-DMPS and 2) if urinary Hg (UHg) excretion increases following use of TD-DMPS. IRB approval was granted for this study.

Methods: Eight healthy adult volunteers provided a 12 hr urine collection for determination of baseline UHg and creatinine (Cr) excretions. The following morning, subjects applied 3 mg/kg TD-DMPS to their arms (time 0). A second 12 hr urine collection began at the time of TD-DMPS application. Blood samples were obtained at times 30 min, 60 min, 90 min, 2 hr, and 4 hr post-application. One subject had additional blood drawn at 24 hr. Plasma and urine were analyzed for presence of DMPS by the FDA Division of Pharmaceutical Analysis (limit of detection 0.74 mcg/mL) and UHg concentrations were measured at the CDC Division of Laboratory Sciences (LOD 0.08 µg/L).

A ninth volunteer ingested 250 mg oral crystalline DMPS and provided pre- and post-chelation 12 hr urine samples as well as blood samples at 30, 60, 90 min, and 2 and 4 hr post-ingestion.

Results: The subject who ingested oral DMPS had DMPS detected in plasma at levels comparable to those reported in the literature (2.7 - 3.9 µg/mL), and had an increase in UHg excretion following chelation. DMPS was undetectable in urine and plasma samples for all eight volunteers who applied TD-DMPS.

Conclusions: DMPS is not detected in plasma or urine following topical application of TD-DMPS, and TD-DMPS does not increase UHg excretion in healthy volunteers.
73. VALIDATION OF A PRE-EXISTING FORMULA TO CALCULATE THE CONTRIBUTION OF ETHANOL TO THE OSMOLAR GAP

Alexander Garrard1, Thomas Kuniaki1, Dawn Sollee1, Ryan Butterfield2, Laura Johansen2, Roger Bertholf2
1Florida/USVI Poison Information Center – Jacksonville, Jacksonville FL USA; 2LIF&Shands – Jacksonville

Background: This study aims to validate the formula derived by Pursell et al. and clarify the clinical controversy of which cofactor to use when accounting for ethanol in the osmolar gap calculation. Osmolar gap is often used to help diagnose toxic alcohol poisoning when these levels are not available.

Methodology: Part I consisted of a retrospective review of 604 emergency department patients who had a concurrent ethanol, basic metabolic panel, and a serum osmolarity drawn. Predicted osmolarity, excluding ethanol, was calculated using the following formula: (2 x Na + (mEq/L) + (BUN [mg/dL])/2.8 + (Glucose [mg/dL])/18). The osmolar gap was determined by subtracting the calculated osmolarity, excluding ethanol, from the measured serum osmolarity. Linear regression was then used to evaluate the relationship between the osmolar gap and the measured ethanol level. In Part II, this experiment was duplicated by adding predetermined amounts of ethanol to serum. Post hoc analysis via linear regression was done on groupings of ethanol concentration versus osmolar gap to see if the factor varied with the ethanol concentration.

Results: We had 604 patients in Part I from which we derived a new formula to calculate the contribution of ethanol to the osmolar gap. The mean ethanol level was 182.54 mg/dL (SD of 116.80 mg/dL) with a range of 10 – 644 mg/dL. Our data had a strong linear relationship with a Pearson coefficient of correlation of 0.93 and a r² value of 0.96. The derived linear regression equation was Osmolar gap = [Ethanol (mg/dL)/3.96] Pursell and colleagues derived a denominator of 3.7 from their 98 observations. The formula for linear regression derived from the in vitro data was Osmolar gap = [Ethanol (mg/dL)/3.85] with a Pearson coefficient of r = .98, p-val < 0.0001 and r² of 0.99. Post hoc analysis revealed a bell-shaped distribution of denominators from 3.9, peak of 5.8, and back to 4.1.

Conclusions: The data from our study suggests that a more accurate equation for calculating the contribution of ethanol to the osmolar gap is the following: Ethanol (mg/dL)/3.96. Further investigation is warranted to see if the ethanol denominator is dependent on concentration.

74. THE EFFECT OF INTRAVENOUS FAT EMULSION ON ORGANOPHOSPHATE TOXICITY

Theodore Bania, Jason Chu, Kimal Medlej
Luke’s-Roosevelt/Columbia University, New York NY USA

Background: Intravenous fat emulsion (IFE) has been successfully used to treat toxicity from several lipid soluble drugs in several animal models and in multiple case reports. Most evidence supports a lipid sink/sponge model as the mechanism of IFE in ameliorating lipid soluble drug toxicity. Intravascular lipid emulsions soak up lipid soluble toxins and prevent the lipid soluble toxin from reaching the site of toxicity. Many nerve agents and organophosphates are highly lipid soluble. It is not known if IFE will have any effect on organophosphate toxicity.

Objective: 1. To determine if intravenous fat emulsion can improve survival in a model of organophosphate toxicity. 2. To determine if intravenous fat emulsion is protective and delays the onset of clinical signs of organophosphate toxicity.

Methods: This was a randomized, controlled investigation. 28 male wistar rats were anesthetized, and instrumented with a tracheostomy, carotid artery and femoral vein catheters. Anesthesia was maintained with IP ketamine and baseline mean arterial pressure (MAP), heart rate (HR) and arterial blood gases (ABG) were recorded. All rats were given parathion (log P 3.47) 30 mg/kg via oral gastric tube. Five minutes later, animals were divided into 2 groups and received 3 doses of 6 ml/kg of 20 % IFE every 2.5 minutes or an equivalent volume of 0.9% saline. MAP, HR and ABG were recorded every 15 minutes. Animals were continuously monitored for apnea lasting > 30 sec or HR decrease by 50%. Survival was determined at 165 minutes if HR remained greater than 10 % of baseline. Data was analyzed using Kaplan Meier analysis, log rank test and ANOVA. A pre-test sample size calculation determined that 14 animals per group would be needed to detect a 15 min change in survival time.

Results: The median survival time for the IFE group was 37 min (95% CI = 26-48) and the NS group was 40 min (95% CI = 37-42) (p = 0.2). The median time to apnea for the IFE group was 29 min (95% CI = 20-38) and the NS group was 24 min (95% CI = 20-28) (p = 0.5). The median time to HR decrease by 50% for the IFE group was 30 min (95% CI = 29-32) and the NS group was 25 min (95% CI = 15-34) (p = 0.2). There was no significant difference in HR and MAP between treatment groups. At 45 min post exposure, pH was higher in the IFE group versus NS group (IFE = 7.24, NS = 6.9, 95% CI of difference 0.06-0.57). There was no significant difference in pCO2, pO2 and oxygen Saturation.

Conclusions: In this model of organophosphate toxicity, post exposure treatment with IFE did not improve survival or result in delayed onset of toxicity as measured by apnea or bradycardia.
Background: Emergency intoxication cases often lack enough historical information to narrow testing to a target group of analytes. An analytical platform that allows comprehensive and untargeted drug screening with a fast turnaround time may be useful for emergency toxicology screening. We have taken advantage of advances in the mass accuracy, resolution, and speed of liquid chromatography-time-of-flight mass spectrometry (TOF LC-MS) to implement non-targeted drug screening on intoxications referred to a poison control center (PCC).

Methods: We developed a rapid, generic, semi-quantitative, non-targeted urine and serum drug screening regimen using TOF LC-MS. The analytical run requires chromatography with gradient elution in a C18 column followed by TOF MS using electrospray ionization. The high accuracy of mass measurement afforded by TOF MS allows unambiguous molecular formula assignment that generates candidate compound hits from a comprehensive forensics database (~7000 compounds) by imposing a compound mass tolerance <10ppm, peak area count >5000, and a target score >70 (numerical comparison of theoretical and measured isotopic patterns). The combination of molecular formula matches to drug and/or drug metabolites from the patient’s urine and serum, and available drug specimens taken by the patient, are used to report presumptive intoxicant data in real time. These data are validated and correlated to a patient’s clinical syndrome by PCC medical toxicologists. Further confirmation of the intoxicating compound’s identity is done by comparing its retention time to its reference standard, after which its quantitative levels are measured. Retention times (RT) for common drug intoxicants (~300 drugs) have been established and compiled to facilitate drug intoxicant confirmation.

Results: The overall analytical turnaround time when the RT of the presumptive intoxicant is available is ~2h. For non-targeted screens yielding a presumptive intoxicant without an established RT, preliminary data for emergency cases can be made available within 3-6 hrs. Using this approach we were able to solve >70% of the 55 cases referred to PCC last year. These cases include 9 unexplained seizures, 10 therapeutic drug overdoses, 15 illicit drug overdoses, 3 new designer drug intoxications, 4 intoxications involving misrepresented drugs and 2 adverse drug reactions.

Conclusion: With its fast turnaround time and very large number of molecules that can be analyzed, semi-quantitative, non-targeted drug screening using TOF LC-MS may be extremely useful in emergency toxicology screening.

77. SYNTHETIC CANNABINOIDS TESTING USING LIQUID CHROMATOGRAPHY-TIME-OF-FLIGHT MASS SPECTROMETRY (TOF LC-MS)

Roy R. Gerona1, Elisabeth Birdsal1, Till Beuerle1, Alan H. Wu1
1Department of Laboratory Medicine, San Francisco General Hospital/University of California, San Francisco, San Francisco CA USA; 2California Poison Control System- San Francisco Division, San Francisco CA USA; 3Institute of Pharmaceutical Biology, Braunschweig University of Technology, Braunschweig Germany

Background: The recreational use of herbal incense has gained popularity in the last year. More than 2000 cases related to its use were reported to Poison Control Centers in the first quarter of 2011. Majority of these products are laced with synthetic cannabinoids (SCs), a group of compounds that mimic the effects of tetrahydrocannabinol (THC). SCs are not detected by THC screens, and setting up a targeted screening for these compounds is challenging because herbal incense manufacturers constantly change the lacing agents they use to avoid prosecution. There are more than 400 different SCs that can be synthesized and used. Only five have been classified as Schedule I substance so far. To allow investigation of SC intoxications, we have set-up a product, serum, and urine testing for SCs using TOF LC-MS.

Methods: We developed semi-quantitative methods for the analysis of 33 SCs and their metabolites in herbal incense, serum, and urine using TOF LC-MS. The method requires chromatography with gradient elution in a C18 column followed by TOF-MS using electrospray ionization. The analytical turnaround time for each method is ~2h. We tested the suitability of these methods for emergency cases involving SCs.

Results: The screening methods are analytically robust. The analytes in the panel have LOD of 0.5-25ng/mL, linear concentration range of 200-5000, %CV of 2-14% and 6-20% for within run and between run precision, respectively, and % recoveries of 80-110%. The methods are currently being used to document levels of specific SCs in herbal incense intoxications. For example, two patients recently presented to the ED with nausea, vomiting, tachycardia, and mentally altered status on separate occasion. Both patients claimed using herbal incense immediately before presenting to the ED. Screening of the products they used revealed a number of SCs (Spike Maxx: JWH-007, 073, and 398; Spike 99: JWH-018, 015, 210, and 122). Most of these compounds have been reported in herbal incense. JWH-007 is an interesting finding as it has never been reported. This compound is not included in our panel but the ability of TOF LC-MS to facilitate non-targeted screening has allowed us to identify this novel lacing agent in Spike Maxx. One of the patient’s serum was found to contain JWH-018 and one of its hydroxylated metabolites.

Conclusions: Use of TOF LC-MS allowed the development of Methods: for screening SCs in herbal incense, and patient’s serum and urine. The ability of TOF LC-MS to facilitate non-targeted screening also allows for discovery of new SCs in herbal incense, a feature useful for screening designer drugs where target compounds are constantly expanding.

78. DEVELOPMENT OF A LC-MS/MS METHOD FOR MEASUREMENT OF NEW PSYCHOACTIVE COMPOUNDS IN THE CLINICAL LABORATORY

Duncan Stephen, Janice Reeve, Iain Clunie, Linda McKay, James Allison, Bernard Croal
Aberdeen Royal Infirmary, Aberdeen, AB2522D UK

Background. There has been an increase in the recreational use of new psychoactive compounds, such as Methyleneedioxy-2-aminoindane (MDAI) and Cathinone (+ derivatives), collectively known as “legal highs” over the last three years. These amphetamine-like compounds have been associated with euphoria, paranoia, arhythmia and have the potential to cause death through toxicity. In April 2010, the UK government scheduled Cathinone (+ derivatives) as Class B drugs under the Misuse of Drugs Act 1971; however, MDAI is still legal. The potential for increasing use of these drugs may be mirrored by requests for analysis. As a toxicological response to the measurement of these compounds, we present an analytical technique applicable for use in the emergency clinical toxicology setting.

Methods: A LC-MS/MS method was established on the Waters® XevoTM TQ MS ACQUITY UPLC® System to measure Cathinone, Methylenedioxymethamphetamine (MDMA), Methylene-dioxymethamphetamine (MDPV) and MDAI Calibration. Calibration was achieved (n=8) using drug-free blood spiked with these compounds and quetiapine (internal standard). Liquid-liquid extracts were injected onto a C8 column and chromatographic separation was performed using gradient elution (methanol/water0.05% formic acid) with MS/MS detection. Linearity, assay imprecision, LOD, LOQ (CV <20%) and efficiency of recovery for each compound was determined.

Results: Linearity was observed over the range 0.005-0.500 mg/L for each compound: Cathinone (R=0.997), Methylenedioxymethamphetamine (R=0.936), Ethylone, Butyrolone Methylene-dioxymethamphetamine (MDPV) and MDAI (R=0.999). Intra- and inter-assay imprecision at 0.03 mg/L was <20% and <25% for all compounds, respectively. LOD were: 0.03 mg/L (Cathinone), 0.001 mg/L (Methylenedioxymethamphetamine, Butyrolone, MDPV, MDAI). LOQ were: 0.01 mg/L (MDPV, Butyrolone), 0.02 mg/L (Methylenedioxymethamphetamine, MDAI), 0.05 mg/L (Cathinone, Ethylone). Preliminary recoveries were: Cathinone (89%), Methylenedioxymethamphetamine (66%), Ethylone (61%), Butyrolone (63%), MDPV (87%), MDAI (65%).

Conclusions: In the absence of commercially available immunoassays for the detection of these novel compounds, we have developed a quick and effective LC-MS/MS method for the measurement of Cathinone and its derivatives in the acute toxicity setting.

79. DETECTION OF NITROMETHANE AND RELIABLE CREATININE ASSESSMENT WITH RACING FUEL INGESTION USING COMMON ANALYTICAL METHODS

CM. Murphy1, MC. Beuther2, Susan Maynard1
1Carolinas Pathology Group, Charlotte NC USA; 2Carolinas Medical Center/Carolinas Poison Center, Charlotte NC USA
Background: Model car racing utilizes fuel consisting of a methanol-nitromethane mixture. In cases of ingestion, nitromethane (NM) is known to cause a false elevation in serum creatinine with standard colorimetric creatinine analysis (Jaffe method). We present a case of racing fuel ingestion in which an alternative method of creatinine determination, as well as semi-quantitation of NM using a common toxic alcohol analysis method was used to confirm exposure.

Case Report: A 42 yo male with a history of alcoholism presented with new onset seizures. On admission, his lactate was > 20 mmol/L; arterial pH, 7.03; serum bicarbonate; 7 mEq/L; chloride, 98 mEq/L; glucose, 246 mg/dL; BUN, 18 mg/dL; and creatinine, 35.04 mg/dL. A point-of-care creatinine level was 1.1 mg/dL. The working diagnosis was alcohol withdrawal, however 15 hours after admission he admitted to drinking Nitromethane Glow Fuel (72% methanol and 20% NM). He was transferred to a tertiary care facility for hemodialysis (HD). Laboratories obtained 8 hours after initial evaluation demonstrated a methanol level of 114 mg/dL, improved serum bicarbonate of 24 mEq/L and creatinine of 33.80 mg/dL. A methanol level obtained just prior to initiation of HD was 51 mg/dL. On repeat labs post HD his methanol level was 4.7 mg/dL; BUN, 2 mg/dL; and creatinine, 6.9 mg/dL. Ultimately, the patient made a full recovery and was discharged home. Toxic alcohol analysis was performed using gas chromatography with a trifluoropropyl methyl polysiloxane capillary column utilizing flame ionization detection. All three toxic alcohol chromatograms demonstrated a peak which was confirmed to be NM. Assuming 1st order clearance, endogenous half-life of NM was determined to be 13.5 hours; this decreased to <10 hours during HD. The highest serum NM level (pre HD) was estimated at 20 mg/dL, however we were unable to complete a full analysis using the original samples.

Case Discussion: This case is unique in that we were able to identify and estimate NM levels using a common alcohols analysis method without special column or oven parameters. We determined an endogenous half-life of NM and demonstrated its increased clearance during HD. Finally, creatinine determination using two Methods demonstrated that point-of-care testing utilizing electrochemical detection of peroxide evolution does not result in the marked false elevation observed with the colorimetric method.

Conclusions: NM can cause extreme false elevations in serum creatinine complicating the diagnosis. However, it is possible to reliably assess creatinine through other Methods and identify the presence of NM in serum using standard gas chromatography Methods.

8.0 SEVERE CARBON MONOXIDE POISONING WITH A FALSELY LOW CARBOXYHEMOGLOBIN LEVEL DUE TO COLORIMETRIC INTERFERENCE FROM HYDROXOCOBALAMIN

Zhanna Livshits, Robert S. Hoffman, Daniel M. Lugassy
The New York City Poison Control Center, New York NY USA

Background: Acute carbon monoxide (CO) toxicity may lead to devastating delayed cognitive effects. Determination of carboxyhemoglobin (COHb) level by co-oximetry is essential for diagnosis and initiation of hyperbaric therapy, and may be inaccurate in the setting of colorimetric interference from hydroxocobalamin.

Case Report: A 41-year-old woman was brought to the ED after being found unconscious in a running vehicle with snow obstructing the car’s tail pipe. In addition to oxygen and for unclear reasons, EMS administered 5g of hydroxocobalamin and 12.5g of sodium thiosulfate. In the ED the patient was awake and alert. Her vital signs were: BP: 101/68 mmHg; HR: 62 beats/min; T: 36.8°F; RR: 20 breaths/min; SpO2: 100%. Physical and neurological examinations were unremarkable. Initial co-oximetry analysis (via Siemens Rapid Lab 1265) demonstrated a COHb level of 2.5%. Based on the history, the patient received hyperbaric oxygen therapy. Subsequent analysis of blood in the CBC tube obtained by EMS prior to hydroxocobalamin administration demonstrated a COHb level of 34.9%.

Case Discussion: Hydroxocobalamin, a red chemical can interfere with colorimetric laboratory analyses. The influence of hydroxocobalamin on co-oximetry has not been well studied. In a rabbit model, COHb levels increased following hydroxocobalamin administration. A recent in-vitro analysis implicated that diagnostic co-oximetry may be altered in the presence of hydroxocobalamin. This is a unique case that demonstrated a marked decrease in COHb level following hydroxocobalamin administration.

Conclusion: Erroneously low COHb levels may be measured in patients treated with hydroxocobalamin. It is important to obtain blood prior to hydroxocobalamin administration to ensure accurate COHb analysis. Clinical judgment in this setting should dictate patient management of carbon monoxide poisoning. Further studies are essential to determine the specific value of hydroxocobalamin interference with COHb level using various instruments.

81. THE DETECTION OF THE PRECURSOR BENZOPHONE FOLLOWING THE USE OF DIPHENYL-2-PYRROLIDINEMETHANOL (D2PM) SUGGESTS AN ALTERNATIVE SYNTHESIS PATHWAY FOR D2PM

David M. Wood1, Maggie Puchnarzewicz2, John Ramsey3, David W. Holt2, Paul I. Dargan1
1Guy’s and St Thomas’ NHS Foundation Trust, London UK; 2St George’s University of London, London UK; 3TICTAC Communications Ltd, London UK

Introduction: Legal highs (novel psychoactive substances) purchased from internet suppliers are thought to be of high purity and the detection of precursors in products sold is unusual. We describe two individuals who used legal highs which contained the Pipradrol Diphenyl-2-pyrrolidinemethanol (D2PM), as well as its precursor benzophenone.

Cases: 33 year old male ingested 8 capsules of the legal high “Benzofury” over the course of an evening. Initially, he had a “high” similar to that he experienced with mephedrone (4-methylmethcathinone). He presented to the Emergency Department (ED) about 56 hours post-ingestion with ongoing symptoms of anxiety/agitation and prolonged insomnia. On examination there were no features of acute sympathomimetic drug toxicity (HR 71bpm, BP 140/95mmHg, Temp 36.0°C, neurological examination normal).

Case 2: 25 year old male ingested a drink containing an “unknown substance”. He presented to the ED 96 hours following ingestion due to ongoing insomnia and visual hallucinations. He had self-medicated with Diphenhydramine due to his symptoms. On examination there were no features of acute sympathomimetic drug toxicity (HR 98bpm, BP 134/98mmHg, Temp 36.4°C, neurological examination normal).

Toxicological Screening: Urine was collected was collected from both patients and analysed by gas-chromatography mass-spectrometry (GC-MS). Case 1: Urine positive for benzophenone and D2PM; Case 2: Urine positive for Diphenhydramine, D2PM and benzophenone.

Discussion: The detection of the benzophenone precursor in biological samples taken from individuals who have used D2PM has not previously been reported. D2PM, a Pipradrol, was thought to be commercially produced from the precursor diphylenylacetoni trile. Detection of benzophenone suggests that the D2PM used was less likely to have been commercially produced. Comprehensive analytical screening in individuals with acute “legal high” toxicity can potentially provide information on the synthetic routes being used for these drugs. This can inform legislative authorities to ensure that appropriate monitoring/control of the relevant precursors is in place.

82. USE OF LEAD ISOTOPE RATIO ANALYSIS TO INVESTIGATE SEVERE, NONACCIDENTAL LEAD POISONING IN A CHILD WITH CHARGE SYNDROME

Lawrence Quang
Rainbow Babies & Children’s Hospital, Cleveland OH USA

Background: We evaluated the clinical use of lead isotope ratio (IR) analysis for a case of severe, non-accidental childhood lead poisoning.

Case Report: We evaluated a 22 m.o. medically complex, technology-dependent child with CHARGE Syndrome for lead poisoning of indeterminate etiology since 5 months of age. The blood lead level was 68 mcg/dL and an abdominal x-ray showed multiple radio-opacities. Environmental history and inspection of the family’s home found no lead source. Non-accidental lead poisoning was suspected and reported to law enforcement, who found a bag...
of white powder in the mother’s backpack. Crime lab analysis of the powder identified crystalline lead nitrate, Pb(NO₃)₂. We performed a lead IR analysis of the confiscated Pb(NO₃)₂ and the child’s blood and urine lead for the ratios of ²⁰⁷/Pb and ²⁰⁸/Pb. The confiscated Pb(NO₃)₂ did not match the child’s lead. This suggested that additional sources of lead were used to poison this child. Law enforcement eventually found 5 additional lead sources from the family’s previous home and the mother’s previous high school science classroom. These included 2 of the child’s lead-contaminated medications from the previous home and two 500 g bottles of laboratory-grade Pb(NO₃)₂ from the mother’s classroom. Additionally, they found evidence that the mother ordered and received a third 500 g bottle of Pb(NO₃)₂ for her classroom which was missing. The mother refused to cooperate regarding its whereabouts. Lead IR analysis of the 2 classroom Pb(NO₃)₂ sources did not match the child.

**Case Discussion:** Diagnostic use of lead IR analysis for childhood lead poisoning has been described only once, when it identified the lead mine responsible for an outbreak of childhood lead poisoning in Peru. In our case, initial lead IR was used in an effort to match the first lead source with the child. Ultimately, it did not identify the exact source among 6 known sources. There are two explanations. First, only 3 of the 6 lead sources were tested. One of the mother’s Pb(NO₃)₂ bottles is missing. The 2 medication bottles could not be tested, as the current lead IR methodology is limited to environmental and biological samples. Second, multiple lead sources will have different lead IRs. If more than one lead source was used throughout the poisoning, lead IR analysis will be unable to produce a match for only one source.

**Conclusions:** In this case, initial lead IR analysis of the first lead sample was consistent with multiple sources of lead used in the poisoning of this child. Indeed, law enforcement discovered 5 additional lead sources directly linked to the mother.

**SESSION II**

**87. SEIZURE 15 HOURS AFTER TRAMADOL EXTENDED RELEASE INGESTION BY AN INFANT**

**John R. Stromness,** Barbara I. Crouch, E. M. Caravati

Utah Poison Control Center, University of Utah, Salt Lake City UT USA

**Background:** Tramadol is a non-narcotic analgesic that binds μ-opiate receptors in the CNS and induces the reuptake of serotonin and norepinephrine. It is metabolized to the M₁ active metabolite, O-desmethyltramadol, via the CYP2D6 isozyme. Peak plasma levels for immediate release tramadol and the M₁ metabolite are seen at 1.5 hours and 1.9 hours, respectively. Peak plasma levels for the extended release formulation occur at 12 hours for the parent compound and at 15 hours for the M₁ metabolite. Tramadol is known to cause seizures and respiratory depression in overdose. A literature search revealed that all reported overdoses have been with the immediate release formulation despite the availability of the extended release formulation since October 2005. We report a case of a child that ingested up to 600 mg of extended release tramadol and experienced delayed respiratory depression and a seizure.

**Case Report:** An 8 month-old boy was referred to the emergency department after ingesting 1-3 tablets of 200 mg extended release tramadol. Approximately 6 hours after ingestion he became sedated with periods of agitation where his heart rate increased to 150 beats/minute. He was admitted for observation. The child had an apneic episode approximately 10 hours after ingestion, was given 1mg of naloxone and responded immediately. Fifteen hours post ingestion the child had another apneic episode and a tonic clonic seizure that lasted approximately 30 seconds. The child had no further episodes of respiratory depression or seizures, his mental status returned to baseline approximately 29 hours after the ingestion and he was discharged home.

**Discussion:** All reported cases of seizures from tramadol have resulted from ingestion of the immediate release formulation of this drug and most occur within 4 hours of ingestion. In our case it is unclear if the seizure was caused by anoxia or drug effect. Delayed onset of respiratory depression and seizure was most likely due to the extended release formulation and a delay in accumulation of the active metabolite. This case is important because it illustrates the potential for delayed effects resulting from the extended release formulation and the importance of a longer observation period for patients exposed to this formulation.

**Conclusion:** Ingestion of extended release tramadol may produce seizures significantly later than has been noted with the immediate release formulations.

**88. DELAYED ARIPIPRAZOLE NEUROTOXICITY IN CHILDREN UNDER AGE 5**

**Michael G. Abesamis,** Michael J. Lynch, Steven J. Walsh, Anthony F. Pizon, Kenneth D. Katz, Denise Kuspsi, Rita Mrvos

¹University of Pittsburgh Medical Center, Pittsburgh PA USA; ²Pittsburgh Poison Center, University of Pittsburgh Medical Center, Pittsburgh PA USA

**Background:** Aripiprazole is an atypical antipsychotic which has been approved for the treatment of schizophrenia, bipolar disorder, and irritability in autism starting at ages 13, 10, and 6, respectively. It has a half-life of 75 hours, and its active metabolite, dehydroaripiprazole, is half-life of 94 hours. Although peak plasma levels occur 3 to 5 hours after ingestion, delayed neurotoxicity in children under age 5 is unique and is discussed in this case series of 4 patients.

**Case Reports:**

1) A 3 year old boy presented to an Emergency Department (ED) 24 hours after ingesting one 20mg aripiprazole pill. Although asymptomatic that evening, he was stumbling and slurring words the next morning. In the ED, the patient was somnolent but arousable, and had intermittent staring spells with right gaze preference and horizontal nystagmus. Electroencephalogram showed diffuse slowing.

2) A 20 month old girl presented to an ED after a witnessed ingestion of 20mg aripiprazole. She had an unremarkable 8 hour observation period and was discharged. Twelve hours later, she returned to the ED with gaze deviation and torticollis which improved with benzodiazepines.

3) A 2 year old girl presented asymptomatic to an ED after a witnessed ingestion of 10mg aripiprazole roughly 3 hours prior. The patient did not stay in the ED and was discharged against medical advice. Twenty-four hours later, the child was transported to a different ED because of somnolence and tremulousness.

4) A 3 year old girl presented to an ED after ingesting 5mg aripiprazole. She was observed for 2.5 hours and sent home asymptomatic. Twelve hours later, the child returned to the ED with somnolence. All the children were admitted, treated symptomatically, and discharged home without sequelae 48-72 hours later.

**Case Discussion:** Literature review of aripiprazole toxicity yielded little published data. Although prolonged toxicity has been described in children, data on delayed neurotoxicity is limited to one case report in an adult. This case series illustrates the potential for young pediatric patients to develop significant neurotoxicity several hours after ingestion without a prodrome. Given the timing of the toxicity, it could be the active drug metabolite that is contributing to the delayed presentation in this population.

**Case Conclusions:** In aripiprazole ingestions, the dose requiring a prolonged observation has not been determined. Young children under age 5 may require a longer observation period before manifesting toxicity. Clinicians and parents need to be aware that symptom onset may be significantly delayed.

**89. GUT GORILLA GLUE?**

**S. Banerji, M. Judd, A. C. Bronstein, T. Valdez**

¹Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA; ²Saint Alphonsus Regional Medical Center, Boise ID USA

**Background:** Poison centers (PCs) routinely answer questions involving pediatric ingestion of common glues and adhesives. Gorilla Glue® (The Gorilla Glue Company, Cincinnati, Ohio) is a polyurethane household glue that foams in contact with moisture. Following ingestion in canines, it can swell 3-4 times its original size and cause gastrointestinal obstruction. There have been no human reports of obstruction following Gorilla Glue®. We report a case of pediatric ingestion of Gorilla Glue® with significant sequelae.

**Case Report:** A previously healthy 19-month old female ingested an unknown amount of Gorilla Glue®. She was brought to the Emergency Department 2 days after ingestion when her parents reported that she had anorexia, nausea, vomiting and diarrhea. On examination, a palpable mass was felt in the epigastric region. The PC was contacted for management information. Abdominal radiography was recommended and revealed a mass filling the stomach. The
child was transferred to another facility for pediatric endoscopy. Vital signs remained normal. Laboratory Results: were remarkable for elevated white blood count (13,800 cells/mL) and hypoglycemia. After unsuccessful endoscopic removal attempts, the child underwent abdominal surgery. The mass was removed in pieces and filled a kidney shaped emesis basin. 24 hours following surgery the child was in good spirits and eating, drinking and playful. The child was discharged on hospital day 3.

Case Discussion: Glues typically pose minimal harm post ingestion. This may not be the case with polyurethane glues that can expand after ingestion. The product lacks child resistant packaging. Children may be more at risk for exposure as the gorilla motif on the bottle makes it appealing. If gastrointestinal obstruction occurs, there is no simple decontamination or removal method.

Conclusion: PCs and healthcare providers need to be aware of the potential risks of pediatric ingestion of these types of expanding polyurethane glues. If a large amount is ingested or the patient is symptomatic, the victim may need health care facility referral for evaluation and possible endoscopic or surgical removal. Further study is needed to estimate toxic dose.

90. DANGEROUS ATTRACTION: A 2-YEAR-OLD GIRL WITH ENTERIC AND MESENTERIC FISTULAE AFTER INGESTION OF SPHERICAL MAGNETIC TOYS
Kevin C. Osterhoudt, William Peranteau, Kathy Shaw, Alan Flake
The Children’s Hospital of Philadelphia, Philadelphia PA USA

Background: Foreign bodies in the intestinal tract of children typically pass uneventfully. However, case reports herald the potential danger of ingestion of more than one magnet, and the U.S. Consumer Product Safety Commission has called magnets a top “hidden home hazard.” The popular brand of spherical magnets implicated in this case was recalled in May, 2010, because at the time it was labeled for “Ages 13 + ,” and mandatory U.S. toy standard F963-08 requires powerful magnets not be sold for children under 14 years. At the time of recall no injuries had been reported with this product.

Case Report: A 2-year-old (10.6 kg) girl had 2 weeks of crampy abdominal pain, anorexia, and malaise with weight loss of 2 lbs. She was afebrile with a heart rate of 136/minute. She appeared pale, ill, and had diffuse abdominal tenderness without guarding or distention. Plain abdominal radiography showed inflamed, dilated loops of small bowel were found to contain the metallic spheres. Two ileal-ileal fistulae, and one ileal-cecal fistula, were identified; and the erosive defects were noted in both the bowel wall and the mesentery. The intraluminal ring of magnets was converted to a kidney shaped emesis basin. 24 hours following surgery the child was in good spirits and eating, drinking and playful. The child was discharged on hospital day three with magnesium level of 2.2 mg/dL.

Case Discussion: This case highlights the occurrence of pseudocoma due to severe hypermagnesemia. Despite his description as comatose and receiving a GCS score of 3, the patient had complete recall of both intubation procedures and painful central line placement. We suspect that hypermagnesemia induced a profound muscle weakness resulting in apparent coma. Limitations of this report include a lack of formal testing of neural function (EEG) and muscular function (train-of-four) during resuscitation.

Conclusion: Profound hypermagnesemia may produce serious manifestations including pseudocoma. Thus, in the setting of magnesium toxicity and apparent coma, sedation and analgesia are requisite to prevent pain and emotional trauma associated with resuscitation procedures.

92. VANCOMYCIN TOXICITY IN A NEONATE SUCCESSFULLY TREATED WITH EXCHANGE TRANSFUSION
Christian P. Lintner1, Heather Ellsworth1, Jamie M. Hess2, Shin K. Kwon2, Jon B. Cole2
1Hennepin Regional Poison Center, Minneapolis MN USA; 2University of Minnesota Medical Center, Minneapolis MN USA

Background: Vancomycin (VCM) is a commonly used antibiotic for treatment of methicillin-resistant Staphylococcus aureus (MRSA) as well as other gram-positive bacteria. VCM poisoning Results: in renal failure and ototoxicity. We report a case of VCM toxicity in a 7 day-old infant with subsequent renal dysfunction that was successfully treated with plasma exchange transfusion (PET).

Case Report: A 7 day-old girl born at 34 weeks, 1 day, weighing 1,847 g was treated with VCM, 12.5 mg/kg every 12 hours, concomitantly with gentamicin for suspected necrotizing enterocolitis (NEC). Her VCM blood level after dose 3 returned at 328 mcg/mL (target range 5-20 mcg/mL) and was similarly elevated to 317 mcg/mL and 323 mcg/mL on 2 additional measurements over the next 6 hours. On her 11th day of life she was transferred to a specialty center due to renal dysfunction; her serum creatinine (SCr) had risen from 0.48 mg/dL at birth to 1.71 mg/dL with a decline in her urine output (UOP) to 1.5 mL/kg/hr. Her gentamicin level was 4.8 (therapeutic range 5-10 mcg/mL). Given the acute renal dysfunction, decreased UOP and significantly elevated VCM level, PET was initiated. The VCM level 13 hours after completion of PET was 130 mcg/mL. A second PET was performed; the VCM level 7 hours later was 44.6 mcg/mL and her UOP improved to 6 mL/kg/hr with the addition of furosemide. Twenty hours after the second PET, the VCM level was 8.8 mcg/mL. She continued to improve and was transferred back to the original hospital after 4 days with a Cr of 0.48 mg/dL. Iatrogenic error was identified as the likely cause of her elevated VCM level.

Discussion: VCM is being used with increasing frequency as clinicians deal with the increased prevalence of MRSA. Renal dysfunction is a known complication of VCM. As VCM is cleared via the kidneys renal failure prolongs the VCM half-life, which in turn causes higher VCM levels and increased toxicity. Case reports of the use of various treatment modalities, including multi-dose
activated charcoal (MDAC), hemodialysis (HD), and PET to treat increased VCM levels exist. Our case presented some unique clinical challenges. HD would have been extremely difficult and potentially dangerous given this child’s size. MDAC was not an option since the child was NPO for her suspected NEC. A previous case report of PET used to treat VCM overdose showed this method to be largely ineffective. Our case, however, illustrates the possible benefit of PET in the appropriate clinical circumstance.

**Conclusion:** Plasma exchange transfusion may be beneficial in the treatment of some cases of vancomycin toxicity in neonates, particularly when other options may not be feasible.

93. MASSIVE UNINTENTIONAL OVERDOSE OF LOPINAVIR/ RITONAVIR IN A 1 DAY OLD PREMATURE NEONATE

Harry C. Karydes, Mark B. Mycyk
Cook County Hospital (Stroger), Chicago IL USA

**Background:** Lopinavir/ritonavir oral solution (lopinavir 80mg/ritonavir 20mg per mL) is a co-formulation of HIV-1 protease inhibitors and used in combination with other anti-retroviral therapy to prevent or reduce perinatal transmission of HIV. Human pediatric experience of acute overdoses with lopinavir/ritonavir is limited. We report a large unintentional overdose of lopinavir/ritonavir in a one day premature neonate.

**Case Report:** A female neonate was born (birth weight 1380 grams) via emergent caesarean section at 29 weeks to a newly diagnosed HIV+ mother with no prenatal care. Patient was immediately intubated for cyanosis, hypoxia and weak tone. In addition to neonatal intensive supportive care, zidovudine 1.5mg/kg every 12 hours IV, lamivudine 2mg/kg every 12 hours IV nasogastric (NG) tube and lopinavir/ritonavir 12mg/kg (16.8 mg) every 12 hours via NG tube was recommended.

Seven hours after birth, patient received 16.8 milliliters (960mg/kg) rather than 16.8 milligrams (12mg/kg) of lopinavir/ritonavir—an 80 fold dosing error. After recognizing that the parent compound and the diluent may cause toxicity, additional lab work was obtained serially. The patient’s AST/ALT peaked 55 hours post-ingestion at 1402 and 418 (U/L) respectively. The neonate’s ethanol level was 172mg/dL 14 hours after administration and undetectable at 35 hours after ingestion. A lactate level of 11.7 mmol/L was noted 13 hours after ingestion. Over the following 3 days, the patient’s arterial pH ranged between 7.144-7.350 with its nadir 15 hours after administration of lopinavir/ritonavir. The patient had no episodes of cardiovascular compromise and no episodes of apnea on the ventilator.

**Discussion:** Lopinavir/ritonavir oral solution contains alcohol (42.4% v/v) and propylene glycol (15.3% v/v). There is little known about lopinavir/ ritonavir overdose especially in the pediatric population. A previously noted neonatal death occurred from a 10 fold dosing error, much lower than in our case. Also, life threatening events such as CNS depression and respiratory compromise have been reported to the FDA adverse events reporting system. Fortunately this patient did not exhibit such significant manifestations.

**Conclusion:** We report a massive unintentional ingestion of lopinavir/ritonavir in a premature neonate with transaminitis, lactic acidosis and a significant ethanol level but no significant cardiovascular or neurological compromise.

94. CHILD ABUSE VIA HEROIN INJECTION

Brian J. Wolk, Matthew Zuckerman, Jarrett Burns, Christina Hernon
University of Massachusetts Medical School, Worcester MA USA

**Background:** Child maltreatment affects over 700,000 children annually in the United States.1 We report on the case of a 3-year old female who was the victim of child abuse by heroin injection.

**Case Report:** An otherwise healthy 3-year-old female with an upper respiratory infection was brought to the emergency department with lethargy, miosis, bradypnea, and vomiting, as well as ecchymosis in the right antecubital fossa. She had been left in the care of the mother’s boyfriend, who is not the biological father. The child reported that the mother’s boyfriend had given her “a shot.” The mother reported that her boyfriend had a history of IV drug abuse, but denied injecting the child when confronted.

A urine drugs-of-abuse immunoassay was positive for opiates (detection limit 300 ng/mL of morphine), but negative for benzoylcegonine, cannabinoids, and barbiturates. A urine gas chromatography-mass spectrometry screen was positive for 6-monoacetylmorphine, morphine, codeine, and dilitiazem, as well as caffeine. The patient was admitted to the pediatric intensive care unit for 24-hour monitoring. Mandated reporting of child abuse was completed, and criminal charges were filed against the mother and the suspected perpetrator. The patient recovered uneventfully from the acute intoxication, was given prophylaxis for HIV and Hepatitis B, and was discharged to foster care. Three months after evaluation, she remained negative for HIV and Hepatitis C.

**Discussion:** The history, clinical presentation, and presence of 6-monoacetylmorphine as well as common regional adulterants of heroin in urine are consistent with injection of heroin by an adult as a manifestation of child abuse. Early childhood exposure to injected heroin has not previously been described in the literature, and is of uncertain clinical and psychosocial significance.

**Conclusion:** To our knowledge, this is the first report of a case of child abuse via heroin injection.

Reference


95. BARBITAL BUFFER POISONING: INHALATION DUE TO MISUSE OF A LABORATORY REAGENT

Rudy Rose, Brandon Wills, Kirk Cumpston
Virginia Poison Center, Richmond VA USA

**Background:** Barbital buffer is a laboratory reagent used in protein gel electrophoresis. It is chemically similar to long-acting barbiturates, with sedative-hypnotic effects, though it is not available as a medication. We report a case of unintentional poisoning via inhalation, dermal and ocular exposure following use in a make-shift home laboratory.

**Case Report:** A 30-year-old doctoral student continued experiments at home in the sink of a poorly ventilated bathroom after his academic lab lost funding. He apparently was using various solvents and reagents that he obtained from work. After about a week, he sought medical care for headaches, confusion, ataxia, slurred speech, incoordination, unsteady gait, impaired attention/memory, and recent fever, nausea, vomiting and diarrhea. On exam he was awake and oriented but confused, was slow to respond and had diffuse muscle weakness and ataxia. Vital signs were normal. Head CT, serum chemistries, hepatic and renal function tests were normal. A urine drug screen was positive for barbiturates. Serum was negative for phenobarbital and the patient denied barbiturate or other drug use. The patient was admitted for observation and received supportive care. Within 24 hours all CNS symptoms had resolved, except for headache, photophobia and nausea.

Upon further questioning, the patient admitted to using barbital buffer powder in his home experiments, from which he had multiple inhalation and dermal exposures. He denied ingestion or use of any personal protective equipment. An assay developed by our hospital clinical toxicology laboratory revealed a serum barbital concentration of 156 mg/L. The patient was discharged in good condition on hospital day three.

**Case Discussion:** Barbital was synthesized in 1902 as the first barbiturate medication. It fell out of favor once phenobarbital was marketed in 1912, and is no longer used in clinical medicine. Barbital differs from phenobarbital in that it is non-phenolic and contains a phenolic group instead of a phenol. No toxic effects are known from percutaneous absorption, and no oral toxicity data exist. Barbital is a weak hypnotic, though it is not available as a medication. We report a case of unintentional poisoning via inhalation, dermal and ocular exposure following use in a make-shift home laboratory.

**Case Report:** A 30-year-old doctoral student continued experiments at home in the sink of a poorly ventilated bathroom after his academic lab lost funding. He apparently was using various solvents and reagents that he obtained from work. After about a week, he sought medical care for headaches, confusion, ataxia, slurred speech, incoordination, unsteady gait, impaired attention/memory, and recent fever, nausea, vomiting and diarrhea. On exam he was awake and oriented but confused, was slow to respond and had diffuse muscle weakness and ataxia. Vital signs were normal. Head CT, serum chemistries, hepatic and renal function tests were normal. A urine drug screen was positive for barbiturates. Serum was negative for phenobarbital and the patient denied barbiturate or other drug use. The patient was admitted for observation and received supportive care. Within 24 hours all CNS symptoms had resolved, except for headache, photophobia and nausea.

Upon further questioning, the patient admitted to using barbital buffer powder in his home experiments, from which he had multiple inhalation and dermal exposures. He denied ingestion or use of any personal protective equipment. An assay developed by our hospital clinical toxicology laboratory revealed a serum barbital concentration of 156 mg/L. The patient was discharged in good condition on hospital day three.

**Case Discussion:** Barbital was synthesized in 1902 as the first barbiturate medication. It fell out of favor once phenobarbital was marketed in 1912, and is no longer used in clinical medicine. Barbital differs from phenobarbital in that it is non-phenolic and contains a phenolic group instead of a phenol. No toxic effects are known from percutaneous absorption, and no oral toxicity data exist. Barbital is a weak hypnotic, though it is not available as a medication.
96. BETA-BLOCKER AND CALCIUM CHANNEL BLOCKER ERRORS REPORTED TO A POISON CENTER: CASE CHARACTERISTICS AND OUTCOMES

Janet L. Blasko1, Curtis J. Elko1
1Washington Poison Center, Seattle WA USA; 2Ohio State University School of Pharmacy, Columbus OH USA

Background: Beta-blockers (BB) and calcium channel blockers (CCB) are commonly prescribed antihypertensives. Therapeutic errors could cause profound cardiovascular effects, yet published information is scant. The present study Objective is to characterize BB/CCB therapeutic errors and identify predictors of toxicity.

Methods: A search of the Poison Center Toxicall® database identified 3071 cases of BB/CCB therapeutic errors from the year 2000 through March 2010. Cases were sorted as to management site and the presence of cardiovascular symptoms (CVS).

Results: The vast majority of cases (2820) were managed at home but 86% of these were not followed to an outcome. Cases at hospitals numbered 251, 106 with (+)CVS, 145 without (−)CVS. The following Tables 1-3 describe characteristics among these groups.

Table 1. Characteristics of cases grouped by site of management and cardiovascular symptoms (CVS).

<table>
<thead>
<tr>
<th>Management site</th>
<th>Mean Age (years)</th>
<th>BB cases</th>
<th>CCB cases</th>
<th>BB+CCB cases</th>
<th>Verapamil or Diltiazem</th>
<th>Dihydropyridine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospitals+CVS</td>
<td>N = 106 (hospital)</td>
<td>65 SD 20</td>
<td>44 (42%)</td>
<td>46 (43%)</td>
<td>16 (15%)</td>
<td>39 (37%)</td>
</tr>
<tr>
<td>Hospital – CVS</td>
<td>N = 145 (hospital)</td>
<td>56 SD 25</td>
<td>68 (47%)</td>
<td>62 (43%)</td>
<td>15 (10%)</td>
<td>38 (26%)</td>
</tr>
</tbody>
</table>

Dihydropyridines vamlodipine, felodipine, nifedipine, isradipine, nicardipine, nimodipine, nisoldipine.

Table 2. Distribution of CVS of Patients at Hospital (N = 106).

<table>
<thead>
<tr>
<th>Asystole</th>
<th>Death</th>
<th>Bradycardia</th>
<th>Conduction/EKG abnormalities</th>
<th>Hypotension</th>
<th>Tachycardia/hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>1*</td>
<td>59</td>
<td>8</td>
<td>49</td>
<td>7</td>
</tr>
</tbody>
</table>

*(case included diltiazem, digoxin, warfarin).

Table 3. Treatments for cases needing more than IV fluids for CVS (N = 30).

<table>
<thead>
<tr>
<th>Pacemaker</th>
<th>Vasopressor(s)</th>
<th>Calcium</th>
<th>Glucagon</th>
<th>Insulin</th>
<th>Atropine</th>
<th>Anti-arrhythmic</th>
<th>Dighibid</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>8</td>
<td>19</td>
<td>16</td>
<td>5</td>
<td>7</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

Discussion: This data shows most therapeutic errors of BB/CCB are managed at home. Hospitalized patients with CVS tended to be older than those without CVS (65 versus 56 years) but the range of ages varied widely. Patients managed at home rather than at hospital were more likely to have taken a BB rather than a CCB (65 versus 29%) and were less likely to have taken a combination of BB/CCB than their hospitalized counterparts (5.6% versus 12.4%). Patients at hospital with CVS also included more exposure to verapamil and/or diltiazem rather than the dihydropyridines (37% versus 17%). Despite significant hypotension and bradycardia, only 30 patients required more than IV fluids as treatment.

Conclusion: Therapeutic errors with BB/CCB are usually managed at home. The predictive factors for those who will require hospitalization for significant CVS remain elusive.

97. PEDIATRIC CAFFEINE TOXICITY FROM A WEIGHT-LOSS AGENT CONFIRMED WITH QUANTITATIVE TESTING

Daniel E. Brooks, Joshua Canning
Banner Good Samaritan Poison and Drug Information Center, Phoenix AZ USA

Background: Caffeine is a ubiquitous xenobiotic found in many beverages and medications, including weight loss agents. As a methylxanthine, caffeine results in adenosine receptor antagonism, release of endogenous catecholamines, and inhibition of phosphodiesterase. In mild toxicity gastrointestinal and tachycardia are common, while significant ingestions may result in seizures and life-threatening tachydysrhythmias.

Case Report: An 11-year-old boy with autism presented to the Emergency Department (ED) after ingesting up to 60 tablets of Apideixin, a non-FDA approved weight-loss agent purported to contain dicaffeine malate. He rapidly developed emesis and altered sensorium. In the ED he was noted to be tachycardic with multiple runs of both ventricular and supraventricular tachycardia, and frequent premature ventricular contractions (PVCs). He was anxious, diaphoretic and mydriatic, with repeated emesis. Benzodiazepines and esmolol were recommended; he only received lorazepam 2 mg intravenously prior to transfer. At our facility he continued to have tachycardia (~150bpm) with PVCs and bigeminy. Pertinent laboratory findings included potassium of 2.2 mmol/L and glucose of 332 mg/dL. Comprehensive urine drug testing revealed caffeine and theophylline. Serum caffeine concentration, obtained approximately 8 hours after ingestion, was reported as 130 μg/mL using dilution technique. Serum theophylline was measured at 2.7μg/mL. The patient was treated with IVFs, benzodiazepines, and a transient insulin infusion. The gastroenteritis, dysrhythmias and mental status improved over 24 hours and he was discharged home on hospital day two.

Discussion: Non-FDA-approved weight-loss medications are advertised to contain substances from various proprietary compounds, amphetamine-like xenobiotics or methylxanthines. It is often difficult to confirm the true contents of these agents. In this case, significant caffeine toxicity was confirmed with quantitative serum concentration. Prior deaths have been reported with caffeine concentrations as low as 80 μg/mL. Based on the delay in obtaining blood and expected pharmacokinetics, it is assumed that our patient’s peak caffeine level was >130 μg/mL.

Conclusion: Weight loss agents continue to be a potential source of significant poisonings. This case illustrates survival with aggressive supportive care despite potentially fatal caffeine ingestion.

98. PROLONGED ALTERED MENTAL STATUS AND BRADYCARDIA FOLLOWING PEDIATRIC DONEPEZIL INGESTION

Fiona M. Garlich1, Syed K. Shah1, Lewis S. Nelson1, Mary Ann Howland2
1New York City Poison Control Center, New York NY USA; 2Bronx Lebanon Hospital, Bronx NY USA; 3St. John’s College of Pharmacy, Queens NY USA

Background: Donepezil is a centrally-acting, reversible acetylcholinesterase inhibitor that is used in the treatment of Alzheimer’s dementia. Altered mental status, nausea, vomiting, and bradycardia have been reported in therapeutic and supratherapeutic ingestions of donepezil in the elderly population. We report a case of altered mental status and prolonged bradycardia in a child with a single-pill ingestion of donepezil.

Case report: A 14 month-old boy was brought to the emergency department three hours after ingesting one of his grandfather’s donepezil tablets (10mg). Upon arrival, he was somnolent and drooling, with multiple episodes of vomiting and diarrhea. Pupils were normal. Initial vitals: temperature, 98.2°F; blood pressure, 103/66 mmHg; heart rate, 140/min; respiratory rate, 36/min; oxygen saturation, 97%. His drooling, vomiting, and diarrhea resolved, but he remained intermittently agitated and was admitted to the intensive care unit for observation. Over the course of the following four days, he had intermittent, episodes of asymptomatic bradycardia to a low of 55/min, primarily when sleeping. A transient episode of junctional rhythm was observed. His bradycardic episodes persisted despite normalization of mental status on hospital day 3. Serum donepezil level 97 hours post ingestion was 10 ng/ ml. He did not require atropine treatment, and was discharged in stable condition on hospital day 5.

Case Discussion: Donepezil has a prolonged elimination of half life in adults of approximately 70 hours. Despite its relative specificity for central acetylcholinesterases, peripheral cholinergic symptoms such as drooling, vomiting, diarrhea and bradycardia have been described, primarily in the elderly population. To our knowledge, this is the first reported case of a symptomatic pediatric ingestion of donepezil.
Conclusions: As a single tablet ingestion, donepezil may cause prolonged altered mental status and bradycardia in the pediatric population.

99. CAPECITABINE
Kensuke Kanie1, Anna Arroyo Plasencia1, William Dribben1, Robert Tremmel2
1Washington University School of Medicine; 2Wellstat Therapeutics Corporation

Background: Capecitabine is a 5'-deoxy-5-fluorouridine prodrug of fluorouracil (FU) approved by the FDA for the treatment of adult colorectal and metastatic breast cancer and currently under investigational study for pediatric gliomas. Despite a lack of approved indications in the pediatric population, it is increasingly used in adult outpatient settings creating an environment for pediatric exposures. We describe a pediatric capecitabine exposure treated with uridine triacetate (UT), an investigational antidote for FU overexposure, granted orphan-drug status by the FDA and EMA.

Case Report: A previously healthy 22 month old male presented to a local hospital after being found with his grandfather’s capecitabine. Two 500 mg capecitabine tabs and a portion of a third were missing. The patient received activated charcoal and was transferred to a tertiary care center. On arrival, he was alert, active, and playful. Baseline vital signs were normal and laboratory values were: WBC 6.5; ANC 1560; Hgb 10.7; Hct 31; plt 328; Cr 0.3; AST 61; and ALT 27. Based on his weight of 12.5 kg, this was a potentially lethal ingestion. An experimental investigational new drug application was submitted and approved by the FDA and UT was obtained emergently from Wellstat Therapeutics Corporation (Guthersburg, MD). Treatment was started approximately 7 hrs after ingestion at an initial oral dose of 11 gm every 6 hours. The patient was unable to tolerate the taste of the oral formulation when mixed with food, and was transitioned to nasogastric tube administration. The dose was adjusted based on body surface area to 3.3 gm every 6 hours to aid in drug administration. The patient completed 20 doses and remained asymptomatic with normal discharge laboratory values: WBC 7.4; ANC 1554; Hgb 11.2; Hct 33; plt 432; Cr 0.3; and ALT 23. Following discharge, his ANC reached a low of 1029 on day 12 and increased to 1664 on day 15. AST was 73 and ALT was 66 on day 15 and both returned to normal by day 19.

Case Discussion: Although our patient potentially ingested an amount of capecitabine that could have caused significant complications, only a mild ANC decrease and a minimal transient rise in transaminases were noted following treatment with UT. ANC returned to pre-exposure levels by day 15. This decrease in ANC was mild compared to the significant myelosuppression possible following FU exposure. Serving as a direct antagonist of 5-FU, UT may have prevented a more serious clinical outcome.

Conclusions: We report a pediatric exposure to capecitabine treated with UT. Our patient demonstrated mild transient granulocyte suppression that could have been much more severe if treatment with UT had not been promptly initiated.

100. CLINICAL EFFECTS AND CLINICAL OUTCOMES FOLLOWING THE INJECTION OF EXTENDED RELEASE GUANFACINE (INTUNIV®) IN CHILDREN
Douglas J. Borys1, Hollie W. Blair1, Ryan P. Morrissey1, David L. Morgan2
1Central Texas Poison Center, Scott and White Healthcare, Temple TX USA; 2Department of Emergency Medicine, Scott and White Healthcare, Temple TX USA

Background: Guanfacine extended release (Intuniv®) was approved in 2009 as a secondary agent in the treatment of attention deficit hyperactivity disorder for patients 6-17 years of age. It is a central alpha agonist and has a long history of use as a prompt release formulation for hypertension. Adverse effects associated with guanfacine use in adults include drowsiness, dizziness and headache. This is the first study of pediatric exposures to guanfacine extended release.

Objective: The purpose of this study was to determine the clinical effects of accidental guanfacine ingestion by young children.

Methods: This was a retrospective, observational study of one state’s poison centers’ calls of exposures to guanfacine (Intuniv®) as a single agent from January 2010 through February 2011 for children age 6 years and younger. All patients were followed to a known outcome.

Results: There were 32 children who met the inclusion criteria. They ranged from 14 months to 6 years in age with a mean age of 3.85 years. Dose ingested ranged from 0.5–15 mg. Nineteen (59.4%; 95% CI: 42.3–74.5%) were symptomat. Common effects noted included: drowsiness (46.9 %), hypotension (21.9%), bradycardia (12.5 %) and agitation (9.38 %). Two patients were noted to be hypertensive. Twenty-nine patients (90.6%) were managed at health care facilities. Of those, 9 were treated with intravenous fluids and 7 were given one dose of activated charcoal. No patient received vasopressors. Thirteen children (40.6%) had no effect from their exposure, 8 (25.0%) had a minor effect and 11 (34.4%) had moderate effects. No patient had a major effect or died (95CI: 0–10.7%).

Conclusions: This is the first study of ingestions of extended release guanfacine in young children. Low numbers, its retrospective nature and reliance on caller information limit the study. About one-third of children developed moderate clinical effects and none developed major effects. More studies are needed to determine the appropriate referral dose and emergency management of this age group.

101. A DESCRIPTIVE STUDY OF ANTI NEOPLASTIC DRUG EXPOSURES IN PEDIATRIC PATIENTS 5 YEARS OLD AND YOUNGER
Stephen L. Thornton1, R. Romasco1, K. Soleymani1, R. F. Clark4, F. L. Cantrell2, H. Sarid3
1Veteran’s Affairs Medical Center, San Diego CA USA; 2California Poison Control System, San Diego CA USA; 3UC San Diego Skaggs School of Pharmacy, San Diego CA USA; 4University of California - San Diego, San Diego CA USA

Introduction: The use of antineoplastic drugs continues to increase, mirroring the increasing prevalence of cancer. There is limited data available on pediatric exposures to these agents. We sought to identify characteristics of pediatric patients 5 years and younger with reported exposure to antineoplastic drugs.

Methods: We performed a retrospective review of a statewide poison system’s database for all cases of pediatric (5 years of age or less) exposures to antineoplastic agents from Jan 2000 – Dec 2009. Data collected include gender, age, agent of exposure, route of exposure, dose, drug concentration, reason for exposure, symptoms, outcomes, interventions, and length of hospital stay.

Results: There were a total of 329 patients, 54% of which were female (n = 179). Eighty percent of the patients were two years of age or younger (n = 265) with a mean age of 2 years. The average reported weight was 12.7 kg (n = 185) Almost all exposures were ingestions (n = 312), took place at home (n = 322), and were not the patients’ medication (n = 299). [LC1] Exposures to 29 different antineoplastic agents were identified. Methotrexate (23%, n = 76), azathioprene (17%, n = 56), mercaptopurine (10%, n = 32), tamoxifen (9%, n = 31), and anastrozole (7%, n = 22) accounted for 66% of the exposures. Seventeen (6%) of the exposures were to veterinary antineoplastic agents. In 319 (97%) of the exposures, no symptoms were reported after the ingestion. [LC2] When symptoms were reported, GI symptoms were the most common (n = 7). There were no deaths [LC3] reported in this series. Only one serious outcome was reported in this study: Pancytopenia and hepatitis developed in a 5 year old child who was mistakenly given 5 times more mercaptopurine (305 mg/m2) than recommended over a 9 day period. Less than 35% (n = 115) of the cases were evaluated at a health care facility, with 20 (6%) admitted to the hospital. Of admitted patients, 10 (50%) had a hospital stay of 1 day or less. Leucovorin was administered 6 times for methotrexate exposures and 1 time for a vincristine exposure.

Conclusions: In this retrospective study, unintentional exposures to antineoplastic drugs among pediatric patients 5 years or younger was associated with minimal complications and no deaths.

102. RETROSPECTIVE REVIEW OF POISON CONTROL CENTER CASES INVOLVING COCAINE EXPOSURES IN CHILDREN, 1997-2010
Michelle Fleurat1, Patil Armenian1, Kent R. Olson1
1California Poison Control System, San Francisco Division, University of California, San Francisco, San Francisco CA USA; 2Alameda County Medical Center: Highland Hospital, Department of Emergency Medicine, Oakland CA USA
Background: Cocaine is a local anesthetic and sympathomimetic drug with the potential to cause morbidity and mortality in inadvertent exposures in children. The purpose of this study was to assess the demographic and clinical characteristics of pediatric cocaine exposures reported to the California Poison Control System (CPCS).

Methods: A 13 year retrospective study was conducted using CPCS electronic records from January 1997 to December 2010 involving pediatric patients less than 6 years of age with cocaine exposures. The exposures were analyzed for age, sex, race, type of toxic exposure, location of incident, complications and eventual disposition.

Results: 86 cases met initial inclusion criteria. Four were excluded because the exposures were prenatal and one was excluded due to incorrect age. Of the 81 remaining cases, 21 were observed at home or did not present to a health care facility, 23 were seen in and sent home from the emergency department (ED), 15 were admitted to unmonitored hospital beds and 18 required intensive care unit admission. In the 46 cases where the route of ingestion was known, 37 were oral, 7 inhalaed and 2 topical. Of the 34 cases where the location of exposure was recorded, the majority (n = 21) occurred in the child’s home. Prenatal and one was excluded due to incorrect age. Of the 81 remaining cases, 21 were observed at home or did not present to a health care facility, 23 were seen in and sent home from the emergency department (ED), 15 were admitted to unmonitored hospital beds and 18 required intensive care unit admission. In the 46 cases where the route of ingestion was known, 37 were oral, 7 inhalaed and 2 topical. Of the 34 cases where the location of exposure was recorded, the majority (n = 21) occurred in the child’s home. Presenting symptoms included agitation in 19, seizure in 25, respiratory distress in 7, ataxia in 4, fever in 2 and in one patient, cardiac arrest. The expression of complications during hospital admission were tachycardia (n = 19), hypertension (n = 7), fever (n = 4), need for endotracheal intubation (n = 3), persistent neurologic symptoms (n = 5). Treatment was provided in 35 patients, 33 were observed only, and 13 had an unknown treatment course. Single dose activated charcoal (SDAC) was given in 17 cases, SDAC and lavage in 3, SDAC and whole bowel irrigation (WBI) in 1 and WBI alone in 2 cases. 14 required therapy for agitation and/or seizures, using barbiturates, benzodiazipines, anticonvulsants and/or deep sedation. Urine drug of abuse screening was recorded in 43 cases, of which 84% were positive for cocaine. Child protective services was contacted in 37 (45.7%) cases. Outcomes were unknown in 17%, no effect in 28.4%, mild effect in 18.5%, moderate effect in 23.5% and severe effect in 12.3%. No deaths were recorded in this time period. 63% (22/35) of cocaine drug screen positive cases had either moderate or severe outcomes.

Conclusion: Although infrequently reported, pediatric cocaine exposures resulted in significant morbidity. Our data supports the current standard of care to refer all pediatric cocaine exposures to hospitals for evaluation and possible treatment.

103. MANAGEMENT OF METHYLERGONOVINE INDUCED RESPIRATORY DEPRESSION IN A NEWBORN WITH NALOXONE

Ross W. Sullivan, Jamie Nelsen, Michael Holland
SUNY Upstate Medical University, Syracuse NY USA

Background: We report a case of a female neonate who developed significant clinical toxicity, including respiratory depression, following the unintentional administration of methylergonovine. The respiratory depression was attenuated with naloxone, preventing the potential need for intubation and prolonged ventilation. To our knowledge this is the first time naloxone has been suggested for methylergonovine toxicity.

Case Report: A full-term female neonate, 3345 grams, was delivered vaginally without issue. The child had an initial Apgar score of 9 at one minute. Approximately 10 minutes after delivery the infant was inadvertently administered 0.1 mg of methylergonovine intramuscularly that was ordered for her mother instead of the intended vitamin K. Thirty minutes later the child appeared mottled with cyanotic extremities. Respiratory effort was diminished with shallow respirations. Her oxygen saturation at that time was 75%. Continuous positive pressure ventilation was applied via bag-mask. Over the next 30 minutes the patient continued to appear ill and her respiratory effort worsened. The Poison Center was contacted and the consulting toxicologist advised naloxone, 0.4 mg IM, to mitigate respiratory depression. Within 5 minutes respirations improved to 40 breaths per minute. The infant’s color improved and she began resisting ventilations and crying loudly. Bag-mask was discontinued and she was placed in a head box oxygen tent. Despite improved respiratory drive, the patient continued to have clenched fists, hypertonus, and jerkiness. The patient was transferred to a neonatal intensive care unit. A single episode of apnea was noted at the referral facility, which responded to physical stimulation. The child continued to improve and was back to baseline that evening.

Case Discussion: We present the first case of a methyergine induced respiratory depression that was managed successfully with naloxone. Methylergonovine toxicity in neonates has been commonly associated with respiratory depression necessitating ventilatory support, although the mechanism remains unclear. In consideration of chemical structural similarity between methylergonovine and morphine, as well as signs/symptoms consistent with opioid-induced respiratory depression, naloxone was suggested.

Conclusions: It appears that naloxone may reverse methylergonovine toxicity in neonates. The identification of a safe and potentially useful antidote to mitigate respiratory depression, potentially avoiding the need for intubation and more invasive interventions in this patient population is important.

104. CHRONIC AMITRIPTYLINE OVERDOSE IN A CHILD

Jennifer Lowry, Alan Clement, Jeremiah Raney, Gary Wasserman
Children’s Mercy Hospitals and Clinics, Kansas City MO USA

Amitriptyline, a tricyclic antidepressant, has a well-described toxicity profile, and acute ingestions are common in pediatric toxicology. However, the literature contains scant information in regard chronic overdose in adults or children. We describe a case of a 6 year old girl who was prescribed amitriptyline, 10 mg tablets X 3 (30mg total) nightly for sleep problems, but was mistakenly dispensed 100 mg tablets X 3 for a total of 15mg/kg nightly for over a month. She was noted to have mental status changes and difficulty reading shortly after starting the medication. After receiving the overdose for a month, she presented to the local children’s hospital in status epilepticus with significant cardia conducation abnormalities on EKG. Her QRS was 134 msec and QTc was calculated at 640 msec. Her total amitriptyline/ nortriptyline level was found to be 1676 ng/ml (normal therapeutic level< 250 ng/ml). She was treated for several days with sodium bicarbonate as her QRS would rebound above 100 msec when Bicarb was discontinued. Within 24 hours of admission, her neurologic status improved and had returned to baseline within several days. Her EKG normalized, but worsened after developing iatrogenic vomiting. Amitriptyline levels increased during this time and were presumed to be secondary to release of drug from fat stores. After fluid correction, she was discharged home without apparent sequelae. Thus, we report a novel case of chronic amitriptyline overdose of a known daily fatal dose. It is presumed that there were possible protective mechanisms that allowed this child to survive this dose.

105. PEDIATRIC INGESTIONS OF DISSOLVABLE NICOTINE PRODUCTS REPORTED TO POISON CENTERS

Mathias B. Forrester 1, Leo Artalejo 2, Emilio Saenz 2
1Department of State Health Services, Austin TX USA; 2West Texas Regional Poison Center, El Paso TX USA

Background: Dissolvable nicotine products are marketed for use when a person is in a setting where they cannot smoke. These products may be similar in appearance to candy, possibly resulting in accidental ingestion by young children. This study described ingestions of dissolvable nicotine products by young children reported to poison centers.

Methods: This retrospective investigation identified ingestions of dissolvable nicotine products by children age 0-5 years reported to a statewide poison center system during 2002-2010. The distribution of ingestions was determined for various demographic and clinical factors.

Results: Fourteen dissolvable nicotine product ingestions were identified. The patients’ age range was 17 months-4 years; 64.3% were female and 35.7% male. All of the ingestions were unintentional and occurred at the patient’s own residence. No ingestions involved other substances. The management site was 42.9% managed on site, 21.4% already at or en route to a healthcare facility, and 35.7% referred to a healthcare facility. The medical outcome was 50.0% no effect, 28.6% minor effect, 14.3% moderate effect, and 7.1% effect considered unrelated to the product. The reported adverse clinical effects were vomiting (14.3%), tachycardia (7.1%), abdominal pain (7.1%), nausea (7.1%), and agitation (7.1%). The reported treatments were administration of activated charcoal (50.0%), cathartic (26.8%), dilution/irrigation/wash (26.8%), food/snack (14.3%), ipecac (7.1%), and IV fluids (7.1%).
Discussion: During a recent 9-year period, 14 dissolvable nicotine product ingestions by young children were reported to a statewide poison center. All of the exposures were accidental and occurred at the child’s own residence. Most of the ingestions were managed at a healthcare facility, and the majority involved no major outcomes.

Conclusion: In spite of the fact that dissolvable nicotine products may resemble candy, so far relatively few accidental ingestions by young children have been reported to poison centers. Those ingestions that are reported may require treatment at a healthcare facility. However, serious medical outcomes are not likely to occur.

106. DEATH FROM CONSTIPATION: OFF LABEL USE OF COLCHICINE RESULTS: IN HIGH MORBIDITY IN ELDERLY
Brett Roth, Melody Gardner
North Texas Poison Center at Parkland, Dallas TX USA

Index Case: An 86 year old female with a history of stroke, hypertension, congestive heart failure, and gastritis presented to her primary provider with chronic constipation. She was given a new prescription for colchicine, #60, and was told to take one every 10 minutes with a maximum of three in order to assist her bowel movements. Instead, she continued taking the colchicine every 10 minutes until her daughter called 4 hours later, at which point she had consumed 49 tablets. The patient was transported by the daughter to the emergency department where nausea and vomiting developed. She was given charcoal and antiemetics in the emergency department and an increase in vomiting and diarrhea was noted over the next day. On hospital day #2 the patient went to the commode and was found slumped over and apneic. She continued to deteriorate over hospital day #3 and she went into asystole in the early afternoon of that day with evidence of multisystem organ failure and intractable acidosis. Toxicology screens were negative.

The above case prompted a review of a statewide poison center database involving colchicine exposures. Out of a total of 96 exposures without co-ingestion, 46% were from therapeutic errors. Twenty percent of these cases were associated with moderate to severe outcomes as defined by the National Poison Data System (NPDS) representing a high incidence of significant morbidity. Three deaths are described. The elderly make up a majority of therapeutic errors.

Conclusion: Therapeutic errors, with significant toxicity, are frequently seen with colchicine. Confusion about dosing regimens is concerning, especially among the elderly population. Future preventative measures should include clear dosing instructions with both patient and family members, or simple avoidance of colchicine entirely in elderly patients.

107. A CASE OF THYROTOTOXICOSIS DUE TO A COMPOUNDING ERROR
Mae F. De La Calzada-Jeanlouie, Howard A. Greller, Mark K. Su, Gar M. Chan
North Shore University Hospital, Manhasset NY USA

Background: The role of the pharmacist is deeply rooted in the specialty of compounding. In the past, pharmacists would compound organic materials to extract medicinal properties believed to be contained within. By the 1950s, large scale distribution by pharmaceutical companies became increasingly prevalent and overshadowed the need for compounding. Today, compounded products are still widely available, from intravenous chemotherapeutic agents to additives available at local retail pharmacies. The importance of this skill-set is treasured at a healthcare facility. However, serious medical outcomes are not likely to occur.

Conclusion: In spite of the fact that dissolvable nicotine products may resemble candy, so far relatively few accidental ingestions by young children have been reported to poison centers. Those ingestions that are reported may require treatment at a healthcare facility. However, serious medical outcomes are not likely to occur.

108. RISK FACTORS FOR HYPOGLYCEMIA IN UNINTENTIONAL INSULIN OVERDOSE
Rachel Gorodetsky1, Sean Ragone1, Robert Geller2, Gaylord Lopez1, Stephanie Hon1
1Georgia Poison Center, Atlanta GA USA; 2Emory University Department of Pediatrics, Atlanta GA USA

Background: In 2009, over 5500 calls involving insulin overdose were made to US poison centers (PC). Of these, 80% were unintentional; 30% were treated in a health care facility (HCF). Of cases that were followed to a known outcome, over 75% experienced minor or no effect. Although most insulin exposures are unintentional, the majority of case reports involve intentional exposures, and little literature exists addressing unintentional overdose. No studies have attempted to identify patients at increased risk of poor outcomes from accidental insulin overdose. If such information was known, it could help to guide triage by poison specialists (SPIs).

Methods: A retrospective chart review of all calls received by our PC involving unintentional insulin overdose and followed to a known outcome between 1/1/2001 and 12/31/2010. Data collected included patient age, type of insulin, units injected, magnitude of overdose, whether the patient was referred to a HCF, blood glucose (BG) nadir and peak, symptoms if any, whether others were home with the patient, and other medications used by the patient.

Results: We identified 439 eligible patients. Of those, 284 (64.7%) involved rapid-acting insulin, 70 (15.9%) regular insulin, 45 (10.3%) mixed insulin, 30 (6.8%) long-acting, and 10 (2.3%) intermediate-acting. Of all patients, 55 (12.5%) experienced a nadir BG < 60 mg/dL. Overall, 91 (20.7%) patients were referred to a HCF at the initial contact with the PC. Patients who overdosed on long-acting insulin were most likely to be referred, with 26.7% of those sent in. Of patients referred to a HCF, 11 (12%) experienced BG < 60 mg/dL, but hypoglycemia occurred in 44 patients at home (80% of patients with hypoglycemia). Patients who were referred to a HCF had a greater mean dose of insulin injected than those who were not (66 units vs. 39 units), but dose did not predict the likelihood of subsequent hypoglycemia. Patients who had BG < 60 mg/dL had a higher median magnitude of overdose than those who did not (4 fold vs. 3.5 fold), and a lower median peak BG (194 vs. 241 mg/dL). The average age of patients was 56 years, and did not differ among groups.

Conclusions: Almost 90% of patients with unintentional insulin overdose did not experience hypoglycemia. The insulin dose was not a predictor of hypoglycemia. Patients with lower peak blood glucose were more likely to experience a nadir < 60 mg/dL, but no threshold of peak values precluded later hypoglycemia. Given the low rate of hypoglycemia, it may not be appropriate to refer all to an HCF. Because hypoglycemia occurs in patients triaged to HCF and in those managed at home, patients should not be managed at home without another adult present and able to assist in initial patient care if hypoglycemia occurs.
109. ONDANSETRON TOXICITY IN A TODDLER RESULTING IN TACHYCARDIA, ALTERED MENTAL STATUS, AND QTc PROLONGATION

Steven C. Setzer, Jon B. Cole, Shin K. Kwon1, Samuel J. Stellpflug, Heather Ellsworth
Hennepin Regional Poison Center, Minneapolis MN USA

Background: Ondansetron is a commonly prescribed 5-HT3 antagonist used in the management of nausea and vomiting. Little is known about its potential adverse effects following an overdose; however, it has a well-established safety profile at therapeutic doses. We describe a case of a toddler who ingested a large quantity of ondansetron resulting in tachycardia, altered mental status, and QTc prolongation.

Case Report: A 15-month-old boy ingested a maximum of 18 ondansetron tablets prescribed to his mother from a bottle containing a mixture of 4 and 8 mg tablets. No other medications were accessible and no emesis was reported. After arrival to the Emergency Department, he was noted to be irritable with flushed skin, and tachycardic with a pulse between 170 and 190 beats/min. His initial ECG demonstrated sinus tachycardia with a QRS duration of 40 msec and a prolonged QTc of 490 msec. The patient subsequently became intermittently somnolent and agitated. Eight hours post-ingestion the patient was much improved with a heart rate of 120 beats/min and behaving normally. The child was observed overnight and discharged to home 19 hours post-ingestion; at the time of discharge, he was asymptomatic, had normal vital signs, and his QTc prolongation had resolved. He received no pharmacotherapy during his hospitalization.

Discussion: The potential for pediatric exposures to ondansetron and risk for subsequent toxicity is increased, parallelizing the rise in physician prescribing patterns for both children and adults. Despite its well-established therapeutic safety profile in adults, knowledge of toxicity in pediatric patients is limited. This case demonstrates that acute ondansetron toxicity in a toddler may result in somnolence, tachycardia, and prolongation of the QTc interval. These symptoms, though rare, have previously been reported in a 12-month-old male who also developed seizures, hepatotoxicity, and serotonin syndrome following the unintentional ingestion of 56-64 mg of ondansetron.

Conclusion: Healthcare providers should recognize the risk for acute toxicity following ondansetron overdose. This case illustrates a probable association between this commonly prescribed medication and the development of tachycardia, altered mental status, and QTc prolongation in an exposed toddler.

110. MEDICAL CHILD ABUSE WITH IPECAK: A VARIANT OF MÜNCHAUSEN SYNDROME BY PROXY

Jon B. Cole1, Robin M. Lloyd2, Heather Ellsworth1, Samuel J. Stellpflug1, Stacey A. Bangh1
1Hennepin Regional Poison Center, Minneapolis MN USA; 2Mayo Clinic, Division of General Pediatrics and Adolescent Medicine, Rochester MN USA

Background: Child maltreatment is a very common problem in the United States; over 702,000 children were victims in 2009. Medical Child Abuse by Proxy (MSbP), an extremely rare variant of child abuse and often requires multiple health care visits to diagnose. We present a case of MSbP involving intentional poisoning with Syrup of Ipecac.

Case Report: An 8 month-old girl had frequent visits to the emergency department (ED) for episodes of cyclical vomiting and constipation. No cause was found despite upper and lower endoscopies with biopsies, abdominal x-rays, MRIs of the head and lumbar regions, urine studies for infection and drugs, and multiple blood tests. The child was referred to a specialty center and her mother, a paramedic, relocated to be closer to this quaternary center. ED visits increased to every 3 days. The mother reported placing a nasogastric tube for fluid supplementation until the child could be formally evaluated. Further workup included: upper GI with small bowel follow through, abdominal ultrasound, extensive metabolic workup, stool studies, and additional screening blood tests. Providers noted symptoms often completely resolved within 12 hours of hospital admission. With an extensive negative work-up the diagnosis of MSbP was entertained. Poison Control was contacted and emetine and cephaeline levels were sent. The urine emetine level returned at 8.4 ng/mL (lowest limit of detection 5 ng/mL) and cephaeline level was undetectable (<0.5 ng/mL). Child Protective Services was contacted and the child was removed from the mother’s care, whereupon her symptoms resolved. Criminal charges against the mother have been filed.

Discussion: Intentional poisoning of children is uncommon. A 2-year prospective observational study in the United Kingdom identified only 65 cases with the most common drugs being anticonvulsants and opioids. Documented cases of intentional Ipecac poisoning are rare, particularly with Ipecac use becoming far less common. Our case is even more unique in that only emetine was detected. Emetine and cephaeline are the active alkaloids in Syrup of Ipecac that induce vomiting. Pharmacokinetic studies of both xenobiotics in humans revealed a half-life (t½) of 3.45-9.4 hours for cephaeline, while the t½ of emetine was longer at 65.4-163 hours. One explanation for this large difference is emetine’s enterohepatic recirculation.

Conclusion: Providers should be aware intentional poisoning with Syrup of Ipecac may be the cause of chronic vomiting. Detection is possible but only with a high level of suspicion and specialized testing. Emetine may be more readily detected than cephaeline given its longer half-life.

111. POISON CENTER MANAGEMENT OF INSULIN THERAPEUTIC ERRORS: CAN WE PREDICT WHO WILL BE HYPOGLYCEMIC?

Douglas J. Borys1, David L. Morgan2, Ryan P. Morrissey1, Richard M. Thompson1
1Central Texas Poison Center, Scott and White Healthcare, Temple TX USA; 2Department of Emergency Medicine, Scott and White Healthcare, Temple TX USA

Background: Poison centers have shown value by assisting in the management of poisoned patients and in keeping those patients at home whenever possible. In a recent retrospective study, Spiller found that poison centers managed 70 % of patients with an insulin unintentional therapeutic error at home. However, that study did not predict which of these patients will develop hypoglycemia. Defining those patients most likely to develop hypoglycemia will allow for improved patient care.

Objective: The purpose of this study is to compare key characteristics of patients who developed hypoglycemia following a therapeutic error with insulin to those of patients that had no hypoglycemia.

Methods: This was a retrospective, case control study of one state’s poison centers’ calls of therapeutic errors with insulin. Dates included January 2010 through February 2011. All patients were followed to a known outcome. Hypoglycemia is defined as a blood glucose less than or equal to 60 mg/dL at any time during follow-up. Age, gender, type of insulin, and initial blood sugar were measured prior to the error was 196.6 mg/dL. When comparing the hypoglycemia group with those that did not develop hypoglycemia we could find no statistical difference pre-error blood sugar, age, gender or type of insulin used. (p> .05).

Results: 105 patients met the inclusion criteria. Average age was 57.1 years. There were 67.3 % female and 32.7 % male. Twenty-seven patients used insulin aspart, 22 insulin lispro, 16 regular insulin, four insulin glargine and 38 used a variety of other insulin products. Twenty one patients (19.6 %; 95%CI: 13.5% - 28.7%) developed hypoglycemia. Where noted the mean measured blood sugar prior to the error was 196.6 mg/dL. When comparing the hypoglycemia group with those that did not develop hypoglycemia we could find no statistical difference in pre-error blood sugar, age, gender or type of insulin used.

Conclusions: This is the first study that utilizes poison center data to differentiate those patients that develop hypoglycemia from those that do not following a therapeutic error with insulin. This study was limited by its retrospective nature and low number of patients. We could find no statistical difference between the two groups across a variety of variables. Although only 19.6 % of patients developed hypoglycemia, all of these patients are at risk. More and larger studies are needed to predict which patients will develop hypoglycemia.

112. LARGEST REPORTED LIRAGLUTIDE OVERDOSE

Carrie A. Truitt, Daniel E. Brooks, Aaron Skolnik
Banner Good Samaritan Poison and Drug Information Center, Phoenix AZ USA
Background: Liraglutide (Victoza®), a glucagon-like peptide-1 receptor agonist (GLP-1 RA), was FDA approved for the treatment of Type II Diabetes Mellitus (DMII) in 1/2010. Similar to other GLP-1 RA (a.k.a. “incretin mimetic”) exenatide (Byetta®), liraglutide is known to stimulate insulin secretion, lower glucagon secretion and slow gastric emptying. It is available in disposable, pre-filled, multi-dose pens. Each pen can deliver 0.6 mg, 1.2 mg or 1.8 mg doses (6 mg/mL, 3 mL). Normal dosing starts at 0.6 mg subcutaneously (SC) daily but can be increased weekly as needed for glycemic control, to a maximal dose of 1.8 mg daily. There is limited information on clinical effects following overdose. We report the second and largest overdose of liraglutide.

Case Report: A 53-year-old man with DMII was given a liraglutide injection pen and told to administer 0.6 mg SC. Other medications included glyburide, metformin, rosiglitazone, valsartan and ezetimibe/simvastatin. On the first use of liraglutide his wife unintentionally administered the entire contents (18 mg) of the pen SC. Within 30 min he experienced dry mouth and abdominal discomfort. He presented to the emergency department (ED) within one hour; initial finger stick glucose (FSG) was 116 mg/dL. His examination was normal with a benign abdomen. In one hour his repeat FSG was 75 mg/dL; 1 amp of 50% dextrose was administered prior to our involvement. Initial CBC, CMP, and lipase were normal except for an AST of 70 IU/L (normal 10-50). He was admitted for observation with continued nausea, emesis and abdominal pain. Serial FSG ranged from 87 to 205 mg/dL; hypoglycemia did not develop and lipase remained normal. In 18 hours he tolerated clear liquids, diet was advanced and he was discharged home at 24 hours. On home follow-up the day of discharge, he reported nausea and headache but was eating normally without abdominal discomfort.

Case Discussion: In clinical trials liraglutide’s most common adverse effects were nausea, emesis and diarrhea. Pancreatitis occurred more frequently than placebo (2.2 v. 0.6 cases/1000 patient-years). Hypoglycemia was infrequently noted, but the incidence increased in patients on a sulfonylurea. In our patient, an overdose of 18 mg did not result in pancreatitis or hypoglycemia. It is unlikely the dextrose he received masked any hypoglycemia due to liraglutide’s long duration of action (elimination half-life of almost 13 hours). We found one other reported overdose (17.4 mg SC). This patient was also hospitalized with nausea and vomiting, and had no hypoglycemia.

Conclusion: A large SC overdose of liraglutide resulted in nausea, emesis, abdominal pain and headache in a DMII patient also taking a sulfonylurea. Hypoglycemia and pancreatitis did not occur.

113. HIGH-DOSE FLUMAZENIL DRIP FOR REVERSAL OF A MASSIVE IATROGENIC BENZODIAZEPINE OVERDOSE: A CAUSE-EFFECT ANALYSIS OF AN ADVERSE DRUG EFFECT AND DISCUSSION OF THE ROLE OF THE CLINICAL PHARMACIST AND MEDICAL TOXICOLOGIST
Timothy Wiegand, Nicole Acquisto
The University or Rochester Medical Center, Rochester NY USA

Background: Sentinel events typically occur after a series of errors, including errors in the very systems designed to prevent them. Iatrogenic drug overdoses are a particular type of adverse drug events that have potential to cause significant patient harm, depending upon the type of event.

Case Report: A 70-year-old gentleman who had been hospitalized for over 180 days with a very complicated course became unresponsive shortly after treatment began with intravenous lorazepam for refractory nausea. The cause was an iatrogenic benzodiazepine overdose. A flumazenil drip was utilized at doses of 1.0 mg/hour for over 48 hours to prevent respiratory depression and airway compromise.

Case Discussion: A clinician intended to order 1 mg of lorazepam IV every 4 hours as needed for nausea or emesis however the wrong formulation of lorazepam was chosen during electronic order entry and the electronic-overring system interpreted the placed order to mean 60 mg every 4 hours rather than 1 mg every 4 hours. In response to the order, however, the system generated two warnings: 1) daily dose 360 mg -Overdose (Max. 16 mg) and 2) single dose 60 mg -Overdose (Max 4 mg). Both the ordering provider and the pharmacist who verified the order overdose these warnings. Subsequently, the nurse who was on duty apparently noticed the order included the entire bag as one dose and asked the provider if she was supposed to give, “the whole thing”. The provider assented but the 60 mg dose was not included in the discussion. Finally, the nurse reviewing this patient’s medications was using bar-code for med ID but the lorazepam was not captured. This would have also triggered warnings for overdose including a high daily dose and high single dose.

Conclusion: Typical of this degree of adverse event a cascade of errors occurred, including failure of several safety mechanisms inherent in the system. Patient outcome was impacted by Medical Toxicology consultation. Reconstruction of the event by Patient Safety Officers and Clinical Pharmacists was instrumental in understanding how this iatrogenic drug overdose occurred and is crucial in implementing improvements in the system necessary to avoid recurrence.

Medical Toxicologists and Clinical Pharmacists have unique and complimentary expertise and they are effective resources for reacting to these events for the care of the effected patient as well as analyzing these events so that patient outcomes are improved and recurrence is prevented.

114. A 48-MONTH RETROSPECTIVE REVIEW OF PREGABALIN INGESTIONS IN CHILDREN LESS THAN 6 YEARS OLD
Grant D. Lackey1, Judith A. Alsop1, Kathy A. Marquardt1, John F. Inciardi1, Timothy E. Albertson2
1California Poison Control System-Sacramento Division, Sacramento CA USA; 2University of California, Davis, School of Medicine, Sacramento CA USA; 3University of California, San Francisco, School of Pharmacy, San Francisco CA USA; 4California Northstate College of Pharmacy, Rancho Cordova CA USA

Background: Pregabalin is an anticonvulsant drug used for neuropathic pain and as an adjunct therapy for partial seizures with or without secondary generalization in adults. There are few published reports of clinical experience with acute ingestions in children.

Method: A 48 month retrospective study of ingestions of pregabalin in children less than 6 years old was performed. CHR approval was obtained and cases were blinded prior to analysis. Inclusion criteria were pregabalin as a single ingestant in children 6 years and younger followed to a known outcome. Data collected were age, sex, amount ingested, clinical symptoms, and patient outcome.

Results: A total of 80 cases of pregabalin ingestions without coingestants were identified. 39% were male, and 61% were female with a mean age of 2.2 years old (range 8mo – 6yo, SD 1.15 yrs). The mean amount ingested was 106 mg (range 25 mg to 450 mg, SD 91 mg). Of the 80 patients, 4 patients (5%) developed drowsiness, 2 patients (2.5%) developed dizziness, and 1 patient (1.3%) vomited. None of the patients developed any other CNS, or cardiovascular symptoms. 22 patients (27.5%) were treated in the ED. Activated charcoal was administered to 8 (36%) of the 22 patients. All 22 patients (100%) in the ED were discharged without sequelae. Outcome: No effect in 73 patients (91%), minor effects in 7 patients (9%) drowsiness, dizziness, and vomiting.

Conclusion: Pregabalin toxicity at doses up to 450mg in children less than 6 years old manifested primarily as drowsiness and dizziness. Pediatric pregabalin ingestions showed favorable outcomes with minimal supportive care and gastric decontamination with activated charcoal in the ED. Continued evaluation of pediatric ingestions of pregabalin is essential to determine more specific thresholds for toxicity.

115. CHILDHOOD ATAXIA FOLLOWING ACCIDENTAL ASA POISONING
Eric VanDePol, Amy Bishop, Bryan S. Judge
GRMEP/MSU Program in Emergency Medicine, Grand Rapids MI USA

Introduction: Ataxia is a relatively common presenting sign in children and can be caused by both benign and life-threatening conditions. In most cases of acetylsalicylic acid (ASA) overdose, ataxia is associated with low-grade fever, hyperpnea, vomiting without diarrhea, drowsiness, and additional changes in mental function. In this case, the presentation of ataxia as the sole neurological symptom after ASA poisoning was unusual.

Case report: An 18-month-old male presented to the emergency department visibly tremulous and unable to walk or stand. The mother reported that the child was breathing fast, had an associated dry cough, and had 4 prior episodes of vomiting. The child had been playful and active until 4 hours prior to presentation. The perinatal and past medical histories were unremarkable. Physical
556 NACCT Abstracts

examination revealed a well-hydrated child who appeared to be in mild respiratory distress and appeared unable to support himself while seated. Vital signs were: oral temperature, 37.0 °C; HR, 142 beats/min; RR, 60 breaths/min; and BP, 88/46 mmHg. Upon cardiovascular examination, the patient was found to be tachycardic without murmur or rubs. The HEENT, lung, and abdominal examinations were unremarkable. Further neurological examination revealed no additional focal neurologic findings. Lab testing revealed an anion gap metabolic acidosis and respiratory alkalosis with a normal pH and normal lactic acid. A serum salicylate level was 97 mg/dL. When the mother was questioned about aspirin exposure, she recalled her daughter dropping a jar of adult aspirin on the floor seven hours earlier in the area where the patient was playing. The patient was subsequently treated with IV fluids and urinary alkalinization. He was discharged to home on the third hospital day without sequelae.

Discussion: Drug ingestion and toxin exposure should be considered early in the differential diagnosis of acute ataxia in children. Salicylates are neurotoxic. Acidosis worsens this toxicity by increasing the amount of salicylate that crosses the blood brain barrier and increases CNS tissue levels. Signs and symptoms of CNS toxicity are myriad and include nausea, vomiting, tinnitus, hyperpnea, and lethargy. Severe toxicity can progress to disorientation, ataxia, seizures, cerebral edema, hyperthermia, coma, cardiorespiratory depression, and, eventually, death.

Conclusions: ASA is a common medication that can be found in many households. Physicians need to be aware of the dangers that pediatric ASA ingestions present and should have a high index of suspicion as a history of ingestion is often not given. If the onset of ataxia is acute, questions regarding the possible ingestion of toxic substances may be diagnostic and are essential.

116. ELEVATED SERUM IRON CONCENTRATIONS FOLLOWING HAND WARMER INGESTIONS IN 4 DOGS

Ahna G. Brutlag, Catherine M. Adams
SafetyCall International and Pet Poison Helpline, Bloomington MN USA

Background: Instant hand/foot warmers and disposable heating pads are common products that contain reduced iron as the active ingredient. Upon exposure to air, oxidation of the iron produces the desired exothermic reaction. The external packaging often declares such products “non-toxic,” providing neither the salt formulation nor amount of elemental iron. Although reduced iron has minimal oral bioavailability, such products are potential sources of iron poisoning in pets, especially dogs. We present a case series of canine ingestions of non-oxidized hand warmers, all resulting in elevated serum iron concentrations and gastrointestinal (GI) signs.

Case Reports:
Case 1: An 8 month old (mo) Labrador retriever ingested one 11.8 gram (g) warmer. Successful decontamination was performed using emesis and whole bowel irrigation. Serum iron concentrations were 348 ug/dL (normal = 98-220 ug/dL), 303 ug/dL, and 85 ug/dL at 4 hours (h), 9.5 h. and 48 h post-ingestion (PI), respectively.
Case 2: A 2 year old (yo) Yorkshire terrier ingested 1 warmer and presented with vomiting, diarrhea and abdominal pain. Therapies included gastric lavage due to the large amount of metallic radiodense material in the stomach. Serum iron concentrations were 432 ug/dL 10-14 h PI and 350 ug/dL 3 days (d) PI. Chelation with deferoxamine was then performed.
Case 3: A 2 yo miniature pincher ingested up to 3 warmers (36 g total). On presentation the dog was asymptomatic with radiographic evidence of metallic material in the small intestine; a 4-6 h PI serum iron concentration was 378 ug/dL. Chelation with deferoxamine was then performed.
Case 4: A 3 yo Doberman pincher ingested 2 warmers (23.7 g total) and presented with spontaneous vomiting. Emesis was performed due to a large amount of metallic radiodense material in the stomach; a 4-6 h PI serum iron concentration was 332 ug/dL.

All dogs exhibited only GI signs, remained stable during hospitalization and had a favorable outcome.

Conclusion: The ingestion of reduced iron in human beings has been reported to cause mild elevation in serum iron concentrations (remaining within the normal range) with minimal clinical effects. To our knowledge, no such data have been reported in dogs. These cases demonstrate that an elevation of serum iron can occur following the ingestion of reduced iron-containing products. Despite the elevated iron concentrations, all dogs suffered mild to moderate clinical signs with full recovery in 12 h - 4 d.

117. METHANOL EXPOSURE IN A TODDLER WITH 3-METHYL-CROTONYL COA CARBOXYLASE DEFICIENCY

Jon B. Cole, Heather Ellsworth, Benjamin S. Orozco, Deborah L. Anderson, Stacey A. Bangh
Hennepin Regional Poison Center, Minneapolis MN USA

Introduction: Methanol exposure is a common toxicologic problem in children, however methanol exposure in a child with a pre-existing metabolic disorder that causes metabolic acidosis represents a unique challenge. A review of the literature reveals no cases of methanol exposure in children with pre-existing metabolic disorders. We report such a case.

Case Report: A 16 month-old girl with a history of 3-Methylcrotonyl CoA Carboxylase Deficiency (3-MCCD) was brought to the emergency department by her parents 2 hours after she accidentally drank a mouthful of windshield wiper fluid that contained 50% methanol. Her parents witnessed the incident and were certain this was an acute isolated exposure. On arrival the patient had no complaints and was noted to be acting normally by her parents. Her physical exam, including vital signs, was normal. Laboratory values were as follows: methanol 6 mg/dL, sodium 145 mEq/L, chloride 110 mEq/L, bicarbonate 16 mEq/L, (anion gap 19), BUN 16 mg/dL, glucose 110 mg/dL, serum osmolality 298 mOsm (mOsm gap 4) and urine drug screen, including high performance liquid chromatography and mass spectrometry, was negative. Because of the wide anion gap metabolic acidosis she was treated with fomepizole and admitted for observation. The following morning her methanol level was zero, her acidosis had resolved (bicarbonate 22 mEq/L, anion gap 9), and she was discharged home.

Discussion: 3-MCCD is an inherited autosomal recessive metabolic disorder resulting in an inability to correctly metabolize the amino acid Leucine. Patients with 3-MCCD have clinical presentations that range from severe to benign but are susceptible to the development of significant catabolic stress resulting in lethargy, apnea, hypotonia, and seizures. Metabolic disturbances of the disease include hypoglycemia, metabolic acidosis, and ketonuria. Methanol ingestion can also result in a significant metabolic acidosis. It is uncertain if this child’s acidosis was the result of her 3-MCCD, her methanol exposure, or both. It is also possible the time of ingestion was not correct, however there was no reason to suspect this clinically. Given the accepted “non-toxic” level of 20 mg/dL has dubious supporting evidence, it is uncertain if any detectable level of methanol in these children can truly be considered non-toxic.

Conclusion: Children with pre-existing metabolic disorders prone to acidosis who ingest methanol present a clinical dilemma. Clinicians who care for these patients should consider treatment with fomepizole, even when measured methanol concentrations fall in the traditionally non-toxic range.

118. A TALE OF TWO BLUE BABIES

Heather Ellsworth1, Christian P. Lintner1, Annalisa K. Rudser2, Jon B. Cole1
1Hennepin Regional Poison Center, Minneapolis MN USA; 2Children’s Hospitals and Clinics of Minnesota, Minneapolis MN USA

Background: Benzocaine is a local anesthetic found in over the counter (OTC) topical medications. Unintentional exposures to these products are common in children; however, the development of symptoms after such exposures is rare, as is severe methemoglobinemia (MHb). When MHb does occur, treatment with methylene blue (MB) is not usually required. We report 2 cases of accidental ingestions of benzocaine-containing products producing MHb requiring MB.

Case 1: A 22 month-old girl presented to the Emergency Department (ED) with cyanosis. Pulse oximeter (SpO2) read 85-86% despite 100% supplemental oxygen (O2) via a non-rebreather facemask. Vital signs were notable for a heart rate of 182 bpm. She was awake and alert with peri-oral, oral, and peripheral cyanosis but had no Objective findings of respiratory distress. Her blood on venipuncture was dark brown. She had received Nysquil® the previous evening but had no other known medication exposures. She received 1 mg/kg of MB via an intravenous line with resolution of her cyanosis and improvement in her
Sinrolimus is a lipid-soluble compound with a V<sub>d</sub> of 4-20L. It was being used to prevent rejection in certain organ transplant patients by binding FK-binding protein 12. This complex inhibits mTOR to regulate cell growth and proliferation. It also inhibits IL2, IL4, and IL15 dependant proliferation of T and B cells and arrests the G1-S phase of the cell cycle.

**Discussion:** Despite wide availability of a significant number of OTC medications containing benzocaine, unintentional pediatric exposures necessitating MB are rare as the anesthetic effects of benzocaine usually limit the amount ingested. Most case reports link the development of MB to the use of benzocaine in oral and endoscopic procedures. These 2 cases highlight the potential for MB to occur in unintentional pediatric ingestions and the importance of history, clinical examination, and physical assessment in making the diagnosis.

**Conclusion:** Methemoglobinemia requiring treatment with methylene blue may occur after unintentional pediatric ingestions of the counter products containing benzocaine.

**119. SIROLIMUS TOXICITY IN A RENAL TRANSPLANT PATIENT**

**Laura W. White<sup>1</sup>, Jean L. Cleary<sup>1</sup>, Wayne R. Snodgrass<sup>1</sup>, Jon D. Thompson<sup>1</sup>**

<sup>1</sup>Southeast Texas Poison Control Center at University of Texas Medical Branch, Galveston TX USA; <sup>2</sup>University of Texas-Austin, Austin TX USA

**Background:** Sirolimus is a macrocyclic lactone produced by streptomyces hygroscopicus used to prevent rejection in certain organ transplant patients by binding FK-binding protein 12. This complex inhibits mTOR to regulate cell growth and proliferation. It also inhibits IL2, IL4, and IL15 dependant proliferation of T and B cells and arrests the G1-S phase of the cell cycle.

**Case Report:** A 58 year old female of unknown race, post kidney transplant arrived eight days prior to the initial contact with the poison control center. It was not known if previous sirolimus levels were done. On day 2, the serum level was 22ng/ml with a goal of 4ng/ml. The sirolimus was discontinued as the patient’s chief complaint was hypoxia and renal failure. She then developed thrombocytopenia and anemia with continued deterioration despite declining sirolimus levels. The patient expired on hospital day sixteen.

**Case Discussion:** Sirolimus is a lipid soluble compound with a V<sub>d</sub> of 4-20L/kg. It is metabolized primarily by CYP3A4 and transported by alpha-glycoproteins. It is a highly protein bound at 92%, with 97% of this being albumin, and to lesser extents alpha-1 acid glycoprotein and other lipoproteins. Sirolimus contributes about 90% of the immunosuppressant activity. It is unknown how long the patient exhibited elevated sirolimus levels, and during the course of her hospital stay, she did have mildly elevated transaminases. She also exhibited the expected toxic symptoms of acute renal failure, thrombocytopenia, anemia and respiratory distress. It was not known if the patient was also receiving other medications (ie: cyclosporine), or what formulation of sirolimus was prescribed. Her dose was prescribed as 6mg/d. Possible explanations include hyperalumeminemia, CYP3A4 polymorphism, unabsorbed drug due to minimal bowel sounds, enterohepatic recirculation of metabolites, unknown drug interactions, and errors in patient compliance.

**Conclusion:** Careful monitoring of clinical toxicity and sirolimus levels is needed in post-transplant patients. Patient education is essential for the well being and compliance of the patient. Early contact with the poison center may have been beneficial.

**120. SYNDROME OF INAPPROPRIATE ANTI DIURETIC HORMONE COMPPLICATING PEDIATRIC INGESTION OF FLUOXETINE**

**Evan S. Schwartz<sup>1</sup>, Stacey L. Hall<sup>1</sup>, Meria Aulds<sup>2</sup>**

<sup>1</sup>University of Texas Southwestern, Dallas TX USA; <sup>2</sup>Meria Aulds MD, PA, TX USA

The syndrome of inappropriate antidiuretic hormone (SIADH) is a known complication with therapeutic usage of serotonin reuptake inhibitors (SSRI); it generally occurs in older patients. We report the youngest patient known to have developed SIADH after an accidental ingestion of fluoxetine.

A 3 years old (y/o) male with a history of febrile seizures and attention deficit disorder on clonidine and methylphenidate presented to the emergency department (ED) after ingesting up to 12 tabs of fluoxetine 20mg (12.6 mg/kg) 30 minutes prior to arrival. His exam and vital signs were normal aside from a tachycardia of 109 beats per minute (BPM); he was discharged 30 minutes later at 19:35. He returned 3 hours later; he was hallucinating and diaphoretic. On exam, he had mydriasis, aphasis, normal reflexes, and left sided rigidity with the following vital signs: pulse 97 bpm, blood pressure (BP) 117/80 mmHg, respirations 16/minute, and temperature of 97 degrees. Laboratories were unremarkable including a normal basic metabolic panel (BMP), fluids were initiated, and he was admitted. He became progressively more tachycardic to 140 bpm, hypertensive with BP of 144/96, and had emesis treated with a 1.25 mg promethazine suppository; he was not hypothermic. He developed tremors and multiple seizures (SZ). The next morning, a repeat BMP was obtained with sodium of 122 mEq/L. Oxygen was administered and the patient was transferred to a regional hospital.

At the regional hospital, he was given diazepam 5 mg IV for SZs; his SZ activity and vitals improved. A head CT was unremarkable. He again developed hemodynamic instability and SZs. Fosphenytoin 20 mg/kg and 55 ml of 3% hypertonic saline were administered; his fluids were changed from D5 ½ NS to D5NS. He was intubated and transferred to a facility with a pediatric intensive care unit (PICU) later that afternoon.

A repeat CT in the PICU showed cerebral edema. He remained intubated and on fluid restriction for the next 48 hours. A brain MRI was normal. His vital signs and sodium level improved; seizure activity stopped, and he was extubated. A serum fluoroxetine level obtained at the initial hospital was 1,465 ng/ml. SIADH from SSRIs most frequently occurs within 2 weeks of drug initiation; it has been documented to occur from 3 days to 4 months after initiation of therapy. Women over 70 years of age are most at risk. A review of fluoxetine ingestions in patients less than 6 y/o did not report any cases of SIADH. Patients less than 6 y/o that ingested an SSRI did not develop SIADH in a different review.

Children that become symptomatic after ingesting fluoxetine should be evaluated for the development of SIADH induced hyponatremia.

**121. LEAD PELLET EMBEDDED IN THE C2 VERTEBRA OF A CHILD: A CLINICAL DILEMMA**

**Kelsey L. Moser, Jon B. Cole, Stacey A. Bangh, Heather Ellsworth, Samuel J. Stellpflug**

Hennepin Regional Poison Center, Minneapolis MN USA

**Background:** Lead continues to be a health hazard in the United States, especially in children, where it may lead to behavioral changes and developmental delay. Most exposures can be traced to the ingestion of a lead-containing substance, but cases of toxic lead levels developing in patients with retained bullets exist. There is no standard for lead foreign body removal, but it is generally accepted lead objects should be removed from synovial fluid and cerebrospinal fluid (CSF). We present the case of a child shot in the neck with an air pellet gun resulting in a lead pellet lodging in his cervical vertebrae, which represented a clinical dilemma.

**Case Report:** A 7 year-old boy presented after being shot in the neck with a lead pellet. Lead content was confirmed with the manufacturer. Surgical exploration revealed the pellet had penetrated the oropharynx and was firmly embedded in the C2 vertebrae at the base of the odontoid process. Extraction required a neurosurgeon and one was not immediately available. The serum lead concentration 24 hours post-exposure was 6 mcg/dL. A CT scan confirmed the pellet’s location to be in the bone of C2 and not the intervertebral disc. As there were significant risks associated with surgical removal of the pellet, the medical toxicologists and neurosurgeons decided to leave it in place. Twenty days post-exposure a repeat CT scan was unchanged. Ten months post-exposure the child had no signs or symptoms of lead toxicity; no more lead testing had been done due to non-compliance of the child’s mother.

**Discussion:** Lead is common to firearm ammunition because it is cheap, malleable, and delivers a high amount of kinetic energy per volume. Toxic
lead levels from retained bullets have only rarely been reported. Bullets that remain lodged in muscle or fat will typically become encapsulated in fibrous tissue, preventing systemic absorption. The literature lends more credence to concern for lead foreign bodies located in spaces such as the intervertebral discs and CSF. The case was unique because there was evidence of the pellet being extruded by bone, but given the lead concentration 24 hours post-exposure there was a definite possibility of exposure to either a disc or CSF. The decision to leave the pellet in place was difficult and involved weighing both the toxicologic and surgical recommendations. Lead toxicity could still develop in this patient, and education and continued monitoring will be important.

**Conclusion:** The decision of whether or not to removed a lead foreign body in or around the vertebral column is a difficult one; toxicologic and surgical risks and benefits need to be weighed against one another. If not removed, there is a possibility for increased lead levels acutely and chronically.

### 122. ACUTE MENTAL STATUS CHANGES AFTER INGESTING LEVAMISOLE

**Evans S. Schwartz, Kurt C. Kleinenschmidt, Melody Gardner**

*University of Texas Southwestern, Dallas USA*

Levamisole is an anthelminthic used by veterinarians. It is used by physicians to treat immune related diseases and cancer. Recently, it was found in illegally imported cocaine. We report the first case of levamisole use associated with acute mental status changes.

A 55 year old male with peptic ulcer disease presented after accidental ingestion of a goat de-wormer that contained levamisole. It was kept in a water bottle. One hour after ingestion, he felt dizzy, had diaphoria, and was diaphoretic. In the Emergency Department (ED), he was anxious and agitated. He denied suicidal ideation or depression. Vital signs (VS) were abnormal: blood pressure (BP)160/99 mmHg, pulse (P) 121 beats per minute (BPM), and respirations @ 24/minute. He was oriented with midsize, reactive pupils, and clear breath sounds. He had increased salivation and emesis but no diaphoresis, lacrimation, or diarrhea. His agitation increased, and he required diazepam 10 mg IV for sedation. His head CT was normal; all laboratory tests were unremarkable including a normal complete blood count (CBC), basic metabolic panel, alcohol level, and urine drug screen (UDS).

His mental status improved after the diazepam; he did not require any further sedation. The next morning he was alert, oriented, and cooperative. VS were improved: BP 107/62 mmHg, P 90 bpm, and R 11. The rest of his exam and a repeat CBC were normal. He was discharged without any further sequelae. Urinary levamisole levels were not obtained.

Demyelinating leukoencephalopathy after treatment with levamisole and 5-fluorouracil (5-FU) is well reported in the literature. Mental status changes develop between 2 and 16 weeks after drug initiation and are associated with abnormal MRIs. Most patients received multiple treatments; not all received 5-FU. A case report described a 3.5 year old female who developed ataxia, behavior changes, and paresthesias after 3 days of treatment. In one case series, 2/16 patients’ neurologic abnormalities began after 1 day of therapy. There are no other reports regarding neurologic developing this quickly that were not associated with illicit drug use.

Other possible etiologies for his presentation include drug use, co-ingen- tants, and psychiatric disease. However he did not have a history of sub- stance abuse or prior psychiatric disease. His history seems dependable as he was not suicidal and it was verified by his wife. While a confirmatory urine test was not obtained, we believe the history that we obtained was accurate.

Levamisole is associated with subacute development of demyelinating leuko-encephalopathy. We report mental status changes developing within hours of ingestion of levamisole that resolved by the next day.

### 123. DIGOXIN TOXICITY AND RENAL FAILURE TREATED WITH DIGOXIN ANTIBODIES AND PLASMAPHERESIS

**Edward Bottei**

*Iowa Statewide Poison Control Center, Sioux City IA USA*

**Background:** Digoxin-digoxin antibody complexes are renally eliminated. In patients with renal failure, dissociation of the digoxin-digoxin antibody complex has occurred and led to recurrent digoxin toxicity. Four case reports describe the use of plasmapheresis or plasma exchange in conjunction with digoxin antibodies to treat patients with both digoxin toxicity and renal failure.

**Case Report** A 70 year old man with a history of atrial fibrillation and placement of a permanent pacemaker, presented to the ED with a paced heart rhythm in the 50’s, a BP of 80/50 mm Hg, and mental status changes. Initial labs included a digoxin level of 5.92 ng/mL, potassium of 6.7 mEq/L and creatinine of 5.7 mg/dL. He was diagnosed with digoxin toxicity secondary to acute renal failure. He was given 240 mg of digoxin antibodies and was started on continuous veno-venous hemofiltration (CVVH). Overnight, dopa- amine was started for worsening hypotension. By the morning of hospital day 2, the patient’s heart rate was in the 110’s and potassium was down to 4.5 mEq/L. Also on hospital day 2, approximately 19 hours after administration of digoxin antibodies, plasmapheresis was started. Post-plasmapheresis digoxin level, obtained on hospital day 3, was 3.01 ng/mL. No further digoxin levels were obtained. On hospital day 3, another 120 mg of digoxin antibodies was given, CVVH and dopamine were discontinued, and his mental status had improved considerably. By hospital day 4, the patient was in atrial fibrillation with a heart rate of 85 and creatinine was 1.4 mg/dL. Later on hospital day 4, the patient underwent a second round of plasmapheresis. The patient was discharged home on hospital 5.

**Discussion** Since only two serum digoxin levels were obtained and the effluent from the plasmapheresis was not tested for digoxin concentration, it is difficult to determine exactly how much digoxin-digoxin antibody complex was removed by plasmapheresis. A previous study has shown that <1% of the total digoxin dose was removed by plasmapheresis. The same study also found that plasmapheresis 2.5 hours after the administration of digoxin antibody removed more digoxin than if the plasmapheresis was delayed until 26 hours post-antibody administration.

**Conclusion** In patients with renal failure, the use of plasmapheresis may help prevent dissociation of digoxin-digoxin antibody complex, which could lead to recurrent digoxin toxicity.

### 124. THE ATYPICALS: UNEXPECTED OUTCOMES IN CHILDREN

**Susan Smolinske, Ron Thomas, Jimmie Leleszi, Lydia Baltarowich, Victoria Tutag Lehr**

**Background:** Atypical antipsychotic prescriptions for pediatrics increased 10-fold since 2007. NPDS single substance atypical exposure in patients less than 19 years of age increased 8% from 2007–2009, while overall exposures in this age group increased 0.4%. Frequency and type of adverse effects associated with pediatric use in therapeutic and overdose scenarios is not well known.

**Objective:** To estimate the frequency and type of atypical antipsychotic exposure in children and associated adverse effect, dose-relationship, and outcomes.

**Design/Methods:** Retrospective analysis of patient exposures reported to a regional poison center. Inclusion criteria were age 2 to <19 years (yrs) as of January 1, 2010, single agent exposure to an atypical antipsychotic, medical outcome of minor, moderate, and date of exposure 2010. Cases not followed or reported no effects from exposure were excluded. Descriptive analyses were conducted using Chi-Square Fisher’s Exact and student’s-t tests.

**Results:** A total of 135 cases met criteria. The sample had mean age 10.8 ± 6.6 yrs; 0.8-18 yrs) with 51% female. Pre-morbid conditions were common, including depression 22%, bipolar 18%, ADHD 9%, substance abuse 5%, and previous suicide 3%. Atypical antipsychotic agents were: quetiapine 43%, aripiprazole 20%, risperidone 13%, olanzapine 11%, clozapine 1%, iloperidone 1%. Intent was self-harm (43.7%), accidental poisoning (14%), suicidal (25%), and ADR (3%). Children with self-harm were older than those with accidental poisoning (16 ± 1.4 vs 2.8 ± 2.0 years; p < .001). Mechanical ventilation most frequently associated with quetiapine (10%) and ziprasidone (14%). ICU admission was more common with quetiapine (27%) and aripiprazole (26%). Prolonged QTc (defined as >450 msec) occurred in 22 cases. Overall, outcomes were 52% mild, 41% moderate and 5% major (2 ziprasidone and 5 quetiapine). Moderate outcomes were reported in 8 children with history of less than AAPCC triage dose exposure.
Case Discussion: TORSADES DE POINTES ASSOCIATED WITH CESIUM

126. TORSADES DE POINTES ASSOCIATED WITH CESIUM

Conclusions: Frequency of poisoning exposures to atypical antipsychotics in children reported to a regional poison center reflected clinical use of these agents according to marketing data. Quetiapine represented the most exposures, highest severity of outcomes, and need for ICU admission. AAPCC published triage data predicted major outcomes, but was not sensitive enough to exclude moderate outcomes. NPDS reported exposures to antipsychotics and self-harm for this age are increasing. Dose related toxicity data for pediatrics may require a prospective study with a larger sample.

Jeffrey Suchard, Michael Habicht
UC Irvine Medical Center, Orange CA USA

Background: Hydroxocobalamin (OHCbl) may interfere with several colorimetric laboratory tests due to the drug’s intense red hue. OHCbl may also rarely interfere in patient management, as when hemodialysis machines misinterpret serum discoloration as hemolysis. We report a case where the management of a trauma and burn patient treated with OHCbl for presumed cyanide poisoning was affected by interference with reporting coagulation study results.

Case Report: A 20-year-old man was the restrained driver in a high-speed car vs. tree accident that killed a passenger. Extrication was complicated by a vehicle fire, including the driver’s seat. The patient arrived with GCS 5, BP 112/62mmHg, head injuries, second-degree chest burns, and full-thickness burns to both legs which had no distal pulses. The melted plastic car seat and floor mats had fused with his buttocks, legs, and feet. The patient was intubated and underwent bilateral leg escharotomy and fasciotomies. Initial labs showed arterial pH 7.14 and serum lactate > 11.1 mmol/L (> upper reporting limit). The patient was given 5g IV OHCbl for potential cyanide poisoning. Head CT scan showed a frontal epidural hematoma and cerebral contusions. The initial PT/INR and PTT (drawn after OHCbl given) were reported only as “hemolyzed”. Neurosurgery delayed insertion of an external ventricular drain pending interpretable coagulation test results. Repeat labs were again reported only as “hemolyzed”. A medical toxicologist requested lab personnel perform the analyses despite serum discoloration from OHCbl; these had not been performed due to presumed hemolysis by visual inspection only. Test Results were all only slightly elevated: PT 13.1 sec, INR 1.35, PTT 38.9 sec. A blood cyanide level drawn 5 hours after arrival was normal (<5 mcg/dL).

Case Discussion: Hemolysis affects many common laboratory assays, including coagulation tests. Hemolysis will raise the PT and lower the PTT, and is a common cause of sample rejection. A “pseudo-hemolysis” occurs with OHCbl administration, which resulted in the lab declining to perform an urgently needed test.

Conclusions: “Pseudo-hemolysis” from the administration of OHCbl as an antidote for cyanide toxicity can interfere with the timely performance and reporting of coagulation test results.

125. HYDROXOCOBALAMIN INTERFERENCE WITH COAGULATION STUDIES IN A TRAUMA AND BURN VICTIM

Jeffrey Suchard, Michael Habicht
UC Irvine Medical Center, Orange CA USA

Background: Hydroxocobalamin (OHCB) may interfere with several coagulation test results. OHCB may also rarely interfere in patient management, as when hemodialysis machines misinterpret serum discoloration as hemolysis. We report a case where the management of a trauma and burn patient treated with OHCB for presumed cyanide poisoning was affected by interference with reporting coagulation study results.

Case Report: A 53-year-old female with a history of Stage IV ovarian carcinoma presented to the emergency department with repeated episodes of syncope. At least one of these episodes was associated with a seizure. She had been treated with surgery, chemotherapy and radiation therapy, which she discontinued in favor of alternative therapies. More recently the patient had been using a variety of alternative therapies, including cesium carbonate. The patient purchased cesium carbonate via the Internet that she mixed 30 grams in 1 quart of tap water. She ingested 1 gram TID for approximately 2 weeks, eventually increasing her dose to approximately 5 grams per day. The patient reported diarrhea and paresthesias prior to the syncopal episodes. She had no known personal or family history of long QT syndrome or sudden death and was taking no medications known to cause QT prolongation. Initial vital signs: HR 77, BP 85/42, RR 16, T 37°C, room air O2 saturation 98%. She was alert and oriented with a nonfocal neurological exam. EKG showed sinus rhythm, a left bundle branch block and a corrected QT interval (QTc) of 722 milliseconds. Her electrolytes, including magnesium, were normal except for potassium of 3.1 mmol/L; serum creatinine was 1.4 mg/dL. Approximately 6 hours after admission the patient suffered three episodes of TdP. She was treated with intubation, defibrillation, potassium, magnesium, isoproterenol and immediately started on PB after which she had no more episodes of TdP her QTc narrowed. Initial blood and urine cesium concentration were 210,000 and 180,000 micrograms/L, respectively. Blood cesium levels trended downward, while urine cesium levels initially increased by 50% before trending downward.

Discussion: Treatment with cesium carbonate resulted in markedly elevated blood and urine cesium concentrations associated with QT prolongation and TdP. Prussian blue and standard treatment for TdP appeared to be successful in stabilizing the patient.

Conclusion: Internet sale of cesium carbonate as an alternative cancer therapy is unregulated and can be associated with life threatening QTc prolongation and TdP. PB appears to be a helpful in the treatment of cesium-induced TdP.

127. DELAYED ONSET OF TACROLIMUS-ASSOCIATED REVERSIBLE MULTIFOCAL LEUKOENCEPHALOPATHY (RML)

Patrick M. Whiteley
1 David P. Randall2, Jerrold B. Leikin2
1Aurora CO USA; 2NorthShore University Health System, Glenview IL USA

Background: Tacrolimus is an immunosuppressive macrolide isolated from Streptomyces tsukubaensis that works primarily by inhibiting T-cell activation and proliferation. RML is a rare complication of cyclosporine A or tacrolimus therapy. If the diagnosis is made promptly and the immunosuppressive agent exposure is reduced, this syndrome is rapidly and completely reversible. Previously reported cases of tacrolimus-associated RML show a median time-of-onset of 9 days after initiation of the drug.

Case Report: A 62 year-old female with a history of liver transplant 22 years prior reported to an emergency department (ED) with fever, myalgias, and altered mental status (AMS). She had been diagnosed with a URI 2 weeks prior by her primary physician, but had no abatement of her symptoms. She increased her dose of acetaminophen/hydrocodone from 0-2 tabs/day to 6-8 tabs/day to treat these myalgias. Upon arrival to the ED she was found to be markedly somnolent with inappropriate answers to questioning. Per husband, the patient had progressive AMS over the previous 2-3 weeks. There was no response to narcan, and negative acetaminophen and ethanol level were noted. An MRI of the brain showed diffuse edema consistent with multifocal leukoencephalopathy. The tacrolimus level was 9.2 ng/mL (2-10 ng/mL) in the ED, a marked increase from multiple previous outpatient levels of 2-4 ng/mL. Viral and bacterial CSF cultures were negative. Tacrolimus was discontinued and the patient’s AMS improved remarkably by day four. Follow-up MRI 1 month later showed marked improvement confirming the diagnosis of RML.

Discussion: This case demonstrates a significantly prolonged delay in onset of tacrolimus-related RML. The metabolism of tacrolimus is thought to involve the CYP3A4 enzyme, and serum concentrations of the drug are known to change in the presence of other agents that utilize this pathway. This patient’s therapeutic range had been tightly controlled in past between 2 and 4 ng/mL. The increased use of hydrocodone in this case may have been a contributing factor to an increased serum concentration since its metabolism is known to involve enzyme CYP3A4. This case highlights the

Copyright © Informa Healthcare USA, Inc. 2011
Potential of a significant delay (>15 years) in the presentation of RML and may demonstrate a drug-drug interaction with hydrocodone.

**Conclusion:** This case highlights the potential for a significant delay in the onset of tacrolimus-associated RML.

### 128. SEVERE ALKALOSIS AND DYSPNEA COMPLICATING THE MILK-ALKALI SYNDROME: A CASE OF BAKING SODA INGESTION IN A PATIENT WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND GASTROINTESTINAL BLEED

**Soumya L. Pandali**, Nahar Alruwali, Timara Shaw-Lutchman, Brent W. Morgan

1Emory University International Postdoctoral Medical Toxicology Fellowship, Atlanta GA USA; 2CDC/Emory University Medical Toxicology Fellowship, Atlanta GA USA; 3Emory University, Atlanta GA USA

**Background:** The milk-alkali syndrome (MAS) is characterized by hypercalcemia, renal failure and metabolic alkalosis. Physiologic compensation for the metabolic alkalosis is impaired in patients with chronic obstructive airway disease (COPD). We present a case of MAS from antacid overdose complicated by COPD and a gastrointestinal bleed (GIB) with one of the highest reported serum pHs in the literature.

**Case Report:** A 57 year old male presented with a history of dizziness and dark stools for three days. He complained of abdominal discomfort and worsening dyspnea. His past medical history included COPD and mild dyspnea at baseline, GIB, and alcoholism. His medications were ranitidine and his significant laboratory test results included: sodium, 136 mmol/L; potassium, 1.9 mEq/L; chloride, 71 mmol/L; bicarbonate, “too high to report”; ionized calcium, 0.5 mmol/L (1.13-1.33 mmol/L); BUN, 24 mg/dL; and creatinine, 3.3 mg/dL. An arterial blood gas (ABG) on room air revealed a pH of 7.72; pCO2 of 46 mmHg; pO2 of 37 mmHg; and bicarbonate of 60.9 mmol/L. His hemoglobin concentration was 10.2 g/dL. The patient was admitted to the intensive care unit and aggressively resuscitated with intravenous fluids. The medical toxicology service was consulted. An ABG 18 hours after presentation showed a pH of 7.56, pCO2 of 57 mmHg, pO2 of 50 mmHg, and bicarbonate of 51 mmol/L on 3 liters of oxygen. The patient clinically improved but left the hospital against medical advice on hospital day number 2.

**Case Discussion:** Patients presenting with acid/base disorders resulting in alkalosis rely on compensatory mechanisms to maintain physiologic homeostasis. Healthcare providers should be aware that respiratory complaints in patients with these conditions may be more severe due to underlying electrolyte abnormalities rather than their underlying lung disease.

**Conclusions:** Chronic ingestion of antacids alone is a risk factor for metabolic alkalosis and MAS. Individuals with co-morbid conditions that result in chronic hypercarbia, contraction alkalosis, or worsening renal function are at increased risk for severe alkalemia and respiratory decompensation.

### 129. HYPERKALEMIA DOES NOT PREDICT OUTCOME FROM DIGOXIN EXCESS IN PATIENTS WITH RENAL DYSFUNCTION

**Charles McKay, Kathy Hart, on behalf of the ACMT ToxIC Case Registry Investigators**

Hartford Hospital, Hartford CT USA

**Background:** The 1973 Clinical Toxicology study “Hyperkalemia in Acute Digitalis Poisoning: Prognostic Significance and Therapeutic Implications” is often quoted to emphasize the importance of assessing hyperkalemia in patients presenting with digoxin toxicity. Bisnath et al reported a 50% mortality rate for patients with potassium between 5 - 5.5 mEq/L and 100% mortality for those with potassium > 5.5 mEq/L. However, this study included a number of young patients (age ranged from 15 - 94 years) with acute digitalis ingestions (10 of 91 “overdose occurred in the course of therapy”) and normal renal function. This differs from the elderly patient on chronic digoxin therapy now more commonly seen. An assessment of digoxin toxic patients seen by medical toxicologists in hospitals throughout the country over a 13 month period (as reported to the ACMT ToxIC registry) indicates that 71% of the 42 digoxin patients were over the age of 65 years.

**Methods:** We performed a single center IRB-approved retrospective chart review of all hospitalized patients with an elevated digoxin serum concentration (> 1.2 ng/mL) and evaluated the correlation between plasma potassium, digoxin concentration and mortality.

**Results:** We identified 70 patients (74% women) in a 2 year period with complete data. The mean age was 81 years (SD 9; range 58-96 years). The mean plasma creatinine was 1.8 mg/dL (SD 0.8) and the calculated GFR (Glomerular Filtration Rate) ranged from 9-81 ml/min. The mean plasma potassium was 4.7 mEq/L (SD 1.1) with a range of 2.6-8.1 mEq/L. The digoxin concentration averaged 2.7 mg/mL (SD 1) and did not correlate with either the potassium or calculated GFR. There were 8 patients who died during their hospital stay. The mortality rates for patients with presenting potassium < 5 mEq/L between 5.0 - 5.5 mEq/L and > 5.5 mEq/L were 9%, 0%, and 31% respectively. Chart review identified digoxin toxicity as a likely primary and proximate cause of death in only 1 patient who presented in Vfib arrest with a potassium of 6.5 mEq/L. Six of the remaining seven deaths occurred in the setting of palliative care and were attributable to underlying chronic disease states. Seventeen patients were treated with digoxin Fab fragments; 6 of these patients had potassium > 5 mEq/L.

**Conclusions:** Hyperkalemia is not a reliable predictor of digoxin toxicity in elderly patients with multiple co-morbidities, nor is it predictive of mortality or the need for therapy with digoxin Fab fragments.

### 130. GOING WITHOUT FORESKIN: BUPIVACAINE TOXICITY FOLLOWING LOCAL ANESTHESIA

**Loren K. French, Anna Cedar, Robert G. Hendrickson**

Oregon Health and Sciences University, Portland OR USA

**Background:** Roughly 50% of US males are circumcised – the majority as neonates. While the most common complication is poor cosmesis, major outcomes such as local anesthetic toxicity can occur. Systemic toxicity from local anesthetics manifests as CNS depression, seizures, and cardiovascular compromise. We describe a case of local anesthetic toxicity in a neonate undergoing circumcision.

**Case Report:** A 2715gm, 21 day-old male presented to his pediatrician’s office for elective circumcision. He had a history of intrauterine growth restriction due to twin gestation and was born at 37-2/7 weeks gestation. A dorsal, penile block was the planned method of anesthesia. Bupivacaine was chosen to prolong the anesthetic effect. He received two, 1 mL doses of 0.5% bupivacaine, spaced apart by 2-3 minutes. The estimated total dose was 3.7 mg/kg. Within minutes of the injections, he became lethargic. An ambulance was summoned and he was delivered to the nearest hospital. On arrival to the emergency department he was afebrile, with a BP 90/60 mmHg, HR 178 bpm, and RR 46. The infant was noted to have disconjugate gaze, intermittent extensoria, and altered consciousness with hypotonia. Serum bupivacaine concentration, obtained 2.5 hours after injection, was 0.76 mcg/mL. He was admitted to the PICU and observed overnight. Five hours after the bupivacaine exposure, he would respond to painful stimuli, but was excessively somnolent and not latching on to breast feed. He did not experience seizures or cardiovascular instability, nor did he require specific antidotes. At 22 hours he was awake and back to baseline. Lipid emulsion therapy was discussed and available in the event of cardiovascular collapse.

**Case Discussion:** Bupivacaine is not currently recommended for use in children by its manufacturers. Due to immature hepatic enzymes and decreased levels of α1-acid glycoprotein, bupivacaine has both a longer half-life and a higher percentage of unbound drug in the neonate compared to adults. This case exemplifies the principle that relatively small volumes of bupivacaine may result in symptoms consistent with local anesthetic toxicity. Lidocaine offers a wider margin of safety and may be the preferred local anesthetic of choice for local anesthesia. In this case, the same volume of 0.5% lidocaine would have been under the maximum recommended dose of 5mg/kg.

**Conclusion:** We describe a case of local anesthetic toxicity associated with bupivacaine injections for a circumcision in a neonate. Practitioners who routinely perform this procedure should be aware of the relatively low doses of bupivacaine needed to produce toxicity and the absence of a recommendation for pediatric use from the manufacture.
131. UNINTENTIONAL CLONIDINE EXPOSURES IN CHILDREN: ESTABLISHING EVIDENCE BASED TRIAGE GUIDELINES

Soumya L. Pandala1, Barrak M. Aldosary2, Stephanie L. Hon2, Alexander Garrard1, Robert J. Geller3
1CDC/Emory University Medical Toxicology Fellowship, Atlanta GA USA; 2Georgia Poison Center, Atlanta GA USA; 3Florida USVI Poison Information Center, Jacksonville FL USA; 4Emory University International Postdoctoral Medical Toxicology Fellowship, Atlanta GA USA; 5Emory University, Atlanta GA USA

Background: Ingestion of small amounts of clonidine has reportedly resulted in severe toxicity in pediatric patients. No evidence-based triage guidelines currently exist that considers the amount ingested per patient weight in determining who is at risk for clonidine toxicity. This lack of standardized guidelines may result in management recommendations based on anecdotal experience and suboptimal outcomes. The objective of this study is to describe adverse drug events (ADEs) related to isolated clonidine exposures reported to three poison centers in children less than 12 years of age.

Methods: In this IRB-approved study, charts were abstracted from each center’s database from January 2000 until December 2009. The search strategy for case identification of clonidine exposure calls included the codes “clonidine” and, “age less than 12 years” and “overdose.” Only calls regarding acute, isolated ingestions of clonidine were included in the analysis. Data collected included gender, age, weight, quantity and/or amount of clonidine ingested. If the patient’s weight was not reported, then weight was estimated by using the 50th percentile in growth charts published by the Centers for Disease Control and Prevention and the World Health Organization. Calculations were made based on the total amount ingested (in micrograms) per patient weight (in kilograms). Characterization of adverse events related to clonidine ingestion were recorded as minor, moderate, or severe as per American Association of Poison Control Centers (AAPCC).

Results: A total of 1854 calls met the search criteria with our study sample representing 43.8% females and 56.2% males. The mean age was 3 years (range, 6 months to 12 years). Exposure amounts ranged between 9 mcg and 30,000 mcg and weights ranged between 7.3 kg and 58.6 kg. Calculated dosages that resulted in no side effects ranged between 1.1 mcg/kg and 591.6 mcg/kg. The lowest estimated dosage that resulted in moderate adverse events could not be reasonably established and results were inconclusive. Major, moderate, minor and no adverse events were reported in approximately 2.3%, 21.9%, 31%, and 28% from this study sample, respectively, and 17.2% were unable to be followed.

Conclusions: In this study population, a dosage in micrograms per kilogram that could be used as a triage guideline could not be safely established. Several limitations to this study include utilization of poison center data including recording errors. Final data analysis is pending and more work is needed to further develop these guidelines.

132. ATYPICAL BOTULISM PRESENTATION FOLLOWING BOTULINUM TOXIN WITH PHENOL THERAPY

Kimberly Hovseth1, Randal Badillo1, Scott Schaeffer1, William Banner1, Ed Wright2
1University of Oklahoma College of Pharmacy Oklahoma Poison Control Center, Oklahoma City OK USA; 2The Children’s Center, Bethany OK USA

Background: Onabotulinum toxin type A is approved by the FDA for use in cervical dystonia, blepharospasm, strabismus, and severe primary axillary hyperhidrosis. A common off-label use is in the treatment of spasticity. In 2009, the FDA issued a black box warning that systemic symptoms of botulinum toxicity might occur with therapeutic use of botulinum toxin. Phenol may be used as an adjunct to promote maximum therapeutic effect while minimizing toxin dose. Kolaski et al noted more frequent adverse events with combination therapy vs. botulinum toxin alone.

Case Report: A 3 year old boy treated with botulinum toxin and phenol injections for leukodystrophy developed bulbar weakness and respiratory problems 9 days post-exposure. He received 200 U of toxin (18 U/kg) total; 50 U into right rectus femoris and 75 U into bilateral gastrocnemius as well as 6 cc total of 6% phenol injections into bilateral hamstrings and obturator nerves. Six hours post-injection, right-sided thigh swelling developed. He was diagnosed with lymphedema, which resolved with application of compression wraps. He presented with somnolence, dehydration and hypoglycemia 7 days post-injection. Posis and dysphagia were present but confounded by small reactive pupils, tearing, sialorrhea, typical gut function, hypertonia/clonus and relatively preserved extremity strength. By day 9 the patient displayed generalized weakness, was intubated and received 1.9 cc of IV Neumastat Botulinum Antitoxin twice over 8 hours. Extremity strength improved the next day. Multiple attempts at extubation failed as extremity and bulbar hypotonia reemerged 12-72 hours post-extubation. Pyridostigmine was given with no benefit. Though bowel and bladder function remained intact, a tracheostomy and gastrostomy were performed. On day 90 he was decannulated; at 6 months post-injection he had gained some strength but not fully recovered.

Discussion: An inflammatory response to phenol, resulting in increased blood flow, may have caused greater absorption and systemic distribution of the botulinum toxin; the compression wraps may have contributed to the distribution via local perfusion changes. The failure of the antitoxin to work may be due to delay in administration or other unknown factors. The early apparent improvement with antitoxin and repeated improvement with mechanical ventilation may have been on the basis of neuronal accumulation of neurotransmitters with decreased muscle work.

Conclusion: Even at recommended doses onabotulinum may cause systemic botulism. Patients developing botulism from the administration of onabotulinum toxin A may not present in typical fashion. Early recognition of impending systemic toxicity is crucial.

133. TEN YEARS OF H2O2 INGESTIONS: ANY MAGIC IN MANAGING MAGIC BUBBLES?

Harry C. Karydes, Sean M. Bryant
Cook County Hospital (Stroger), Chicago IL USA

Background: Ingestion of low concentrations of hydrogen peroxide is generally considered benign. However, serious toxicity, such as venous/arterial gas embolism, hemorrhagic gastritis, gastrointestinal bleeding, shock and death, has been reported with higher concentrations. There are limited data on the necessary and appropriate imaging studies and treatment for suspected ingestions. We report hydrogen peroxide exposures over the course of 10 years managed by our regional poison center (RPC).

Methods: Cases from our RPC coded as hydrogen peroxide from January 1, 2001 to December 31, 2010 were retrospectively searched. All patients with gastrointestinal (GI) exposure to hydrogen peroxide (i.e those with suicide intent, volumes ≥ “mouthful”, rectal enema, and/or high concentrations) and managed at a healthcare facility were included. General data were recorded including types of imaging modalities, management strategies and outcome.

Results: A total of 760 cases were reviewed. Twenty-four patients met the inclusion criteria. The mean age was 33 years old (50% male) and 5 (21%) were suicide attempts. Of the study population, 5 (21%) were exposed to 35% hydrogen peroxide orally or via rectal enema, 3 (13%) had oral ingestions of 6-12% hydrogen peroxide, 11 (46%) had oral exposures of 35% hydrogen peroxide and 4 (17%) with unknown concentration. Ten (42%) patients had plain film x-rays (chest or abdominal x-ray) of which 9 (90%) were reported as normal and 1 (10%) reported gastric distention. One patient had an abdominal computed tomography (CT) which revealed portal venous air and subsequently received hyperbaric oxygen therapy with complete resolution on repeat imaging. Finally, 9 (82%) patients who ingested 35% hydrogen peroxide received an esophagogastroduodenoscopy (EGD) of which 7 (78%) revealed mild inflammation in esophageal and/or gastric mucosa. No cerebrovascular accidents or deaths occurred in our study group.

Discussion: Like other caustic ingestions, significant or unknown GI exposures to hydrogen peroxide, EGD has historically been advocated to determine any mucosal injury. Plain films and CT scans have also been utilized to ascertain any abnormal accumulations of air.

Conclusion: Over a 10-year period our RPC managed 24 cases of hydrogen peroxide GI exposures. Eleven consumed 35% hydrogen peroxide with a large majority of patients resulting in mild GI mucosal injury without any known significant morbidity. A prospective study may be warranted to evaluate the utility of recommending EGD for this population of patients. Additionally, further
data would help assign value to the various imaging studies regularly pursued in these patients.

134. NON-ST ELEVATION MYOCARDIAL INFARCTION AND STRESS CARDIOMYOPATHY ASSOCIATED WITH ACUTE MONOAMINE OXIDASE TOXICITY

Jarrett M. Burns, Kathryn W. Weibricht, Jeanine A. Ward, Brian J. Wolk, Giza M. High, Edward W. Boyer
Emory University School of Medicine, Atlanta GA USA

Background: To describe non-ST elevation myocardial infarction and stress cardiomyopathy associated with acute monoamine oxidase toxicity.

Case Report: A 46 year old female with bipolar disorder, maintained on isocarboxazid, lithium, lorazepam and trazodone, presented with MAO toxicity after ingesting an herbal dietary supplement, Phenylethylamine HCI. Worsening depression in the patient compelled her husband to administer 500 mg of phenylethylamine. Within 20 minutes, the patient developed severe headache, palpitations, diaphoresis, nausea and wheezing. At presentation she complained of chest pain; vitalis were normal except for BP 220/100 mm Hg. An EKG revealed NSR @ 88 bpm, QTc 508 msec, and ST-segment depressions in the inferolateral leads (II, III, aVF and V3-V6). A chest radiograph showed bilateral vascular congestion and pulmonary edema. The patient received aspirin 325 mg as well as intravenous furosemide, nitroglycerin infusion and lorazepam. She was transferred to the regional toxicology center. Her symptoms resolved as a second EKG, performed approximately 2.5 hours after the first, showed complete resolution of the ST-segment depressions. Laboratory testing showed elevated troponin-I of 0.69 ng/mL that peaked at 6.29 ng/mL. Creatine kinase peaked at 273 U/L, with a CK-MB of 39.8 ng/mL (index: 14.6). A 2D echocardiogram demonstrated markedly reduced left ventricular systolic function (ejection fraction of 30-35 %). Cardiac catheterization revealed no coronary artery disease. A diagnosis of stress cardiomyopathy was made. A second echo performed 2 weeks post discharge from the hospital demonstrated resolution of wall motion abnormalities, normal systolic function, and ejection fraction 60-65 %.

Case Discussion: Isocarboxazid is an irreversible nonselective monoamine oxidase inhibitor (MAOI) of the hydrazine class used as an antidepressant and anxiolytic. Phenylethylamine, a natural monoamine alkaloid, is a precursor of many psychoactive drugs with stimulant effects. As a dietary supplement, phenylethylamine is purported to have benefits related to improved mood and weight loss. The combination of these two agents may produce severe MAO toxicity, cardiac ischemia, and stress cardiomyopathy.

Conclusion: Patients must use caution when combining MAO inhibitors with any dietary supplements.

135. PROFOUND HYPOCALCEMIA IN A WOMAN STARTING RIFAMPIN

Michael Schwartz
Emory University School of Medicine, Atlanta GA USA

Background: Drug-induced hypocalcemia may result from treatment with a number of pharmaceutical agents. Though usually mild, hypocalcemia may occasionally manifest with severe symptoms and EKG changes. Hypocalcemia is a well-known adverse drug reaction associated with anti-convulsants, anti-infectives, and proton-pump inhibitors. We describe a case of profound hypocalcemia secondary to rifampin therapy.

Case Report: A 42 year-old Nepalese woman presented to the ED complaining of musculoskeletal pain, profound weakness (4 / 5 on Objective strength testing in all extremities), fatigue, and diffuse abdominal pain without nausea or vomiting. She had immigrated to the US five months previously. Routine immigration health screening revealed a positive PPD (BCG status unknown) and she was started on rifampin 1 month prior. An EKG revealed a prolonged QTc (580 msec); chest radiographs showed no active - or evidence of prior – TB pulmonary disease. The calcium was 5.1 mg/dl (ionized Ca 3.5 mg/dl). The patient began aggressive IV calcium gluconate and oral potassium repletion. Common causes of hypocalcemia were ruled out: primary hypoparathyroidism (PTH = 18.6 pg/ml) and thyroid disease (T4 = 10.5 ng/dl). Active Vitamin D intermediates were profoundly depressed (1, 25-Vit D2 = 17 pg/ml; 25-Vit D2 < 4 ng/ml, total 25-Vit D = 9 ng/ml). The patient was admitted overnight and received a total of 8 gm calcium gluconate IV with resolution of her weakness and ECG changes (QTc = 477 msec). Radiographic survey and AFB cultures revealed no evidence of TB disease. She was discharged on INH and Vitamin D and calcium supplementation.

Discussion: Drug-induced hypocalcemia may result from impaired calcium absorption (PPIs), secondary to drug-induced hypomagnesemia (aminoglycosides, cisplatin), from direct chelation of calcium (citrate, HF) or impaired bone resorption (bisphosphonates), or by impaired Vitamin D synthesis and trafficking (anti-convulsants). Rifampin is thought to cause Vitamin D deficiency through enhanced metabolism of active Vitamin D via P450 induction. Early researchers demonstrated reduced circulating levels of active 25-OH and 1,25-OH Vitamin D intermediates in subjects taking rifampicin (Brodie, 1982; Williams, 1985).

Conclusion: We report a case of rifampin-induced hypocalcemia with demonstrated Vitamin D deficiency. Many classes of drugs are associated with hypocalcemia; both clinicians and toxicologists should be aware of, and monitor for, electrolyte abnormality ADRs resulting from commonly prescribed medications.

136. BULLOUS ERUPTION FOLLOWING EXTRAVASATION OF IV ACETYLCYSTEINE: A PREVIOUSLY UNREPORTED ADVERSE EVENT

Laura Tormoehlen1, Louise Kao1, James Mowry2
1Indiana University School of Medicine, Indianapolis IN USA; 2Indiana Poison Center, Indianapolis IN USA

Background: Intravenous acetylcysteine (IV-AC) is an effective treatment for acetaminophen-induced hepatotoxicity. It is also indicated therapy for transaminitis in the setting of unreliable ingestion history. We report a case of extravasation of IV-AC, resulting in a bullous skin eruption.

Case Report: A 36-year-old man was transferred from a referring hospital after he was found to be unresponsive by EMS. Polysubstance ingestion was suspected in this skin reaction were apparent. The IV catheter was removed, warm compresses were applied, and the arm was elevated. The 21-hour course of IV-AC was completed at an alternative site without complication. Serum transaminases returned to normal prior to discontinuation of therapy. Upon further questioning, he reported intentional ingestion of alprazolam, oxycodone, and amitriptyline in the setting of chronic methamphetamine use. The bullae were drained, and the patient was discharged from the hospital on a course of oral cephalexin. Resolution of the skin reaction cannot be verified as the patient was lost to follow-up evaluation.

Conclusion: A novel adverse reaction to IV-AC is reported. Extravasation of IV-AC may result in a severe bullous skin reaction.

137. FATAL HYPERTHERMIA ASSOCIATED WITH TOPIRAMATE

Stephen W. Borron, Robert Woolard, Susan Watts
Paul L. Foster School of Medicine, El Paso TX USA

Background: Topiramate is indicated for partial-onset and generalized tonic-clonic seizures and for the prevention of migraines. It blocks voltage-dependent
Comprehensive urine drug testing revealed the presence of caffeine, lidocaine, oxycodone, and hydrocodone, the latter of which were administered in the hospital. A plasma colchicine level, obtained 3.5 days after colchicine administration was 2.3 ng/mL.

Over the next 2 weeks, near complete aloppecia and painful neuropathies involving the lower extremities developed. Gabapentin therapy was begun.

Discussion: The use of complementary and alternative medications can be associated with undesired and unexpected complications. The naturopathic physician did confirm that he “stocked up” on injectable colchicine, before it was removed from the US market. The history, along with quantitative levels confirm colchicine as the etiology of the toxicity.

Conclusion: Iatrogenic toxicity from colchicine infusions is rare. This case illustrates colchicine toxicity resulting in substantial morbidity following intentional administration for the treatment of fibromyalgia.

139. CLUSTER OF CHRONIC LITHIUM TOXICITY IN A CORRECTIONAL FACILITY
Ronald I. Kirschner1, Claudia L. Barthold1
1University of Nebraska Medical Center, Omaha NE USA; 2Nebraska Regional Poison Center, Omaha NE USA

Background: Upon entering a correctional facility (CF) patients may be seen by a variety of providers and have changes made to their medications. This can be problematic with drugs such as lithium (Li) which have a narrow therapeutic index and multiple drug interactions. We report 3 cases of Li toxicity from the same CF presenting within a 2 month period.

Case 1: A 54 year old woman was sent to the Emergency Department (ED) for altered mental status (MS). She was started on Li at the CF 2 weeks prior and had been refusing to eat or drink for 2 days. Medications included Li, metformin, lisinopril, simvastatin, and aspirin. She was sleepy but arousable with BP 148/84, P 100, dry skin/mucous membranes, hyperreflexia, ankle clonus, and fine tremor. Labs were notable for new onset renal insufficiency and a Li level of 3.2 mmol/L. After treatment with hydration and hemodilysis, she was discharged to the CF with normal MS and renal function on hospital day 6.

Case 2: A 52 year old man from the CF presented to the ED with altered MS and headache. He had refused food and drink for 2 days. Medications included Li, ranitidine, diclofenac, sulfasalazine, and diflucan. He was agitated with unintelligible speech, BP 141/97, P 104, T 38.1, and dry skin/mucous membranes. Labs included BUN/creatinine 49/2.4 mg/dL (baseline unknown), and Li 3.0 mmol/L. Renal failure resolved after rehydration. His Li level declined, and MS normalized.

Case 3: A 43 year old man was sent from the CF for tremors and an elevated Li level. He had been started on Li 3 weeks earlier. Medications included lisinopril, hydrochlorothiazide, bupropion, ibuprofen, and Li. He was alert and oriented with mild tremor and ataxia, but normal vital signs. Labs were notable for increased BUN/creatinine and Li 2.7 mmol/L. After hydration, BUN/creatinine and Li level improved, and he returned to the CF the next day.

Discussion: Prisoners may have untreated psychiatric conditions for which medications are started after incarceration. It was common practice at this CF for a psychiatrist to order Li without consulting other medical staff. Once the previously undetected pattern of Li toxicity was noted by the poison center, CF medical staff was contacted. With poison center consultation, it was agreed that Li would only be given after the CF medical director reviewed inmates’ medical histories and medications. Subsequently, there have been no known cases of chronic Li toxicity at this CF.

Conclusion: Incarceration may be a previously unrecognized risk factor for chronic Li toxicity. Additionally, the poison center is able to detect toxicity patterns that may not be apparent to local medical providers.

140. SELF-MEDICATION WITH AMITRAZ FOR DELUSIONS OF PARASITOSIS; DERMAL EXPOSURE AND A DELAYED PRESENTATION
Jeena Jacob, Tammi H. Schaeffer
Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA
Background: Amitraz is an insecticide and acaricide used in veterinary medicine. It is a neurotoxin and alpha-2 adrenergic agonist. We report a case of a dermal amitraz exposure and an emergency department (ED) presentation 16 hours later consistent with alpha-2 agonist toxicity. Case Report: A 33-year-old previously healthy woman on no medications presented to the ED with weakness and lightheadedness. Sixteen hours prior to presentation, she had applied her dog’s amitraz 12.5% “from head to toe” in one time application for treatment of perceived mites over her entire body. She had tried ivmectin and permethrin in the past without improvement. The lightheadedness started at home approximately 8 hours after dermal application. Upon arrival the ED her blood pressure (BP): 82/47 mmHg, heart rate (HR): 61 bpm. Physical exam including mental status was normal. She did not have any other signs of hypoperfusion such as abnormal creatinine or EKG changes. EKG showed sinus bradycardia. Approximately 2 hours after presentation, her BP dropped to 66/28 mmHg despite receiving 1 liter 0.9% normal saline (NS). She subsequently responded to a total of 6 liters NS and her BP improved to 103/53 mmHg though she remained persistently bradycardic and lightheaded. Her HR ranged between 45-55 bpm for most of her ED stay. She was transferred to the intensive care unit (ICU) approximately 12 hours after ED presentation at which time her BP: 85/52 mmHg and HR: 51 bpm. Her bradycardia and hypotension improved with intravenous fluids during the course of her first hospital day in the ICU to BP: 120/85 mmHg and HR: 85 bpm. She remained asymptomatic with normal vital signs for the remainder of her hospital stay. Intrinsic causes for her symptoms including electrolyte abnormalities, cardiac disease, and endocrine abnormalities were ruled out by history or laboratory tests. She was transferred on the second hospital day to an inpatient psychiatry facility for presumed delusions of parasitosis.

Discussion: This is a case of a dermal amitraz exposure with a patient presenting with hypotension and bradycardia 16 hours after application that continued for 30 hours post-exposure. Studies of human dermal toxicity have shown onset of symptoms within 30-150 minutes and cases of human ingestions have reported a 30-90 minute onset of action. The limitations of this report include reliance on the patient history and unavailable laboratory results to confirm exposure though her clinical presentation is very consistent with amitraz toxicity.

Conclusion: We alert clinicians to the possibility of delayed symptoms of hypotension and bradycardia after dermal exposure to the veterinary pharmaceutical, amitraz.

141. CARDIOGENIC SHOCK ASSOCIATED WITH THERAPEUTIC IV FOSPHENYTOIN LOAD
Erine O. Fong1, Benjamin S. Orozco2, Jon B. Cole1, Stephen W. Smith2
1Hennepin Regional Poison Center, Minneapolis MN USA; 2Hennepin County Medical Center, Minneapolis MN USA

Introduction: Patients in the emergency department (ED) often require IV loading of anticonvulsants for seizures. Phenytin is a first line agent for status epilepticus, however rapid administration is associated with cardiogenic shock believed to be due to the diluent, propylene glycol. Fosphenytoin was developed as a safer alternative, however cardiotoxicity still occurs. We present a case of cardiotoxicity from therapeutic IV fosphenytoin loading despite use of the recommended infusion rate.

Case Report: A 49 yo man with a history of coronary artery disease (CAD) and seizure disorder arrived to the ED in status epilepticus. Blood glucose was 128 mg/dL. His medications included carvedilol and amiodarone. His seizures were refractory to 2 mg of lorazepam, thus he was given an 18 PE/mL (reference range 5-15 μL/mL). Urinalysis noted 0-2 RBC, 15-20 WBC, protein 100 mg/dL, without casts. The patient had his stat labs drawn including a coagulation screen, BMP, and heparin dosing titration. A patient with CAD, Hx of atrial fibrillation on dabigatran, and extensive recent surgery. While he was in the ED, he developed visual hallucinations, myoclonus, and confusion which worsened and serum bicarbonate levels declined prompting hemodialysis (HD) on hospital day 3. Within one week of admission, and after her second round of HD, she was back to baseline mental status with complete resolution of myoclonus.

Case Discussion: Beta-lactam antibiotics have been associated with neurotoxicity. Clinical findings with cefepime-induced neurotoxicity (CIN) include encephalopathy and myoclonus, though coma and non-convulsive status epilepticus have been described. A common feature, and risk factor for neurotoxicity, is renal insufficiency. Hemodialysis (HD) is a viable option and may reduce cefepime half-life from 13.5 hours to 2.3 hours.

Conclusion: We report a case of cefepime induced neurotoxicity associated with acute kidney injury. Hemodialysis has been shown to reduce cefepime half-lives and should be considered in these patients.

143. MI AFTER USE OF YOHIMBINE
Beth Y. Ginsburg, Swathi Nadindla, Lara Bomba, Alex F. Manini
Mount Sinai School of Medicine, Elmhurst Hospital Center, Elmhurst NY USA

Discussion: Fosphenytoin is a water-soluble molecule developed to address some of the undesirable effects of phenytoin, including impaired myocardial contractility, decreased peripheral vascular resistance and depressed myocardial conduction. Despite the lack of propylene glycol, cardiovascular complications have also been described with rapid and/or supratherapeutic IV administration of fosphenytoin. Our case is unique, however, in that cardiogenic shock occurred despite a therapeutic dose at the recommended rate of 150 PE/min. While a prevailing theory is toxicity from fast cardiac sodium channel blockade, in our case QRS widening was not observed on ECG, making another uncertain mechanism more plausible. With no evidence of focal ischemia on ECG or echo, our patient’s CAD is very unlikely to have contributed to his cardiogenic shock, however it is conceivable his antihypertensives may have rendered him more susceptible.

Conclusion: IV fosphenytoin can result in bradycardia, hypotension, and cardiogenic shock. Providers who administer IV fosphenytoin should be aware of these rare but life-threatening side effects.
Background: Yohimbine, found in unregulated products sold as stimulants and aphrodisiacs, is an α2-adrenergic antagonist with adverse effects related to sympathomimetic properties. We report a case of myocardial infarction (MI) associated with yohimbine use.

Case: A 60 year-old man presented to the ED with chest pain, palpitations, diaphoresis, and dyspnea on exertion for 2 weeks. He admitted to first-time use of yohimbine as an energy supplement 2 weeks prior. He developed weakness and dizziness 30 minutes after ingestion, followed by chest pain, palpitations, tinnitus, and diaphoresis lasting several hours. He denied subsequent yohimbine use. He reported cocaine use over 30 years ago and last tobacco use 1 year ago. He denied any medical or relevant family history. His presenting physical examination was normal. ECG revealed Q waves in leads V5 and V6. Chest radiography showed mild congestion. Laboratories were remarkable for troponin I of 0.37 ng/mL and HDL of 18 mg/dL. He was admitted to the CCU and started on aspirin, heparin, metoprolol, and rosvastatin. Cardiac catheterization revealed 2-vessel disease, ejection fraction 50%, and lateral wall hypokinesia. PCI was performed with stent placement in OM1 (90-95% thrombotic stenosis). Echocardiogram also revealed LHV. The hospital course was uncomplicated and clopidogrel was added to his medications.

Discussion: α2-adrenergic receptors are found in the CNS and PNS. Blockade of peripheral presynaptic receptors on noradrenergic nerves, postsynaptic receptors in the nucleus tractus solitarius, and receptors in the locus ceruleus may result in enhanced sympathomimetic output. As a result, α2-adrenoceptor antagonists such as yohimbine mediate a variety of cardiovascular effects including coronary vasoconstriction which may lead to myocardial ischemia. Agitation and other sympathomimetic effects of acute yohimbine toxicity should be treated with benzodiazepines. Use of β-adrenergic antagonists may result in worsening vasoconstriction due to unopposed α1-adrenergic activity and should be avoided. Clonidine, an α2-adrenergic agonist, is a potential antidote. Our patient had risk factors for coronary disease including tobacco use and possibly underlying hypertension as evidenced by LHV. Yohimbine was likely a precipitating factor in his MI via its vasocostrictive effects and may have destabilized an atherosclerotic plaque. Our patient did not require antidotal therapy for yohimbine toxicity since his last exposure was 2 weeks prior.

Conclusions: We believe that this is the first report of MI associated with yohimbine use. Physicians should be aware of yohimbine’s unintended cardio-toxicity, especially in the setting of coronary disease.

144. COMPARATIVE CLINICAL TOXICITY OF INDIVIDUAL ANTIPSYCHOTIC DRUGS AFTER OVERDOSE

Salem M. Fathalla1, Janine Gray1, Simon H. Thomas2
1 Janine Gray Statistical Consultancy, East Bolden UK; 2Newcastle University, Newcastle UK

Background: Antipsychotic drugs are widely prescribed to people at high risk of drug overdose, with increasing recent use of atypical (second generation) types. Although the clinical features of antipsychotic poisoning are well characterized, there is limited information on their differential toxicity. This study was therefore performed to compare clinical effects of overdose with individual antipsychotic drugs.

Methods: Observational cohort study involving adult patients with first presentation with antipsychotic drug overdose (Newcastle, 2000 to 2008). Multivariate analysis was used to investigate the relationship between drugs involved in overdose and prespecified clinical outcomes of interest, which were reduced level of consciousness (Glasgow Coma Scale ≤ 9), seizure, hypotension (blood pressure < 90 systolic or 60 diastolic), arrhythmias and death.

Results: There were 443 first presentations with reported antipsychotic overdose involving 224 females (51%, mean age 32) and 219 males (mean age 35). Hypotension occurred in 44 episodes and was more common with co-ingestion of other antihypertensive agents (Odds Ratio (OR) 4.86, 95% CI 1.37, 17.33). There were no significant independent effects of age, sex, cardiovascular disease or any individual antipsychotic drug. Reduced GCS occurred in 35 episodes and with risk significantly higher for olanzapine (OR 4.21, 95% CI 1.73, 10.26). Seizures occurred in 6 episodes, bradycardias in 5 (1 chlorpromazine, 4 haloperidol) and cardiac arrest in 1 (quetiapine). There were no documented episodes of ventricular tachycardia or torsade de pointes and no deaths. These numbers are too low for detailed statistical analysis.

Conclusions: Serious consequences of antipsychotic overdose are uncommon. Reduced level of consciousness may be more common with olanzapine but otherwise no significantly different risks of events of interest between drugs were documented. Study limitations include reduced statistical power, especially for less common outcomes, retrospective methodology with incomplete availability of data, unavailability of analytical confirmation and lack of reliable information on doses taken. Although adjustment was made where possible, the possibility of residual confounding or bias remains.

145. REPORTING MORTALITY FROM ANIMAL ENVENOMATION: DISCREPANCIES BETWEEN THE CDC WONDER DATABASE AND THE AMERICAN ASSOCIATION OF POISON CONTROL CENTER NATIONAL POISON DATA SYSTEM ANNUAL REPORTS

Joseph D. Forrester, Nathan P. Charlton, Heather A. Borek, Christopher P. Holstege
University of Virginia School of Medicine, Charlottesville VA USA

Background: The total number of US deaths reported each year due to envenomation is unclear. The Objective of this study is to compare envenomation fatality data between the two largest US databases.

Methods: The Centers for Disease Control and Prevention WONDER database (CDC) and the American Association of Poison Control Center National Poison Data System Annual Report (NPDS) were compared. The CDC was queried to return all fatalities between 1999 and 2007 from contact with venomous snakes and lizards (ICD-10:X20), contact with venomous spiders (ICD-10:X21), contact with scorpions (ICD-10:X22), and contact with hornets, wasps & bees (ICD-10:X23). Results were grouped by age. A review of the NPDS was performed for 1999 to 2007. The information pertaining to deaths from bites and stings was extracted from the summaries of fatal exposures reported in the toxic exposure surveillance system tables for each of the respective years. This NPDS data was then age grouped and organized into categories that corresponded to the CDC ICD-10 codes.

Results: There were at total of 59 and 26 deaths reported from contact with venomous snakes and lizards reported by the CDC and the NPDS, respectively. There were 70 reported deaths from contact with venomous spiders in the CDC, but only 5 reported for the exact same time period in the NPDS. The CDC reported 5 deaths due to contact with scorpions and NPDS reported 2. The largest discrepancy between any of the four queried causes of death was found in those who died due to contact with hornets, wasps and bees: the CDC reported 499 deaths and NPDS only 8 deaths. The Table 1 below documents CDC and NPDS fatalities by age group for each envenomation.

Table 1.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>CDC Spiders &amp; Lizards</th>
<th>NPDS Spiders &amp; Lizards</th>
<th>CDC Spiders</th>
<th>NPDS Spiders</th>
<th>CDC Scorpions</th>
<th>NPDS Scorpions</th>
<th>CDC Hornets, Wasps, Bees</th>
<th>NPDS Hornets, Wasps, Bees</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1-4</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>5-9</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>10-14</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>15-19</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>20-24</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>25-34</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>35-44</td>
<td>16</td>
<td>10</td>
<td>11</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>45-54</td>
<td>12</td>
<td>7</td>
<td>17</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>145</td>
<td>0</td>
</tr>
<tr>
<td>55-64</td>
<td>7</td>
<td>2</td>
<td>17</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>91</td>
<td>1</td>
</tr>
<tr>
<td>65-74</td>
<td>5</td>
<td>0</td>
<td>9</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>75</td>
<td>0</td>
</tr>
<tr>
<td>75-84</td>
<td>3</td>
<td>0</td>
<td>7</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>45</td>
<td>1</td>
</tr>
<tr>
<td>&amp; 85</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Totals</td>
<td>59</td>
<td>26</td>
<td>70</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>499</td>
<td>8</td>
</tr>
</tbody>
</table>
146. EPIDEMIOLOGY OF INCAPACITATED SEXUAL ASSAULT IN A COMMUNITY-BASED POPULATION

Brook Woolley, Colleen Bush, Bryan S. Judge, Linda Rossman
GRMEP/MU Program in Emergency Medicine, Grand Rapids MI USA

Background: Although women’s substance use is believed to contribute to sexual assault vulnerability, few studies have compared the characteristics of rape that occurs through incapacitation or intentional intoxication from those that are due to perpetrator physical aggression. The current study compares the prevalence, epidemiology, and injury patterns of these two forms of rape in a community-based population.

Methods: This case-series analysis evaluated consecutive female patients presenting to a sexual assault clinic over a seven-year study period. Sexual assault victims presenting directly to four downtown emergency departments were routinely referred to the sexual assault clinic for evaluation after triage and initial assessment. The clinic is associated with a university-affiliated emergency medicine residency program and is staffed by forensic nurses trained to perform medicolegal examinations using colposcopy with digital imaging. Patient demographics, assault characteristics, and injury patterns were recorded using a standardized classification system. Comparative data from the two patient groups (incapacitated vs. forcible rape) were analyzed using chi-square, t-test, and multivariate analysis.

Results: Case files of 1786 cases of sexual assault were reviewed; 19% (339/1786) of the victims were documented as incapacitated. Alcohol consumption in the hours prior to the assault was reported by 90% of incapacitated victims; 19% described recreational drug use and 9% reported using prescription medications. A total of 28% (95/339) of these women reported rape due to covert drug administration by the perpetrator. Incapacitated rape incidents differed from forcible rape on several contextual variables, including relationship to perpetrator, location and time of assault, history of childhood sexual abuse, and frequency of non-genital injuries. Incapacitated victims had significantly fewer anogenital injuries (39% vs. 73%, P<0.001), although the overall injury pattern was not statistically different (P=0.79). Incapacitated victims also had a longer delay to treatment (26 hours vs. 17 hours, P<0.001), and were less likely to consent to a forensic examination or file a police report.

Conclusion: Findings suggest that one-fifth of sexual assault victims are mentally incapacitated through alcohol and/or drug use. The epidemiology of sexual trauma in this group is unique and may pose special challenges to emergency health care providers.

147. THE POISON CENTER AS A REPORTING AGENCY TO THE MEDICAL EXAMINER

Suzanne Doyon1, Mary Ripple2, Charlotte Noranbrock2, David Fowler2
1Maryland Poison Center, University of Maryland School of Pharmacy, Baltimore MD USA; 2Office of the Chief Medical Examiner, Baltimore MD USA

Background: In 2008, the Maryland Poison Center (MPC) became a reporting agency to the Office of the Chief Medical Examiner (OCME). Once per 24 hours period the MPC database is scanned for deaths. Identification of death(s) triggers an immediate email to designated recipients at the OCME. The OCME has access to the MPC electronic medical records via a secure portal system. Poisoning deaths so captured will have a death certificate (DC) completed by the OCME. Deaths not so-captured will bypass the OCME and have a DC completed by the treating physician or funeral home. In both instances the DCs are filed with the Department of Vital Records for the state.

Methods: The MPC database was queried for all deaths where the MPC sent an email and where the MPC was the primary and sole reporting agency from January 1 2008 to December 31 2010 (3 years). DCs were obtained for all these deaths. The cause and manner of death recorded in the DC were compared to the relative contribution to fatality (RCF) determined by the fatality review process in NPDS. Agreement of opinion was: NPDS RCF was probably or clearly responsible while the RCF determined in the DC was probably or clearly responsible (Agreement of opinion: 15/19 (79%) vs. 2/19 (10%) for all cases). Agreement of opinion was: NPDS RCF was probably or clearly responsible and the RCF determined in the DC was probably or clearly responsible (Agreement of opinion: 15/19 (79%) vs. 2/19 (10%) for all cases).

Discussion: The MPC reported 100 deaths via email to the OCME during the 3 year period. The MPC was the primary and sole reporting agency in 29 cases. The OCME captured 19/29 cases and did not capture 10 deaths. The OCME completed 19 DCs.

Conclusions: The implementation of the automated email system increased the capture rate by the OCME and resulted in greater agreement between OCME and MPC/NPDS opinions on causes of deaths. Reporting systems vary from state to state and any method that increases the probability that a medical examiner’s case will be caught is of benefit to the state.

148. EPIDEMIOLOGY OF UNINTENTIONAL CARBON MONOXIDE (CO) EXPOSURE IN THE UNITED STATES (US) 2000-2009: A REVIEW OF NPDS

Henry A. Spiller1, Svetla S. Slavova2, Terry L. Bunn2
1Kentucky Regional Poison Control Center, Louisville KY USA; 2Kentucky Injury Prevention and Research Center, University of Kentucky, Lexington KY USA

Background: CO exposure is common in the US with most reports focusing on regional events, treatment controversies or toxic mechanisms. We evaluated a large temporal and geographic area (US over ten years) to better elucidate the epidemiology of CO exposures.

Method: retrospective review of NPDS unintentional human exposures for CO 2000-2009. A scan statistic was utilized to detect space-time clusters. Additionally in selected states we performed temporal analysis involving a discrete Poisson model.

Results: 163,435 CO exposures were reported, of which 72% were acute exposures. 16,464 patients (10%) were chronic exposures of 1 to 30 days. Exposure site was primarily residence (79%) and workplace (11%). 69,470 CO exposures (43%) were treated in a HCF. Severe outcomes reported were: Death 474 (0.3%) Major 1,643 (1%) and Moderate13,703 (8%). Mean CO exposures/year was 16,344 with a range of 14,206 to 17,658. Peak years were 2001 and 2004, with a consistent decreasing trend after 2004. For the total US there was a seasonal trend with recurring peaks in winter months, mainly January, Northern states (33 states, pop 153.3 million) had twice the number of reported exposures per 100,000/pop as southern states (18 states, Pop 141.4 million).7.8 vs. 3.8 respectively. Fifteen clusters were detected (p<0.001) in northern states of which 10 (67%) occurred during cold weather months. Seven clusters were detected (p<0.001) in southern states of which 3 (42%) occurred during colder weather months. Temporal analysis of Florida and Louisiana (cluster size of 15 days), identified 11 likely clusters.

Discussion: CO exposure remains common in the US with an overall cyclical seasonal pattern. Significant temporal clusters were identified, suggesting a role for timely targeted public health efforts in response to local weather/disaster events, when the message has the highest probability to be noticed. Spatial-temporal analysis is a useful statistical method to detect CO related clusters and helps to understand existing patterns of exposure.
Conclusion: poison center recognition of CO clusters may help public health agencies improve response to disasters and future education/prevention efforts.

149. PRIISON POISONINGS REPORTED TO A REGIONAL POISON CENTER

Stephanie L. Hon1, Linda Ekereri1, Gaylord P. Lopez1, Robert J. Geller2
1Georgia Poison Center, Atlanta GA USA; 2Emory University, Atlanta GA USA

Background: Poisonings while imprisoned have been reported to poison centers (PC); however, these cases have not been categorized in publication. We report prison exposure trends over a 4 year period as reported to our PC and the medical outcomes associated with these cases.

Method: This retrospective study reviewed adult prison poisonings reported to our PC from January 2007 to December 2010. Because there is no coding option in NPDS for site of exposure described as a “jail,” “prison,” or “penitentiary,” an automated query tool was used to identify cases where the exposure site was coded as “Other (Other)” or caller site as “US Penitentiary.” A manual chart review was then conducted to further identify and confirm the site of the exposure as a prison.

Results: Of the 366,837 exposures reported over 4 years, 522 (0.14%) originated from prisons. Most exposures were initially reported to the PC by health care professionals in 186 hospitals (37% of reports) as the caller site. Hospitals were also the site where a majority of the cases were managed (59%); 104 (20%) cases were handled within the prison itself. The mean age of subjects was 29.9 years; 448 (86%) were male. Most of the reported reasons for exposure were intentional, with 310 (59%) suspected suicide attempts and 30 (5.7%) reported abuse; 8 (1.5%) were reported as accidental. There were a total of 808 substances reported within the 522 exposure cases; 362 (69%) cases involved 1 substance, 160 (31%) involved 2 or more substances. Ingestion was the most reported route (90%). Of the 756 substances known at the time of the call, 522 (65%) were pharmaceutical agents, 78 (10%) substance were cleaning agents, and 78 (10%) were drugs of abuse. Antipsychotics (13%) were the most common reported pharmaceutical agent; antidepressants (12%) and analgesics (8.5%) followed. The top three drugs of abuse included sedative hypnotics (3%), opioids (2%), and cocaine (1.4%). Based on PC documentation, 116 (22%) cases involved patients who intentionally exposed themselves to their own prescribed medication, 158 (30%) cases involved patients obtaining the substance from another source, and the source was unknown for the remainder of the patients (48%). While no deaths were reported in this study population, 104 (20%) reported a moderate outcome. Follow-up was incomplete in 40% of cases.

Discussion: Prison exposures reported to the PC accounted for <1% of our annual call volume. Most of the demographic data and common substance types do not reflect typical PC poisoning trends.

Conclusion: Reviewing current prison poison inquiries and the demographics associated may help poison centers recognize and better manage the challenges of prison drug use and overdoses.

150. A REVIEW OF BATH SALT EXPOSURES REPORTED TO SIX REGIONAL POISON CENTERS

Brandon Warrick1, Meredith Hill1, Benjamin Lehr2, James Mowry3, David Gunmin1, Deborah Anderson4, Michael Wahl5, Robert Goetz6, Susan Smolinski4
1Children’s Hospital of Michigan Regional Poison Center, Detroit MI USA; 2Indiana Poison Center, Indianapolis IN USA; 3Wisconsin Poison Center, Milwaukee WI USA; 4Minnesota Poison Center, Minneapolis MN USA; 5Illinois Poison Center, Chicago IL USA; 6Cincinnati Poison Center, Cincinnati OH USA

Background: A new trend of marketing cathinone derivatives, such as Methylenedioxypolyvaleraone (MDPV) and mephedrone as “bath salts” became known to poison centers centers around November 2010. A new feature of the National Poison Data System (NPDS) that allows mutual sharing of de-identified data through a special projects report was implemented for the Deepwater Horizon and then Enbridge oil spills, greatly enhancing public health toxicosurveillance. A sharing agreement and special project report was implemented by six Great Lakes Regional Poison Centers to enhance public health surveillance regarding this rapidly evolving substance abuse trend. The ability to rapidly assess differences and patterns in the method of use, patient presentation, and optimal treatment was the goal of this project.

Methods: A retrospective analysis of “bath salt” cases reported to the Regional Poison Centers of six Great Lakes states was conducted from November 2010 through March 2011. Assessment was limited to patients treated in a health care facility. Patient information was shared through National Poison Database Systems (NPDS), and enhanced by each center to include brand name information when available, and verification of route, substance, and clinical effects. Data was extracted from the NPDS report, which includes clinical effects, therapies, other substances involved, patient age, and 3 digit zip code.

Results: A total of 219 hospitalized cases from 6 states were evaluated. Mean age was 29.83 ±9.2 (range 14–55 years). Inhalation (either smoking or smoking) was reported in 117 (55%), ingestion in 23%, injection, in 16%, and 6% had unknown route. There was significant regional differences between route and state (p<.0001), with one state seeing a predominance of parenteral abuse. Twenty brand names were implicated. Outcome was moderate in 56.6%, minor in 16.4%, major in 6.4% with one death, where MDPV was confirmed at autopsy as well as in the substance used. Clinical effects were predominantly sympathomimetic, with agitation in 35%, tachycardia in 34%, hypertension in 19%, and hallucinations in 15%. Benzodiazepines were the most commonly used therapy (49.8%), with other sedatives used in only 7%. In all states, the number of cases increased incrementally as each month progressed, with no signs of abatement.

Conclusions: “Bath Salt” abuse appears to be an emerging public health problem with clinical effects similar to MDMA and methamphetamine. A unique data sharing arrangement allowed for tracking this trend, and may assist with legislative efforts in the Great Lakes Area. At this time, only mephedrone is a controlled drug in one state. Sharing data with FDA, DEA. And state and local health departments may be optimized by regional comparisons and cooperation.

151. HOME STORAGE OF ACETAMINOPHEN (PARACETAMOL) EXPLAINS THE LIMITED IMPACT OF UK PACK-SIZE LEGISLATION ON ACETAMINOPHEN POISONING

Paul I. Dargan, David M. Wood
Guy’s and St Thomas’ NHS Foundation Trust, London UK

Background: Acetaminophen (paracetamol) is the commonest drug taken in overdose in the UK and responsible for 100 to 150 deaths per year. Legislation was introduced in 1998 to limit acetaminophen pack-sizes in an attempt to reduce the number and severity of acetaminophen overdoses. Studies have shown that overall the legislation has had a limited impact on the number and severity of acetaminophen overdoses. The aim of this study was to assess the amount of acetaminophen that individuals have stored, at home and in other areas, to investigate whether this may explain the limited impact of the legislation.

Methods: An internet market research portal was used to identify individuals who had acetaminophen tablets/capsules stored in at least one location. Data was then collected on participant age and sex, where they had acetaminophen stored and the number of tablets/capsules and blister packets/bottles of acetaminophen in each location.

Results: 1013 individuals completed the survey (50.2% male, 49.9% female), 9.5% aged 16–24, 22.7% 25–34, 23.2% 35–44, 21.6% 45–54, 15.5% 55–64, 7.5% aged 65+. 95.1% had acetaminophen in blister packs and 16.9% in bottles. 98.5% had acetaminophen stored at home, 28.3% at work, 16.6% in their car and 9.5% aged 16–24, 22.7% 25–34, 23.2% 35–44, 21.6% 45–54, 15.5% 55–64, 7.5% aged 65+. 95.1% had acetaminophen in blister packs and 16.9% in bottles. 98.5% had acetaminophen stored at home, 28.3% at work, 16.6% in their car and 42.0% in their handbag or briefcase. Home storage was in the kitchen in 68.5%, bathroom in 26.2%, bedroom in 30.0% and in another location in 12.6%. The mean ± SD (max) number of tablets in the home in blister packs was 17.3 ±24.7 (150) and in bottles was 29.8±39.6 (300). The percentage of individuals who had ≥24 tablets stored in the home environment in blister packs and/or bottles was 31.1%; those with ≥32 tablets was 21.9%, ≥64 tablets was 8.2% and ≥100 tablets was 5.2%. The Table 1 shows the mean ± SD number of packs of different sized blister packets in the home.

Conclusions: This study showed that almost all individuals have acetaminophen stored at home and the majority also have it stored in another location. Most have
multiple blister packets of acetaminophen in the home environment. A significant proportion had a potentially hepatotoxic amount of acetaminophen in the home environment, should they all be consumed in an impulsive overdose. This study suggests that the 1998 UK acetaminophen pack-size legislation has been ineffective in decreasing the availability and storage of acetaminophen, available for self-poisoning, both in the home and other locations and this may in part explain the limited impact of the legislation on acetaminophen overdoses.

152. INMATE EXPOSURES TO POISONS IN CALIFORNIA PENAL INSTITUTIONS: AN EPIDEMIOLOGY

Richard Geller, Serena Huntington, Robert Kezirian, S. Arain, Terry Carlson, Diane Strong
Children’s Hospital Central California, Madera CA USA

Background: Although the CPCS frequently observes them, poisonings occurring in penal institutions are poorly described in the medical literature.

Methods: CPCS Dotlab records underwent computerized search of free-text entries. Search terms included jail, prison, detention, correctional, institution, inmate and prisoner. A manual examination of computer-selected records excluded all but inmate poisoning exposures occurring while incarcerated in CA’s penal institutions, including jails, prisons and juvenile halls.

Results: We identified 627 inmates with 881 poison exposures in 2009 and 2010. Average age was 48.6 years. Gender was 85% M and 15% F. Identified exposures were to drugs (666) and non-drugs (141). The most frequent exposures were to antidepressants (90), antihypertensives (56), ibuprofen (53), acetylsalicylic acid (49), atypical antipsychotics (45), nuclo cleaning agents (40), unknown drugs (23), methamphetamine (19), lithium (18), diphenhydramine (17), indomethacin (17), aspirin (16), beta blockers (15), phenothiazines (15) and heroin (12). Reasons for exposure were: intentional self-harm (540), unintentional (106), abuse (75), misuse (67), therapeutic error (37), unknown (20), adverse reaction (13) and drug withdrawal (3). Outcomes were death in 3 cases, major effect in 14 and moderate effect in 114. Additionally, 44 inmates in custody required medical attention for an exposure which occurred before arrest, 15 for methamphetamine, 7 for cocaine and 7 for heroin ingestion. Also, 13 former inmates required medical attention for an overdose within days of being released from prison. 23 individuals were taken to the hospital because of drug use or overdose.

Conclusions: Our study provides a significant epidemiologic glimpse into inmate poisonings occurring in penal institutions in California. Most of the exposures were to OTC analgesics, psychotropic medications, antidepressants, cleaning agents and street drugs. In-custody methamphetamine and heroin use was surprisingly high. These cases had an unusually high rate of hospital utilization (58%) and a mortality rate of 0.48%. (Overall, the annual mortality rate for poisonings reported to US poison control centers averages about 0.05% in NPDS data.) Recently arrested individuals who become ill should be suspected of recent street drug use, particularly with methamphetamine, heroin or cocaine. Recent parolees may be at significant risk of overdose. Finally, it appears that jail confinement is in some instances substituted for substance abuse treatment in California.

153. SUICIDE FADS: FREQUENCY AND CHARACTERISTICS OF VICTIMS OF HYDROGEN SULFIDE SUICIDES IN THE UNITED STATES

Sarah Jane Reedy, Michael D. Schwartz, Brent Morgan
Emory University Department of Emergency Medicine, Atlanta GA USA

Background: Hydrogen sulfide (H2S) suicides appear to be more common in the United States (US) since the technique became popular in Japan and on suicide web sites in 2007. However, there has not yet been a comprehensive survey to capture these incidents. By describing the characteristics of H2S suicide victims in the US, we hope to shed light on this disturbing trend and further understand the implications for first responders attempting to extricate victims from these toxic environments.

Methods: To ascertain the frequency of intentional H2S related deaths in the US prior to the start of the Japanese trend in 2007, we searched the multiple-cause-of-death data from the National Vital Statistics System (NVSS). To collect as much information about the victims as possible, we sent an email to the National Association of Medical Examiners (NAME) listserv asking for their cooperation in identifying cases of H2S suicide. To identify cases that were not voluntarily reported by medical examiners but were reported by the media, we conducted Google searches utilizing the search terms: “hydrogen sulfide suicide,” “H2S suicide,” “detergent suicide,” “chemical suicide,” and “suicide fad.”

We obtained all available autopsy reports and abstracted information including the site of the incident, the presence of a note warning others about the toxic gas, and the demographic characteristics of the victims. We contacted medical examiners who potentially had custody of the cases which were identified through media reports and requested autopsies of these victims. When unable to obtain the autopsies, we gathered information from the media reports.

Results: An inquiry of the NVSS resulted 45 deaths from H2S exposure that occurred in the US from 1999 to 2007; all were deemed unintentional. Responses from the NAME listserv yielded autopsy reports for 11 victims and Google searches revealed an additional 19 H2S suicides in the United States since 2008. Overall (n = 30), 2 cases were identified during 2008, 10 cases in 2009, and 18 cases in 2010. The majority of victims were white males, younger than 30 years old, left a note warning responders of the risk of exposure to H2S, and were found in cars. There were 5 reports of injuries to first responders, but no secondary fatalities.

Conclusions: H2S suicides are increasing in the US and their incidence is probably underestimated by public health officials and physicians. First responders are at risk when assessing these victims due to the severe toxicity of the gas. Emergency providers must be aware of H2S suicides to educate others and care for the rare survivor.

154. HOMICIDAL POISONINGS IN THE UNITED STATES: AN ANALYSIS OF THE FEDERAL BUREAU OF INVESTIGATION UNIFORM CRIME REPORTS FROM 2000 TO 2009

Kiera N. Sibbald1, Christopher P. Holstege2, Brent Furbee1, Thomas Neer3, Greg B. Saathoff3
1Indiana University School of Medicine, Indianapolis IN USA; 2Federal Bureau of Investigation, Quantico VA USA; 3University of Virginia School of Medicine, Charlottesville VA USA

Background: Documented homicidal poisonings are rare in U.S. society. As overall homicide rates in the U.S. have steadily declined over the past 3 decades, homicidal poisoning rates have reportedly increased from the decade of 1980-1989 to 1990-1999. This study was conducted to determine the total number of homicidal poisoning cases reported in the last decade (2000-2009) and compare with those numbers reported in the two previous decades.

Methods: The Federal Bureau of Investigation Uniformed Crime Reports (UCRs) from 2000 through 2009 were reviewed and compared in the areas of Overall Homicide, Homicide by Poison, Homicidal Poisoning by Age of Victim, and Homicidal Poisoning by Circumstance. These findings were compared to the Results of the previous two decades.

Results: From 2000-2009, there were a total of 142,916 U.S. homicides reported to the UCRs. Of those, 107 were homicidal poisonings (0.075%). This represented a significant drop in total number of homicidal poisonings and the percent of total homicides from the previous 2 decades: 1980-1989 (292 total and 0.14%); 1990-1999 (346 total and 0.18%). By year, there has been no significant change in the percentage of homicides facilitated by poison over the past decade (average of 10 cases per year). There were twice as many adult victims reported (total 71) when compared to minor victims (total 35), with one age listed as unknown. The largest reported group of victims was over the age of 75 (17 total; 16% of total). The majority of victims were white males, younger than 30 years old, left a note warning responders of the risk of exposure to H2S, and were found in cars. There were 5 reports of injuries to first responders, but no secondary fatalities.

Conclusions: This study of the UCRs demonstrates a significant decrease in the total number of reported homicidal poisoning cases and percentage

Table 1.

<table>
<thead>
<tr>
<th>Number of tablets/capsules per packet</th>
<th>Number (%) respondents</th>
<th>Mean ± SD number of packets</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>185 (18.3%)</td>
<td>1.9 ± 6.0</td>
</tr>
<tr>
<td>16</td>
<td>471 (46.5%)</td>
<td>4.4 ± 10.1</td>
</tr>
<tr>
<td>24</td>
<td>106 (10.5%)</td>
<td>1.3 ± 5.0</td>
</tr>
<tr>
<td>32</td>
<td>110 (10.9%)</td>
<td>2.0 ± 7.7</td>
</tr>
</tbody>
</table>
of reported total homicide cases over the past decade. There is a disproportionately high number of victims over the age 75. It is important to note that the UCR only documents detected poisonings; a significant number of these crimes potentially go undetected and unreported.

References

155. HOSPITAL ADMISSIONS FOR DRUGS OF MISUSE IN THE US AND ENGLAND, 2001-2010
Alvin C. Bronstein1, Stacey Wyke1, Daniel A. Spyker3
1International Research and Development Group, Centre for Radiation, Chemicals and Environmental Hazards, Health Protection Agency, UK;
2Rocky Mountain Poison Center, University of Colorado School of Medicine, Denver CO USA;
3Department of Internal Medicine, Uniform Services University of Health Sciences, Bethesda MD USA

Objective: US Poison center calls for methadone and other drugs of misuse have exhibited distinct changes over time. We compared these with patterns in England (Eng) for 5 substance categories: cocaine, heroin, LSD, marijuana, and methadone.

Methods: We examined hospital admissions (HA) for 2000-2009 in Eng using the 4-digit ICD-10 code, and in the US, all calls where the patient was either already enroute to or referred to a health care facility. We defined HA per million population at risk as HA/m. HA/m for each substance was examined: graphically vs time, statistically (linear and quadratic regression) vs time, prediction of 2010 US values, and correlation coefficient for England vs US.

Results: Eng reported 30,037 HA (9 y) with a 2009 population of 51.8 m, compared to 122,750 US HA with a population of 311.4 m. Mean HA/m for the 9 y were similar for LSD and methadone, but were higher in Eng for heroin, and higher in the US for cocaine and marijuana. HA/m over time for the 9 y (quadratic regressions) were statistically significant (p < 0.05) for 4 of the substances for Eng and for 4 for the US. Regressions at year 2009 showed decreases in 3 and increases in 1 for the Eng and decreases for 2, increase for 1 and a peak for 1 in the US.

Table 1.

<table>
<thead>
<tr>
<th>Substance</th>
<th>HA/m, Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Eng</td>
</tr>
<tr>
<td>Cocaine</td>
<td>13.7 ± 5.01</td>
</tr>
<tr>
<td>Heroin</td>
<td>36.0 ± 3.27</td>
</tr>
<tr>
<td>LSD</td>
<td>0.580 ± 0.120</td>
</tr>
<tr>
<td>Marijuana</td>
<td>3.24 ± 0.895</td>
</tr>
<tr>
<td>Methadone</td>
<td>12.4 ± 3.02</td>
</tr>
</tbody>
</table>

Table 2.

<table>
<thead>
<tr>
<th>Substance</th>
<th>HA/m, Change at 2009: p-values 1st, 2nd order</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Eng</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Decrease: p=0.001, 0.034</td>
</tr>
<tr>
<td>Heroin</td>
<td>undetermined: p=ns, ns</td>
</tr>
<tr>
<td>LSD</td>
<td>Decrease: p=0.013, 0.019</td>
</tr>
<tr>
<td>Marijuana</td>
<td>Decrease: p=0.034, ns</td>
</tr>
<tr>
<td>Methadone</td>
<td>Increase: p=0.0001, ns</td>
</tr>
</tbody>
</table>

2010 data were available for the US HA/m and were consistent with predictions based on 2001-2009 data for 3 of the 4 substances. HA/m over time exhibited similarities for cocaine (last 3 y), LSD (last 8 y), and distinct similarity for methadone (all 9 y). The correlation coefficient for methadone HA/m for Eng vs US was r = 0.958 (p < 0.0001, N = 9). Limitations of these analyses include: HA were determined by different methodologies (hospital ICD-10 code [Eng] and NPDS calls [US]) and different annual reporting windows (Apr-Mar in the UK and Jan - Dec in the US).

Conclusion: Both the temporal patterns and the absolute rates of HA/m showed similarities for the 5 substances examined. Methadone and cocaine showed the most consistent pattern of change over time. Longitudinal international comparisons of morbidity and mortality may help us better understand exposure patterns and needed interventions.

156. POISON CENTER CALLS FOR INTENTIONAL EXPOSURES TO OPIOIDS ARE HIGHLY CORRELATED WITH RETAIL AVAILABILITY IN THE RADARS SYSTEM POISON CENTER PROGRAM
E. J. Winter, C. Buchholtz, J. M. Davis, S. G. Severtson, R. C. Dart, RADARS System Poison Center Group
Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA

Background: Calls to poison centers (PCs) involving prescription opioids have increased in recent years. The strength of the relationship between PC intentional exposure call (IEC) volume and drugs with higher retail availability has not been measured. The purpose of the current study was to compare retail availability of prescription opioids to the number of IECs received by PCs for these drugs.

Methods: The RADARS System Poison Center Program captures acute drug exposures from participating US PCs. PCs use a standard electronic system to record spontaneous calls from the public; the coordinating PC performs quality control checks to verify product coding accuracy. IECs are coded as suspected suicide, misuse, abuse, intentional unknown, or withdrawal exposures. Total calls by quarter from 2003-2010 involving at least one target opioid mention (hydrocodone, oxycodone, fentanyl, hydromorphone, morphine, buprenorphine, methadone) were included in the analysis (N = 166,180). Retail availability is represented by Unique Recipients of Dispensed Drug (URDD) within the reporting PC coverage area for each quarter. Mixed effects modeling correcting for autocorrelation was used to determine the relationship of retail availability and overall PC IEC volume.

Results: RADARS System Poison Center US population coverage ranged from 21.2% in 1Q2003 to 75.6% in 4Q2010. Individual opioid class IECs ranged from 2,765 (hydromorphone) to 84,582 (hydrocodone). The expected call volumes to PCs per 100,000 URDD are: hydrocodone 12.2 (95% CI: 5.4-27.9), oxycodone 26.6 (15.5-45.6), fentanyl 36.0 (28.7-45.2), hydromorphone 42.2 (37.7-47.1), morphine 83.1 (68.1-101.5), buprenorphine 98.1 (88.2-109.1), and methadone 168.6 (141.8-200.4). Inclusion of an interaction term suggests that across all drug classes there is some variability in the association between IEC calls and URDD, but higher quarterly URDD are a strong predictor of greater quarterly IECs across all drug classes (p < .001).

Conclusion: Retail availability is a strong predictor of PC IEC volume when considering drugs of varying levels of availability. Overall PC IEC volumes are sensitive to changes in retail availability, and in light of increasing retail availability of all opioid products, PCs will continue to provide a valuable public health service.

157. MASS POISONING WITH SODIUM AZIDE (NAH3) AT A RESTAURANT
Evan S. Schwarz1, Paul M. Wax1, Kurt C. Kleinschmidt1, Wendy Chung2, Gabriela Cantu2
1University of Texas Southwestern, Dallas TX USA; 2Dallas County Health and Human Services, Dallas TX USA
Sodium azide inhibits oxidative phosphorylation and increases the production of nitric oxide, a vasodilator. Ingestion can cause profound hypotension and syncope. It was once used to treat hypertension; now, it is used in laboratories and in airbags. We report a cluster of poisonings where iced tea was contaminated with sodium azide.

Four of the 5 presented to the same hospital; the same physician evaluated 3 of the patients. The hospital alerted the health department (HD) and had the restaurant voluntarily closed. The HD did not believe the outbreak was from an infectious source and contacted the poison center (PC). The PC personnel performed a site evaluation, reviewed medical records obtained by the HD, and did a follow-up clinic visit with the victims. The tea had a common water source with the soda machine and was brewed from pre-packaged bags; leading to the belief that the tea was tampered with after it was brewed. The HD conducted a case control study and concluded that people who drank the iced tea were 65 times more likely to become ill than those that did not drink it (OR 65). Through the use of their syndromic surveillance system, they found the fifth victim who received care at a different hospital. Two samples of iced tea from the contaminated urn, and one control sample of tea from a previous urn, were sent to the Federal Bureau of Investigations (FBI) for analysis. The FBI found sodium azide in the first two samples but not in the control sample. Tests for arsenic and cyanide were negative. The biologic specimens from the patients were not tested. This is the second group of poisonings from sodium azide in the last year. Multiple employees at a renowned medical school were poisoned with coffee contaminated with sodium azide. Due to its ready availability on-line and profound symptomatology, some fear sodium azide could be used in a terrorist attack. Physicians should consider sodium azide as a possible etiology when multiple patients present from a common location with rapid-onset of syncope, nausea, and hypotension.

158. ENERGY DRINK EXPOSURES IN THE AMERICAN ASSOCIATION OF POISON CONTROL CENTERS' NATIONAL POISON DATA SYSTEM

Sara M. Seifter1, Judith L. Schaechter1, Eugene R. Hershorn1, Steven A. Seifter1, Alvin C. Bronstein3, Steven E. Lipshultz1

1Department of Pediatrics, University of Miami Miller School of Medicine, Miami FL USA; 2University of New Mexico School of Medicine and New Mexico Poison and Drug Information Center, Albuquerque NM USA; 3American Association of Poison Control Centers, Alexandria VA USA

Background: Energy drinks (ED) typically contain caffeine and other stimulants. Self-report surveys show they are consumed by 30-50% of 0-25 yos. The American Beverage Association states that “children and teens are not large consumers of energy drinks” and “total caffeine consumption from energy drinks among pre-teens is nearly zero” (http://www.amerivibe.org/news-media/news-releases-statements/more/237/). The American Association of Poison Control Centers recently created codes to track these exposures in the National Poison Data System (NPDS). Alcohol-containing products were banned by the FDA in 12/2010.

Methods: We retrospectively studied all human ED exposures reported to the NPDS from 10/1/10 - 2/28/11. ED were categorized as alcohol (A) or non-alcohol (NA) containing; NA, caffeine-containing (CC); NA, non-caffeine containing (NC); or NA, unknown formulations (UNK).

Results: There were 1,568 ED exposures: 1,466 NA/ED and 102 A/ED. 662 NA/ED cases were single product (SP) exposures: 492 (74%) CC; 8 (1%) NC; and 162 (24%) UNK. Of SP exposures, 25% were coded as resulting from “abuse” or “misuse”. SP/CC/ED exposures disproportionately involved those < 20 yos compared with other substance exposures in the NPDS (79% v 65%; p < 0.0001; OR 2.022; 95% CI 1.626 - 2.514). SP/CC/ED exposures were also disproportionately male compared with other substance exposures (63% v 49%; p < 0.0001; OR 1.777; 95% CI 1.480 - 2.134). Age groups in SP/CC/ED exposures were: Children < 6 yos = 47%; 6-12 yos = 13%; 13-19 yos = 19%; 20-25 yos = 12% and > 25 yos = 9%. Among SP/CC/ED exposures with a known outcome (n = 223), 38% had no-, 44% had mild-, 17% had moderate-, and 1% had major effects. Among all age groups, 13-19 yos had the highest proportion of moderate- or major effects (19%). Major effects included cardiac conduction/rhythm disturbances, hypertension and hyperthermia. There were no deaths. 93% of SP/NA/ED exposures were treated and released; 4% admitted to a critical care unit; and 3% admitted to a non-critical care unit. Effects duration was ≤ 24 hours in 98%. Benzodiazepines were the most commonly used specific therapy. A/ED exposures declined by almost 50% in the first two months following the FDA ban.

Conclusions: Reported exposures to energy drinks are disproportionally higher in males, and in young children, pre-teens, and adolescents compared with other toxic exposures in the NPDS. Toxicity most likely reflects the dose of caffeine and other stimulants in energy drinks.

159. HISTORICAL PREVALENCE OF MURDERS BY POISON FOUND ON SEARCH OF A NATIONAL NEWSPAPER OVER THE LAST CENTURY

Victoria R. Hall, Heather A. Borek, Christopher P. Holstege
University of Virginia School of Medicine, Charlottesville VA USA

Background: Throughout history poisons have been utilized to commit murder. We hypothesized that the number of incidents of murder by poisoning has decreased over the past century as reported in mainstream media and that trends in specific poison use for murder as reported in the media follows the timing of specific regulation of such agents.

Methods: The Historical New York Times (1900-2000), as a representative U.S. national newspaper, was searched using the Internet-based newspaper repository ProQuest. Searches were performed within ‘citation and abstract’ criteria of each article. An initial search using the terms “poison” and “murder” was performed for each decade and the total number of articles were recorded and graphed to demonstrate trends. Additionally, three representative poisons (arsenic, cyanide, and strychnine) were searched in association with the term “murder” as representative specific examples.

Results: The Results of the search “poison” and “murder” can be found in the chart below for each decade (416 total articles). There was a spike of 121articles found from 1921-1930 and a steady decline after 1930 to 2000. For arsenic, there was a peak in articles during the 1930s (39 total), with a steady decline from that time to less than 5 articles per decade over the past 5 decades. There were numerous murders reported due to cyanide from 1900-1920, followed by a significant drop in reports except for the decade of the 1980s when cyanide was utilized in a number of product tampering cases of over-the-counter medications. From 1900-1910 there were 17 articles pertaining to strychnine and murder; after 1910, there was a steady decline with only one reported article from 1950 to 2000 corresponding with the decreased use and regulation of strychnine in products.

Table 1.

<table>
<thead>
<tr>
<th>Decade</th>
<th>The New York Times Number of Article Found with Search “Poison” AND “Murder”</th>
</tr>
</thead>
<tbody>
<tr>
<td>1901-1910</td>
<td>72</td>
</tr>
<tr>
<td>1911-1920</td>
<td>60</td>
</tr>
<tr>
<td>1921-1930</td>
<td>121</td>
</tr>
<tr>
<td>1931-1940</td>
<td>99</td>
</tr>
<tr>
<td>1941-1950</td>
<td>20</td>
</tr>
<tr>
<td>1951-1960</td>
<td>12</td>
</tr>
<tr>
<td>1961-1970</td>
<td>4</td>
</tr>
<tr>
<td>1971-1980</td>
<td>10</td>
</tr>
<tr>
<td>1981-1990</td>
<td>11</td>
</tr>
<tr>
<td>1991-2000</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>416</td>
</tr>
</tbody>
</table>
Conclusions: There are significant historical observable trends in murders committed with the use of poisons spanning the last century as covered in the media, with a significant drop of such reports in the modern era. The trend data of specific poisons utilized in murder corresponds with a historical timeline of agent use regulation.

160. THEMES AND TRENDS IN SERIOUS INTENTIONAL SELF-POISONING

Erica E. Smolcic1, Kamal Sachdeva2, Amanda G. Cresswell2, J. W. Donovan3, Joseph J. Rasimas1
1 Penn State College of Medicine, Hershey PA USA; 2 Pinnacle Health Toxicology Center, Harrisburg PA USA

Background: Intentional self-poisoning is the most common method by which patients attempt to end their lives. Increases in availability of prescription medications are expected to alter overdose trends. The present study aimed to investigate the substances employed by and experiences of patients who come to treatment after self-poisoning.

Methods: A retrospective search of the Toxicology Investigators Consortium (ToxIC) Registry was performed to characterize patients with intentional exposures that led to care by a medical toxicologist. A one-year prospective study of an inpatient toxicology service was also conducted, categorizing patients by toxicologic diagnoses. This study gathered data on medications available to each patient (pharmacy records), their comorbidities (medical records), and courses of treatment.

Results: In the ToxIC Registry, 3032 (51.5 %) of the 5885 cases represented intentional exposures. Of those self-poisonings, the single class of medications involved in the highest percentage was over-the-counter analgesics (26.7 %). Sedatives and muscle relaxants are grouped together in the registry; they were involved in 19.2 % of cases. Antidepressants (17.1 %), opioids (9.4 %), antipsychotics (9.3 %), and anti-convulsants (5.6 %) were commonly ingested. Toxicity from ethanol was reported in 13.5 % of the registry’s self-poisonings. Prospective study of 1026 patients ranging in age from 2 to 89 years (51.9 % female) revealed 655 cases (63.8 %) of intentional self-poisoning. Of those patients, 382 (58.3 %) were female, and rates of comorbid conditions including obesity, chronic pain, and substance abuse were high. 74.8 % of self-poisonings involved at least one psychiatric medication, and 28.1 % involved a narcotic with a somatic indication (opioid analogues, muscle relaxant, etc.). Acute intoxication from ethanol and/or illicit substances was found in 16.6 % of cases. Only 49 cases (7.5 %) did not involve CNS-active substances—29 of those patients continued to have suicidal ideation after coming to medical care. After recovering neurocognitive function, most patients were not actively suicidal following self-poisoning with CNS-active compounds.

Conclusions: Patients choose agents for self-poisoning that are readily available. Over-the-counter drugs are still frequently ingested, but CNS-active medications are now more available and therefore used at high rates in overdose. Substances of abuse are also involved in many cases of self-harm. The study emphasizes that access to chemical dependency treatment, alternatives to medication and substance misuse, and careful prescribing and medication distribution practices are all serious unmet healthcare needs in North America.

SESSION III

169. MULTIPLE FATALITIES FOLLOWING INGESTION OF SEA TURTLE MEAT

Ronald L. Kirschner2, Kathy L. Jacobitz1
1 Nebraska Regional Poison Center, Omaha NE USA; 2 University of Nebraska Medical Center, Omaha NE USA

Background: Chelonitoxin is an uncommon food poisoning associated with ingestion of sea turtle meat. The etiology is thought to be biotoxin accumulation through the food chain, with no effect on the turtles themselves. We report multiple fatalities following consumption of hawksbill turtle (Eretmochelys imbricata) at a family gathering on a remote island in Micronesia.

Case Report: A healthy 21 year old man became nauseated several hours after ingesting turtle meat. The next day he had multiple episodes of emesis, but on post-ingestion (PI) day 2 felt well enough to go fishing. At sea he developed hematemesis. During evacuation he became restless and disoriented. At the hospital on PI day 3, he was noted to be confused with BP 120/70, heart rate 78, respiratory rate 24, and normal temperature. He complained of abdominal pain but the abdomen was soft. On neurologic exam he had hyperreflexia in all 4 extremities. Initial chemistries and CBC were normal. Treatment included IV fluids, promethazine, ranitidine, dexamethasone, mannitol (for possible sugitauria), diazepam, and ceftriaxone. The poison center was contacted by hospital providers. The patient became more obtunded, developing sustained clonus and upward plantar reflexes. Elevated liver enzymes (ALT 99 U/L, AST 152 U/L), amylase (756 U/L) and creatinine (1.5 mg/dL) were noted. He was intubated for unresponsiveness and respiratory distress. At family’s request he was extubated, and died on PI day 7.

A 22 year old man, brother of the first patient, had a similar course, and died on the same day.

Four children, ages 2–4, died prior to reaching the hospital. One 2 year old ate no turtle, but was breastfed by the mother who had consumed turtle meat. A second 2 year old ate a small amount of meat and was also breastfed. Altogether some 90 individuals were sickened and 6 died following this event.

Discussion: Sea turtle is a common food source in the outer Micronesian islands. Chelonitoxin has been associated with ingestion of hawksbill (E. imbricata) and green sea turtle (Chelonia mydas). Both species are found in tropical oceans worldwide. Nausea, vomiting, dysphagia, and abdominal pain are typical, with coma and multiorgan involvement seen in severe cases. Children appear to be more susceptible, and poisoning has been reported in breastfed children who did not consume turtle meat. Lyngbyatoxins from marine algae have been implicated in poisoning from C. mydas. Causative agents remain unclear for the carnivorous E. imbricata. Diagnosis is clinical and treatment is supportive care.

Conclusion: Ingestion of sea turtle meat is associated with an uncommon, but potentially fatal poisoning syndrome characterized by gastrointestinal and neurologic effects.

170. LONG-TERM FUNCTIONAL OUTCOME OF RATTLESNAKE ENVENOMATIONS TO THE HAND

Joshua N. Nogar1, Angela S. Wittuck2, Aaron B. Schneir3
1 University of California San Diego, San Diego CA USA; 2 Mesa Physical Therapy, San Diego CA USA

Background: Little information exists on long-term outcomes after rattlesnake envenomations (RSEs). RSEs often occur on the hand, a location that could potentially lead to long-term functional disability. Our aim was to analyze hand function at a time remote from the envenomation.

Methods: All admitted patients with hand RSEs at 3 hospitals were identified for a 10 year period. A minimum of 6 months was required between RSE and study evaluation. A chart review recorded various RSE characteristics. Those willing to participate who were >15 years old were evaluated with the Michigan Hand Function Questionnaire (MHQ), a validated subjective hand comparison test. Additionally, all participants were prospectively examined by a certified hand therapist blinded to the RSE location. Examination of both hands included: sensation (two-point tactile acuity in each fingertip), range of motion (ROM) (active flexion/extension of all finger joints and wrist), and strength (grip and pinch). Differences were considered to be significant if >20% decrement was identified between the envenomated hand and the control hand in the MHQ, as well as for the Objective parameters. When evaluating strength, we used >10% decrement to account for the normal variance that exists between dominant and non-dominant hands.

Results: 68 hand RSEs were identified. Nine patients have been evaluated with 2 exclusions for potentially confounding conditions that occurred in the control hand (carpal tunnel syndrome, cervical radiculopathy). Mean age at the time of envenomation was 33 years (SD 28.4) [range 1.25-72]; mean time from envenomation to evaluation was 50 months (33.9) [6-90]; mean quantity of FabAV was 12 vials (8.8) [4-24], and mean modified snakebite severity score was 2.6 out of 6 (2.89) [2-4]. Location of envenomation was index (2), middle (3), thumb (1), and dorsum of wrist (1). No patient had a significant difference between hands on the MHQ. A total of 4 patients had at least one significant difference on

Copyright © Informa Healthcare USA, Inc. 2011
Table 1.

<table>
<thead>
<tr>
<th>Patients With Significant Differences (N=4)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensation (hand composite)</td>
<td>2</td>
</tr>
<tr>
<td>Sensation (envenomated digit)</td>
<td>3</td>
</tr>
<tr>
<td>ROM (hand composite)</td>
<td>0</td>
</tr>
<tr>
<td>ROM (envenomated digit)</td>
<td>1</td>
</tr>
<tr>
<td>Strength (hand composite)</td>
<td>1</td>
</tr>
</tbody>
</table>

Objective parameters: one patient with 3, one patient with 2, and two patients with 1 [see Table 1]. The one patient with 3 differences was the only patient who had suffered tissue loss; he was skin-grafted on the thumb. Conclusion: Although some Objective differences were found on blinded Objective hand evaluation remote from the time of envenomation, no significant subjective functional differences were found. The study is limited by a small number of patients and potential selection bias.

171. INCREASED RISK OF CHRONIC KIDNEY DISEASE AMONG USERS OF NON-PRESCRIBED CHINESE HERBAL MEDICINE IN TAIWAN

Chuan-Fa Hsieh1, Song-Lih Huang2, Chien-Lung Chen1, Wei-Ta Chen1, Huan-Cheng Chang1, Ming-Ling Wu3, Chen-Chang Yang2
1Taiwan Landseed Hospital, Taoyuan County Taiwan; 2National Yang-Ming University, Taipei Taiwan; 3Taipei Veterans General Hospital, Taipei Taiwan

Background: Taiwan is a country with the highest prevalence and incidence of chronic kidney disease (CKD) and end-stage renal disease (ESRD) worldwide. Chinese herbal medicine (CHM) is commonly used in Taiwan and previous studies had linked the use of CHM with the occurrence of CKD/ESRD. The aim of this study was to better understand the association between specific types of CHM use and the risk of CKD.

Methods: We conducted a matched case-control study at the Landseed Hospital in Taoyuan, Taiwan from August 2006 through December 2009. Case patients were recruited from the outpatients of the Division of Nephrology who were aged 20 years and above, had a first-time diagnosis of CKD, and did not have cancer or specific pre-existing renal diseases. The controls were randomly selected outpatients from the same division who did not have CKD and were 2:1 matched to the cases by age (within 5 years), gender and date of outpatient visit. All cases and controls were interviewed by a trained research nurse using a structured questionnaire to obtain participants’ demographic data, personal medical history and regular use of CHM within past five years. Detailed laboratory and diagnostic data were obtained from the medical records. Conditional logistic regression models were used for statistical analysis.

Results: A total of 484 participants, including 214 cases and 270 controls, were eligible for final analysis. Their mean age was 65.7 years old. 37.8% of the cases reported having regular use of CHM, while 30% of CHM use was reported among the controls (p = 0.10). Notably, 23.4% of the cases took non-prescribed CHM, as compared to 6.7% among the controls (p < 0.001). Multivariate analysis found that illiteracy [adjusted odds ratio (OR) 4.7, 95% confidence interval (CI) 1.7-12.6], diabetes mellitus (OR 2.2, 95% CI 1.4-3.5), hypertension (OR 6.2, 95% CI 3.8-10.0), and use of non-prescribed CMH (OR 3.3, 95% CI 1.7-6.4) were associated with the risk of CKD; whereas having a habit of regular exercise was associated with a lower risk of CKD (OR 0.4, 95% CI 0.3-0.7).

Conclusions: We found that regular use of non-prescribed CHM rather than the use of CHM was associated with the development of CKD. This finding may have an important policy and clinical implication in future prevention of CKD in Taiwan.

172. PATTERNS OF ILLNESS AND HEMOLYSIS IN PATIENTS WITH ACUTE LOXOSCELES ENVENOMATION

Saralyn R. Williams, Daniel S. Smith, Donna L. Seger, John G. Benitez
Vanderbilt University, Nashville TN USA

Background: Brown recluse spider bites (BRSB) may result in systemic loxoscelism that is associated with fever, nausea, myalgias, and rashes in addition to the lesion at the bite site. Hemolysis is the life threatening complication of systemic loxoscelism. Current theories discuss a venom induced complement mediated pathway that results in destruction of erythrocytes. There are rare case reports of hemolysis occurring in a delayed fashion.

Methods: A 5 years retrospective review (2006-2010) of patients diagnosed with brown recluse spider bites was undertaken. Only cases of BRSB that were verified by direct examination by a medical toxicologist were included. Presumed BRSB were not included. Extracted data included demographics, signs and symptoms, clinical course, laboratory Results, number of transfusions, and outcome.

Results: There were 63 cases that were verified and included in the study. Average age was 27.6 years with a range of 1.1 years to 62.7 years. Of these, 35 were managed as outpatients although 10 of these manifested additional symptoms of systemic loxoscelism (fever, myalgias, nausea, or other rashes). There was one death in a 3.7 year old due to severe hemolysis < 24 hours after BRSB despite being given massive transfusion protocol. Thirteen patients were observed in the hospital and required no transfusions. One patient was observed in the intensive care unit due to severe facial swelling and airway concerns. Another patient had mild hemolysis within 48 hours of his BRSB but did not require transfusion. Of the 12 patients who required transfusions, early hemolysis (< 48 hours from BRSB) occurred in 4 patients, 3 of which were direct Coombs negative (one not done). One patient did not have hemolysis < 48 hrs but was readmitted with moderate hemolysis 5 days after BRSB. Direct Coombs was not done. The remaining 7 patients had delayed hemolysis (> 5 days), 5 of which had positive direct Coombs, 1 had negative direct Coomb’s, and one was not done. Although the sample size is very small, the 7 patients with delayed hemolysis were ages 15 and older.

Conclusions: While the majority of BRSB do not manifest hemolysis requiring transfusions, our pilot data suggests that there is a bimodal pattern to the moderate to severe hemolysis. Hemolysis that starts within 48 hours of envenomation may be due to a direct venom effect while the hemolysis that is delayed in onset may be more commonly due to an autoimmune process. Patients who are evaluated early in their course with systemic loxoscelism may not manifest the early hemolysis should have clear instructions for return if the hemolysis occurs as the delayed hemolysis can be severe.

173. CONTINUOUS INFUSION OF CROTALIDAE POLYVALENT FAB (OVINE) IN A PATIENT WITH RATTLESNAKE ENVENOMATION

Sean P. Bush1, Steven A. Seifert2, Sarah R. Pearl1
1Department of Emergency Medicine, Loma Linda University School of Medicine, Loma Linda CA USA; 2University of New Mexico School of Medicine, and New Mexico Poison and Drug Information Center, Albuquerque NM USA

Background: Since the introduction of CroFab (Crotalidae Polyvalent Immune Fab, Ovine, Protherics, UK; FabAV) in 2000, a phenomenon of recurrent, persistent or late, new onset hematologic abnormalities (primarily hypofibrinogenemia and/or thrombocytopenia), has been described. Hematologic abnormalities that occur post-hospital-discharge may lead to morbidity or mortality and the optimal strategy for managing such patients has yet to be determined. There are no published reports of continuous FabAV infusion. We describe a patient with severe hematologic effects treated with a continuous infusion of FabAV in an effort to prevent or lessen the severity of persistent/recurrent venom effects.

Case Report: A 16-year-old previously healthy male presented with a right distal lower extremity. He developed symptoms of systemic loxoscelism (fever, myalgias, nausea, or other rashes). The patient was treated with standard dosing of FabAV, as well as platelet, FFP and RBC transfusions until initial control was achieved and hematologic abnormalities corrected. The patient had normalization of the platelet count, fibrinogen and PT/INR, but had recurrent and persistent thrombocytopenia starting one day after treatment. He was placed on a continuous infusion of FabAV, initially at 2 vials per day, increased to 4 vials per day when the platelet trend remained downward. The platelet trend reversed, with
platelets normalizing on day 4, and the infusion of FabAV was stopped on day 8 without further recurrence.

**Case Discussion:** Persistent and recurrent hematologic effects following crotaline envenomation are thought to be due to recurrent venemism. Since only the circulating venom is apparently able to be neutralized, maintaining a continuous, antivenom serum concentration sufficient to neutralize venom in, and coming into, circulation is optimal. In the absence of the ability to measure venom and/or antivenom concentrations, titration of the antivenom infusions to clinical effect and periodic cessation and observation are required to determine the needed rate and duration of antivenom treatment.

**Conclusions:** A continuous IV infusion of FabAV, titrated to effect and maintained for 8 days post-envenomation, was associated with a good clinical outcome in a patient with recurrent and persistent thrombocytopenia.

174. MYOCARDIAL INFARCTION AND REFRACTORY THROMBOCYTOPENIA COMPLICATING RATTLESNAKE ENVENOMATION

Michael Levine1, Anne-Michelle Ruha1, Victor Sein2, Timothy Byrne2

1 Department of Medical Toxicology, Banner Good Samaritan Medical Center, Phoenix AZ USA; 2 Department of Cardiology, Banner Good Samaritan Medical Center, Phoenix AZ USA

**Background:** North American rattlesnake envenomation (RSE) commonly produces edema and hematologic toxicities. Myocardial infarction (MI) following RSE has not previously been described. The use of antplatelet or anticoagulant agents (AAA) in patients with RSE can be particularly problematic.

**Case Report:** A 68-year-old male with a history of hypercholesterolemia and cardiac stent placement 20 years prior, on no cardiac medications was bitten on the left wrist. He immediately developed subternal chest pressure with associated nausea. He was transported to an ED where he had anterolateral ST segment elevations, consistent with an acute MI. He received 6 vials of Crotalidae Polyvalent Immune Fab (FabAV). Due to concern for severe venom-induced thrombocytopenia and coagulopathy, he was transferred to a tertiary care facility without receiving any AAA.

Upon arrival in the cardiac catheterization suites he was chest pain free. Initial platelet count in the ED was then reported as 23 K/mm³ and there was no coagulopathy. After FabAV, platelets were 61 K/mm³. A high-grade occlusion involving the proximal to mid left anterior descending artery, with TIMI 2 flow, was found. He received 10,000 units heparin, 600 mg clopidogrel, and 325 mg aspirin during the catheterization. Following aspiration thrombectomy and placement of a drug-eluting stent, TIMI 3 flow and a left ventricular ejection fraction of 20-25% was noted. The troponin peaked at 129 ng/mL. Clopidogrel and aspirin were continued to prevent stent occlusion, but venom-induced thrombocytopenia was persistent refractory despite 76 vials FabAV. At discharge, 11 days post envenomation, his platelets increased to 51 K/mm³, from a post-admission nadir of 19 K/mm³.

**Discussion:** Thrombosis is not a typical feature of RSE. In fact, eptifibatide, a glycoprotein 2b/3a inhibitor, was originally derived from rattlesnake venom. Given the chest pain began almost immediately following the RSE and resolved prior to any pharmacologic intervention, we hypothesize that patient’s endogenous vascular plasminogen activator may have facilitated vessel opening, and the subsequent venom-induced thrombocytopenia may have been protective by preventing re-occlusion.

Despite the increased risk of bleeding with AAA, the use of such drugs in patients with cardiac stents is sometimes necessary. Active venom-induced thrombocytopenia as well as the potential for delayed onset of coagulopathy after treatment with FabAV makes management of RSE patients who require AAA especially challenging.

**Conclusion:** Acute MI complicating RSE has not been previously described. This case illustrates the potential challenges in managing RSE victims requiring AAA.

175. SUMMARY OF THE SAFETY ALERTS FROM THE FDA MEDWATCH PROGRAM CONCERNING DIETARY SUPPLEMENTS SINCE 2000

Brenna Farmer

Wen Cornell Medical College, New York NY USA

**Background:** The Dietary Supplement Health and Education Act of 1994 requires that supplement manufacturers ensure their supplements are safe for marketing. The FDA only takes action after an unsafe dietary supplement reaches the market. With the use of the voluntary reporting of possible harm or toxicities related to dietary supplements to the MedWatch program, the FDA has been able to issue multiple safety alerts. The Objective of this project is to summarize the FDA’s Safety Alerts related to dietary supplements.

**Methods:** Using the FDA.gov website, all Safety Alerts for Human Medical Products were examined by year from 2000 to present. Each year’s listed products were read for alerts related to dietary supplements. The name of the product and type of alert (unapproved/undeclared ingredient, toxicity, or drug interactions) were determined for each dietary supplement. For the alerts regarding unapproved/undeclared ingredients, the ingredient was listed.

**Results:** From 2000 to present, there have been 761 safety alerts (as listed on the FDA Safety Alerts for Human Medical Products website). Forty-four of those alerts are in regards to dietary supplements. Forty of those alerts are due to dietary supplements containing unapproved or undeclared ingredients, most often a form of prescription drug. The most common undeclared ingredients include: 21 alerts for PDE 5 inhibitors, 6 alerts for sibutramine, 5 alerts for anabolic steroids. Other alerts mention: glyburide, bumetanide, aromatase inhibitors, and 3 alerts due to “numerous prescription drugs” as undeclared ingredients. There have been 4 alerts related to aristocholic acid. Two reports have been related to possible poisoning due to lead or chromium with selenium.

**Conclusion:** While the FDA cannot require safety testing of dietary supplements prior to their reaching the market, they have been able to issue safety alerts for the general public based on voluntary after market reporting to the MedWatch program. Most often these safety alerts are for undeclared ingredients that can be harmful. As toxicologists, we must ensure that we do our part through reporting possible toxicities related to dietary supplements.

176. RECREATIONAL USE OF A BODY-BUILDING SUPPLEMENT RESULTING IN SEVERE CARDIOTOXICITY

Lauren Salinger1, Brock Daniels2, Bernard Sangalli1, Marc Bayer1

1University of CT Health Center, Farmington CT USA; 2 Yale New Haven Hospital, New Haven CT USA

**Background:** Performance enhancing supplements vary by composition and continue to gain in popularity, while little remains known as to their safety and effectiveness. We report a case of an adult male who developed significant cardiomyopathy temporally associated with the use of a popular bodybuilding product, advertised as the 2010 supplement award winner.

**Case Report:** A 24-year-old male presented to an emergency department one hour after ingesting Jack3D® which contains caffeine, arginine alpha-ketoglutarate, creatine, beta-alanine, schizandrol A and 1,3-dimethylamylamine (DMAAA). History was significant for an untreated viral illness during the previous month and negative for preexisting cardiac disease. On arrival, he complained of headache, palpitations, nausea/vomiting and chest pain. He was diaphoretic with a heart rate in the 130’s and a peak blood pressure as high as 180/100 mm Hg; there was no evidence of respiratory distress. After infusion of IV fluids, the patient decompensated requiring sedation and intubation and was transferred to a tertiary level center. A chest radiograph was suggestive of pulmonary edema. Additional therapy included nitroglycerin, electrolytes, antibiotics, antihypertensives, sedatives and diuretics. An echocardiogram revealed a severely increased left ventricular (LV) chamber, hypokinesis of basal segments with apical sparing and an ejection fraction (EF) of <20 %. A cardiac MRI confirmed LV hypokinesis and ruled-out an infective or infectious process. Two separate urine toxicology screens were negative for cocaine. The patient was extubated on hospital day (HD) 5; however his hospital course was prolonged due to “medical stress” delirium. On HD 14, he had improved cardiac function and was discharged. A follow-up echocardiogram at one month revealed near-normal EF of 55 %.

**Case Discussion:** The patient’s clinical presentation was consistent with a variant of Takotsubo cardiomyopathy, confirmed by echocardiography and MRI. Interestingly, the patient worked for the distributor of Jack3D® and reportedly spiked the DMAA concentration 1.5 times, for personal use, to achieve a more...
Background: Rattlesnake envenomation (RSE) is commonly associated with edema and hematologic toxicity, but tissue necrosis resulting in partial or entire digital amputation is uncommonly described and rarely seen in our practice. Of 57 patients with RSE who presented to our tertiary care hospital in 2010, 4 developed tissue necrosis resulting in the loss of a digit or portion of a digit.

Case Series:

Case 1: A 53-year-old man with tobacco and ethanol use, was bit to the base of the left thumb. He was given 8 v AV at 2.5 hr. He received 24 v AV for edema. 24 hr later the hand and thumb were tense with normal compartment pressures in the forearm. At 36 hr he had no sensation at the thumb tip and the thumb was cool with a small bleb at the bite site. Operative neurovascular decompression (NVD) and thenar fasciotomy resulted in improved sensation. Outcome: multiple surgical incision and debridement (I&D) procedures & bone graft over 16 weeks.

Case 2: A 54-year-old man with HTN, psoriasis and tobacco use; on nifedipine and etanercept; was bitten on the right middle finger (RMF). At 2 hr a blister was noted at the bite site. He received 6 v AV at 3 hr, and 24 v in total for edema, low platelets (Plt) and low fibrinogen (Fib). Large bullae developed over 36 hr, but color and sensation of the fingertip were normal. Outcome: I&D within 48 hr; excision and reconstruction of the volar digit at 33 days.

Case 3: A healthy, non-smoking 29-year-old man was bitten on the distal RMF. He enrolled in an antivenom RCT and received initial drug at 2 hr and a 2nd control dose at 5.5 hr for edema. Bullae developed over 24 hr to become necrotic, with decreased sensation at the fingertip but good capillary refill. Outcome: I&D at 30 hr; amputation at 10 days.

Case 4: A non-smoking 62-year-old man with HTN; on ASA, irbesartan and HCTZ; was bitten on the tip of the LIF. Within 2.5 hr the distal LIF became cyanotic. 6 v AV was given at 4 hr, with a total of 20 v for edema and coagulopathy. At 7 hr, the distal 2/3 of LIF was cyanotic, without capillary refill or sensation. Outcome: NVD with I&D of fingertip at 9 hr.

Case Discussion: Loss of a digit or portion of a digit is a recognized but rarely reported complication of RSE in the US. It is unknown how often necrosis necessitating amputation occurs or if any patient characteristics may predispose to this outcome. AV given after RSE has not been shown to treat or prevent tissue necrosis. In this series, there were no common factors identified other than sex and puncture site on the digit. AV was given early and all patients received above-average doses.

Conclusion: We describe 4 cases of RSE-related tissue necrosis leading to digital amputation or significant digit tissue loss. All patients were men with direct bites to the digit.

178. HEAVY METAL AND PESTICIDE CONTENT IN COMMONLY PRESCRIBED INDIVIDUAL RAW CHINESE HERBAL MEDICINES

Eric S. Harris1, Shugeng Cao1, Bruce A. Littlefield1, Jane A. Craycroft3, Robert Scholten1, Ted Kapchuk1, Yanling Fu2, Wenquan Wang2, Yong Liu2, Hubiao Chen1, Zhongzhen Zhao1, Jon Clardy1, Alan D. Woolf3, David M. Eisenberg1

1Hong Kong Baptist University, Hong Kong Hong Kong; 2Beijing University of Chinese Medicine, Beijing China; 3Harvard Medical School, Boston MA USA

Background: Heavy metal and pesticide contamination has previously been reported in Chinese Herbal Medicines (CHMs), in some cases at potentially toxic levels. The main Objectives of this study were to determine general patterns of heavy metal and pesticide contamination in a broad sample of raw CHMs and to provide an interpretation of the significance of contaminant levels.

Methods: Three hundred thirty four samples representing 126 species of commonly used CHMs were meticulously collected throughout China and examined for arsenic, cadmium, chromium, lead, and mercury. Of the total, 294 samples representing 112 species were also tested for 162 pesticide residues.

Results: At least 1 metal was detected in all 334 samples (100%). Pesticides were detected in 108 samples (36.7%). According to a likely scenario of how raw herbs are often consumed, only 2 samples (1%) with heavy metals and 12 samples (4%) with pesticides were found at concentrations that could contribute to elevated background levels of contaminant exposure. Wild collected plants had higher contaminant levels than cultivated samples.

Conclusions: Based on our assumptions of the likely mode of consumption of raw CHMs, the vast majority of the 334 samples in this study contained levels of heavy metals or pesticides that would be of negligible concern. However, more research and monitoring of heavy metals (especially cadmium and chromium) and pesticide residues (especially chlorpyrifos) in both wild and cultivated raw HM are advised.

179. RESPIRATORY FAILURE FOLLOWING CROTALEIN VENOM INGESTION

Thomas Arnold1, Mark L. Ryan1, Frank Humphries2, Partick McGauly2

1Louisiana Poison Center, Shreveport LA USA; 2LSU Health Sciences Center - Shreveport, Shreveport LA USA

Background: Pit vipers (subfamily Crotalinae) are responsible for most of the venomous snakebites in the USA, affecting approximately 9000 people every year and resulting in about 5 deaths. Typical reactions to venom injection may include nausea, vomiting, localized tissue edema, compartment syndrome, coagulopathy, thrombocytopenia. Other severe effects may include hypotension, cardio toxicity, and neurotoxicity. Very few reports of respiratory failure following a pit viper bite exist and no reports exist on the effects of snake venom ingestion.

Case Report: 40 year-old male patient was brought into the ED by helicopter following respiratory failure in the field. The patient was camping with friends when a second camper was bitten in the hand by a Copperhead snake. Our patient then attempted to suck out the poison orally as he had seen in movies. He attempted to spit out the venom after sucking on the wound. Several minutes later he began to have respiratory distress requiring pre-hospital intubation. On arrival to the ED the patient, without a history of asthma or emphysma, had diffuse wheezes throughout his lung fields and oxygen saturation near 85%. Chest X-ray demonstrated vascular congestion. CroFab was administered in the ED and continued in the intensive care unit for a total of 12 vials. During his treatment, he experienced hypotension requiring pressor support. The patient recovered rapidly after antivenom and was extubated within the first 24 hours. No evidence of coagulopathy was noted during the hospitalization. The patient was discharged on his second hospital day.

Case Discussion: Little is known regarding the effects of ingestion of crotaline venom. This patient presented with wheezing, possibly indicating a reactive airway process that required intubation and ventilator support. The rapid improvement of the patient within 24 hours suggests responsiveness to antivenom treatment.

Current snakebite treatment guidelines recommend against incision and suction, mainly for protection of the snakebite victim from further harm. This case highlights the potential risk to the first-aid provider applying the oral suction technique.

The original snakebite victim was discharged directly from the ED after being treated with antivenom while his “Good Samaritan” friend was admitted and cared for in the critical care unit.

Conclusion: We present a rare case of snake venom ingestion with subsequent respiratory failure.
BITE TO THE FACE

Carrie A. Truitt, Anne-Michelle Ruha
Department of Medical Toxicology, Banner Good Samaritan Medical Center,
Phoenix AZ USA

Background: Mithridatism describes the belief in acquired immunity to a poison after gradually self-administering small amounts of the same poison. Some snake enthusiasts believe in mithridatism with snake venom. We describe a patient with a rattlesnake bite who had attempted to self-immunize against the same snake's venom.

Case Report: A 44 year-old man was bitten on the thumb by his pet Western Diamondback rattlesnake. He had severe swelling that was treated with Crotalidae Polyvalent Immune Fab (AV). Upon discharge he began milking the snake’s venom and injecting it in his lower legs every few weeks to gain immunity in case of a future bite. Six months later he was bitten on the face by the same snake. He immediately injected more venom into the right lower leg (RLE). In the ED 45 min later, he had emesis, a puncture wound near each medial canthus, and facial edema with eyes swollen shut. A 2 cm erythematous and indurated lesion was present on the RLE at the site of venom self-injection. Progressive head and neck swelling necessitated intubation 2 hr after envenomation. Initial platelets were 27 K/mm³, PT peaked at 16.1 sec, and fibrinogen fell to a nadir of 174 mg/dL. A total of 26 v of AV were given. No leg edema developed. He was not extubated until day 6, due to combined effects of edema, aspiration pneumonia, and COPD. He left against medical advice after extubation. On follow-up visit 9 days after the bite he indicated a strong belief that the immunization worked because each time he had injected venom the local reaction was smaller, and because he had not died from the facial bite. He expressed intent to continue the injections. Photos document dramatically improved facial edema from presentation to follow-up.

Case Discussion: Successful immunization against snake venom was not achieved in this case. At least one animal study has demonstrated moderate effect of toxoid immunization in preventing serious venom toxicity. However, we found no reports of acquired immunity to snake venom in humans. In fact, a recent report described venom toxicity despite documentation of high titers of human IgG against venom from the same snake species.

Conclusion: Despite a previous systemic envenomation and repeated injections of venom from the same snake over 6 months, our patient was not protected from venom toxicity. Successful mithridatism has not been convincingly documented in humans.

181. WEEVER FISH ENVENOMATION - ANALYSIS OF ENQUIRIES TO THE UK NATIONAL POISONS INFORMATION SERVICE (NPIS) FROM 2004 - 2011

Eleri Thomas, Jeff Dyas, Channa V. Krishna, John P. Thompson
National Poisons Information Service (Cardiff), Cardiff and Vale University Health Board, Cardiff UK

Background: The Weever fish (Echichthys vipera) is one of very few aquatic creatures found around the coast of the UK that can cause significant envenomation likely to require attendance at hospital. Symptoms can include severe pain at the puncture site, dizziness, sweating, headache, chest pain and swelling of the affected limb.

Case series: Enquiries to the NPIS relating to Weever fish stings in the period April 2004 – April 2011 were analysed to examine the severity of symptoms reported and which treatments were recommended, particularly in relation to the time of the enquiry post exposure.

Discussion: Over this 7-year period, 45 enquiries were received with a high proportion, as expected, being stings to the feet. Sixteen enquiries (36%) were referred to NPIS within 24 hours of exposure, 12 of these (27%) being referred within the first 5 hours. Twenty enquiries (44%) were received more than one day following exposure, 9 of these within 5 days, a further 7 within 2 weeks and surprisingly 4 enquiries were received more than 3 weeks post exposure, one of these being after 10 weeks. Of the exposures referred within 24 hours, 6 patients complained of both pain and swelling at the site of envenomation, some referring to the pain as severe. Four patients reported pain alone and another 3 only swelling. A further 3 callers reported visible oedema of the affected limb. One patient complained of having difficulty breathing. Hot water immersion was recommended as treatment in 9 of these cases, the majority being patients who presented within 5 hours of exposure. The remainder received advice regarding appropriate analgesia and supportive care. Of those patients referred more than 24 hours post exposure, 7 were still experiencing pain at the site, 2 of these were 3 weeks after exposure. A further 8 still had swelling, one with an oedematous limb. Nausea, chest pain, headache, sweating, dizziness and skin reactions were also reported by some patients. Subcutaneous spines were evident in one patient two weeks after being stung. Treatment in these patients was generally supportive with appropriate analgesia, antihistamines and antibiotics.

Conclusions: Weever fish toxin is protein-based and readily denatured by hot water. The mainstay of treatment is to immerse the affected limb in hot water as soon as possible after envenomation and until the pain has eased. Appropriate antibiotic and analgesic treatment can be instigated at the same time. The high proportion of patients (44%) presenting well after the effective treatment interval yet still with appreciable pain and discomfort leads us to recommend that medical attention should be sought immediately after Weever fish envenomation.

182. DEATH DUE TO CROTALID ENVENOMATION IN A CHILD TREATED WITH POLYVALENT IMMUNE FAB

Adesi 1, Obafemi 1 , Melody Gardner 1 , Amy C. Young 2 , Ryan P. Morrissey 3
1North Texas Poison Center, Dallas TX USA; 2 UT Southwestern Medical School, Dallas TX USA; 3 Central Texas Poison Center at Scott & White, Temple TX USA

Background: Crotalid bite fatalities are rare in the US: about 10 deaths per 7000 bites annually. No pediatric deaths have been reported since the advent of crotaline Fab. We report the death of a 23 month old girl due to crotalid envenomation.

Case Report: A 23 month old girl screamed at a playground. Her parents found her near a small rattlesnake with blood on her left ankle. Within one minute she vomited, became unresponsive, and had generalized convulsions. EMS transported her to a pediatric hospital within two hours, receiving O2 and IV NS en route.

Initial VS were: BP, 106/85 mmHg; HR, 184/min; RR, 46/min; SpO2, 100% on room air. Initial laboratory findings included: WBC 33 k/mm³; Hct 22%; Plt 10 k/mm³; hemoysis on peripheral smear; INR 19; PTT > 300 s; fibrinogen < 70 mcg/ml; D-dimer > 22 mcg/ml; FDP 320 mcg/ml; ABG: 7.09/31/116/-19. IV crotaline Fab was begun 3 hours post bite. The patient received 6 vials as well as 4 units PRBC, 5 units plasma, 2 units platelets, and 10 units cryoprecipitate. In PICU at 3.5 hours post bite she received factor VII per hematology consultation. She became hypotensive and acidotic (pH 6.9), requiring resuscitation with multiple vasopressors. Poison control was consulted 4.5 hours post bite and advocated repeat doses of crotaline Fab. Prior to a second dose the patient became pulseless. She was pronounced dead less than five hours post bite.

Postmortem findings included: four fang marks anterior to the medial left malleolus; cutaneous and serous petechiae; and acute hemorrhagic pancreatitis and gastritis. The cause of death was listed as hemorrhagic diathesis due to snake bites.

Discussion: Pediatric death from snake envenomation is extremely rare in the US. We found no case reported in the literature since the advent of crotaline Fab. In this case the snake was not formally identified, but the history, examination and laboratory findings were most consistent with crotalid envenomation. The rapidity of systemic symptoms and clinical deterioration suggested envenomation directly into a blood vessel. The possibility of an anaphylactoid reaction could not be ruled out. Extended transportation time may have contributed to mortality, but it is unclear whether earlier treatment would have prevented death given the extreme presentation.
**Background:** The California Poison Control System (CPCS) notified county health authorities after an apparently new hazard was identified. CPCS received calls regarding four separate incidences involving Almendara quema grasa, a dieting aid manufactured in Mexico. Three adult females and one male toddler developed mild to moderate adverse effects after ingestion of seeds from this product. We report the clinical details of this emerging hazard, and the Results from three analytical Methods available to identify this agent.

**Methods:** This product is typically packaged as a “fat-burning almond” (“almendra quema grasa” in Spanish). After collating clinical data reported by local hospitals, (Table 1), our team recovered a specimen of the product in the form of seeds from a local “botanica,” a medical herbalist.

The agent was analyzed with three methodologies: digoxin immunoassay in patient serum samples, liquid chromatography-mass spectrometry (LC-MS) and LC-MS-TOF (time-of-flight) analyses performed directly on the plant material.

**Table 1.**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Symptoms</th>
<th>Peak Digoxin Level (ng/mL)</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (37 yo F)</td>
<td>VO, DI, HA, DZ, BR, PA</td>
<td>2.4</td>
<td>IVFs, KX</td>
</tr>
<tr>
<td>B (27 yo F)</td>
<td>VO, BR</td>
<td>0.6</td>
<td>IVFs</td>
</tr>
<tr>
<td>C (2 yo M)</td>
<td>VO, DH</td>
<td>0.5</td>
<td>IVFs, AE</td>
</tr>
<tr>
<td>D (24 yo F)</td>
<td>VO, BP</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>

**Key:** VO: vomiting; DI: diarrhea; HA: headaches; DZ: dizziness; BR: bradycardia; PA: palpitations; DH: dehydration; BP: back pain; IVF: intravenous fluids; KX: kayexalate; AE: antiemetics.

N/A: not applicable; this patient was lost to PCC followup after calling from home to report exposure.

**Results:** Serum digoxin levels in patients ranged from 0.5 to 2.4 ng/mL. LC-MS Analysis on the seed specimens confirmed the presence of peruvoside, a cardiac glycoside found in yellow oleander (*Thevetia peruviana*) plants. LCMS-TOF also detected several other compounds which appear to be cardenolides found in yellow oleander.

**Conclusions:** Poison control center surveillance can piece together disparate and isolated elements of a public poisoning hazard in order to avert further illness. In our case series, clinical presentations involved predominantly GI toxicity, although the digoxin levels measured in our patients had no correlation with illness severity. Lab Results initially confirmed the presence of cardiac glycosides from this new dietary supplement. Further analysis confirmed the presence of peruvoside, a cardenolide found in yellow oleander, and other related compounds. These findings suggest that different analytical Methods can yield different Results about the nature and quantity of herbal cardenolides. These issues should be considered when formulating diagnostic and therapeutic strategies involving herbal and plant toxins.

**Background:** Apple seeds (AS) contain cyanogenic glycosides (amylgdalin) which are a naturally occurring source of cyanide (CN). Unintentional ingestion of seeds of 1 or 2 apples is unlikely to result in CN toxicity. One reference states that ingestion of 50 AS may cause toxicity, while a cupful of AS has been reported to be fatal in an adult. We present a case of an intentional ingestion of 80-100 AS by an adult resulting in no toxicity.

**Case Report:** A 66-yr/o 50 kg, non-smoking female, with an unremarkable PMHx, presented to the ED 3 hours after thoroughly chewing and swallowing about 80-100 AS from 15 large Granny Smith apples on an empty stomach. She had read an internet post claiming cancer protection from regular AS consumption. Upon ED presentation, she was slightly anxious but otherwise asymptomatic. The temperature was 98.2°F, the blood pressure 151/105 mm Hg, the pulse 78 beats per minute, the respiratory rate 14 breaths per minute, and the oxygen saturation 98% on room air. The physical examination was otherwise normal.

The electrocardiogram was normal. Activated charcoal (AC) 50 gm with sorbitol, was given 4 hours post ingestion. An intravenous of normal saline was started and labs were drawn about 3.5 hours post ingestion.

**Results:** Venous pH 7.37, Na 138 meq/L, K 3.9 meq/L, Cl 102 meq/L, total CO2 27 mmol/l, AG 9, BUN 19 mg/dl, creatinine 0.79 mg/dl, glucose 98 mg/dl, and lactate 0.9 mmol/l. A stat CN level was < 20 ng/ml, using Conway diffusion and a colorimetric spectrophotometer (below reference range for non-smokers).

The patient was observed for 4 hours. No CN antidotes were administered. She left in good condition. A phone follow up revealed that the patient had experienced no subsequent adverse effects. Specimens of the AS received from the patient were analyzed for CN content.

**Case Discussion:** One prior study reported CN content of AS to be 0.61 mg/gm, as determined by absorbance spectrophotometry. Our one gram (23 AS) sample from the patient, analyzed by reflux-distillation followed by flow injection absorbance spectrophotometry analysis, yielded 0.410 mg/gm of total CN. Thus, the 80-100 AS ingested by this patient would contain 1.4-1.8 mg CN, which is low compared to the estimated lethal dose of CN in adults of approximately 50-300 mg. It is unlikely that the late GI decontamination performed in this case contributed to a benign outcome, since toxic symptoms of ingested cyanogenic plant material usually appear within 4 hours.

**Conclusions:** In this case, acute ingestion of up to 100 AS was a non-toxic exposure.

**185. SHOULD WE BE CONCERNED ABOUT THE INGESTION OF UNIDENTIFIED PLANTS?**

**Edward P. Krenzelok, Rita Mrvos**

**Pittsburgh Poison Center, Pittsburgh PA USA**

**Background:** Inquiries about plant exposures are common calls to poison information centers. The majority of inquiries involve the ingestion of a plant, the identity of which is generally known by a common or botanical name to the caller. However, a challenge to specialists in poison information (SPI) is the management of patients who have ingested plants that are unidentified. The purpose of this project was to investigate the morbidity and mortality associated with the ingestion of unidentified plants.

**Methods:** All plant ingestions (human) managed by SPI at AAPCC member poison information centers from 2000-2009 served as the database for this analysis. The database was provided by the AAPCC as a data grant. The data set included ingestions by month, age, reason and outcome. Descriptive statistics were used to characterize the Results.

**Results:** The database included 668,111 plant ingestion exposures. Unidentified plants accounted for 131,700 (19.7%) of all ingestion exposures. Ingestions by children less than six years of age were responsible for 81.6% of the exposures and 72.9% of all ingestions occurred during the seven month period of April-October. There were 127,072 (96.5%) unintentional ingestions and 3,091 (2.3%) were intentional. When the outcome was known, 78.3% had no effect and 19.0% had a minor effect. Moderate effect outcomes accounted for 2.6% of the ingestion exposures. There were 77 (0.2%) patients with major (life-threatening or disfiguring) effect outcomes—intentional 35.1%, 40.3% unintentional and 19.5% adverse reactions. Seven (0.005%) patients had fatal outcomes—three involved...
children less than age five (two were adverse reactions and one had a malignant etiology) and four fatalities occurred in adults over the age of 20.

**Conclusions:** The majority of unidentified plant ingestion exposures occurred in children less than six years of age. Uniformly, the exposures were associated with very low morbidity and mortality.

186. **THE TRUTH ABOUT COLLOIDAL SILVER PRODUCTS: A CASE OF SYSTEMIC ARGYRIA FROM SILVER THERAPY**

**Sophia Sheikh, Rizwan Riyaz, Brent Morgan**

**Background:** Colloidal silver products are widely available and marketed as safe alternative medications. Manufacturers of such products claim efficacy for major illnesses like AIDS without sufficient supporting evidence. Silver is a natural substance and not regulated by the FDA. Many such products are of unknown quality/effectiveness posing significant health risks to the public. Our case illustrates the dangers colloidal silver products pose to the public. We report a case of argyria from a silver colloidal product called NutraSilver®.

**Case Report:** A 49-year-old female came to our clinic in December of 2010 after developing permanent skin darkening while using NutraSilver® for treatment of Morgellons. She started NutraSilver®, product claiming composition of 0.36% colloidal silver, in January of 2008. Her initial oral dose of 20 drops three times/day (TID) was increased to 30 drops TID then 50 drops TID until her symptoms improved. Her skin discoloration began 6 months into treatment. She discontinued treatment in fall of 2009. She consumed estimated total of 3.7 mg of silver/day. Her exam revealed blue-silver discoloration of sun-exposed areas. Her plasma silver level was 72 mcg/L (ref. 5), blood silver 77 mcg/L (ref < 5), and spot urine for silver was undetectable. We purchased a bottle of NutraSilver® and sent it to Industrial Analytical Services, Inc in Leonminster, MA. Subsequent analysis revealed total silver of 3,754.88 ppm.

**Discussion:** Silver is widely used in colloidal form in alternative or holistic medicine practices. The term colloidal silver first used in 1861 to describe metallic silver nanoparticles suspended in liquid/gel. These suspensions did not use ionized forms of silver or silver complexes. However, today the term colloidal can be misleading from original meaning and simply refers to products containing silver. There is confusion over which forms of silver cause argyria. Many claim only ionic silver or silver salts cause argyria, however cases involving elemental silver exist. In 1999, FDA declared non-prescription colloidal silver products as unsafe. FDA later stated as silver is a natural substance it could be marketed as a dietary supplement. NutraSilver® is advertised as a supplement for many conditions and claims zero toxicity with a silver composition of 3,754.88 ppm. Our analysis revealed results of 3,754 ppm of silver.

**Conclusion:** Alternative supplements are popular and use of colloidal silver products is quite common. There is no cure for argyria. Prevention of this disorder is the focus and should include public awareness. Manufacturers of such products claim efficacy for serious conditions and claims zero toxicity with a silver composition of 3,754.88 ppm. Our analysis revealed results of 3,754 ppm of silver.

187. **DANGEROUS DIET: A CASE SERIES OF ALMENDRA QUEMA INGESTION**

**A. G. Cima, A. A. Monte, S. Banerji, A. C. Bronstein**

**Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA**

**Background:** AQG is marketed as a dietary supplement for weight loss sold in flea markets, swap meets and on the internet. The supplement contains the cardiac toxin yellow oleander (Thevetia peruviana). We report 2 cases of AQG ingestion, one acute and one chronic, leading to symptoms consistent with cardiac glycoside intoxication.

**Case 1:** A 41 year old male with no past medical history was taken to the emergency department (ED) with 12 hours of abdominal pain, nausea, and vomiting. He reported ingesting a whole “nut” of AQG 14 hours prior to arrival. The patient mistook the supplement as a snack nut. His wife had purchased the nut as a weight loss agent at a local swap meet. Upon arrival to the ED his heart rate (HR) was 26 bpm and blood pressure 97/67 mmHg. The ventricular escape rhythm converted with atropine to atrial fibrillation with a HR in the 80s. Laboratory analysis revealed a serum potassium of 7.3 mEq/L, a serum creatinine of 1.3 mg/dL and a serum digoxin concentration of 0.4 ng/mL. Third degree heart block and digitalis effect were present in ECG. HR again declined to the 40s. He was given 5 vials of Digoxin Fab without improvement of HR or BP. Following intravenous fluids, repeat potassium was 5.5 mEq/L. Serum troponin peaked at 0.11 ng/ml on hospital day (HD) 1. During hospitalization he required dopamine to maintain HR above 40 bpm. On HD 4 dopamine was discontinued and the heart rate remained 60-70 bpm. On HD 5 he was discharged in good condition with a normal ECG and echocardiogram.

**Case 2:** A 16 years old male with no past medical history presented to the ED with anxiety, dizziness, chest pain, and palpitations for 4 days prior to arrival. The patient reported taking AQG, obtained from a Mexican herbal shop, for 7 days as a weight loss measure. His vitals signs were 109/52 mmHg, 90 bpm, with respirations of 18 per minute. The last dose was two days prior to presentation. ECG showed normal sinus rhythm with scooping of the ST segments. The patient was treated with IV fluids, antiemetics, and lorazepam. Laboratory analysis revealed a digoxin level of 0.2 ng/mL and potassium of 3.4 mEq/L. The patient improved over 4 hours and was asymptomatic at the time of discharge.

**Discussion:** AQG contains yellow oleander. Acute or chronic ingestion can mimic digitalis poisoning. Exposure in both cases was confirmed by patient report, pictures of the product label, and measurable serum digoxin concentrations with no alternative reason for endogenous digoxin-like compounds to be present. Both patients experienced symptoms from the exposure.

**Conclusions:** AQG ingestion can lead to cardiotoxicity. Serum digoxin concentrations may be detectable. Digoxin Fab did not improve HR. Poison centers should be aware of toxicity due to this supplement.

188. **HAUNTING RETRIBUTION: ENVENOMATION BY A POSTMORTEM AGKISTRODON CONTORTRIX**

**Kirk L. Cumpston, Brandon K. Wills, Rutherford Rose**

1Virginia Commonwealth University Medical Center, Virginia Poison Center, Richmond VA USA; 2Virginia Commonwealth University Medical Center, Richmond VA USA

**Background:** Envenomation by dead rattlesnakes is well-known, however post-mortem Agkistrodon contortrix (northern copperhead) envenomation has not been reported. A. contortrix is a species that generally causes local tissue destruction without systemic effects as seen in rattlesnakes. We report a case of significant envenomation by a recently deceased A. contortrix.

**Case Report:** A 23 year old male was drinking beer outside and discovered a living A. contortrix. He quickly killed the snake and diced it into three pieces. While handling the head a fang inadvertently punctured the index finger of his right hand three times. At hospital presentation approximately one hour post-envenomation the hand was edematous to the proximal forearm and extremely painful. Four vials of Crotalidae Polyvalent Immune Fab (Crobat) were given, and an additional 4 vials were given due to progressive swelling and ecchymosis. Upon transfer to a tertiary care hospital the vital signs were: T 38.7 C, BP 130/69 mm Hg, HR 85, RR 22 and SpO2 96%. He received 2 additional vials of Crofab and continued hand elevation. On hospital day 2 swelling had decreased but a 4 cm hemorrhagic bulla developed over the proximal interphalangeal joint limiting his range of motion (ROM). Laboratory testing included normal blood counts and coagulation studies. His ROM improved and the patient was discharged home on hospital day 3 and was lost to follow-up.

**Case Discussion:** Like rattlesnakes, a deceased A. contortrix can deliver toxic amounts of venom causing significant local tissue destruction and functional morbidity. Many reports of post-mortem rattlesnake envenomation are due to reflexive bites from decapitated snake heads. This case did not appear to be a reflex envenomation. Because venom is still contained in the venom gland, requiring antivenin, after the demise of the snake. This snake should be avoided whether animate or inanimate.

189. **NO WONDERLAND FOR ALICE: A RARE CASE OF SEIZURES AFTER AMANITA MUSCARIA INGESTION**

**Patrick M. Whiteley**

1Toxikon Consortium, Chicago IL USA; 2Cork County Hospital (Stroger), Chicago IL USA; 3Illinois Poison Center, Chicago IL USA

**Background:** Amanita muscaria is a species that typically causes gastrointestinal illness, behavioral changes, and hallucinations, but rarely convulsions. The true death rate is unknown, but fatalities are rare. Ten cases of amanita ingestion in adults were found in the literature, involving convulsions in three cases and seizure activity in one case. We report a case of amanita muscaria ingestion with convulsions.

**Case Report:** A 36 year-old woman was involved in a car accident while ingesting mushrooms. She arrived to the emergency department (ED) approximately 1 hour after ingestion. She was alert, oriented, and fully responsive. Physical examination revealed normal vital signs and no focal neurological deficits. She was treated with IV fluids, lorazepam, and meperidine for nausea, vomiting, and agitation. She was discharged home from the ED after 2 hours of observation.

**Discussion:** Amanita muscaria ingestion can lead to seizures. Early recognition and treatment can prevent serious complications. Additional cases of amanita muscaria ingestion with convulsions are needed to better understand the clinical presentation and management of this rare condition.
Introduction: Amanita muscaria (fly agaric) is a muscimol- and ibotenic acid-containing mushroom, identified by its emblematic brilliant red or tan cap with white flecks. Although hallucinogenic effects are the primary goals of intentional ingestions, various symptoms from CNS somnolence to excitation can occur. Seizures in pediatric patients have been reported with large ingestions, but only one previous case of seizures in an adult was found in the English literature.

Case Details: A 61 year-old female with hypothyroidism presented to the Emergency Department seizing approximately three hours after frying and ingesting an unknown quantity of unidentified mushrooms she found growing in grass on the roadside in a Midwestern US state. Nausea and vomiting started immediately, followed by diarrhea, bowel incontinence, and tonic-clonic movements of the extremities. On EMS arrival, midazolam was given for generalized seizure activity.

Initial vital signs were: P: 68/min, BP: 92/63 mmHg, RR: 16/min, and T: 95.6°F. The patient was postictal, unresponsive, with marked salivation, and was consequently intubated and given activated charcoal. Neurologic exam was limited due to mental status. Further history revealed a medication list including naproxen, levothyroxine, and several homeopathic medications, although none were new or changed. Pertinent studies: CT brain: negative; EKG: normal sinus rhythm; serum toxicology: negative; urine toxicology: benzodiazepines positive; Chemistry: Na + 150, K + 2.9, CO2- 19, creatinine 0.7, glucose 99, calcium 6.3, AST 27, ALT 31. An EEG was unrevealing.

Photographs of the mushrooms taken by the husband were identified by Poison Center mycologists as Amanita muscaria. No subsequent seizures were observed. The patient was extubated on day 2 and discharged from the ICU on day 3 with normal mental status.

Discussion: Amanita muscaria is found throughout the Northern Hemisphere. Immature forms are occasionally mistaken for edible mushrooms. The two psychoactive ingredients muscimol (from decarboxylation of ibotenic acid) and ibotenic acid, bind GABA and glutamic acid, respectively. The opposing effects of these toxins can produce an unpredictable constellation of symptoms from somnolence and hallucinations to excitatory manifestations such as myoclonus, although the excitatory manifestations usually predominate in pediatrics. Serious toxicity in adults is uncommon. Supportive care is the mainstay of treatment. Recovery is usually complete within 24 hours. No antidote exists.

Conclusion: This represents a rare case of seizures in an adult following Amanita muscaria ingestion.

191. HUMAN POISONING AFTER INGESTION OF EGGS FROM A LONGNOSED GAR FISH (LEPIOSTOSEUS OSSEUS)

Edward Botti1, Gary LaFleur, Jr.2

1Iowa Statewide Poison Control Center, Sioux City IA; 2Nicholls State University, Thibodaux LA USA

Background: Many internet websites state that eggs from the gar fish (Lepisosteus species) are very toxic to humans. While a blog article in Field & Stream describes the effects on an Arkansas family who ate gar eggs, there is nothing in the recent scientific literature describing the clinical effects caused by gar egg ingestion.

Case Report: We managed a case of a husband and wife who ate cooked eggs from a longnosed gar fish that had been caught the previous day. The husband estimates he ate 30 teaspoons of eggs and the wife estimates she ate 3 teaspoons. Two hours post ingestion, both had the sudden onset of nausea, vomiting, diffuse abdominal pain, cramping and profuse watery diarrhea. Fish eggs were seen in the diarrhea. They presented to the ED 3 hours after the onset of symptoms. Neither had any other complaints in the ED. The husband’s admission vitals: HR 54 bpm, BP 112/74 mm Hg, RR 20 bpm, Temp 35.3°C. His admission labs were notable for BUN of 24 mg/dL and creatinine of 1.47 mg/dL. The wife’s admission vitals: HR 104 bpm, BP 108/88 mm Hg, RR 18 bpm, and Temp 36.4°C. Her admitting labs were unremarkable. Both were treated with IV fluids, pantoprazole and as-needed ondansetron. By the next morning, both had resolution of the nausea, vomiting, diarrhea, abdominal pain. The husband labs revealed BUN 20 mg/dL and creatinine 1.12 mg/dL. However, both patients’ calcium levels had fallen from admission: the husband’s from 9.5 to 8.0 mg/dL and the wife’s from 9.1 to 7.5 mg/dL. The two were discharged home on hospital day two.

Discussion: An extensive search of the National Library of Medicine’s PubMed failed to find any publications describing the effects of human consumption of gar eggs. The only English-language article found describing gar egg poisoning in humans was published in the North Western Medical Journal in 1851. The specific gar fish ichthyotoxin has not yet been identified, but is believed to be a protein. The toxin is hypothesized to affect calcium channels in neuro-muscular junction. Chickens have been reported to have died after being fed gar fish eggs. A study published in 1980 found that negative chronotropism and negative inotropism resulted when the toxin was applied to intact turtle hearts. This may account for the bradycardia seen in the husband, but this is purely speculative. Why the patients’ calcium levels fell is unknown but may be related to the possible effect of the toxin on the calcium channel.

Conclusion: This is probably the first case report in 160 years describing the clinical effects resulting from humans eating gar fish eggs. While this ingestion is apparently rare, there is much to be learned about the effects of gar fish ichthyotoxin on humans.

190. EARLY GI SYMPTOMS DO NOT PREDICT ANTIVENOM USE OR BITE SEVERITY IN RATTLESNAKE ENVENOMATION

Stephen L. Thornton1, K. Nguyen1, S. Shen1, C. Tomaszewski2, R. F. Clark1, F. L. Cantrell2

1UC San Diego Skaggs School of Pharmacy, San Diego CA; 2California Poison Control System, San Diego CA USA; 3Veteran’s Affairs Medical Center, San Diego CA USA; 4University of California - San Diego, San Diego CA USA

Background: North American rattlesnake envenomations are frequently cited to cause gastrointestinal (GI) symptoms. There is some thought that GI symptoms such as nausea, vomiting, and diarrhea may be indicators of systemic envenomation and bite severity. We sought to identify whether the presence of early GI symptoms, defined as occurring within 4 hours of the bite, could be used to predict antivenom use and/or bite severity.

Methods: We performed a retrospective review of a statewide poison system’s database for all cases of rattlesnake envenomation from January 2000-December 2009. Data collected included gender, age, location of bite, presence of nausea, vomiting, diarrhea, unusual taste, or oral paresthesia, antivenom use, and bite severity. GI symptoms were further classified as early (within 4 hours) or late. Bite severity was determined using the minimal-moderate-severe scoring system. This system was chosen over more Objective scoring systems because the inherent limitations of a retrospective review. Data was then analyzed with a Chi-square test to evaluate for statistically significant association between early GI symptoms and either antivenom use or bite severity.

Results: There were 2570 reported rattlesnake exposures in the database. Sixty-four (2.5%) of these had GI symptoms reported. Of these patients, 55 (86%) were older than 18 years of age and 49 were male (77%). A majority were bitten with the hand (56%, n = 36). Forty (62%) of the patients with GI symptoms developed them within 4 hours of the reported rattlesnake bite. A total of 50/64 patients (78%) were given antivenom. Six (9%) of the bites were evaluated as severe, while 30 (46%) were moderate and 28 (44%) were rated as minimal severity. Early GI symptoms were seen in 33/50 (66%) of those receiving antivenin versus 7/14 (50%) of those not receiving antivenom (p = 0.27). For severity, early GI symptoms were seen in 4/6 (66%) of the severe envenomation group, 20/30 (66%) of the moderate group, and 16/28 (57%) of the mild group (p = 0.43).

Conclusion: Gastrointestinal symptoms are rare after rattlesnake envenomations reported to a statewide poison center. In this retrospective study, the presence of early GI symptoms after a rattlesnake bite did not predict the use of antivenom or bite severity. Early GI symptoms may not be an accurate marker of systemic or serious envenomation.
192. NAJA KAOUTHIA ENVENOMATION

Robert L. Alunday, Anna M. Arroyo Plasencia
Washington University School of Medicine

Background: The American Association of Poison Control Centers reported 3652 total snake bites in 2009. With 41 exposures, known poisonous exotic snakes accounted for around 1% of reported bites. Because of the relative infrequency of exposure, antivenom specific to the exotic snake may not be immediately available. Here we report a case of envenomation by an exotic snake treated by antivenom obtained through use of the Antivenom Index.

Case Report: A 32-year-old male with a history of Bipolar disorder presented to a hospital after sustaining a bite to the base of his left index finger while handling his pet Naja kaouthia. Swelling at the bite site was noted on initial evaluation. The patient was transferred to a tertiary care center for further evaluation. On route, he became progressively tachypneic, developed angioedema of the tongue, and was emergently intubated. On arrival, vital signs demonstrated tachycardia and mild hypertension (HR 113, BP156/110). Laboratory data obtained 3 hours following the envenomation were: WBC 15.2; Hgb 14.4; Hct 41.7; Plt 181; Cr 1.03; AST 55; ALT 79; and ETOH 86 mg/dL. Continued facial edema with proximal progression of swelling from the bite site was noted. Toxicology was consulted and used the Antivenom Index to verify availability of antivenom at a local zoo. After consultation with the local zoo, Polyvalent Antivenom from South African Vaccine Producers (Pty) Ltd. was obtained. The patient received a total of 3 vials of antivenom. Left upper extremity edema progressed to the level of the elbow before improvement in swelling was noted. Facial angioedema improved early, and the patient was extubated on day 2. Minimal wound necrosis was noted on day 3. No signs of coagulopathy or rhabdomyolysis were seen.

Case Discussion: Here we present an envenomation by a nonindigenous snake, Naja kaouthia. In addition to significant swelling at the bite site, our patient developed respiratory distress with angioedema of the tongue. An allergic reaction caused by previous exposure to his snake’s venom may have contributed to the development of angioedema. The Antivenom Index was used to obtain antivenom from a local source. The patient did well during his hospitalization and was discharged with close follow-up of his wound.

Conclusion: We report an envenomation by a non-indigenous snake. The Antivenom Index was referenced to locate antivenom for Naja kaouthia. This case helps remind practitioners of the importance of the Antivenom Index when treating non-indigenous snakes.

193. ENERGY DRINK USE AND ADVERSE EFFECTS AMONG EMERGENCY DEPARTMENT PATIENTS

Sean Patrick Nordt1, Edward Castillo1, Melissa Galinao1, Vincent Nguyen1, Richard F. Clark2, F. Lee Cantrell3, Theodore Chan1, Gary M. Vilke1
1University of California, San Diego; 2California Poison Control System - San Diego Division; 3University of Southern California, Keck School of Medicine, Los Angeles CA USA

Background: The number and usage of energy drinks has increased over the past decade. These beverages contain caffeine or other stimulants. The objective of study is to evaluate demographics, prevalence, reasons and adverse effects with consuming energy beverages.

Methods: Patients greater or equal to 18 years of age utilizing emergency department (ED) services for any reason at a university hospital consented to participate in questionnaire about energy drink usage including frequency, reasons, adverse events and demographic data. Descriptive statistics utilized.

Results: 2158 patients participated, 860 answered "no" and not analyzed. 1298 answered "yes" to previous use, 52.6% male and 47.4% female. Ethnicity: Caucasian 48.3%, African American 17%, Hispanic 18%, Other 16.7%. Age ranges: 18-29 y (38.4%), 30-44 y (49.6%), greater or equal to 55 y (12%). Seventy-eight percent report use 0-1/wk, 11% 2-4/wk 6.6% 4-6/wk, greater than 7/wk 4.5%. Income levels: less than $20K 53.8%, $20-$40K 19.5%, $40-$80K 15.3%, greater than $80K 11.4%. Education levels: 39.7% high school or less, some college 34.9%, at least 4 year degree 25.4%. Reasons for use: 57% to “increase energy”, 9.5% for studying/work projects, 2.4% while prolonged driving, improve sports performance 2%, with ethanol 6.3%, “other” reasons 22.1%.

Adverse reactions reported in 33.5% (429) patients including: 280 report “shaky/jittery”, insomnia in 136, palpitations in 150, gastrointestinal upset in 82, headache in 68, chest pain in 39, impaired sexual performance in 7, seizures in 6, and “other” adverse effects in 97 patients. Eighty-five patients reported co-ingestion with illicit "stimulants" including cocaine, methamphetamine, MDMA.

Conclusion: Our data demonstrates that energy drink use crosses a number of age ranges and incomes. Of concern, we show one-third of patients report at least one adverse effect. Whilst most of these are not severe, a small number were serious e.g., seizures. In addition, some report purposely ingesting with illicit drugs. Further study needed to fully elucidate our findings.

194. AN AMANITA PHALLOIDES POISONING IN WASHINGTON STATE CONFIRMED BY POISON CENTER FIELD COLLECTION

Robert J. Freeman1, Curtis J. Elko1, Thomas G. Martin1, Joseph F. Ammirati2
1Washington Poison Center, Seattle WA USA; 2University of Washington, Seattle WA USA; 3University of Washington School of Pharmacy, Seattle WA USA

Background: Confirmed cases of Amanita phalloides poisoning are rare in Washington State, in part due to the sparse fruiting body distribution but also in the difficulty identifying the specific mushrooms involved. This report details a case of suspected amatoxin poisoning that was confirmed by the Washington Poison Center (WPC) and the local mycologists through a field collection and identification of the culprit, Amanita phalloides.

Case Report: The WPC was contacted by a hospital critical care about a 45 year old female with nausea, vomiting, diarrhea, and signs of hepatotoxicity. She had dined on 4 mushrooms that she had foraged from a local park and began having the symptoms after about 12 hours. She was initially treated at a local hospital with anti-emetics but returned when her symptoms continued. Her alanine aminotransferase (ALT) increased from 100 to 5600 over the subsequent 2 days and her international normalized ratio (INR) was 1.5. Before contacting the WPC she had received activated charcoal, N-acetylcysteine and penicillin. At the suggestion of the Poison Center, the patient subsequently received IV silibinin obtained through the current phase II clinical trial. Her liver panel had begun to resolve markedly before it was initiated. Before her discharge on day 8, the elevations of total bilirubin, ALT and INR resolved, although she continued to have abdominal pain and diarrhea. Before discharge the patient was able to explain which public park the mushrooms had been foraged. The pharmacy extern on rotation at the Poison Center collected specimens from the park, tentatively identifying them as Amanita phalloides. After confirmation of the identification by a mycologist at the University of Washington, the local county health department was contacted and a press release was issued to warn the public.

Case Discussion: Local mycologists have noted that 2010 was a prolific year for Amanita phalloides in the Seattle area. These mushrooms are non-native ectomycorrhizal fungi originally introduced into the US from strains in Europe. Horticulture is the likely vector aiding its spread up the west coast, but once established it may become endemic. The specimens in this case were smaller than average for this species and were found associated with young oak trees and bark mulch. The limited amount of material ingested likely contributed to this patient’s favorable outcome.

Conclusion: This Poison Center reports their first case in Washington State where a suspected amatoxin poisoning was confirmed through field collection and mycologist identification of the ingested mushroom, Amanita phalloides.

195. PROFUND HYPOKALEMIA AND WEAKNESS FOLLOWING CHRONIC LICORICE INGESTION FOR WEIGHT LOSS

David H. Jang1, Robert S. Hoffman1, Lewis S. Nelson1, Herbert Schaumburg2
1New York University School of Medicine, New York NY USA; 2Albert Einstein College of Medicine-Bronx, New York NY USA

Background: Chronic licorice ingestion is a rare cause of hypokalemia. The active metabolite of licorice is glycyrrhizic acid that may cause a metabolic syndrome similar to hyperaldosteronism. Glycyrrhizic acid inhibits 11-β-hydroxysteroid dehydrogenase resulting in high cortisol concentrations. This leads to hypertension, metabolic alkalosis, and hypokalemia. Licorice-induced hypokalemia usually has a mild progression but can be severe leading to profound weakness and paralysis.
While pediatric plant ingestions are frequently asymptomatic, we present a case of an unlikely ingestion which presented with classic symptoms of an ancient poison.

**Case Report:** A 4 year old boy, while on a walk with his family in an urban park, ate 4-5 handfuls of what his parents believed to be wild carrots. As they returned home during the ensuing 30 minutes, he was noted to have twitching in his legs, strabismus, and ptosis of his right eye. The poison center was contacted and recommended evaluation in an emergency department. On arrival, the patient was initially alert and oriented with stable vital signs (BP 81/63 mmHg, HR 96/min, RR 20/min). He was given activated charcoal and then developed tachycardia with a heart rate of 118/min. However, he became progressively tachycardic and developed bradycardia, increased oral secretions, and respiratory depression requiring endotracheal intubation and intravenous atropine (2 mg) administration 2 hours after ingestion. Initial laboratory evaluation, including serum electrolytes, hepatic transaminases, creatinine kinase and complete blood count, were normal. He was transferred to a tertiary facility where he remained hemodynamically stable and was extubated the following day. He was discharged within 48 hours of his ingestion without sequelae. A sample of the plant provided by the family was identified as poison hemlock.

**Conclusion:** Chronic licorice consumption is occasionally used as a dieting agent and should be considered in a patient who presents with unexplained hypokalemia.

**196. THE AILS OF SOCRATES IN MODERN DAY AMERICA**

*Jayne Santeflemé, Anna Plasencia*

1. Washington University School of Medicine; 2Indiana Poison Center

**Background:** While pediatric plant ingestions are frequent occurrences, the majority remain asymptomatic. Here, we present a case of an unlikely ingestion which presented with classic symptoms of an ancient poison.

**Case Report:** A 42-year-old male science teacher contacted the Poison Center after consuming 5 pods of fried milkweed for lunch. He prepared *Astragalus syriaca* prepared according to an online recipe. Similar exposures may occur after the consumption of innovative dishes available at progressive eateries. We present the case of a science teacher who had a therapeutic digoxin concentration after eating fried milkweed (*Asclepiadaceae syriaca*) prepared according to an online recipe.

**Case Report:** A 42-year-old male science teacher contacted the Poison Center after consuming 5 pods of fried milkweed for lunch. He prepared the milkweed according to a recipe he had discovered online while doing research for his class. After returning to work, a colleague alerted him to the possible dangers associated with consumption of the plant and he became alarmed. His AST/ALT remained in the 5,000–7,000 range for 18 days, and he displayed refractory acidemia and vasopressor–dependent hemodynamic instability; however, histamininitis eventually resolved while his mental status never recovered. He developed hemodialysis–dependent renal failure on HD #21. He was made comfort care only, and died following a gastrointestinal hemorrhage 43 days after ingestion.

**Conclusion:** We report a mycologist-confirmed case of fulminant *A. virosa* toxicity, which is uncommon in North America.

**197. SEVERE AMINITA VIROSA TOXICITY**

*Steven J. Walsh, Michael G. Abesamis, Anthony F. Pizon*

*University of Pittsburgh Medical Center, Pittsburgh PA USA*

**Background:** *Aminita virosa* toxicity is characterized by delayed gastrointestinal effects and hepatotoxicity. This poisoning is uncommon in North America. We describe a case of severe, prolonged, fatal *A. virosa* toxicity.

**Case:** Our case was a 53-year-old man with metastatic melanoma who presented to an outside facility with nausea and vomiting after eating mushrooms that he picked. His vital signs and physical examination were reportedly normal. AST and ALT were “elevated,” but symptoms resolved with intravenous (IV) fluids. He was given a dose of oral n-acetylcysteine (NAC) and provided with a prescription for additional doses before discharge home. 48 hours later, he re-presented with abdominal pain, nausea, and vomiting. His laboratory studies displayed AST 4,718 IU/L, ALT 5,454 IU/L, total bilirubin 2 mg/dL, and INR 5.4. Abdominal CT showed fatty liver without tumor burden. He was administered 150 mg/kg of IV NAC and transferred to our center.

Upon arrival to our facility, vital signs were normal save for minimal tachycardia. His laboratory studies displayed AST 4,178 IU/L, ALT 5,454 IU/L, total bilirubin 2 mg/dL, and INR 5.4. Abdominal CT showed fatty liver without tumor burden. He was administered 150 mg/kg of IV NAC and transferred to our center.

While admitted, he developed severe abdominal pain and anorexia. His coagulopathy worsened, requiring large amounts of fresh-frozen plasma and vitamin K. His mental status deteriorated, requiring intubation on hospital day (HD) #5. Head CT revealed cerebral edema but no mass. He was ruled ineligible for hepatic transplant secondary to his malignancy. His AST/ALT remained in the 5,000–7,000 range for 18 days, and he displayed refractory acidemia and vasopressor–dependent hemodynamic instability; however, his transaminitis eventually resolved while his mental status never recovered. He developed hemodialysis–dependent renal failure on HD #21. He was made comfort care only, and died following a gastrointestinal hemorrhage 43 days after ingestion.

**Conclusion:** We report a mycologist-confirmed case of fulminant *A. virosa* toxicity, which is uncommon in North America.
SAFFRON FOR THOSE OF BEAR’S GARLIC

Background: In spring more and more German people pick healthy and savoury plants themselves. Unfortunately, the young leaves of bear’s garlic resemble those of the poisonous lily of the valley and of the very toxic meadow saffron plant. Too many people carelessly trust the olfactory test which involves a high risk: Lingering on fingers, the garlic-like smell may be perceived erroneously from leaves of the lily of the valley or meadow saffron. Such mistakes have regularly resulted in poisoning accidents in spring, sometimes even with fatal outcome. Since 1990 we have documented 4 fatal cases, often with a typical history as follows: An elderly couple had picked the leaves for a natural salad, which they thought to be those of bear’s garlic. The woman ate a smaller amount of this salad than did her husband. About four hours after the meal, the couple noticed marked symptoms consisting of increasing episodes of vomiting and diarrhoea. Case histories: Husband: Due to persisting diarrhoea and severe deterioration of his general condition, the 70-year-old male had to be admitted to a hospital with marked signs of exsiccosis ca. 36 hours after the lunch. Within four hours, the patient’s condition deteriorated dramatically. He developed progressive cardiogenic pulmonary oedema refractory to therapy. Despite all available measures, the patient died ca. 64 hours after the ingestion. Wife: In contrast to her husband, manifestations in the wife came to a halt after one day. Together with her husband she was admitted to hospital with marked signs of exsiccosis 36 hours after ingestion. She revealed only mild leukocytosis and a slight increase in transaminases. She could be discharged on the following day.

Discussion: Colchicine poisoning could be confirmed based on the history, the symptomatology, the increase in blood levels and the presence of colchicine in the urine. The husband died from this poisoning ending with uncontrollable multiple organ failure. All measures of intensive medical treatment including an attempt of continuous secondary detoxification by means of haemodialysis remained unsuccessful. The poisoning was associated with a pronounced increase in D-dimer levels during the near-death period as a consequence of an activation of coagulation and the concomitant fibrinolysis. Details of pathophysiological mechanisms with regard to colchicine poisoning have not yet been fully elucidated.

DEET TOXICITY IN A NUDIST

Background: N,N-diethyl-m-toluamide (DEET) is a widely used insect repellent available in numerous topical preparations of varying strengths. It is poorly absorbed through intact skin, and is felt to be extremely safe with few reports of toxicity.

Case Report: 69 year old man who lives as a nudist in a cabin in the woods reported that he would spray his entire body liberally with 30% DEET containing insect repellent multiple times daily. Three weeks before presentation he stated he was having increasing weakness and nausea. On the day of incident he used a new bottle, but due to a faulty nozzle, the entire bottle discharged in a single spray onto his chest and back. He soon became ill with nausea, vomiting, and diarrhea. On presentation to the ED he was confused, short of breath and generally weak. Pulse 112, respiratory rate 22, Pulse Ox 94% on 2L nasal cannula, with pertinent labs including Lactate 6.5 mmol/L, CO2 18 mmol/L and Cr 2.3 mg/dL. Fluid rehydration was begun, but due to increasing weakness he was intubated and mechanically ventilated. He was briefly hemodialyzed for worsening renal insufficiency and lactic acidosis. GC-MS blood drug screen was positive only for DEET, and a subsequent blood DEET level of 130 ppb was obtained. He recovered after a prolonged hospital stay, without other cause for illness identified. No seizures were noted, and he made a complete neurological recovery.

Case Discussion: We report a novel case of DEET toxicity with altered mental status, weakness, and lactic acidosis. We hypothesize that the combination of multiple daily liberal sprayings of his entire body, along with an acute large dose secondary to spray can malfunction led to his illness. DEET toxicity has mostly involved children, where neurotoxic symptoms including confusion, agitation, and seizures predominate. Symptoms are generally self limited, though case reports of deterioration leading to respiratory failure and death have been reported. The mechanism of action of DEET neurotoxicity is poorly understood. Animal studies have suggested it may disrupt the blood-brain barrier, and induce neuronal apoptosis. Treatment involves only supportive care. The focus has been on prevention by limiting exposure through federal regulation of topical DEET concentrations, especially for children.

Conclusion: The patient had an acute on chronic DEET exposure with elevated levels, and no other reasonable explanation. Studies to determine safe DEET dosing are generally performed on intact skin of the forearm. Our patient as a nudist would spray his entire body including genitals multiple times daily, likely leading to toxicity.

BLOOD LEAD LEVEL ELEVATION FOLLOWING EXPLOSIVE CHARGE EXPOSURE IN BREACHERS

Background: Soldiers, law enforcement agents, and others are repeatedly exposed to blast events in the course of carrying out their duties during training and combat operations. Very little data exists on the effect of this exposure on the physiological function of the human body. Both military and law enforcement dynamic entry personnel, “Breachers”, began expressing sensitivity to the risk of injury as a result of multiple blast exposures. Breachers apply explosives as a means of gaining access to barricaded or hardened structures. These specialists can be exposed to as many as a dozen 0.3 to 10 pound lead-encased charges per day during training exercises, and even larger numbers per night during military operations. This observational study was performed by the Breacher Injury Consortium to determine the impact of short-term exposure to blasts by Breachers on whole blood lead levels (BLLs) and zinc protoporphyrin levels (ZPPPs).

Methods: Two 2-week Basic Breaching training classes were conducted by the United States Marine Corps’ Weapons Training Battalion Dynamic Entry School (USMC-WTB/DES). Each class included 14 students and up to 3 instructors, with 6 non-breaching Marines from the WTB serving as a control group. To evaluate for lead exposure, venous blood samples were acquired from study participants on the weekend prior and following training in the first training class, whereas the second training class had an additional level performed mid-training. No greater than 10 cc of blood were collected during each acquisition utilizing the collecting technique advocated by the CDC. Laboratory testing of samples included BLLs and ZPPPs. Testing was performed at the Mayo Medical Lab. BLLs and ZPPPs were measured in a whole-blood sample using the furnace atomic absorption method and hemato-furometer method respectively.

Results: Analysis of this blast injury data indicated students demonstrated significantly increased BLLs post-explosion (mean = 7 mcg/dL, SD 2.42, p < 0.001) compared to pre-training (mean = 3 mcg/dL, SD 1.60) and control subjects (mean = 3 mcg/dL, SD 2.73, p < 0.001). Instructors also demonstrated significantly increased BLLs post explosion (mean = 6 mcg/dL, SD 1.95, p < 0.02) compared to pre-training (mean = 3 mcg/dL, SD 1.14) and control subjects (mean = 3 mcg/dL, SD 2.73, p < 0.001). Student and instructor ZPPPs were not significantly different in post-train compared to pre-training or control groups.
Conclusions: The observation from this study that Breachers are at risk of mild increases in BLLs support the need for further investigation into the role of lead following repeated blast exposure with munitions encased in lead.

202. DRUG-ASSOCIATED HEAT ILLNESS

Michael Levine1, Anne-Michelle Ruha1, Frank LoVecchio1, Gregory Chu2, Pedro Roque3
1Department of Medical Toxicology, Banner Good Samaritan Medical Center, Phoenix AZ USA; 2Department of Critical Care Medicine, Banner Good Samaritan Medical Center, Phoenix AZ USA; 3Department of Emergency Medicine, Maricopa Medical Center, Phoenix AZ USA

Background: Previous case series of heat stroke suggest that xenobiotics which impair heat dissipation or increase endogenous heat production can predispose individuals to develop heat illness. Little is known if patients taking these medications have worse outcomes. The primary Objective is to determine if patients taking xenobiotics that impair heat dissipation or increase heat production experience more severe illness than those not taking such xenobiotics. A secondary Objective was to determine the overall prevalence of such xenobiotic use among heat stroke patients.

Methods: A retrospective chart review was performed for all patients discharged with a diagnosis of heat illness (ICD-9: 992) from two urban medical centers in Arizona over a 5 year period. Patients were excluded if bacteremia or presumed sepsis was present on admission. Patients in whom no medication history or no urine drug screen (UDS) were performed were excluded. Patients taking sympathomimetics, anticholinergics, diuretics, or neuroleptics (users) were compared to those not taking these agents (non-users) for outcome parameters of death and length of hospital stay. Data were abstracted onto a pre-designed data abstraction sheet following a brief training session and trial run. Descriptive statistics were used, with Student’s t test and Mann-Whitney U test performed as appropriate. Multivariate logistic regression was performed to adjust for confounding variables.

Results: 78 patients were identified, but neither a medication history nor a UDS was performed on 4 patients, leaving 74 patients for analysis. The mean (+/- SD) age was 50.2 (+/- 19.5) years. Nine subjects (12%) were female. Forty-three (55%) were non-users, and 31 (40%) were users. 6 (8%) subjects died. An additional 14/74 (19%) required rehabilitation or skilled nursing care upon discharge. The overall prevalence of xenobiotic utilization was 41.9% (31/74). The median length of stay (LOS) was 3.0 days for non-users and 9.0 days for users (p = 0.005). Age, initial temperature, initial mean arterial pressure, creatinine kinase, creatinine, white blood cell count, and prothrombin time were not predictive of a statistically significant increased LOS or mortality when corrected for multiple comparisons. Multiple logistic regression found the following variables to be associated with increased LOS: initial heart rate and need for mechanical ventilation (p = 0.018, 0.047 respectively).

Conclusion: The overall prevalence of pre-defined xenobiotic utilization was 41.9% in this series of heat stroke patients. Subjects using these xenobiotics experienced a nearly three-fold longer LOS without any associated increase in mortality.

203. A 2-YEAR OBSERVATIONAL STUDY ON ALUMINUM PHOSPHIDE POISONING IN MAZANDARAN: A COMMON TOXICITY IN THE NORTH PART OF IRAN

Nasrin Pakravan1, Aref Hoseinian1, Ali Reza Rafie2, Seyed Mehdi Feyz Baksh2
1Iman Teaching Hospital-Mazandaran University of Medical Science, Sari Iran; 2School of Medicine, Mazandaran University of Medical Science, Sari Iran

Background: aluminum phosphide (ALP) is highly effective insecticides and rodenticides used frequently to protect stored grain. Acute poisoning with this compound is common in some countries including India and Iran, and it is a serious health problem. Northern part of Iran is an agricultural area where they use ALP to protect their products, particularly rice.

Aim: The Objective of this study was to survey ALP poisoning and the outcome in a referral poisoning hospital in Mazandaran province, northern part of Iran.

Method: the study was a cross-sectional study from March 2007 to February 2008. Records of all patients admitted and hospitalized to a referral teaching hospital during 2-year period were collected. Information including gender, age, cause of toxicity, amount of ALP consumed (each tablet of ALP: 3g), route of exposure, time between exposure and hospital admission, signs and symptoms of toxicity at admission, therapeutic intervention, laboratory tests, and outcome were extracted from the patients’ notes. Patients who died and survived were compared using appropriate statistical tests. Data was presented as mean ± standard deviation (SD). p < 0.05 was considered as significant.

Results: during two-year period 102 patient, 46 men and 56 women, were admitted with ALP poisoning. The mean (± SD) age was 28.5 ± 12.4 y. The most common signs and symptoms at admission were nausea (79.4%), vomiting (76.5%), and abdominal pain (31.4%).

41.1% of patient showed metabolic acidosis in the arterial blood gas. The mean (± SD) number of ingested tablet and length of hospital stay was 1.5 ± 1.3 and 2.14 ± 5.02 days (min:1 day and max: 51 days), respectively. Suicidal intention was the most common cause of poisoning (97%) leading to 19 (18.6%) death. Comparing to patients who survived, death group had taken higher amount of ALP tablet (2.2 ± 2.4 vs. 1.4 ± 1.0, p < 0.05), had poor liver function test (p < 0.0001) and severe metabolic acidosis (pH: 7.17 ± 0.19 vs. 7.33 ± 0.08, p < 0.0001). More than 80% of patients who died had EKG abnormalities. Gastric washing with sodium bicarbonate, potassium permanganate, or coconut oil followed by charcoal and sorbitol was performed immediately after admission. Hemodynamic, acid-base, electrolytes and EKG abnormalities were treated conservatively for all patients as required.

Conclusion: ALP poisoning is a common toxicity in Iran causing high morality. This is a serious health problem in agricultural region where ALP is readily available. Withdrawal of aluminum phosphate tablet from the market and introduction of safer products as rodenticides and insecticides is recommended.

204. AMANITA PANTHERINA INGESTION RESULTING IN REFRACTORY STATUS EPILEPTICUS AND DEATH

Shin K. Kwon, Heather Ellsworth, Christian P. Lintner, Jon B. Cole
Hennepin Regional Poison Center, Minneapolis MN USA

Background: Amanita pantherina ingestions may cause a variety of effects, including sedation, hallucinations, agitation, and seizures, but fatal exposures are rare. Symptoms typically occur within 2 hours of ingestion but may be delayed for up to 6 hours. We report a case of A. pantherina ingestion resulting in refractory status epilepticus and death.

Case Report: A 68-year-old woman with a history of diabetes mellitus, hypertension, and chronic kidney disease (CKD) with baseline creatinine (Cr) 2.5 mg/dL presented to an Emergency Department with status epilepticus after consuming a mushroom she had foraged. The mushroom was later identified as A. pantherina by a mycologist. She was markedly hypertensive upon arrival with systolic BP’s between 230 and 240 mm Hg. Endotracheal intubation was performed and a propofol infusion started. Her initial labs were: sodium 140 mEq/L, bicarbonate 21 mEq/L, anion gap 8, glucose 84 mg/dL, and Cr 3.14 mg/dL. Her head CT was negative for acute pathology. Due to persistent hypertension, a nitroprusside drip was started. Electroencephalogram (EEG) monitoring confirmed continuous seizure activity. Her hypertension resolved and norepinephrine was required for persistent hypertension after hospital day (HD) 1. Despite administration of lorazepam, a therapeutic phenytoin concentration (14.7 mcg/mL), a therapeutic levetiracetam concentration (46.9 mcg/mL), and a slightly sub-therapeutic phenobarbital concentration (14.6 mcg/mL), the patient’s seizures persisted for 6 days, and life support was withdrawn on hospital day (HD) 7. She had no prior seizure history.

Case Discussion: A. pantherina does not contain amatoxin or other cyclopeptide toxins; rather it contains ibotenic acid and muscimol. Ibotenic acid has neurologic effects analogous to those of glutamate and may cause agitation, seizures, and delirium via D-aspartate (NMDA) receptor.
205. SERUM CONCENTRATIONS OF PERFLUOROALKYL CHEMICALS (PFC) IN FEMALES OF CHILDBEARING AGE IN THE GENERAL US POPULATION: 2007-8

Richard Wang, Lee-Yang Wong, Antonia Calafat
NCEH/CDC, Atlanta GA USA

Purpose: To characterize the serum concentrations of PFCs in non-pregnant females of childbearing age in the United States.

Method: In 2007-2008, National Health and Nutrition Examination Survey (NHANES) used a complex, stratified, multistage probability sampling design to provide a representative probability sample of the civilian, noninstitutionalized population of the United States based on age, gender, and race/ethnicity. Hispanic-Mexican American (MA), Non-Hispanic black (NHB), and Non-Hispanic white (NHW) were the major groups represented in the survey.

For the 2007-2008 survey, PFC concentrations were measured in 2100 participants aged 12 years and older who had been selected to represent the U.S. population for the survey. Sampling weights were created in NHANES to account for the complex survey design, including oversampling, survey non-response, and post-stratification.

Females between 15 years and 45 years participating in NHANES 2007-8 were identified and their demographic variables, pregnancy status, and serum concentrations of perfluoroalkyl chemicals were abstracted. LC/MS/MS was used to measure 11 PFCs in serum. For concentrations below the LOD, they were imputed by LOD/sqrt(2). Geometric means were calculated for PFCs with a detection frequency of at least 60% for the population.

Results: PFCs detected in at least 60% of females of childbearing age included Me-PFOSA-AcOH (66.4%), PFDoA (70.5%), PFHxS (99.5%), PFNA (99.2%), PFNA (100%), and PFOS (99.9%). Serum concentrations (mcg/L) of PFCs at the 50th percentile (95% CI) were Me-PFOSA-AcOH 0.3 (0.2-0.3), PFDoA 0.3 (0.2-0.3), PFHxS 0.16 (0.1-1.9), PFNA 1.4 (1.3-1.6), PFGA 4.3 (3.8-4.3), and PFOS 11.4 (10.5-13.0); and at the 95th percentile (95% CI) were Me-PFOSA-AcOH 1.3 (0.9-1.7), PFDoA 0.9 (0.6-1.3), PFHxS 10.0 (6.2-14.9), PFNA 4.1 (3.1-5.0), PFGA 9.1 (7.5-10.3), and PFOS 36.1 (30.9-39.7). The geometric mean serum concentrations (mcg/L) (95% CI) for Me-PFOSA-AcOH were 0.3 (0.2-0.3), PFDoA 0.3 (0.2-0.3), PFHxS 1.6 (1.4-1.9), PFNA 1.4 (1.3-1.6), PFGA 4.3 (3.8-4.3), and PFOS 11.4 (10.5-13.0); and at the 95th percentile (95% CI) were Me-PFOSA-AcOH 1.3 (0.9-1.7), PFDoA 0.9 (0.6-1.3), PFHxS 10.0 (6.2-14.9), PFNA 4.1 (3.1-5.0), PFGA 9.1 (7.5-10.3), and PFOS 36.1 (30.9-39.7). The geometric mean serum concentrations (mcg/L) (95% CI) for selected PFCs by race/ethnicity were as follows: for PFOA MA, 3.6 (3.3-3.9), NHB, 3.5 (3.1-3.9), NHW, 4.2 (3.9-4.5); and for Me-PFOSA-AcOH MA, 0.2 (0.2-0.3), NHB, 0.4 (0.3-0.5), NHW, 0.3 (0.3-0.4).

Conclusions: Females of childbearing age in the general U.S. population have a high prevalence of exposure to certain perfluoroalkyl chemicals based on serum concentrations for chemicals selected in this survey. Although differences in level of exposure to PFOA and Me-PFOSA-AcOH appear to exist among race/ethnicities for this population, additional data analyses are needed for confirmation. Further work is necessary to determine the human health effects at these exposure levels because they are not currently known.

206. THE STUDY OF EVOKED POTENTIALS IN ORGANOPHOSPHATE (DVP) INTOXICATION

Zeynep Kekeç, Filiz Koç
1Cukurova University Medical School, Adana Turkey; 2Cukurova University Medical School, Department of Emergency Medicine, Adana Turkey

Aim: Organophosphorous compounds used as insecticides widely cause many acute and chronic intoxications in man. Organophosphorous insecticides are neurotoxic poisons inhibiting acetylcholine esterase (AChE) activity in neuromuscular junction and secretar glands. Reported neurological findings include confusion, delirium, psychosis, headache, tremor, speech disorder, delay in cognitive processes, ataxia, generalized weakness, pyramidal signs, seizure, neuropathy, axonopathy and myelinopathy. In animal models, hearing disturbances had been reported associated with acute intoxication. In this report, visual (VEP), auditory (BAEP), and somatosensory (SEP) evoked potentials were studied in patients with acute-subacute organophosphorous intoxication.

Material and Methods: Evoked potentials were studied in 24 patients (12 men and 12 women at a mean age of 33.8 ± 17.4) diagnosed with organophosphorous intoxication (OPI). BAEP were studied in 22 patients VEP and SEP was studied in 24 patients.

Findings: Latency and/or amplitude asymmetry of VEP in 8, SEP in 7 and BAEP in 6 patients were found. In patients with VEP abnormality, 6 of them had latency asymmetry or prolonged latency and 2 had amplitude asymmetry. In 3 of SEPs, there was latency asymmetry and in 4 of them, there was amplitude asymmetry. In BAEP study, 4 patients had prolonged 5th wave and 2 had additionally first and third wave involvement.

Results and Discussion: In literature, electromyography, repetitive nerve stimulation and P300 studies have been found in patients with OPI, however, evoked potentials study could not be found. The pathological findings found in some of our patients suggest an involvement not only in periphery but also in visual, sensorial and auditory tracts in OPI presenting with general neurological findings.
Discussion: Analysis of changes in scores reveals some recovery in working memory skills that were presumably affected by neurotoxic effects of Hg. Improvements in problem-solving, sequencing, and inhibitory control were evident. Parents noted improvement in behavior and memory. DMSA lowered Hg total body burden.

Conclusion: An adolescent with mercury poisoning and neurotoxicity, treated with DMSA, showed resolution of some symptoms and cognitive function within two years after therapy.

208. EYE BURNS AFTER EXPOSURE TO CAR BATTERY ACID - A CATEGORIZATION OF THE CIRCUMSTANCES OF ACCIDENTS AND THE SEVERITY OF HEALTH IMPAIRMENT
Hahn Axel, Kathrin Begemann, Miriam Friedemann, Marita Gessner
Federal Institute for Risk Assessment (BfR), Berlin Germany

Objectives: German physicians have to give information about cases of poisoning (§ 16 Chemicals Act) to the BfR Centre for Documentation and Assessment of Poisonings (DocAssCenter Poisoning). Owing to a good cooperation with physicians, hospitals and German Poison Centres, the BfR also receives sufficient accident data. Based on a sample of well-documented cases of car battery accidents we implemented an accident categorization for eye burns due to exposure to car battery acid in the year 2001. To re-evaluate this new approach in the documentation of poisonings, we analyzed 1,118 cases of exposure to car battery acid in the period from 01/01/2001 to 31/12/2010.

Method: The reported cases were completed to a standard file, assessed by the BfR expert judgement together with the Poison Severity Score (PSS), collected and analyzed by the routine procedures of the DocAssCenter Poisoning system EVA (SAS). The mode of accident was assessed and categorized in defined groups. For 2001 we selected a sample of 100 out of 176 cases, for the re-evaluation the random sample consisted of 20 cases from the 2001 to 2010 period.

Results: Nearly all of the 1,118 cases were occupational poisonings. Most of them involved gas deflagration or explosions where acid originating from the batteries caused burns to the eyes and/or face. Even if the notifications did not always contain all the details of the course of accidents, we could again identify three frequent causes of accidents: 1. Electrical short circuits when inserting or removing batteries. 2. Sparking during recharging or when pushing batteries over floors/carpets and 3. Major jerking/shock, when batteries are put down roughly or dropped. The figures steadily went down from 176 cases in 2001 to 78 cases in 2010, maybe due to constructive measures discussed with the German industry (de-aeration holes were too small). In 78% of the cases the eyes were affected. The severity of symptoms and signs of eye exposure to acid splashes was classified as ‘minor’ in 91% and ‘moderate’ in 4%. Only in 3 cases most severe burns of the eye occurred such as corneal erosions, hyphaema, glaucoma, ‘fish eye’ etc.

Conclusion: The re-evaluation showed that an accident categorization in poisonings could give clear indications on the type of accident, possible preventive measures and the trend. The principle should be extended to further scenarios.

209. A CASE SERIES OF LEAD TOXICITY FROM ADOPTED CHINESE INFANTS
Brandon Warrick, Bram Dolcourt

Background: China is leading source for United States international adoption. There is increasing awareness of lead based pollution in China with significant exposure to children. Although pediatric lead poisoning is prevalent within China, this information is not readily available to adoptive US parents. We present a cluster of pediatric lead poisoning in recent US adoptees from a single adoption agency in from Jing Xi Provence in China.

Case Series: Of a group of nine children adopted in February 2011 with ages less than 13 months, we identified six children with lead intoxication. Two children were admitted to the hospital for chelation with blood lead levels (BLL) of 54 and 57 mcg/dL respectively, 2 had BLL between 30-40 mcg/dL, 3 had BLL between 5-30 mcg/dL, and 2 less than 5 mcg/dL. The 11 month old admitted to our facility for chelation (BLL 54 mcg/dL) presented with developmental delay, dense lead lines on long bone radiographs and anemia consistent with prolonged lead exposure. The child with a BLL of 57 mcg/dL has weakness of legs requiring physical therapy.

Discussion: The rapid industrialization of China and poor governmental oversight has led to pervasive environmental lead contamination. Chinese children are at significant risk for lead poisoning. Lead testing should be a routine part of post-adoption medical screening.

Conclusions: Adoption agencies should be more cognizant of potential medical risks and a take proactive action to improve safety in foreign orphanages. More research is needed to describe the scope and severity of the problem.

210. PHOSGENE EXPOSURE: A CASE REPORT
Anthony F. Pizon1, Lewis Hardison1, Edward Wright2, James M Turner3
1Charleston Area Medical Center, Charleston WV USA, 2Charleston Area Medical Center, Charleston WV USA, 3Charleston Area Medical Center, Charleston WV USA, 4University of Pittsburgh School of Medicine, Pittsburgh PA USA

Background: Phosgene is a rare exposure, but has strong clinical implications due to its potential use in terrorism. We report a phosgene exposure that resulted in the patient’s death.

Case Report: A 58 year-old man had phosgene gas blown into his face after a hose developed a leak. He was immediately decontaminated on site. A badge worn by the patient revealed an exposure >50, but <100 ppm*min. He arrived to the emergency department 1 hour after the exposure and only complained of a sore throat. Initial vital signs were blood pressure 175/118 mmHg, heart rate 98/min, respirations 12/min, and oxygen saturation of 93% on room air. Physical exam revealed few scattered rhonchi, but without signs of distress. His remaining exam was normal. Initial arterial blood gases (ABG’s) revealed pH 7.42, pCO2 43 mmHg, pO2 68 mmHg, HCO3 27 meq/L, and oxygen saturation of 93% on room air. Initial chest x-ray taken 2 hours after the exposure demonstrated clear lung fields. Approximately 2.5 hours after the exposure, he began complaining of dyspnea and restlessness, with oxygen saturation of 99% on 2 L oxygen by nasal cannula. His oxygen saturation began to drop below 90% 15 minutes later; he was more restless and began coughing. At this point he received nebulized albuterol, 1 gm IV methylprednisolone, and 100% oxygen via face mask. Minimal improvement was noted and he was intubated. The post intubation chest x-ray 3.5 hours after the exposure revealed diffuse alveolar infiltrates. One hour after intubation, repeat ABG’s revealed pH 7.19, pCO2 72 mmHg, pO2 67 mmHg, HCO3 26 meq/L, and oxygen saturation of 87% on 100% FiO2. Norepinephrine and vasopressin were initiated for hypotension. He required frequent endotracheal succioning. Changing to pressure control ventilation improved oxygenation only temporarily, and repeat ABG’s on this ventilation, approximately 11.5 hours post exposure, revealed pH 7.0, pCO2 71 mmHg, pO2 66 mmHg, HCO3 20 meq/L, and oxygen saturation of 83% on 100% FiO2. Acetylcysteine, terbutaline, and IV methylprednisolone were continued without improvement. Prone positioning was attempted, yet no improvement in oxygenation. Unfortunately his status declined very quickly. The patient died 30 hours after the exposure.

Case Discussion: There are many misunderstandings concerning phosgene due to its rare presentation. The human LD50 has been reported at 500 ppm/min and this patient was exposed to far less. Furthermore, traditional phosgene treatments were unsuccessful. Extracorporeal membrane oxygenation has not been studied with phosgene exposures and was not attempted in this case, but may be a good adjunct in severe exposures.

Conclusion: This case serves as a learning tool for toxicologists due to its unlikely presentation, but potential use in terrorism.

211. “OMG! GOT STNG BY SKRPION”: SCORPION ENVENOMATION IN TODAY’S IWORLD
Michael R. Christian1, Connie B. Fischbein2, Jenny J. Lu3
1The Toxikon Consortium, Chicago IL USA; 2Illinois Poison Center, Chicago IL USA; 3Cook County Hospital (Stroger), Chicago IL

Background: Envenomation by non-native scorpion species in the US is unlikely presentation, but potential use in terrorism.

A 58 year-old man had phosgene gas blown into his face after a hose developed a leak. He was immediately decontaminated on site. A badge worn by the patient revealed an exposure >50, but <100 ppm*min. He arrived to the emergency department 1 hour after the exposure and only complained of a sore throat. Initial vital signs were blood pressure 175/118 mmHg, heart rate 98/min, respirations 12/min, and oxygen saturation of 93% on room air. Physical exam revealed few scattered rhonchi, but without signs of distress. His remaining exam was normal. Initial arterial blood gases (ABG’s) revealed pH 7.42, pCO2 43 mmHg, pO2 68 mmHg, HCO3 27 meq/L, and oxygen saturation of 93% on room air. Initial chest x-ray taken 2 hours after the exposure demonstrated clear lung fields. Approximately 2.5 hours after the exposure, he began complaining of dyspnea and restlessness, with oxygen saturation of 99% on 2 L oxygen by nasal cannula. His oxygen saturation began to drop below 90% 15 minutes later; he was more restless and began coughing. At this point he received nebulized albuterol, 1 gm IV methylprednisolone, and 100% oxygen via face mask. Minimal improvement was noted and he was intubated. The post intubation chest x-ray 3.5 hours after the exposure revealed diffuse alveolar infiltrates. One hour after intubation, repeat ABG’s revealed pH 7.19, pCO2 72 mmHg, pO2 67 mmHg, HCO3 26 meq/L, and oxygen saturation of 87% on 100% FiO2. Norepinephrine and vasopressin were initiated for hypotension. He required frequent endotracheal succioning. Changing to pressure control ventilation improved oxygenation only temporarily, and repeat ABG’s on this ventilation, approximately 11.5 hours post exposure, revealed pH 7.0, pCO2 71 mmHg, pO2 66 mmHg, HCO3 20 meq/L, and oxygen saturation of 83% on 100% FiO2. Acetylcysteine, terbutaline, and IV methylprednisolone were continued without improvement. Prone positioning was attempted, yet no improvement in oxygenation. Unfortunately his status declined very quickly. The patient died 30 hours after the exposure.

Case Discussion: There are many misunderstandings concerning phosgene due to its rare presentation. The human LD50 has been reported at 500 ppm/min and this patient was exposed to far less. Furthermore, traditional phosgene treatments were unsuccessful. Extracorporeal membrane oxygenation has not been studied with phosgene exposures and was not attempted in this case, but may be a good adjunct in severe exposures.

Conclusion: This case serves as a learning tool for toxicologists due to its unlikely presentation, but potential use in terrorism.

Clinical Toxicology vol. 49 no. 6 2011

Clinical Toxicology Downloaded from informahealthcare.com by University of Zuerich on 09/15/11 For personal use only.
luggage. Additionally, toxicity varies widely among non-US scorpions, with some species having a primarily neurotoxic venom and others a hemotoxic venom. With advances in technology, arachnologist involvement for assistance with species identification and management is more feasible than ever.

**Case Report:** A 22 year-old man was stung on the right third digit by a scorpion while unloading a cargo ship from China. The patient captured the scorpion and brought it to a local emergency department. He initially complained of finger pain and numbness radiating to the axilla, along with a sensation of numbness in his throat. Initial exam revealed mild erythema and swelling extending 1cm from the site of the sting on dorsal side of distal interphalangeal joint. CBC, BMP, coagulation studies, and EKG were normal. A digital image of the captured scorpion was sent via text message to the consulting toxicologist, who forwarded the photograph to an arachnologist on staff at a large zoo. The scorpion was identified as a Chinese Golden Scorpion (*Mesobuthus martensii*), which has primarily a neurotoxic venom with an LD50 at 0.75mg/kg. This particular species is also noted to be aggressive and a good climber. The British Tarantula Society characterizes the venom as toxicity being close to that of the African Fat-Tailed Scorpion (*Androctonus amurensis*), which is considered one of the most toxic and medically significant species. The patient was admitted for observation and discharged the following morning.

**Case Discussion:** While envenomations from rare stowaway arachnids have been reported, the diagnosis and management of such envenomations have been revolutionized in today’s technology-ubiquitous society. Real-time transmission of photographs to arachnologists located anywhere remotely can provide quick species identification to help guide symptom-directed treatment and facilitate prudent disposition decisions.

**Conclusions:** We describe an exotic scorpion envenomation in the US where rapid species identification through real-time image transmission provided clinically useful information.

### 212. DROPPING ACID AT SCHOOL: HCl EXPOSURES IN CHEMISTRY CLASS

**Sean M. Bryant, Steven E. Aks**

**Toxikon Consortium, Cook County Hospital (Stroger), Illinois Poison Center, Chicago IL**

**Background:** Injuries secondary to HCl may vary in several ways. The purpose of this study is to report 9 years of our regional poison center’s (RPCs) experience with HCl exposures occurring in school chemistry classes.

**Methods:** All HCl exposures from 1/1/2002 through 12/31/2010 were retrospectively searched from our RPC database. Cases meeting inclusion criteria were those exposures occurring in the context of a chemistry class or experiment (grades 1-12). Demographic data, identity of the caller to the RPC, the type and context of exposure, whether or not the patient was referred to a health care professional, therapy recommended, and outcome were recorded.

**Results:** Over the course of 9 years, a total of 830 cases of HCl exposures were coded by our RPC with 127 (15.3%) taking place during a chemistry class. The majority occurred in females (63%) with mean age being 16 years (median 15 years; range 11-68 years). Three adult teachers aged 27, 44, and 68-years-old were included in the series. A school nurse was the most frequent caller (87%). Others included a parent (8), nanny (1), family physician (1), pharmacist (1), & unknown (3). All exposures were accidental except one malicious event involved a chemistry class mishap. These exposures were generally benign, rarely required referral, and commonly treated with dilution or fresh air alone. Poison centers may provide a valuable resource regarding on site management of chemistry class toxic exposures.

**Conclusion:** School chemistry classes are defined settings of HCl exposure. Over 15% of HCl exposures reported to our RPC were secondary to chemistry class mishaps. These exposures were generally benign, rarely required referral, and commonly treated with dilution or fresh air alone. Poison centers may provide a valuable resource regarding on site management of chemistry class toxic exposures.

### 213. PRAIRIE RATTLESNAKE ENVENOMATION: A POISON CENTER’S 5-YEAR EXPERIENCE

**Kirk A. Hughes, Heather Ellsworth, Shin K. Kwon, Christian P. Lintner, Jon B. Cole, Daniel E. Keyler**

**Hennepin Regional Poison Center, Minneapolis MN USA**

**Background:** *Crotalus viridis viridis*, commonly known as the Prairie Rattlesnake (PR), is responsible for nearly all *Crotalus* envenomations in the Northern Great Plains of the United States. A paucity of literature exists regarding PR envenomations. A review of data from a single poison center (PC) is presented to augment current knowledge.

**Methods:** A retrospective review of all cases of humans envenominated by PRs reported to a PC from 2006 to 2010 was performed. Data recorded included age, site of bite, clinical effects (local and systemic), CroFab® administration and duration of hospitalization.

**Results:** 45 total cases of PR exposures were identified with 39 included in the final analysis. 6 cases were excluded due to inconsistent data. All patients survived. Twelve (31%) patients were pediatric (ages 4-13 y), and 27 (69%) were adults (ages 18-76 y). The site of the bite was the upper extremity in 12 cases (31%) and the lower extremity in 27 cases (69%). Isolated local effects were described in 10 patients (20%). Commonly reported local effects included edema (n = 32, 82%), pain (n = 24, 62%), ecchymosis (n = 19, 49%), erythema (n = 10, 26%), paresthesias (n = 2), and bullae (n = 2). Systemic effects were reported in 29 patients (74%). The most commonly reported systemic effects included thrombocytopenia (n = 10, 26%), hypofibrinogenemia (n = 8, 20%), hypotension (n = 7, 18%), and gastrointestinal effects such as vomiting (n = 6) and nausea (n = 4). Additional systemic effects included coagulopathy (n = 4), tachycardia (n = 4), peri-oral paresthesias (n = 5), and significant airway associated complications such as oropharyngeal swelling (n = 4), tongue swelling (n = 3), lip edema (n = 1), and angioedema (n = 1). Intubation was required in 6 patients (15%). Diaphoresis (n = 3), fecal incontinence (n = 1), diarrhea (n = 1), and rash/hives (n = 1) were also observed. CroFab® was administered to 33 patients (85%); the average number of CroFab® vials administered per patient was 14 (range 3-30). Thirty-six patients (92%) were admitted to the hospital with an average stay of 4.6 days.

**Conclusion:** Similar to other *Crotalus* species, Prairie rattlesnake envenomations result in hematological disturbances. However, the risk of airway compromise from oropharyngeal swelling appears to be an important consideration in the envenomation profile and subsequent treatment. This case series of PR envenomations demonstrates that significant morbidity may occur but mortality is rare. This is consistent with the relative mid-strength LD₅₀ of Prairie rattlesnake venom of 2.1 mg/kg as compared to other *Crotalus* species such as the Mojave (*Crotalus scutulatus*, 0.24 mg/kg LD₅₀) and Western diamondback (*Crotalus atrox*, 5.0 mg/kg LD₅₀).

### 214. ACRODYNIA: THREE ADOLESCENT BROTHERS WITH SEVERE INORGANIC MERCURY INTOXICATION

**Joshua Canning, Steven Curry**

**Banner Good Samaritan Medical Center, Phoenix AZ USA**

**Background:** Inorganic mercury intoxication may present with personality changes, weight loss, hypertension, neuropa thy, and desquamation of the extremities. Acrodynia within the same family has been rarely described.

**Case Series:** Two brothers (14 & 15 years old) living in the same home presented with 4-week history of 15 lb weight loss, rashes, night sweats, arthralgias, weakness, and severe neuropathic pain. On admission, both boys were irritable and erethismic, tachycardic, and significantly hypertensive, requiring 3-drug anti-hypertensive therapy. Examination revealed muscle wasting, desquamation and erythema of the palms and soles as well as a fine maculopapular rash on the trunk.

---

**NACCT Abstracts 585**

*Clinical Toxicology* Downloaded from informahealthcare.com by University of Zuerich on 09/15/11

Copyright © Informa Healthcare USA, Inc. 2011
Laboratory studies were remarkable for serum creatine kinase of 860 IU/L in one boy. Extensive infectious and autoimmune work-up was negative. EMG and nerve conduction studies in one boy demonstrated acute, axonal, length-dependent motor neuropathy. Acute Hg toxicity was considered and confirmed with 24 hour urinary Hg collection (30.6 and 17.4 mcg Hg/g Cr). Further history revealed that 3 months prior to admission the boys had found a small glass vial of liquid Hg outside their home that they broke open and possibly heated inside the bedroom they shared. Further inquiry led to hospital admission of a 3rd brother (11 years old) from the same household who had similar but less severe toxicity and elevated urine Hg excretion. All three boys underwent chelation with a 14-day course of 30 mg/kg/day DMSA and demonstrated an expected post-challenge increase in 24hr urinary Hg excretion to as high as 89 mcg Hg/g Cr. Two evaluations of the home using real-time air Hg monitors by toxicologists, hazardous materials experts, and public health personnel 3.5 months after the boys had “lost” the Hg in their bedroom failed to detect remaining Hg. The boys were discharged on antihypertensives and opiates for severe pain. Repeated 24hr Hg collections 10 days after completion of chelation therapy showed <10mcg Hg/g Cr in two boys and 23.7mcg Hg/g Cr in the most severely affected child. On follow-up 4 weeks later, all boys were significantly improved.

Discussion: Acroderma may be easily mistaken for a variety of infectious, endocrine, nutritional and toxicologic disorders. It appears best explained as a hypersensitivity syndrome with some sort of genetic disposition, since only a minority of individuals develop the illness after Hg exposure, and almost all cases are in children.

Conclusion: While much less common today, this case series illustrates that acroderma should still be included on the differential of any child presenting with a clinical picture suggestive of pheochromocytoma, neuropathy, personality changes or dermatologic complaints.

215. SILVER-MERCURY AMALGAM DENTAL FILLINGS EXPOSE PATIENTS TO HIGH LEVELS OF MERCURY VAPOR
Don Robbins, Kathleen Boyle

Introduction: Dental amalgam fillings are alloys, containing 50% mercury (Hg). In the mouth, these alloys corrode and are exposed to heat, acids, and abrasion from eating, chewing, and bruxism. Hg fillings expose patients to Hg vapor continuously.

Objective: Quantitate oral levels of Hg vapor released from Hg dental fillings before and after chewing for 3 min and after removal of Hg dental fillings.

Methods: All patients provided consent for treatment each time they visited this dental practice; demographic information, number of Hg fillings (surfaces), and cumulative readings, a 4-fold increase compared to resting. Removal of Hg fillings resulted in an immediate and sustained decrease in oral Hg levels to <0.08mcg/m³ resting (p<0.001) and <0.15 post-chew (p<0.001).

Results: Data collected between Sept 2004 and March 2011 from >1000 patients were analyzed. Number of Hg surfaces ranged from 0-42; 37% of patients had ≥3 oral Hg levels recorded. Resting levels of oral Hg vapor increased at a rate of 0.73 mcg/m³ per surface for initial and cumulative readings. Post-chew levels increased at a rate of 3.3 mcg/m³ per surface for initial and cumulative readings, a 4-fold increase compared to resting. Removal of Hg fillings resulted in an immediate and sustained decrease in oral Hg levels to <0.08mcg/m³ resting (p<0.001) and <0.15 post-chew (p<0.001). Inclusion of fish in the diet during the previous week did not affect oral Hg levels.

Conclusions: Patients with dental fillings are constantly exposed to Hg vapor levels which far exceed OSHA/EPA maximum safe levels for work or residential settings. Hg vapor readily accesses all body tissues, gaining access via circulation to all organs and the brain. While the FDA considers reclassifying Hg amalgams, patients and healthcare providers need to be made aware of the extent of exposure to Hg caused by Hg dental amalgam and healthcare providers need to consider Hg toxicity when evaluating patients.

216. LABORATORY VARIATION IN REPORTING OF URINE ARSENIC LEVELS
Evan S. Schwarz, Paul M. Wax, Kurt C. Kleinschmidt
University of Texas Southwestern, Dallas TX USA

Heavy metal testing for arsenic is ordered on patients for many reasons. Misinterpretation of the testing

Results: In misdiagnosis and unnecessary emotional stress. Laboratories utilize different methodologies in their testing and report their results in a variety of ways. As such, tests can be difficult to interpret.

An internet search was conducted to find any laboratory in the United States that offered 24 hour urinary arsenic testing. Any laboratory that was found, in addition to any laboratory known by one of the authors that offered the appropriate testing, was contacted and surveyed. The survey instrument was made by the authors and reviewed by colleagues before it was used. The instrument was then trialied at a few laboratories for clarity before being sent to the rest of the eligible laboratories. Laboratories that did not initially respond were sent another copy of the survey and then called. Technicians from one laboratory were at a conference attended by one of the authors and were personally surveyed.

A total of 23 laboratories were found. Of those 23, only eight conducted their own testing. The other 15 outsourced the testing to one of the eight laboratories. Of those eight, four completed the survey; one laboratory answered a few questions over the phone. Only three of the laboratories speciated their Results. The laboratories were not consistent regarding which species were classified as organic versus inorganic arsenicals. Two laboratories measured total arsenic but did not do their own speciation; neither reflexively offered speciation if the testing was positive for arsenic. If speciation was required, it was outsourced to one of the three laboratories that offered speciation. The methodologies used by the laboratories were also different.

Few laboratories offer urinary arsenic testing. Of those that do, very few laboratories perform their own testing. There is a large amount of inter-laboratory variation in how the results are reported. Comparing results from different laboratories can be very challenging and difficult to interpret.

217. INTRAPERITONEAL ELEMENTAL MERCURY EXPOSURE FROM A MERCURY WEIGHTED BOUGIE
Fermin Barrueto1, Stephen McKenna2, Sonny Kim2, Margaret L. Bleeker4
1University of Maryland School of Medicine, Baltimore MD USA
2Upper Chesapeake Health Systems - Harford Memorial Hospital, Havre de Grace MD USA

Background: We report a rare case of an elemental mercury (Hg) spill into the intraperitoneum of a patient undergoing a laparoscopic Roux-en-Y gastric bypass procedure. Case Report: 64 morbidly obese man underwent laparoscopic Roux-en-Y gastric bypass procedure. To perform a leak test after construction of the anastomosis, a bougie was used to assist with passage of orogastric tube. A stitch was placed through the tip of the bougie which was not labeled to contain Hg. The procedure was completed and bougie removed. An OR tech noticed silvery beads in the sink, the floor and finally on the bougie. Hazmat was notified and took ambient air Hg measurements with an Lumex RA-915 + Zee-man Mercury Spectrometer. Elevated readings were noted in all 4 ORs which were shutdown for cleaning and the patient was notified after full recovery from the initial surgery. CT of the abdomen/pelvis showed the mercury both within the GI tract and intraperitoneal. Laparoscopic and fluoroscopy assisted removal of the intraperitoneal mercury 2 days later was performed. Some mercury was removed and the patient recovered from the surgery without complications. Intraoperative readings of ambient air in the OR was taken with the same Hg spectrometer throughout the procedure and with intermittent readings taken from the exhaust from the laparoscope. Ambient air remained <200ng/m³ throughout the procedure. Initial reading when abdomen filled with gas from laparoscopy was 2849ng/m³. Maximum reading came after 141 min of operating and was 98169ng/m³ after multiple bead recovery of mercury with suction. The patient’s serum Hg levels were: day 0, 25ug/L; day 22, 146ug/L; day 39, 122ug/L; day 43, 125ug/L; day 51, 101ug/L; day 24 Urine Hg on day 0, 9 ug/L; day 43, 227 ug/L (Vol 600cc); day 51, 196ug/L (Vol 850cc, UCr 1.77g/24h thus corrected Urine Hg 110.73ug Hg/g creatinine). Patient underwent neurologic
testing that revealed pre-existing diabetic neuropathy and essential tremor with no neurotoxic effect from mercury. 3 month follow up shows patient has lost 60lbs of weight from the surgery and is asymptomatic from the exposure.

**Case Discussion:** This case illustrates the need for hospitals to remove and audit their stock for mercury containing medical devices for patient safety, especially when viable alternatives exist for Hg weighted esophageal bougies.

**Conclusion:** This case represents the first known case of intentional mercury self-poisoning as a means to obtain workers’ compensation benefits.

---

**218. BLOOD LEAD LEVEL AMONG 6-YEAR OLD PALESTINIAN CHILDREN**

Ansam Sawalha1, Robert O. Wright2, David Bellinger3, Chitra Amarasingwardena4, Waleed Sweileh5, Adham Abu Tahah6

1An-Najah National University, Nablus Israel; 2Harvard university, Boston MA USA

**Background and Objective:** Lead is among the toxic heavy metals and is of major environmental and health concern. Leaded gasoline is still used in the major environmental and health concern. Leaded gasoline is still used in the Occupied Palestinian Territories. To assess the degree of Palestinian children’s exposure to this toxic metal, blood lead was measured.

**Methods:** First-grade students were selected from both private schools and schools in the Palestinian refugee camps (administered by United Nations Relief and Work Agency [UNRWA]). Venous blood was collected after obtaining parental consent. Hemoglobin was also measured, along with body weight and arm circumference. A questionnaire was sent with each child before blood collection to his/her parents to be filled out regarding demographic and life conditions.

**Results:** Blood samples were collected from 177 children, 140 males and 37 females. Seventy-four children were from private schools and were living in the city while 103 were from UNRW A schools and were living in refugee camps. The concentration of organic arsenic was 4332 mcg/L, and no inorganic or methylated arsenic was detected. A repeat 24-hour urine collection was performed after the patient abstained from eating seafood, and the total arsenic level was 50 mcg/day (reference range 0-50) with concentrations of 3 mcg/L (reference range 0-35) and 21 mcg/g creatinine.

**Case Discussion:** Our patient demonstrates the highest level of arsenobetaine reported in the literature. The majority of the arsenic consumed in the human diet is the organic arsenic compound arsenobetaine. Arsenobetaine is considered non-toxic and is present in many forms of seafood. Although seafood is recognized as the main source of arsenobetaine in the diet, other foods such as mushrooms also contain this compound.

The level manifested by our patient is higher than would be expected for a person who had not consumed seafood for five days prior to testing. He reported that he ate two lobster tails and did not recall any other fish or seafood in the interval between that meal and when he tested his urine. Arsenobetaine clears the body rapidly, with the majority being excreted two days after ingestion. The high levels may be due to consumption of food that he did not recognize as possibly containing arsenobetaine just prior to testing, or that his clearance of the arsenobetaine from the ingested lobster is slower than published ranges.

**Conclusion:** This case demonstrates the importance of speciation when measuring urine arsenic levels. Total urine arsenic levels are difficult to interpret without knowing how much is due to the non-toxic organic arsenic compound arsenobetaine, even if the patient’s dietary habits are known before testing.

---

**220. METAL FOR MONEY: FACTITIOUS OCCUPATIONAL MERCURY EXPOSURE**

Loren K. French2, Brent T. Burton1, Nathanael J. McKeown1

1Oregon Poison Center, Portland OR USA; 2Oregon Health and Sciences University, Portland OR USA

**Background:** Mercury is available in 3 distinct forms of toxicologic importance: elemental, organic and inorganic. While human poisonings are infrequent, occupational exposure remains an important consideration for workers, employers and toxicologists. We report a case of intentional mercury self-poisoning as a means to obtain workers’ compensation benefits.

**Case Report:** A 39 YO commercial lab worker presented to an urgent care clinic for evaluation of rash, myalgias, hair loss and photosensitivity. Her medical history was significant for an unconfirmed history of dermatomyositis ten years earlier. Her current symptoms were initially considered to represent a relapse. However, over the next two years she failed to improve despite several trials of immune-modulating medications. Instead, she reported that only large doses of opiates controlled her pain and fatigue complaints. Though she had a positive ANA, the remainder of her workup including a normal ESR, muscle biopsy and EMG could not definitively demonstrate a rheumatologic origin for her symptoms. During this time she remained unemployed and on disability status. The origin of her symptoms seemed to remain unclear until a 24-hour urine collection revealed a mercury level of 1030 μg/L. A repeat level less than a month later was 685 μg/L. The patient filed a claim for an occupational disease related to mercury exposure. An industrial hygiene survey at her workplace revealed insignificant airborne concentrations of mercury raising a question about a relationship to workplace exposure. When the exam findings revealed evidence of skin inflammation of the foot, consultation with the Poison Center medical toxicologist prompted a radiographic survey of the extremities. This led to a finding of extensive subcutaneous mercury deposits in the foot and antecubital fossae. A subsequent chest radiograph revealed evidence of mercury emboli. Despite the obvious source of mercury, the patient steadfastly asserted that she had been poisoned from a work exposure. Nonetheless, she received prophylactic chelation treatment with DMSA prior to undergoing surgical debridement of the mercury deposits.
Case Discussion: Inhalation of mercury vapor would be the expected route of occupational mercury exposure. The only conceivable route for exposure in this case, however, is subcutaneous injection. While psychiatric disease is a common undertone in previously published cases of elemental mercury injection, it appears that the primary goal for this patient was monetary compensation and disability status.

Conclusion: We report case of factitious mercury poisoning by intentional injection for the purpose of acquiring worker compensation benefits and/or disability status.

221. NICKEL DERMATITIS AFTER SWALLOWING A COIN: CASE REPORT AND REVIEW OF THE LITERATURE

Aaron Ziegler, Rebecca Andrews, Bryan S. Judge
GRMEP/MSU Program in Emergency Medicine, Grand Rapids MI USA

Introduction: Metal contact dermatitis is a relatively common condition, with nickel most frequently involved. Symptoms usually develop following cutaneous contact with the metals, are pruritic, and appear as an erythematous vesicular or eczematous rash. We present a case of a child with a known nickel contact sensitivity that accidentally swallowed a nickel containing coin and subsequently developed a widespread dermatitis.

Case Report: A seven year old female presented to the emergency department with a generalized rash after accidentally swallowing a quarter. She noted that the accidental ingestion occurred two days ago and then began developing a generalized pruritic rash the following day. Additionally, the patient had a documented history of contact dermatitis with nickel. A chest x-ray confirmed the presence of a gastric metallic foreign body. Examination revealed a generalized fine erythematous macular-papular rash on all skin surfaces as well as excoriations and scleral injection. Abdominal x-ray showed the coin in the distal gastric body. Pediatric surgery was consulted and the patient was given 50 mg of IV methylprednisolone and 25 mg of IV diphenhydramine. She was admitted to the surgical service and observed overnight. The next morning an EGD was performed and the coin was easily identified within the stomach and removed.

Discussion: The US Washington quarter is composed of 92% copper and 8% nickel. A review of the literature revealed five previous case reports of individuals that exhibited systemic rash following ingestion of nickel containing foreign bodies. Two of the cases involved children with previously known nickel sensitivities. Additionally, one patient had an associated gastroitis noted on endoscopy, and another patient also had a lung infiltrate felt to be related to the nickel allergy. In all but one of the patients the foreign body was removed via endoscopy or surgery, and in all cases the systemic symptoms receded once the metal was no longer within the GI tract.

Conclusion: Treatment algorithms have been proposed for the management of foreign body ingestion in children, however given the preceding cases, additional consideration should be made for early endoscopic removal if there is a known contact allergy to the foreign body.

222. NON-ACCIDENTAL, LIFE-THREATENING, LEAD POISONING IN A CHILD

Anthony F. Pizzi1, Lawrence S. Quang2, Steven J. Walsh1, Michael J. Lynch1
1University of Pittsburgh School of Medicine, Pittsburgh PA USA; 2University Hospitals Case Medical Center, Cleveland OH USA; 3University of Pittsburgh School of Medicine, Pittsburgh PA USA; 4University of Pittsburgh School of Medicine, Pittsburgh PA USA

Background: We describe a case of non-accidental, life-threatening, repeated, lead (Pb) toxicity in a child with CHARGE syndrome.

Case: Our patient was a 5-month-old girl with CHARGE syndrome with a trabeculothy and gastrostomy tube who presented to our center as an outpatient for altered mental status, seizures, and explosive diarrhea. Her vital signs were normal; examination was remarkable for lethargy and bluish skin discoloration. She was admitted for further evaluation. Methemoglobin concentration was 18%, and electroencephalogram/other laboratory studies were non-diagnostic on admission. Imaging revealed radiopaque material coating the stomach and small bowel without prior contrast administration. She was treated for infectious etiologies until a blood Pb concentration returned at 135 μg/dL.

Chelation with BAL and CaNa2EDTA was immediately initiated. Whole-bowel irrigation was complicated due to an ileus, but was eventually successful. Her symptoms improved while hospitalized. She was discharged on succimer with a Pb level of 35 μg/dL. After an extensive search, the only environmental Pb source was contamination in two out of three of the girl’s medications. Both contaminated medications were produced by different manufacturers and were filled in two different pharmacies. Furthermore, the Pb contaminated medicines did not account for the girl’s entire lead burden. Over the next 16 months, her Pb levels oscillated between 25-97 μg/dL, and she was intermittently given PO succimer or IV CaNa2EDTA. Each inpatient IV chelation markedly reduced her Pb level. However, at-home PO chelation frequently resulted in abrupt rises in her Pb levels. Serial abdominal x-rays showed 2 small persistent radiopaque areas in the stomach confirmed to be Pb-burdened gastric mucosa via endoscopy-directed biopsies. We could not determine if the high spiking Pb was due to PO chelator-assisted absorption of the gastric Pb or ongoing malicious Pb poisoning. A long investigation left the child in the care of her family which complicated effective treatment.

Case Discussion: After successful removal from the home, her Pb levels trended downward and she has responded to PO chelation without need for surgical debridement of her gastric mucosa. Police search revealed lead nitrate in the home which explained the methemoglobinemia upon admission and the patient’s mother has been convicted of the poisoning.

Conclusion: This case highlights two important points. First, removing the child from the source (in this case, the mother) remains the mainstay of treating Pb toxicity. Second, suspicion that gastric Pb absorption may be enhanced with oral chelator administration does not appear to be the case here after retrospective review.

223. CARDIOTOXICITY IN THE SETTING OF MONOCLED COBRA ENVENOMATION

Colleen M. Birmingham, Robert S. Hoffman, Lewis S. Nelson
NYU Hospital Center/NYC Poison Control Center; New York NY USA

Background: Cobra venom is best known for its neurotoxic components. Animal models of cobra envenomation have also demonstrated cardiotoxicity. We report a case of human cardiotoxicity following Monocled Cobra envenomation.

Case Report: A twenty-five year old man purchased an albino Monocled Cobra (Naja kaouthia) which was reportedly “devenomated.” The snake bit his right hand and within three hours the patient was found apneic. EMS intubated the patient and brought him to a local hospital. He was promptly transferred to the regional snake bite center where his initial vital signs included: BP, 156/103 mmHg; HR, 112 beats/min; RR, 17 breaths/min; Temp. 96.6°F; SpO2, 100% on a ventilator. On physical examination, the patient was unresponsive and flaccid. His right fifth finger had two pinpoint puncture marks and his hand was moderately edematous. Through collaboration with the local zoo, the treating physician obtained a polyvalent equine antivenom (Haffkine) that was produced in India for cobra envenomations and stocked at the zoo in case of envenomation of snake handlers. The patient received 15 vials of antivenom over six hours in the ED while becoming increasingly tachycardic to 150 beats/min. Another 10 vials were infused in the ICU where the patient’s lactate peaked at 5.9 ng/dL, his CPK at 1653 U/L, and his troponin at 0.38 ng/mL. He developed an abscess at the site of the snake bite which resolved with routine therapies. The patient was extubated on hospital day three and was well two weeks after discharge.

Case Discussion: Snake venom is a complex mixture of toxic proteins, metal ions, and other small molecules which lead to an array of cellular sequelae. Classically, cobra venom causes flaccid paralysis as seen in this case by blockade at the motor end plate, a result of the alpha neurotoxin component of venom. While the basic science literature also describes a number of cardiotoxins found in cobra venom, our search did not reveal any reports of human cardiotoxicity in the setting of Naja kaouthia envenomation. Cardiotoxins, only yet noted in the venom of cobras and rhingals, are single-chain short polypeptides that bind to excitatory cells leading to depolarization...
224. **DO PATIENTS RECOGNIZE OVER-THE-COUNTER (OTC) AND PRESCRIPTION (RX) PRODUCTS CONTAINING ACETAMINOPHEN (APAP) AND TOXICITY RELATED TO APAP CONTAINING PRODUCTS?**

Brenna Farmer1, Sunday Clark2
1Weill-Cornell Medical College, New York NY USA; 2University of Pittsburgh, Pittsburgh PA USA

**Background/Objectives:** Previous survey studies have shown that patients often do not know which OTC and RX products contain APAP. This needs assessment survey was conducted to determine if our patients recognized pictures of OTC and RX products containing APAP and APAP toxicity. A secondary goal was to determine if education made a difference in the recognition of APAP products.

**Methods:** An anonymous convenience survey was conducted as a needs assessment of the population at an academic medical center in New York City over a 2-month period. Age, gender, and education level were collected for each participant. Using pictures of 17 OTC and RX products, the participants were asked if the product contained APAP. They were allowed to answer: Yes, No, or I don’t know. They were also surveyed about APAP toxicity. Using Kruskal-Wallis non-parametric testing, the number correct in each category (APAP vs. non-APAP) was compared to the number correct across the education groups. A $P<0.05$ was considered significant. Further analysis is pending.

**Results:** 198 surveys were completed. The average age was 49 (range 19-88 years old); 57% were women; 41% were men; 2% did not specify. Education was divided as such: 3% did not graduate, 12% high school education, 37% college education, 45% graduate school education, and 3% did not answer. Overall, 39% of responses regarding APAP products were correct while 52% of responses regarding non-APAP products were correct. There were 69% correct responses for Tylenol brand APAP compared to 25% and 18% correct responses respectively for non-Tylenol OTC APAP and RX APAP. 58% of participants recognized that APAP causes poisoning. 67% recognized the liver as the poisoned organ. 64% knew death could result.

The median number correct for all APAP products (11 total) did not differ significantly across the 3-level education groups (high school education or less: 4 [IQR, 2-5]; college education: 4 [IQR, 3-6]; graduate school education: 4 [IQR, 3-6]; $P = 0.33$). The number correct for non-APAP medications (6 total) was also similar across the education groups (high school education or less: 2 [IQR, 0-5]; college education: 3 [IQR, 1-6]; graduate school education: 4.5 [IQR, 1-6]; $P = 0.08$).

**Conclusions:** Participants do not know which OTC and RX products contain APAP unless labeled by the brand name Tylenol. However, most participants do recognize that the liver is the organ poisoned and that death can result from overdose. Education level is not a significant factor in the recognition of APAP products. With this information, a module will be developed to better educate our patients upon discharge from the ED.

225. **EXAMINING THE RISK OF METHANOL POISONING FROM METHYL ACETATE-CONTAINING PRODUCTS**

Alicia Minns1, Alycia Clark2, Anesia Zeller3, Richard F. Clark1, Lee Cantrell1
1UCSD, San Diego CA USA; 2California Poison Control System, San Diego CA USA

**Background:** Methyl acetate (MA) is used as a nail polish remover and as a solvent in glues and paints. It is hydrolyzed to acetic acid and methanol. Due to the concern for methanol poisoning following ingestion, the poison centers (PC) have been referring patients to health care facilities (HCF) to monitor for metabolic acidosis. Clinical experience with methyl acetate and the incidence of metabolic acidosis has not been previously reported.

**Methods:** We queried a statewide PC database between January 1997 and December 2010. Inclusion criteria were isolated methyl acetate ingestions. Data collected included: age, gender, amount ingested, reason for ingestion, symptoms, medical outcome, treatment site, laboratory evidence of metabolic acidosis, and therapy.

**Results:** 83 cases were identified. Females made up 52% of all patients. 32 cases (39%) were less than 2 years of age; 43 cases (52%) were between 2 and 5 years; 2 cases (2%) were between 6 and 9 years; 2 cases (2%) were between 10 and 18 years; 4 cases (5%) were over 19 years of age. The majority (93%) of the ingestions were unintentional. Amount ingested was difficult to discern with 11 cases (13%) estimated to be more than a mouthful. Sixty-two cases (75%) were referred to a HCF. Of these, 46 (75%) had no effect and 25% of cases had minor effects consisting of vomiting; 12 (14%) cases; throat/oral irritation- 3 cases; drowsiness, abdominal pain and ataxia in one case each. No moderate effects, major effects or deaths were reported. One case received single dose activated charcoal and one case received intravenous fluids as therapy. All other cases were observed only. Of all cases, including home and HCF cases, 63 (76%) had no symptoms. 53% of cases referred to HCF had at least one chemistry panel done (27% had more than one chemistry panel done). One asymptomatic patient had a mild metabolic acidosis (bicarbonate of 17), which resolved after a short period of observation alone.

**Conclusion:** Methyl acetate exposures are reported to poison control centers due to the perceived risk of in vivo conversion to methanol. In our study, methyl acetate ingestions were not associated with the development of a clinically significant metabolic acidosis and observation at home is reasonable for unintentional ingestions.

226. **UTILITY OF THE INITIAL TROPONIN FOR COCAINE-ASSOCIATED CHEST PAIN IN THE ED**

Jane M. Prosser1, John Cienk3, Marco L. Sivilotti4, Andrew Stolbach4, Lewis S. Nelson5, Robert S. Hoffman6
1Weill Cornell Medical Center, New York NY USA; 2Jackson Memorial Hospital, Miami FL USA; 3Queen’s University, Kingston ON Canada; 4John Hopkins University, Baltimore MD USA; 5New York City Poison Center, New York NY USA

**Background:** Although cocaine abuse often causes chest pain (CCP), myocardial infarction (MI) occurs in <10% of cases. Current recommendations by the AHA, based largely on a single study, support outpatient management if the ECG is unchanged and the troponin normal over a 9-12 hour interval. Our Objective was to investigate the value of the first serum troponin at presentation for identifying cocaine-associated MI.

**Methods:** This prospective observational cohort study enrolled patients aged 18-85 years presenting to 4 tertiary care emergency departments (ED) with CCP and report of recent (<7 days) cocaine use or a positive urine cocaine. Patients were excluded for an other non-ischemic diagnosis, for dysrhythmia which the ECG is unchanged and the troponin normal over a 9-12 hour interval. Our Objective was to investigate the value of the first serum troponin at presentation for identifying cocaine-associated MI.

**Results:** Eighty-one patients were enrolled from 3/09-2/11. Seventy-one were male, with a median age of 42. Two patients were enrolled twice for separate CCP visits, totaling 83 patient encounters. There were 42 African Americans, 19 Caucasians, 19 Hispanics, 1 unknown. Five patients sustained an MI on initial visit, all with an abnormal troponin at presentation. Of the remaining 78 encounters, 24 had a normal troponin >9 hours after presentation. Another 31 had a second troponin measured less than 9 hours after presentation, and 8 had a single troponin measured; all 39 were known to be alive and MI-free at 30 days. Fifteen patients were deemed lost to follow-up by 30 days. Of these, 8 had a negative second troponin 3-8 hours after arrival, and 7 had a single negative troponin. A total of 8 patients had troponin.

**Results:** in the indeterminate range, but MI was subsequently excluded by serial measurements in seven and after stress testing in one patient. Of
these the indeterminate troponin was present initially in 6. One patient had a very mild elevation on the second troponin that was negative on third measurement. A second patient had a mild elevation on second measurement and was diagnosed with transient ischemia due to vasospasm after normal exercise stress testing. No patients were known to have died within 30 days of index visit.

**Conclusions:** The initial troponin in patients presenting to an ED with CCP following cocaine use appears to identify most with MI. These data are preliminary, subject to incorporation and workup bias, but reflect real-world practice. The findings are encouraging, supporting further study currently underway to produce adequately powered results.

228. MASSIVELY ELEVATED BLOOD MERCURY LEVELS AFTER ELEMENTAL MERCURY EXPOSURE WITH MINIMAL MORBIDITY

**Stephen L. Thornton**, A. Minns, R. F. Clark

1University of California - San Diego, San Diego CA USA; 2Veteran’s Affairs Medical Center, San Diego CA USA

**Background:** Exposure to elemental mercury can cause significant morbidity and occasional mortality. We report a case of an elemental mercury exposure with massively elevated mercury blood levels with minimal morbidity. Case Report: A 42 year old female with a history of essential tremor, eczema, and celiac disease was found by her primary care physician to have a blood mercury level of 1201.8 mcg/L (normal: 0-10 mcg/L) on day 0. Approximately 40 days after a mercury containing thermostat “exploded” in her bedroom. She attempted to vacuum up the mercury. She continued to live in the apartment after the event. She then developed generalized fatigue, GI upset, a nonproductive cough, sore throat, rash and worsening tremor approximately 2 weeks after the incident. On day 3, mercury vapor levels in her bedroom were found to exceed 200 mcg/m3 and it was quarantined. County HazMat reported a total of 114 mL of elemental mercury was recovered from her bedroom, significantly more than would be found in a typical thermostat. On day 5 she was evaluated in the medical toxicology clinic and found to have an eczematous rash and a nonproductive cough. She was noted to have a blood pressure of 135/90. Otherwise her vital signs and physical exam were normal. In particular, no tremor was noted on exam and no injection sites were seen. Her chest x-ray, abdominal x-ray, complete blood count (CBC), serum chemistry, liver function tests, and urine analysis were all normal. A repeat blood Hg level was 793 mcg/L. Twenty-four hour urine mercury level was 4012 mcg/L (normal: 0-10 mcg/L). She was treated with a 15 day course of DMSA. Subsequently her blood Hg level declined to 215 mcg/L on day 20 and 181 mcg/L on day 26. Repeat CBC, serum chemistry, and liver function tests remained normal on those days. Her blood pressure remained elevated (up to 151/94 on day 26) but her physical exam remained unchanged. In addition, her urine mercury levels declined to 1241 mcg/L on day 20. She continued to complain of cough, nausea, and fatigue so an additional 14 day course of DMSA was prescribed on day 27. The patient was rechecked on day 55 and found to have a blood mercury level of 61 mcg/L and urine mercury level of 339 mcg/L. Her blood pressure was now normal (117/73) and her symptoms had improved. Her CBC, serum chemistry, and liver function tests were normal. On day 86 she had a serum mercury level of 24 mcg/L and her symptoms had almost completely resolved.

**Conclusion:** To our knowledge this is the highest reported non-fatal mercury level of index visit.

229. A COST COMPARISON OF FOMEPIZOLE AND HEMODIALYSIS IN THE TREATMENT OF METHANOL AND ETHYLENE GLYCOL TOXICITY

**Heather Ellsworth**, Kristin M. Engebretsen, Lisa M. Hlavenka, Andy K. Kim, Jon B. Cole, Carson R. Harris, Samuel J. Stellplug

Regions Hospital, St Paul MN USA

**Background:** Fomepizole (F), alone or in combination with hemodialysis (HD), may be used in the treatment of toxic alcohol exposures such as methanol (M) and ethylene glycol (EG). There is a paucity of data regarding the financial cost of each treatment approach. Using patient charge estimates specific to our institution, we present the Results of an analysis comparing F and HD for treatment of M and EG levels of 50 mg/dL.

**Methods:** Patient charges associated with treatment of toxic alcohol exposures in 2010 at our institution were reviewed and averaged with respect to the cost of the following: F dose, HD session, daily rate of a general care (GC) bed, and daily rate of an intensive care unit (ICU) bed. All other costs were assumed comparable irrespective of treatment received. Based on available pharmacokinetic (PK) data for M and EG in the presence of F, the duration of treatment was projected.

**Results:** The average patient charge for a dose of F was $1,267, HD session $765, GC bed (daily rate) $915, and ICU bed (daily rate) $1,524. For an EG or M level of 50 mg/dL treated with HD, patient charge would approximate $4,823; this cost was based on the administration of 2 doses of F, 1 HD session, and 1 day of hospitalization in the ICU. In contrast, the estimated cost associated with treatment of an EG level of 50 mg/dL, with F only, based on a t½ of 12.9 h, to an endpoint of 20 mg/dL, was $5,631. This was based on a treatment duration of 25.8 h and charges for 3 doses of F and 2 days of hospitalization in a GC bed. Similarly, for a M level of 50 mg/dL treated with F only, with an estimated t½ of 54 h, the estimated cost was $17,245 based on administration of 10 doses of F and 5 days of hospitalization in a GC bed.

**Discussion:** Based solely on financial considerations, HD is a more favorable treatment approach for levels of M and EG > 50 mg/dL than F alone. This is especially true for M, which has a significantly longer t½ than EG. Limitations include not accounting for the cost of clinically significant complications related to HD such as vascular injury, infection, and thrombosis. However, data suggest these complications are rare. Another limitation is the failure to account for individual variability with respect to PK as well as patient weight,
which may influence the number and volume of F doses required. Accounting for these parameters could make the cost difference between F and HD more favorable for HD, considering reports of extremely long EG and M 91's treated with F alone and patients with weights that necessitate larger doses.

Conclusion: Hemodialysis is a more cost effective approach to the management of melphalan and ethylene glycol toxicity than fomepizole alone if levels exceed 50 mg/dL.

230. MORBIDITY AND MORTALITY FROM ACETAMINOPHEN POISONING: A 30-YEAR EXPERIENCE

1University of Calgary, Calgary AB Canada; 2Queen's University, Kingston ON Canada; 3University of British Columbia, Vancouver BC Canada; 4McGill University, Montreal QC Canada; 5University of Ottawa, Ottawa ON Canada; 6University of Alberta, Edmonton AB Canada; 7University of Western Ontario, London ON Canada; 8Université de Montreal, Montreal QC Canada; 9University of Toronto, Toronto ON Canada; 10Dalhousie University, Halifax NS Canada; 11University of California-San Francisco, San Francisco CA USA; 12University of Colorado School of Medicine, Denver CO USA

Background: Acetaminophen (APAP) poisoning is the most common etiology of acute liver failure in North America. Early identification of patients at risk of death remains challenging. As part of a study designed to compare different prognostic indicators, we sought to describe the characteristics of hospitalized patients who died or were referred for liver transplant for APAP poisoning.

Methods: We performed a structured medical record review of patients hospitalized for APAP poisoning at one of 34 hospitals in eight Canadian cities from 1980-2010. We identified all cases of in-hospital death, liver transplant, or admission for APAP poisoning.

Results: One hundred seventy-three patients met our inclusion criteria. Eighty patients (10%) were admitted from 1980-1990, 80 (46%) from 1991-2000, and 75 (43%) from 2001-2010. The median age was 42 years, and 106 (61%) were female. Sixty-nine (40%) co-ingested ethanol, and 93 (54%) were described as chronic alcoholics. NAC was given in 147 (85%) patients; 138 (94%) were started on the 21-hour intravenous (IV) protocol. Median time from start of APAP ingestion to treatment with NAC was 110 hours (Interquartile range [IQR] 313 hours) and median duration of NAC therapy was 29 hours (IQR 47 hours). Median values for the following variables at hospital presentation were: aminotransferases 2242 IU/L, INR 3.9, serum pH 7.28, phosphate 1.6 mmol/L, lactate 9.1 mmol/L, creatinine 169 μmol/L. Eighty-one (47%) patients developed cerebral edema. One hundred fifty-seven patients (91%) died, 4 (2%) survived, and 12 (7%) had unknown outcomes (transfer to another facility for liver transplant consultation. Patient demographics, ingestion history, N-acetylcySTEINE (NAC) therapy, laboratory values, presence of cerebral edema, and outcome were abstracted and summarized. The presence of cerebral edema was identified using clinical, radiologic, therapeutic, and post-mortem criteria developed by consensus among all investigators.

Conclusions: Hepatotoxicity, coagulopathy, renal failure, acidosis and cerebral edema were frequently present during hospital admission in this cohort of APAP-poisoned patients. Liver transplantation was performed infrequently and survival post-transplant was uncommon. Further research on early recognition of patients who may benefit from aggressive liver care, including transplantation, is required.

231. USE OF THE INITIAL ACETAMINOPHEN CONCENTRATION TIMES SERUM AMINOTRANSFERASE PRODUCT TO PREDICT SIGNIFICANT LIVER ENZYME ELEVATIONS AFTER ACETAMINOPHEN OVERDOSE

Steve Offerman1, Michael Young2
1Kaiser Permanente Northern California, Sacramento CA USA; 2Kaiser Permanente Northern California, San Jose CA USA

Background: Patients presenting with acute acetaminophen (APAP) overdose are at risk assessed using the Rumack-Mathews (RM) nomogram. The serum APAP level times aminotransferase (AT) product has been proposed as another predictor of risk. Our aim was to determine whether the APAP x AT product at the time of initial labs predicted significant liver enzyme elevations in patients treated for acetaminophen overdose.

Methods: We retrospectively analyzed data from patients admitted for isolated APAP overdose to any Kaiser Permanente Northern California (KPNC) hospital from July 2003 through December 2007 to assess whether the APAP x AT product at the time of initial labs predicted significant AT elevations (defined as AST or ALT > 1000 IU/L). Any patient admitted for treatment of acute, isolated APAP overdose was eligible for study inclusion. Only those cases presenting within 24 hours of ingestion and with serum APAP level above the RM nomogram and/or initial AT elevation were included. To assess for association and identify the optimal cutoff for the APAP x AT product, a logistic regression model and Receiver operating characteristic (ROC) curve were constructed.

Results: The cohort of APAP overdose patients admitted to KPNC hospitals in the study period included 435 patients. Of these, 216 patients met study inclusion criteria. The mean age was 25.9 years. There were 161 (74.5%) females. The mean time from APAP ingestion to presentation was 7.5 hours (range 0.25-23.0). All study patients received NAC therapy. The mean time from ingestion to NAC was 10.1 hours (range 1.3-24.3). There were no deaths or liver transplants in the study group. The APAP x AT product predicted significant AT elevation (p = 0.003) with an area under the ROC curve of 0.716. Based on the ROC curve, the most appropriate cutoff value was 10,000. Using this cutoff, the APAP x AT product had a sensitivity of 44% and specificity of 92% to predict significant AT elevation. The PPV was 44% and NPV was 92%.

Conclusion: The initial APAP x AT multiplication product does appear to be a predictor of significant AT elevation. Using a cutoff of 10,000, we found a reasonably high specificity. Use of serial APAP x AT products might improve the value of this prediction tool.

232. THE ROLE OF THE EMERGENCY DEPARTMENT PHARMACIST IN THE TIMING OF MEDICATION DELIVERY

Kelly P. Owen, Christine S. Castro, Brian Fusco, Daniel K. Nishijima, Deborah B. Diercks, Timothy E. Albertson, Mark E. Sutter
University of California Davis, Sacramento CA USA

Background: It has been shown that having a pharmacist in the emergency department (ED) decreases medication errors and cost. We tested the hypothesis that the presence of a pharmacist in the ED improves time to medication administration to ED patients.

Methods: This is a study of adult patients seen in a single, academic ED. A retrospective chart review was performed using data from the electronic medical record (EMR). Medication order and delivery times were recorded and the time to medication delivery was analyzed with and without the presence of a pharmacist. Secondary analysis included the time to drug delivery for medications stored in an automated dispensing system situated in the ED (Pyxis™) versus those stored in the central pharmacy.

Results: We evaluated 16,699 separate patient encounters resulting in 30,782 medication orders. There was a pharmacist on duty 51.4% of the time (n = 15,941 drug administrations). Overall the mean-time to drug delivery was 33.8 minutes (Range 0-720 minutes). We found no significant difference between overall drug delivery time with a pharmacist on versus off duty. (33.9 min vs 33.7 min p > 0.78). There was a significant difference in time to administration of medications kept in the Pyxis™ versus those that were not in the Pyxis (33 min vs. 54 min, p < 0.001). Having a pharmacist on duty significantly decreased the time to drug delivery for medications not found in the Pyxis™ (47 min vs 61 min p < 0.001).

Conclusions: Overall, there was not a significant decrease in time to medication delivery with a pharmacist on duty. When evaluating the subset of medications not located in the ED Pyxis there was a significant decrease in time to medication delivery with a pharmacist on duty. Further prospective studies are needed to evaluate the role of the pharmacist in the ED.
235. PUMPING PARTIES, PUMP UP YOUR DERRIERE
Twyla A. Kimball, Denise Becker
Florida Poison Information Center, Tampa, Tampa FL USA

Background: To get hourglass figures in just one hour, Floridians visit illegal “pumping parties” where nonprofessionals inject various substances such as hydroxyurea, Fix-A-Flat®, superglue, mineral oil, vegetable oil, peanut butter, hydrogel and other unknown products to provide or enhance curves in all the right places. We present four cases of “pumping party” victims, who almost became “pumping party” fatalities.

Case Series/Reports: Within one year, two hospitals sought the advice of this poison center on four emergency room patients. Each was admitted for sepsis resulting from injections in the buttocks. The injectable substances ranged from hydroxyurea, mineral oil, vegetable oil, superglue, hydrogel, peanut butter, Fix-A-Flat® and other unknown products. They presented to the emergency rooms at various post exposure days. Two were seven days, one was ten days and one was one month. Each had multiple injection sites and was in moderate to severe septic states. All required surgical intervention with repeated irrigation and drainage of the infected wounds. All required intravenous antibiotics, central line placement plus blood transfusions. Two developed renal failure with creatinine levels of 11.8mg/dl and 13.1mg/dl. Both required dialysis. Their length of hospital stays were 14, 20, 26 and 33 days. All were discharged with ongoing needs such as wound care, intravenous antibiotic management, multi-system concerns and home healthcare.

Discussion: Treatment and outcomes of the four cases were delayed and complicated because of the illegal component of the exposures. The techniques of the injections were varied, non-sterile and the injected substances were partially unknown. Healthcare was sought only after extreme symptoms surfaced. Contacting the poison center may have minimized further complications.

Conclusion: The desire to be “beautiful” is an age-old phenomenon. It is now a multimillion-dollar industry. For many, it is considered expensive, but worthwhile. For others, the high cost drives them to seek alternative Methods: in which the market for back alley, illegal Fix-a-Flat® style injections thrive. Pumping parties are not free. The cost for those seeking “beauty” seems within financial reach from these “back-alley” providers. It is the enormous immeasurable cost of pain, suffering and bad outcomes that become the burden long after the “providers” have moved on.

236. CLINICAL AND EXPERIMENTAL STUDY OF ACUTE INTOXICATION AS A RESULT OF INGESTION OF PARAPHENYLENEDIAMINE CONTAINING STONE HAIR DYE
Hatem A. Ahmed

Hair dyes containing para-phenylenediamine were used in some communities for criminal purposes and more frequently for attempted suicide and sometimes accidentally ingested. The aim of the present work was to analyze the various aspects of acute poisoning through a retrospective study of fatalities
investigated by Assiut Laboratory of Medico-legal Department, Ministry of Justice in four governorates in Upper Egypt as a result of black hair dye ingestion and to detect the systemic effects on experimental animals as a result of its ingestion and if there is dose-effect relationship.

Methods: The records of acute poisoning cases of hair dye ingestion investigated by Assiut Forensic Chemical Laboratory in the period from January 2002 to December 2009 were examined as regarding type of poison, pattern, incidence, sex, geographical distribution and mode of poisoning. The studying of the systemic effects on ingestion of hair dye was conducted 30 rats divided into five groups each contain 6 animals. The first group was the control and the other groups were subjected to oral administration of either stone hair dye or para-phenylenediamine in two doses (10 and 20 mg). The animals were sacrificed after 24 hours and haematological, histopathological (liver and kidney) and biochemical examinations were performed.

Results: revealed that 72.29% from Qena, 14.45% from Aswan, 12% from Sohag and 1.2% from Assiut. The highest incidence of poisoning was found in 2006 (19.3%) followed by 2008, 2009 (15.7%), then 2004, 2005 (13.3%), 2007 (12%), 2002 (7.2%) and lastly 2003 (3.6%). The majority of victim’s were females and most of cases were suicides. The experimental study revealed that the liver is the target organ of para-phenylenediamine toxicity. There was significant increase in the plasma enzymes AST, GPT. The liver tissues showed many degenerative changes in the form of vacuolated cytoplasm and irregular deeply stained nuclei of the hepatocytes with vascular congestion and lymphocytic infiltration. The levels of ALT and AST showed a significant increase in all treated groups while serum level of creatinine was insignificant decreased. There were insignificant changes in RBCs count in all groups and insignificant decrease Hb concentration while WBCs count, very significantly increased. The results confirmed that Para-phenylenediamine is the main toxic ingredient in stone hair dye. This compound is highly toxic when taken by mouth and the outcome depends mainly on the dose taken. The study recommends that the sale and use of PPD containing dyes, SHD and henna should be prohibited.

237. COMPARISON OF DATA IN THE TOXICOLOGY INVESTIGATORS CONSORTIUM (TOXIC) REGISTRY WITH THE NATIONAL POISON DATA SYSTEM (NPDS)

Kurt Kleinschmidt, Paul Wax1, Jeffrey Brem2
1University of Texas Southwestern Medical Center, Dallas TX USA;
2University of Colorado School of Medicine, Denver CO USA

Background: The American Association of Poison Control Centers (AAPCC) has the first and largest data set on patients with toxic exposures; the National Poison Data System (NPDS). In Jan 2010 the American College of Medical Toxicology’s Toxicology Investigators Consortium (Toxic) began a Registry that captures descriptive data on patients seen at the bedside by medical toxicologists (MTs). While this data registry is much smaller than the NPDS, it reflects the poisoning epidemiology among patients consulted by MTs. While this data registry is much smaller than the NPDS, it reflects the poisoning epidemiology that focuses on patients consulted by MTs.

Methods: The records of acute poisoning cases of hair dye ingestion investigated by Assiut Forensic Chemical Laboratory in the period from January 2002 to December 2009 were examined as regarding type of poison, pattern, incidence, sex, geographical distribution and mode of poisoning. The studying of the systemic effects on ingestion of hair dye was conducted 30 rats divided into five groups each contain 6 animals. The first group was the control and the other groups were subjected to oral administration of either stone hair dye or para-phenylenediamine in two doses (10 and 20 mg). The animals were sacrificed after 24 hours and haematological, histopathological (liver and kidney) and biochemical examinations were performed.

Results: revealed that 72.29% from Qena, 14.45% from Aswan, 12% from Sohag and 1.2% from Assiut. The highest incidence of poisoning was found in 2006 (19.3%) followed by 2008, 2009 (15.7%), then 2004, 2005 (13.3%), 2007 (12%), 2002 (7.2%) and lastly 2003 (3.6%). The majority of victim’s were females and most of cases were suicides. The experimental study revealed that the liver is the target organ of para-phenylenediamine toxicity. There was significant increase in the plasma enzymes AST, GPT. The liver tissues showed many degenerative changes in the form of vacuolated cytoplasm and irregular deeply stained nuclei of the hepatocytes with vascular congestion and lymphocytic infiltration. The levels of ALT and AST showed a significant increase in all treated groups while serum level of creatinine was insignificant decreased. There were insignificant changes in RBCs count in all groups and insignificant decrease Hb concentration while WBCs count, very significantly increased. The results confirmed that Para-phenylenediamine is the main toxic ingredient in stone hair dye. This compound is highly toxic when taken by mouth and the outcome depends mainly on the dose taken. The study recommends that the sale and use of PPD containing dyes, SHD and henna should be prohibited.

Table 1.

<table>
<thead>
<tr>
<th>Agent</th>
<th>ToxIC (%)</th>
<th>NPDS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>11.6</td>
<td>3.2</td>
</tr>
<tr>
<td>Analgesics</td>
<td>26.6</td>
<td>11.8</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>4.2</td>
<td>1.6</td>
</tr>
<tr>
<td>AntiDepressants</td>
<td>11.2</td>
<td>3.6</td>
</tr>
<tr>
<td>AntiCholinergic/Histamines</td>
<td>6.8</td>
<td>3.3</td>
</tr>
<tr>
<td>AntiMicrobials</td>
<td>0.6</td>
<td>2.5</td>
</tr>
<tr>
<td>Antipsychotics-SedHyps</td>
<td>19.5</td>
<td>5.8</td>
</tr>
<tr>
<td>Plants</td>
<td>0.9</td>
<td>2.1</td>
</tr>
</tbody>
</table>

Conclusion: The ToxIC registry’s population is older than that in the NPDS. Gender distribution in the registry is equal while the NPDS contains more females. While analgesics were the most common exposure category for both databases, there were significant differences between the types of exposures in the ToxIC registry and the NPDS. The registry provides a new source of information about poisoning epidemiology that focuses on patients consulted on by MTs.

283. CASE SERIES OF SEVERE CALCIUM CHANNEL BLOCKER TOXICITY TREATED WITH HIGH CONCENTRATION INFUSION OF HIGH DOSE INSULIN THERAPY

Suprat Saely1, Megan E.Musselman1, Keenan Bora2
1Detroit Receiving Hospital, Detroit MI USA, 2Children’s Hospital of Michigan Poison Control Center, Detroit MI USA

Background: High dose insulin (HDI) therapy is becoming well recognized as an effective therapy for severe calcium channel blocker (CCB) toxic ingestions. At most institutions, the standard concentration of insulin (SCI) infusion is 1 unit/mL. The dosing strategy for HDI therapy is 1 to 10 units/kg/hr; this may result in patients becoming fluid overloaded. To avoid this, a high concentration insulin infusion (HCI) of 10 units/mL was standardized at our institution.

Methods: A descriptive case series of three patients sequential with CCB toxicity who received HCI to manage blood pressure (BP) in addition to conventional therapy is presented.

Results: Case #1: A 28 year-old female intentionally ingested –1200mg of immediate release diltiazem. Her initial BP was 60/palp and heart rate (HR) was 68. HDI was started with an insulin bolus of 1 unit/kg and HCI at 1 unit/kg/hr and max rate of 2 units/kg/hr (13 mL/hr). During HCI therapy, mean arterial pressure (MAP) was maintained between 60-80 for the entire therapy. In addition to HDI, calcium and dextrose was given. The patient was transferred out of the ICU within 24 hours and discharged at 48 hours without incident.

Case #2: A 49 year-old female intentionally ingested an unknown amount of verapamil. Upon presentation, BP was 70/30 and HR was 52 with third degree AV block. Patient received calcium, glucagon and a norepinephrine (NE) infusion. In addition, HDI was initiated with a 1 unit/kg bolus, followed by HCI at a rate of 1.5 units/kg/hr and max rate of 4 units/kg/hr (30 mL/hr) along with a transvenous pacemaker. Within 2 hours of HDI, a MAP >70 was maintained and NE was discontinued without consequence. Patient remained on HDI for ~20 hours and patient was discharged without incident.

Case #3: A 56 year-old female accidentally ingested an unknown amount of verapamil. Initial vitals were a BP of 50/palp and HR of 29. Therapy included calcium bolus and infusion. Immediately following, HDI was started with a bolus of 1 unit/kg and HCI was started at 1.5 units/kg/hr (10 mL/hr) and no titration was needed to maintain MAP between 60-80 during the 12 hours of therapy. Patient was transferred out of the ICU within 48 hours and discharged home without incident.

Conclusion: A series of case reports involving the first three CCB toxic ingestions where HCI (10 units/mL) was successfully used in place of SCI (1 unit/mL). All patients survived. No crystallization of HCI was observed during therapy. Our institution has standardized HCI (10 units/mL) solution for use in CCB toxic ingestions due to concerns over amount of volume administered. Further studies on the long-term stability and efficacy of insulin at similar concentrations are warranted.
239. THE CASE OF THE MASQUERADING OCTANE BOOSTER
C. M. Deutsch1, S. Banerji1, A. C. Bronstein1, D. Tremblay2
1 Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA
2 Poudre Valley Hospital, Fort Collins CO USA

Background: Methylcyclopentadienyl manganese tricarbonyl (MMT) is an organomanganese compound used as a fuel additive/octane booster. It was banned in the United States from 1977-1995 but is currently available as a fuel additive. Human exposure data is lacking.

Case Report: A 50 year old generally healthy male called the Poison Center (PC) 5 minutes after drinking a 1-ounce bottle of LHP Liquid Horsepower (Manhattan Oil, New Braunfels, Texas). He mistook the octane booster for a 1-ounce convenience store “energy shot.” After consuming, he reported feeling “shaky.” The PC obtained the MSDS from the manufacturer which listed the following active ingredients: MMT 60-100%; Solvent Naptha, Petroleum, Heavy Aromatic 30-60%; 1,2,4-Trimethylbenzene 1.0-4.9%; Napthalene 1.0-4.9%; and 1,3,5-Trimethylbenzene 0.1-0.5%. The PC referred the caller to the emergency department (ED). Within 1 hour of ED arrival vital signs were: heart rate 77 beats per minute, respiratory rate 17 respirations per minute, and room air oxygen saturation 94%. He complained of cough and upper airway irritation. Aluminum/magnesium antacid was given. Chest radiograph was consistent with pre-existing COPD. Within 3 hours post ingestion, he experienced a 2-minute generalized seizure that responded to lorazepam. Following the seizure he was weak and ataxic. He developed fluorescent-red diarrhea (the color of the product). Symptoms resolved with supportive care. After no further seizure activity, he was discharged the following day.

Case Discussion: MMT is available in octane boosters in high concentrations. Seizures have been reported in experimental animals with MMT exposure, but the incidence is not well described. In this case, product packaging resembled a widely advertised energy “shot.”

Conclusion: PC and consumer awareness of this “look alike” product is important as the trend in energy “shots” and drinks continues to increase. Product appearance should be considered by manufacturers, stores, and regulatory agencies especially when automotive products with similar packaging are sold near food and drink.

240. REPEATED VANILLA EXTRACT OVERDOSE: A SIMPLE CASE OF ETHANOL TOXICITY?
Joshua W. Russell1, L. K. French2, Nathanael J. McKeown2
1 Oregon Health and Science University, Portland OR USA; 2 Oregon Poison Center, Portland OR USA

Background: Ethanol is a commonly used solvent in many commercial food flavorings and additives. These preparations generally contain > 30% ethanol and are available for purchase without restriction. We describe an atypical case of ethanol intoxication resulting from vanilla extract ingestion.

Case Report: A 37 YO man with history of alcoholism and prior intoxication with vanilla extract a month earlier, was found unresponsive with 33 mg/dL serum ethanol. PC obtained the MSDS from the manufacturer which listed the following active ingredients: 2 fl oz. Pure Vanilla Extract (Manhattan Oil, New Braunfels, Texas). He mistook the octane booster for a 1-ounce convenience store “energy shot.” After consuming, he reported feeling “shaky.” The PC obtained the MSDS from the manufacturer which listed the following active ingredients: MMT 60-100%; Solvent Naptha, Petroleum, Heavy Aromatic 30-60%; 1,2,4-Trimethylbenzene 1.0-4.9%; Napthalene 1.0-4.9%; and 1,3,5-Trimethylbenzene 0.1-0.5%. The PC referred the caller to the emergency department (ED). Within 1 hour of ED arrival vital signs were: heart rate 77 beats per minute, respiratory rate 17 respirations per minute, and room air oxygen saturation 94%. He complained of cough and upper airway irritation. Aluminum/magnesium antacid was given. Chest radiograph was consistent with pre-existing COPD. Within 3 hours post ingestion, he experienced a 2-minute generalized seizure that responded to lorazepam. Following the seizure he was weak and ataxic. He developed fluorescent-red diarrhea (the color of the product). Symptoms resolved with supportive care. After no further seizure activity, he was discharged the following day.

Case Discussion: MMT is available in octane boosters in high concentrations. Seizures have been reported in experimental animals with MMT exposure, but the incidence is not well described. In this case, product packaging resembled a widely advertised energy “shot.”

Conclusion: PC and consumer awareness of this “look alike” product is important as the trend in energy “shots” and drinks continues to increase. Product appearance should be considered by manufacturers, stores, and regulatory agencies especially when automotive products with similar packaging are sold near food and drink.

241. USING THE TOXIC REGISTRY TO INVESTIGATE THE BEDSIDE DIAGNOSIS AND TREATMENT OF SEROTONIN SYNDROME
Kristin M. Engebretsen1, Anthony F. Pizorn2, Christopher J. Misfeldt1
1 Regions Hospital, St. Paul MN USA; 2 University of Pittsburgh School of Medicine, Pittsburgh PA USA; 3 University of Minnesota College of Pharmacy, Minneapolis MN USA

Background: ACMT’s ToxIC group developed a registry of bedside consultations in January of 2010. Over five thousand cases seen by toxicologists nationwide have been entered into this database. This constitutes a database that can be used to help understand and advance the practice of clinical toxicology.

Objective: With the increased use of serotonergic medications, the diagnosis of serotonin syndrome (SS) has continued to increase. Our objective is to describe the characteristics surrounding the diagnosis and treatment of cases entered into the “Toxic Registry” as SS.

Methods: The ToxIC Case Registry was searched from January 1, 2010 through April 4, 2011. All cases entered as SS were further classified based upon demographics, agents involved, signs and treatments.

Results: A total of 105 cases were identified as having SS. Forty-six cases (43.8%) were males and 59 (56.2%) female. Twenty nine cases (27.6%) involved a single agent, 72 (68.6%) had multiple drug ingestions and 4 cases (4%) had no agents documented. The pharmacological classes most commonly associated with SS were antidepressants (70%), antipsychotics (15%) and anticonvulsants (13%). The top serotonergic drugs involved were citalopram (n = 22; 21%), dextromethorphan (n = 12; 11.4%), bupropion (n = 12; 11.4%), lithium (n = 11; 10.5%), sertraline (n = 9; 8.6%), venlafaxine (n = 9; 8.6%) and tramadol (n = 8; 7.6%). Medications not commonly thought to cause SS that were reported as associated with SS included acetaminophen and codeine in 9 (8.6%) cases, meperidine (n = 1); lithium (n = 1); and cyproheptadine in 7 (6.7%) cases.

Pharmacological treatments consisted of benzodiazepines in 51 (48.6%) cases and cyproheptadine in 47; 44.8%) and agitation (n = 43; 41%). The most common sign reported was hypertension (n = 56; 53.3%) followed by tachycardia (n = 51; 48.6%), delirium (n = 47; 44.8%) and agitation (n = 43; 41%). Hyperthermia was reported in 16 (15.2%) cases. The data collection sheet of the online registry does not have a checkbox for ocular clonus/nystagmus or clonus. A text box is available for documentation, but no cases had these symptoms manually entered. Given the lack of documentation of ocular clonus/nystagmus or clonus, we were unable to compare how many patients met Sternbach’s or Hunter’s Criteria for diagnosis.

Pharmacological treatments consisted of benzodiazepines in 51 (48.6%) cases and cyproheptadine in 7 (6.7%) cases.

Conclusions: The ToxIC Registry was a useful tool for review of SS and allowed identification of medications not commonly thought to cause SS. Future iterations of the ToxIC Registry may wish to include some data fields currently not collected.

242. SUMMARY OF THE RISK EVALUATION AND MITIGATION STRATEGIES (REMS) APPROVED BY THE FOOD AND DRUG ADMINISTRATION (FDA) SINCE THE START OF THEIR USE IN 2007
Brenna Farmer
Weill Cornell Medical College, New York NY USA
Background/Objective: The FDA gained authority to require manufacturers to provide strategies to ensure risks were outweighed by benefits for drugs and biological products through The Food and Drug Administration Amendments Act of 2007. The Objective of this project is to summarize the types of products and REMS components that have been produced since as toxicologists can use these components when considering a risk mitigation strategy for drugs and biologicals on their hospital formulary that do not have FDA REMS.

Methods: The FDA website was used to find what products have REMS and the most common REMS components implemented since the start of the program in 2007. The types of drugs or biological products requiring REMS were determined. The number of each REMS component(s) (as set by the FDA) was determined: medication guides for patients and healthcare providers, communication plan, elements to ensure safe use, and an implementation system. Further summarization is pending.

Results: 184 products have had REMS in place, with 1 of these products being released from requiring a REMS (Sucralfate Oral Solution Oral Solution due to no increased risk of allergy related adverse events). Products are from a wide range of classes: antibiotics (mainly fluorquinolones), antivirals, antiretrovirals (usually containing abacavir), chemotherapeutics, immune modulators, antidepressants, anticonvulsants, sedatives, opioids (fentanyl, oxycodone, buprenorphine), opioid antagonists (naltrrexone products); anti-diabetic agents, sedatives, butotolinum toxin, bowel preps for procedures, isotretinoin, testosterone creams, and erythropoiesis stimulating agents. Specifically, the REMS components used include: 180 products have medication guides, 49 products have communication plans, 22 products have elements to ensure safe use, and 18 products have implementation systems. 62 products have more than 1 component as part of their REMS. 6 products have all 4 components (Aranesp®, Epogen/Procrit®, Neulasta®, Nplate®, Onsolis®, Zyprexa Relprev®). 12 have 3 components (Abstral®, Entereg®, Extraheal®, Isotretinoin, Letairis®, Luponex®, Lumyzyme®, Promacta®, Revlimid®, Suboxone®, Thalomid®, and Tracleer®), and 44 have 2 components. Conclusions: The FDA has used its REMS program to focus on 183 drugs and biologics that are considered high risk due to toxicity and to increase their possible benefits. Medication guides are the most common component of REMS. Toxicologists can use these REMS as examples for how to develop a risk mitigation plan for (high risk/toxic) products that have not been required to implement a REMS by the FDA.

SESSION IV

243. ENHANCING SURGE CAPACITY: BUILDING UPON A PARTNERSHIP FORGED WITH STATE PUBLIC HEALTH

Robert J. Geller1, Gaylord P. Lopez1

1Georgia Poison Center, Atlanta GA USA; 2Emory University-Department of Pediatrics, Atlanta GA USA

Introduction: The specter of pandemic influenza has elevated planning for surges in call volume. State and Federal planners have recognized the value of medically sophisticated contact centers, such as poison centers, in addressing this. Our current support for and interactions with State Public Health created the opportunity for our poison center (PC) to collaborate further.

Background: Our state public health colleagues asked us to develop a strategy and plan that provides the capability of answering a sudden large influx of calls. Many entities plan around the “1% of the service area’s population calls each day” metric.

Methods: We used a systems approach to address this, building on existing IT and telephony infrastructure and PC operational templates. Working with technology partners, a cost-effective, novel application of existing technology was utilized to handle a large call volume occurring rarely, suddenly, and unpredictably. In order to be prepared for an outbreak, rigorous planning must develop and validate a set of operating principles. First is the availability of adequate phone/data capacity to receive a large number of calls and contacts, which must incorporate current trends in social networking. Staffing must also be addressed: Who will answer the phones and where will they be located while they answer the calls? What percent of callers will utilize automated messaging, and what percent will insist on talking to a person? Finally, just-in-time-training and data collection must also be included.

Results: We selected a premises-based approach, given our experiences and expertise in managing this strategy. High density on-demand call traffic flow (utilizing SIP trunking, a type of VOIP) was added to our existing telephony infrastructure, which already permits teleworking of a distributed workforce. Combined with voice recognition and text-to-speech tools, this provides initial automated response to 480 simultaneous calls. We anticipate that some callers will insist on talking to a live person, and this solution simultaneously permits 100 to speak to state staff located at diverse locations across the state. The system will have the capability of playing multiple messages in English or Spanish, will route calls to designated sites or individuals based on caller location, and will automatically collect epidemiologic data.

Conclusions: Building upon a long collaboration with State Public Health, we developed an approach to handling call surges which is also applicable to many sophisticated contact centers. The PC gets additional call handling capabilities and enhanced caller interaction. State Public Health gets medically sophisticated call surge capability.

244. UTILIZING KEY PERFORMANCE INDICATORS IN MEASURING EFFICIENCY AMONG TELEWORKERS

Gaylord P. Lopez1, Natika Wright1, Robert J. Geller2, Stephanie L. Hon1

1Georgia Poison Center, Atlanta GA USA; 2Emory University-Department of Pediatrics, Atlanta GA USA

Introduction: Teleworking has become an emerging initiative among many companies in an effort to better utilize their current workforce. By globalizing and evolving their scheduling strategy, poison center managers presuppose cost savings for their company, lowered carbon emissions, a plan of action for emergency preparedness, and improved job satisfaction among their employees. Assessing productivity of employees while teleworking as opposed to an in-office workplace will be critical in evaluating the effectiveness of this program. Equally as important is their efficiency as compared to other colleagues with a comparable workload.

Background: In February of 2009, our center implemented 10-hour work shifts for our staff of Specialists in Poison Information (SPIs). Four months later, we launched our telework initiative for 10 SPIs who met predetermined criteria. As we look to expand this initiative to a broader staff base, Key Performance Indicators (KPIs) and a corresponding standard of measure have been established to assess the overall program’s degree of efficiency.

Methods: Utilizing data from previously produced monthly SPI productivity reports, KPIs were established for the 21 SPIs we analyzed in this study. These KPIs included the number of cases handled per SPI, time SPI was available to receive a call, time SPI was idle, rate of incoming calls per hour, and average wrap-up time per call. Automatic call distribution (ACD) and workload data was gathered for each KPI in three different settings. We compared seven teleworkers productivity during a telework shift and a non-telework shift to other SPIs during a comparable non-telework shift.

Results: Comparison between teleworkers working at home and in the office, and teleworkers compared to office-based staff, showed less than 10% variation on most KPI measures. Only availability to answer ACD calls varied significantly. Teleworking SPIs were available for a larger amount of time during their teleworking shift compared to their own shifts in-office and to office-based staff (for 10 hr shifts, 9 hr 4 min, vs. 8 hr 38 min and 8 hr 33 min respectively; p < 0.05 by ANOVA).

Conclusions: By establishing a core set of Key Performance Indicators, we were able to establish a means of evaluating the effectiveness of the telework initiative and setting standards before expanding it to a wider workforce. Teleworkers proved to be just as productive during their teleworking shift than their non-teleworking shift, and some were even more productive.

245. US POISON CENTER PUBLIC HEALTH 2011 JAPAN EARTHQUAKE RESPONSE

Stuart E. Heard1, Alvin C. Bronstein2

1California Poison Control System/UCSF, San Francisco CA USA; 2American Association of Poison Control Centers, Alexandria VA USA

Background: Poison Centers (PCs) are uniquely positioned as 24/7 public health resources. PCs, through AAPCC, have an established relationship with CDC to collaborate on the surveillance of emerging public health events. PCs partnered with CDC and other public health entities to assist with public health
247. AN IVR MEDICATION IDENTIFICATION SYSTEM—IT IS WHAT IT IS!

Edward P. Krenzelok1, Rita Mrvos
Pittsburgh Poison Center, Pittsburgh PA USA

Background: The 2009 AAPCC NPDS report identified 1,057,632 medication identification requests (MIR) to poison information centers. This represents 24.7% of all calls to US poison information centers. While MIR contribute valuable insight into the misuse and abuse of prescription medications, the sheer volume of MIR presents a logistical, and sometimes ethical, challenge to specialists in poison information (SPI) and a potential burden on poison information center efficiency. To reduce the impact of MIR on a poison information center, an RPIC developed and implemented an automated medication identification system that utilized an interactive voice response (IVR) system. The Objective of this project was to describe how the IVR affected the RPIC MIR call volume and workload of the RPIC SPI staff.

Methods: All documented MIR inquiries from January 1, 2007 through February 28, 2011 were extracted from the RPIC Visual Dotlab electronic medical record system. The inquiries were tabulated by frequency by month. Descriptive statistics, presented as means, were used to characterize the monthly call volume inquiries.

Results: Over the 19 months preceding the implementation of the IVR MIR system, there was a mean of 4,389.6 MIR per month that required manual electronic documentation by SPI. In the immediate 12 months following the IVR MIR system implementation, a mean of 2132.6 inquiries per month were managed by the IVR and SPI documented manually a monthly mean of 190.6 MIR. During the most recent 12 month period there was a monthly mean of 712.9 IVR MIR and a mean of 118.1 required manual electronic documentation by SPI. The triage of MIR to the IVR resulted in a dramatic reduction in workload (as measured by the number of inquiries that required manual documentation into the electronic medical record) from 4309.6 per month to a current monthly volume of 118.1. The combined monthly total of MIR managed by SPI and the IVR decreased to 830.1, representing an annual volume decrease from 52,675 to 9,961. While the IVR reduced workload it also had the unintended consequence of reducing the overall volume of MIR by over 50% and essentially changed the culture of those who sought the identification of prescription medications. This has a multitude of implications, both positive and negative.

Conclusions: The IVR was successful in reducing the number of MIR that required manual electronic documentation by SPI and freed up a substantial amount of time for SPI to perform other critical patient care-related responsibilities.

248. SEVEN YEAR FOLLOW UP OF A STATE-BASED POISON CONTROL CENTER COST EFFECTIVENESS STUDY

Edward Bottei, Linda Kalin
Iowa Statewide Poison Control Center, Sioux City IA USA

Background: Poison control centers (PCC) continue to need to justify their existence in these difficult financial times. In 2004, at the request of our state health department, our PCC performed a cost effectiveness study within our state. We have repeated this study on an ongoing basis and now present the Results of our 2010 study.

Methods: In 2010, 317 callers to our PCC, who were appropriately managed at home, were asked what alternative action they would have taken had the PCC not been available. Callers were also asked what type of health care insurance they had. The final disposition of the 317 callers was determined by an algorithm developed in 2004 and updated regularly. The average cost of an emergency department visit, a doctor’s office visit and a 5-mile ambulance run were determined. ED visit charges were based solely on hospital and physician charges; no charges for pharmacy, nursing, observation or interventions were included. ED and physician charges were an average of level 2 and level 3 acuity (CPT codes 99282 and 99283). Office visit charges were based on level 3 acuity (CPT code 99213).

Results: Overall, 73% of the callers would have ended up in an ED, 8% would have gone to their doctor’s office, 6% would have called 911, and 19% would have been managed at home. Our very conservative estimate of the total charges for these medical services would be $127,657, or $402.70 per call. The actual
OIL SPILL RESPONSE

Kathy L. Jacobitz

CENTERS FOR REVENUE

249. THE EFFECT OF HOSPITAL BILLING ON POISON CENTER REVENUE

Kathy L. Jacobitz1, Claudia L. Barthold2
1Nebraska Regional Poison Center, Omaha NE USA; 2University of Nebraska Medical Center, Omaha NE USA

Background: A U.S. poison center (PC) serving multiple regions initiated a billing process for calls from all acute care hospitals (ACHs) located in one state. This was done to supplement the PC's budgetary shortfall. These ACHs were notified by letter that a set fee would be charged for each case for which they contacted the PC. They were invoiced for ACH-initiated contacts on a per-case basis. The fee covered unlimited calls to the PC for each case. Multiple exposures were billed as one case. ACHs were not charged for PC-initiated calls. ACHs were also given the option of contacting the PC to discuss an alternate contractual arrangement. Contract amounts were individualized by ACH and based on prior call volumes. The purpose of this study is to evaluate the financial impact of this ACH billing process.

Methods: A retrospective examination of all ACH invoices and contracts over a 17-month period (10/1/09 to 2/28/11) was performed to determine the revenue generated and its impact on the PC’s overall budget.

Results: ACHs were invoiced a total of $362,684 over a 17-month period. Thirty-five percent ($128,538) of the total amount was for contacts and 100% was collected. Sixty-five percent of the total amount ($234,146) was invoiced on a per-case basis and 98% ($229,142) was collected. During the study period, the PC collected a mean of $21,040 per month. The median was $20,990. This funded 16% of the PC’s annual operating expenses. As a result, the PC’s budgetary shortfall was reduced by 32%. The number of ACHs with contracts gradually increased. By the end of the study period, 40% of ACHs had agreed to annual contracts, paying a monthly fee for unlimited calls. Thirty-nine percent of critical access hospitals (as defined by the Centers for Medicare and Medicaid Services) and 30% of non-critical access hospitals had contracts. Ninety-four percent of the ACHs with annual contracts elected to renew them.

Conclusions: Contributions by ACHs provided sufficient revenue to support 16% of a regional PC’s annual operating expenses during the study period. This funded 32% of its budgetary shortfall. Annual contracts allowed unlimited calls to the PC and provided a steady source of income for the PC. Critical access hospitals were more likely to have contracts than non-critical access hospitals. The majority of income came from ACHs that paid on a per-case basis. Individualized ACH contracts and per-case billing are both viable Methods: of generating income for PCs.

250. THE ROLE OF POISON CENTERS IN THE 2010 GULF OIL SPILL RESPONSE

Mark L. Ryan1, Thomas C. Arnold2
1Louisiana Poison Center, Shreveport LA USA; 2LSU Health Sciences Center, Shreveport LA USA

Background: An explosion occurred on the drilling rig Deepwater Horizon on April 20, 2010. The release of oil that occurred in the months after the rig sank is among the largest in history, with some estimates exceeding 200 million gallons before the well was finally capped on July 15, 2010. An oil slick was noted at the site of the accident on April 22, touching off massive response efforts involving tens of thousands of individuals.

Conclusions: We have demonstrated the feasibility of promoting CO safety practices within the pediatric primary care setting, and have successfully increased the number of installed CO detectors within our clinic population.
Participants in the program reported enhanced knowledge of CO dangers. This program shows the promise of advocacy education for health care providers, and provides a model for future collaborative partnerships between primary care centers, health departments, poison centers, and the community. Future goals are to investigate measures to increase enrollment rates, and to expand programs and educational outreach further into the region.

252. POISON CENTER OPERATES PUBLIC INFORMATION HOTLINE FOR 2009/2010 NOVEL H1N1 PANDEMIC

G. M. Bogdan1, G. Lewis2, A. M. Seroka1, N. Enyart3
1Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA; 2Colorado Department of Public Health and Environment, Denver CO USA

Background: In April 2009, the state public health department needed our center to provide 2009/2010 Novel H1N1 pandemic information via a separate and established public information hotline. Within hours of activation, staff were trained on the evolving “swine flu” pandemic and began taking calls. State and local public health agencies provided information on their specific H1N1 response efforts to improve communications with public and lessen burdens on their agencies. Our center has been part of our state’s public health emergency response strategy since 2003 and has handled over 300,000 calls related various public health incidents. Our Objective is to describe the changing information needs during two waves of the 2009/2010 H1N1 pandemic.

Methods: Our toll-free public health information hotline has the capability to take 1,000 calls/hour using telephony and integrated voice response technology. Callers listened to voice recordings 24 hrs and/or spoke to a live agent regarding from 7am to 11pm. Agents used approved Frequently Asked Questions (FAQs) from state epidemiologists to answer inquiries. Our database tracked caller zip code, reason for call, FAQs provided and data on self reported cases of influenza-like illness. We use descriptive statistics to report on call metrics and information inquiries for the two waves of pandemic.

Results: During the first wave of H1N1 (04-26-09 to 05-29-09) we received 5,763 calls requesting information. Call volumes increased to 29,916 callers for the second wave of H1N1 (08-02-09 to 01-10-10). Half the callers in the first wave relied primarily on continually updated recorded messages while a majority (68%) of second wave callers waited to speak to an agent. The maximum weekly call volume was 3,981 and occurred week of 11-08-09. Throughout the H1N1 response, caller inquiries changed from “What are the symptoms of novel H1N1 flu in people” and “What should I do if I get sick?” during the first wave to “When will 2009 novel H1N1 Influenza vaccine become available?”. “Where can I get a H1N1 vaccination” during the second wave. Call metrics and situational awareness data were regularly provided to state and local public health agencies to assist them in their surveillance and disease control strategies.

Conclusions: Our public health information capabilities allowed our local and state public health departments to focus on H1N1 response activities while providing the public with accurate and timely information during the two waves of this pandemic. This model indicates another role poison centers can fulfill during public health emergency events.

253. POISONINGS REPORTABLE TO HEALTH DEPARTMENTS WITHIN THE UNITED STATES AND ITS TERRITORIES

Robert French1, Daniel E. Brooks3, Kenneth Komatsu1, Robert England2
1Arizona Department of Health Services, Phoenix AZ USA; 2Maricopa County Department of Public Health, Phoenix AZ USA; 3Banner Good Samaritan Medical Center, Phoenix AZ USA

Background: Select poisonings may represent a public health threat and thus warrant involvement by public health authorities. This study’s aim was to determine the extent to which five poisonings are reportable to State or Territorial Health Departments (SHD). Based on local poison center and SHD experience we identified five poisonings where an individual case may indicate an ongoing public health threat: carbon monoxide (CO), envenomation by a non-native species (ENNS), mercury (Hg), unexplained methemoglobinemia (MetHgb), and methanol (MeOH).

Methods: All SHDs identified by the Council of State and Territorial Epidemiologists were included. Lists of reportable diseases from SHDs’ websites were reviewed for the presence of the five poisonings. Data related to any other reportable poisonings were also recorded. Categorical variables are reported as percentages. Institutional review board approval was not required for this review of public data.

Results: Lists of reportable illnesses were found for 53 of the 57 SHDs (92.9%); American Samoa, Northern Mariana Islands, Puerto Rico, and the US Virgin Islands lists were not found. Six (11.3%) SHDs require reporting of CO poisoning (Connecticut, Florida, Iowa, Louisiana, Missouri, and New York City). Eleven (20.8%) SHDs require reporting of Hg poisoning (Connecticut, Florida, Iowa, Louisiana, Missouri, Nebraska, New Jersey, New Mexico, New York State, New York City, and Wisconsin). Five (9.4%) SHDs require reporting of MetHgb (Iowa, Missouri, and Nebraska; New Mexico and Wisconsin limit this to infants). None require reporting of ENNS or MeOH toxicity. Of note; New York City and Mississippi list all poisonings called to the poison control center as reportable diseases; Utah requires reporting of all chemical poisonings that result in death or hospitalization.

Discussion: Select poisoning-related illnesses may represent ongoing public health threats. Despite this, few SHDs require reporting of five identified illnesses. Limitations to this study exist. Inclusion of only these poisonings introduced bias. The lists found on the SHDs’ websites may not accurately represent actual reporting requirements. Lastly, poisonings may be reportable to agencies other than the SHD, such as local or tribal health departments, and thus may have been missed.

Conclusion: A small proportion of SHDs require reporting of five poisonings that may represent a public health threat. Poison Control Centers may serve a role in enhancing public health situational awareness and possible response.

254. ALL SMOKE AND NO FIRE? A RETROSPECTIVE ANALYSIS OF FIREWORKS INGESTIONS REPORTED TO A SINGLE POISON CENTER

Michael R. Christian1, Brian C. Fort1, Mark B. Myck3
1The Toxikon Consortium, Chicago IL USA; 2University of Illinois at Chicago, Chicago IL USA; 3Cook County Hospital (Stroger), Chicago IL USA

Background: Ingestion of fireworks is commonly reported to North American poison centers, but published data is limited on the extent of this toxicologic problem and how best to manage these ingestions. Fireworks may contain various metals (e.g., arsenic) and other metabolic toxins (e.g., barium), and a recently published case report described significant prolonged toxicity and hospitalization in a patient after ingestion of consumer-grade black snake fireworks.

Objectives: To determine how often fireworks ingestion is reported to a large regional poison center, describe the patient and clinical characteristics associated with fireworks ingestion, and describe the clinical course of these patients.

Methods: We performed a retrospective analysis of consecutive fireworks ingestion cases between 1/1/2002 and 12/31/2010 managed by a regional poison center with an annual call volume >100,000 cases. All clinical data were prospectively entered in real time into a structured database. Variables and outcomes were determined a priori and data abstraction was performed in a systematic manner according to the guidelines of Gilbert.

Results: Out of 198 cases of fireworks exposures, 172 met study inclusion criteria. The median age was 2 years (range: 6 months–49 years); 98 were male (56%), 123 (71.5%) of exposures occurred in June, July, or August. Fireworks involved were: snakes (80; 46.5%), sparklers (36; 20.9%), firecrackers (21; 12.2%), pop-pops (18; 10.5%), and other (17; 9.9%). 169 patients were asymptomatic, but 13 (7.6%) patients had nausea/vomiting and 4 (2.3%) patients had abdominal pain. Only three patients were lost to follow-up. 18 (10.5%) patients were evaluated at a health care facility, but only one patient was hospitalized and was discharged the following day. 154 (89.5%) cases were categorized as “no effect,” 18 (10.5%) as “minimal effect,” and no case met moderate, major, or death severity by NDPS criteria.

Conclusions: Most fireworks ingestions occur in young children who remain asymptomatic. Even symptomatic cases did well with minimal treatment. Future research should confirm if unintentional firework ingestion by children who are asymptomatic can remain home without emergency department evaluation.
255. DEVELOPMENT AND IMPLEMENTATION OF A MEDWATCH® REPORTING PROGRAM BY A LARGE REGIONAL POISON CENTER

Ashley N. Webb1, Anna Rouse Dulaney2, Sallie A. Dea1
1School of Pharmacy and Pharmaceutical Sciences, University at Buffalo, SUNY, Buffalo NY USA; 2Carolinas Poison Center, Charlotte NC USA

Background: The FDA Medwatch® reporting system provides continued safety of medications through post-market analysis. In keeping with their goal to “protect public health”, Medwatch® allows careful review of adverse events. Although reporting is voluntary, healthcare providers are encouraged to report a “clinically significant” event even when they are unsure of its relationship to a “serious event”. This allows the FDA to track patterns of events and determine if additional steps are warranted to improve product safety. Poison Centers are ideal participants in Medwatch® due to the calls they manage concerning adverse drug events (ADEs), access to data and clinical staff to determine significance, and specialists in Poison Information (SPIs) trained to gather information. Here, we report the frequency of Medwatch® submissions at one Regional Poison Center.

Methods: In 2008, 9,568 calls were recorded in October, with 120 cases coded as ADEs. Upon review, 10 cases met criteria for Medwatch® reporting. The reportable cases comprised 0.1% of the total call volume for the month; decidedly not a significant time burden on the SPIs. The staff was then educated on Medwatch® and appropriate coding for reportable cases, and a box was made available in the data collection software for flagging a potential Medwatch® case for review. Several months were studied to review the SPIs’ selection and appropriateness of coded cases. A second educational meeting provided feedback to the SPIs to improve coding and documentation.

Results: From August 2009 through April 2011, 53 cases tagged for Medwatch® were reviewed and 21 were submitted. Of the cases not reported, 25% documented known and expected ADEs, 13% documented symptoms deemed unrelated to the exposure, 11% were suicide attempts, and 0.04% were misuse of the product. Only one case did not include enough data for submission. While this is below the expected volume of reportable cases, it was the SPIs’ selection and appropriateness of coded cases. A second educational meeting provided feedback to the SPIs to improve coding and documentation.

Conclusions: Limitations include the need for staff re-education and the time required of clinical staff to review cases. Case submission is not a factor, as this is accomplished electronically by a poison information provider. To address these deficiencies, pharmacy students completing their experiential rotations in toxicology/drug information are now being utilized to review cases and will reduce the time burden on clinical staff. Additionally, brief re-education sessions at several monthly staff meetings throughout the year will improve the overall number and appropriateness of cases flagged for Medwatch® by SPIs.

256. USE OF ROBOTIC TELEPRESENCE FOR POISON CENTER BASED MEDICAL TOXICOLOGY CONSULTATIONS: TELETOXTM

A. C. Bronstein1, P. Huang2, S. Banerji1, A. Gilbreath2, T. Whitmore2
1Rocky Mountain Poison & Drug Center-Denver Health, Denver CO USA; 2Saint Alphonsus Regional Medical Center, Boise ID USA

Background: Regional Poison Centers (PCs) routinely provide hospital and telephonic medical toxicology consultation. Although telephonic consults are limited to voice, hospitals can fax/email test results, and substance/clinical pictures on request. However, this process does not match bedside consultation. To improve on this provision of care, we piloted toxicology consultation via remote presence robots at a metropolitan and rural hospital system: TeleTox™.

Methods: We partnered with an 8 hospital telehealth network (metropolitan hub and 7 rural hospitals; bed range 10 - 361) serving one state using either RP-7 or RP-Lite telepresence robots (InTouch Health, Santa Barbara, CA) to converse with hospital emergency departments and critical care units. Training with the robots was conducted for all participating medical toxicology fellows and attendings. RP-7 Robots with 30 frame/second video for real-time motion capture can sense obstacles and move under manual video control to safely gain clear patient access. An initial robot laptop base station was replaced with a hardwired, encrypted desktop and T1 line in the PC. Consultations were initiated by the patient’s hospital by contacting the hub-hospital’s Access Center, which then contacted the toxicologist using the PC 800-number. Special patient consent was not required as no video recording was made. Initially Access Center personnel drove the robots as directed by the consulting toxicologist, and were available to coordinate transport if necessary. Headphones were available for both the toxicologist and SPI. Consult notes were entered into the PC case management system. The treating health care provider remained in charge of the patient’s care with the toxicologist making recommendations as usual. Initially, TeleTox™ consults were available 9 am - 5 pm Monday – Friday.

Results: A series of 2 mock cases was conducted with each toxicologist and ED staff from participating hospitals. Toxicologists also “beamed” in to 8 staff meetings to meet the remote ED physicians and staff. TeleTox™ went live on 14 February 2011. A total of 2 consultations have been done. TeleTox™ consult length appears longer than similar telephone consultations. Data continues to be accumulated. All TeleTox™ patients survived.

Conclusions: Robotic toxicology telepresence (TeleTox™) is a promising PC application. TeleTox™ has been well accepted by PC staff, physicians, and patients. Expansion to SPI follow-up calls and extended consultation hours with individual laptop systems outside the PC is possible. TeleTox™ may also have applicability for cross-town PC consultation with multiple hospitals or during public health events.

257. A CSPI’S USE OF SPEECH RECOGNITION SOFTWARE: A SUCCESS STORY

Donna Smyth, Anne M. Gallo, Heather Hudson
Ontario Poison Centre, The Hospital for Sick Children, Toronto ON Canada

Background: Computers have become an integral part of Poison Centre functioning. Not coincidentally, the occurrence of musculoskeletal disorders (MSDs), including repetitive strain injury (RSI), has become more prevalent. RSI has been linked with the overuse of muscles and tendons associated with the fine hand manipulation required in keyboarding and use of a computer mouse. As a result, a provincial Workplace Safety Insurance Board has identified that MSDs account for 42% of all lost time claims.

Case Report: After many years of being employed as a full time Certified Specialist in Poison Information (CSPi) at a Poison Centre (PC) serving a population of 13 million, a CSPi developed a significant RSI. This RSI involved pain and decreased mobility in the fingers of both hands. As poison case management occupied 100% of this CSPi’s time, this resulted in a significant impediment to this CSPi’s former productivity. In order to manage the symptoms, medical treatment included a three week leave of absence, anti-inflammatory drugs and cortisone injections. Workplace modifications included shortening the length of worked shifts, assigning a permanent workstation and purchasing a split-key soft touch keyboard. As these efforts were only partially successful in managing the symptoms, the decision was made to install Speech Recognition software for this CSPi’s exclusive use. Prior to the installation the CSPi spent a considerable amount of time researching information about the software; including reviewing the manufacturer’s user guide and attending webinars to increase familiarity and proficiency. Subsequently, the CSPi was able to utilize the software in managing 100% of the CSPi’s call volumes within three days of the installation. Since the installation, the software has learned and adapted to the CSPi’s speaking style to further increase efficiency. The CSPi has returned to full-time status and is able to manage call volumes that are above average when compared to colleagues who are manually entering cases. The CSPi no longer experiences the stress that previously came from an inability to function at a pre-injury level, and has seen a decrease in physical discomfort.

Case Discussion: Specific features of this Speech Recognition Software that contributed to the success of the pilot included its ability to recognize medical terminology, diverse accents, create templates for future use, the ability to customize the pre-set vocabulary and its compatibility with the PC’s documentation software.

Conclusion: The use of Speech Recognition Software is a viable solution for poison centre staff suffering from RSIs to return to a productive and efficient work level.
258. **COLLABORATION OF POISON CENTER WITH NURSING ADVICE LINE WAS ASSOCIATED WITH INCREASE IN CAPTURE OF THERAPEUTIC ERRORS AND ADVERSE DRUG REACTIONS**

Nancy G. Murphy1, Stacy Ackroyd Stolarz2, Kimberley Sheppard1, MaryAnne Carew2

1WK Regional Poison Centre, Halifax NS Canada; 2Dalhousie Department of Emergency Medicine, Halifax NS Canada

**Background:** Poison Centers are poised to become a valuable resource in capturing therapeutic errors (TEs) and adverse drug reactions (ADRs). This data may make a significant contribution to the identification of areas for preventive strategies as well as post-marketing surveillance, both of which may improve patient safety. The public may not be aware that poison centers are a resource for information regarding TEs and ADRs. Most Canadian provinces have access to 24 hour nursing advice lines for non-emergent health queries from the public. Prior to the implementation of a province-wide nursing advice line in Nova Scotia, our poison center collaborated with those responsible for the service to ensure that all drug-related calls were transferred to us in the interest of optimal patient care. We present data that shows this collaboration to be temporally associated with a significant change in the number of calls categorized as TEs and ADRs.

**Methods:** As part of a quality assurance project, the annual poison center statistics were generated from the electronic database, Visual DotLab. The increase in TEs and ADRs was noted; the same statistics generated from 2008 were then compared with 2010 statistics. No statistical analysis was performed.

**Results:** The following compares the number of calls in 2008 with 2010 (before and after implementation of the nursing advice line): the total number of ADRs increased from 69 to 406. The total number of TEs increased from 639 to 1063. In terms of outcomes, moderate and major effects from ADRs increased from 9 to 71, and for TEs, from 15 to 35. The most common drug category implicated in ADRs was antimicrobials, and for TEs, hormone and hormone antagonists (eg insulin).

**Conclusion:** The increase in drug-related errors and adverse reactions described may not be exclusively related to the collaborative relationship with a new nursing advice line. However, cited reasons for the general increase in reported TEs and ADRs, such as polypharmacy, an aging population, or co-morbidities, would be unlikely to change significantly in such a short time period. Collaborative partnerships with other health care agencies may improve capture of therapeutic errors and adverse drug reactions. Poison Centers can provide valuable data that may guide preventive strategies and improve patient safety via enhanced post-marketing surveillance.

<table>
<thead>
<tr>
<th>Time period</th>
<th>Calls received</th>
<th>Rabies PEP recommended (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>May 1 2007 – April 30 2008</td>
<td>1356</td>
<td>436 (32.2%)</td>
</tr>
<tr>
<td>May 1 2008 – April 30 2009*</td>
<td>1758</td>
<td>554 (31.5%)</td>
</tr>
<tr>
<td>May 1 2009 – April 30 2010</td>
<td>1608</td>
<td>567 (33.4%)</td>
</tr>
</tbody>
</table>

*Rabies vaccine restricted.

260. **TOXICOLOGIC HAZARD VULNERABILITY ANALYSIS: A TIMELY TOOL**

Anthony J. Tomassoni1, Greg Bogdan2, Vinita Sinha3, Christopher Lee1

1Yale School of Medicine, Department of Emergency Medicine, New Haven CT USA; 2Rocky Mountain Poison and Drug Center, Denver CO USA; 3Yale New Haven Hospital, New Haven CT USA

**Purpose:** Natural and manmade disasters have heightened awareness of risks from chemical and radiological disasters/events (CRE). In order to respond well to these incidents, good planning that is tailored to the special circumstances of such events needs to occur. For example, previous studies document inadequate regional stockpiling of antidotes to treat exposures such as cyanide or cesium releases. Federal resources such as the Strategic National Stockpile may not be immediately available. Therefore communities must prepare themselves for likely CRE incidents. A Systematic Hazard Vulnerability Analysis (HV A) facilitates hazard identification and resource triage, informing planners to reduce risks, plan effective response to CRE and make best use of scarce resources/funds. HVA requires knowledge of local hazards and history. Regional poison centers/toxicologists hold unique knowledge, connections and positions to perform a “Toxicological” HVA (THVA), interpret results; and plan for protection of responders and patients. With enhancements to existing HVA tools a serviceable THVA could be created.

**Methods:** CRE vary by the agent(s) involved, the number of individuals affected (ex. single drug overdose vs. mass casualty HAZMAT disaster), the urgency with which chemical countermeasures (CCM) must be administered, CCM availability, deployment time, staff training in recognition & treatment of toxic hazards & other variables. We modified a popular HVA tool to include CRE risks and related variables including preparedness measures in spreadsheet format. This tool helps prioritize CCM needs, by inventorying chemicals and drugs in and passing through the community using data elements including agents, CCM, quantities on hand, event magnitude, probability, risks to life, health & social fabric, urgency of treatment, and mitigation undertaken to minimize impact and optimize recovery.

**Results:** A THVA tool for use by toxicologists and other medical planners has been developed to improve the planning process for CRE mitigation and response. It highlights agents for which CCM and/or special training may be made available.

**Conclusion:** A focused THVA tool may facilitate planning/prioritization for CRE to optimize training, design CCM stockpiles, prioritize purchases, develop staging and administration protocols for force protection.
of responders and patients. Although focus on CRE is expected to improve results attainable by many planners, limitations of the THVA are similar to those for HVAs: performance depends upon the quality of data entered, final interpretation and implementation of the results.

261. RETROSPECTIVE REVIEW OF POISON CENTER DATA FOR UNINTENTIONAL BETA-BLOCKER AND/OR CALCIUM CHANNEL BLOCKER INGESTIONS

P. Dommer1, C. A. Truitt1, D. E. Brooks2, F. LoVecchio3
1Banner Good Samaritan Poison & Drug Information Center, Phoenix AZ USA; 2Department of Emergency Medicine, Maricopa Medical Center, Phoenix AZ USA

Background: Beta-blocker (BB) and calcium channel blocker (CCB) toxicities are common poisonings reported to poison control centers (PCCs) and may lead to significant morbidity and mortality. Outcomes following unintentional supratherapeutic ingestions of these agents have not been well studied.

Objective: We conducted a retrospective chart review of cases referred to our PCC in hope of better understanding patient outcome after unintentional BB/CCB ingestions.

Methods: Reviewers underwent standard training for systematic electronic chart review. Crystal Reports™ (2010, Williamsville, NY) search engine was run on all PCC charts from 1/1/100 through 10/31/09 (> 1 million calls) for cases relating to BB and/or CCB ingestions. This subset of charts was manually reviewed by trained investigators. Cases involving unintentional supratherapeutic ingestions of a BB and/or CCB were recorded on a data abstraction sheet. Patient age, gender, all medications, doses, time since last dose, symptoms and outcome were recorded. Exclusion criteria included intentional ingestion and ingestion of someone else’s medication. Outcomes were defined as the development of symptoms (hypotension, bradycardia, dizziness/lightheadedness or abnormal glucose), hospital admission and death. Ten percent of reviewed cases were reviewed by a second investigator to ensure validity.

Results: 448 charts met inclusion criteria. 421 (94%) had complete data. 17 patients (3.8%) refused medical advice/treatment; 10 cases (2.2%) were lost to follow-up. Mean age was 64.5 yrs (range 2 to 97; SD = 16.3 yrs) from data recorded in 435 (97.1%) of all charts. Gender was documented in 447 (99.7%) of charts; 292 (65.3%) were women. 84 (19.9%) patients were evaluated in an ED/hospital; 51 (60.7%) of these were referred in by our PCC. Symptoms developed in 42 (9.9%) of patients. 25 (5.9%) of all patients were admitted for a direct result of the ingestion. Of admitted patients, 2 (8%) received treatment (glucagon) specific for the ingestion and 14 (42.4%) had no effect or were discharged within 24 hours. Only 2 patients initially watched at home were later referred to an ED; neither required treatment or hospital admission. There was one death unrelated to the CCB ingestion.

Conclusions: Approximately 10% of cases involving unintentional, supratherapeutic ingestions of a patient’s own BB or CCB resulted in symptoms; very few of which required treatment. Home observation of asymptomatic patients is safe in the majority of cases.

262. POISON CENTER SURVEY OF PRIMARY CARE OFFICES: TRIAGE OF POISONING CALLS WITHOUT A REGIONAL POISON CONTROL CENTER

Daniel E. Brooks, Travis Austin, Sharyn Welch, Frank LoVecchio
Banner Good Samaritan Poison and Drug Information Center, Phoenix AZ USA

Background: Poison control centers (PCCs) save health care resources, yet annual threats of funding cuts continue. Our PCC routinely conducts quality assurance surveys to identify our need and role within our community.

Objective: We hypothesized that primary care providers (PCPs) would refer most poisoning-related calls to either 911 services or an emergency department (ED) if our PCC were to close.

Method: Adult and/or pediatric PCPs within our region were identified via an internet search. We developed a survey with three poisoning-related scenarios based on common calls to our PCC. Trained investigators contacted PCP offices via phone and asked to speak with an office manager or triage personnel. The first 100 consecutively completed surveys were recorded and analyzed.

Survey Question: ‘Assuming there were no regional poison control centers,’

Table 1. PCC Survey of PCP offices.

<table>
<thead>
<tr>
<th>Survey Question</th>
<th>Responses to Questions (n = 100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>The patient had an accidental ingestion of an unknown pill?</td>
<td>Come to the office 33 Call 911 59 Go to the ED 7 Other 911 or ED 3</td>
</tr>
<tr>
<td>The patient had an accidental exposure to fumes from an oven cleaner and has eye and throat irritation?</td>
<td>Come to the office 45 Call 911 45 Go to the ED 7 Other 911 or ED 3</td>
</tr>
<tr>
<td>The patient was stung on the foot by a scorpion and is having localized pain and paresthesias?</td>
<td>Come to the office 28 Call 911 18 Go to the ED 52 Other 911 or ED 2</td>
</tr>
</tbody>
</table>

Results: The survey questions and Results are in Table 1. Responses included in the “Other” category were ‘referral to an urgent care or pharmacy’, and ‘discuss with a physician.’ We also asked “would there be a difference in handling these scenarios for after-hours calls?” Responses to this question included: refer all patients to an ED (46%), no difference (25%), page the oncall physician (17%), 911 (11%), and call a nurse line (1%).

Conclusions: Based on our survey, 82.5% of poisoning-related calls to primary care offices in our region would be referred to 911 or an ED if our poison center closed. These Results further support the role of poison control centers in saving health care resources.

263. NARRATIVE TEXT IMPROVES PERFORMANCE OF POISON CENTER PREDICTIVE MODELS

Mollie R. Cummins, Anusha Mathukutty, Seth Latimer, Heather K. Bennett, E. Martin Caravati, Barbara I Crouch, Lee Ellington University of Utah, Salt Lake City UT USA

Background: If PCC staff can quickly identify callers who are unlikely to adhere to emergency department (ED) referrals, the staff may be able to more effectively manage poison exposures. To achieve this purpose, PCC staff may use tools that predict adherence on the basis of routinely collected data. We constructed predictive models of adherence using National Poison Data System (NPDS) data elements and narrative text entered by PCC staff, in order to determine whether inclusion of narrative text increased model performance.

Methods: Our sample consisted of all human exposure cases occurring at one PCC during a three-year period for which the cases were referred to a health care facility for emergency evaluation. We created coded representations of the narrative text using singular value decomposition. We included only narrative text entries within 10 minutes of the initial call to the PCC. We used automated search Methods to identify the optimal subset of coded NPDS data elements and/or narrative text for prediction of non-adherence in three age groups: all ages, children (age < 18), and adult (age ≥ 18). Using artificial neural networks, decision tree induction, and logistic regression, we induced multiple possible predictive models of adherence for the three age groups using: only NPDS data elements, only narrative text, and combined NPDS data elements/narrative text. We validated models using one year of reserved data. We calculated non-parametric area under the receiver operating characteristic curve (AROC) for model comparison.

Results: AROC values for predictive models based on NPDS data elements ranged from 0.64-0.71. AROC values for predictive models based on both NPDS data elements and narrative text ranged from 0.53-0.73. For 11 of 14 models, the inclusion of narrative text resulted in increased performance. The mean difference in AROC = 0.02.

Conclusions: Coded representations of narrative text improved the performance of predictive models of caller adherence to PCC referral to EDs. The NPDS data elements accurately described key caller and exposure characteristics, and so the narrative text may reflect additional PCC staff concerns and impressions resulting from the communication process. In clinical decision support applications, PCCs should consider the modest improvement in predictive modeling.
realized with additional natural language processing in the context of clinical implications and increased processing times necessary to include narrative text.

264. EXOTIC VENOMOUS SNAKEBITE DRILL  
Rittirak Othong1, Sophia Sheikh1, Nahar D. Alruwaili1, Rachel Gorodetsky1, Brent W. Morgan1, Brad Lock1, Ziad N. Kazzi2

Background: The National Poison Data System has reported 41-93 patients/ year exposed to exotic venomous snakes from 2005 to 2009. According to the Association of Zoos and Aquariums’ recommendation, institutions housing venomous animals should have protocols in place for appropriate and timely transport of envenomated individuals to hospitals. The Objective of this study was to use a functional exercise to evaluate the existing exotic snakebite protocol (ESP) that is used by our local zoo.

Methods: Prior to the exercise, all involved parties were contacted, including the poison center (PC), zoo, emergency medical services (EMS), receiving hospital emergency department (ED) and pharmacy. A checklist of all required actions in the ESP was created and used during the exercise. The exercise was divided into 4 phases that were evaluated by independent observers: zoo, EMS, PC, and hospital ED. The zoo component was further divided into 3 action lists (victim, assistant, and zoo dispatch). After the drill, we held a debriefing session and generated an after action report that was submitted to the zoo, PC, and hospital administrations.

Results: We found that the ESP contained procedures for zoo personnel that were easy to follow, but the procedures for hospital personnel lacked details regarding signs and symptoms expected from each species; indications, dosing, reconstituting and forms (liquid vs. powder) of each antivenin (AV). Zoo personnel performed almost all required actions (93%-victim, 100%-assistant, 93%-zoo dispatch). EMS and ED personnel completed 90% and 78% of the listed tasks while PC personnel completed 25% of the tasks. Additionally, we discovered that pharmacy was not included in the ESP, even though reconstituting and dosing the exotic AV consumed time (22 minutes). Finally, we encountered problems communicating the ESP to the ED and pharmacy due to the PC phone system problem. Despite the identified shortcomings, the time from simulated envenomation to AV administration was under an hour.

Conclusions: This drill identified several potential issues that led us to revise our protocol by adding signs and symptoms expected from each species; indications, dosing and reconstituting of each AV, and a pharmacy section. We also identified suboptimal PC response in the application of the ESP. This may be due to the fact that some of the PC staff were aware this was a drill and may have not followed the ESP. Finally, we encouraged PC staff to find alternative ways communicating the protocol to the ED and pharmacy such as pre-positioning it in the ED and pharmacy, calling and talking directly to the treating physician instead of relying on faxing. This drill provided a good opportunity to evaluate and improve our existing system.

265. DEMOGRAPHICS AND OUTCOME OF UNINTENTIONAL INSULIN OVERDOSES MANAGED BY POISON CENTERS  
Michael C. Beuhler1, Henry A. Spiller2, Alfred Alegus3

Background: The number of patients requiring insulin for diabetes management is growing, as are the numbers of types of insulin, thus potentially increasing the frequency of insulin dosing errors. Insulin dosing errors can lead to dangerous hypoglycemia. Poison Centers (PCs) assist in managing many of these patients, but the characteristics and outcomes of these exposures managed by PCs are not known.

Methods: Case notes of insulin exposures with reason coded as therapeutic error, unintentional general or unintentional unknown from 1/1/2009 to 10/31/2010 were reviewed from three poison centers (PC). Polypharmacy not involving insulin and cases with routes of exposure other than parenteral were excluded. Data abstracted included age, type and dose of insulin, presence of clinical hypoglycemia, lowest blood sugar, administration of food, management site, exposure site, and outcome. Information in the case text notes superseded coded data. Insulin types were grouped into short, medium, and long duration as well as combination products. Variables are expressed as mean ± standard deviation.

Results: A total of 642 cases were included; average age was 57.5 years and 59.5% of exposures were female. Site of exposure was own residence in 97.5% of cases; 77.9% of these exposures were managed on site without health care facility (HCF) referral. The most common time of day of exposure was 9 pm. When known (98.4%), the average exposure amount of insulin was 46.8 ± 47.8 units, with short duration insulin being the most common insulin group type (64.3%). Average follow up time (excluding those not followed) was 6.9 ± 5.0 hours. The average total number of calls made by the PC for each case was 4.2 ± 1.9. The benign outcomes of no effect, minor, confirmed non-exposure and unrelated effect occurred 81.9% of the time. Moderate outcome occurred 8.4% of the time. Only 4.8% of cases had outcome of unable to be followed, judged as potentially toxic by the specialist. Short duration insulin exposures were associated with an increased frequency of moderate outcomes compared to non-short duration insulin exposures (10.2% vs. 5.2%, p=0.037). Clinical or numerical (blood sugar <70) hypoglycemia occurred 15.9% of the time with all cases.

Conclusion: Insulin dosing accidents are routinely managed at home by PCs. Even with an average of 4 PC calls per case, this may represent a cost savings over the required prolonged observation (likely more than 6 hours) in an HCF. Outcomes were good with no major outcomes or deaths and the majority of cases (90.3%) followed to a known outcome. Insulin dosing accidents can be managed at home by PCs and they have a low rate of symptomatic or numerical hypoglycemia.

266. CALLERS’ WILLINGNESS TO USE POISON CENTERS AS DISASTER TELEMEDICINE PROVIDERS  
Cassandra N. Harrison1, Arthur Chang1, Daviesha L. Rice1, Sarah J. Reedy1, Robert J. Geller1, Stephanie L Hon2

Background: During a public health emergency, such as an epidemic H1N1 situation or radiological crisis, telemedicine has great potential to address hospital surge capacity by managing care for patients who have mild or no symptoms. Poison Control Centers (PCC) can serve a large number of patients using set treatment guidelines under the supervision of medical professionals. In order to optimize call center disaster telemedicine services, it is important to understand public opinions.

Methods: Participants were selected each month from recent callers to the Georgia Poison Center (GPC). Eligibility included members of the general public who were at least 18 years old, had placed a call to the GPC between two weeks and two months prior to study enrollment date, and received management relating to their exposure at home. The research employed was a cross-sectional survey design. The survey consisted of 21 items which assessed the demographic characteristics of participants, knowledge and attitudes towards telemedicine, and aspects related to a hypothetical epidemic of H1N1 flu.

Results: Based on the eligibility, 491 PCC callers were eligible for enrollment. Individuals were then called at random and invited to participate. In total, 116 individuals completed the telephone survey and were enrolled in the study. Of these participants, 84.5% were female and the mean age was 38 years old; 69.8% indicated they were white, 22.4% black, and 2.6% Hispanic. Level of education varied, but 73.3% of respondents indicated having completed some college or more. Many callers (45.7%) reported living in a suburban area. Based on the telephone survey, 66.4% of respondents indicated they were aware of telemedicine and 72.4% indicated they were likely or very likely to use telemedicine services. In addition to demonstrating knowledge and likelihood, 67.2% of participants said they were very or extremely willing to follow medical care instructions through telemedicine for treating themselves and 61.2% for treating other family members.

Discussion: During a public health crisis, telemedicine may provide medical advice for patients with minor or no symptoms and has potential to alleviate capacity constraints at acute care hospitals and medical care facilities. The majority of participants had knowledge of telemedicine, were likely to use it, and were willing to follow telemedicine instructions for themselves or other family members during a public health emergency.
Conclusion: This study supports the use of poison centers as potential telemedicine providers during a public health emergency. Telemedicine could serve as an additional resource for treatment during times of overwhelming demands on the acute care system.

267. WHAT DO OTHER COUNTRIES WANT FROM THE ACMT? RESULTS OF THE AMERICAN COLLEGE OF MEDICAL TOXICOLOGY INTERNATIONAL COMMITTEE SURVEY

David M. Wood¹, Paul Wax², Lewis Nelson³, Rais Vohra³, Paul I. Dargan¹
¹Guy’s and St Thomas’ NHS Foundation Trust, London UK; ²American College of Medical Toxicology, Phoenix AZ USA; ³New York University, New York NY USA; ⁴UCSF-Fresno Medical Center, Fresno CA USA

Background: The International Committee of the American College of Medical Toxicology (ACMT) was established as a forum for members from outside of the USA and to provide support to its International members. Since it was not clear what the exact needs of medical toxicologists around the world were, an Internet-based survey was designed to determine these needs and we describe here the interim Results of this survey.

Methods: A multi-page self-completion on-line questionnaire was designed and trialled by the ACMT International Committee. Delivery was through an on-line survey portal, with individuals invited to complete the questionnaire by personalised electronic mail invites to key individuals within 81 different countries. The key individuals were identified using personal contacts through the ACMT, and where possible only one representative individual per country was invited to complete the questionnaire. The questionnaire included sections on medical toxicologist recognition/accrual and training, poison centres, antidote availability, laboratory support, training and teaching, and a needs assessment including how the ACMT could support these needs.

Results: Responses were received, within the first month, from individuals representing 43 different countries (response rate 53.1%). Medical toxicology was a recognised speciality in only 12 (27.9%) countries, however, medical toxicologists (physicians performing clinical toxicology) were recognised in 21 (48.3%) countries. Recognition of medical toxicologists was through collegiate recognition 13 (61.9% of countries), membership of national or international societies (7,33.3% and 5, 23.8% respectively). Although 13 (30.2%) countries had a recognised training programme, only 7 (16.3%) recognised specialists by formal certification. Common themes identified in the current and anticipated needs in relation to medical toxicology amongst those who responded, included the need for training, antidote supply, laboratory services, development of poison centres and research funding. It was felt that recognition of medical toxicologists through Fellow-ship of ACMT (FACMT) would be beneficial in countries where specialists were not formally recognised.

Conclusions: There are a number of potential streams of support that ACMT and other International toxicology societies could undertake to provide support and improve the status of the speciality of medical toxicology across the world. In addition, it appears that whilst these workstreams may require some funding, it appears that whilst these workstreams may require some funding, the needs of those who responded does not appear to be purely financial.

268. POISON CENTER UTILIZATION RESULTS IN AVOIDANCE OF MEDICAL CARE EXPENDITURES

Marsha D. Ford, Michael C. Beuhler, Rebecca S. Rembert
Carolina Poison Center, Charlotte NC USA

Background: Each year most human poisoning exposures called to poison centers (PCs) are managed on-site at a non-healthcare facility (HCF); in 2009, 72.5% were managed in this manner (source: National Poison Data System). Previous studies indicate 33-70% would seek medical care if PCs were not available. This study utilizes caller survey data, from a subset of calls made to a large public health PC that serves over 9 million people and receives over 100,000 calls annually, and medical care cost data to determine cost avoidance when a PC manages these cases.

Methods: People who contacted a single PC from 1/18/2011 to 3/31/2011 for human exposures [coded: exposure site – own residence; management site - managed on-site (not a HCF)] were called and asked to participate in a survey. The survey was administered by Specialists and Providers to callers from 10 to 21 days earlier. The survey explanatory script, questions, and caller answers were recorded in Survey Monkey™. Callers were asked what they would have done if there was no PC [called 911, gone to MD office, gone to urgent care, gone to emergency department (ED), stayed at home, not sure or other] and what insurance coverage the patient had (private, Medicare, Medicaid, Tricare, none). The costs of seeking medical care outside the home (external care) were: 911 transport-$532; MD office or urgent care visit-$98; ED visit-$332. A random sampling bootstrap method with replacement utilizing 200 iterations was performed to determine average and standard deviation of the sample; subgroup analysis was performed for the Medicaid-covered patients. Using the number of calls of similar type for one year, the annual cost to manage these exposures was extrapolated; similar calculation was performed for the Medicaid subgroup. Values are expressed as mean ± 1.96 standard deviations.

Results: There were 679 complete survey responses out of 3165 cases called/attempted to call; the latter represents 61% of all the calls to the PC with the same exposure and management site as the surveyed calls (n=5184) that occurred during the survey period. Those stating they would seek external care if the PC was unavailable were 82.0% ± 4.1%. In 2010, 47,029 calls had the same exposure and management site as the surveyed calls. The estimated cost for external care in the absence of a PC was $8,905,000 ± $919,000; cost for the Medicaid subgroup was $4,003,000 ± $795,000.

Conclusion: Based on caller self-reporting of seeking medical care outside the home if the PC was not available, management by a PC of human exposures occurring in their own residence, managed on site (not HCF) avoided annual medical care costs of nearly $9 million; in the Medicaid subgroup, annual medical care costs avoided were $4 million.

269. HEALTH POLICY AND POISON CONTROL CENTERS: PROVIDING ANALYSIS UTILIZING A LOGIC MODEL

Teresa Dodd-Butera¹, Molly Broderick²
¹CSU San Bernardino, San Bernardino CA USA; ²California Poison Control System, San Diego CA USA

Background: Despite evidence that poison control centers (PCCs) provide cost-effective public health measures and access, threats for decreased public funding necessitates improved evaluation Methods. A logic model provides an illustration of an underlying conceptual framework and the logical connection within and between systems.

Methods: A logic model was utilized to analyze the policy of decreasing public spending for PCCs. An underlying assumption was that PCCs provide essential public health services. Inputs included stakeholders and resources impacted by the policy to decrease public spending. The logic model also considered negative outcomes for both outpatient and inpatient services which are impacted if decreased funding levels lead to the elimination of access to PCC services.

Results: Immediate impact of decreased public spending would deny access to certain essential public health services, particularly significant to vulnerable populations. Short and long-term outcomes would include decreased utilization of appropriate resources; and increased costs and imposition on emergency services and resources, respectively. In addition, there is a potential for increased morbidity and mortality due to an absence of preventive services to the public and impediments to training of clinical toxicologists.

Conclusions: Logic models for analysis of public health policy for PCCs can be effective in establishing causal models and connectedness of systems. In addition, it provides a conceptual and visual frame of reference for professionals, politicians, and the public which increases transparency for fiscal decisions; and eliminates the appearance of budget reduction in the case of decreased public spending for PCC services, which would actually increase spending for other health services.
FU calls also serve to determine patient outcome in relation to the acute toxic episode. With increasing workloads both at the CAPQ and in the critical care units, our specialists in poison information (SPIs) were questioning the relevance of maintaining some FUs. The SPIs were also asking for an update of the FU criteria. They wanted more Objective criteria that would help them in making clinical decisions as to whether or not to initiate or discontinue FU. As an organization, we aimed to improve our patient outcome data.

Method: The CAPQ created a workgroup that surveyed all of the SPIs and the toxicologists for their specific suggestions to improve our FU criteria. The workgroup also reviewed the American Association of Poison Control Centers’ certification criteria concerning the outcome data, and examined the FU procedures from four Canadian, two American and one European poison control centres. Based on these data, the workgroup crafted a brand-new FU procedure which was tested by the SPIs during the months of December 2010 and January 2011.

Results: Following the test period, the workgroup came up with a modified proposal that took into account the SPIs’ comments in terms of workload; this satisfied the majority of the staff. In terms of outcome data, we compared January 2010 and January 2011 and found that we had a 47% increase in the number of cases with known specific outcomes and a 43% decrease in the number of “potentially toxic – no known outcome” cases.

Conclusion: The SPIs, especially the less experienced ones and the trainees, are quite satisfied with the improved and more precise FU procedure. They feel that they can rely on more Objective and specific criteria in deciding to initiate or discontinue the FUs. We can also confidently say that this new FU procedure is more helpful in gathering outcome data than our previous procedure.

271. THE IMPACT OF INCENTIVES ON PARTICIPATION IN A REGIONAL POISON CENTER’S CUSTOMER SATISFACTION SURVEY PROGRAM

Lloyd Herrington, Stephanie Hon1, Natika Wright1, Robert J. Geller2
1Georgia Poison Center, Atlanta GA USA; 2Emory University, Atlanta GA USA

Background: Customer Satisfaction Surveys (CSS) are valuable for assessing customers’ needs and expectations and the quality of services provided. The American Association of Poison Control Centers’ rules for Certification of Regional Poison Centers (RPC) state, “the Certified Poison Center should demonstrate monitoring of customer satisfaction,” but provide no criteria. Past attempts to increase the number of CSS completed have included SPI incentives and daily quotas for medical secretaries working in this RPC. The purpose of this study was to determine the impact of an incentive to callers for participation in a CSS.

Method: At the end of the initial call, SPIs were to offer callers the option of taking a live CSS then, on follow-up, or to complete the CSS on-line at the RPC website and told about a drawing for a $25 gift card. Callers who declined the live option were referred to the Center’s website for survey completion. On-hold messages alerted callers to the CSS as well. The SPI was to enter the caller’s response into the chart. Non-exposure calls for information were also eligible. Calls that required immediate medical attention and calls originating from a healthcare facility or healthcare workers were excluded.

Results: Of the 10,047 eligible cases from an 8 week period, 972 (9.7%) were offered participation in the CSS. Of those, 339 (34.9%) accepted live, 412 (42.5%) were referred to the website, 220 “declined to participate”. The actual number of CSS completed live was 395 and 43 were completed on the website. Only 64% of the live CSS participants opted to enter the drawing. For the same period one week apart, we had a 47% increase in the number of cases with known specific outcomes and a 43% decrease in the number of “potentially toxic – no known outcome” cases.

Conclusion: The SPIs, especially the less experienced ones and the trainees, are quite satisfied with the improved and more precise FU procedure. They feel that they can rely on more Objective and specific criteria in deciding to initiate or discontinue the FUs. We can also confidently say that this new FU procedure is more helpful in gathering outcome data than our previous procedure.

272. RIDING THE WAVE OF CHANGE: REFUGEES TRIUMPH IN POISON PREVENTION EDUCATION

Gayle Finkelstein1, Karen McKnight1, Melissa Heinen2
1Fletcher Allen Health Care, Burlington VT USA; 2Salus Consulting, LLC, Minneapolis MN USA; 2Northern New England Poison Center, Portland ME USA

Background: It can be difficult for refugees who are not native English speakers to understand the poison center’s role and when to call. Lack of awareness and language or cultural barriers may contribute to underutilization of poison center services. It is important to develop materials and provide education to overcome these barriers.

Methods: Three refugee groups (Bhutanese, Burmese and Swahili- or Kirundi-speaking central Africans) in a rural state were educated about poison center services and poison prevention in 2010. The groups represented both newly resettled and established refugee populations. This IRB-approved project involved two sessions, one week apart. The trainings were promoted by a non-profit resettlement agency and consisted of interpreted pre- and post-surveys, a pre-recorded PowerPoint presentation and review of key prevention behaviors. Graphic-based magnets and translated postcards were distributed. A follow-up survey was administered one week later to assess knowledge retention and behavioral changes. All de-identified data were analyzed for statistical significance (at p â‰£ 0.05) using T-Tests with SPSS software. As an incentive for completing the one-week follow-up survey, a water bottle was given to each participant.

Results: Initially there were 64 participants (18 Burmese, 34 Bhutanese and 12 Swahili- or Kirundi-speakers). The pre-survey found that only 19% (n = 12) had ever heard of the poison center. There was variation between groups. The Swahili-speakers, a more settled group, had the greatest initial awareness (46%) and the Bhutanese the least (5%). Post-survey Results: showed a statistically significant improvement in knowledge of ways poisons can enter the body and when to call the poison center versus 911 for a child medication-related poisoning. 73% (n = 47) completed the one-week follow-up survey. Participants were asked to complete five prevention steps. All of these steps were completed by at least 84% of participants and two were completed by 100%.

Conclusion: Poison prevention education to non-English speaking refugees is effective in increasing awareness of poison center services and changing behaviors. Participants’ feedback showed strong interest in the topic and available resources. Several Swahili-speaking participants requested to become future peer educators. Some limitations include: possibly confusing and poorly designed questions; variation in interpretation skills; potentially affecting participants’ responses; possible sharing of answers; small study size; significantly lower participation percentage for the one week follow-up session; and inability to match follow-up responses with pre- and post-surveys.

273. MASS CARBON MONOXIDE POISONING IN A SCHOOL: A CALL TO ACTION

Katherine Hart, Amy L. Hanoian-Fontana, Lauren M. Salinger, Marc J. Bayer
Connecticut Poison Control Center, Farmington CT USA

Background: Our Poison Control Center (PCC) was contacted about a mass carbon monoxide (CO) exposure in a public elementary school serving grades K - 5. Thirty-two fifth grade students, ages 8 to 11 years old, and 3 adult staff members were treated at two local emergency departments for CO poisoning. Symptoms in most patients included headache, nausea and dizziness, and carboxyhemoglobin levels up to 12% were measured after some treatment with 100% oxygen. CO levels of 40 to 125 ppm were measured inside the school. All patients were treated with normobaric 100% oxygen for 4 - 6 hours; symptoms resolved and all were discharged home. Investigation revealed the source was a malfunctioning boiler, and also revealed that carbon monoxide detectors were not present in the school.

We discovered that there is no state requirement for carbon monoxide detection / alarm equipment in schools. CO, a colorless, odorless, potentially deadly gas, is produced during incomplete combustion of any carbon-based fuel (e.g., fossil fuels, coal, wood, etc.) CO poisoning produces immediate symptoms, but in up to 40% of cases, delayed neurological sequelae may occur, sometimes with devastating cognitive impairment. CO detectors are a crucial secondary prevention strategy to alert individuals to the presence of CO before harm occurs.
Methods: Immediately after the school exposure, our PCC and state Department of Public Health (DPH) collaborated to prepare a public health alert about the risks of CO poisoning in schools; this was distributed to school superintendents and school nurse supervisors. We researched US state and local requirements for CO detection / alarm equipment and, with DPH, testified in support of a bill which would expand our state’s mandate for CO detection equipment in residences to include public and non-public schools.

Results: Twenty-four states require CO detectors in residential buildings, but no state requires this protection in schools. A bill which will require CO detection equipment in schools has received legislative committee approval, and budgetary review was also favorable, reflecting modest cost to implement and maintain CO detectors; action by the full legislature is expected in spring of 2011.

Conclusions: CO poisoning can occur anywhere that incomplete combustion of carbon-based fuel can occur. While many states require CO detection / alarm equipment in dwellings, legislation requiring this same protection for school students is lacking. A small monetary investment in CO detection / alarm equipment for all schools can protect students from CO poisoning and its potentially lifelong effects. PCCs and other public health partners can collaborate to achieve this important public health goal.

274. FIELD TEST RESULTS OF A MEDICINE SAFETY GUIDEBOOK FOR OLDER ADULTS IN ENGLISH, SPANISH AND CHINESE

Lauren Schwartz1, Luz Martinez1, Jean Louie1, Maria Mercurio-Zappala2, Mary Ann Howland2, Robert S. Hoffman1
1NYC Poison Control Center, New York NY USA; 2St. John’s University College of Pharmacy, Queens NY USA

Background: We previously conducted a needs assessment with program directors serving multilingual older adults to assess their needs for a medicine safety program. Based on this assessment we developed a medicine safety guidebook to be used as content for educational sessions. The medicine safety guidebook was produced in English, Chinese and Spanish. Before large scale distribution of materials and program expansion in multiple languages, it is essential that content is evaluated for understanding by the target audience. Herein we report the results of our field testing of this guidebook as an essential tool for assuring its utility.

Methods: The Poison Control Center (PCC) educators conducted a field test of the guidebook utilizing a structured interview technique with older adults who self-identified as native speakers of English, Spanish or Chinese dialects. The Learner Verification and Revision technique (Doak, Doak and Root, 1996) was incorporated into the field test questions to ensure understanding of the written content. Each question in the interview was categorized according to a specific element of this technique: attraction (graphics), self-efficacy, cultural acceptability and persuasion. After reading the medicine safety guidebook, each participant was asked open-ended questions by the PCC educator. After completion of the survey, participants received a tote bag with incentives including PCC information, magnet, pen, medicine box and a round trip MetroCard.

Results: In June 2010, 60 interviews were completed with older adults at three senior centers; twenty in each language. Overall, the medicine safety guidebook was well understood in all languages and 87% of respondents reported that they would call the PCC to ask questions about their medicines. In contrast, 80% of the participants had trouble with the meaning of “drug interaction” and 75% misunderstood the auxiliary labels shown in the guidebook, particularly those participants in the Spanish and Chinese groups. In addition, feedback about the booklet cover images led to a redesign.

Conclusions: An educational program utilizing the medicine safety guidebook will emphasize teaching areas that were identified during the field test as confusing. In particular, the meanings of drug interactions and auxiliary labels will be clarified to assure comprehension.

275. DEVELOPING A MOBILE APP TO PROMOTE THE POISON CONTROL SERVICES

Iana M. Simeonov, Kristina M. Hamm, Stuart E. Heard
UCSF-California Poison Control System, San Francisco CA USA

Background: Poison exposure is the 2nd leading cause of injury and death to children 0-5 years in the U.S. However, parents are largely unaware of poisoning risks, often confused about poisoning prevention strategies and unfamiliar with poison control centers. Over 300,000 mobile apps have been developed in the last three years. The most used mobile apps in the U.S. are games (“gaming”), news, maps, social networking and music. The average smartphone user has 40 apps on their phone. Data from the American Association of Poison Control Centers shows over-the-counter (OTC) and prescription medications accounting for the majority of exposures in children. In a series of recent focus groups, parents also reported concern around colorful “pills” that can be mistaken for a treat.

Objective: Choose the most popular mobile application (app) platform, create and launch a free mobile app, and determine if could be a valuable part of an overall marketing strategy for a poison control program.

Methods: Consumer feedback about how people were playing a popular online poison awareness game led to the consideration of a mobile app. Adult consumers described playing along with their children, teachers reported using the game in elementary and middle school classrooms, and pharmacy students related playing against each other. An large number of consumers reported using an internet browser on a mobile device to play. This led to the conception, design and coding of an app version of the game in 3 weeks. Users can also add 1-800-222-1222 to their contacts list with one click. The app was submitted to Apple, vetted and placed in the Apple App Store in 10 days. A Spanish version has been submitted. A promotion and media strategy heavily targeting bloggers, tech websites, and health reporters was implemented.

Results: The free English app, launched for National Poison Prevention Week 2011, had 200 downloads in its first week, reached 1039 downloads within 3 weeks and has a 5 star rating from reviewers.

Conclusions: The first gaming app for poisoning prevention and service promotion generated over 25 media mentions and articles on traditional news and blogging sites nationally, raising the visibility of poison centers and the issue of poisoning prevention. Gaming has enormous potential to help disseminate public health messages in a fun, engaging and creative way.

276. POISON CENTER ROLE IN RESPONDING TO A CRITICAL DRUG SHORTAGE

Cynthia Lewis-Younger1, Denise Becker2, Michelle Rutledge1, JoAnn Chambers-Emerson1, Katelyn Dervay2, Henry J Pittman1
1Florida Poison Information Center- Tampa, Tampa General Hospital, Tampa FL USA; 2Tampa General Hospital, Tampa FL USA

Background: Coral snake envenomation is rare. Even where bites occur most frequently, few hospitals average more than one bite per year. The underlying rate of morbidity and mortality is unknown, but bites are potentially deadly. Only 1 death has occurred since the approval of the antivenin, but people who have received delayed treatment have had neurotoxicity. Some have required ventilator support. In 2003, Wyeth Pharmaceuticals discontinued manufacturing of North American Coral Snake Antivenin. All remaining product was to expire October 31, 2008. No other FDA approved product is available. As a result, the product has been tested and received three extensions of its expiration date: but the supply continues to dwindle. In discussing the discontinuation, it became apparent that lack of information was common among health care providers in the region. Many health care providers believed that the supply was already exhausted.

Methods: The Poison Center developed a coral snake antivenin discon- tinuation task force to develop a systematic approach to the crisis. The plan included determining how to maximize the current supply, identifying barri- ers to using various alternative antivenins, educating health care providers, and surveying regional hospitals to identify locations of existing antivenin supplies. A webpage was posted and information about the antivenin shortage was faxed to all regional EDs.

Results: The Center established contact with the manufacturer to determine the existing supply of the antivenin. The taskforce identified 20 hospitals in the Poison Center’s region with an existing supply of the antivenin. From April to September, 2010, outreach was conducted by the Medical Director at each of those hospitals providing information about the regional supply, extended expira- tion date, the method for ordering antivenin and current treatment guidelines. The poison center coral snake webpage received 456 pageviews the first month it was posted. There was an increase in exposure calls for coral snakebites from 10 to 17 in the year of the outreach. After the outreach, antivenin stock in the region increased by 50%, from 70 vials to 108 vials.
Conclusion: The Poison Center developed a comprehensive plan to address a significant regional issue. It served a pivotal role in temporarily improving the supply of coral snake antivenin in the region where antivenin was most needed during 2010. It improved the recognition of the poison center as a resource for addressing this regional issue.

277. IMPACT OF A COMMUNITY EDUCATION PROGRAM ON POISON CENTER UTILIZATION IN GHANA
Sari Soghoian1, Caesar Nyadedzor1, Edith Clarke1, Rishi Vohra2, Phyllis Caces2, Lewis R Goldfrank2
1Ghana Poison Control Centre, Accra Ghana; 2New York University School of Medicine, New York NY USA

Background: Opened in 2002, the Ghana Poison Control Centre (GhPCC) is one of only eight poison centers in Sub-Saharan Africa. The core function of the GhPCC is a telephone hotline service offered to healthcare workers and members of the general public seeking information or advice about poisoning cases. Currently this service is underutilized. In 2002-2009 the GhPCC received an average of 15.6 calls annually, of which 74% were from doctors at the Ridge Hospital in Accra, where the GhPCC is located. The present study was conducted to test the hypothesis that a community outreach program to raise awareness of poisoning risks and local resources would improve utilization of the GhPCC hotline.

Methods: This was a prospective longitudinal study evaluating changes in GhPCC call volume and distribution after the implementation of a community health education program on poisoning prevention in the home. Nurses were trained to conduct interactive health talks with mothers of young children using picture-based flip-charts to deliver key messages about kerosene poisoning prevention. Wallet cards printed with the GhPCC contact number were provided for distribution to mothers who received the talks. Nurses were contacted three-months after distribution of the teaching materials to determine program uptake and implementation. Differences in monthly call volume and percent calls from outside the Ridge Hospital in the six months before and after program implementation were compared using the Wilcoxon rank sum test.

Results: 76 nurses working in Ghana Health Services Maternal and Child Health clinics throughout the Greater Accra Region were trained, and 23 were interviewed for the three-month program evaluation. These nurses each gave an average of four talks monthly, reaching an estimated 282 mothers per month. The other nurses had either left their posts (16) or could not be reached by phone for follow-up. Average monthly call volume at the GhPCC increased significantly from three to eight and a half calls per month in the six months pre- and post-implementation (two-tailed p = 0.016). Percent calls from outside the Ridge Hospital also increased from 27% to 62%, but this change was not statistically significant (two-tailed p = 0.093).

Conclusions: Nurses who gave the health talks effectively reached large numbers of mothers. Overall call volume to the GhPCC increased significantly in the six months after program implementation. A trend toward increased utilization of the GhPCC hotline by community members and healthcare providers working outside the Ridge Hospital was also noted. More work is needed to raise public awareness of GhPCC services in Ghana, and to understand the barriers to PC utilization in the region.

278. COMPARISON OF EDUCATIONAL OUTCOMES IN RESIDENTS RECEIVING INTERNET ACETAMINOPHEN CURRICULUM VS. LIVE LECTURE
Andrew Stolbach
Johns Hopkins University, Baltimore MD USA

Background: A great number of poisoned patients are treated by emergency physicians without subspecialty training in toxicology. Medical Toxicology is co-sponsored as a subspecialty by the American Board of Emergency Medicine. Many toxicologists gain interest in their subspecialty during EM residency training. Despite the relationship between EM and medical toxicology, many EM residencies do not have a toxicologist on faculty or a dedicated toxicology rotation. Our Objective was to compare educational outcomes in residents attending a live lecture on acetaminophen poisoning with those completing an internet learning module.

Methods: We developed an internet-based acetaminophen education module. The module was formatted as a series of clinical vignettes. The module concludes with a posttest which was formatted in 4-choice best answer format. Items were written with the assistance of a specialist in education assessment and reviewed by outside toxicologists. The same posttest was administered to three separate groups: 1) “No training”- 33 residents and medical students who had no instruction 2) “Live lecture”- 26 residents who had a live lecture delivered by a senior non-toxicology faculty 3) “Internet module”- 9 residents who took the internet curriculum. The first two groups were a convenience sample of EM residents. The third group consisted of EM residents. The “live lecture” and “internet module groups completed the posttest immediately after the didactics. Three months after completing the module, the “internet module” group curriculum took a second follow-up test with items testing same Objectives as the initial posttest. Outcomes measured were posttest scores (all groups) and 3-month posttest scores (“Internet mod- ule” group only). Results were compared using two-tailed t-test.

Results: The mean score for the “no training” group was 46% (95% CI: 38%-54%). The mean posttest score for the “live lecture” group was 51% (95% CI: 42%-60%) The mean posttest score for the “internet module” group was 77% (95% CI: 65%-91%) and the mean 3-month follow-up score for the “internet module” group was 83% (95% CI: 71%-95%). In the internet learning group the mean posttest score and 3-month follow-up score were significantly higher (p < 0.001) than the other groups.

Conclusions: For emergency medicine residents, internet didactics written by a medical toxicologist resulted in a superior posttest score compared to a live lecture delivered by non-toxicologist faculty. The improved score was maintained 3 months after the internet didactics.

279. RADIOLOGICAL PREPAREDNESS- AND ATTITUDES: A CROSS-SECTIONAL SURVEY OF EMERGENCY MEDICINE RESIDENTS AND PHYSICIANS AT 3 ACADEMIC INSTITUTIONS
Ziad N. Kazz1, Lisa McCormick2, Spencer Adolf3, Frank Walter4
1Emory University; 2University of Alabama, Birmingham AL; 3Penn State-Hershey, Hershey 4University of Arizona, Tucson AZ

Background: Previous research has shown that emergency responders felt unprepared to respond to radiological or nuclear disasters.

Methods: An electronic survey was sent to a total of 309 emergency medicine residents and physicians at 3 U.S. academic institutions. The study was approved by the institutional review board.

Results: The survey response rate was 37%. 52% of respondents were residents and 48% were attending physicians. Only 37% and 28% of respondents had attended any training in radiological preparedness in the preceding 5 years or any training in radiation detection, respectively. In the event of a radiological or nuclear emergency, 48% of respondents felt uncomfortable or very uncomfortable caring for victims in the emergency department and performing decontamination. 56% of respondents felt the same about performing a radiation detection survey on their patients. Additionally, 52% and 68% felt uncomfortable or very uncomfortable diagnosing acute radiation syndrome and internal contamination respectively. When asked about their familiarity with some of the available therapies, 89%, 81% and 65% of respondents were unfamiliar with the use of DTPA, Prussian blue and Filgrastim respectively. Moreover, 65% of respondents stated that they would not care for a critically injured patient until radiological decontamination was performed. Forty-one percent believed that a patient can be externally contaminated with radiological material without being exposed to radiation. Similarly, 79% of respondents believed that a patient can be exposed to radiation without being contaminated with radiological material.

Conclusions: Our Results suggest a need for additional radiological-nuclear preparedness training for emergency medicine residents and physicians. Such training should include radiation decontamination, detection, patient management, and existing therapies. Emphasis should be placed on explaining the secondary hazards from contaminated victims and the differences between radiation exposure and contamination. Our Results show that classroom teaching at
280. UNUSED MEDICATIONS: A CASE REPORT—SHOW ME THE MONEY

Jeanie E. Jaramillo1, Ronica D. Farrar1
1Texas Panhandle Poison Center, Amarillo TX USA; 2Texas Tech UHSC School of Pharmacy, Amarillo TX USA

Background: Unused medications are a known source for poisonings, abuse, misuse, and environmental contamination. In 2009, our poison center and host university began implementing periodic community unused medication take back days to facilitate the removal of these items from households. At each event, detailed information is collected. This information is entered into the Pharmaceutical Collection Monitoring System™, a web-based tool developed by Computer Automation Systems, Inc. to facilitate the on-going collection and reporting of consistent data regarding the collection of unused medications. This information includes drug name, strength, original quantity, collected quantity, fill date, use by or expiration date, manufacturer/distributor, and identifies each product as a sample, a factory sealed item, or an item that appears to be a mail-order. We report an uncommon case in which a large number of items were brought from one household. Although many more items were brought, only the collected controlled substances are reported here.

Case Report: An individual brought the medications of deceased parents to our take back event in the open bed of a pickup truck. These items were contained in two cardboard boxes that were approximately 3 ft x 3 ft and 2 ft deep. Items contained prescription labels, thus allowing them to be identified as prescribed to family members. Identifiers were obscured and not recorded. See Table 1 for results.

Table 1.

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Strength</th>
<th># Containers</th>
<th>Qty Collected</th>
<th>AWP</th>
<th>Low-High End Street Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>temazepam</td>
<td>30mg</td>
<td>66</td>
<td>10,080</td>
<td>$5,352.48</td>
<td>$80,640 - $302,400</td>
</tr>
<tr>
<td>oxycodone/APAP</td>
<td>5/500mg</td>
<td>15</td>
<td>802</td>
<td>$94.64</td>
<td>$802 - $4,010</td>
</tr>
<tr>
<td>methadone</td>
<td>15mg</td>
<td>11</td>
<td>685</td>
<td>$176.50</td>
<td>$1,765 - $2,765</td>
</tr>
<tr>
<td>hydrocodone/APAP</td>
<td>7.5mg</td>
<td>2</td>
<td>32</td>
<td>$102.67</td>
<td>$2,051 - $4,102</td>
</tr>
<tr>
<td>morphine sulfate</td>
<td>10mg</td>
<td>12</td>
<td>144</td>
<td>$7.15</td>
<td>$86 - $350</td>
</tr>
<tr>
<td>oxycodone/APAP</td>
<td>5/500mg</td>
<td>10</td>
<td>600</td>
<td>$94.64</td>
<td>$946 - $4,010</td>
</tr>
<tr>
<td>Lorcet®</td>
<td>10mg</td>
<td>3</td>
<td>180</td>
<td>$363.48</td>
<td>$1,040 - $10,400</td>
</tr>
<tr>
<td>methadone</td>
<td>15mg</td>
<td>4</td>
<td>200</td>
<td>$13.99</td>
<td>$56 - $100</td>
</tr>
<tr>
<td>diazepam</td>
<td>5mg</td>
<td>18</td>
<td>2,320</td>
<td>$167.04</td>
<td>$2,320 - $46,400</td>
</tr>
<tr>
<td>MS Contin®</td>
<td>75mg</td>
<td>6</td>
<td>48</td>
<td>$226.72</td>
<td>$420 - $1,680</td>
</tr>
<tr>
<td>hydrocodone/APAP</td>
<td>10/325mg</td>
<td>4</td>
<td>520</td>
<td>$94.64</td>
<td>$802 - $10,400</td>
</tr>
<tr>
<td>MS Contin®</td>
<td>15mg</td>
<td>2</td>
<td>30</td>
<td>$16.67</td>
<td>$30 - $100</td>
</tr>
<tr>
<td>oxycodone/APAP</td>
<td>5/500mg</td>
<td>15</td>
<td>802</td>
<td>$94.64</td>
<td>$802 - $4,010</td>
</tr>
<tr>
<td>temazepam</td>
<td>30mg</td>
<td>111</td>
<td>13,994</td>
<td>$6,255.79</td>
<td>$85,498 - $368,808</td>
</tr>
</tbody>
</table>

Case Discussion: This report reveals a case in which a large number of prescribed medications remained unused. Many were dispensed by a mail-order pharmacy. If auto-refill processes were involved in this case of extreme wastefulness, a plausible theory, an investigation into such processes seems warranted. The controlled substance items brought to this collection event from a single household had an AWP of over $6,000 with low- and high-end street values of over $85,000 and $368,000. 111 containers holding 13,994 pills came from one household. While we hope this scenario is a rare occurrence, this case leads us to believe that similar cases likely exist.

Conclusions: This case reveals an extreme example of medication waste and displays the importance of collecting and reporting data regarding unused medications.

281. CONSUMER SOURCES AND PREFERENCES FOR HEALTH INFORMATION AND IMPLICATIONS FOR POISON CENTER OUTREACH

Iana Simeonov, Kristina Hamm, Stuart E. heard
University of California San Francisco, San Francisco CA USA

Background: Consumers are increasingly searching online for health information and treatment advice as well getting and giving advice about health through social media. This change has important implications for poison center telephone advice services.

Objective: To explore consumer behavior and its implications for poison center services and determine if the this trend offered an entry point for outreach or promotion opportunities.

Methods: Ten focus groups each with 8-10 participants lasting 90 minutes were fielded in 4 markets in the center’s coverage area. Groups consisted of parents from 4 priority market segments: low-income African-American, Monolingual Latino and Bilingual Latino parents, and an ethnically-divers median income group.

Results: Participants report using a search engine to gather medical information more frequently than going to a specific website. They were more likely to search after an alarming incident with their children than for prevention strategies. For general information, respondents headed to trusted websites such as BabyCenter, WebMD, Mayo Clinic, Ask MD or iVillage.

Conclusions: A 24/7 expert resource for poisoning questions and non-emergency situations remains popular with consumers from all backgrounds. However, as more time is spent online or on a mobile device, preferences for consuming medical information have shifted and poison centers must adapt quickly to remain relevant.

282. ARE EMERGENCY PHYSICIANS PREPARED TO MANAGE SYNTHETIC CANNABINOID INTOXICATION?

Patrick M. Lank1, Elizabeth Pines2, Mark Myrcyk3
1Northwestern University, Department of Emergency Medicine, Chicago IL USA; 2Northwestern University, Department of Preventive Medicine, Chicago IL USA; 3Toxikon Consortium, Chicago IL USA

Background: The use of synthetic cannabinoids (SC) has become more prevalent in the United States. The SC Spice/K2 has been shown to produce more pronounced psychosis, agitation, and withdrawal than would routinely be seen in acute marijuana intoxication. Despite increasing ED visits associated with Spice/K2 use, emergency physicians’ (EP) knowledge of this drug of abuse is unknown. The purpose of this study is to assess attending and resident EP knowledge of SC.

Methods: To perform a needs assessment of EP, we conducted a self-administered, anonymous, voluntary, internet-based survey instrument of resident and attending EP at a large urban ED. The survey instrument used closed-format questions to assess familiarity with the terms Spice and K2, basic knowledge of SC, and practice patterns when managing SC intoxication in the ED. Responses were categorical, binary, or on a 4-point Likert scale depending on question type. Data were analyzed using descriptive statistics and significance tests for comparing proportions.

Results: Of 83 physicians invited to participate, 88% (n = 73) completed the survey instrument, including 47 residents and 26 attending physicians. Only 34.2% of residents had heard of Spice and 49.3% had heard of K2 prior to this study, and these percentages did not differ significantly by age or resident/attending status. Of these, 70% had heard of SC only from non-medical sources—most commonly from lay publications and the internet. Among those who stated previous knowledge of SC, 25% were not aware it is a synthetic drug, 16.7% did not know it is most similar to marijuana, and 50% did not know the active components of Spice and K2 are the same or similar. Surprisingly, 5.6% of respondents believed Spice and K2 are most commonly obtained by physician prescription. Respondents familiar with the terms Spice and K2 scored significantly higher on
knowledge-based questions than those not familiar with these terms (5.56 vs. 4.22, P < 0.0001). Among all participants, 80.2% responded that they feel unprepared caring for a patient in the ED who has used SC. Most physicians (91.5%) responded that they would like more education on emerging drugs of abuse patterns.

Conclusion: Despite significant media coverage and political attention in the last year to SC, clinically active EP are still unfamiliar with these products. Even those who stated they heard of SC scored poorly on basic knowledge questions. More education is needed among emergency physicians of all ages and levels of training on synthetic cannabinoids.

283. PILLS vs. CANDY: A POPULAR ONLINE GAME TO PREVENT POISONINGS

Iana M. Simeonov, Kristina M. Hamm, Stuart E. Heard
UCSF-California Poison Control System, San Francisco CA USA

Background: Poison exposure is the 2nd leading cause of injury and death to children 0-5 years in the U.S. Nationally, nearly 80% of poison exposures occur at home and 51% involve children under 5. However, parents are largely unaware of poisoning risks, often confused about prevention strategies and unacquainted with poison centers as a resource. Data from the American Association of Poison Control Centers shows OTC and prescription medications accounting for the majority of childhood exposures. In focus groups, parents report concern around colorful “pills” being mistaken for treats.

Objective: In 2010, online games overtook personal email to become the 2nd biggest activity behind social networks. To leverage the popularity of games for promotion, “Pills vs. Candy” was developed and launched within three weeks. The game engages players in determining if pills or candy is present in each image. Player choices are tabulated and can be posted on Facebook or Twitter. OTC medications and candy were photographed professionally, a designer created a simple, clean user interface and a developer custom-coded the game using the programming language PHP. The game has its own .com and .org web addresses for English and Spanish versions.

Methods: Players identify seemingly identical pairs of medicine and candy by tapping on a photo. Answers are shown with the next pairing, player scores are tabulated and can be posted on Facebook or Twitter. OTC medications and candy were photographed professionally, a designer created a simple, clean user interface and a developer custom-coded the game using the programming language PHP. The game has its own .com and .org web addresses for English and Spanish versions.

Results: In its first 2 months, the game had 12,000 visits. Most visitors played to the end and scored 65% correct. The initial “bounce rate” (percentage who did not continue) was a low 22%. At first, most traffic was direct, but promotion efforts have increased the number of visitors referred from other sites by 200%. Most recent data shows 409 visits in March 2011 alone, a lowered bounce rate and Spanish versions.

Conclusions: For budget-conscious promotion, “Pills vs. Candy” was developed and launched within three weeks. The game engages players in determining if pills or candy is present in each image. Player choices are tabulated and can be posted on Facebook or Twitter. OTC medications and candy were photographed professionally, a designer created a simple, clean user interface and a developer custom-coded the game using the programming language PHP. The game has its own .com and .org web addresses for English and Spanish versions.

284. A RISKS TOY

S. Denise Holzman, Andrea Clements, Keith Boesen, Farshad Shirazi
Arizona Poison and Drug Information Center, University of Arizona, Tucson AZ USA

Background: Rare earth magnets have powerful properties, such as strong magnetic field and great resistance to demagnetization. These attributes result in compact, light magnets, and make them desirable for use in many products. They have also been incorporated into toys. The same properties that make these magnets valuable in other applications make them hazardous if ingested. Cases of potentially life-threatening effects as a result of magnet ingestions have been reported.

Case report: We present a 3 year old who swallowed 13 rare earth magnets. The magnets, called Buckyballs, were 5 mm each, spherical and smooth. An initial x-ray showed 2 foreign bodies located near his epiglottis and 11 foreign bodies, linked together in a linear fashion, located near the upper portion of his small intestine. The 2 magnets in the upper oropharynx were removed. For three days he received MiraLax 17 grams twice daily, and for two days he was given a sodium phosphate/biphosphate pediatric enema, 66 milliliters (mL) per dose. X-rays were done twice daily to monitor the progression of the magnets. There was no significant change in the position of the magnets until the third day of therapy, when they moved over the right lower quadrant of the abdomen. At this time, Polyethylene Glycol-Electrolyte Solution (PEG-ES), 4000 mL, was given. The PEG-ES was completed by 02:00 on the fourth day of therapy. The next x-ray, taken at 06:00, showed that the location of the magnets was unchanged. The child spiked a temperature, and a decision was made to take him to surgery. An exploratory laparoscopy was initiated, but the magnets were not found. At this time, 13:30, another x-ray was done. This image showed that the magnets had moved into the rectum. The chain of 11 magnets was removed digitally via the rectum.

Case Discussion: This case demonstrates the powerful properties of the magnets, in which 11 of them remained linked together despite three days of therapy. It is notable that on the fourth day the magnets passed into the rectum, still linked together. It may have benefited the child to have the 11 magnets remain together, since the risk of injury would have likely increased if the magnets had become separated.

Conclusions: Because of their small size, and powerful properties, these magnets are easily swallowed and present serious risks, especially to children. The warning label on Buckyballs says to “keep away from all children,” and that if swallowed they “can stick to intestines causing serious injury or death.” Unfortunately, small children are not likely to be able to read or understand such warnings. As these rare earth magnets become more popular, parents and health care providers need to be aware of the potential hazard these toys present.

285. IS IT REALLY AN ALLERGY?

Amanda Parker, Jennifer Lowry
Children’s Mercy Hospital, Kansas City MO USA

Background: Adverse drug reactions (ADR) affect 10-20% of hospitalized patients and more than 7% of the general population. Published rates in hospitalized children appear substantially lower than this largely because of fewer drug exposures and communication barriers. They may also be the result of discrepancies in the event categorization and inconsistent reporting. Standardized definitions describing adverse drug reactions of a non-allergic nature (side effect) have not been routinely utilized in the medical record. Increasingly, descriptions of adverse reactions extend beyond standard drugs and include foods as well as personal or religious preferences. Additionally, patients and families often misinterpret allergies as side effects, preventing rechallenge with the medication. From a quality and safety perspective, by using specific definitions and vigilantly verifying the event details, the patient’s care will be improved.

Methods: This study was deemed exempt from IRB review. For hospitalized patients who have a positive history of any adverse drug reaction, a team consisting of a clinical pharmacologist and a pharmacist interviewed the family to document a detailed reaction history. Each reaction was assigned to one of the following adverse reactions: allergy/hypersensitivity, side effect, precaution, preference or unknown.

Results: During a 4 month period, 274 historical reactions were evaluated in 141 pediatric patients by the inpatient Drug Safety Service. Seventy-seven percent were initially categorized as “allergies”. Of those, 76/211 (36%) could be reclassified into more accurate adverse reaction categories. The offending medication could be tolerated again in 25 of the 59 evaluated side effects.

Table 1

<table>
<thead>
<tr>
<th>Interventions</th>
<th>n = 274 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADR’s initially categorized as “allergies”</td>
<td>211/274 (77%)</td>
</tr>
<tr>
<td>% changed to side effects</td>
<td>59/211 (28%)</td>
</tr>
<tr>
<td>% of patients that could tolerate the medication</td>
<td>25/59 (42%)</td>
</tr>
<tr>
<td>% changed to precautions</td>
<td>15/211 (7%)</td>
</tr>
<tr>
<td>% changed to preference</td>
<td>2/211 (1%)</td>
</tr>
<tr>
<td>ADR’s not been previously documented</td>
<td>15/274 (5%)</td>
</tr>
</tbody>
</table>
(42%) given specific considerations (premedications, slowed infusion times, etc.). Most importantly, 15 previously unrecorded ADR’s were detected during patient interview. In addition, we have documented a 350% increase in detected ADR’s occurring on an inpatient basis.

**Conclusions:** Parents may inaccurately present a patient’s adverse reaction profile, and the nature of the ADR may not be verified by the health care provider. This perpetuates the problem and precludes the patient from receiving an appropriate medication for the patient and the disease. Utilization of a program that accurately characterizes the reaction and medication with the reaction may allow patient’s to receive appropriate medications in their future.

### 286. PARTNERSHIP WITH PUBLIC HEALTH AGENCIES CREATES OPPORTUNITY TO EDUCATE POPULATIONS UNDERUTILIZING POISON CENTER SERVICES

**Lizette Villarreal,** Virginia Barrera-Garcia, Carmen Jimenez

**University of Texas Health Science Center San Antonio, San Antonio TX USA**

**Background:** Integrating public health into poison center (PC) activities has become a focus for PCs across the country. By participating in Operation Lone Star (OLS), our PC provided poison prevention education to parents, caregivers, and others in counties that are majority Hispanic, Spanish-speaking, and medically-underserved areas of South Texas.

**Methods:** Our PC partnered with public health agencies across the state to increase awareness related to the importance of utilizing PC services among OLS attendees. This two-week event is a joint project between various agencies including state health and human services agencies, TX State Guard, Army and Air National Guard, county health departments, local service groups and civilian volunteers that provide medical services such as free medical and dental exams, vaccines, and immunizations to people in the community and the operation serves as training for emergency planning. This intensive event covers six counties and is the largest humanitarian effort of its kind in the US. Due to wait times, our PC took the opportunity to educate those attending the event.

**Results:** A major disparity exists in the language of callers utilizing PCs versus the actual percent of the population in our state that speaks Spanish. PC call data for our state indicates that only 1% of calls are from the Spanish-speaking community; although, 29% of the population 5 years old and over speak Spanish in their home. The population reached by OLS was a majority Spanish speaking. Over a two-week period, community educators targeted six counties and reached 12,179 people. Table 1 provides census data on the composition of each county and a breakdown of the number reached. Spanish language calls originating in our region have increased by 220% from 444 in 2001 to 1,421 in 2010. The increase in 2010 may be attributed to educational projects such as Operation Lone Star that provide education aimed at the Spanish speaking population.

**Conclusions:** Since regional PCs encompass a large group of counties across a state, it is important that partnerships with public health entities be established to reach people that are currently underutilizing PC services. This project highlights the effectiveness of an initiative that reached a specific population over a relatively short period of time and attributed to positive outcomes.

<table>
<thead>
<tr>
<th>County</th>
<th>Number of Attendees</th>
<th>% Hispanic or Latino (census data)</th>
<th>% Spanish Language Spoken at Home (census data)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cameron</td>
<td>4,203</td>
<td>86%</td>
<td>72%</td>
</tr>
<tr>
<td>Willacy</td>
<td>1,273</td>
<td>87%</td>
<td>46%</td>
</tr>
<tr>
<td>Hidalgo</td>
<td>2,890</td>
<td>89%</td>
<td>82%</td>
</tr>
<tr>
<td>Webb</td>
<td>2,150</td>
<td>95%</td>
<td>91%</td>
</tr>
<tr>
<td>Starr</td>
<td>696</td>
<td>99%</td>
<td>96%</td>
</tr>
<tr>
<td>Zapata</td>
<td>967</td>
<td>88%</td>
<td>84%</td>
</tr>
</tbody>
</table>

### 287. WIKIPEDIA INFORMATION FOR TOXICOLOGIC EMERGENCIES INVOLVING HOUSEHOLD PRODUCTS, PLANTS AND ENVENOMATIONS: HOW RELIABLE IS IT?

**Kimberly Barker Ayes**, Christina Hantsch Bardsley, John M Jantz, William A Frederick

1. **Lipscomb University College of Pharmacy, Nashville TN USA;**
2. **Loyola University Medical Center, Maywood IL USA**

**Background:** Online resources such as wikis remain popular sources of information. Wikipedia is an unrestricted general information wiki that is often among top Results from web search engines. Lay public and healthcare professionals may utilize this resource for toxicologic information. Previous study on the reliability of Wikipedia for toxicologic information regarding medications found a lack of information on many common medications and two potentially serious instances of misinformation. Medications make up 58% of the top 25 exposure categories reported to National Poison Data System (NPDS) and non-medications the other 42%.

**Objective:** To review toxicology information on Wikipedia for non-medication exposures.

**Methods:** NPDS data was accessed to determine common non-medication exposures. Sixteen specific substances including envenomations, household products, and plants were selected for review. Toxicology information was divided into 4 categories: Mechanism of Toxicity, Toxic Dose, Symptoms of Toxicity, and Treatment. The grading scale developed for the previous study on Wikipedia and toxicologic information for medication exposures was utilized. Content was compared between Wikipedia and Poisindex. Poisindex was selected as a common professionally updated reference utilized by Poison Specialists. The purpose was to compare wiki content with a traditional database, not determine appropriateness of Poisindex. Two toxicologists and two pharmacy students independently compared content using the grading scale. Each substance was reviewed by all authors to reach agreement. Only information in the overview section of Poisindex that was directly related to the substance was included for comparison.

**Results:** Wikipedia content varied greatly on depth of toxicity information. Wikipedia pages for only 9 of 16 substances had information in all categories. There were 6 instances of incorrect information found in Wikipedia. At least 4 of these instances could lead to potential patient harm if the recommendation was followed. The categories most likely to contain information were mechanism of toxicity and toxic dose. The category most likely to lack information was treatment.

**Conclusion:** Wikipedia has limited toxicology information on non-medications commonly involved in exposures. Healthcare professionals and the lay public may have little baseline knowledge of non-medications involved in exposures and be less likely to recognize misinformation. Wikipedia is not a reliable resource, especially for treatment information, for toxicologic emergencies involving common envenomations, household products, and plants.

### 288. CONSUMER RESEARCH ON POISONINGS AND THE POISON CONTROL SERVICE: KEY THEMES

**Iana M. Simeonov,** Kristina M. Hamm, Stuart E. Heard

**UCSF-California Poison Control System, San Francisco CA USA**

**Objective:** Understand parents’ level of concern and responses to poison exposure, assess parents’ use of online sources for healthcare information, evaluate existing marketing materials, and gather insights for any adjustments to current practices across key demographic segments within the larger underserved or at-risk population (defined as at or below Federal Poverty Guidelines).

**Methods:** Field and collect findings from 2 focus groups in each market segment (low-income African-American, Monolingual Latino, and Bilingual Latino, and a mixed group of participants of average income for the state). Consumer research was managed through professional marketing agencies that recruited participants and held 14 focus groups. Two-hundred surveys were also conducted.

**Results:** Parents felt dangers in their homes were well managed and poisonous products stored safely. Of all respondents, 80% had heard of poison control, some commenting “It saved a trip to the ER.” The other 20% had a very poor good understanding of services, commenting that it was not for emergencies or medical information, including some types poisonings.
Mothers reported using search (“just Googling”) when they or a family member are ill. As a resource, respondents overwhelmingly preferred the center’s Facebook page to its website, considering it more useful and user-friendly. Facebook was the most used social network across all demographic groups and a majority of respondents from all groups had active profiles on the site.

Poison center education materials, including the center’s current offering, the most recent AAPCC brochure, and a new prototype, were reviewed and discussed. Participants preferred the center’s current brochure, but indicated a desire for further visual examples (“I need to see it”), less text (“so people who don’t read or speak English can understand”) and more robust colors (“make it stand out”). Respondents also indicated they could distinguish between and preferred genuine images over stock photos (“you can buy pictures of doctors on the internet”).

Conclusions: Education materials were revised to reflect consumer insights and include references to our active presence on popular social networking sites and use of new media.

289. A DESCRIPTIVE CROSS SECTIONAL SURVEY OF MEDICAL TOXICOLOGY CLERKSHIPS AT U.S. ALLOPATHIC AND OSTEOPATHIC EMERGENCY MEDICINE RESIDENCY PROGRAMS

Nahar D. Alruwaili1, Roman A. Hill2, Brent W. Morgan1, Philip Shayne1, Ziad N. Karrzi1
1Emory University/Georgia Poison Center; 2Synergy Medical Education Alliance/Michigan State University, Saginaw MI USA; 3Emory University, Atlanta GA USA

Background: Medical Toxicology (MT) is a core content area of the emergency medicine (EM) curriculum and a recognized subspecialty by the American Board of Medical Specialties and the American Osteopathic Board of Emergency Medicine.

Due to the lack of a uniform presence of MT faculty in EM residency programs, we aimed to describe the current methods and delivery of MT education at US EM residency programs.

Methods: A survey was sent to the program director of every Allopathic and Osteopathic EM program identified.

Results: The survey was entered in a secure electronic database. This study was exempted from IRB review at our institution

Results: The survey was completed by 57% (113/197) of the programs that we identified. Eighty four programs were allopathic while 25 and 4 were osteopathic and dual respectively. The length of the program was 3, 4 and 2 years in 62, 37 and 1% of respondents. A MT rotation was mandatory in 66%, a selective in 12% and not offered in 21% of responding programs. When offered, the duration of the MT rotation was 1 month or 4 weeks in 85% and 3 weeks or less in 15% of programs.

In 40% of respondents, the rotation was offered onsite and 60% offered it off site. When offered as a selective, 71% of respondents reported that less than 50% of their residents took advantage of that offer. Additionally, 88% of programs that did not offer a rotation, allowed their residents to pursue it as an away rotation.

When examining the structure of the MT rotation, 92% of programs had a poison center component while 71% had a bedside consult service and 57% had home call duties.

Medical toxicology lectures were given by program-affiliated toxicologists in 63%, visiting medical toxicologists in 36%, EM physicians in 59%, online lectures in 11.5% and other methods in 5% of respondents. Ninety four percent of respondents were amenable to using online education.

Thirty seven percent of respondents had no affiliated toxicologists while 39, 20 and 3% had 1-2, 3-5 and greater than 5 respectively. Twenty three percent of programs that did not have a toxicologist responded that they had plans to hire one in the future.

Conclusions: MT training is heterogeneous in US EM programs. Selective and off site rotations provide the training when not available at the primary institution. Alternative methods such as online education needs to be further evaluated.

290. INTERNET USE BY CALLERS TO POISON CENTERS

Kathleen M. Roche, Iris Barko, Bernard Sangalli, Marc Bayer
UCONN Health Center, Farmington CT USA

Background: Poison Specialists identified trends in consumers’ comments indicating that internet searches were often performed prior to calling the poison center.

We theorized that apparent decreases in call volume to our center during the past two years related to the use of web-based resources. We speculated, a priori, that consumers satisfied by information on the internet may not call a poison center, while those unsatisfied or wishing to verify accuracy, may call. We surveyed callers to determine the extent of internet utilization prior to calling the poison center.

Method: A survey of lay callers was conducted over a ten week period. Staff determined which callers to question based on call volume and time constraints.

Callers were queried post-exposure management. The survey included the following questions: 1) did you use the internet before calling the poison center? 2) did the information found on the internet differ from the information given by the poison center? 3) which internet site did you use? 4) how did you obtain the poison center phone number?

Results: Of those surveyed, 36% utilized the internet prior to calling. 48% of these callers searched the internet for information related to their exposure and another 48% only used the internet to obtain the poison center number. Callers provided specific reasons for calling the poison center after searching the internet: 25% did not find the information needed; 21% found the information “concerning” and/or they sought to verify its accuracy.

20% of callers that responded to question 2 stated that the information found on the internet differed from the information that the poison center staff provided. Others relayed that the poison center information was the same and some felt the poison center provided more detailed information.

Although most callers “just Googled it” (the exposure substance), the second most common response to question 3 was “manufacturer’s website.” Many callers did not remember the site used and only 3% of internet users searched our poison center website.

In addition to the callers who used the internet to obtain the poison center number, 33% of callers who did not use the internet were referred to the poison center by their pediatrician or other health care provider or service. Only 3% of non-internet users searched a telephone book to obtain the poison center number.

Conclusion: Apparent decreases in poison center call volumes may reflect consumer use of the internet as a first line resource. Callers consult the web, whether to obtain a poison center phone number or research substance information. Poison centers may need to consider alternative avenues to promote their services as the ultimate resource, to compete, and increase public access.

291. POISONINGS ASSOCIATED WITH MEDICATION ORGANIZERS

Rita Mrvos, Edward P. Krenzelok
University of Pittsburgh Medical Center, Pittsburgh PA USA

Background: Pill organizer container usage is becoming more prevalent in homes. The majority of these organizers are not child resistant and targeted for use in the elderly population to encourage compliance with medication regimes.

To determine the prevalence of exposures involving these types of containers all exposure calls over a 4 month period involving pill organizers reported to a RPIC were reviewed.

Methods: All poisoning exposure calls received at a RPIC were documented in the usual manner. If during the interview the caller mentioned that the medication was in a pill organizer when the exposure occurred, the information was documented on the chart. No further questioning about the container was done. The case was then tagged for review. Using Crystal Reports the medical record database was queried for these tagged cases and data collection included age, gender, exposure site, patient flow, hospital flow, outcome and substance(s). The data was analyzed using descriptive statistics.

Results: 75 exposures involving pill organizers were identified during the 4 month study period. 24 (32%) were adults and 51 (68%) were children less than 6 years of age. Multiple medications were involved in 47 (62.6%) of the exposures including 1 case with 10 substances, 12 patients were exposed to 3 different medications and 19 to 2 different medis. 28 of the adults (33%) were admitted to a critical care facility. 2 were admitted to a critical care unit, 2 were admitted to a non critical care unit, 3 were treated and released and 1 case was lost to f/u. Of the 51 pediatric exposures, 19 (37%) required evaluation in a health care facility. Of these, 13 (68%) were treated and released from the emergency department, 1 (5%) was admitted to critical care, 3 (16%) were admitted to a non critical care unit and 2 (11%) cases were lost to follow-up.
Conclusion: Although improved medication compliance in an aging population is highly desirable, many factors play a role in medication safety including pill identifiers. Education as to the proper way to take, administer and store medications remains a vital part of ongoing education.

292. INCONSISTENCIES IN NON-PHARMACEUTICAL COMMERCIAL PRODUCT LABELS

Lee Cantrell1, Sean Nordt2, Jamey Krauss3
1California Poison Control System- San Diego Division, CA; 2USC Keck School of Medicine, CA; 3Univ. of Calif. San Francisco School of Pharmacy, CA

Background: Human exposures to non-pharmaceutical commercial products occur frequently. Many product labels contain instructions for managing unintentional exposures, however the information provided varies considerably. US government regulatory agencies do not specifically mandate the type or location of information that must be contained in case of accidental exposure. The AAPCC has published guidelines for industries to use the national toll-free poison center (PC) telephone number, however there is no published data to determine if companies are adhering to these guidelines. While other authors have described the inadequate quality of product first aid instructions, the purpose of this study was to characterize the type and physical location of first aid instructions and consumer referral information.

Methods: A random, convenience sample of commercial product label information was obtained from a variety of local retail stores over a 6 month period. Twelve common non-pharmaceutical product categories with large numbers of annual human exposures were identified from NPDS data. A minimum of 10 unique products for each category were required. The following information was collected on a standardized Excel spreadsheet: product name and manufacturer, location on container, presence and type of route-specific treatment, medical assistance referral information.

Results: A total of 259 product labels were examined. First aid/contact information was located on container: rear-162 (63%), side-28 (11%), behind label-14 (5%), front-3 (1%), bottom-2 (0.7%), missing entirely-50 (19%). Specific first aid instructions were identified for the following routes of exposure: dermal (38%), ocular (70%), ingestion (52%), and inhalation (12%) exposures. Fifty-five products (21%) lacked any first aid instructions. Suggested contacts for accidental poisoning: physician 144 (56%), PC 102 (39%), manufacturer 44 (17%), 911 10 (4%). Only 2 (0.8%) products displayed the toll-free poison center telephone number, both of which complied with AAPCC guidelines.

Conclusion: Inconsistencies among the location and types of first aid information on commercial product labels were abundant. This may lead to confusion among consumers and delays in treatment. Clinical toxicologists should utilize their expertise to produce standardized commercial product label language and locations for poison-related information. Federal regulatory agencies should then be approached regarding the adoption of these recommendations.

293. THE EMERGENCY MEDICALIZATION OF MEDICAL TOXICOLOGY

Paul M. Wax, Lewis S. Nelson
American College of Medical Toxicology, Phoenix AZ USA

Background: From 1975-1992 the American Board of Medical Toxicology (ABMT) conferred board certification in Medical Toxicology (MT). In 1993 the American Board of Medical Specialties recognized MT as a distinct medical specialty. A subboard to oversee exam development was convened with representatives from American Boards of Emergency Medicine, Pediatrics, and Preventive Medicine. These sponsoring boards reflected the heterogeneity of those with a MT interest and the broad spectrum of toxicological issues that impact human health. Many of the early pioneers in MT were pediatricians responding to the challenges of caring for poisoned children. Also, occupational and public health physicians had become interested in workplace and environmental exposures. The development of emergency medicine (EM), starting in the 1970s, parallels the development of MT as a distinct medical specialty. We investigated whether there have been changes in the primary medical specialty of those board certified in MT over the past 35 years.

Methods: The membership directory of all ABMT diplomates from 1975-1992 and the ABMS directory of Subboard certified MT was compared to determine the primary specialty of their diplomates. Given that ACGME recognition of MT fellowship programs began in 2002, a subanalysis was performed comparing those who attained board certification after ACGME fellowship training became compulsory.

Results: 212 physicians received board certification by the ABMT between 1975-1992. 400 physicians received board certification by the Subboard between 1994-2010. 200 of these were fellowship trained between 2002 and 2010. Many of those board certified in MT have multiple primary board certificates (eg. Internal Med and EM). The primary board(s) of the MT diplomates are as follows:

Conclusions: Over the last 35 years there has been a shift in the primary boards of medical toxicologists towards EM. Prior to 1994 38% of medical toxicologists had primary boards only in EM, and since the fellowship requirement began in 2002 this has increased to 85%. Medical Toxicology has become a specialty predominantly of EM physicians. Pediatricians account for fewer than one in ten recent MT diplomates, and the number of occupational medicine and internal medicine physicians who have become board certified in MT since ACGME fellowship training became compulsory is negligible. The implication of this shift require thoughtful deliberation.

294. COMMUNITY PARTNERS AND NATIONAL POISON PREVENTION WEEK: LESSONS LEARNED

Molly Broderick1, Teresa Dodd-Butera2
1California Poison Control System, San Diego CA USA; 2CSU San Bernardino, San Bernardino CA USA

Background: Unintentional poisoning is one of the leading causes of unintentional injury death in the United States. Establishing community partnerships for National Poison Prevention Week offers the opportunity to educate community agencies and the public on prevention strategies, while increasing awareness of Poison Control Centers (PCCs); and presenting community resources for poisoning emergencies. The purpose of this qualitative report is to describe the barriers and opportunities encountered in establishing community partners for disseminating materials and educating the public on poison prevention.

Methods: Selected community organizations were notified prior to National Poison Prevention Week (2011) about the opportunity to collaborate and celebrate the week of educational activities. Participating organizations involved a youth center, public health departments, health clinics, and community resource centers. Dissemination of educational pamphlets, telephone stickers, and information sessions were arranged for National Poison Prevention Week.

Results: Both obstacles and opportunities presented themselves in this experience. At the youth center, educational materials were required to be placed in a designated community section which was in an open area, but not well-recognized; and was crowded with other literature. There was no information table or opportunity for a question and answer session with the CSPI. The public health department actually encouraged dissemination of materials and patient interaction, but the numbers of encounters were low. The community resource center was available to many participants who took advantage of obtaining information pamphlets, but participants were

Table 1.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Emerg Med</td>
<td>118 (56%)</td>
<td>151 (76%)</td>
<td>102 (86%)</td>
<td>71 (87%)</td>
</tr>
<tr>
<td>Pediatrics</td>
<td>51 (24%)</td>
<td>26 (13%)</td>
<td>6 (5%)</td>
<td>9 (11%)</td>
</tr>
<tr>
<td>Occup Med</td>
<td>20 (9%)</td>
<td>17 (9%)</td>
<td>9 (8%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Internal Med</td>
<td>56 (26%)</td>
<td>34 (17%)</td>
<td>8 (7%)</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>Emerg Med only</td>
<td>81 (38%)</td>
<td>117 (59%)</td>
<td>94 (80%)</td>
<td>70 (85%)</td>
</tr>
</tbody>
</table>

Copyright © Informa Healthcare USA, Inc. 2011
there to receive food stamps, and were somewhat disinterested in discussing poison prevention. Partnering with the professionals was informative, and allowed for assessment of the agency awareness of PCC services. It also allowed for outreach to revisit future partnerships throughout the year.

**Conclusions:** Opportunities were varied for raising awareness and disseminating poison prevention materials. The PCC is an essential source for community information on treatment and prevention of poisonings. National Poison Prevention week offers the opportunity for outreach and education. The lessons learned from the experience include the potential for increasing awareness of PCCs outreach for prevention and services in the community. Barriers presented include the need for community and client assessment. Partnering with the professionals began the assessment of agency potential, and established continuing partnerships for further education and collaboration.

### 295. EFFECTS OF AN OUTDOOR ADVERTISING CAMPAIGN IN A RURAL COMMUNITY ON PUBLIC AWARENESS OF A POISON CENTER

**Evelyn Waring, S. Rutherford Rosé**  
Virginia Poison Center, VCU Medical Center, Richmond VA USA

**Background:** Poison center (PC) education program delivery to low population density rural areas presents unique challenges, and these areas often have lower PC call penetrance values than suburban and urban areas. The purpose of this study was to evaluate the impact of an outdoor advertising campaign on public awareness of the PC in a rural area.

**Methods:** A telephone survey of 300 randomly selected households in four rural counties was conducted prior to an outdoor advertising campaign. Billboard ads displaying the 1-800-222-1222 number were then placed in those counties for an eight week period. Two months later, the survey was repeated in the targeted counties. The survey assessed awareness of the PC, identification of key PC characteristics, description of the best and next best place to call if a poisoning occurs and access to the PC emergency number.

**Results:** The number of respondents knowing a “lot” or “a little” about the PC was high both prior to (55%) and after (57%) the campaign. After the campaign, 13% reported having seen billboards about poison control in the past few months, up from 6% prior to the campaign.

The survey documented improved knowledge of three key PC characteristics: 24 hour availability, toll free access, and medical expertise. The number of respondents who correctly identified that help is given by healthcare professionals, not by trained citizens, remained about the same but the number of incorrect responses to this question increased after the campaign. The mean number of correct responses across these four items rose from 2.04 to 2.33, with statistical significance at .05.

There was a 10% increase in the number of respondents who identified calling the PC as either the best, or next best, action to take after a poison ingestion. The ad campaign had no apparent effect on the number of people who reported having the PC number posted at home or entered into their phones, or on the number of people who reported having ever called a PC.

PC calls were analyzed for changes. Although total annual call volume to the PC decreased by 4% in the year that the project was implemented, calls from the targeted counties had decreased by 14.6% in the four months prior to the campaign. In the four months following it, calls from those counties had returned to within 1% of the previous year volume.

**Conclusions:** A brief outdoor advertising campaign had a modest but measurable impact on awareness of PC characteristics, and of the PC as an appropriate place to call in the event of an exposure.

### 296. RACIAL AND ETHNIC DIFFERENCES IN POISONING EXPOSURES REPORTED TO A REGIONAL POISON CENTER

**S. Alrashdi, R. Riyaz, S. Hon, RJ. Geller, L. Herrington, ZN. kazzi**  
Georgia Poison Center and Emory University, atlanta GA USA

**Background:** The effect of race and ethnicity on health is well documented in the medical literature. It has been shown that racial disparities exist in different aspects of injuries, care and outcomes. Poisoning is a form of intentional or unintentional injury.

**Objectives:** Describe various aspects of poisoning by race and ethnicity, including age of patient, substances involved and reason for exposure.

**Methods:** A cross-sectional descriptive study of poisoning exposures reported to a regional poison center (RPC) over 6 months. Specialists in Poison Information System were asked to collect the race and ethnicity of the patient per the caller, when possible. Age, race, ethnicity, substance(s) involved, and reasons for the exposure were extracted for analysis from the cases meeting the inclusion criteria.

**Results:** A total of 3621 exposures were analyzed. Race distribution represented 70% White, 21% Black, 5% Multiracial, 2% Asian, 2% Pacific Islanders; 1% refused to answer. Ethnicity was 90% non-Hispanic. The distribution of race and ethnicity of the study group was reflective of the racial and ethnic composition of our service area. Among exposures, the two most common substance categories were cosmetics and analgesics in all groups except Pacific Islander, where foreign body was the second most common substance category. Unintentional exposure was the most common reason and children 0-5 years age the most common age group. This was the same amongst all races and ethnicities and consistent with national data. Of the top 25 exposures, 70% of patients were White; about 25% Black. Suspected suicide accounted for 70% of intentional exposures amongst Blacks and 55% in Whites. Blacks represented 15% of all bites and stings compared to 82% Whites. Of mushroom exposures, Whites accounted for 80% of the exposures followed by Pacific Islanders at 20%.

**Conclusions:** There was little difference in the racial and ethnic composition of our two most common substance categories, reason for exposure, and age groups. It is feasible and valuable to collect racial and ethnic data to further identify, address, and prevent potential disparities in poison injuries.

### 297. TOX TOOLBOX: AN APP FOR IPHONE, IPAD, AND ANDROID

**Rais Vohra², John H. Trestrail IIII**  
¹Center for the Study of Criminal Poisoning, Los Lunas NM USA;  
²UCSF-Fresno Medical Center, Fresno CA USA

**Background:** Tox Toolbox is a clinical toxicology software application (“app”) for iPhone, iPad, and Android smartphones. This program is designed to be used by healthcare workers and as a board-review resource for commonly tested toxicology topics. We authored the fixed clinical contents, and in January 2011, the 1.0 version became available for online purchase and download. Four months later, a companion version of the software was released for use on Android smartphones. Featured app contents include: PCC call button; toxic syndrome assessment; decontamination techniques and indications; essential management for 25 common overdoses; direct links to PCC homepages, pill identification sites, and other websites for envenomations, plant/mushroom identification, and veterinary poisonings; and a “Tox History Calendar,” featuring notable or infamous moments from toxicology history.

Detailed descriptions and screenshots of the app can be viewed here: http://itunes. apple.com/us/app/tox-toolbox/id412067576?mt=8

**Methods:** Vendor data accessed from the iTunes App Store, a division of Apple Inc (Cupertino, CA). We collated weekly information statistics about the number of app downloads, and their country of origin.

**Results:** There were 251 downloads to 25 countries in the first 3 months after the app’s release. Countries of download included the following (in descending order of the number of downloads): USA, Great Britain, Australia, Canada, Switzerland, Mexico, South Africa, New Zealand, Germany, Hungary, Italy, Saudi Arabia, Sweden, and Taiwan; these were collectively responsible for 241 of the total downloads. Ten other countries had one download each.

**Conclusions:** Since their inception, poison control centers have successfully incorporated emerging communications platforms. In the current era, app software featuring instant, handheld access to the basics of clinical toxicology management provides new opportunities for toxicologists to connect with clinical workers as well as with the general public. Our experience confirms that app software is a viable modality for clinical toxicology education and outreach efforts. The distribution of these unsolicited app subscribers spanned 25 countries. These first users, the so-called “early adopters” in the infotech nomenclature, are crucial to the emergence and success of new media and technologies. Our data indicate that there is a broad geographic demand for toxicology-related apps. App software promises to generate many new tools for
298. VARIATIONS IN PHYSICIAN-PATIENT NSAID RISK COMMUNICATION BY CYCLO-OXYGENASE (COX) SELECTIVITY

Cornelis A. Van den Boeger¹, Michael J. Miller², Daniel J. Cobaugh¹, Lang Chen¹, Kenneth G. Saag¹
¹UAB CERTs, Birmingham AL USA; ²University of Oklahoma, Tulsa OK USA; ³ASHP Foundation, Bethesda MD USA

Objectives: To examine differences between COX-2 selective NSAID and partially/nonselective NSAID users in patient-reported nonsteroidal anti-inflammatory drug (NSAID) risk communication by physicians.

Methods: A telephone survey of NSAID users aged ≥50 years was used to collect demographic information, comorbidities, type of NSAID agent used and recall of physician-patient risk communication about gastrointestinal (GI) events, hypertension, heart attack and kidney disease. A summary index of total risks communicated (0–4) was also created for analysis. For this summary index, the total number of the 4 risks communicated was dichotomized at 1 with 0 or 1 risk communicated versus 2 or more risks communicated for analysis. NSAID users were divided into two categories based on previously reported Agency for Healthcare Research and Quality definitions: 1) users of only celecoxib (highly selective) and 2) users of either a partially selective (meloxicam, nabumetone or etodolac) or a nonselective NSAID (other available prescription NSAIDs). Bivariate analyses were used to evaluate differences in risk communication between the two NSAID user groups. Multivariable logistic regression models were subsequently used to evaluate differences in risk communication by NSAID user group while adjusting for previously identified covariates.

Results: Among the 83 selective NSAID users and 200 partially/non-selective users, physician-patient risk communication was lower for partially/nonselective NSAID users (range: 26%-38%) compared to selective NSAID users (range: 37%-52%). GI and hypertension risk communication was lower for partially-/nonselective NSAID users. Overall, NSAID risk communication should be improved for both selective and nonselective NSAIDs.

299. A CASE OF MISTAKEN IDENTITY: UNINTENTIONAL INGESTION OF A CAMPHOR CONTAINING SOLUTION

J. Michael Kowalski, Matthew S. Salzman
Albert Einstein Medical Center, Philadelphia PA USA

Unintentional poisoning can occur when a harmful product is mistaken for a benign or therapeutic medication. Misidentification may occur due to identical packaging, similarly spelled name or inappropriate storage. A 30-year-old man accidentally ingested “a mouthful” of Vicks brand VapoSteam® liquid. He was self-medicating at home with Nyquil Cold and Sinus® to treat flu-like symptoms. He awoke during the night and reached for what he believed was a bottle of Nyquil Cold and Sinus® on his nightstand. Mistakenly, the patient drank liquid from a bottle of VapoSteam® instead. He did not recognize the taste as that of the Nyquil® he was used to. When he turned on the lights to examine the substance, the man realized that he had mixed up the bottles. The patient did not use a measuring device so he was unsure of the exact amount that he consumed. The patient went to his local health care facility for evaluation. In the ED his vital signs were 92 bpm, 132/75 mm Hg, 37.2°C, and 16 respirations per minute. Physical exam revealed an alert, oriented, well-developed 30-year-old male in no distress. The remainder of the exam was within normal limits. After an observation period of six hours in the ED, the patient was discharged home without sequelae.

300. OVERALL IMPACT OF A POISON PREVENTION PROGRAM ON DOCTOR OF PHARMACY STUDENTS AND THEIR ATTITUDE TOWARD TOXICOLOGY AS A DISCIPLINE FOR PHARMACISTS

Yusuf Khan², Christina Hew², Peter M Brody², Ashley N Webb²
¹Upstate New York Poison Center, Syracuse NY USA; ²School of Pharmacy and Pharmaceutical Sciences, University at Buffalo, SUNY, Buffalo NY USA

Background: In 1996, the poison prevention program was developed and added to the didactic pharmacy curriculum at our University. First year (P1) students were utilized during National Poison Prevention Week to achieve a goal of community outreach and education in poison prevention. Funding from the State Attorney General’s Office has led to consistent program growth. Today, the program demonstrates the pharmacy student’s role in poison prevention via education of young children and distribution of poison center materials.

Methods: Educational outcomes of this mandatory course are to foster an interest in poison prevention and toxicology and to enhance communication skills. Through coordination with the poison center, P1 students are trained in poison prevention and education of high risk age groups for poisonings (4-6 years). P-1 Student groups provide pre-K and elementary school classes with presentations encompassing 5 routes for poisoning, locations of common poisons, and actions to take in a poisoning emergency. The presentations are unique but evaluated by faculty and poison center staff for appropriateness prior to implementation. Surveys completed by the P1 students provide insight to their perception of the program’s value.

Results: In 2010, P1 students were queried for their impressions of the program’s benefit. We achieved 45% participation and more than half of the respondents gave positive answers (agree or strongly agree): while only 63% felt prepared to educate young children, 78% gained an enhanced ability to communicate with them and 87% felt the children benefitted; 70% improved their ability to function in a group, 81% agreed future P1 students should participate, and 78% had a positive experience. We decided to further evaluate how pharmacy students’ attitudes toward the program and their realization of benefits change by the end their didactic careers. At the conclusion of the 2011 program, a survey was distributed to the current first, second, and third year students to evaluate their perceived benefit, their current interest in toxicology / poison prevention as well as toxicology as a career choice.

Conclusions: While the previous P1 students agreed that all stakeholders in the program benefit at various levels, we intend to show that over time, students at the end of their didactic careers have a different appreciation for poison prevention than during participation in the program. We also intend to show that initial exposure to toxicology in the first pharmacy year, fosters an interest in further education in toxicology through rotations, collaboration with poison centers, and consideration of fellowships.
301. ACUTE CARDIOMYOPATHY FOLLOWING A MILNACIPRAN OVERDOSE

M. Levine, C. A. Truitt, A D O’Connor
Department of Medical Toxicology. Banner Good Samaritan Medical Center, Phoenix AZ USA

Background: Milnacipran (MIL) is an SNRI, approved in the US for the treatment of fibromyalgia. Severe toxicity following acute overdose is rare. The following case describes acute cardiomyopathy and suspected serotonin syndrome following acute overdose.

Case Report: A 59-yr woman presents 30 min after ingesting 3g of MIL. Initially she was awake and normotensive. One h later, she was unresponsive with a BP 70/50, and a HR 60. She was intubated, and started on norepinephrine (NE), which was weaned off 5 h post ingestion. Six h post ingestion, the patient developed atrial fibrillation with a ventricular rate of 135 and BP 249/145. She received 20 mg of diltiazem over 5 min. The HR decreased to 100. Fifteen min. later, her BP decreased to 66/54, HR remained 100. She received IV calcium and 2L of saline without improvement in BP. NE was re-started, and titrated to 20 mcg/min to maintain a MAP of 60-65. An ECHO revealed an LVEF of 30-35% with moderate to severe diastolic dysfunction. Dobutamine was started at 10 mcg/kg/min.

Seven h post ingestion, she developed a fever of 38.9 C, and had tremor without rigidity or clonus. Drug screen (immunoassay) revealed the presence of THC and benzodiazepines. Comprehensive drug testing via GC/MS identified paroxetine and nicotine in the urine. A serum MIL concentration 5 h post ingestion, determined by LC tandem MS was 8400 ng/mL (therapeutic 150 ng/mL). The patient required NE for 38 h, and dobutamine for 66 h. Troponin peaked at 0.45 ng/mL. A repeat ECHO two days post ingestion revealed an LVEF of 60-65% with moderate regional wall motion abnormalities.

Cardiac catherization four days post ingestion revealed a normal coronary angiogram and an EF of >70%.

Discussion: History, pill counts and the area under the curve on GC/MS spectra support therapeutic paroxetine levels. Quantitative MIL levels confirmed acute MIL toxicity was responsible for this patient catecholamine-induced stress cardiomyopathy. Furthermore, the patient displayed altered mental status, autonomic instability, hyperthermia and tremor suggestive of possible serotonin syndrome.

Conclusion: Overdoses of MIL are rare. This case illustrates acute cardiomyopathy, and probable serotonin syndrome following an overdose of MIL in a patient taking paroxetine therapeutically.

302. DOUBLE TROUBLE: INFANT INTRAVENTRICULAR HEMORRHAGE AND DEATH AFTER MATERNAL BRODIFACOUM POISONING

Scott Schmeising1, Kris Nanagas1, Lynn Ballentine2, Mary Wermuth1
1Indiana University School of Medicine, Indianapolis IN USA; 2Indiana Poison Information Center, Indianapolis IN USA

Background: Brodifacoum (4-hydroxycoumarin) is a long-acting anticoagulant that inhibits Vitamin K1.2 epoxide reductase, an enzyme needed for the activation of Vitamin K in the production of clotting factors II, VII, IX, and X. It is marketed as a rodenticide under the trade name “d-Con.” In human ingestions the estimated half-life is 24 days and follows zero-order kinetics. Treatment includes long-term Vitamin K1 (phytodaidone). Fresh frozen plasma (FFP) or prothrombin complex concentrate is used for life-threatening bleeding.

Case Report: A 34 year-old Chinese female attempted suicide by ingesting 7 boxes of d-Con while 33 weeks pregnant. She was treated with oral phytodaidone (up to a maximum dose of 40 mg twice daily) and experienced no hemorrhage or complications. Her PT/INR peaked on day 3 post-ingestion at 87.1/7.14. She was released from the hospital on day 43 with a normal PT/INR, and continued to take phytodaidone.

On day 8 post-ingestion, the fetus was delivered emergently by c-section after experiencing decelerations and had AFGARs of 1 and 7 at delivery. The female baby began seizing and required intubation. She bled significantly from the cord and endotracheal tube. Coagulation studies showed a PT >225, PT >140, and INR >11.4 with a hematocrit of 25. Vitamin K IV and FFP were given to correct anticoagulation and control bleeding. A CT scan of the head showed intraventricular subarachnoid, subdural hemmorhages, and diffuse cerebral edema. Despite aggressive medical treatment, including ventricular drainage, the infant’s condition deteriorated, and life support was withdrawn resulting in death 3 days after delivery.

Case Discussion: Warfarin crosses the placenta freely but it is unclear how well brodifacoum crosses. Vitamin K1 has been shown in human studies to poorly cross the placenta, including one study in 1982 that found no detectable levels in the cord blood of term infants although adequate levels were present in mothers. Vitamin K1 during pregnancy is generally indicated for maternal hypoprothrombinemia and for prevention of hemorrhagic disease of the newborn induced by maternal drugs. There is only one documented case report of a pregnant poisoning with brodifacoum (at 22 weeks gestation) and there were no fetal complications.

Conclusions: This is the first case report of infant demise after maternal ingestion of brodifacoum. Brodifacoum in high doses significantly crosses the placenta, and phytodaidone orally in standard doses does not cross the placenta enough to reverse anticoagulation.

303. COMPARATIVE ELECTROCARDIOGRAPHIC EFFECTS OF ANTIpsychotic DRUGS AFTER OVERDOSE

Salem M. Fatullah1, Janine Gray1, Simon H. Thomas2
1Janine Gray Statistical Consultancy, East Bolden UK; 2Newcastle University, Newcastle UK

Background: Antipsychotics drugs are commonly involved in episodes of drug overdose. Some antipsychotics may delay cardiac repolarisation, leading to QT prolongation and an increased risk of torsade de pointes. Effects on the QRS interval, reflecting sodium channel function and conduction velocity, have also been reported. This study was performed to compare EKG effects of individual antipsychotic drugs after overdose.

Methods: Observational cohort study using EKGs collected at 25 mm/s from all adult patients with first presentation of antipsychotic drug overdose in Newcastle between 2000 and 2008. EKGs were analysed blind to patient characteristics and exposure using a digitizer. Relationships between antipsychotic drugs and QT and QRS intervals were examined by multivariate analysis.

Results: Of 443 first presentations with reported antipsychotic overdose, 364 had at least one EKG available. In 46 (12.6%) episodes QTc prolongation (>450 ms in males, >470 ms females) was demonstrated but there were no QTc intervals >500 ms. QT intervals were correlated with RR interval and age, inversely correlated with plasma potassium concentration and were longer in females and those taking thyroidiazone in overdose (Regression coefficient 16.25, 95% CI 7.7, 24.7 ms). No significantly different QT effects were detected for other individual antipsychotics. There was a weak positive relationship between QRS interval and age. QRS intervals were shorter with haloperidol (Regression coefficient 7.0, 95% CI -12.4 to -1.6, ms) and longer with quetiapine (Regression coefficient 6.0, 95% CI 2.2 to 9.8, ms).

Conclusions: Severe QTc prolongation is uncommon after antipsychotic overdose. Lengthening of the QT prolongation was associated with thyroidiazone overdose, which has also been linked with QT prolongation and torsade de points in normal therapeutic use. The apparent link between haloperidol and quetiapine overdose and QRS interval changes merits further research. Study shortcomings include limited statistical power, retrospective methodology with incomplete availability of data, lack of analytical confirmation and of doses taken and often absent of baseline EKGs.

304. MASSIVE AMANTADINE OVERDOSE RESULTING IN STATUS EPILEPTICUS AND DEATH

Shin K. Kwon, Heather Ellsworth, Christian P. Lintner, Samuel J. Stellpflug, Jon B. Cole
Hennepin Regional Poison Center, Minneapolis MN USA

Background: Amantadine has been used in the treatment of the influenza virus, Parkinson’s disease, and drug-induced extrapyramidal symptoms. Toxic doses have produced cardiac arrhythmias and neuropsychiatric effects in animal studies and have also been demonstrated in human case reports. We report a case of...
Case Report: An asymptomatic 33 year-old woman with a history of bipolar disorder and multiple sclerosis presented to the ED 1.5 hours after ingesting 6.2 g of amantadine in an attempt at self-harm. Four hours post-ingestion, she became acutely disoriented, developed ventricular tachycardia, seized, and had a cardiac arrest treated successfully with CPR, intubation, lidocaine and amiodarone. Her potassium was 2.5 mEq/L, sodium 135 mEq/L, glucose 88 mg/dL, bicarbonate 26 mEq/L and creatinine 0.58 mg/dL. Supplementary magnesium and potassium were administered. Her amantadine concentration 1.5 hours post-ingestion was 3,960 ng/mL, 10.5 hours post-ingestion was 20,508 ng/mL, and 21.8 hours post-ingestion was 15,508 ng/mL. She was extubated on hospital day (HD) 2, but remained confused and was hallucinating. Due to hypoxia and concern for airway compromise, she was re-intubated on HD 3. She developed acute respiratory distress syndrome and septic shock with positive blood cultures. Levofloxacin and continuous infusions of norepinephrine, propofol, midazolam and vecuronium were administered during hospitalization. Due to refractory seizure activity confirmed by electroencephalogram (EEG), pentobarbital was administered on HD 14. On HD 21 life support was withdrawn after EEG monitoring revealed 252 hours of persistent seizure activity.

Discussion: A therapeutic range of amantadine has not been established, however the expected steady state concentration in patients receiving therapeutic doses is thought to be 200-1,000 ng/mL. Time to peak serum concentrations is 3.3 hours, which approximated the onset of toxicity in this case. Reported effects in overdose are cardiac dysrhythmias (including arrest), hypertension, and CNS toxicity. Much of the acute toxicity is attributed to antimuscarinic effects and usually observed when serum concentrations exceed 2,000 ng/mL. This case highlights the rare occurrence of refractory status epilepticus following an acute overdose of amantadine.

Conclusion: Toxicologists and emergency providers should be aware that amantadine toxicity may result in lethal cardiac dysrhythmias and refractory seizure activity.

305. ACIDOSIS AND PROLONGED ATAXIA FOLLOWING MASSIVE IBUPROFEN OVERDOSE

Ronald I. Kirschner1, Karah M. Debroux1
1University of Nebraska Medical Center, Omaha NE USA; 2Nebraska Regional Poison Center, Omaha NE USA

Background: Coma, acidosis, and seizures are sometimes seen with large ibuprofen overdoses. We report a patient who had persistent ataxia until 5 days after ingestion, followed by full recovery.

Case Presentation: A healthy 17 year old female was found unresponsive by family, with seizure-like activity. Initial vital signs were normal except for a heart rate of 127. In the Emergency Department, she had tonic contracture of the right face and received lorazepam 2 mg. Pupils were equal and sluggishly reactive. She withdrew to pain. Heart, lungs, and abdomen were unremarkable. She was intubated for airway protection.

Initial labs were significant for arterial pH 7.12, pCO2 41, pO2 220, Na 138 mmol/L, K 3.6 mmol/L, Cl 104 mmol/L, CO2 16 mmol/L, AG 18, BUN 7 mg/dL, creatinine 0.6 mg/dL, glucose 198 mg/dL, Ca 9.2 mg/dL, ALT/AST 31/51 U/L, lactate 5.1 mmol/L, CK 91 U/L, ketones negative, urine: no crystals, ECG: ST 111, QRS 82, Qtc 413. Ethanol, acetaminophen, salicylate, methanol, and ethylene glycol were undetectable. Head CT, CSF cell counts and chemistries were normal.

After transfer to a children’s hospital, EEG showed diffuse slowing, consistent with encephalopathy. A bicarbonate infusion was begun for acidemia, and stopped 2 days later when the patient was extubated. Family found a bottle of ibuprofen 800 mg in her room with 70 pills (56 g) missing. Serum ibuprofen level was > 250 mcg/mL (therapeutic 10-50). The patient developed transient hyponatremia to 159, which resolved by hospital day 2.

At transfer from the ICU, she had uncoordinated movements of all extremities. By post-ingestion (PI) day 3, mental status had normalized, but she was unable to ambulate due to ataxia. She received physical therapy twice daily. On PI day 4 she could ambulate with assistance. On PI day 6, she was able to tandem walk without deficit. She recovered fully and was transferred to a psychiatric unit.

Discussion: Acidosis, seizure, and CNS depression have been reported with large ibuprofen overdoses. In this case, a previously unreported finding of gait ataxia was prominent, requiring physical therapy and prolonging hospital stay. Ataxia resolved by PI day 6, making anoxic injury an unlikely etiology. Comprehensive drug screening was not done, but according to the patient, her family, and pharmacy, she had no access to other medications. Although the mechanism is unclear, other etiologies of ataxia seem unlikely.

Conclusion: Ataxia may be a previously unrecognized complication of severe ibuprofen toxicity. Further study is warranted to elucidate the mechanism.

306. ACUTE ACEPROMAZINE OVERDOSE: CLINICAL EFFECTS AND TOXICOKINETIC EVALUATION

D. Adam Algren, Amber Ashworth

Background: Acepromazine, a phenothiazine derivative, is used as sedative in veterinary medicine. Cases of acepromazine overdose are infrequently described and toxicokinetic data has not been previously reported. We report a case of intentional acepromazine overdose resulting in central nervous system (CNS)/respiratory depression and hypotension with confirmatory toxicokinetic data. Case Report: A 54 year old woman reported intentionally ingesting 950mg of her dog’s acepromazine. She denied other co-ingestants. Her past medical history was significant for depression, anxiety, and hypothyroidism. She denied current medication use. She was evaluated in an emergency department within 1 hour of her ingestion and was alert with normal vitals signs. Physical examination was unremarkable. Activated charcoal was administered. CBC, electrolytes and LFT’s were normal. Serum acetaminophen, salicylate and alcohol were undetectable. Urine drug screen was negative for drugs of abuse. An EKG demonstrated normal intervals. Over the proceeding two hours the patient developed increasing somnolence, tachycardia and hypotension. The HR ranged from 100-130/min and the systolic BP was 70 mmHg. ABG demonstrated a mild respiratory acidosis. Bicap and a norepinephrine drip were instituted. Clinically she improved over the next 12 hours at which time the Bicap and norepinephrine were discontinued. Repeat laboratory studies and EKG’s remained normal and the patient was transferred to a psychiatric facility the following day. Serial plasma acepromazine levels were determined using GC/MS. The initial acepromazine level (1 hour post-ingestion) was 63 ng/ml. Follow up levels at 8, 10.5 and 13.5 hours post ingestion were 7.6 ng/mL, 8.9 ng/ml and 6.3 ng/ml, respectively, resulting in an elimination half-life of 3.1 hours.

Case Discussion: Acepromazine is structurally similar to chlorpromazine. It is used in dogs and horses for anxiety, treatment of motion sickness and as a perioperative sedative. Previous cases have documented findings consistent with phenothiazine intoxication including: CNS depression, anticholinergic toxidrome, sinus tachycardia, and hypotension. Cardiac conduction delays have not been reported. Our patient’s toxicity resolved within 12 hours and is consistent with other cases in which toxicity resolved over 6-18 hours. The toxicokinetic data demonstrate that acepromazine appears to have a short elimination half-life.

Conclusion: Acepromazine overdose is uncommon and clinical toxicity is similar to other phenothiazines, however toxicity often resolves within hours and the elimination half-life is shorter than most phenothiazines.

307. SUICIDE ATTEMPT BY INJECTION OF MERCURIC CHLORIDE

Ronald I. Kirschner1, Joseph T. Poterucha1, Debra A. Reilly1
1University of Nebraska Medical Center, Omaha NE USA; 2Nebraska Regional Poison Center, Omaha NE USA

Background: Self-harm attempts with mercuric chloride (HgCl2) are typically ingestions. Intravenous (IV) injection of elemental mercury has been described in case reports. To our knowledge, parenteral HgCl2 self-administration has only been reported once, with a fatal outcome. We report a case of HgCl2 injection resulting in significant local injury and moderate renal failure with full recovery.

Case Presentation: A 57 year old woman mixed an unknown number of HgCl2 tablets with 10 mL of water and injected the mixture into her left antecubital fossa (AF). She had been a nuclear medicine technician with...
experience performing antecubital injections. Unable to complete the process due to pain, she injected the remaining HgCl2 into the right AF, then drove to the Emergency Department. She denied ingestion of chemicals or pills. Vital signs were unremarkable. On exam she had gray plaques and surrounding erythema at both AF with normal pulses, motor, and sensory function of the distal upper extremities (UEs). She was neurologically intact. Labs were notable for BUN 39 mg/dL and creatinine 2.12 mg/dL, approximately 16 h after injection. Whole blood Hg was >800 ng/mL (normal <10), and 24 hour urine Hg was >800 mcg/L. Elbow films showed increased soft tissue density suggestive of foreign material.

The patient was treated with IV fluids and oral dimercaptosuccinic acid (DMSA) 10 mg/kg TID x 5 days, then BID x 14 days. Renal function normalized by day 4. She underwent debridement of necrotic tissue, including muscle and tendon, followed by local flaps and skin grafting of both UEs. Findings at surgery: Full-thickness injury including biceps muscle and tendon of both UEs with coagulative necrosis of multiple deep subcutaneous arteries, veins, fat, fascia, and muscle. Blood Hg after DMSA was 70 ng/mL (24 h urine Hg 100 mcg/L). A second course of DMSA was given. Neurologic and renal function remained normal. After discharge she was followed by her primary physician and plastic surgery. Graft sites have healed with preserved UE function, but prominent scarring. There have been no pulmonary issues.

Discussion: HgCl2 ingestion is typically associated with caustic mucosal injury and renal failure. Although the patient believed she injected most of the HgCl2 intravenously, severe soft tissue injury occurred. Despite a markedly elevated blood Hg, renal failure resolved in <96 h, and neurotoxicity was not observed. This may have been due to DMSA chelation and/or limited Hg++ distribution into the CNS.

Conclusion: Parenteral HgCl2 self-injection in this case resulted in significant local injury and renal toxicity that normalized within 4 days.

308. SEVERE SODIUM CHANNEL BLOCKADE AND CARDIOVASCULAR COLLAPSE DUE TO A MASSIVE LAMOTRIGINE OVERDOSE

Joshua N. Nogar1, Alicia B. Minns2, Binh T. Ly3
1Veteran’s Affairs Medical Center - San Diego, San Diego CA USA; 2UC San Diego Division of Medical Toxicology, Department of Emergency Medicine, San Diego CA USA; 3California Poison Control System - San Diego Division, San Diego CA USA

Background: Lamotrigine (LTG) is an antiepileptic drug that inhibits seizure activity by blocking sodium channels. CNS depression, seizures, and QRS prolongation are well reported, but these are typically self-limited events that respond to supportive care, benzodiazepines, or NaHCO3 administration, respectively. Cardiac arrest has been reported after co-ingestion with bupropion, but severe sodium channel blockade in the absence of other toxins has not been described. Case Report: A 48 year-old female who ingested 7.5 gm of LTG was brought to the ED after having a tonic-clonic seizure. Vital signs were HR 131, BP 107/68, T 99.4°F, RR 16. Physical exam was significant for lower extremity clonus, hyperreflexia in all extremities, GCS 8, moist mucous membranes, and normal pupils. She was intubated with rocuronium. Initial ECG demonstrated sinus rhythm at 97 bpm, QRS 106, TqTc 500. Her CBC, electrolytes, creatinine, VBG, ethanol, LFTs, salicylate, and acetylaminoephene were unremarkable. Comprehensive LC/MS/MS drug screen was negative for TCAs, cocaine, meperidine, propoxyphene, methadone, amphetamines, MDMA, and PCP. Her psychiatrist verified that LTG was the patient’s only known prescription medication. The patient was hemodynamically stable while sedated with lorazepam boluses. Three hours after intubation she developed status epilepticus, a wide-complex tachycardia at 143 bpm. QRS 148, and became pulseless. She underwent 45 minutes of resuscitation, during which she received: 500 mEq NaHCO3, 300 mg lidocaine, 300 mg amiodarone, 2 gm calcium gluconate, 300 ml 20% lipid emulsion, and two 200 J cardioversions. Pulses were measured by day 4. She was treated with IV fluids and oral dimercaptosuccinic acid (DMSA) 10 mg/kg TID x 5 days, then BID x 14 days. Renal function normal.

Discussion: LTG was brought to the ED after having a tonic-clonic seizure. Vital signs were unremarkable. On exam she had gray plaques and surrounding erythema at both AF with normal pulses, motor, and sensory function of the distal upper extremities (UEs). She was neurologically intact. Labs were notable for BUN 39 mg/dL and creatinine 2.12 mg/dL, approximately 16 h after injection. Whole blood Hg was >800 ng/mL (normal <10), and 24 hour urine Hg was >800 mcg/L. Elbow films showed increased soft tissue density suggestive of foreign material.

The patient was treated with IV fluids and oral dimercaptosuccinic acid (DMSA) 10 mg/kg TID x 5 days, then BID x 14 days. Renal function normalized by day 4. She underwent debridement of necrotic tissue, including muscle and tendon, followed by local flaps and skin grafting of both UEs. Findings at surgery: Full-thickness injury including biceps muscle and tendon of both UEs with coagulative necrosis of multiple deep subcutaneous arteries, veins, fat, fascia, and muscle. Blood Hg after DMSA was 70 ng/mL (24 h urine Hg 100 mcg/L). A second course of DMSA was given. Neurologic and renal function remained normal. After discharge she was followed by her primary physician and plastic surgery. Graft sites have healed with preserved UE function, but prominent scarring. There have been no pulmonary issues.

Discussion: HgCl2 ingestion is typically associated with caustic mucosal injury and renal failure. Although the patient believed she injected most of the HgCl2 intravenously, severe soft tissue injury occurred. Despite a markedly elevated blood Hg, renal failure resolved in <96 h, and neurotoxicity was not observed. This may have been due to DMSA chelation and/or limited Hg++ distribution into the CNS.

Conclusion: Parenteral HgCl2 self-injection in this case resulted in significant local injury and renal toxicity that normalized within 4 days.

309. CARDIOVASCULAR COLLAPSE AND SHOCK FOLLOWING IBUPROFEN INGESTION

Steven C. Curry, Michael Levine, Robert French, Aaron Skolnik
Department of Medical Toxicology, Banner Good Samaritan Medical Center, Phoenix AZ USA

Background: Ibuprofen (IBU) is a commonly used analgesic and anti-inflammatory agent. While acute overdoses are common, life-threatening toxicity is rare.

Case Report: A 30-year-old HIV positive male, on efavirenz, emtricitabine, and tenofvir called emergency medical services (EMS) 4 hours after an ingestion of 96g (1066 mg/kg) of IBU. Upon arrival of EMS, the patient was normotensive and awake. 2.5 hours after admission, the patient had a prompt decline in blood pressure, a rise in heart rate, and became comatose. He was intubated, received 4 L crystalloids, and vasopressors were commenced. A PA catheter showed initial CO > 20 L/min. SVR < 148 dyne*s/cm5, CVP 16 mm Hg, and PACWP 18 mm Hg. Calculated oxygen consumption was >350 mL O2/min. (CO was higher, but equipment would only report a CO of up to 20 L/min.) Over the next 2 days, he received infusions of numerous vasopressors, including epinephrine (50 mcg/min), norepinephrine (20 mcg/min), dopamine (40 mcg/kg/min), phenylephrine (100 mcg/min) and vasopressin (0.08 units/min) in failed attempts to maintain a MAP of 60 mm Hg. Methylene blue, 2 mg/kg IV, was given in an attempt to attenuate effects of nitric oxide, and had no effect on BP.

Initial laboratory studies had revealed no evidence of acidosis or impaired renal function (serum bicarbonate 23 mmol/L, creatinine 0.9 mg/dL). Comprehensive urine drug testing (with GC-MS) revealed only the presence of IBU and ethanol. Serum ethanol concentration was 159 mg/dL. An IBU level 8 hours post ingestion was 590 mg/L. On the 2nd day, the patient developed acidosis (lactate 10.9 mEq/L), and renal failure (creatinine 2.4 mg/dL).

Discussion: IBU ingestions of less than 400 mg/kg are typically relatively benign, with GI symptoms and occasional metabolic acidosis. Coma and convulsions follow large overdoses. High cardiac output distributive shock with a course similar to our patient has only been previously described once in the English literature following isolated IBU ingestion.

After recovery, our patient admitted to ingesting only ibuprofen, a finding supported by pill counts. Lactic acidosis can occur with nucleoside reverse transcriptase inhibitors. However, lactic acidosis occurring in this case was most likely due to β agonism from some vasopressors rather than the antiretrovirals.

Conclusion: Life-threatening IBU ingestions with cardiovascular collapse are rare. This patient developed coma, high output distributive shock, metabolic acidosis, and renal failure, but ultimately made a full recovery.

310. DELAYED TRANSAMINASE ELEVATION AFTER ACETAMINOPHEN OVERDOSE: A CASE REPORT

Jennett E. Madsen, E. M. Caravati, Barbara I. Crouch
Utah Poison Control Center, University of Utah, Salt Lake City UT USA

Background: Serum transaminases begin to rise 12-24 hours after acute acetaminophen overdose in patients who develop hepatotoxicity. Normal transaminases at the end of the 21 hour NAC infusion imply no liver injury from the overdose.

We report a case of acetaminophen overdose where serum transaminases did not start to rise until 34 hours after arrival to the emergency department (ED).
Case Report: A 16 year-old girl was brought to the ED with persistent vomiting after ingesting an unknown amount of acetaminophen. Time of ingestion was unknown. Upon arrival she was obtunded, acidic (serum CO2 11 mmol/L) with an initial serum acetaminophen concentration 661 mcg/mL, AST 12 U/L and ALT 27 U/L. Intravenous (IV) acetylecysteine (NAC) was started in the ED. Patient’s complete AST/ALT levels (U/L) are listed below. The AST and ALT were not >50 U/L until 34 hr and 44 hrs post admission, respectively. Serum acetaminophen concentration was less than 10 mcg/ml at 41 hours post admission and IV NAC was discontinued 2 hours later and the patient was considered medically cleared. An AST rechecked at 54 hours post admission was 423 U/L and ALT was 550 U/L. IV NAC was restarted. Transaminase levels peaked at 67 hours post presentation and returned to normal approximately 275 hours post admission.

Discussion: The elevation of transaminase levels approximately 34-44 hours post admission was delayed relative to usual rise seen with hepatic injury and led to premature discontinuation of IV NAC therapy.

Conclusion: Onset of rise of AST/ALT beyond 24 hours post ingestion may occur in large acetaminophen overdose.

Table 1.

<table>
<thead>
<tr>
<th>Hours from admission</th>
<th>0</th>
<th>18</th>
<th>24</th>
<th>34</th>
<th>44</th>
<th>54</th>
<th>67</th>
<th>75</th>
<th>83</th>
<th>95</th>
<th>107</th>
<th>275</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST</td>
<td>12</td>
<td>31</td>
<td>23</td>
<td>38</td>
<td>70</td>
<td>423</td>
<td>973</td>
<td>979</td>
<td>820</td>
<td>598</td>
<td>544</td>
<td>96</td>
</tr>
<tr>
<td>ALT</td>
<td>27</td>
<td>34</td>
<td>42</td>
<td>62</td>
<td>58</td>
<td>550</td>
<td>997</td>
<td>742</td>
<td>539</td>
<td>258</td>
<td>207</td>
<td>29</td>
</tr>
</tbody>
</table>

311. PROFOUND HYDROXYCHLOROQUINE TOXICITY WITH RESISTANCE TO SEDATION WITH HIGH DOSE DIAZEPAM

Melissa Halliday1, Kris Nanagas1, Zack Worley1, David Burns2

1Indiana University School of Medicine, Indianapolis IN USA; 2Indiana Poison Information Center, Indianapolis IN USA

Background: Hydroxychloroquine is a synthetic 4-aminquinoline derivative used as an antirheumatologic agent. Hydroxychloroquine ingestions are rarely seen, but result in rapid toxicity and high rates of mortality.

Case Report: A 32 year-old female attempted suicide by ingesting 18g of hydroxychloroquine. On initial presentation her blood pressure was 66/47 with a heart rate of 90, however quickly fell to 31/16 despite 2 liters of crystalloid replacement. She was intubated, placed on an epinephrine drip and given a heart rate of 154, blood pressure 165/67 mmHg, and T 98.0 oF. He received lorazepam 6 mg IV with minimal response and was subsequently intubated and sedated. Whole bowel irrigation (WBI) was instituted. Approximately 5 hours after arrival, he developed generalized tonic clonic seizures refractory to 30 mg lorazepam. He was started on propofol, midazolam and phenytoin infusions which were unsuccessful in terminating seizure activity.

A phenobarbital infusion halted muscular activity but EEG showed occasional epileptiform activity. On day 2, he completed WBI and phenobarbital was discontinued. He subsequently had increasing EEG epileptiform activity and valproic acid was added followed by levetiracetam, phenytoin, propofol and midazolam infusions. Anticonvulsant medications were continued for 6 days after which his family instituted comfort measures and he expired on day 7.

Conclusion: 4-AP is a new drug with a narrow therapeutic index resulting in refractory seizures. As the incidence of multiple sclerosis is increasing, physicians will encounter this complication. Treatment options are limited and research is needed to elucidate effective therapy for 4-AP intoxication.

313. INJECTION OF HOUSEHOLD BLEACH (SODIUM HYPOCHLORITE) INTO THE CENTRAL VENOUS CIRCULATION

J. M. Hildebrand, J. M. Burns, S. H. Rhyee, E. W. Boyer

Background: Ingestion of household bleach (sodium hypochlorite, NaOCl) is a common and often benign toxicological phenomenon. However, few reports describe large volume parenteral injection of bleach. We report the case of a patient who ingested approximately 100 milliliters of bleach into her central venous catheter in an apparent suicide attempt.

Case Report: An 18 year old female with a diagnosis of chronic Lyme disease was receiving long-term antibiotic therapy via a tunneled catheter. In a suicidal gesture the patient injected approximately 100mL of Clorox Lemon Fresh® Bleach (NaOCl 1-5%, NaOH 0.1-1%, pH 12.5) into her central venous circulation. On arrival she complained of thirst and body aches, vital signs were BP 103/32, HR 107, RR 20, O2 sat 100% on room air. For 12 hours after presentation, blood samples exhibited 4 + hemolysis, preventing the laboratory from reporting testing.

Results: She received fluid resuscitation for oliguria (less than 100mL in the first 24 hours). During her hospitalization she exhibited a significant hemolytic anemia (initial hematocrit 43.4%, low of 19.0% 6 days later) and required blood transfusion. She also experienced acute renal failure (peak creatinine 10.9 mg/dL) that required hemodialysis. In addition, the patient developed hypoxemia that resolved following dialysis. Renal biopsy showed changes consistent with acute tubular necrosis and necrotic epithelial cells likely representing hemo-lyzed erythrocytes. After three weeks her creatinine had stabilized in the 2.3 mg/dL range and dialysis was discontinued. She was transferred to the psychiatric service for further care. At a clinic visit 6 weeks post-exposure her creatinine had returned to a value of 0.97 mg/dL.

Case Discussion: Several case reports describe accidental injection of small quantities of NaOCl by IV drug users who commonly use bleach to clean syringes, but large injections are rare. Oxidative injury and glutathione depletion lead to deranged erythrocyte deformability, with resulting hemolysis. Destruction of circulating erythrocytes was demonstrated in this case leading to anemia and renal failure. Initial hypoxia resolved with dialysis, suggesting fluid overload instead of direct lung injury. With supportive care and dialysis the patient’s renal function returned to normal over a period of 6 weeks.

Conclusions: Sodium hypochlorite injection, especially in large quantities, is a rare but potentially serious toxic exposure. The mechanism of toxicity is likely similar to other oxidative processes. Due to hemolysis our patient experienced anemia and acute renal failure necessitating blood transfusion and hemodialysis.
314. LAMOTRIGINE POISONING WITH SEVERE NEUROTOXICITY AND CARDIAC DYSRHYTHMIAS, TREATED WITH HEMODIALYSIS

Saralyn R. Williams, Brad Watkins
Vanderbilt University, Nashville TN USA

Introduction: Lamotrigine (LTG) may exert severe neurotoxicity in patients with an acute overdose. Cardiac effects have rarely been reported. There is pharmacokinetic data to suggest hemodialysis may shorten the elimination half-life in patients with therapeutic levels.

Case Report: A 34 year old woman who was approximately 22 weeks pregnant presented after a history of an intentional ingestion of LTG, fluoxetine, and ethanol. She was obtunded and required intubation. Her ethanol level was 179 mg/dL. She developed generalized and persistent tonic seizures with twitching and tremors. She also manifested intermittent runs of a wide complex tachycardia (WCT). She received a total of diazepam 15 mg intravenously (IV), lorazepam 5 mg IV, phenobarbital 1000 mg IV, magnesium sulfate 2 grams IV, sodium bicarbonate 25 mEq IV, and propofol infusion. During transfer, she had periodic tonic seizure activity with tremors alternating with 30 seconds of no jerking movements. A total of midazolam 6 mg IV and additional phenobarbital 820 mg IV were administered. WCT occurred and was unresponsive to additional sodium bicarbonate IV. In the intensive care unit, she had periodic tonic seizures despite 4 mg of lorazepam IV and 1000 mg levetiracetam IV. Her blood pressure dropped into the 80’s systolic requiring norepinephrine to maintain her mean arterial pressure. She continued to have a WCT at 140 beats/minute. Given the volume of distribution and protein binding of LTG, hemodialysis was performed for 3.5 hours. Within 45 minutes of starting hemodialysis, the seizure activity ceased and the WCT resolved to a sinus tachycardia. An electroencephalogram after the cessation of the seizure activity demonstrated severe cerebral dysfunction with no ictal activity. By hospital day (HD) #2, she required sedation for agitation and was extubated by HD#3. She was discharged with an acetyl group attached to the cyclopentadiene ring. To date, there are no reports of acetylferrocene poisoning in humans. Case Details: A 30 year-old woman was transferred to a liver transplant center two days after presenting to another hospital for abdominal pain which had progressed to acute liver failure. Her labs: pH (ABG) 6.9, lactate 19.9, AST 8752, ALT 9271, INR 9.0, creatinine 1.7, glucose 23, and negative hepatitis/ANA/Anti-mitochondrial Ab panels. Aminocephalin level was 26 mcg/mL. An abdominal CT demonstrated diffuse areas of low attenuation in the liver, suspicious for an infiltrative process. The patient subsequently decompensated and was intubated, and N-acetylcysteine (IV NAC) and bicarbonate infusions were initiated. At the transplant center, she remained comatose, despite discontinuation of propofol, and her ammonia level reached 315 ug/dL. She was jaundiced, with anasarca and lower extremity clonus. As per toxicology recommendations, IV NAC was continued to treat a presumed late acetaminophen poisoning. However, while the liver enzymes decreased, total bilirubin trended upward, from 3.8 mg/dL initially. A head CT showed diffuse cerebral edema, which resolved after one week with mannitol and hypertonic saline. EEG was negative for epileptiform discharges, but revealed marked diffuse slow wave abnormalities in the frontal regions. Her course was complicated by MRSA pneumonia and a right-sided pleural effusion. On day #10, she began to exhibit purposeful movements and, in subsequent days, continued to progress and was eventually extubated. IV NAC was discontinued after 14 days. A repeat abdominal CT showed a cirrhotic liver. While in the inpatient rehabilitation unit, the patient finally confessed to intentionally ingesting 1-2 grams of acetaminophene powder, which she obtained from the laboratory where she worked as a graduate chemistry student. She stated that, within minutes of the ingestion, she had developed nausea with emesis and abdominal pain and had taken a non-toxic dose of acetaminophene. On day 34, the patient completed rehabilitation and was discharged home. At discharge, total bilirubin was 30 mg/dL and INR was 2.7.

Case Discussion: Acetylferrocene (CAS 1271-55-2) is an orange crystalline substance listed to be “highly toxic” and irritating to the skin, respiratory, and digestive tracts. Little is known about toxicity in humans.

Conclusion: We describe the clinical course of a human overdose with acetylferrocene which has not previously been reported.
<table>
<thead>
<tr>
<th>Author</th>
<th>Page Numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aaron CK</td>
<td>312</td>
</tr>
<tr>
<td>Abdelmalek J</td>
<td>48, 57, 88, 197</td>
</tr>
<tr>
<td>Ackermann B</td>
<td>165</td>
</tr>
<tr>
<td>Ackroyd Stolarz S</td>
<td>258</td>
</tr>
<tr>
<td>Acquisto N</td>
<td>113</td>
</tr>
<tr>
<td>Adams AB</td>
<td>1</td>
</tr>
<tr>
<td>Adams CM</td>
<td>116</td>
</tr>
<tr>
<td>Adoff S</td>
<td>279</td>
</tr>
<tr>
<td>Ahmed HA</td>
<td>236</td>
</tr>
<tr>
<td>Aks SE</td>
<td>212</td>
</tr>
<tr>
<td>Al Jumaan M</td>
<td>16, 126</td>
</tr>
<tr>
<td>Albert D</td>
<td>6, 46</td>
</tr>
<tr>
<td>Albertson TE</td>
<td>114, 232</td>
</tr>
<tr>
<td>Aldosary BM</td>
<td>131</td>
</tr>
<tr>
<td>Aleguas A</td>
<td>265</td>
</tr>
<tr>
<td>Algren DA</td>
<td>83, 306</td>
</tr>
<tr>
<td>Allison J</td>
<td>78</td>
</tr>
<tr>
<td>Aloisio N</td>
<td>233</td>
</tr>
<tr>
<td>Alpern E</td>
<td>234</td>
</tr>
<tr>
<td>Alrashdi S</td>
<td>296</td>
</tr>
<tr>
<td>Alruwaili ND</td>
<td>264, 289</td>
</tr>
<tr>
<td>Alruwali N</td>
<td>128</td>
</tr>
<tr>
<td>Alsop JA</td>
<td>114</td>
</tr>
<tr>
<td>Alunday RL</td>
<td>192</td>
</tr>
<tr>
<td>Amarasiriwardena C</td>
<td>218</td>
</tr>
<tr>
<td>Ammirati JF</td>
<td>194</td>
</tr>
<tr>
<td>Anderson CP</td>
<td>1</td>
</tr>
<tr>
<td>Anderson D</td>
<td>150</td>
</tr>
<tr>
<td>Anderson DL</td>
<td>117</td>
</tr>
<tr>
<td>Andrews R</td>
<td>221</td>
</tr>
<tr>
<td>Ann Howland M</td>
<td>274</td>
</tr>
<tr>
<td>Arain S</td>
<td>152</td>
</tr>
<tr>
<td>Armenian P</td>
<td>76, 102</td>
</tr>
<tr>
<td>Arnold T</td>
<td>13, 179</td>
</tr>
<tr>
<td>Arnold TC</td>
<td>163, 250</td>
</tr>
<tr>
<td>Artaelejo L</td>
<td>105</td>
</tr>
<tr>
<td>Ashworth A</td>
<td>306</td>
</tr>
<tr>
<td>Audld M</td>
<td>120</td>
</tr>
<tr>
<td>Austin T</td>
<td>262</td>
</tr>
<tr>
<td>Axel H</td>
<td>199, 208</td>
</tr>
<tr>
<td>Ayes KB</td>
<td>287</td>
</tr>
<tr>
<td>Babu KM</td>
<td>17</td>
</tr>
<tr>
<td>Badillo R</td>
<td>42, 132</td>
</tr>
<tr>
<td>Bailey B</td>
<td>230</td>
</tr>
<tr>
<td>Baird JR</td>
<td>17</td>
</tr>
<tr>
<td>Bakhsh SMF</td>
<td>203</td>
</tr>
<tr>
<td>Ballentine L</td>
<td>302</td>
</tr>
<tr>
<td>Barrera-Garcia V</td>
<td>286</td>
</tr>
<tr>
<td>Barreto F</td>
<td>217</td>
</tr>
<tr>
<td>Bartels M</td>
<td>167</td>
</tr>
<tr>
<td>Bartels MJ</td>
<td>166</td>
</tr>
<tr>
<td>Barthold CL</td>
<td>5, 139, 249</td>
</tr>
<tr>
<td>Barton N</td>
<td>38</td>
</tr>
<tr>
<td>Baumbacher T</td>
<td>23</td>
</tr>
<tr>
<td>Bayer M</td>
<td>176, 290</td>
</tr>
<tr>
<td>Bayer MJ</td>
<td>273</td>
</tr>
<tr>
<td>Bebarta VS</td>
<td>7</td>
</tr>
<tr>
<td>Becker D</td>
<td>235, 276</td>
</tr>
<tr>
<td>Begemann K</td>
<td>199, 208</td>
</tr>
<tr>
<td>Bellinger D</td>
<td>218</td>
</tr>
<tr>
<td>Benitez JG</td>
<td>172</td>
</tr>
<tr>
<td>Bennett HK</td>
<td>263</td>
</tr>
<tr>
<td>Benson B</td>
<td>84</td>
</tr>
<tr>
<td>Berlin RJ</td>
<td>230</td>
</tr>
<tr>
<td>Bertholf R</td>
<td>73</td>
</tr>
<tr>
<td>Besenhofer LM</td>
<td>167, 168</td>
</tr>
<tr>
<td>Beuerle T</td>
<td>77</td>
</tr>
<tr>
<td>Beuhlter MC</td>
<td>79, 265, 268</td>
</tr>
<tr>
<td>Biary R</td>
<td>105</td>
</tr>
<tr>
<td>Biland EF</td>
<td>44</td>
</tr>
<tr>
<td>Birdsall E</td>
<td>77</td>
</tr>
<tr>
<td>Birmingham CM</td>
<td>223</td>
</tr>
<tr>
<td>Bishop A</td>
<td>115</td>
</tr>
<tr>
<td>Biswas K</td>
<td>72</td>
</tr>
<tr>
<td>Blair HW</td>
<td>100</td>
</tr>
<tr>
<td>Blais R</td>
<td>270</td>
</tr>
<tr>
<td>Blasko JL</td>
<td>96</td>
</tr>
<tr>
<td>Bleeker ML</td>
<td>217</td>
</tr>
<tr>
<td>Bodmer M</td>
<td>6, 43</td>
</tr>
<tr>
<td>Boesen K</td>
<td>284</td>
</tr>
<tr>
<td>Bogdan G</td>
<td>260</td>
</tr>
<tr>
<td>Bogdan GM</td>
<td>252</td>
</tr>
<tr>
<td>Bomma L</td>
<td>143</td>
</tr>
<tr>
<td>Bonn R</td>
<td>60</td>
</tr>
<tr>
<td>Bora K</td>
<td>238</td>
</tr>
<tr>
<td>Borek HA</td>
<td>145, 159, 315</td>
</tr>
<tr>
<td>Borron SW</td>
<td>137</td>
</tr>
<tr>
<td>Borys DJ</td>
<td>100, 111</td>
</tr>
<tr>
<td>Bottei E</td>
<td>4, 123, 191, 248</td>
</tr>
<tr>
<td>Bouchard N</td>
<td>56</td>
</tr>
<tr>
<td>Bourdreaux S</td>
<td>7</td>
</tr>
<tr>
<td>Boyer EW</td>
<td>134, 313</td>
</tr>
<tr>
<td>Boyle K</td>
<td>215</td>
</tr>
<tr>
<td>Brent J</td>
<td>126, 237</td>
</tr>
<tr>
<td>Brighurst J</td>
<td>200</td>
</tr>
<tr>
<td>Broderick M</td>
<td>269, 294</td>
</tr>
<tr>
<td>Brody PM</td>
<td>300</td>
</tr>
<tr>
<td>Bronson AC</td>
<td>16, 89, 155, 158, 187, 239, 245, 256</td>
</tr>
<tr>
<td>Brooks DE</td>
<td>97, 112, 253, 261, 262</td>
</tr>
<tr>
<td>Brutlag J</td>
<td>116</td>
</tr>
<tr>
<td>Bryant SM</td>
<td>25, 26, 30, 133, 212</td>
</tr>
<tr>
<td>Buchanan JA</td>
<td>161</td>
</tr>
<tr>
<td>Bucher-Bartelson B</td>
<td>86</td>
</tr>
<tr>
<td>Buchholtz C</td>
<td>156</td>
</tr>
<tr>
<td>Bui A</td>
<td>86</td>
</tr>
<tr>
<td>Bunn TL</td>
<td>148</td>
</tr>
<tr>
<td>Burda AM</td>
<td>184</td>
</tr>
<tr>
<td>Burda T</td>
<td>61</td>
</tr>
<tr>
<td>Burkart K</td>
<td>72</td>
</tr>
<tr>
<td>Burns D</td>
<td>311</td>
</tr>
<tr>
<td>Burns J</td>
<td>94</td>
</tr>
<tr>
<td>Burns JM</td>
<td>134, 313</td>
</tr>
<tr>
<td>Burton BT</td>
<td>220</td>
</tr>
<tr>
<td>Bush C</td>
<td>146</td>
</tr>
<tr>
<td>Bush S</td>
<td>62, 68, 69</td>
</tr>
<tr>
<td>Bush SP</td>
<td>173</td>
</tr>
<tr>
<td>Butterfield R</td>
<td>73</td>
</tr>
<tr>
<td>Byrne T</td>
<td>174</td>
</tr>
<tr>
<td>Caces P</td>
<td>277</td>
</tr>
<tr>
<td>Calafat A</td>
<td>205</td>
</tr>
<tr>
<td>Caldwell K</td>
<td>72</td>
</tr>
<tr>
<td>Canning J</td>
<td>97, 138, 214</td>
</tr>
<tr>
<td>Cannon RD</td>
<td>59</td>
</tr>
<tr>
<td>Cantrell FL</td>
<td>49, 101, 190, 193</td>
</tr>
<tr>
<td>Cantrell L</td>
<td>3, 225, 292</td>
</tr>
<tr>
<td>Cantrell LF</td>
<td>47</td>
</tr>
<tr>
<td>Cantu G</td>
<td>157</td>
</tr>
<tr>
<td>Cao S</td>
<td>178</td>
</tr>
<tr>
<td>Caravati EM</td>
<td>65, 87, 263, 310</td>
</tr>
<tr>
<td>Carey M</td>
<td>258</td>
</tr>
<tr>
<td>Carlberg DJ</td>
<td>315</td>
</tr>
<tr>
<td>Carli L</td>
<td>86</td>
</tr>
<tr>
<td>Carlson T</td>
<td>152</td>
</tr>
<tr>
<td>Carr W</td>
<td>201</td>
</tr>
<tr>
<td>Carstairs SD</td>
<td>2</td>
</tr>
<tr>
<td>Cassidy AR</td>
<td>207</td>
</tr>
<tr>
<td>Castillo E</td>
<td>193</td>
</tr>
<tr>
<td>Castro CS</td>
<td>232</td>
</tr>
<tr>
<td>Cedarl A</td>
<td>130</td>
</tr>
<tr>
<td>Cescchi A</td>
<td>43</td>
</tr>
<tr>
<td>Cetarek E</td>
<td>126</td>
</tr>
<tr>
<td>Chambers-Emerson JA</td>
<td>276</td>
</tr>
<tr>
<td>Champagne L</td>
<td>177</td>
</tr>
<tr>
<td>Chan GM</td>
<td>107</td>
</tr>
<tr>
<td>Chan T</td>
<td>193</td>
</tr>
<tr>
<td>Chang A</td>
<td>266</td>
</tr>
<tr>
<td>Chang H-C</td>
<td>171</td>
</tr>
<tr>
<td>Charlton NP</td>
<td>145</td>
</tr>
<tr>
<td>Chen C-L</td>
<td>171</td>
</tr>
<tr>
<td>Chen H</td>
<td>178</td>
</tr>
<tr>
<td>Chen J</td>
<td>49</td>
</tr>
<tr>
<td>Chen L</td>
<td>298</td>
</tr>
<tr>
<td>Chen W-T</td>
<td>171</td>
</tr>
<tr>
<td>Christian MR</td>
<td>53, 63, 211, 254</td>
</tr>
<tr>
<td>Chu G</td>
<td>202</td>
</tr>
<tr>
<td>Chu J</td>
<td>74</td>
</tr>
<tr>
<td>Chung W</td>
<td>157</td>
</tr>
<tr>
<td>Cienki J</td>
<td>226</td>
</tr>
<tr>
<td>Cima AG</td>
<td>187</td>
</tr>
<tr>
<td>Clardy J</td>
<td>178</td>
</tr>
<tr>
<td>Clark A</td>
<td>225</td>
</tr>
<tr>
<td>Clark RF</td>
<td>101, 190, 193, 225, 227</td>
</tr>
</tbody>
</table>
NACCT Author index

Clark S 224
Clarke E 277
Cleary JL 119
Clement A 104
Clements A 284
Cline J 178
Cobough DJ 298
Cocchio C 41
Cohle SD 14
Cole JB 1, 22, 34, 44, 55, 66, 92, 109, 110, 117, 118, 121, 198, 204, 213, 229, 304
Conley B 42
Conklin L 165
Cookman L 36
Corneli HM 32
Cortes DR 184
Cottrell BV 17
Craig DG 8
Craycroft JA 178
Cresswell AG 160
Croal B 78
Crouch BI 87, 246, 263, 310
Croucher BI 65
Cummins MR 246, 263
Cumpston K 50, 95
Cumpston KL 31, 164, 188
Curry S 214
Curry SC 72, 309
Daniels B 176
Dargan PI 12, 40, 81, 151, 267
Darracq MA 49
Dart RC 6, 86, 156
Davis JM 156
De La Calzada-Jeanlouie MF 107
Deal SA 255
Dear JW 8
Debroux KM 305
Degrandi C 39
Derau KV 36
Dervay K 276
Dixon P 7
Dodd-Butera T 269, 294
Dolcourt B 209
Dommer P 261
Donaldson K 27
Dong KA 230
Donovan JW 160
Dorval M 270
Dougdherry PP 85
Doyon S 15, 147
Dribben W 99
Dulaney AR 33, 255
Durrani AR 33, 255
Durrani T 51
Dux J 181
Edwards EM 22, 66
Eisenberg DM 178
Ekperi L 149
Elko CJ 96, 194
Ellington L 263
Ellsworth H 1, 22, 34, 55, 66, 92, 109, 110, 117, 118, 121, 198, 213, 229, 304
Engebretsen KM 1, 55, 229, 241
Engelund J 91
Enyart N 252
Erickson TB 316
Farmer B 175, 224, 242
Farmer BM 56
Farrar RD 280
Fathalla SM 144, 303
Feldstein E 234
Fenzi M 165
Fenzi MJ 166
Finkenstein G 272
Fischbein C 189
Fischbein CB 211
Fletcher A 90
Forrester JD 145
Forrester MB 29, 35, 105
Fort BC 254
Foster HR 162
Fowler D 147
Frederic WA 287
Freeman RJ 194
French LK 67, 142, 220, 240
French R 253, 309
Friedemann M 208
Friend K 27
Fu Y 178
Furbee B 154
Fusco B 232
Galinato M 193
Gallo AM 257
Garcia-Orr R 138
Gardner M 106, 122, 182
Garlich FM 98
Garrard A 73, 131
Gee AS 230
Gebi A-J 41
Geller R 108, 152, 183, 259
Geller RJ 131, 149, 243, 244, 266, 271, 296
Gernino KW 37
Geron R 3, 21
Geron RR 76, 77
Gessler M 39
Gessner M 208
Gesteland P 246
Gilbreath A 256
Ginsburg BY 143
Gochnour J 19
Goetz R 150
Goldfrank LR 277
Goldring C 8
Gordetsky R 108, 259, 264
Gosselin S 230
Gray J 144, 303
Green J 6, 46, 86
Greenberg MI 58
Greene SL 12, 40
Greller HA 107
Gross V 270
Gummin D 150
Ha E 69
Habicht M 125
Hall SL 120
Halcomb S 24
Hall M 234
Hall VR 159
Halliday M 311
Ham K 281
Hamm KM 275, 283, 288
Haney E 230
Haneaian-Fontana AL 273
Harcumford P 11
Hardison L 210
Harris CR 229
Harris ES 178
Harrison CN 266
Hart K 129, 273
Hart K 6, 16, 46, 126
Haur SE 245, 275, 281, 283, 288
Heinen M 272
Hendrickson RG 67, 130, 142
Henretig F 52, 251
Heronon C 94
Herrington LJ 271, 296
Hershorin E 158
Hess JM 92
Hew C 300
High GM 134
Hildebrand JM 313
Hill M 150
Hill RA 289
Himebauch A 52
Hlavenka LM 1, 229
Hofer KE 43
Hoffman RS 56, 80, 195, 223, 226, 274
Holger JS 1
Hollan M 103
Holstege CP 145, 154, 159, 201, 315
Hoyle DW 11
Holzeman SD 284
Honn S 108, 259, 271, 296
Honn SL 131, 149, 244, 266
Hussainian A 203
Hovesth K 132
Howerd MA 56, 98
Hoyte C 16
Hsiah CF 171
Huang P 256
Huang SY 171
Hudson H 257
Hughs KA 213
Humphries F 179
Hunt DR 166
Huntington S 152
I
Inciardi JF 114
Isaacson BA 44
Jackson DA 17
Jacob J 16, 140

Clinical Toxicology Downloaded from informahealthcare.com by University of Zuerich on 09/15/11
For personal use only.
Jacobitz K 5
Jacobitz KL 169, 249
James LP 162
Jang DH 195
Jansen J 13
Jantz JM 287
Jaramillo JE 280
Jenkins A 28
Jimenez C 286
Johanssen L 73
John N 48
Johnson DW 230
Jolliff H 19
Jones JS 14, 233
Jovane R 165
Judd M 89
Judge BS 14, 115, 146, 221, 233
K
Kaiser BM 51
Kalimullah EA 316
Kaln L 4, 248
Kamali M 38
Kanie K 99
Kao L 136
Kaptchuk T 178
Karydes HC 93, 133, 164
Katz KD 88
Kazmi 75
Kazmi N 71
Kazzi ZN 264, 279, 289, 296
Kekic Z 206
Kerns II W 91
Keyler DE 213
Kezirian R 152
Khan Y 300
Khatti VK 181
Khanlari M 274
Kirschner R 5
Kirschner RI 139, 169, 305, 307
Klein-Schwartz W 15, 84, 85
Koe F 206
Komatsu K 253
Kowalski JM 54, 299
Krauss J 292
Kremzholok EP 185, 247, 291
Kreshak AA 2
Krishna CV 181
Kusnitski T 73
Kupferschmidt H 39, 43
Kupsisp D 88
Kwon SK 92, 109, 204, 213, 304
L
Lackey GD 114
LaFleur, Jr G 191
Lairet J 7
Landry GM 167
Lank PM 282
Latimer S 263
Laverty H 8
Lavonas EJ 161
Leder C 18
Lee C 260
Lee S 15, 85
Lehr B 150
Leikin JB 61, 127, 164
Lelesi J 124
Letarte A 270
Levine M 138, 174, 177, 202, 301, 309
Lewis G 252
Lewis PS 8
Lewis-Younger C 276
Linakis JG 17
Lipton CP 44, 92, 118, 204, 213, 304
Lipshultz SE 158
Litovitz T 84
Littlefield BA 178
Lu J 178
Livshits Z 80
Lloyd RM 110
Lo JC 47
Lock B 264
Long C 24
Long CL 28
Lopez G 108
Lopez GP 28, 149, 243, 244
Lord JA 230
Louie J 274
LoVecchio F 72, 261, 262
Lorwy J 83, 104, 285
Lu J 189, 211, 316
Lugassy DM 80
Lung D 76, 85
Luo J 5
Ly A-V 62, 68, 69
Ly BT 308
Lynch MJ 57, 59, 88, 222
M
Mackenzie TD 161
Macy M 234
Madrigal V 52
Madsen JE 310
Manini AF 143
Marquardt KA 114
Martin TG 194
Martinez L 274
Maynard S 79
McAtee DK 183
McCain KR 162
McCloskey J 52
McCormick A 189
McCormick L 279
McGaul P 179
McKay C 129
McKay L 78
McKenna S 217
McKewon NJ 67, 142, 220, 240,
McKnight K 272
McLaren MC 168
McMartin KE 165, 166, 168
Medley K 74
Merchant RC 17
Mercurio-Zappala M 274
Meyer H 199
Milizia R 56
Miller M 36
Miller MJ 298
Miller SN 58
Miller T 251
Milone M 27
Minns A 225, 227
Minns AB 308
Misfeldt CF 241
Moggs J 8
Monte AA 16, 187
Moran JH 162
Morgan B 153, 186, 259
Morgan CW 128, 226, 284, 289
Morgan DL 100, 111
Morrissey RP 100, 111, 182
Moser KL 121
Morey J 20, 136, 150
Mrvos R 88, 185, 247, 291
Murphy CM 79
Murphy NG 60, 230, 258
Murray S 21
Musselman ME 238
Muthukutty A 263
Mydchak MB 53, 93, 254
Myers RP 230
Myracle AB 6
N
Nadindla S 143
Nanagas K 302, 311
Nanagas KA 219
Narasimhan S 45
Nasr S 72
Neer T 154
Nelsen J 103
Nelson L 56, 267
Nelson LS 98, 195, 223, 226, 293
Nesbitt 11
Nettel-Aguirre A 230
Nguyen K 190
Nguyen T 62, 68, 69
Nguyen V 193
Nirenberg T 17
Nishijima DK 232
Nogar CN 170, 308
Noranbrock C 147
Nord S 292
Nord SP 193
Nyadedzor C 277
O
O’Connor AD 301
Obafemi AI 182
Offerman S 231
Offerman SR 51
Olives TD 34, 198
Olson KR 76, 102
Omid M 64
Ormsby A 18
Ozorozou BS 117, 141
Ozturk J 11
Osterhoudt KC 52, 90, 234, 251
Othong R 264
Owen KP 232
P
Pakravan N 64, 203
Palermo P 251
Pallasch EM 53
Pandalai SL 128, 131
Park K 8
Parker A 285
Pearl SR 173
Perala A 165
Perala AW 166
Peranteau W 90
Perrone J 27
Peterson L-K 70
Pettrey J 50
Phillips S 126
Pines E 282
Pittman JH 276
Pizon AF 57, 88, 197, 210, 222, 241
Plascencia A 196
Plascencia AA 99
Plascencia AMA 192
Platt V 8
Plumb J 32
Poterucha JT 307
Prosser JM 56, 226
Puchnarewicz M 81
Purssell RA 230
Q
Quang L 45, 71, 75, 82
Quang LS 222
R
Rafie AR 203
Ragone S 28, 108
Ramsey J 81
Randall DP 127
Raney J 104
Rao R 56
Rasimas JJ 160
Rasmussen M 5
Rauber-Luthy C 43
Reedy SJ 153, 266
Reeve J 78
Reilly DA 307
Rembert RS 268
Rentmeester LL 14
Rey-Casserly C 207
Riley B 200
Ripple M 147
Riyaz R 186, 296
Robbins D 215
Roberts MR 184
Roche KM 290
Rogers D 126
Romasco R 101
Roque P 202
Rose R 31, 50, 95, 188
Rose SR 295
Rosenbaum CD 28
Rossman L 146
Roth B 106
Rudser AK 118
Ruha A-M 72, 174, 177, 180, 202
Rumack BH 6, 230
Russell JW 240
Rutledge M 276
Ryan ML 9, 13, 163, 179, 250
Rynni KO 41
S
Saag KG 298
Saatloff GB 154
Sabel A161
Sabogal R 165
Sachdeva K 160
Saely S 238
Saenz E 105
Salingar L 176
Salingar LM 273
Salzman MS 299
Sanchez A 48
Sanchez CA 165
Sanftleben J 196
Sangalli B 176, 290
Sarid H 101
Sawalha A 218
Scalzo AJ 24, 28, 37
Schaecker JL 158
Schaeffer S 42, 132
Schaeffer TH 86, 140
Schaumburg H 195
Schier JG 165, 166
Schlichting A 18
Schneissing S 302
Schneir A 23, 48
Schneir AB 170
Scholtens R 178
Seifert SA 173
Seifert SA 158
Seifertl SM 158
Sein V 174
Sejvar JI 165
Seroke AM 252
Setzer SC 109
Severtson SG 156
Shah SK 98
Shaw K 90
Shaw-Luchman T 128
Shayne P 289
Sheikh S 186, 228, 264
Shen S 190
Sheppard K 60, 258
Shinar B 138
Shirazi F 284
Sibbald KN154
Simeonov L 281
Simeonov JM 275, 283, 288
Simpson KJ 8
Simpson S-E 54
Sinha V 260
Siviglioti ML 226, 230
Sivigliotti MLA 6
Skinner CG 36
Skolnik A 112, 309
Slavova SS 148
Small R 251
Smith DS 172
Smith J 72
Smith P 251
Smith S 62, 68, 69
Smith SW 141
Smolcic EE 160
Smolinske S 124, 150
Smollin C 21, 76
Smyth D 257
Snodgrass WR 119
Sogohian S 277
Soleymani K 101
Sollee D 73
Sonier T 60
Spencer JW 24
Spiller HA 9, 13, 148, 265
Spyker DA 155, 230
Stellplug SJ 1, 34, 44, 55, 109, 110, 121, 229, 304
Stephen D 78
Stolbach A 226, 278
Stone J 201
Stromness JR 87
Strong D 152
Su MK 107
Suchard J 3, 125
Sullivan RW 103
Sutter ME 232
Sweet P 49
Swiecieh W 218
T
Taha AA 218
Taik Ha ET 62, 68
Tanan D 7
Tanan DA 2
Thomas E 181
Thomas KC 65
Thomas R 124
Thomas RG 32
Thomas SH 144, 303
Thompson JD 119
Thompson JP 181
Thompson M 230
Thompson RM 111
Thompson TM 63
Thornton SL 48, 101, 190, 227
Tomassoni AJ 260
Tomaszewski C 190
Topeff JM 22, 66, 198
Tormoehlen L 20, 136
Tormoehlen LM 219
Tremblay D 239
Tremmel R 99
Trestallin III JH 297
Truitt CA 112, 177, 180, 261, 301
Tsatooka B 51
Tullloch F 163
Turner JM 210
Tutag Lehr V 124
U
Ubaldo C 47
V
Valdez T 89
Valento M 30, 61
Van den Bogert CA 298
VanDePol E 115
Vargas T 7
Vecchio FL 202

Clinical Toxicology Downloaded from informahealthcare.com by University of Zuerich on 09/15/11
For personal use only.
<table>
<thead>
<tr>
<th>Name</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Victoria G</td>
<td>165</td>
</tr>
<tr>
<td>Victorino C</td>
<td>230</td>
</tr>
<tr>
<td>Vilke GM</td>
<td>193</td>
</tr>
<tr>
<td>Villarreal L</td>
<td>286</td>
</tr>
<tr>
<td>Vohra R</td>
<td>183, 267, 277, 297</td>
</tr>
<tr>
<td>Wahl M</td>
<td>53, 150</td>
</tr>
<tr>
<td>Waliko T</td>
<td>201</td>
</tr>
<tr>
<td>Walsh SJ</td>
<td>57, 88, 197, 222</td>
</tr>
<tr>
<td>Walter F</td>
<td>279</td>
</tr>
<tr>
<td>Walters H</td>
<td>83</td>
</tr>
<tr>
<td>Wang R</td>
<td>205</td>
</tr>
<tr>
<td>Wang W</td>
<td>178</td>
</tr>
<tr>
<td>Ward JA</td>
<td>134</td>
</tr>
<tr>
<td>Wares C</td>
<td>91</td>
</tr>
<tr>
<td>Waring E</td>
<td>295</td>
</tr>
<tr>
<td>Warrick B</td>
<td>150, 209</td>
</tr>
<tr>
<td>Wasserman G</td>
<td>83, 104</td>
</tr>
<tr>
<td>Watkins B</td>
<td>314</td>
</tr>
<tr>
<td>Watts S</td>
<td>137</td>
</tr>
<tr>
<td>Wax F</td>
<td>38, 237, 267</td>
</tr>
<tr>
<td>Wax PM</td>
<td>157, 216, 255</td>
</tr>
<tr>
<td>Webb AN</td>
<td>255, 300</td>
</tr>
<tr>
<td>Weber JA</td>
<td>24, 28, 37</td>
</tr>
<tr>
<td>Weerasuriya D</td>
<td>228</td>
</tr>
<tr>
<td>Weibrecht KW</td>
<td>134</td>
</tr>
<tr>
<td>Welch S</td>
<td>262</td>
</tr>
<tr>
<td>Wermuth M</td>
<td>302</td>
</tr>
<tr>
<td>Westenberger B</td>
<td>72</td>
</tr>
<tr>
<td>Weston RG</td>
<td>9, 13</td>
</tr>
<tr>
<td>White LW</td>
<td>119</td>
</tr>
<tr>
<td>Whiteley PM</td>
<td>127, 164, 189</td>
</tr>
<tr>
<td>Whitmore T</td>
<td>256</td>
</tr>
<tr>
<td>Wiegand T</td>
<td>38, 70, 113</td>
</tr>
<tr>
<td>Willhite LA</td>
<td>22</td>
</tr>
<tr>
<td>Williams SR</td>
<td>172, 314</td>
</tr>
<tr>
<td>Wills B</td>
<td>50, 95</td>
</tr>
<tr>
<td>Wills BK</td>
<td>31, 164, 188</td>
</tr>
<tr>
<td>Winter E</td>
<td>156</td>
</tr>
<tr>
<td>Witucki AS</td>
<td>170</td>
</tr>
<tr>
<td>Wolf CE</td>
<td>31</td>
</tr>
<tr>
<td>Wolk BJ</td>
<td>94, 134</td>
</tr>
<tr>
<td>Wong L-Y</td>
<td>205</td>
</tr>
<tr>
<td>Wood DM</td>
<td>12, 40, 81, 151, 267</td>
</tr>
<tr>
<td>Woolard R</td>
<td>137</td>
</tr>
<tr>
<td>Woolf AD</td>
<td>178, 207</td>
</tr>
<tr>
<td>Woolley B</td>
<td>146</td>
</tr>
<tr>
<td>Worley Z</td>
<td>311</td>
</tr>
<tr>
<td>Wright RO</td>
<td>218</td>
</tr>
<tr>
<td>Wright E</td>
<td>132, 210</td>
</tr>
<tr>
<td>Wright N</td>
<td>244, 271</td>
</tr>
<tr>
<td>Wu A</td>
<td>3</td>
</tr>
<tr>
<td>Wu AH</td>
<td>76, 77</td>
</tr>
<tr>
<td>Wu M-L</td>
<td>171</td>
</tr>
<tr>
<td>Wyckoff A</td>
<td>246</td>
</tr>
<tr>
<td>Wyke S</td>
<td>155</td>
</tr>
<tr>
<td>Yang C-C</td>
<td>171</td>
</tr>
<tr>
<td>Yarema MC</td>
<td>230</td>
</tr>
<tr>
<td>Ye W</td>
<td>72</td>
</tr>
<tr>
<td>Young AC</td>
<td>182</td>
</tr>
<tr>
<td>Young LA</td>
<td>201</td>
</tr>
<tr>
<td>Young M</td>
<td>231</td>
</tr>
<tr>
<td>Zahnd R</td>
<td>39</td>
</tr>
<tr>
<td>Zeager M</td>
<td>207</td>
</tr>
<tr>
<td>Zeiler A</td>
<td>225</td>
</tr>
<tr>
<td>Zell-Kanter M</td>
<td>61</td>
</tr>
<tr>
<td>Zhao Z</td>
<td>178</td>
</tr>
<tr>
<td>Ziegler A</td>
<td>221</td>
</tr>
<tr>
<td>Zuckerman M</td>
<td>94</td>
</tr>
</tbody>
</table>
NACCT Keyword index

1,4 Butanediol 31
2C-E 66
3-dimethylamylamine (DMAA) 177
4-aminopyridine 176
4-AP 312

A
Abuse
Accident categorization 208
Accidental 131
Acepromazine 306
Acetaminophen 6
Acetaminophen overdose 46
Acetylcholinesterase inhibitor 98
Acetylferrocene 316
Acetylsalicylic acid 115
Acidosis 225
Acute cardiomyopathy 301
Adherence 263
Adolescent 17
Adoption 209
Adulteration 157
Adverse Drug Event 255
Adverse effects 39
Adverse Events 86
Agkistrodon contortrix 188
Alabama NSAID Patient Safety Study 298
Alcohol 6
Alcohol abuse 232
Alcohol dehydrogenase 31
Alcohol intoxication 14
Alcohol Withdrawal 70
Allergy 285
Almendra Quema Grasa 187
Alpha 2-adrenergic antagonist 143
Alternative Medicine 126
Aluminum Phosphide Toxicity 203
Amanita muscaria 189
Amanita pantherina 204
Amanita Phalloides 194
Amanadine 304
Amatadine 51
Aminita virosa 197
Aminotransferase 231
Amiritpyline 104
Amphetanamne 22
Amputation 177
Amygdalin 184
Analog drugs 9
Anticholinergic 44
Anticonvulsants 141
Antidote 168
Antiemetic 120
Antihelminthic 122
Antineoplastic 101
Antipsychotic drugs 144, 303
Antivenom 2, 65, 68, 69, 179, 190, 192, 223, 234
Anxiety 71
APAP 224
App
Appendectomy 42
Appendolith 42
Apple seeds 184
Arachnologist 211
Argyria 186
Arriprazole 88
Arrhythmia 144
Arsenic 216, 219
Atuxia 115, 305
Atropine 44
Atypical antipsychotic 124
Awareness 272
B
Barbital 95
Bath salt 13, 150
Benzoephonene 81
Beta Blocker 1
Billing 5, 249
Biomarkers 82
Biomonitoring 205
Biosurveillance 183
Bleach 313
Blood 218
Blood Levels 227
Board certification 293
Body Packer 64
Bodybuilding supplement 176
Bone Lead Compartment 45
Botulinum 132
Brodifacoum 302
Bronchoalveolar lavage 52
Bupivicaine 130
Buprenorphine 15

C
Caffeine 97, 158
Caffeine toxicity 97
Calcium Channel Blocker 96, 238, 261
Call Center Response 252
Call Volume 5
Cannabiniod 16, 26, 27, 100, 150, 161, 226
Capcitabine 29
Car battery 208
Carbamazepine 34
Carbon monoxide 148, 251
Carbon monoxide poisoning 273
Carboxyhemoglobin 80
Cardiac arrest 19, 66
Cardioactive Steroid 198
Cardiogenic Shock 1, 55, 141
Cardiomyopathy 134, 176
Cardiotoxicity 314
Cardioversion 223
Cardiovascular collapse 309
Case-control 166
Cathinone 163
Cathinone (+ derivatives) 78
Cathinone analog 13
Caucitc 212
Cefepime 142
Cesium 126
Change over time 155
Charcoal 50, 52
CHARGE Syndrome
Chelation 72, 222
Chelation therapy 214
Chelonitoxism 169
Chemical disaster 260
Chemical suicide 153
Chemistry Class 212
Chest pain 226
Child Abuse 16, 110
Children 84, 124
Children < 6 years old 114
China 209
Chinese Golden Scorpion 211
Cyanide 7, 125
Cyanogenic glycoside 184
Cyanokit 48
Cytosin 34
CVVHD 63
Cyanide 7, 125
Cyanogenic glycoside 184
Cyanokit 48

624
Dalmefradrine 312
Death 30, 145, 182, 202, 305
Decision support 263
Decontamination 60
DEET 200
Delayed 88
Delayed hepatotoxicity 310
Delayed seizure 87
Delirium 122
Delirium Tremens 70
Delphi 246
Demographics 237
Dental fillings 215
Dermal 140
Dermatitis 221
Designer drugs 13
Detergent suicide 153
Dexmedetomidine 70
Dextromethorphan 37
Diazepam 311
Dietary supplements 175
Diethylene glycol 165, 166, 167
Dieting agent 195
Differences 296
Diglycolic acid 166
Digoxin 59
Digoxin toxicity 123, 129
Digoxin-specific antibody fragment 59
Dinner or monitoring acid 207
Dissolvable nicotine products 105
Disulfiram 56
Dental mercury 311
DMPS 72
DMSA 207, 227
DNR 228
Dog 116
Donepezil 98
Drill 264
Drug Availability 156
Drug Eruptions 136
Drug interactions 139
Drug level 59
Drug of abuse 35
Drug shortage 276
Drug-induced Liver Injury 136
Drugs 242
Drugs of Abuse 9, 21, 25

E
Education 161, 224, 278, 288, 294
Electronic information exchange 246
Emergency Department
Emergency medicine 206, 293
Emergency response 250,
Emergency toxicology screening 76
Emergency Training 289
Emetine 110
End-stage renal disease 171
Energy drink 193, 239
Energy Drinks 158
England 155
Enhanced Elimination 49, 67
Enteral fistula 90
Envenomation 145, 170, 173, 174, 182,
188, 192, 213, 234
Environmental exposure 119
Epidemiology 148

F
Fab antivenom 173
Face 180
Factitious Disorder 34, 220
Fat emulsion 74
Fatality 145
FDA 175, 242
Ferrocene 316
Fibromyalgia 138
Fireworks 254
Fix-a-Flat 235
Flumazenil 113
Fluorouracil 99
Fly agaric 189
Fomepizole 7, 80, 125
Foreign bodies 90
Foreign body reactions 235
Forensic toxicology 14
Fosphenytoin 141
Fulminant Hepatic Failure 197

G
Gamma-hydroxybutyrate 31
Gar fish egg poisoning 191
Gastrointestinal 133
Gastrointestinal symptoms 190
Geriatric drug abuse 26
Geriatrics 25
Glucagon-like peptide-1 112
Glucocorticoid 34
Glues and Adhesives 89
Gorilla Glue 89
Guanciacine 100

H
H1N1 Pandemic 252
Hand/foot warmers 116
Hazard vulnerability analysis 260
HCl 212
Health Emergency 252
Health information 281
Heat Stroke 137, 202
Heavy Metals 178, 216
Hematologic recurrence 173
Hemlock 196
Hemodialysis 48, 49, 57, 63, 142, 314
Hemolysis 83
Hemolytic anemia 172
Hemorrhage 302
Hepatic injury 39
Hepatotoxicity 46
Herbal incense 77
Herbal marijuana alternative 28
Herbal toxins 183
Hg exposure 215
Hg vapor 215
High Concentration Insulin Infusion 238
High Dose Insulin 1, 55, 238
Home safety 251
Homeopathic medicine 138
Homicide 146
Hospital admission 155
Huffing 32
Human Exposure 140
Hydrocode drug interaction 127
Hydrogen peroxide 133
Hydrogen sulfide suicide 153
Hydroxocobalamin 7, 80, 125
Hydroyurea 235
Hypercarbia 126
Hyperkalemia 129
Hypermagnesemia 91
Hypermatemia 315
Hypothermias 137
Hypocalcemia 135
Hypokalemia 195, 211
Hyponatremia 27
Hypophosphatemia 11
Hypovitaminosis D 135

I
Ibuprofen 305, 309
Ichthyootoxin 191
Identification 247, 299
Illicit drug 64
Illicit drug use 36
Illicit drugs of abuse 9
In Utero Exposure 51, 75
Incapacitated rape 146
Incarceration 139
Incentive 271
Information sources 290
Information technology 297
Ingestion 179, 254
Ingredients 3
Inhalant abuse 32
Inhalation 95
Inmate 152
Insect repellent 200
Insulin 108, 111, 265
Insurance company 4
International 155, 267
Internet 278, 290
Intoxication 240
Intraperitoneal 217
Intravenous fat emulsion 41, 55, 66
Intravenous fat emulsion therapy 53
Ipecac 110
IVR 247

J
JWH 162

Copyright © Informa Healthcare USA, Inc. 2011