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ABSTRACTS

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1. Comprehensive drug analysis of "bath salts" purchased in the United States

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Background: In the last few years "bath salts", sold over the internet at various shops, have emerged as new products of abuse. Qualitative product analysis and testing of patients who have used them have revealed multiple cathinones. One U.S. study analyzed 11 products marketed as "bath salts" purchased from separate stores in 2 states. The presence of 3 synthetic cathinones (mephedrone, MDPV, and methylone) were found alone or in combination. Caffeine was found in 1 product and an "unknown compound" in another. Our goal was to analyze both qualitatively and quantitatively a number of products sold as "bath salts" in the U.S. in a comprehensive manner.
Methods: A variety of products sold as "bath salts" were purchased over the internet from U.S. sites, and in person at multiple retail stores in locations throughout California. At each internet site or store differently named "bath salts" were purchased. Product labels including warnings and description of effect or intended use were recorded. The drug content of each product was assayed using liquid chromatography-time-of-flight mass spectrometry (Agilent LC 1200-TOF MS 6230). Triplicates of each sample were run for quantitative analysis.
Results: A total of 36 products were purchased (26 from stores) between August 11 and December 15, 2011. Prices ranged from $9.95–49.99. The majority of products (29) had a label "not for human consumption" and 3 others had a similar comment. Nine products detailed they should not be sold to minors and 4 products instructed doctor contact if consumed. To date qualitative analysis has been performed on 29 products with quantification completed on 8. Every product contained at least 1 cathinone. Nine different cathinones were found [cathinone (# products found in)]: {MDPV (14), methylone (7), ethylone (6), mephedrone (3), flephedrone (3), ethcathinone (3), α-PVP (3), pentedrone (2), and butylone (1)}. Three designer amphetamines were also found (fluoroamphetamine, ethylamphetamine, 5-iodo-2-aminoindane) in separate products. Other drugs identified included caffeine (6), lidocaine (3), and doxylamine (3). Heterogeneous composition was observed during quantitative analysis which was resolved by thorough mixing of each product. A wide range in total quantity of principal cathinone in products existed (106 to 334 mg).
Conclusions: Similar to the previous U.S.-based study MDPV, methylone, mephedrone, and caffeine were all identified. Our comprehensive analysis also identified the presence of multiple other cathinones, some designer amphetamines, lidocaine, and doxylamine. Additionally, quantification has to date revealed a wide range in quantity of the principal cathinone in each product.
Keywords: Cathinone, Bath salts, MDPV

2. The epidemiology of mushroom ingestion calls to US poison control centers: 2001–2011

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Background: There have been neither national multiyear analyses of mushroom ingestions handled by poison control centers (PCCs), nor spatial analyses of these ingestions.
Objectives: To describe the epidemiology of mushroom ingestion calls to PCCs. To perform a GIS/Spatial Epidemiology analysis of mushroom ingestion calls.
Methods: A retrospective analysis of PCC call data. NPDS database search was performed, from 2001–2011 with matching mushroom codes. Demographics, toxin group, specific species ingested, clinical outcome, treatment location, and caller zip code were identified. Descriptive analysis was performed with Excel and Stata; Spatial analysis was performed with SaTScan and ArcGIS.
Results: 83140 mushroom ingestions were reported to US PCCs from 2001–2011. 64534 (77.6%) were pediatric ingestions and 48437 (58.3%) were under 6 years old. 48423 (58.2%) of patients were male. 65255 (78.5%) of ingestions were unintentional. Identification was made in only 4232 (5.1%) exposures and included 185 distinct species. The 5 most common species (number of identifications): 1. Morchella angusticeps (507) 2. Chlorophyllum molybdites (374) 3. Amanita muscaria (319) 4. Lycoperdon candidum (228) 5. Calvatia lepidophora (175). The toxin group was identified in 12147 (14.6%) of ingestions. The toxin groups (number of identifications) were: 1. Cyclopeptides

(512) 1a. Orellanine (41) 2. Muscimol (Ibotenic Acid) (480) 3. Monomethylhydrazine (MMH) (448) 4. Muscarine and Histamine (284) 5. Coprine (141) 6. Hallucinogenics (Psilocybin and Psilocin) (8375). 7. Gastrointestinal Irritants (1866). Of the symptomatic cases, effects were minor in 10953 (56.5%), moderate in 7804 (40.3%), major in 568 (2.9%), and death in 45 (0.2%). Of the 614 cases of major effect or death, a species was identified in 64 (10.4%). The 5 most common species (number of cases) identified as responsible for major effects or death were: Amanita phalloides (27), Amanita muscaria (22), Amanita pantherina (9), Amanita Smithiana/Proxima/Pseudoporphyria (5), & Amanita bisporigera (3). Of the 97 cases where Amanita phalloides was identified, 23 (23.7%) resulted in major effects and 4 (4.1%) in death. In the 59 cases of Amanita phalloides ingestions where clinical effects were confirmed, 23 (39.0%) resulted in major effects and 4 (6.8%) in death. Maps demonstrating ingestion broken down by species of interest, toxin group, and clinical effects will be presented.
Conclusions: Calls to US PCCs regarding mushroom ingestions occur commonly. However, the mushroom species or toxin group is infrequently identified. Major effects or death are rare, but symptomatic Amanita phalloides ingestions appear to be at the highest risk.
Keywords: Mushroom poisoning, Epidemiology, National Poison Data System

3. Diglycolic acid, the nephrotoxic metabolite of diethylene glycol, produces cytoxicity via molecular mimicry and metabolic disruption

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Background: Diethylene glycol (DEG) is an organic solvent used in antifreeze blends, brake fluid, and the production of various polymers allowing the risk of consumer exposure. The hallmark of DEG poisoning is acute renal failure caused by cortical tubular degeneration and proximal tubule necrosis. DEG is metabolized to two primary metabolites, 2-hydroxyethoxyacetic acid (2-HEAA) and diglycolic acid (DGA). The main hypothesis of this study is that DGA produces proximal tubule cell necrosis seen in DEG poisoning via a molecular mimicry mechanism leading to metabolic disruption, and ultimately mitochondrial dysfunction.
Methods: For transporter inhibition studies, confluent cells were incubated with N-(p-amylcinnamoyl)anthranilic acid (ACA), a sodium dicarboxylate transporter (NaDC-1) inhibitor or 2, 3-dimethylsuccinate (2,3-DMS), an NaDC-3 inhibitor, and increasing concentrations of DGA (0–100 mmol/L) for 48 h. Necrotic cell death was measured via ethidium homodimer uptake. Oxygen consumption was determined in whole cells using a standard Clark-type electrode in cells pre-treated with DGA for 6 h. ATP levels were determined using luciferase-coupled bioluminescence at various time points (0–36 h). Reactive oxygen species formation from 0–12 h was detected by the conversion of dihydrorhodamine to the fluorescent product rhodamine 123. Succinate dehydrogenase (SDH) and fumarase activities were measured spectrophotometrically using purified enzyme in the presence of increasing DGA concentrations.
Results: ACA, but not 2,3-DMS, decreased the degree of DGA-induced cell death by approximately 50% suggesting a specific role of NaDC-1 in DGA intracellular uptake. DGA decreased oxygen consumption by approximately 30% in DGA-pretreated cells compared to controls. This effect corroborated the severe DGA-induced ATP depletion seen at 36 h, which most likely resulted from decreased oxidative phosphorylation in the mitochondria. DGA significantly decreased SDH activity at 50 and 100 mmol/L, but had no effect on fumarase activity suggesting the ability of DGA to mimic succinate, but not fumarate or L-malate. DGA also produced a concentration and time dependent increase in total cellular oxidant production indicating disruption of cellular redox status.
Conclusions: These results indicate that after internalization, DGA likely induces proximal tubule cell dysfunction by specific mitochondrial-mediated processes, which lead to decreased energy production and ultimately cellular necrosis.
Keywords: Mitochondrial dysfunction, Kidney failure, Dicarboxylate transporters

4. Investigation of an outbreak of photokeratitis

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Background: Photokeratitis and burns caused by ultraviolet (UV) radiation have been reported from damaged metal halide bulbs when the protective outer bulb casing is not intact. On December 5th, 2011, an emergency physician contacted the regional poison center after evaluating a cluster of 13 patients who had presented to her emergency department (ED) in the middle of the night with complaints of eye pain. The patients had attended the same high school event earlier that day but no common exposure could be identified. A medical toxicologist was consulted and surveillance for other patients in surrounding EDs was performed by the poison center CSPI. A Hazmat investigation was initiated. Ninety nine persons presented to area EDs and skin and eye decontamination was performed.
Methods: The Department of Public Health conducted an investigation to characterize the exposure and symptom history of approximately 1,000 event attendees. IRB approval was not obtained because it was a public health investigation. City Police, Fire, and Health Departments completed an in-depth examination of the high school gymnasium, which included reviewing video footage and testing for hazardous chemicals. A web-based survey was distributed on December 7th to capture attendees' exposure and symptom history. A case was defined as a person with 2 or more acute eye symptoms who attended the competition.
Results: Surveys were completed by 760 persons, representing approximately 76% of attendees. Two hundred forty two (32%) respondents met the case definition. Acute eye symptoms among cases included burning eyes (96%), red eyes (89%), tearing eyes (82%), foreign body sensation (82%), blurry vision (57%), and eyelid swelling (51%). Of the 127 individuals who sought care, 99 (78%) went to EDs. The median time between arriving at the gym and symptom onset was 9 hours (range: $<$ 1–72 hours). Risk of becoming a case was increased in persons who spent longer time in the gym – a mean of 5 hours versus 3 hours respectively ($p < 0.01$), and in those who sat in the bleachers (RR = 1.65; 95% CI = 1.31–2.08). Persons wearing contact lenses or glasses were

at decreased risk (RR = 0.66; 95% CI = 0.49–0.89). In the gymnasium, tests for chemical agents were negative and a metal halide bulb with a broken outer casing was found. Video footage of the event confirmed that the bulb was in use during the competition.
Conclusions: UV radiation from a damaged metal halide bulb in a school gymnasium caused this outbreak of photokeratitis. Such UV exposures can be prevented through use of self-extinguishing metal halide bulbs and enclosed fixtures. These exposures can be reduced by targeted education about maintenance and safety for metal halide bulbs.
Keywords: Photokeratitis, Metal halide bulbs, Ultraviolet light

5. Poison center consultation and hospital length of stay

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Background: Poison Centers' (PCs) impact on inpatient care is not easily quantified. While hard endpoints are elusive, the ability to measure and quantify value is increasingly relevant as competition for healthcare dollars intensifies. Hospital length of stay (LOS) is an accepted metric that encompasses both cost and efficiency in healthcare facilities (HCFs). Reduced LOS may reflect "streamlined" care at lower cost and greater efficiency. But reduced LOS might also translate into reduced revenue opportunity for the treating HCF – not a popular concept in today's market.
Objective: Determine the impact of poison center consultation on hospital LOS and charges for poisoned or potentially poisoned hospital patients in one state.
Methods: All hospitals within the PC's designated region report billing and discharge coding data to the state Hospital Association (HA) database. Cases tabulated by the HA between 7/1/2008 and 6/30/2011 were directly matched against those within the PC case database. Included cases were hospitalized under an external cause of injury (E code) for poisoning. Exclusion criteria (psychiatric or rehab admissions, medical complications noted by certain ICD9 codes, adverse drug events) ensured that outliers were minimized and only cases hospitalized primarily for poisoning were included. "Matched" cases were found in both the PC and the HA databases during a specific hospitalization. "Unmatched" cases appeared only in the HA records (i.e. the PC was not consulted). Mean LOS and charges were compared between the groups. Payor status was also documented and tracked.
Results: Government payors covered 55% of hospitalizations, while 3rd party insurers covered 32% (Table 1) see above.
Conclusions: This is the first attempt to determine the impact of one state's PC on LOS and charges over a 3 year period while excluding potential confounders. PCs were 2–3 times more likely to be called if the case involved a child or adolescent. On average, cases including a PC consultation left the hospital nearly 1/2 day sooner. The greatest improvement in LOS (one day) was seen in the adolescent group. Charges were not significantly different between the two groups.
Keywords: Poison center, Length of stay, Charges

Table 1. Results for abstract 5.

	Matched (95% CI)	Unmatched (95% CI)	Δ; p value
Mean LOS (days) for all cases	2.42 (2.33,2.5) n = 4,227	2.87 (2.81,2.93) n = 14,026	0.45; < 0.005
Mean LOS (days) for age <12 yrs	1.41 (1.20,1.63) n = 257	1.74 (1.33,2.14) n = 268	0.33; 0.19
Mean LOS (days) for age 12–18 yrs	1.72 (1.58,1.86) n = 734	2.69 (2.53,2.85) n = 1,127	0.97; < 0.005
Mean LOS (days) for age >18 yrs	2.65 (2.55,2.76) n = 3,236	2.91 (2.85,2.97) n = 12,631	0.26; < 0.005
Mean Charges ($) for all cases	12,335 (11,872,12,799) n = 4,227	12,930 (12,667,13,194) n = 14,026	595; 0.57

6. Incidents of public health significance identified by National Surveillance of Poison Center Data

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The Centers for Disease Control and Prevention and the American Association of Poison Control Centers conduct public health surveillance on data collected by the National Poison Data System (NPDS) to identify incidents of public health significance (PHS). Automated surveillance algorithms run continuously in NPDS to identify instances when the number of hourly calls to a PC (call volume incidents or CVIs), or the daily, cumulative count of any reported sign, symptom, or laboratory abnormality (clinical effect incidents or CEIs) exceed a historical baseline. Incidents are reviewed daily by clinical toxicologists and epidemiologists to determine if an incident is of PHS and if further public health action is needed. The objective of this report is to characterize incidents of PHS identified by national surveillance from 1/2007 to 8/2011.
Methods: All incidents of PHS identified from 1/1/2007 to 8/1/2011 were reviewed. PHS was left to the individual judgment of the surveillance team member. Outcome measures for CVIs included mechanism of chemical exposure, setting, and substance type: mechanism of chemical exposure, clinical syndrome, specific illness and substance reported was reported for CEIs.
Results: A total of 830 CVIs were identified and the majority were of PHS (n = 721; 86.9%). Airborne exposures (e.g., gases and fumes) were the most reported mechanism of chemical exposure for each year, and in total (n = 312; 42.2%). The most common exposure setting for all years when combined was occupational (n = 128, 27.9%), followed by school (n = 126, 27.5%). The most frequently reported chemical exposure every year was carbon monoxide (n = 115, 15.5%), except in 2007 (when an outbreak of Salmonella contaminated peanut butter occurred). A cumulative total of 383 CEIs were identified and the majority were of PHS (n = 339; 88.5%). Airborne exposures were the most common mechanism of exposure for all years combined (70, 21.8%), followed by product contamination/tampering (68, 19.7%). The most common syndrome reported was gastrointestinal (n = 124, 20.9%); diarrhea was the most frequently reported illness (n = 42, 7.1%). Contaminated peanut butter (2007 and 2009 outbreaks) was the most frequently reported substance overall (n = 61, 18%).

Conclusions: Carbon monoxide releases were the most frequently reported incidents of PHS and gastrointestinal syndromes and symptoms were the most commonly reported illness manifestations. Surveillance of NPDS data can be used to characterize incidents of PHS and possibly identify risk factors for future interventions. These surveillance strategies can be replicated at the regional level which would be of interest to state health departments.
Keywords: Public health, Surveillance, Epidemiology

7. Massive acetaminophen overdose treated with immediate hemodialysis: Is the antidote removed faster than the toxin?

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Background: The role of hemodialysis for acetaminophen (APAP) overdose is debated. While APAP (MW 151 daltons) is readily dialyzable, its antidote acetylcysteine (MW 163) is as well. We occasionally recommend hemodialysis when it can be started immediately for the rare patient with features of so-called "mitochondrial paralysis" (lactic acidosis, altered mental status, depressed catabolism) following massive APAP overdose. We prospectively measured serum acetylcysteine concentrations during hemodialysis in such cases.
Case series: Three patients (ages 20 to 65 years) each presented unconscious after ingesting more than 60 grams of APAP. One coingested prednisone, another ibuprofen/chlorpheniramine/pseudoephedrine, and two were hypothermic (lowest 31.5°C rectal). Serum lactate ranged from 7 to 12.5 mM (pyroglutamic acid not measured). Each was intubated, and initial APAP concentrations were as high as 5980 μM (900 μg/mL). Intravenous acetylcysteine was initiated between 10.8 and ~18 hours post ingestion, and hemodialysis ~4 hours later. While dosing regimens varied, all patients were empirically administered additional acetylcysteine to compensate for possible antidote removal during hemodialysis. In each case, a single course of hemodialysis (duration 3.2 to 4.2 hours; blood flow ~400 mL/min) reduced serum APAP concentrations by 56 to 84% (mean elimination half-life 110 minutes), and also resulted in marked improvement with rapid resolution of the lactic acidosis. Serum acetylcysteine concentrations ranged from 220 to 1980 μM during hemodialysis, and the extraction ratio of acetylcysteine across the dialysis circuit ranged from 73% to 87%. All three patients recovered fully, with only one developing ALT >1000 IU/L, and none developing coagulopathy or other signs of liver failure.
Case discussion: As expected, both APAP and acetylcysteine are effectively and rapidly cleared during hemodialysis. Based on the kinetics observed in this case series, we estimate that hemodialysis more than doubles the overall clearance of acetylcysteine. Doubling the rate of the 4-hour component of the Prescott protocol to 25 mg/kg/hr, or administering intermittent doses of 70 mg/kg every 2 hours instead of every 4 hours, should help compensate for this removal.
Conclusions: When massive APAP ingestion is accompanied by coma and lactic acidosis, immediate hemodialysis can result in rapid biochemical and clinical improvement. Recognizing that acetylcysteine dosing is largely empirical, we recommend that the dosing of acetylcysteine be at least doubled whenever patients being treated with acetylcysteine undergo hemodialysis.
Keywords: Acetaminophen (paracetamol), N-acetylcysteine, Hemodialysis

8. Effectiveness of low dose naloxone to reverse respiratory depression in opioid intoxication

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Background: Although naloxone reverses the respiratory and CNS effects of opioids, care must be utilized to avoid precipitating acute opioid withdrawal syndrome (OWS). The commonly recommended initial naloxone dose of 0.4 mg is based on studies in a non-opioid-dependent population undergoing anesthetic reversal. However, in opioid-dependent patients, this dose predictably results in acute withdrawal. Despite limited data demonstrating the efficacy of a lower dose of naloxone, 0.04 mg is often recommended in medical toxicology and anesthesia texts as safe and effective. The objective of this study was to evaluate the clinical effects of naloxone 0.04 mg (with titration) on the respiratory depression noted in ED patients with acute opioid overdose.
Methods: A prospective observational study of the current ED practice of naloxone use in opioid intoxicated patients was conducted. Thirty consecutive ED patients, 18 years and older, with clinical findings of opioid intoxication (e.g., history, CNS depression, pinpoint pupils, RR <8/minute) requiring naloxone were included. The primary outcome was improved RR. Secondary

Table 1. Results for abstract 8.

	Drug history	Clinical findings	Naloxone (mg)	Clinical response
Patient A	Methadone	RR: 6/min RASS: −4 O_2sat: 87% CO_2: 50 mmHg	IV: 0.04 × 2	RR: 14/min RASS: 2 O_2sat: 95% CO_2: 42 mmHg
Patient B	Methadone Benzodiazepine	RR: 7/min RASS: −4 O_2sat: 95% CO_2: NR	IV: 0.04 × 3 Infusion: 0.04 mg/hr	RR: 9/min RASS: −1 O2 sat: 99% CO_2: NR
Patient C	Methadone	RR: 6/min RASS: −5 O_2sat: 80% CO_2: NR	IV: 0.04 Infusion: 0.05 mg/hr	RR: 12/min RASS: 0 O_2sat: 92% CO_2: 29 mmHg
Patient D	Methadone	RR: NR RASS: −5 O_2sat: 40% CO_2: 60 mmHg	IV: 0.04 × 2 Infusion: 0.1 mg/hr	RR: 13/min RASS: 2 O_2sat: 99% CO_2: NR OWS: vomitting
Patient E	Methadone	RR: 3/min RASS: −4 O_2sat: 92% CO_2: 55 mmHg	IV: 0.04 × 2 Infusion: 0.05 mg/hr	RR: 6/min RASS: −2 O_2sat: 94% CO_2: 50 mmHg

NR: not reported

outcomes were end-tidal CO2 level (CO2), pulse oximetry (O2 sat) on room air, Richmond Agitation Sedation Scale (RASS), and OWS.
Results: Preliminary results involving five ED patients are summarized in the Table 1. All five patients experienced coma with hypoventilation due to methadone use. On average, patients required two naloxone doses of 0.04 mg. One patient (patient D) experienced acute OWS with a total naloxone dose of 0.08 mg.
Conclusions: A naloxone dose of 0.04 mg with appropriate dose titration can effectively reverse both respiratory and CNS depression. A total dose of 0.08 mg may be required, but titration would limit the risk of acute OWS. Initial results indicate the commonly recommended initial dose of 0.4 mg may be excessive.
Keywords: Naloxone, Opioid, Overdose

9. A 10 Year Retrospective Review of Digoxin Immune Fab Use in Digoxin Exposures

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Background: Digoxin (DIG) is a cardiac glycoside indicated for the treatment of both systolic heart failure and atrial fibrillation. Although the use of DIG has been declining in the past 10 years, reported DIG overdose cases reported to the Statewide Poison System have remained stable. An analysis of the appropriate use of digoxin immune FAB (DIF) was initiated.
Methods: A 10-year retrospective poison center data study was completed on all cases of DIG exposures for a period of January 1, 2001 to December 31, 2011. IRB approval was obtained and cases were blinded prior to analysis. Inclusion criteria were DIG as a single ingestant and cases followed to a known outcome. The parameters used in the case analysis were DIG as a single substance, age, sex, year of reported cases, serum DIG level, serum potassium, serum creatinine, FDA indications and dose for DIF, and patient outcome.
Results: A total of 798 cases of DIG ingestion without coingestants were identified. Of the 798 exposures, 543 (68%) were female, and 258 (32%) were male with a mean age of 72 years old (range 4 days–99 yo, SD 20 yrs). The mean DIG level reported was 4.3 ng/mL (range 0.1–37 ng/mL, SD 3.0 ng/mL). The mean serum potassium reported was 5.1 mEq/L (range 2.3–8.7 mEq/L, SD 1.3 mEq/L) while the mean serum creatinine reported was 2.4 mg/dL (range 0.4–15.2 mg/dL, SD 1.8 mg/dL). Of 798 exposures, the use of DIF was reported in 292 cases (37%). Of these 292 cases, 259 cases (89%) met the FDA indication for DIF use while 33 (11%) did not. The remaining 506 exposures (63%) did not use DIF. Of the 506 cases, 293 (58%) met the indication for DIF use while 213 (42%) did not. Of the 293 cases of patients who did not receive DIF but met the FDA indication for use, 284 (97%) survived while 9 (3%) expired. Of the 213 (42%) cases that did not receive DIF and did not meet the FDA indication for use, 212 (99.5%) survived while 1 (0.5%) died. The odds ratio calculated for the cases that did not use DIF with clinical survival was 0.149 with a P value of 0.05 and CI of 0.007–1.154 and the number needed to treat (NNT), was 38. 59 (20%) patients that used DIF were under dosed with 55 (93%) survived and 4 (7%) expired. In 94 (32%) of the patients who were dosed correctly, 88 (94%) survived and 6 (6%) expired (P value 0.92).
Conclusions: Healthcare practitioners should consult with their regional Poison Control Center and closely follow the recommended FDA treatment guidelines regarding the use of digoxin immune FAB.
Keywords: Antidote, Cardiac glycoside, Adverse drug event

10. Successful management of olanzapine-induced anticholinergic agitation and delirium with a continuous intravenous infusion of physostigmine in a pediatric patient

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Background: Physostigmine effectively reverses anticholinergic delirium. However, continuous IV infusion of physostigmine is rarely used due to concern for cardiotoxicity. We report the successful use of continuous IV physostigmine in a 6-year-old male with anticholinergic delirium.
Case report: A 6 y/o 30 kg male with ADHD ingested 15 to 20 5-mg olanzapine tablets. He was agitated and was treated with lorazepam at a local hospital. His heart rate was 148, respiratory rate 32, blood pressure 111/70, temperature 96.8°F, and O_2 saturation of 98% on room air. His pupils were 5–6 mm and his skin was warm but not flushed. He had hypoactive bowel sounds. Blood chemistries were normal. A 12-lead ECG showed sinus tachycardia with normal QRS and QT intervals.

The agitation worsened and did not respond to benzodiazepines. The patient was then given 2 doses of physostigmine 0.6 mg with reversal of the agitation; but the effect only lasted 45 minutes. The patient's mother was disappointed that the physostigmine did not last longer and asked for continuous physostigmine infusion.

The patient was transferred to the PICU and 6 mg of physostigmine was placed in 250 cc of D_5W and administered at 0.5 milligrams/hour. Overnight the patient became more agitated. But instead of titrating the physostigmine drip, the PICU team discontinued it and started IV dexmedetomidine 0.2 micrograms/kilogram/hour at 2100. The patient became over-sedated with pinpoint pupils resulting in discontinuation of the dexmedetomidine at 0400. The patient again became agitated and developed visual hallucinations. Three 1 mg boluses of physostigmine were administered over 45 minutes and the physostigmine infusion was restarted at 1 milligram/hour for 16.5 hours. He received a total of 19.5 milligrams of physostigmine with no return of agitation or delirium. He was discharged home without further sequelae.
Discussion: There are limited data describing physostigmine to reverse anticholinergic delirium. There are no studies involving its use in children. Pentel described asystole complicating physostigmine use in TCA overdoses. Hence, physostigmine has been rarely used. Stern was the first to report a case of a 20-year-old female with anticholinergic delirium successfully reversed with 77 mg of physostigmine in a continuous infusion. Our patient received a total dose of 19.5 mg with complete resolution of symptoms.
Conclusion: To our knowledge, this is the first case report using continuous infusion of physostigmine to reverse anticholinergic delirium in a pediatric patient.
Keywords: Anticholinergic, Delirium, Physostigmine

11. Massive TCA ingestion treated successfully with a prolonged infusion of 20% lipid emulsion

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Background: Intravenous lipid emulsion (ILE) is increasingly used for severe intoxications of lipophilic substances. However, data is limited on the optimal duration of treatment with ILE. We present a case of prolonged use of ILE for a tricyclic antidepressant (TCA) overdose.
Case report: A 42 year-old man was admitted hypothermic and hypotensive with QRS widening to 200 ms. He was also comatose, seizing, and suffered recurrent cardiac arrest, ultimately developing vasopressor-dependent shock with persistent QRS widening despite repeated administration of IV sodium bicarbonate and active rewarming. An initial [TCA] returned at 7400 mmol/L. 20% ILE was administered as a 250 mL bolus followed by a 24 h infusion at 100 mL/h. Despite prompt normalization of the QRS interval and improvement in blood pressure, the QRS widened following discontinuation of the infusion. ILE was resumed at 100 mL/h with QRS normalization once again. Due to persistent toxicity and markedly elevated TCA levels (8858 mmol/L day 2), ILE was continued at 18 mL/h, for 19 days, until levels were no longer toxic. Whole bowel irrigation with polyethylene glycol was also performed due to concern for persistent GI absorption after the levels increased on the second day. The patient survived to discharge. Apart from a lipemic serum, there were no apparent complications from the prolonged ILE infusion.
Case discussion: We suspect that ongoing drug absorption due to prolonged ileus and delayed elimination due to acute renal failure contributed to the persistent toxicity and elevated TCA levels.
Conclusions: Prolonged ILE infusions may be useful and effective in massive TCA ingestions with delayed absorption and/or clearance of the drug. In our patient, a prolonged ILE infusion (19 days) appeared to be safe and effective when used along with standard therapy for TCA toxicity.
Keywords: Lipid therapy, Tricyclic antidepressants, Intoxication

12. Benefits of symptom-triggered phenobarbital therapy in refractory delirium tremens: A case report

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Background: Delirium tremens (DTs) can manifest days after the cessation of chronic alcohol abuse. Symptoms such as change in cognition, disturbance of consciousness, and autonomic instability are usually treated with benzodiazepines, however other modalities may be required. We present a case of a patient with benzodiazepine refractory DTs who responded to symptomtriggered phenobarbital therapy.
Case report: A 30 year-old male with a history of polysubstance abuse arrived to the Emergency Department (ED). The extremely agitated patient reported using alcohol one day prior. The patient is known to use alcohol chronically. The patient was sedated in the ED with lorazepam 4 mg IVP every 15 minutes as needed. Four hours later, he was transferred to the ICU. Within hours, DTs quickly developed. Symptoms resolved with lorazepam 4 mg IVP every 15 minutes (cumulative dose of 26 mg) and he was placed on a CIWA protocol. On hospital day 2, DTs returned and intensified. A total of 46 mg of lorazepam was given over 4 hours with minimal improvement; a lorazepam drip was then initiated. Despite all efforts to treat the patient with high-dose lorazepam (>40 mg in 2 hours), the DTs persisted. A phenobarbital protocol was initiated. A total of 1170 mg of phenobarbital was given on hospital day 2. The patient remained sedated with no DT's. On hospital day 3, a total of 1300 mg of phenobarbital was administered for agitation. At this time, due to a back order on phenobarbital, supplies were exhausting and could not be replenished. A CIWA-A protocol was used to supplement the remaining phenobarbital.

When phenobarbital supplies exhausted, the patient was intubated and started on a propofol infusion. With minimal symptom relief, approximately 38 mg of midazolam was given over 18 hours in addition with propofol, to control withdrawal symptoms. The remainder of the hospital course was complicated by ventilator-associated pneumonia and ICU delirium.
Case discussion: This patient responded well to symptom-triggered phenobarbital therapy. It was not until after phenobarbital supplies were exhausted that mechanical ventilation with propofol was necessary. The patient's DTs were uncontrolled with the change in therapy. Due to the misfortune of exhausted hospital supplies, we were forced to intubate, begging the question; would have mechanical ventilation been necessary if phenobarbital was continued?
Conclusion: Phenobarbital delayed the need for mechanical ventilation and reduced benzodiazepine use. In this case, phenobarbital was the drug of choice for refractory DTs.
Keywords: Withdrawal, Alcohol, Delirium

13. Dexmedetomidine as an adjunct in patients undergoing treatment for ethanol withdrawal in the critical care setting

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Background: The ideal management of ethanol withdrawal, a potential medical emergency, still eludes the practitioner. The stigmata of ethanol withdrawal have not been fully comprehended and involve the interplay of numerous neurotransmitters including γ-aminobutyric acid (GABA), glutamic acid, and norepinephrine. The benzodiazepines have been the mainstay in treating patients withdrawing from ethanol because of their effect on GABA and glutamate receptors. The adjunctive role of dexmedetomidine, a presynaptic α_2-agonist, has yet to be elucidated in managing the hyper-adrenergic state of patients undergoing ethanol withdrawal.
Methods: All adult patients in a suburban hospital critical care unit who exhibited the stigmata of ethanol withdrawal and who received continuous intravenous infusions of dexmedetomidine as part of their pharmacologic management were studied. Medical records were retrospectively examined from July 2009 until November 2011 for the following parameters: demographic

information, outcome, complications, and the Richmond Agitation and Sedation scores (RAS).
Results: Seventeen patients fulfilled the criteria and were studied. There were 16 males (age range 34–79 years, mean 55.4) and 1 female. Ethanol withdrawal was the primary diagnosis in 6 patients. Dexmedetomidine was infused for an average of 67.2 hours (range 1.5–167 hours). One patient received 5 separate infusions, 2 patients received 2 infusions. Fourteen patients had RAS scores before and after dexmedetomidine therapy; all scores had improved with therapy. Transient asymptomatic bradycardia occurred in 2 patients; 1 patient developed bradycardia and 5 beats of ventricular tachycardia necessitating cessation of dexmedetomidine. One patient developed supraventricular tachycardia requiring 3 doses of adenosine. Intubation was avoided in 5 patients; dexmetedtomidine facilitated extubation in 1 patient.
Conclusions: Dexmedetomidine appears to be a worthwhile adjunct in the treatment of patients with ethanol withdrawal in the critical care setting. Airway management was facilitated in 6 patients (35%). Four of 17 patients developed either changes in pulse rate or rhythm while receiving dexmedetomidine. Patients treated with dexmedetomidine for ethanol withdrawal should receive cardiac monitoring.
Keywords: Ethanol, Withdrawal, Dexmedetomidine

14. Physostigmine for reversal of iatrogenic atropine overdose from a dental procedure

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Background: Atropine is described in the dental literature for use as an anti-sialogogue, with a recommended dose range of 0.4–0.8 mg in adults. Typically this is administered as 1–2 drops of ophthalmic atropine, which has a concentration of 10 mg/mL. This is the first reported case of iatrogenic overdose of oral administration of ophthalmic atropine treated with physostigmine. Overdose of opthalmic atropine is sparsely reported in the literature and in the only reported case of dental administration resulting in toxicity went unrecognized, and resulted in a fatality.
Case: A 57-year-old male presented to the ED with acute delirium after he was seen by his dentist at 7 a.m. and fitted for a dental prosthesis. A coworker brought him to the ED four hours later with dysarthria, and concern for possible stroke. On arrival, his heart rate was 128 BPM, blood pressure 148/94 mmHg, and temperature 37.7 C. His physical exam was notable for marked mydriasis, dry mucus membranes, tachycardia, dysarthria, and a distended bladder. The dentist was contacted, and reported that he had administered almost 2 mL of ophthalmic atropine (10 mg/mL) sublingually to assist in drying the gingiva for moulding of the prosthesis. We gave physostigmine 2.5 mg IV with resolution of his anticholinergic syndrome. We observed him in the ED, and at 6 hours after atropine administration, his anticholinergic symptoms recurred. He was given a second dose of physostigmine 2.5 mg, again with resolution of his symptoms. Due to the efficacy of treatment with physostigmine, he did not require an extensive work-up for his acute delirium. He was observed for 24 hours, with no further recurrences.
Case discussion: Atropine is a tropane alkaloid extracted from several plants of the Solanaceae family, and was used centuries ago to induce mydriasis for cosmetic effect. Currently, atropine is used for therapeutic mydriasis, as well as for its cardiovascular effects. The ophthalmic preparation has been adopted for use by dentists and oral surgeons to minimize salivation during procedures. Atropine toxicity is characterized by anticholinergic crisis with delirium, tachycardia, dry skin, and mydriasis. Physostigmine is a carbamate acetylcholinersterase inhibitor effective at reversal of atropine induced anticholinergic syndrome. Repeat dosing is safe and effective for recurrent symptoms.
Conclusions: Ophthalmic atropine drops are highly concentrated, and can result in symptomatic overdose with relatively small volume administration. Anticholinergic delirium should be considered in cases of acute altered mental status after dental procedures. This case highlights the therapeutic and diagnostic utility of physostigmine.
Keywords: Iatrogenic, Atropine, Overdose

15. Successful treatment of severe diethylene glycol poisoning with fomepizole and hemodiaylsis

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Background: Several instances of mass poisoning by diethylene glycol (DEG) due to medication contamination have been described. However, there are few case reports of intentional ingestion and even fewer reports of successful treatment.
Case report: We report a case of a 43 year old woman who ingested most of a 32-ounce can of "Prestone Super Heavy Duty High Temperature Break Fluid" in a suicide attempt. The patient was found unresponsive and with agonal respirations. She had three episodes of PEA arrest en route to our hospital. After initial resuscitation she arrived at the emergency department unresponsive, hemodynamically unstable, and severely acidotic. Her initial heart rate was 130 and systolic blood pressure was 65. Pupils were fixed and dilated. Initial laboratory investigation was remarkable for pH of 6.95, HCO_3 of 15, BE of −18, potassium of 2.7, and osmolal gap of 30. Aggressive treatment was undertaken with 3.5 liters of normal saline, 100 mEq of sodium bicarbonate, and 1200 mg of fomepizole. Emergent hemodialysis was initiated and after 6 hours of dialysis the acidosis had nearly resolved, hemodynamic status was improved, and the osmolal gap had decreased to 12. Ultimately, resuscitation and treatment were successful and the patient was discharged neurologically intact 15 days after arrival.
Case discussion: Prestone Super Heavy Duty Break Fluid is a complex mixture of glycol ethers. Notably, it contains up to 8% diethylene glycol. DEG is known to be extremely toxic, but most reports in the literature describe accidental contamination. Cases of large-volume intentional ingestion are rare. It has been proposed that such ingestions be treated similar to other toxic alcohols, mainly utilizing fomepizole and hemodialysis. This is largely based on animal models, and to our knowledge there is only one report of such treatment in humans. Our case illustrates successful treatment of a life-threatening DEG ingestion using these treatment modalities.
Conclusions: DEG is an extremely toxic substance and ingestion can be life-threatening. Fomepizole and hemodialysis, along with aggressive supportive care, may be an effective treatment.
Keywords: Diethylene glycol, Fomepizole, Ingestion

16. Treatment of severe toxic alcohol and glycol poisoning in the UK

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Background: This prospective study was undertaken to investigate the management of systemic exposures to toxic alcohols and glycols reported to the UK National Poisons Information Service (NPIS).
Methods: Details of exposures to products containing toxic alcohols and glycols reported to NPIS were collected from 1 January 2010 to 31st December 2010 and cases of significant systemic exposure were followed up to obtain information on antidote use and patient outcome.
Results: Of the 488 individual exposures to toxic alcohols and glycols resulting in 608 enquiries to NPIS over the 12 month period, 182 met the criteria for case follow up. Antidote treatment was provided to 99 patients. 36 patients received fomepizole alone, 49 ethanol alone, and 14 both ethanol and fomepizole. Extracorporeal elimination by intermittent haemodialysis and/or continuous haemodiafiltration was instituted in 33 patients - alone without antidotal treatment in 3, in conjunction with antidotes in 29 patients. Antidotal and/or extracorporeal treatment was instituted in 36 of the 39 patients where the PSS was recorded as severe. Outcomes were generally favourable regardless of treatment modality. However there were 4 cases resulting in patient death (2 were treated with ethanol and 2 did not receive an antidote). 5 patients developed renal failure (4 were treated with ethanol and 1 with fomepizole).

Adverse reactions to antidotes were reported in 10 patients: 8 receiving ethanol and 2 receiving fomepizole. Adverse reactions reported as probably related to ethanol use were intoxication (4), reduced conscious level (1), agitation (1) and withdrawal symptoms (1). Adverse reactions were possibly related to fomepizole in 2 patients (shaking in 1 patient which recovered with supportive treatment and angio-oedema which recovered following fomepizole discontinuation in 1 patient). Problems reported with use of ethanol during the study included difficulty in making the infusion, calculating and adjusting infusion rate, difficulty in obtaining ethanol concentrations to monitor treatment, sub-therapeutic ethanol concentrations, lack of availability or inadequate stocks to complete full treatment. Problems reported with use of fomepizole included lack of availability or inadequate stocks and lack of familiarity with its reconstitution and dosing schedule.
Conclusions: Antidote and/or extracorporeal treatment for severe toxic alcohol or glycol poisoning is required infrequently in the UK with around 100 reported cases per year. Ethanol and fomepizole are used in similar numbers of patients without any differences in outcome. Ethanol use may be associated with more frequent adverse reactions and practical difficulties with its use.
Keywords: Ethylene glycol, Antidote, Hemodialysis

17. Successful treatment of massive ethylene glycol poisoning with fomepizole only

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Background: Treatment of ethylene glycol (EG) poisoning consist of administration of an alcohol dehydrogenase inhibitor such as fomepizole or ethanol and the possible addition of hemodialysis (HD) to enhance elimination of the parent compound and metabolites. Several cases have been published where patients exhibiting high blood levels of EG who presented prior to the development of metabolic acidosis and nephrotoxicity were treated with fomepizole alone. We present a case that is the highest EG level reported to date in a patient who was successfully treated with fomepizole without HD. Case report: A 43 y/o, 75 kg female presented to the ED 1 hour after ingestion of a 1/2 gallon of a 50:50 mixture of EG antifreeze and water along with 20 fluoxetine tablets and alcohol. Her physical exam was unremarkable other than appearing slightly drowsy with slurred speech. Initial vitals signs were BP 137/88 mmHg, HR 92 beats/minute, RR 12 breaths/minute, PaO_2 96% on room air. Her labs were: Na 136 mEq/L, K 3.7 mEq/L, Cl 99 mEq/L, CO_2 22 mEq/L, BUN 2 mg/dL, Cr 0.7 mg/dL, glucose 110 mg/dl, osmolality 411 mOsm/kg, and ethanol 71 mg/dl. Her anion gap was 15 with an osmolar gap of 117 mOsm/kg. A loading dose of 15 mg/kg fomepizole was initiated followed by 4 doses of 10 mg/kg q 12 hours then a repeat dose of 15 mg/kg. The patient's ethylene glycol level on arrival was 944 mg/dl. Subsequent levels were 405 mg/dl, 94 mg/dl and non-detectable at 5.5, 30, and 64 hours post ingestion respectively. The patient's acid/base status and renal function remained normal throughout her hospital stay. The patient was admitted to the intensive care unit for the first 24 hours then transferred to a general medical floor. Case discussion: Fomepizole effectively blocks the metabolism of EG to its toxic metabolites. Although treatment with the alcohol dehydrogenase inhibitor, fomepizole prolongs the elimination half-life of EG, complete elimination within three to five days is possible in the setting of normal renal function. It would be impractical to treat asymptomatic patients with very high methanol levels with fomepizole alone since the elimination half-life would extend to 40–60 hours thus requiring a protracted length of stay in the hospital.
Conclusions: We present a case of EG poisoning with a peak blood level of 944 mg/dl treated with fomepizole alone. This case, together with other published reports suggest that patients with massive EG poisoning who arrive early and demonstrate no evidence of a wide anion gap metabolic acidosis or presence of toxic metabolites maybe safely treated with fomepizole as sole therapy without HD.
Keywords: Ethylene glycol, Fomepizole, Antifreeze

18. Predictors of death and renal failure in ethylene glycol poisoning

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Background: Despite the severity of ethylene glycol poisoning, little is known about what factors influence survival and prolonged renal insufficiency. Our study aims to describe clinical signs, times of door-to-antidote (D2A) administration, and times of door-to-dialysis (D2D) associated with the outcomes of death and prolonged renal insufficiency.

Methods: This was a retrospective, observational California Poison Control System (CPCS) study. Over a ten-year period (1999–2008), the CPCS database included 517 consultations for ethylene glycol exposure. Of these, we studied 121 patients who had all of the following: recorded serum level of ethylene glycol, arterial pH measurement, serum creatinine measurement, and times of patient arrival, and antidote (ethanol and or fomepizole) and dialysis initiation. We defined as "cases" 59 patients who died (9) or had prolonged renal insufficiency (50). Prolonged renal insufficiency was defined as kidney injury in which dialysis was required for greater than 3 days after hospital presentation. The remaining 62 patients were "controls." In addition to descriptive statistics, to evaluate the association of D2A and D2D with dying or prolonged renal insufficiency, we estimated odds ratios (ORs) and 95% confidence intervals (CIs) using logistic regression models.

Results: Cases, compared to controls, were more likely to present comatose (57.6% compared to 12.9%), have seizures (40.7% compared to none), and require vasopressors (13.6% compared to 4.8%).

Cases presented with a higher mean ethylene glycol level of 240.4 mg/dL (SD 307), compared to 171.8 mg/dL (SD 168.3) for controls.

Cases presented with a lower mean initial arterial pH of 7.03 (SD 0.20), compared to 7.27 (SD 0.14) for controls. Cases had a higher peak creatinine within the first 24 hours of hospitalization (2.4 mg/dL, SD 1.0), compared to controls (peak Cr 1.1 mg/dL, SD 0.5).

Compared to patients with a D2A within 3 hours, patients with a D2A greater than 3 hours had a higher odds of dying or having prolonged renal insufficiency (OR = 3.34, 95% CI = 1.21–9.26).

Compared to patients with a D2D within 6 hours, patients with a D2D greater than 6 hours had a lower odds of dying or having prolonged renal insufficiency (OR = 0.36, 95% CI = 0.15–0.87). However, no patient who received dialysis within 3 hours of arrival died.

Conclusions: Compared to survivors without any prolonged toxic sequelae, patients poisoned with ethylene glycol who died or had prolonged renal insufficiency were more likely to exhibit severe clinical signs such as coma, seizures, hypotension, and acidosis. Earlier antidote administration appears to lead to better outcomes. Benefit from earlier dialysis initiation could not be demonstrated.

Keywords: Ethylene glycol, Fomepizole, Hemodialysis

19. Clinical and economic impacts of hemodialysis in the treatment of phenobarbital overdose

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Background: In most instances, patients with phenobarbital (PB) overdose (OD) can be managed with supportive care, activated charcoal and urine alkalinisation. Given the extended half-life of PB, patients may be unconscious for several days even with aggressive symptomatic care. Hemodialysis (HD) may be useful in such cases. We attempt to determine the clinical and economic impacts of HD in severe PB OD.

Case report: A 29 year old female was found unresponsive. She was known to take quetiapine and aripiprazole; no pills were missing. She was intubated on arrival. Causes of coma were eliminated with time, standard laboratory tests, CT scan and lumbar puncture. Patient remained comatose during the next 4 days. On day 4, a serum toxicological screen requested by our Poison Centre revealed PB at 378 umol/L. HD was recommended until return to therapeutic PB level or awakening. Dialysis duration was of 4 h and the patient regained consciousness 2.5 hours after it began. She was extubated 9 h after HD ended and she recovered with no deficits.

Results: Multiple serum samples were analyzed for PB prior to dialysis (5), during (4) and after dialysis (4). Apparent half-lives were 71 h pre HD, 7 h during HD and 26 h post HD. No clearance data was available at the time of writing.

Discussion: HD is generally recommended in cases where [PB] serum is ≥ 500 umol/L, and for patients who are hemodynamically unstable. According to local data, the cost of 24 h in ICU is 9300$ excluding expenses related to complications such as venous thromboembolism or ventilator associated pneumonia which increase with intubation duration (Grap, 2012). One episode of HD at our institution is 1332$. Complications of HD are minimal and mostly related to catheter insertion (Randriamanantsoa, 2011). Considering the length of PB-associated coma following OD, it seems that HD is a cost-effective, risk-minimizing solution for clinical and economical perspectives. In our case it decreased apparent half-life by 10-fold while shortening intubation and ICU stay. It could have been even more effective if started shortly after admission.

Conclusions: Our case shows that HD significantly decreases the plasma half-life of PB. One episode of HD costs a fraction of an ICU stay while minimizing risk of complications due to prolonged coma. Measuring plasma PB, early in coma of unclear origin would help clinicians weigh the risks and benefits of HD early after admission. Evaluating the efficiency of other treatment modalities such as multiple doses activated charcoal against HD is warranted.

Keywords: Hemodialysis, Coma, Intoxication

20. Usefulness of late haemodialysis in paraquat poisoning

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Case presentation: A 53-year-old man accidentally drank Gramoxone containing 37% paraquat (PQ) 90 minutes before the call to Poison Control; he had already vomited 12 times. Paramedics were on site but not interested in any recommendation. According to family, the Gramoxone was an old product kept for professional use. Product was diluted by son and poured into a drinking glass. The patient mistook the glass as his own and "accidentally drank and spat out a mouthful". He was admitted to a community hospital. Poison Control recommended activated charcoal, IV fluids, and antiemetics for persistent vomiting. Haemodialysis (HD) was not initially recommended given the time elapsed since ingestion. At 18 hours (hrs.) post-ingestion (PI) the Poison Centre toxicologist recommended N-Acetylcysteine (N-AC) administration, and transfer to tertiary care hospital for ICU admission. Patient arrived at the tertiary care facility 23 hrs. PI. He began to deteriorate at about 45 hrs. PI. Treatment included IV steroids for progressive upper airway oedema, and haemodialysis for renal failure. At 52 hrs. patient was hypoxic and chest X-ray revealed pulmonary infiltrates. A cyclophosphamide IV

protocol was started in addition to N-AC and antioxidants. At 69 hrs. patient was intubated in the OR. At 89 hrs. sepsis was suspected from aspiration pneumonitis. HD and Continuous Venovenous Haemodiafiltration (CVVHDF) were administered in alternating cycles. At 115 hrs. severe hypoxia and refractory hypotension developed. Comfort care measures were implemented. Death was pronounced 117 hrs. PI. PQ levels in plasma, urine and dialysate were measured before, during and after extracorporeal treatment (ECTR). The apparent half-life was 25 hrs. Estimated PQ body burden on admission was 204 mg. PQ removal by HD was 13 mg in 6 hrs. (HD#1) and 18 mg in 15 hrs. (HD#2). CVVHDF removed 4.2 mg in 26.4 hrs, whereas urine excretion was 2.5 mg in 96 hrs. Calculated clearances were 236 ml/min (HD#1), 250 ml/min (HD#2), 43.8 ml/min (CVVHDF#1), 26.7 ml/min (CVVHDF#2) and 5.4 ml/min (urine).
Discussion: While PQ use and intoxications are more common in developing countries, it is still used by the landscaping industry in North America. HD has been advocated only in the first few hours after ingestion. However, analysis of our toxicokinetic data seems to indicate that significant amounts of PQ are removed by ECTR even at a late stage, and that HD is more effective than CRRT. Given that PQ ingestions are potentially lethal, that alternative treatments have no definite benefit, and that HD has been associated with survival, we suggest that HD for poison removal should be considered for all PQ ingestions, as it is unknown at which point in time it becomes futile.
Keywords: Clearance, Hemodialysis, Paraquat

21. Hydroxocobalamin hinders hemodialysis

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Background: Hydroxocobalamin, an antidote used for cyanide exposure, has been FDA approved since 2006. It functions by binding cyanide, forming cyanocobalamin, which is then excreted in the urine. Its side effect profile is limited, however it can alter accuracy of laboratory measurements and impact hemodialysis. We present a patient whose hemodialysis treatment was delayed due to a false "blood leak" alarm following hydroxocobalamin administration.
Case report: A 22 year-old male was running from police, and upon being caught he suffered a respiratory arrest. He was transported by ambulance with respirations assisted by bag valve mask. On arrival to the emergency department (ED) he had palpable pulses and Glasgow Coma Scale (GCS) score of 3. ED providers promptly intubated the patient and initiated other resuscitative measures. Vital signs included heart rate 70 beats/minute, blood pressure 76/50 mmHg, and O_2 saturation 100%. Blood drawn from both peripheral IVs and a central line was bright red; venous blood gas revealed pH $<$ 6.8, pCO_2 47.5 mmHg, pO_2 245 mmHg, bicarbonate 6.4 mmol/L, and O_2 saturation 97%. Lactate concentration was 16.4 mmol/L. With the constellation of low GCS, hypotension, bright red venous blood with high O_2 content, and lactic acidosis, cyanide poisoning was considered and 5 gm hydroxocobalamin was given. Hemodialysis was initiated based on persistent acidosis, hyperkalemia, and renal insufficiency. The dialysis machine experienced an auto-shutdown due to a "blood leak" alarm. This alarm persisted and finally had to be manually overridden, at which point dialysis commenced. The patient ultimately deteriorated and expired 24 hours after initial presentation.
Discussion: A known effect of hydroxocobalamin administration is the discoloration of body fluids. During dialysis it also discolors the dialysate, creating a problem because of an internal safety measure in the dialysis machine. Normally blood passes on one side of a semi-permeable membrane and dialysate on the other. Photosensors monitoring the dialysate are meant to alarm and shut down the machine if red blood cells leak across the membrane. Hydroxocobalamin changes the light refraction in the dialysate enough to set off the alarm and cause the auto-stop. This "blood leak" phenomenon of hydroxocobalamin has been reported previously in a case report; in that case it appears as though manual override was not done in time to perform dialysis.
Conclusions: Hydroxocobalamin administration can induce a false "blood leak" alarm preventing hemodialysis until manually overriding the alarm. Providers caring for patients treated with hydroxocobalamin should be aware of this as inability to perform dialysis may make for poor outcomes.
Keywords: Cyanide, Hydroxocobalamin, Dialysis

22. Physostigmine continuous infusion for the treatment anticholinergic toxicity in combined diphenhydramine and bupropion overdose

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Background: Once a popular and widely used antidote for the treatment of anticholinergic toxicity, the publication of case reports reporting an association of physostigmine use with asystole in severe tricyclic antidepressant poisoning resulted in a paradigm shift and dramatic decrease in physostigmine use beginning in the early 1980s. Recent literature reviews and cohort studies have demonstrated that physostigmine, either administered alone or with benzodiazepines, is a safe and effective antidote for the treatment of anticholinergic toxicity. Despite this, there is minimal information in the literature regarding the use of physostigmine administered as a continuous intravenous infusion for persistent anticholinergic toxicity. We present a case of continuous physostigmine infusion, used with adjunctive GABAergic medications, for the treatment of hyperthermia and agitation in a combined bupropion and diphenhydramine overdose.
Case report: A 13 year-old female presented with severe agitation and hyperthermia after ingesting a large amount of diphenhydramine and bupropion. Despite incremental and increasing doses of lorazepam, the patient remained febrile (up to 39 C via foley catheter). In conjunction with additional doses of benzodiazepines, 2 mg of physostigmine was administered over 10 minutes. Coinciding nearly with the end of the infusion, along with the formation of sweat, there was marked improvement in the patient's temperature and a decrease in agitation. Two additional doses of physostigmine 2 mg were administered for recurrence of symptoms. Ultimately, a continuous infusion of physostigmine at 2–4 mg/hour was required for a period of 6.5 hours to maintain control of her agitation and attenuation of hyperthermia. The physostigmine was discontinued when the patient was able to maintain her ability to sweat and her agitation remained under control, albeit with high-doses of adjunctive benzodiazepines. The patient did not experience vomiting, seizures, bradycardia or conduction block.

Case discussion: A combination of sympathomimetic and anticholinergic poisonings may be particularly problematic due to the impairment of evaporative heat loss in the setting of an agitated delirium. Severe anticholinergic symptoms may not respond well to benzodiazepines.
Conclusions: Physostigmine administration allowed for evaporative heat loss to occur in our patient. Due to the severe and persistent toxicity, along with the relatively short half-life of physostigmine, a continuous infusion was required and used safely.
Keywords: Physostigmine, Anticholinergic, Infusion.

23. Lipid rescue for tricyclic antidepressant toxicity: A case report

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Background: Intravenous fat emulsion (IFE) therapy is accepted therapy for local anesthetic toxicity. Its use has expanded to other lipophilic drug poisonings, including calcium channel blockers, beta adrenergic antagonists, antidepressants and neuroleptics. We report the case of an intentional amitriptyline overdose with cardiovascular collapse, which was reversed following the administration of intravenous fat emulsion.
Case report: A 54 year old man was found unresponsive following a 2.25 gram amitriptyline ingestion of fifteen 150 mg tablets. Upon admission, He became hypotensive with a wide complex tachycardia at 106 bpm, and having a QRS of 166 ms, and a QTc of 566 ms. Resuscitation consisted of 4 L of IV normal saline, Na bicarbonate, norepinephrine and phenylephrine infusions. The patient remained hypotensive with prolonged QRS and QTc following resusitation. A 100 ml bolus of 20% IFE was given followed by an infusion of 0.25 ml/Kg/min over 60 min. Within 9 minutes of the IFE bolus, the QRS narrowed to 114 msec and vasopressor support was weaned over the next several hours. The patient was discharged neurologically intact on hospital day 11.
Case discussion: Our case describes severe cardiac instability following a massive overdose of the lipophilic, antidepressant amitriptyline. The patient had significantly improved cardiac function and hemodynamics following initiation of IFE therapy. The wide complex tachycardia, previously refractory to sodium bicarbonate, narrowed within 9 minutes of the IFE infusion. Most previous reports of tricyclic ingestions with hypotension and QRS widening were refractory to vasopressors and sodium bicarbonate, have shown improvement in blood pressure with IFE therapy. Many cases do not report significant narrowing of the QRS complex. Our patient had refractory hypotension with prolongation of the QRS complex that was refractory to sodium bicarbonate but narrowed after the IFE was administered. The patient also had improved blood pressure with reduction in his vasopressor requirements following IFE.
Conclusions: IFE may be a valuable adjunct to treating patients with acute ingestions of lipophilic drugs, such as tricyclic antidepressants, refractory to traditional therapy. Further investigation is necessary to determine if these benefits of IFE therapy can demonstrate a consistent response in other acute lipophilic drug poisonings.
Keywords: Lipid therapy, Overdose, Antidepressant

24. Intravenous fat emulsion (IFE) for refractory hypotension due to venlafaxine overdose

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Background: Venlafaxine is a serotonin-norepinehprine reuptake inhibitor, with a relatively well-described overdose syndrome, including seizures and serotonin-like syndrome. Cardiac dysfunction, including tachyarrhythmias, Takot-subo morphology, and refractory hypotension are also described. We report the first case of IFE for treatment of refractory hypotension due to intentional venlafaxine overdose.
Case report: A 43-year-old woman presented to an outside hospital with altered mental status after ingestion of alcohol, venlafaxine and zolpidem. She was intubated for declining mental status. On arrival to a teritiary ED, she had a heart rate of 108 BPM, BP of 82/45 mmHg, temperature of 96 F, and was sedated with lorazepam. Serum ethanol was 0.05 mg/dL. She was given 1 L of intravenous normal saline. Her blood pressure dropped to 51/27 mmHg. Naloxone 2 mg was given. A dopamine drip of 1 mcg/kg/min was ordered but not available before 150 mL of IFE 20% was administered. Within 5 minutes her BP improved to 120/47 mmHg, and she never required pressors. A urine drug screen that utilizes liquid chromatography and mass spectrometry was positive only for zolpidem and venlafaxine. Her BP remained normal throughout the rest of her hospitalization, and she was extubated the next day and transferred to psychiatry.
Case discussion: Venlafaxine toxicity has been well reported in the literature. A syndrome of altered mental status, seizures, and hypertension is typical initially, followed by hypotension in severe cases. Treatment with single-dose activated charcoal, whole bowel irrigation, and pressors have been used with variable success in treating hypotension due to venlafaxine overdose. The experimental logP hydrophobicity of venlafaxine is 2.8, which is similar to other compounds that are known to be successfully treated with IFE in overdose. This contriuted to our decision to give IFE, as well as the few contraindications to IFE. To our knowledge, this is the first reported case of IFE for hypotension due to venlafaxine overdose.
Conclusions: IFE may be useful for treatment of hypotension due to venlafaxine overdose. This case also supports the theoretical efficacy of IFE in patients with toxicity due to a compound with similar hydrophobicity.
Keywords: Venlafaxine, Intravenous Fat Emulsion, Overdose

25. Novel use of intralipid in the treatment of severe baclofen toxicity

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Backround: We present a case of severe baclofen toxicity, with hemodynamic instability, and decreased level of consciousness that was treated successfully with intralipid.
Case report: A middle-aged female was brought by paramedics to a rural hospital, after an overdose of an unknown amount of baclofen tablets. The patient was initially stable, however her GCS declined en route to hospital. Upon arrival to the ED, the patient had a GCS of

8, BP: 101/60 P 65 R 16 O_2 saturation of 99% on 3 L nasal prongs. Initial laboratory studies were unremarkable, in particular there was no increased anion gap, and no salicylates or acetaminophen were detected. An ECG demonstrated a sinus rhythm with first degree AV block, but normal QRS and QTc intervals. Over the next hour, the patient deteriorated, with a declining GCS, and BP of 81/39. A fluid bolus was initiated with no improvement. The patient was intubated for airway protection, and the next BP was 59/32. Intralipid was administered as a 20% solution at 1.5 cc/kg as a bolus followed by 0.25 cc/kg/min infusion for 30 minutes. The BP rose to 89/57 within 30 minutes of the bolus being given. The blood pressure continued to normalize with no pressor support. The heart rate ranged between 44 to 58 bpm. Within 2 hours of intralipid administration, the patient's hemodynamic status stabilized. She was subsequently extubated the following day and discharged.

Discussions/conclusion: This case demonstrates the first reported use of lipid emulsion therapy in the treatment of hemodynamic instability due to a baclofen overdose. Baclofen is a GABA agonist, and is often associated with neuromuscular dysfunction such as hypotonicity, decreased LOC, and coma like states. Baclofen has been shown to cause hypotension[1], but it is rare[1,2]. Lipid emulsion therapy provided rapid resolution of the cardiovascular collapse, associated with the baclofen ingestion in this patient.

Keywords: Lipid therapy, Overdose, Baclofen

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26. Immediate chelation with DMSA for acute arsenic overdose prevents chronic toxicity from arsenic poisoning

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A 52 yo male with severe mental illness overdosed on trazodone, DEET, and an old arsenic-containing pesticide found in his garage. The ED initially was not concerned about the pesticide, since the product was >40 yrs old, and therefore was felt to be non-toxic.

Patient presented with severe watery vomiting and diarrhea, but no blood was seen. VS were stable with only slight tachycardia, which responded to fluid replacement. EKG showed no abnormalities, no QTc prolongation. Vomiting had resolved once Poison Center was called for treatment advice.

Medical Toxicology consult through the Poison Center recommended obtaining a stat whole blood As and urine arsenic, then to start immediate chelation with DMSA due to signs and symptoms consistent with acute inorganic As poisoning. The patient was immediately started on DMSA 10 mg/kg q 8 hr (30 mg/kg/d) for 5 days, then 10 mg/kg q 12 hr (20 mg/kg/d) pending lab test results.

The initial stat whole blood Arsenic was 186 µg/L (nl = 2–23), and spot urine Arsenic was 1,936 mcg/L (nl <50 mcg/L), all inorganic arsenic. Continued DMSA chelation was recommended until urine and whole blood arsenic were normal.

The following lab results were obtained while chelation was ongoing:

Repeat whole blood arsenic on day 5 was 54 µg/dL (nl = 2–23)
24 hr Urine arsenic from day 9 was 131 µg/dL (nl <50 mcg/l)
24 hr Urine arsenic on day 14 was 112 µg/L (nl <50 mcg/l)
24 hr Urine arsenic on day 29 was normal, <50 µg/L, and no inorganic arsenic was detected via speciation

DMSA therapy was stopped after results came back normal, with the patient asymptomatic; he developed no signs or symptoms of chronic arsenic posioning, with no alopecia, and no peripheral neuropathy. He remained well after follow-up examination.

Keywords: Arsenic, Chelation, DMSA

27. Clinical outcomes in newer anticonvulsant overdose

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Objectives: Clinicians have limited experience with treatment of overdose from newer anticonvulsant medications. The aim of this study was to evaluate clinical effects of newer anticonvulsants in overdose, determine if a relationship exists between dose and clinical effect, and if there are drug(s) particularly more toxic in overdose.

Methods: This was a retrospective study using poison center data, evaluating clinical outcomes. The Toxicall™ database from 1/1/2002–12/31/2011 was queried using key words: gabapentin, lamotrigine, levetiracetam, tiagabine, topiramate, zonisamide, pregabalin, and oxcarbazine. Polypharmacy overdose and children less than 15 years of age were excluded. Charts were reviewed by two abstractors for pharmaceutical, reported dose, clinical outcome score, recorded clinical signs/ symptoms, and vital signs.

Results: Out of 501 cases identified, 347 met final inclusion criteria. There was one death reported from intentional overdose of topiramate. Proportion of overdoses exhibiting a clinical effect by pharmaceutical are listed in Table 1.

Data were modeled by cumulative logit ordinal regression on a 3-level ordinal scale response (0 no effect, 1 minor effect, ≥2 moderate to severe effect) using log-transformed standardized doses. There was no significant effect of dose on severity of outcome ($\beta = 0.12$, $p = 0.23$). However risk of a more severe outcome score was significantly increased with tiagabine relative to other drugs ($\beta = 2.8$, $p = 0.001$).

Table 1. Results for abstract 27.

	Seizure	AMS	Tachy/brady	Hypotension	GI
Gabapentin	2	41	11	16	6
Lamotrigine	5	45	16	9	36
Levetiracetam	0	60	0	7	7
Tiagabine	20	67	13	20	7
Topiramate	0	25	11	7	4
Pregabalin	0	35	22	4	13
Oxcarbazepine	4	53	31	5	24

Ranking of drug toxicity based on clinical signs and symptoms was performed by calculating an information index H based on the proportional occurrence (1/0) of clinical outcome signs and symptoms in nine categories of toxicity (seizures, AMS, pupillary response, neuromuscular, GI, dermatological, cardiac, hypotension, metabolic). Lamotrigine ranked highest in terms of toxicity ($H_T = 1.66$) and number of interventions performed ($H_I = 1.17$), and levetiracetam the lowest ($H_T = 0.98$; $H_I = 0.88$).
Conclusions: Overdose of newer anticonvulsants frequently results in altered mental status. Seizures appear to be more common with tiagabine, lamotrigine, and oxcarbazepine. We could not identify a dose-effect in these data which likely reflects the limitations of self-reported doses. Risk of more severe outcome score was higher with tiagabine overdose while lamotrigine overdose appears to result in more reported signs, symptoms, and interventions.
Keywords: Anticonvulsant, Overdose, Poison center

28. Recommendation and use of high-dose insulin and intralipid following beta and calcium channel blocker toxicity

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Background: Calcium channel (CCB) and beta-receptor (BB) blockers may cause significant morbidity and mortality in overdose. Hyperinsulinemic euglycemia (HIE) and intravenous fat emulsion (IFE) may be beneficial in treating toxicity from CCBs and BBs. Many poison control centers (PCC) now recommend these modalities. However, health-care providers and may not institute them despite PCC recommendations. We sought to determine how often HIE and IFE are recommended by a statewide PCC in CCB and BB toxicity, how often those recommendations are implemented, and if a faxable information sheet is associated with improved adherence.
Methods: We performed a retrospective review of a statewide poison system's database for cases of CCB and BB exposures from January 2005–July 2011. Gender, age, drug type, co-ingestants, interventions, and outcomes were recorded. HIE or IFE recommended by PCC, whether therapy was implemented before or after recommendations, and whether a written protocol was faxed to the treating physician was also abstracted. Exclusion criteria included an incomplete PCC record.
Results: There were 215 CCB and/or BB exposures identified during the study period that met criteria for evaluation. Patients were predominantly female (64%) with an average age of 54 years. Death occurred in 25 (12%) cases. The PCC recommended HIE in 71 cases and it was started in 1 case prior to PCC discussion. HIE was subsequently used in 30 of these cases after PCC recommendation. IFE was recommended by the PCC in 30 cases. IFE was implemented 10 times. In no case, was IFE started prior to discussion with PCC. In 6 cases both HIE and IFE were implemented, all after recommendation. A protocol for HIE was faxed to the treating provider in 7 cases while a protocol for both HIE and IFE was faxed in 13 cases. HIE (8 cases) or IFE (5 cases) was implemented in 13 cases following faxed protocol. There was no statistical difference between HIE or IFE being implemented when a faxable protocol was available vs just when PCC advised by phone. See Table 1.
Conclusions: HIE and IFE is increasingly recommended by PCC for the treatment of CCB and BB toxicity. An increased use by treating providers follows a similar temporal trend. The presence of a faxable protocol was not associated with significant increased adherence to PCC recommendations regarding the use of HIE or IFE.

Additional efforts to improve clinician education regarding HIE and IFE as well as implementing improved faxable protocols may be required to increase the utilization of these potentially life-saving antidotes.
Keywords: Beta blocker, Calcium channel blocker, Insulin

Table 1. Results for abstract 28.

	2006	2007	2008	2009	2010
HIE recommended	8	14	5	17	27
HIE given	2	3	4	5	16
IFE recommended	–	–	1	10	19
IFE given	–	–	0	2	8

29. Critical care management of verapamil and diltiazem overdoses at a single center

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Background: Overdose (OD) of the calcium channel blockers (CCB) verapamil and diltiazem is associated with severe illness. Traditional management has used vasopressors (VASO), although recently hyperinsulinemic euglycemia (HIE) has become popular. We describe a large series of patients (pts) with CCB OD.
Methods: Retrospective review of all adult pts (age >14) admitted to one toxicology service over 22 yrs, for treatment of non-dihydropyridine CCB OD. Pts were identified via review of pt encounter logs. Data were abstracted using standard methodology; analyses were mostly descriptive, although medians (interquartile range (IQR)) were determined as appropriate.
Results: 59 verapamil or diltiazem ODs were identified. The mean (SD) age was 50 (+/−16) yrs; 48% were men. 53% were verapamil ODs. Median (IQR) initial and max glucose were 150 (123–189) and 219 (159–330) mg/dL, respectively. Median (IQR) initial and nadir K+ were 3.7 (3.3–4.1) and 3.2 (2.9–3.5) mEq/L, respectively. 41% presented with acute kidney injury (creatinine >1.5 mg/dL). VASO were used in 61% pts; mean number used 2.1. VASO use was as follows (n pts/total; mean max dose +/−SD; max dose): norepinephrine (24/59; 26+/−26 mcg/min; 100 mcg/min;), dopamine (22/59; 20+/−20 mcg/kg/min; 100 mcg/kg/min), epinephrine (16/59; 47+/−85 mcg/min; 325 mcg/min;), isoproterenol (12/59; 16+/−18 mcg/min; 60 mcg/min;), phenylephrine (5/59; 282+/−296 mcg/min; 800 mcg/min); 9/56 received dobutamine. HIE (1 U/kg bolus, then 1 U/kg/hr) was used in 3 pts who were already on VASO. An intra-aortic balloon pump was placed in 1 pt. 5 ischemic complications occurred in 4/59 pts: 3 pts had GI bleeds (GIB), 2 of which were apparent prior to starting VASO. 1 pt who was slow to awaken had a brain MRI which revealed mild ischemia, without clear evidence of infarction. Another pt had ischemic bowel, which was suspected on admit (lactate 9.3 mmol/L

with abdominal pain). Of note, HIE was used in 1 pt with a GIB present on admit, as well as in the pt with possible stroke who had GIB during therapy. 2 pts had in-hospital cardiac arrest and were resuscitated. 1 pt died due to complications from pulmonary artery catheter placement. All pts with ischemic complications were discharged to in-patient psychiatry, fully recovered. 4 pts were sent to short-term rehab, including 1 with in-hospital cardiac arrest, due to deconditioning, but all were neurologically intact.

Conclusions: In this case series of 59 non-dihydropyridine CCB ODs, hypotension was common, and often managed with multiple high-dose VASO, without HIE. Ischemia was uncommon, and death occurred in only 1 pt due to a procedural complication. VASO use following CCB OD was associated with good outcome in this series

Keywords: Calcium channel blocker, Insulin, Vasopressor

30. Effect of acetaminophen overdose on prothrombin time

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Background: APAP toxicity can result in transaminitis, coagulopathy, liver failure and death. This study examined the test characteristics of a high initial PT and AST for hepatic injury, death or liver transplant.

Methods: We conducted a chart review of patients ≥14 years old with known APAP overdose, treated with NAC between 1 Jan 2006 and 31 Dec 2010 at a tertiary care referral center. Inclusion criteria were APAP overdose established by history of APAP ingestion plus either an APAP level above the treatment line on the Rumack-Matthew nomogram or elevated transaminases in cases of suspected delayed presentation. Exclusion criteria were concurrent use of warfarin, history of hepatitis with previously documented transaminitis,or CK >1000 IU/L. Standardized data abstraction methods were utilized. We defined: high PT as a PT value > the upper limit of reference normal; high AST = >50 IU/L; liver injury = AST >1000 IU/L. Initial PT was performed at various labs so the specific reference ranges for normal were used.

Results: 304 pts were included, mean age 31 (range 14–88), with 67% women. The mean (SD) and median (IQR) initial AST was 1208 (+/−3186) and 37 (22–217) IU/L, while the mean (SD) and median (IQR) max AST was 2426 +/−4429) and 48.5 (25–2991) IU/L respectively. The mean (SD) and median (IQR) initial PT was 17.4 (+/−13.2) and 13.8 (11.8–16.4) sec. while the mean (SD) and median (IQR) max PT were 21.7 (+/−17.7) and 15.4 (13.8–21) sec. respectively. 13 pts died or had a liver transplant (D/OLT). Test characteristics are in Table 1. 8 pts had initially normal AST but high PT and developed hepatic injury. 8 others had initially normal PT but high AST and developed hepatic injury.

Conclusions: In this cohort of patients treated for APAP toxicity, an elevated initial PT and AST each had similar test characteristics for predicting the outcome of d/OLT. A high initial PT has little clinical relevance, but a normal initial PT is associated with a very high NPV for predicting d/OLT. Neither a normal initial AST nor an initial normal PT can definitively exclude the risk of developing hepatic injury.

Important limitations are small numbers of pts with the endpoint of d/OLT, and all pts were treated with NAC. Further study is needed to determine if an initial normal PT can be used in the decision to treat.

Keywords: Acetaminophen (paracetamol), Prothrombin time, Coagulopathy

31. Occurrence of thrombocytopenia following acetaminophen toxicity

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Background: Thrombocytopenia has been described following APAP ingestions. Theories proposed, include antibody formation to APAP metabolites and altered concentrations of thrombopoietin. Neither the prevalence of thrombocytopenia, nor the significance has been investigated. This study examines the prevalence of thrombocytopenia in a consecutive case series of adults admitted with confirmed APAP overdose.

Methods: A retrospective analysis of data from a prospectively-derived registry of adults (age >14 yrs) treated for confirmed APAP ingestion over a 1-yr span was performed. Primary outcome was to determine the occurrence of thrombocytopenia. Secondary outcome included correlation of thrombocytopenia and AST >1000. Standardized data abstraction methods were used. Median and interquartile range (IQR) were calculated for categorical variables. Fisher's exact test was used to assess the correlation between thrombocytopenia (PLT <100,000/mm^3) and hepatic injury (AST >1000 IU/L).

Results: 92 consecutive pts were identified and included in the analysis. The median (IQR) age was 28.5 (19–52) yrs. The majority were women (65%) with acute ingestions (85%). Platelet (PLT) counts 21 hrs post-admission were available in 91.3% of cases. The median (IQR) initial and nadir PLT counts were 285,000 (203,000–312,000)/mm^3 and 198,000 (139,000–239,000)/mm^3. With nadir PLT count <100,000/mm^3 in 12/84 (14.3%) and <50,000/mm^3 in 9/84 (10.7%). The mean (+/−SD)

Table 1. Test characteristics for development of d/OLT.

	Sensitivity	Specificity	PPV	NPV
Death/OLT				
Initial elevated PT	100 (75.3–100)	47.2 (41.2–53.2)	8 (4.3–13.3)	100 (97.3–100)
Initial elevated AST	100 (75.3–100)	61.7 (55.9–67.3)	10.4 (5.7–17.3)	100 (98–100)
Liver injury				
Initial elevated PT	91.7 (84.2–96.3)	62.8 (55.7–55.9)	54.3 (46.3–62.2)	94 (88.5–97.4)
Initial elevated AST	91.7 (84.2–96.3)	82.6 (76.7–87.5)	71 (62.1–78.8)	95.5 (91.4–98.1)

and median (IQR) initial AST values were 1195 (+/−3334) IU/L and 38 (23–175) IU/L respectively. The mean (+/−SD) and median (IQR) max AST values were 1822 (+/−3945) IU/L and 42 (25–1391) IU/L. The initial AST was >1000 IU/L in 19/92 (20.7%), while the max AST was >1000 IU/L in 26/92 (28.3%). Among the 26 patients with a maximal AST >1000 IU/L, 16/26 (61.5%) had nadir platelet counts <100,000/mm^3 vs. 10/26 (38.4%) with a nadir platelet count >100,000/mm^3 ($p < 0.001$). When excluding those patients whose presenting AST was >1000 IU/L, 2/58 (3.4%) of patients whose maximal AST was <1000 IU/L had PLT <100,000/mm^3 vs. 2/7 (28.5%) of patients whose maximal AST was >1000 IU/L developed PLT <100,000/mm^3 ($p = 0.054$). 1/75 (1.3%) of pts with a PLT nadir >50,000/mm^3 died, while 3/9 (33%) of pts with a nadir PLT <50,000/mm^3 died; $p = 0.003$.

Conclusions: Thrombocytopenia following APAP overdose in this study population was relatively common with 14.3% developing PLT< 100,000/ mm^3 and 10.7% with PLT <50,000/mm^3. Decrease in PLT below either 100,000 or 50,000/mm^3 was strongly associated with the likelihood of peak AST> 1000 IU/L. Etiology and prognostic utility of thrombocytopenia could not be evaluated by the current study future investigation is needed.

Keywords: Acetaminophen (paracetamol), Thrombocytopenia, Platelets

32. Pharmacokinetics of N-acetylcysteine (NAC) during CVVH, HD and in the absence of renal replacement therapy

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Background: APAP induced fulminant hepatic failure (FHF) is associated with kidney injury, acidosis and severe fluid and electrolyte imbalances. As a result, varying renal replacement therapies (RRT) are increasingly used in severe APAP poisonings. Although there is a potential for NAC extraction with RRTs, data are lacking and it is unclear whether NAC dosing should be altered during RRT. We report extensive data from the first patient in a multicenter pharmacokinetic study of the extracorporeal removal of NAC by RRTs in patients with APAP induced FHF.

Methods: A 29 year old, 74 kg woman presented with right upper quadrant pain and vomiting after unintentional chronic supratherapeutic dosing of APAP and oxycodone-APAP. Initial lab values: [APAP] 31 mcg/mL, AST 3972 U/L, ALT 10270 U/L, total bilirubin 2.7 mg/dL, INR 3.1, creatinine 3.2 mg/dL. She was started on a standard IV NAC regimen and over a 48 h period we studied the pharmacokinetics of NAC during a maintenance infusion of 6.25 mg/kg/hr IV. Kinetics were determined while she was on CVVH, in the absence of RRT, and lastly during HD (with minor ultrafiltration). At nine points in time (T1–T9) simultaneous urine, blood, dialysate and/or ultrafiltrate specimens were collected for [NAC], and measured via HPLC. During RRTs blood was collected from both access and return, to obtain average [NAC] (Cp). Standard pharmacokinetic calculations were applied.

Results: NAC concentrations in the absence of RRT (T3–6) were consistent with published literature on standard NAC kinetics and averaged 31.6 mg/L. CVVH effect was minimal, with extraction ratios of 0.03 and 0.09 (T1 and T2 respectively). This produced fractional clearances of 5% and 3%, and removed 24.2 mg and 15.1 mg hourly. However, HD clearance was substantial, with extraction ratios of 0.30 (T8) and 0.34 (T9). This produced fractional clearance 61% (T8) and 85% (T9) and removed in the dialysate (plus ultrafiltrate) 279 mg and 395 mg of the 462.5 mg hourly NAC dose, (T8 and T9). Relatively low HD clearance at T7 was likely due to sampling prior to equilibration. By extrapolation, 24 hours of CVVH and 4 hours of HD would have removed 470 mg and 1348 mg of NAC, respectively.

Conclusions: These results suggest that CVVH clearance of NAC is minimal and unlikely to require altered dosing. In contrast, HD extracted a significant amount of NAC. If more than 50% of the hourly NAC dose is removed in dialysate during HD, a substantial dose adjustment may be indicated. These data are limited based on the number of samples and the use of a single patient whose clinical condition was changing over time. Ongoing patient enrollment and data collection will enable us to better define NAC dose adjustments during RRTs.

Keywords: N-acetylcysteine, Pharmacokinetics, Acetaminophen (paracetamol)

33. Refractory hypoglycemia in an acetaminophen, acetyl-salicylic acid, and glyburide overdose

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Background: Refractory hypoglycemia is a well-described consequence of overdoses involving acetaminophen (APAP), aspirin (ASA), and sulfonylureas. We describe a 17 day course of recurrent hypoglycemia after an overdose of APAP, ASA, and glyburide (GBU).

Case: A 57 year-old man was found unresponsive by EMS. Initial capillary blood glucose was 52 mg/dL. Despite 25 g of dextrose by EMS, glucose upon arrival to the ED was 42 mg/dL. Vital signs were normal. GCS was 10T. The rest of his examination was normal. ABG: pH 7.36, PaCO2 30 mmHg, and bicarbonate 16 mEq/L. Serum APAP and salicylate measured 396 ug/mL and 61.4 mg/dL, respectively. The patient was initiated on 5% dextrose (D5W) with 150 mEq/L $NaHCO_3$ infusion, 21-hour N-acetylcysteine (NAC), and subcutaneous octreotide (OCT) (50 ug q 8 hr). INR peaked at 4.3 on hospital day (HD) 2 and AST peaked at 1098 IU/L on HD 4. $NaHCO_3$ infusion was discontinued on HD 4 and NAC on HD 5. Despite resolution of ASA toxicity and evidence of adequate hepatic synthetic function, the patient continued to have episodes of hypoglycemia requiring dextrose boluses. Therefore, his D5W infusion was increased to D20W on HD 6. Serum sulfonylurea panels on hospital days 1, 6, 12, 13, and 17 screened positive for GBU (>3 ng/mL). On HD 7 his insulin level was 26 uIU/L [normal high <25 uIU/L] and C-peptide was 10.6 ng/mL [0.8–4 ng/mL] during a documented episode of hypoglycemia. On HD 10, OCT was converted to an infusion at 25 mcg/hour. The treatment goal was a blood glucose of 80–200. The following titration parameters were utilized: 1) if glucose is at goal, increase OCT by 5mcg/hr and decrease D20W by 10 mL/hr. 2) If glucose is <80 increase OCT by 5 mcg/hr and increase D20W to previous hour's

rate and restart titration in 1 hour. 3) If glucose is >200, decrease OCT by 5 mcg/hr and decrease D20W by 10 mL/hr. Verapamil 80 mg PO TID was initiated on HD 13 and continued until HD 19. His last episode of hypoglycemia was on HD 17. He was discharged to home on HD 21.
Discussion: The etiology of refractory hypoglycemia in this case is multifactorial. Contributing are acute liver injury, impaired metabolism of GBU, depletion of glycogen stores, and catabolism of acute illness. We also postulate that verapamil aids in reducing GBU-related secretion of insulin by antagonizing pancreatic L-type calcium channels.
Conclusions: We describe the longest duration of refractory hypoglycemia due to sulfonylurea poisoning and the first reported uses of an octreotide infusion and oral verapamil in sulfonylurea poisoning. We propose that higher doses of octreotide may be required for severe cases of sulfonylurea poisoning. Adjunctive therapy with verapamil may also be considered.
Keywords: Hypoglycemic, Sulfonylurea, Aspirin

34. The ascending hegemony of IV N-Acetylcysteine for acetaminophen poisoning: Why?

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Background: After the introduction of Acetadote®, in 2004, US use of IV N-acetylcysteine (NAC) dramatically increased, and has surpassed that of the oral preparation as reflected in NPDS data. We find this surprising.
Methods: Reasons cited for the preferential use of IV NAC include purportedly shorter therapy (along with lower cost), convenience and less emesis. We examined, from 1985–2010, NPDS (US PCC) yearly totals of patients receiving IV v. PO NAC. We informally surveyed local hospitals re: relative acquisition cost of Acetadote versus generic oral NAC. We searched the medical literature to compare relative incidence (IV v PO) of serious ADRs and deaths, and to assess relative efficacy. Finally, we examined if there really is a difference in average duration of treatment.
Results: The US NPDS logged, from 1985–2003, 158,332 patients who received NAC PO and 10,787 who received NAC IV, a ratio of 15:1. By2010, IV NAC cases (16,961) now out-numbered PO NAC cases (8,362) by a ratio of 2:1. Local hospitals report in 2012 an average acquisition cost per gram of $30.21 for Acetadote, and $1.40 per gram for generic NAC 20% oral solution. While vomiting is more frequent with PO versus IV NAC, serious ADRs are more common with IV administration. Multiple deaths have been reported since 2004 caused by Acetadote dosing errors, which appear more consequentially grave than PO dosing errors. Cumberland, Acetadote's manufacturer, has reported no data indicating that Acetadote is safer than the formerly administered generic oral NAC formulation through a Millipore filter. Both PO and IV NAC are of established efficacy. While it has been suggested that IV NAC may be more efficacious when initiating treatment within 12 hours of ingestion, and that PO 72 hour protocol more effective when initiating after 18 hours, these differences are not firmly established and, if they exist, are probably small. It is conceptually untrue that all IV NAC patients need only 21 hours of therapy, or that PO NAC patients need 72 hours of therapy. Current literature advocates that duration of NAC therapy be patient-tailored, with endpoints of both no remaining acetaminophen and improving liver health. The California Poison Control System (CPCS) makes no durational distinction between PO v IV NAC, and stops either one when both endpoints are reached, as early as after only 20 hours of therapy.
Discussion/Conclusion: When examining relative acquisition cost, frequency of severe ADRs with IV NAC, comparable efficacy and comparable duration of treatment, we find no justification for the preponderant preferential use of IV NAC. We suggest routinely employing a 20 hour oral regimen, and reserving the use of IV NAC for patients unable to take the drug by mouth.
Keywords: Acetaminophen (paracetamol), Overdose, Antidote

35. Acetaminophen overdose in profound hypothermia

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Background: There are few reports of acetaminophen overdose in hypothermic patients and fewer in patients with profound hypothermia. The metabolism of acetaminophen, the risk of hepatotoxicity with delayed treatment, and the kinetics and possible dose adjustments of N-acetylcysteine therapy are not well-studied in this setting.
Case report: A 37-year old female was found unconscious outside during the winter. She was transported by EMS to a tertiary care facility where she was noted to be profoundly hypothermic, with a core rectal temperature of 17oC. Her initial serum acetaminophen concentration was 232 mcg/mL. Active rewarming was started immediately and in the absence of guidelines from the manufacturer regarding the use of IV N-acetylcysteine (N-AC) in profound hypothermia, it was initiated with the standard treatment protocol. The patient did not develop signs of hepatic or renal injury or of N-acetylcysteine toxicity. She later confirmed that she had taken an overdose of acetaminophen/diphenhydramine ("Tylenol PM") 18 hours prior to discovery.
Discussion: Despite the significant ingestion of acetaminophen and delayed presentation, the patient did not develop any signs or symptoms of liver or kidney injury. Acetaminophen's combination with diphenhydramine may have delayed systemic absorption initially and her profound hypothermia likely slowed heptatic and renal metabolism once acetaminophen concentrations had reached peak levels. Although there are no guidelines for its use in profound hypothermia, N-acetylcysteine was administered in a standard dose during her rewarming period. Despite a blood concentration of acetaminophen greater than 200 mcg/mL and a late presentation, she did not develop hepatotoxicity or nephrotoxicity and there were no observable adverse effects from IV NAC.
Conclusions: Profound hypothermia may be protective of hepatic and/or renal injury in acetaminophen overdose by slowing its metabolism. IV N-acetylcysteine was given in a standard dose to a profoundly hypothermic patient without apparent adverse effects and appeared to be protective of acetaminophen-induced hepatic and renal toxicity during rewarming.
Keywords: Acetaminophen (paracetamol), Environmental, N-acetylcysteine

36. Safety of non-therapeutic atomoxetine ingestions-A National Poison Data System study

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Background: The safety of medications used to treat attention deficit-hyperactivity disorder has been questioned resulting in US Food & Drug Administration "black box" warnings for increased suicidality and cardiovascular events. The purpose of this study is to describe epidemiology and clinical course of non-therapeutic atomoxetine exposures reported to the US National Poison Database System (NPDS).

Methods: Retrospective database study of non-therapeutic single agent ingestions of atomoxetine in children and adults reported to the NPDS between January 1, 2002 and December 31, 2010. Adverse drug reactions (ADRs) were excluded from the analysis. Severity and clinical effects were recorded by poison center providers using a standardized data collection system; effects noted only in case notes were not captured.

Results: 20,032 atomoxetine exposures were reported during the study period, and 12,500 of these were single agent, non-ADR exposures. The median age was 9 years (IQR 2, 14) and 7,454 were males (59.6%). 8,933 (71.5%) were acute exposures, 3,325 (26.6%) acute-on-chronic, 166 (1.3%) chronic, and the remaining 76 (0.6%) cases were unknown chronicity. In 10,732 (86.2%) cases, exposure was unintentional, in 1,082 (8.7%) were suicide attempts, and in 630 (5.1%) abuse. The majority of cases were managed on-site [n = 8,619 (69.0%)], and 3,667 (29.3%) were referred to/managed in health care facilities. Acute on chronic ingestion was associated with an increased risk of a suicidal reason for exposure in comparison to acute ingestions (OR 1.44, 95% CI 1.26, 1.65). The most common clinical effects were drowsiness/lethargy (709 cases; 5.7%), tachycardia (555; 4.4%), and nausea (388; 3.1%). Exact dose information was available for 2,749 cases (22.0%). The maximum ingested dose was 48,000 mg in an 18 year male in a suicide attempt resulting in a moderate outcome. Major toxicity was observed in 21 cases; seizures in 9 (42.9%), tachycardia in 8 (38.1%), coma in 6 (28.6%), and ventricular dysrhythmia in one case (4.8%). There were no deaths.

Conclusions: Non-therapeutic atomoxetine ingestions reported to US poison centers are largely safe and significant overdoses were tolerated with few clinical effects, however, seizures were rarely observed. Major cardiovascular events were exceedingly rare in our study.

Keywords: National Poison Data System, Overdose, Adolescent

37. Massive Ibuprofen ingestion with refractory hypotension, hyperlactemia, and CNS depression

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Ibuprofen, a propionic acid NSAID is one of the most commonly used non prescribed medications in the world. Although the pathophysiology of the common complications of NSAIDS such as GI and renal effects are clearly defined, the data on the cardiovascular risk and toxicity remain controversial. We report on the case of a 16 year old girl with refractory hypotension and hyperlactemia following Ibuprofen overdose.

Case report: A previously well 16 year old girl ingested 300 to 350 tablets of 200 mg (60–70 grams) Ibuprofen in a suicide attempt following an argument with her parents. She was found unconscious with two empty bottles of Ibuprofen. She vomited several times on the way to the hospital without hematemesis.

She arrived at the emergency department (ED) 6 hours post ingestion with GCS of 11. BP 68/27 HR 110, RR, 18, Temp 36.6 C. She denied abdominal pain or diarrhea and no fever, cough or shortness of breath. Initial venous blood gas was PH 7.27/PCO_2 46.8/PO_2 62/BE −5. She was bolused with 2 liters of fluid without improvement in her blood pressure and subsequently started on dopamine drip at 5 mcg/hr. She was then transferred to PICU on cardiac monitor.

Other laboratory results showed, Na 146 Meq/L, K 3.8 Meq/L, HCO_3 18 Meq/L, Chloride 102 Meq/L, Anion gap 26. Comprehensive Urine drug screen was only positive for Ibuprofen and her serum Ibuprofen level 7–8 hrs post ingestion was > 250 mcg/ml (normal 10–50 meq/L); serum lactate 2.7, TSH 1.44. Acetaminophen, salicylate, creatinine and ethanol levels as well as coagulation profiles were normal. Her EKG showed sinus rhythm with a QTc of 455. Chest X-ray was normal.

Her blood pressure improved initially to 107/50 and then dropped down to 82/35, necessitating increased dopamine dose to 10 mcg/hr to maintain MAP at > 60. She subsequently sustained a stable blood pressure and dopamine drip was turned off 18 hours from admission. She was then transferred to psychiatry for inpatient treatment two days post admission.

Discussion: While Ibuprofen overdose usually present with GI, renal and electrolyte disturbances, massive ingestion can present with refractory hypotension, QT prolongation, anion gap metabolic acidosis and neurological effects. Our patient's serum Ibuprofen level was reported as greater than 250 mcg/ml the highest level this particular laboratory could determine. The exact mechanism of the cardiovascular toxicity of Ibuprofen is not known but there is no evidence of direct cardiotoxic effect.

Conclusion: Massive Ibuprofen overdose can present with refractory hypotension and neurologic effects, but the exact mechanism of the cardiotoxic effect is not known.

Keywords: Ibuprofen, Refractory hypotension, Metabolic acidosis

38. Long term cohort of patients with Lamotrigine toxicity

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Background: Lamotrigine (LTG) is a phenyltriazine antiepileptic drug which inhibits voltage-gated sodium channels, decreasing presynaptic release of glutamate and aspartate. Additionally, a concentration dependent inhibition of serotonin uptake by lamotrigine has been reported in rats. Reports of toxicity from the literature are limited to case reports and primarily involve coingestants.

Case series: Fifty-three patients were identified between 2003 and 2012 with possible LTG toxicity at a single toxicology center. Forty-four patients (83%) had coingestant toxicity with one fatality. Nine patients had LTG as the only significant ingestant. Two of the 9 patients had seizures during transport or while in the emergency department. Six of the 9 patients demonstrated myoclonic-type movement disorders. At presentation, 5 of the 9 patients were hypertensive. Four patients were tachycardic with rates greater than 100 bpm. Four of the patients were tachypneic with rates greater than 24 per minute. Mental status was assessed using the Riker sedation scale, and scores ranged from 1–7 with an average of 3.6. Four of the 9 patients were hyperreflexic and 3 had inducible clonus. Six of the 9 patients had an electrocardiogram, and two of these patients had QRS greater than 100 and the average QRS duration was 101 ms. Five of the patients had QTc > 460 and the average QTc was 487. Therapeutic blood levels of LTG are less than 12 mg/L, and 6 of the 9 patients had LTG blood levels with confirmed toxicity (> 12.0 mg/L). Three patients did not have documented LTG blood levels. Initial LTG levels ranged from 17.1 to 90 mg/L (mean 33.8) with half-lives varying from 6.9 to 51.7 hours. The patients who experienced seizures had initial LTG levels of 26 mg/L and 90 mg/L (both had seizure disorders). The patients with myoclonic movement disorders had LTG levels from 17.8 to 90 mg/L.

Discussion: This is the largest cohort of LTG ingestions reported to date. The degree of LTG toxicity was variable, likely reflecting dose-dependency. Mental status ranged from coma to severe agitation, but usually was described as lethargic with intermittent myoclonic type restlessness. Patients generally were hypertensive, tachycardic, and tachypneic. Some patients with LTG toxicity have seizures, clonus and myoclonic movement disorders as well as QRS and QTc prolongation.

Conclusions:

1) LTG toxicity was associated with altered mental status
2) LTG toxicity was associated with increased vital signs and neuromuscular irritability
3) LTG toxicity was associated with prolonged QRS and QTc
4) These clinical presentations are consistent with serotonin toxicity and sodium channel blockade, both pharmacologic effects of LTG.

Keywords: Lamotrigine, Pharmacokinetics, Toxicity

39. Predicting mortality in severe aluminium phosphide poisoning

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Background: Aluminium Phosphide poisoning is an important cause of morbidity and mortality in India. In this study, tried to develop a model for predicting mortality in patients with severe aluminium phosphide poisoning.

Methods: A prospective study including all patients with aluminium phosphide poisoning was carried out in the medical emergency attached to Nehru Hospital at our centre from June 2010 till December 2011. At admission, heart rate, blood pressure, blood sugar, electrocardiogram, blood gases, liver functions, renal functions were recorded and all the patients was followed up till discharge or death. Primary outcome was mortality. Parametric variables like blood pressure, pulse rate, heart rate was analyzed by student's t test. For categorical data like mortality, relative risk of death was calculated with 95% confidence intervals, with chi square test. Further correlation was carried out by univariate, multivariate and multiple logistic regression analysis.

Results: A total of 105 patients were enrolled during the study period. majority patients were males (62%) in the age group 15–35 years (78%). The mean dose consumed was 3.6 grams. 51% patients died in the study group. Important parameters, after multivariate analysis, correlating with mortality were Blood pH < 7.2, Systolic blood pressure < 90, blood sugar < 50 or > 200 mg/dl and Glasgow coma scale < 4. On multivariate regression, none of these parameters were independently associated with mortality. The odds ratio was 12.614 for pH < 7.2, 17.600 for SBP < 90 mmHg, 13.23 for Blood sugar < 50 or > 200 and 18.621 for GCS < 4. A score of 2 each was assigned to SBP < 90 and pH < 7.2 and score of 1 was assigned to Blood sugar < 50 or > 200 and GCS < 4. The maximum score was 6. When applied to the study group it was noticed that 96.4% with score of 0 and 69% with score 2 survived, while 70% with score of 4 and 100% with a score of 6 died.

Conclusions: pH < 7.2, Systolic blood pressure < 90, blood sugar < 50 or > 200 mg/dl and Glasgow coma scale < 4 are important predictors of poor outcome in patients with severe aluminium phosphide poisoning and a scoring system based on these parameters may be a useful adjunct in assessing severity of poisoning.

Keywords: Pesticide, Shock, Medical toxicology

40. Salicylate induced proximal tubular dysfunction

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Background: Fanconi Syndrome (FS) is characterized by multiple proximal tubular transport defects resulting in aminoaciduria and glycosuria despite euglycemia, hypouricemia and phosphaturia. The absence of one or more manifestations defines partial FS. We report a case of a 14-year-old female with salicylate toxicity who present with persistent glycosuria despite euglycemia.

Case report: A 14-year-old female ingested 9.75 g aspirin in a suicide gesture. She presented 14 hours post-ingestion with tinnitus, nausea, abdominal pain and hyperpnea.

Initial vital signs were: BP 121/65 mmHg; temperature 37.1^{0c}; HR 105 beats/minute; respirations 24 breaths/minute; weight: 53.1 kg; height: 161 cm. She had fresh lacerations on her wrists.

Initial laboratory results showed a serum salicylate 53 mg/dL; venous blood gas pH 7.46, CO_2 29 mmHg. Serum electrolytes showed sodium (Na) 140 mEq/L, potassium (K) 3.4 mEq/L, bicarbonate 20 mEq/L, blood urea nitrogen 14 mg/dl, creatinine 0.9 mg/dL, glucose 100 mg/dl. A comprehensive urine drug screen was negative. Urinalysis showed glucose 301–1000 mg/dl, 3+ proteinuria, and negative ketones.

The patient was treated with multi-dose activated charcoal and intravenous sodium bicarbonate. Her serum salicylate was monitored and gradually declined to 1 mg/dL. However, her glycosuria and proteinuria persisted. She also had hypouricemia with serum uric acid 1.6 mg/dL. Urine chemistry showed phosphorus 7.8 mg/dL; K 33 mEq/L; Na 102 mEq/L; uric acid 10.7 mg/dL. She

remained normoglycemic during her hospital stay. The patient was discharged home after 2 days with persistent glycosuria and proteinuria. Three weeks later, she was asymptomatic and her glycosuria and proteinuria resolved.
Discussion: Drug induced FS has been described but the literature about the effects of salicylate toxicity on proximal tubular function is rare. A case series of 5 patients with reversible aminoaciduria following salicylate toxicity was reported in 1961. The only case of reversible salicylate-induced FS in a pediatric age group was reported in a 17-year-old female who ingested 12.5 gram of aspirin. Our patient had reversible salicylate-induced proximal renal tubular dysfunction with significant glycosuria (despite euglycemia), proteinuria and hypouricemia. The mechanism is unknown but interference with proximal tubular basolateral Na-K-ATPactivity is a possible mechanism.
Conclusion: Clinicians should be aware that reversible proximal renal tubular dysfunction can be rarely associated with salicylate toxicity. The mechanism is not known.
Keywords: Salicylate, Glycosuria, Renal tubular dysfunction

41. Takotsubo cardiomyopathy associated with opiate and benzodiazepine overdose

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Background: Takotsubo cardiomyopathy ("broken heart syndrome") is a clinical syndrome defined by left ventricular dysfunction, elevated serum troponin and ischemic patterns on electrocardiogram but with patent coronary arteries. This syndrome was first described by Japanese investigators in 1991 and is classically seen after severe emotional stress. We report a case associated with drug overdose.
Case report: A 34 year old woman with past medical history of depression and multiple suicide attempts was found unresponsive with empty pill bottles (zolpidem 10 mg #60, oxycodone 15 mg #90 and lorazepam 1 mg #120). On the day prior to presentation, she was noted to have normal mental status. In the ED, the patient had temperature 99.0 F, heart rate 146 bpm, blood pressure 159/100 mmHg, and oxygen saturation 94% on room air. Two doses of 0.4 mg naloxone were given without effect. Physical examination revealed an unresponsive woman without evidence of trauma. Pupils were 3 mm and sluggishly reactive to light bilaterally. The remainder of the exam was significant for the presence of 11 fentanyl patches placed dermally. Her Glascow Coma Scale was 7 and she was intubated. Laboratory findings showed a Troponin-I of 6.53 ng/mL that peaked on day 2 at 15.33 ng/ml. Urine toxicology was positive for benzodiazepines and negative for other drugs of abuse, including cocaine and stimulant amines. A 12-lead electrocardiogram showed T-wave inversion in leads V1–V3. An echocardiogram revealed severe apical left ventricular hypokinesis and an ejection fraction of 10–20%, consistent with Takotsubo cardiomyopathy. The patient recovered without event and required only supportive management. Follow up echocardiogram 6 days post-ingestion showed improvement of left ventricular function with an ejection fraction of 50%.
Discussion: Drug related Takotsubo has been rarely associated with sympathomimetics like epinephrine, vasopressors, amphetamine salts, nasal decongestants and with withdrawal from opiates or benzodiazepines. These scenarios are consistent with the presumed pathophysiology of catecholamine induced myocyte toxicity. We present a unique case of Takotsubo cardiomyopathy that was associated with acute opiate and benzodiazepine overdose, which is not explained by the commonly supported mechanism of sympathetic nervous system induced myocyte toxity.
Conclusions: This patient had Takotsubo cardiomyopathy associated with severe opiate and benzodiazepine overdose. In the overdose setting, there is potential for emotionally induced sympathetic nervous system up-regulation, thus the provider should be wary of a "broken heart".
Keywords: Overdose, Cardiac toxicity, Benzodiazepine

42. A massive overdose of dalfampridine

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Background: Currently, about 400,000 Americans are living with multiple sclerosis (MS). MS is an immune mediated inflammatory disease that attacks myelinated axons in the central nervous system. One of the newer medications on the market is Dalfampridine or 4-aminopyridine (4-AP). Ampyra® was approved by the FDA In January 2010 for treatment of patients with MS.
Case report: Our patient was a 34 year old male with a past history of MS, who was brought to the Emergency Department after being found unresponsive with bottles of valacyclovir, temazepam and dalfampridine at his side. The patient had no other pertinent past medical history. His current medications were valacyclovir, temazepam, dalfampridine (4-AP) and a tysabri IV infusion. The patient had a temperature of 97.4, pulse 106, respiratory rate 24, BP 155/82. The patient was tremulous, awake, but not responding to questioning or following simple commands. He only responded to tactile stimulation by localizing to pain. The remainder of the physical exam was only remarkable for tachycardia and diaphoresis with bedside blood glucose of 144. Fifteen minutes after arrival the patient lost consciousness and began to have a seizure. The patient was administered 8 mg of lorazepam IV without control of his seizures. He was intubated and placed on a lorazepam drip and started on a propofol drip and loaded with phenytoin and phenobarbital (bolus and drip). The patient continued to have seizures. The patient's lab values were positive for a white blood count of 31.1×10^3/mcL. A urine toxicology screen was positive only for benzodiazepines. The CT of the brain and the ECG were both normal. The 4-AP level was 530 ng/mL (25 ng/mL and 49 ng/mL). The valacyclovir level was 7.5 mcg/mL (2.0–4.0 mcg/mL). The patient stopped seizing on hospital day 3 and was discharged 14 days later with normal mental status and neurologic exam.
Discussion: 4-AP is a potassium channel blocker that blocks the potassium ion current of repolarization following an action potential. The blockade of the potassium channel at the level of the membrane widens the action potential and enhances the release of acetylcholine, thus increasing post-synaptic action potentials. Due to the ability of 4-AP to reverse synaptic blockade it has been used as a treatment in many different neuromuscular disorders. The 4-AP level of our patient was 530 ng/mL. The treatment of patients with 4-AP overdose is supportive. Animal data suggests that patients with toxic levels of 4-AP may respond to phenytoin. Our patient did not respond to phenytoin.
Conclusion: Our case illustrates the highest recorded level of 4-AP in an overdose. Our patient appeared to be refractory to a combination of high doses of anticonvulsants and only improved after the drug was metabolized.
Keywords: Dalfampridine, 4-Aminopyridine, Seizure

43. The minoxidil brothers: Severe hypotension following intentional ingestions of a 5% topical preparation

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Background: Topical minoxidil (Rogaine®) is an over-the-counter medication used to treat male-pattern baldness. Minoxidil, which was initially marketed as an anti-hypertensive medication, is a vasodilator that works by relaxing arteriolar smooth muscle, increasing cutaneous blood flow, and stimulating resting hair follicles. This topical product became available without a prescription in 1996 as a 2% solution, and a 5% solution was approved in 1997. Because these formulations are highly concentrated, ingestions may result in significant toxicity. We present 2 cases of intentional minoxidil ingestions causing profound hypotension requiring vasopressor support.
Case report: Two brothers, ages 50 and 46, presented to the ED with nausea, vomiting, and lightheadedness within 30 minutes of drinking unknown amounts of a 5% topical minoxidil solution. Both had a history of alcohol abuse and drank the solution for its ethanol content. At presentation, the elder brother had a blood pressure (BP) of 74/30 mm Hg and a heart rate (HR) of 127 bpm, and the younger brother had a BP of 60/32 mm Hg and a HR of 113 bpm. Intravenous fluid resuscitation was promptly initiated; due to persistent hypotension, dopamine infusions were started and transfers to a tertiary care center arranged. The elder brother required vasopressor support with dopamine overnight. His initial EKG was notable for ST depression in the lateral leads and non-specific T wave changes. Cardiac biomarkers were undetectable. The younger brother required dopamine and norepinephrine for blood pressure support for >48 hrs. His troponin peaked at 0.12 ng/mL; an echocardiogram revealed an EF of 65–70% and no gross wall motion abnormalities. Both brothers developed alcohol withdrawal during hospitalization but were discharged on hospital days 4 and 5 in stable condition with no permanent sequelae.
Discussion: Minoxidil is well absorbed and peak effects are typically seen within 1 hour of ingestion. Despite a half-life of 4.2 hours, hypotensive effects may persist for more than 24 hours. A 60 ml bottle of 5% solution contains 3 g of minoxidil; this is 30 times the maximum daily dose. Both of our patients presented with significant and persistent hypotension and tachycardia. Although both brothers were initially started on dopamine, a vasopressor with strong a1 agonist activity, phenylephrine or norepinephrine would be preferred in a minoxidil overdose, as the use of positive chronotropes may worsen tachycardia.
Conclusions: Healthcare providers should be aware that ingestions of topical minoxidil preparations have the potential to cause severe, life-threatening hypotension requiring vasopressor support and intensive cares.
Keywords: Minoxidil, Overdose, Vasodilator

44. Apparent oral methylphenidate fatality with postmortem concentrations

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Methylphenidate (MPD) is a widely prescribed stimulant used primarily for the treatment of attention-deficit/hyperactivity disorder (ADHD). While typically used in the pediatric/adolescent population, there has been an increase in therapeutic use among adults in recent years. Suicide attempts involving methylphenidate ingestion have been well described; however deaths attributed solely to MPD ingestion have not been reported. We report a case of a suspected fatal MPD intoxication with corresponding postmortem liver, gastric, vitreous, peripheral and central blood concentrations.
Case report: A 62-year-old female was last known to be alive three days prior to a welfare check when she was found dead on the floor near her bed with obvious rigor mortis. Her past medical history included schizophrenia, depression, hypertension, fibromyalgia, knee osteoarthritis, chronic lower back pain, Hepatitis C and mitral valve prolapse. She had a distant history of ethanol abuse and her medications found at the scene included: acyclovir, atenolol, duloxetine, furosemide, gabapentin, potassium chloride, levalbuterol tartrate, levothyroxine, MPD, morphine, oxycodone/APAP, conjugated estrogen, prochlorperazine, ranitidine, and trazodone. The 10 mg MPD IR tablets were dispensed 12 days prior to death, and 331 of 360 were missing. There were no other significant medication discrepancies.

An autopsy revealed only mild coronary artery and aortic atherosclerosis with mild fatty changes and moderate chronic inflammation in the liver. Histopathological studies were unremarkable. Postmortem specimen analysis revealed MPD concentrations: peripheral blood 1.1 mg/L, central blood 0.98 mg/L, liver 3.6 mg/kg, vitreous humor 0.80 mg/L and 1 mg in stomach contents. Diphenhydramine was confirmed at less than 0.10 mg/L in peripheral blood, no other drugs were detected.
Discussion: Deaths related to excessive isolated MPD ingestion have not been previously reported. Reported MPD-attributable deaths have all involved self-administration via parenteral or nasal insufflation routes in an effort to abuse MPD rather than to commit suicide. As with our case, the exact mechanism by which excessive exposure to MPD results or directly contributes to death is unclear. Reports of postmortem MPD concentrations are rare and in our case we found markedly elevated blood MPD concentrations, higher MPD liver concentrations than previously reported and the first postmortem MPD vitreous humor concentration reported.
Conclusion: With no other identifiable cause of death, we report the first isolated MPD ingestion associated with a fatality.
Keywords: Methylphenidate, Overdose, Death

45. Baclofen overdose resulting in prolonged alteration of mental status

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Background: Baclofen is a gamma-aminobutyric acid (GABA) analogue that acts on $GABA_b$ receptors in the central nervous system (CNS). There are reports of baclofen overdose causing prolonged coma mimicking brain death with serum baclofen levels 2–6 times higher than the upper limit of therapeutic. We report a patient with an intentional baclofen overdose who had altered mental status for 7 days and a corresponding urine baclofen level.
Case report: A 52 year old female with a history of hypertension and alcohol abuse was brought to the hospital for altered mental status. On exam she was hypertensive, alternatively agitated and somnolent,

unable to follow commands, hypotonic and hyporeflexic. Evaluation including head CT and serum testing for ethanol, salicylates and urine screening for drugs of abuse was negative. EEG showed occasional frontal intermittent rhythmic delta activity (FIRDA) consistent with cerebral dysfunction and possible toxic or metabolic encephalopathy. During admission the patient remained disoriented, agitated, hypertensive and minimally verbal. She was treated with benzodiazepines for presumed ethanol withdrawal. Her family then discovered a new empty baclofen pill bottle in the patient's home. Urine baclofen level from the day of admission was 79 mcg/ml (minimum level of detection 2 mcg/ml). At 5 days her sensorium began clearing and she returned to her baseline 7 days after admission. She then admitted to consuming 12 baclofen tablets of unknown dosage prior to admission.

Case discussion: Previous case reports describe prolonged sedation and coma from central nervous system depressant effects of baclofen despite a short half-life of 2–4 hours. Half-life in overdose has ranged from 8 to 36 hours. The etiology of this prolonged clinical effect is unknown, and animal studies seem to refute the theory of delayed CNS clearance. Baclofen overdose and withdrawal are known to present similarly, and patients who present with overdose may progress to withdrawal. Urine levels of 760 mcg/ml and 774 mcg/ml were reported in two baclofen overdose cases resulting in fatality. While we cannot completely exclude concomitant ethanol withdrawal in this patient, the presence of baclofen in the urine, the neuromuscular physical exam findings, and the ingestion history provided by the patient all suggest that her prolonged symptoms were due to baclofen overdose.

Conclusions: Baclofen is a GABA analogue that in overdose may result in prolonged CNS effects by an unknown mechanism. We report an overdose of at least 120 mg of baclofen with a urine level of 79 mcg/ml resulting in 7 days of alteration of mental status and complete recovery.

Keywords: Baclofen, Overdose, Delirium

46. 4-Aminopyridine: A novel toxidrome

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Background: 4-Aminopyridine (4-AP) selectively blocks voltage-gated potassium channels, prolongs the action potential, increases presynaptic calcium influx, and subsequently, enhances interneuronal and neuromuscular synaptic transmission. It also enhances the release of acetylcholine from nerve endings and has been shown to increase dopamine transmission in rat striatum. This medication has been studied and used in many disease processes with poor neuronal transmission including multiple sclerosis (MS), spinal cord injuries (SCI), botulism, Lambert-Eaton syndrome, and myasthenia gravis. To date, there have been limited reports of either intentional or accidental 4-AP toxicity in humans.

Case report: A 37 year old man with a medical history of chronic progressive MS and depression was brought to a local emergency department (ED) after he was found by his brother naked and confused with pills both in his mouth and scattered on the floor. The patient's prescribed medications were 4-aminopyridine, paroxetine, and fingolimod. Upon arrival to the tertiary care hospital ED, initial vital signs included: blood pressure 125/104 mmHg, heart rate 133/minute, respiratory rate 30/minute, temperature 36.8°C and room air SpO_2 of 97%. Physical examination revealed the patient to be agitated and delirious with slurred and incomprehensible speech. Other physical exam findings included: mid-range and reactive pupils, dry mucous membranes, diffuse diaphoresis, choreoathetoid movements and tremulousness with diffuse hyperreflexia. A 4-AP level obtained six hours after suspected ingestion was 140 (ref range 17.3–42.7 ng/mL) with a level 24 hours later measuring 6 ng/mL. He was treated with diphenhydramine (DPH) and diazepam for presumed dystonia. A comprehensive urine drug screen by gas chromatography-mass spectrometry (GC/MS) identified small amounts of DPH, caffeine, nicotine, and acetaminophen metabolites.

Discussion: The toxidrome associated with 4-AP agrees with what is known about its mechanism of action, combining cholingergic features (diaphoresis, agitation, and seizures) with dopamine-related movement abnormalities (tremor, choreathetosis, and dystonias). This case combines and exemplifies the clinical features described in previous case reports. Management should be centered upon airway management, controlling agitation and seizures with benzodiazepines, and judicious use of antipsychotics and/or anticholinergics for the dopaminergic and cholinergic effects. As 4-aminopyridine recently received Federal Drug Administration approval for the treatment of ambulation in patients with MS, physicians should be keenly aware of this novel toxidrome, it's mechanism of action, and it management in overdose.

Keywords: Neurotoxicity, Delirium, Overdose

47. Accidental use of mistaken insulin products: A retrospective review of poison center data

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Background: There are limited data concerning accidental mis-identification and administration of insulin. We hypothesized that home monitoring by a poison control center (PCC) was safe for asymptomatic patients who use the wrong insulin product.

Methods: A retrospective review of PCC charts from 1/1/00–12/31/10 identified 652 cases of accidental mis-identification and administration of insulin. Patients already taking at least one form of insulin were included; intentional exposures were excluded. Data abstracted from charts included: patient age and gender, caller site, 'intended' and 'mistaken' insulin formulations and doses, use of oral diabetic agent, management site, emergency department (ED) referral, symptoms, blood glucose values and treatments. Outcomes were defined as the development of symptoms (altered sensorium, dizziness, diaphoresis or nausea), hypoglycemia (< 60 mg/dL; compared to 'normoglycemia' $= 60$–300 or 'hyperglycemia' $= > 300$), management site, admission to a health care facility (HCF) and death. Descriptive statistics were used with continuous variables reported as means and standard deviations, and categorical variables are reported as percentages; multiple logistic regression was used to determine outcome predictors. The validity of data abstraction was assessed by a secondary evaluation of 70 (10.7%) charts.

Results: A total of 652 charts met inclusion criteria. Mean age was 56.4 years (range 2–92; SD 15.4) and most (58.5%) were women. Accuracy of data abstraction was 98.2% (95% CI 97.2–98.9). Most ($n = 580$; 89%) calls originated from home, 70 (10.7%) from

a HCF and 2 (0.3%) from EMS. Overall, 397 (60.9%) cases were managed at home and 201 (30.8%) were immediately referred to a HCF; 26 (4.0%) patients refused referral or were lost to follow up. Two (0.5%) patients initially managed at home were later evaluated by EMS; neither became hypoglycemic or required admission. Symptoms developed in 56 (8.6%) of all patients; 14/41 (34.1%) of those with hypoglycemia, 32/402 (8.8%) with normoglycemia, and 10/135 (7.4%) with hyperglycemia. Dextrose (5–50%) was used in 69 (10.6%) cases. Of the 40 (6.1%) patients admitted to a HCF only 18 (45%) had hypoglycemia. There were no deaths. Hypoglycemia (OR 5.94; $p < 0.001$) and insulin dose administered (OR 1.04; $p < 0.001$) were risks for HCF referral. The type and dose of insulin administered did not predict symptom development.
Conclusions: Based on a retrospective analysis of PCC data, home observation of asymptomatic patients following the unintentional use of a wrong insulin formulation appears safe. Further work is required to improve risk assessment.
Keywords: Insulin, Adverse drug event, Poison center

48. Adverse events with cough/cold product use in children: Four years of surveillance

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Background: In efforts to analyze the risks associated with the use of cough/cold (CC) medications in children, a multisystem surveillance model was developed to detect and evaluate adverse events (AEs) associated with exposures to these medications. This study provides an update on the safety profile of these medications based upon 4 years of active surveillance.
Methods: Five case sources are routinely monitored for CC medication exposures in children: National Poison Drug System, FDA/Adverse Event Reporting System, English language medical literature, news/media reports and manufacturer internal safety reports. Case inclusion criteria: age < 12 y, exposure to ≥ 1 oral CC ingredient, ≥ 1 AE which occurred in the US. The Pediatric CC Medication Safety Surveillance Team reviewed each case to reach consensus on the causal relationship of each AE to each reported ingredient, estimate dose involved in the exposure, judge therapeutic intent of the exposure, and identify contributing factors.
Results: The Team reviewed 3140 unique cases. 2368 (75%) cases were determined at least potentially related to a CC ingredient: 38 (2%) fatal and 2330 (98%) non-fatal. Of the 2368 cases, 63% were < 4 years of age. The majority (68%) involved accidental unsupervised ingestions (AUIs). 2% were off-label administration by others (i.e. use for sedation or other off-label use). Use for labeled indications was reported in 22% and unknown indication in 9%. Dose was estimated supratherapeutic (75%), therapeutic (9%), or unknown (17%). The total number of exposures has decreased from 756 in 2008 to 519 in 2011. While 38 fatal cases were detected during this study period, only 8 deaths actually occurred since 2008 (5 in 2008, 1 in 2009, 2 in 2010). All deaths involved supratherapeutic or unknown doses.
Conclusions: While AEs are reported with appropriate use of CC products in children, the vast majority (75%) of AEs were determined to involve a supratherapeutic dose. Reason for exposure is critical when understanding what can be done to reduce these AEs. AUI was the most common root cause of supratherapeutic dosing among the cases we reviewed. Further interventions are underway to educate consumers about safe product storage and to discover advanced packaging technology to minimize the frequency and amount of drug accessed during AUIs. Continued surveillance will help determine the success of these interventions.
Keywords: Drug safety, Pediatric, Surveillance

49. Arginine Hydrochloride overdose in an infant

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Background: Arginine hydrochloride is used diagnostically to test for growth hormone deficiency and therapeutically for treatment of metabolic alkalosis. We report a 5 month old who was given an inadvertent overdose developing a metabolic acidosis, mental status changes, and a subdural hemmorhage.
Case report: A 5 month old male with ornithine-transcarbamylase deficiency was admitted to the hospital with URI and mild metabolic crisis and was started on an arginine infusion. Instead of the intended dose of 200 mg/kg arginine, the patient was inadvertently given 2 grams/kg. He developed hyperglycemia with peak glucose 900 mg/dL, acidosis (pH 7.20), hypernatremia (peak 152 mmol/L), hyperchloremia (peak 125 mmol/L), elevated lactate (peak 5.8 mmol/L), hyperosmolality (346 mOsm/kgH_2O), and hyperammonemia (peak 636 umol/L). He was given two 1 meq/kg sodium bicarbonate boluses. He became increasingly irritable, had several episodes concerning for seizure activity, and was subsequently intubated for mental status changes. CT showed a small subdural hemorrhage. The patient underwent one episode of hemodialysis immediately after the error was recognized, for the hyperammonemia, acidosis and metabolic abnormalities, which subsequently improved. At time of discharge he had returned to baseline neurologic status.
Case discussion: There is only one previous case report of a pediatric arginine hydrochloride overdose. It resulted in the death of a 21 month-old girl who received a dosing error of 3.9 g/kg over 30 minutes during a provocative test for growth hormone deficiency. She developed cardiac arrest at the end of the infusion with subsequent spontaneous return of circulation. She then developed seizures and central pontine myelinolysis with death after 8 days. In our case, the patient received half that amount given over 2 hours. Although he did develop seizures and a small amount of intracerebral bleeding, the patient experienced improvement in mental status. In normal human subjects given arginine, metabolic abnormalities have included transient asymptomatic hyperkalemia, hyponatremia and hypophosphatemia. It is presumed that the life threatening effects of an arginine hydrochloride are due to extreme hyperosmolality of the infusion (950 nmol/L) and the potential to cause intracellular sodium shifts. It is possible that early hemodialysis prevented a fatal outcome in this case and should be considered as soon as a therapeutic error is recognized.
Conclusions: We report an infant who was given an inadvertent overdose of arginine hydrochloride, developing mental status changes and metabolic derangements, which improved with

hemodialysis. This case reaffirms the rare but serious nature of Arginine Hydrochloride overdoses.
Keywords: Hemodialysis, Pediatric, Adverse drug event

50. Lead poisoning in an infant from a Nigerian traditional remedy

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Background: Childhood lead poisoning is most often due to lead-based paint exposure and typically develops after a child learns to walk. We present an unusual case of an elevated blood lead level (BLL) in a 6-month-old infant due to a Nigerian traditional remedy applied to the infant's eyes.
Case report: A 6-month-old infant born in the US to Nigerian parents was found to have an elevated BLL (13 mcg/dL). The infant was started on iron and referred to the Region 1 New England Pediatric Environmental Health Specialty Unit (PEHSU), where one week later the confirmatory BLL was 13 mcg/dL. Other laboratory indices were normal except for microcytosis. The infant was asymptomatic, with normal developmental milestones. Other potential sources of lead poisoning in the family's residence and environment were ruled-out. Further questioning revealed that since age 2 weeks, a Nigerian folk remedy and cosmetic called 'tiro' had been applied to the infant's eyelids 3–4 times weekly to promote visual development and improve attractiveness. A grandparent had purchased the 'tiro' from a street vendor in Ilorin, a city in Kwara State, Nigeria. The PEHSU recommended immediately ceasing application of 'tiro' to the infant. Three months later, the BLL had fallen to 8 mcg/dL. Scanning electron microscopy of the 'tiro' was consistent with lead sulfide and quantitative analysis found the 'tiro' consisted of 82.6% lead (826 mg/g). A single application was estimated to deliver approximately 8 mg of lead to the infant's face.
Case discussion: Several factors implicate 'tiro' as the agent responsible for this infant's elevated BLL: other sources of lead were ruled out, the infant's developmental age precluded him from moving independently to potential hazards, the 'tiro' was $>80\%$ lead, and when the practice was discontinued, the BLL dropped. This case was reported to public health authorities at the Centers for Disease Control & Prevention (CDC) in 2011. CDC officials have subsequently been working with the Nigerian Ministry of Health to conduct outreach education discouraging this cultural practice in specific districts of Nigeria.
Conclusions: The Nigerian folk remedy and cosmetic, 'tiro', can be a source of lead poisoning in infants. Clinicians should carefully query caregivers regarding their health beliefs and culturally-based practices. Use of ethnic remedies on infants should prompt clinicians to consider BLL screening before the age typically recommended by federal and state guidelines.
Keywords: Lead, Pediatric, Public health

51. Phenol toxicity following cutaneous exposure to Creolin®

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Background: Phenol is a caustic that may cause cutaneous or gastrointestinal burns, depending on the route of exposure. Significant absorption may result in systemic toxicity. We present a case of cutaneous phenol exposure resulting in cutaneous and systemic phenol toxicity.
Case report: A 9-year-old girl was exposed to Creolin®, a general purpose disinfectant containing 40–50% phenol, when her mother applied this product to her head and upper torso in an attempt to delouse the child. The patient presented to the emergency department with depressed mental status requiring endotracheal intubation and skin erythema in the distribution of contact with the cleanser. An initial EKG revealed sinus tachycardia with brief runs of monomorphic premature ventricular contractions. On hospital day (HD) 1, the area of erythema extended to both upper extremities and hyperpigmentation developed over the affected areas, which continued to darken during the hospital course. The patient was extubated late on HD 1. On HD 2, the patient's urine was noted to be a dark green color that resolved later that day. By HD 3, areas of desquamation and decreased sensation were noted in areas of maximal contact with the cleanser. The patient developed a mild transaminitis with peak AST and ALT levels of 84 units/liter and 99 units/liter, respectively. The patient was stable through the remainder of her hospital course and was discharged to home on HD 4.
Case discussion: Our patient presented with signs of cutaneous and systemic phenol toxicity characterized by dermal burns, depressed mental status, cardiac dysrhythmias and elevated hepatic transaminases. Phenol exposure is known to cause systemic toxicity following even limited dermal exposure. This may include CNS depression, seizures, hypotension, dysrhythmias, hypothermia, metabolic acidosis, methemoglobinemia, and direct renal and hepatic injury. Patients with significant phenol exposure often present with decreased mental status or seizures and require prompt airway management. After stabilization, decontamination is of critical importance. Exposed skin should be copiously irrigated with soap and water. If available, a mixture of 75% low-molecular-weight polyethylene glycol and 25% ethanol is recommended by the manufacturer and may be superior to irrigation with water alone. Patients with significant dermal toxicity may require referral to a regional burn center.
Conclusions: Phenol is still used widely in manufacturing processes and is also present in less concentrated formulations such as topical and oral analgesics. High concentration formulations such as Creolin® are still available and misinformation about their appropriate use can have adverse consequences.
Keywords: Pediatric, Caustic, Neurotoxicity

52. A 10-year retrospective review of venlafaxine ingestions in children under 4 years of age

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Background: Serotonin-norepinephrine reuptake inhibitors (SNRIs) are a widely utilized class of neuropsychiatric medications used for

anxiety, major depression, obsessive compulsive disorder, and attention deficit hyperactivity disorder. Venlafaxine (Effexor®) use has been associated with cardiovascular problems, cognitive decline, and seizures with overdose in adults. There are few studies on venlafaxine exposures in pediatrics and currently the drug is not FDA approved in this population.

Objective: The goal of this study was to describe adverse events related to pediatric ingestion of venlafaxine.

Methods: A 10-year retrospective poison center study of ingestions of venlafaxine in children 4 years of age or younger from January 2001 to December 2011 was performed. IRB approval was obtained and cases were blinded prior to analysis. Inclusion criteria were venlafaxine as a single ingestant in children 4 years and younger followed to a known outcome. Amount ingested, age, sex, weight, time to call, clinical symptoms and patient outcomes were collected.

Results: A total of 99 cases of ingestion were identified. 53 (53%) were female and 47 (47%) were male with a mean age of 1.83 years (range 7 months to 4 years, SD 0.75 years). The mean amount ingested per weight was 15.37 mg/kg (range 2.23 to 120.97, SD 17.60). The mean time to call was 51 minutes (range 5 to 600 min, SD 102). Of the 99 children, 11 (11%) had hypotension $<$ 90/60, 2 (2%) had hypertension $>$ 140/90, 53 (53%) experienced heart rate $>$ 100, 4 (4%) had a temperature greater than 100, 29 (29%) had a respiratory rate $>$ 20, and 1 (1%) had a seizure. Activated charcoal was administered to 60 (60%) of the patients.

Outcomes: No effect in 65 (65%), minor effects in 13 (13%), minimal effects possible in (20) 20%. moderate effect in 1 (1%), and major clinical effects in 1 (1%). The incidence of adverse effects was: 11 (11%) lethargy/drowsiness, 5 (5%) mydriasis, 5 (5%) nausea/vomiting, 4 (4%) activation, 3 (3%) somnolence, 3 (3%) diaphoresis, 1 (1%) ECG change, and 1 (1%) seizure.

Conclusions: Venlafaxine toxicity at doses up to 60 mg/kg in children 4 years of age and younger manifested primarily as minor effects such as lethargy and drowsiness. One patient (1%) at 110 mg/kg manifested symptoms such as hypotension and mild tachycardia. One patient (1%) at 120 mg/kg (1500 mg) manifested symptom of a tonic-clonic seizure. In this age group ingestions $<$ 60 mg/kg showed favorable outcomes with supportive care and gastric decontamination with activated charcoal.

Keywords: Antidepressant, Pediatric, Selective serotonin reuptake inhibitors

53. First case report of pediatric dabigatran poisoning

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Background: Dabigatran etexilate (Pradaxa®) is an oral, direct thrombin inhibitor used to prevent stroke and embolism in patients with non-valvular atrial fibrillation. This is the first report of pediatric poisoning with laboratory confirmation.

Case report: A 2 year old African American F was found chewing on a maximum of one Pradaxa® 150 mg capsule from a medication tray. Most of it was retrieved but some was eaten. The Poison Control Center was called and referred patient to the Emergency Department. Vital signs (1 hr post ingestion): BP 107/76, HR 99, RR 20, O_2 saturation 99% on room air, T 36.2 C; weight 13.15 kg. Physical exam showed an alert, well-appearing child without vomiting, bruising, bleeding or other complaints. She had no significant past medical history. Labs (3 hrs post ingestion): aPTT (activated partial thromboplastin time) 45.1 seconds (normal range 23.5–34.2 seconds), BUN 10, creatinine 0.3, total protein 7.3, albumin 3.7, alkaline phosphatase 234, aspartate aminotransferase 37 IU/L, alanine transaminase 37 IU/L, hemoglobin 12.1, hematocrit 36, platelets 315. At four hours, normal saline 264 mL and activated charcoal 25 grams with sorbitol were given. She was transferred to a tertiary pediatric hospital for observation. Her 18 hr aPTT = 37.5 seconds; 24 hr aPTT = 34.8 seconds. She was discharged on Day 2, 30 hrs post ingestion in her normal state of health. Serum samples (from 3 hrs post ingestion) were analyzed for dabigatran utilizing high resolution mass spectrometry; a concentration of 25 ng/ml dabigatran (active form) was measured.

Discussion: Dabigatran, a direct thrombin inhibitor, may pose a significant bleeding risk following inadvertent pediatric ingestion. Overdose experience is limited and, to date virtually nothing is known about the effects and metabolism in pediatric patients. Dabigatran etexilate, in adults, is rapidly hydrolyzed by esterases in both plasma and the liver to its active form (dabigatran) with peak levels at 1.5–3 hrs. However, biotransformation in the pediatric population is unknown. In our 2 yr old patient, dabigatran (active form) was measured in the serum 3 hours post ingestion. Notably, none of the dabigatran etexilate (prodrug) was detected, indicating efficient biotransformation in the pediatric population. Our patient experienced slight aPTT elevation at 3 hrs with no clinical symptoms of anticoagulation after ingestion of $<$ 150 mg capsule with complete resolution by 24 hrs.

Conclusions: This case highlights the potential danger of dabigatran ingestion in pediatric patients, the usefulness of aPTT monitoring given the early elevation following ingestion and evidence that biotransformation of dabigatran etexilate in the pediatric population is similar to adults.

Keywords: Pediatric, Dabigatran, Metabolism

54. Severe pediatric lead toxicity after ingestion of three intact rifle cartridges

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Background: This is the first pediatric report and second ever report of lead toxicity due to ingestion of intact cartridges.

Case report: A developmentally delayed, non-verbal 15-year-old male with a history of pica behavior presented to the ED after vomiting a piece of a plastic fork. Abdominal X-Ray revealed a foreign body in the stomach. EGD was performed to retrieve what was presumed to be the remainder of the fork, but 3 intact, partially corroded 30 mm rifle cartridges were found and removed. No fork remnant was identified. The medical toxicology service was consulted. Upon focused questioning of the parents, it was discovered that the patient had access to rifle shells on a single day 1 month prior to presentation. He had exhibited a decreased level of activity, anorexia, diarrhea, constipation and nausea with emesis for the

past 3 weeks. Labs demonstrated a new normocytic anemia with heavy basophilic stippling and mild renal insufficiency. The blood lead level (BLL) returned 12 hours after EGD and was 146 mcg/dl. The patient was chelated with a regimen of British anti-lewisite (BAL) IM and CaNa2-EDTA IV. On day 4 the BLL was 71 mcg/dl, so therapy was transitioned to PO Succimer. The patient was discharged on hospital day 9 on PO Succimer with a BLL of 53 mcg/dl. 36 days after removal, the BLL was 51 mcg/dl. At this time, the patient had completed his course of Succimer and was at his baseline per parents with resolution of the renal insufficiency and improving anemia. The BLL was 38 mcg/dl on post EGD day 98. The patient is currently being followed with serial lead levels and multivitamin with iron supplementation.

Case discussion: This case was challenging due to the patient's developmental delay and baseline nonverbal state, as determining symptoms of encephalopathy was difficult. However, the subtle behavioral changes in concert with the extremely high lead level led to the decision to treat with BAL/CaNa2-EDTA for severe plumbism. It is of interest that the cartridges remained in the stomach for a month without breaking apart or passing the pylorus. The patient's persistently elevated BLL is likely due to lead uptake into the RBC and bone compartments. This patient did well with rapid transition from parenteral to oral chelation therapy and appears to have returned to baseline neurologic status. X-Ray image and endoscopy photos will be presented.

Conclusion: This is the first pediatric case of lead toxicity due to intact rifle cartridge ingestion and the first time intact cartridges have been removed by endoscopy. Intact rifle cartridges can result in severe toxicity if ingested, but can be safely removed from the stomach by EGD. Determining the severity of lead toxicity can be challenging in the pediatric population.

Keywords: Lead, Chelation, Pediatric

55. Pink disease resurfaces

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Background: A case of acrodynia is described, a rare presentation of chronic mercury (Hg) exposure in a child.

Case report: A previously healthy 5-year-old boy presented with 7 days of fatigue, dull abdominal pain, anorexia, and red rash on his face, trunk, and extremities. His vital signs (VS), exam, and office workup were unrevealing. He was diagnosed with viral syndrome. Over the next 4 weeks, further symptoms developed, including increased fatigue, irritability, profuse sweating, and extremity pain. A 5-pound weight loss, hypertension (127/97 mmHg) and tachycardia (155 bpm) were noted. He was admitted; workup revealed: WBC, 25,800 (nl differential); Hg, 16.1 g/dL; Hct, 47.8%; metanephrine, 90 pg/mL (nl $\leq$ 57 pg/mL); normetanephrine, 235 pg/mL (nl $\leq$ 148 pg/mL); VMA, 4.6 mg/24 h (nl $\leq$ 2.3 mg/24 h); normal CMP, UA, CRP, TFT, gastrin and lead levels; and normal abdominal ultrasound. He was discharged with presumed viral syndrome.

One week later, he developed excessive drooling, and a red rash on his trunk, hands, and feet. Exam revealed an extremely irritable child who repeatedly rubbed his hands and feet. VS were BP 122/86, HR 160, RR 18, T 98.3. He had a blanching, erythematous, pinhead papular rash on his trunk, and nonblanching, erythematous, excoriated macular rash on his palms and feet.

Dad, a HVAC contractor, previously brought home 3 Hg switches. Mom recalled vacuuming elemental Hg from the kitchen floor 1 week prior to symptom onset. His 9-year-old sister had a concurrent similar illness. The EPA was contacted, and subsequently both children were hospitalized and toxicology was consulted. The patient's blood Hg level was 12 mcg/L (nl $<$ 10 mcg/L). Home and vacuum exhaust Hg air levels were 5,000 ng/m^3 and 37,000 ng/m^3, respectively (nl $<$ 1,000 ng/m^3). Succimer was administered per standard protocol. After treatment and environmental remediation, the patient's symptoms resolved.

Case discussion: Acrodynia (Gr: "painful extremities") was not uncommon in children the early 20 th century, when teething powders often contained calomel. Since not all exposed children developed the systemic and cutaneous symptom complex, an idiosyncratic hypersensitivity reaction was postulated. Cutaneous findings include initial bright pink hue of the fingers, toes, and nose ("pink disease"); subsequent duskiness, swelling and desquamation may occur. Evanescent truncal rashes also occur. Extremity pain and pruritis are common. Systemic symptoms include irritability, insomnia, fatigue, anorexia, photophobia, and hypersalivation. Catecholamine excess accounts for sympathetic over activity, and is related to Hg's inhibition of catechol methyl transferase.

Conclusions: This case highlights classic symptoms of a now rare disease.

Keywords: Mercury, Pediatric, Acrodynia

56. Lisinopril ingestions in children less than 6 years of age

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Background: Lisinopril is an angiotensin converting enzyme inhibitor used for hypertension, congestive heart failure and acute myocardial infarction. For adults, the maximum recommended dose is 40 mg/day. Reports of clinical experience with pediatric ingestions are minimal.

Method: A 13 year retrospective study of lisinopril ingestions in children was analyzed. Cases included children less than 6 years (yrs) who were treated at a HCF. CHR approval was obtained and cases were blinded. Inclusion criteria: lisinopril as a single ingestant, age $<$ 6 yrs, treatment site HCF, case followed to a known outcome. Data collected: age, sex, weight, amount (amt) ingested (reported as exact, estimate, maximum, unknown amt), vital signs, symptoms, and outcome.

Results: A total of 296 cases of lisinopril ingestions without coingestants were identified. Demographics: male 51%; mean age 1.97 yrs (range 9 mo–5 yo, SD 0.65 yrs). Mean amt ingested was 7.0 mg/kg (range 0.1 mg/kg to 76.2 mg/kg, SD 10.5 mg/kg) and 84.3 mg total (range 2.5–800 mg, SD 132.0 mg). Therapy: activated charcoal (AC) 151 patients (pts) (51.1%); gastric lavage 3 pts (1.0%); and IV fluids (IVF) 9 pts (3.0%). Of the 296 pts, 8 pts (2.7%) developed hypotension (HOTN) (range 55–74 systolic and 22–48 diastolic), 6 pts (2.0%) developed tachycardia (range 159–174 bpm), and 4 pts (1.4%) developed bradycardia (range 67–91 bpm). Of the 9 pts who developed HOTN, 3 pts (30%) were drowsy. The lowest blood pressure of 55/22 was recorded in a 1.8 yr male who ingested an

estimated 120 mg (13.3 mg/kg). The lowest dose of lisinopril causing HOTN was 50 mg or 3.9 mg/kg. 282 pts (95.3%) were treated and released from the ED, 14 pts (4.7%) were admitted.

Ingestion of an exact amount of lisinopril was reported in 61 pts (20.6%); mean amt ingested 3.0 mg/kg (range 0.1–10.9 mg/kg, SD 2.7 mg/kg); and 33.4 mg total (range 2.5–160 mg, SD 32.3 mg). None of the pts with exact lisinopril ingestions developed HOTN although tachycardia (range 160–170 bpm) was noted in 3 pts (4.9%). No pts received IVF or were admitted (100% treated and released). Therapies in the exact group: AC 20 pts (32.7%), lavage 1 pt (1.6 %), and ipecac administered prior to ED arrival in 1 pt (1.6%).

Of all pts, no effect was observed in 256 pts (86.5%). Cardiovascular effects were seen in 19 total pts (6.4%). Minor unrelated effects were seen in 21 pts (7.1%): drowsiness, vomiting, diarrhea from GI decontamination.

Conclusions: The lowest dose of lisinopril to cause HOTN was 50 mg or 3.9 mg/kg. Although continued evaluation of pediatric lisinopril ingestions is essential to determine more specific thresholds for toxicity, these results indicate that lisinopril ingestions <50 mg total or <4 mg/kg may be safely managed at home.

Keywords: Lisinopril, Pediatric, Hypotension

57. A review of pediatric exposures to anti-dementia drugs reported to a state wide poison control system

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Background: There are currently four anti-dementia drugs commonly used in the United States. Three (donepezil, galantamine, and rivastigmine) are centrally acting acetylcholinesterase inhibitors. The other, memantine, is an NMDA antagonist. Their use has increased over the last decade in response to the growing prevalence of dementia. There is a paucity of literature regarding toxicity from these agents, especially in pediatric patients. We sought to characterize pediatric exposures to these anti-dementia drugs.

Methods: We performed a retrospective review of a statewide poison system's database for all cases of pediatric (less than 19 years of age) exposures to anti-dementia drugs from Jan 2001–Sept 2011. Data collected included age, sex, drug name, route of exposure, reason for exposure, adverse effects, interventions, hospitalization, and death.

Results: There were 126 cases identified. Sixty-eight (54%) were male. One hundred and one (80%) patients were two years of age or less. The mean age was 2.3 years. One hundred and twenty three (98%) cases were unintentional exposures. Donepezil was involved in 59 (47%) cases, memantine in 37 (29%) cases, galantamine in 15 (12%) cases, and rivastigmine in 15 (12%) cases. Four of the rivastigmine cases involved the transdermal patch. All cases were single substance exposures to the anti-dementia drug. No symptoms were reported in 103 (82%) of the cases. Reported symptoms were GI upset ($n=16$), sedation ($n=7$), drooling ($n=5$), and diaphoresis ($n=3$). No other symptoms were reported. In cases with known amounts ingested, the average dose and number of symptomatic cases were: donepezil 5.4 mg, 2; memantine 6.2 mg, 1; galantamine 6.2 mg, 0; rivastigmine 6 mg, 1. Symptoms were reported in 7 of the 11 exposures to oral rivastigmine. Forty-seven (37%) cases were evaluated at a healthcare facility including 9 of the 11 oral rivastigmine exposures. Four patients (3%) were admitted for an average of 1 day. Activated charcoal was given 13 (10%) times while atropine was given once. There were no serious outcomes or deaths in this series.

Conclusions: In our review, pediatric exposures to anti-dementia drugs resulted in minimal symptoms and no serious outcomes or death. Exposures to oral rivastigmine appear to be associated with a higher rate of symptoms and health-care evaluation.

Keywords: Pediatric, Poison center, Ingestion

58. Compounded ointment results in severe toxicity in a pediatric patient

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Introduction: Dermal drug delivery is becoming more common, as evidenced by the increased numbers of compounding pharmacies preparing topical products for chronic pain management. Consumers may not appreciate the potency or dangers associated with some of the drugs in these preparations. Pediatric patients are especially at risk for significant toxicity with accidental exposures due to their relatively large skin surface area, allowing significant systemic absorption from even small dermal exposures. We report a case of severe toxicity in an 18 month old male from exposure to his father's compounded pain ointment.

Case: An 18 month old previously healthy child had an ointment applied topically to a diaper rash by his mother, consisting of a single pump of a prescription ointment that her husband received from a compounding pharmacy for neck pain. Approximately 20 minutes later, when the child had been put down for a nap, he had gasping respiration but was otherwise unresponsive. EMS was called and the child was unresponsive. In the ED, vital signs were pulse of 57 bpm , BP 74/35 mmHg, RR 21 breaths/min, O_2 saturation 98% on a non-rebreather. Fingerstick glucose was 105 mg/dL. In the ED, physical examination was significant for: unresponsiveness; pinpoint pupils and hyporeflexia. The patient's mental status continued to deteriorate with depressed respirations and he was intubated. Laboratory results were non-contributory. Electrocardiogram revealed only sinus bradycardia.

The patient was transported to a pediatric intensive care unit. He did well over the next several hours with supportive care and had return to normal vital signs over the following 12 hours. He was extubated the following morning without problems.

Blood taken at the time of ED presentation had a serum clonidine level of 9.2 ng/ml (reference range 0.5–4.5 ng/ml) and a norketamine level of 41 ng/ml (reporting limit >20 ng/ml).

Discussion: Dermal absorption of drugs leading to significant toxicity in children is well-known. Our patient had toxicity from a topical pain medication compounded with several potent drugs known to cause CNS depression.

Conclusions: There has been an increase in the use of this drug delivery system for management of chronic painful conditions.

The popularity and attractiveness of such preparations may be the perception that they are somehow safer and more natural than taking pills. This perception, the fact that these are not dispensed in child-proof containers, and are often mailed to the patients without pharmacist counseling, can lead to increased inadvertent exposures in the pediatric population.

Keywords: Pediatric, Dermal toxicity, Adverse drug event

59. Encephalopthy from unintentional donepezil and memantine ingestion in a 2 year child with confirmatory serum levels

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Background: Donepezil and memantine are commonly prescribed anti-dementia drugs. There is a paucity of literature concerning pediatric ingestions of these drugs. We describe a case of 2 year old child who developed an encephalopathy after an unintentional ingestion of donepezil and memantine.

Case report: A previously healthy 2 year old female was found by family agitated and reporting visual hallucinations. Medications in the house were donepezil, memantine, orlistat, a calcium supplement, acetaminophen, and an unknown antibiotic. They were unable to provide strengths or dosages. There was no witnessed ingestion. She was taken to an emergency department. There she became sedate and had rightward eye deviation but no tonic-clonic activity. A head CT, lumbar puncture (LP), and urine drug screen were normal. She was transferred to a children's hospital and remained sedated with occasional rightward eye deviation and rapid breathing, concerning for seizure activity. Her vital signs were: heart rate 150 bpm; blood pressure 97/36 mmHg; respiratory rate 17 breaths per minute; SaO_2 98% on room air; temperature of 37.8°C. She was given intravenous lorazepam and started on levetiracetam. A CT head, MRI brain and repeat LP were normal. An EEG showed no seizure activity but was consistent with mild diffuse encephalopathy of nonspecific etiology. Other blood and chemistry tests, including a viral encephalopathy panel, were normal. Twenty-four hours later she was more appropriate. She continued to improve and was discharged 72 hours after admission with the diagnosis of unknown encephalopathy. Serum collected approximately 24 hours from the onset of her symptoms was sent for comprehensive drug testing. Memantine and donepezil were detected at concentrations of 32 ng/ml and 470 ng/ml, respectively. Therapeutic memantine levels after a single 20 mg dose vary between 80 and 120 ng/ml. Therapeutic donepezil serum levels vary from 25–50 ng/ml depending on dose.

Case discussion: There is a paucity of published literature detailing pediatric exposures to these drugs or other anti-dementia agents. Memantine is a NMDA receptor antagonist. There are no prior pediatric exposures reported. Donepezil is a centrally acting acetylcholinesterase inhibitor. There is only one pediatric case report in the literature; a 2 year old reportedly ingested a 10 mg donepezil tablet and developed sedation, drooling, and fasciculations that resolved with observation. No donepezil serum level was reported.

Conclusions: This case demonstrates that unintentional ingestions of memantine and donepezil can cause significant neurological symptoms in pediatric patients.

Keywords: Pediatric, Ingestion, Neurotoxicity

60. Circumstances and characteristics of poisoning in infants up to three months of age

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Introduction: 70% of all poisonings occurring in children ≤12 years involve toddlers 12 to 36 months of age. Infants ≤3 month are rarely involved in cases of poisoning. Moreover, the causes of exposure to toxicants are different. The aim of this study was to identify the circumstances of exposure, involved substances, and characteristics of cases of poisoning in infants ≤3 month.

Methods: A retrospective case study of all inquiries involving infants ≤3 month reported to the Swiss Toxicological Information Centre (STIC) between Jan 2001 and Dec 2010. Analysis of poisoning severity, included only cases with written feedback from the treating physicians.

Results: A total of 136945 pediatric (<16 y/o) poisonings were registered by the STIC during the study period. 1096 (0.8%) cases involved children ≤3 month – 432 females and 384 males (unknown 280). Exposure was acute in 968 (88.3%) cases, chronic in 106 (9.6%), and unknown in 22. The caller was a layperson in 603 (55%) cases, a physician in 454 (41.4%), and another health professional in 39 (3.6%) cases. Pharmaceuticals were involved in 650 (59.3%) cases, household products in 223 (20%), cosmetics in 70 (6.4%), food/beverages in 47 (4.3%), and other agents in 106 (9.7%) cases. The routes of exposure were oral (776, 70.8%), inhalational (109, 9.9%), rectal (44, 4.0%), intravenous (42, 3.8%), and other (125, 11.4%). Agents involved in more than 10 cases were cholecalciferol (154), essential oils (108), detergents (67), smoke gas (55), acetaminophen (40), amidosulfonic acid (26), acetic acid (19), phytomenadione (18), chlorhexidine (18), alcohol (16), amoxicillin (14), and zidovudine (11). The most frequent circumstances of exposure were confusion of medicinal drops with essential oils or other pharmaceuticals (147, 13.4%), milk/tea prepared with water contaminated with a decalcifier or dish detergent (92, 8.4%), 10-fold dose error (58, 5.3%), exposure related to breast-feeding (54, 4.9%), administration of 1 pipette instead of 1 drop (48, 4.4%), giving the wrong strength of the drug (47, 4.3%), and administration of substances by an older sibling (46, 4.2%). Severity of poisoning (based on 174 cases with written medical follow-up) was mild in 41, moderate in 9, and severe in 6 cases. 118 children were asymptomatic. No fatalities were reported. Substances that caused severe symptoms were cholecalciferol (2 cases), dimenhydrinate (2 cases, twins), digoxin (1), and illicium anisatum (1).

Conclusions: Infants up to 3 months of age were involved in less than 1% of all pediatric poisonings. Severe symptoms were rarely observed, and the most common source of error was confusion of medicinal drops with essential oils or other pharmaceuticals.

Keywords: Infants, Cholecalciferol, Essential oils

61. Iatrogenic error: Survival after Amphotericin B overdose

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Background: Amphotericin B deoxycholate (AB) is an IV polyene macrolide antifungal agent isolated from Streptomyces nodosus. AB is still considered the drug of choice for most systemic fungal infections. Normal dosing is 0.25–1.5 mg/kg/day. Adequate tissue concentrations are needed for treatment success, especially in immunosuppressed patients. Therefore, dose limiting nephrotoxicity is common. Newer AB preparations are available with lower renal toxicity profiles at therapeutic concentrations. Liposomal amphotericin B (LAB) is a lipid complex formulation. LAB is dosed higher than AB which results in enhanced efficacy and reduced toxicity. The similar names of the 2 drugs can be the basis of dispensing and other iatrogenic errors.
Case report: 60-year old, 57 kg female renal transplant recipient presented with suspected fungal infection. 250 mg of intravenous conventional AB (4.4 mg/kg) was inadvertently substituted for LAB [Day 0]. The poison center was contacted 16 hours later and recommended hemodialysis followed by plasmapheresis and/or exchange transfusion. Labs – see Table 1.

The patient received multiple rounds of plasmapheresis and hemodialysis during a 15-day period. Her baseline creatinine was 2.1 mg/dl and peaked on Day 1 at 3.0. Highest serum potassium level was 5.1 meq/L which also occurred on Day 1. Serum amphotericin levels peaked at 4.9 mcg/ml on Day 1, and dropped to 0.2 mcg/ml on Day 8 (therapeutic: 1–2 mcg/ml). She remained hemodynamically stable throughout the hospital course. Urine output was not compromised. Her mental status remained normal. She received 1 course of packed red blood cells for a drop in hemoglobin and hematocrit and did not require subsequent transfusions. Broad spectrum antibiotics were started for fever on Day 4. Corticosteroids were not administered. She was discharged on Day 15 with serum blood urea nitrogen and creatinine of 18 mg/dl and 1.4 mg/dl respectively.
Discussion: AB overdose can be fatal. Aggressive treatment with repeated hemodialysis and plasmapheresis most likely limited nephrotoxicity in our patient. Enhanced elimination was successful as reflected by the rise and fall of serum AB levels. Drug dispensing errors involving drugs with similar names are common and problematic with drugs such as AB.
Conclusions: Aggressive treatment with hemodialysis and plasmapheresis may limit morbidity and mortality of AB overdose. The different formulations of amphotericin B marketed have a variety of dosing recommendations. Hospitals and pharmacies must maintain vigilance for look-alike drug names to avoid errors.
Keywords: Amphotericin, Dosing error, Renal toxicity

Table 1. Data for abstract 61.

Hospital day	Cr (mg/dl)	Amphotericin (mcg/ml)
0	2.1	
1	2.9	4.9
		1.7 after plasmapheresis
2	3.0	1.5
3		1.1
5	2.1	0.4
8		0.2
15	1.4	

62. Triad of toxicity following topical application of EMLA

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Background: Topical anesthetics are commonly used in pediatrics. EMLA Cream (AstraZeneca; Wilmington, DE) is a eutectic mixture of prilocaine (2.5%) and lidocaine (2.5%) packaged in 5 and 30 gram tubes intended for use on intact skin. Although generally well tolerated, systemic absorption can result in a clinical triad: seizures, methemoglobinemia (MetHb), and respiratory failure. We report a case of an infant who suffered serious sequelae after excessive EMLA application.
Case report: 4-month-old female with Klippel-Trenaunay syndrome was undergoing a series of laser treatments for port wine stains (torso and lower extremities). Despite pre-treatment with 15 gm of EMLA and occlusion with plastic wrap, she had significant discomfort after first two sessions. Parents were advised to apply 60 gm of EMLA with occlusion prior to third visit – resulting in EMLA covering 50% of body surface area (BSA). While waiting in the dermatology clinic, 75 minutes after EMLA application, the patient became cyanotic and limp. She was unresponsive with no respiratory effort and displayed generalized tonic clonic movements. Resuscitation included bag-mask ventilation and lorazepam. She was intubated and transported to the pediatrics ICU. Seizure activity terminated after 5–10 minutes. Serum lactate was 1.56 mmol/L. Methemoglobinemia (22.8%) was treated with methylene blue (1.5 mg/kg). One dose of IV lipid emulsion was given. Brain CT scan was normal and EEG was negative for continued seizure activity. MetHb fell to 6% one hour after methylene blue. Initial plasma lidocaine concentration was 7.7 mcg/ml (therapeutic: 1.5–5 mcg/ml). Prilocaine was undetectable. G6PD deficiency was not detected. She was extubated 48 hours after presentation with normal neurological exam and discharged 4 days after admission.
Discussion: There are several contributing factors that lead to EMLA toxicity in this patient. Manufacturer's directions specify a maximum of 2 grams applied over 20 cm^2 BSA. Our patient received 60 grams on 150 cm^2 BSA. Prilocaine and lidocaine are amide-linked local anesthetics that bind to sodium channels, blocking sodium current responsible for nerve conduction. In concentrations exceeding therapeutic ranges, lidocaine may cause CNS and cardiovascular toxicity. MetHb may occur from systemic prilocaine absorption. While our patient exhibited toxicity from both agents due to excessive dosing, it is also postulated that absorption was enhanced due to her vascular malformation.
Conclusions: Dermal absorption of topical anesthetics can cause a systemic toxicity triad. Providers need to be aware that this risk is elevated with excessive application. Dermal vascular abnormalities may further enhance absorption.
Keywords: Topical anesthetics, Pediatric, Methemoglobin

63. Prolonged hypocalcemia refractory to calcium gluconate following ammonium bifluoride ingestion

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Background: Ammonium bifluoride is used in glass etching cream and has caused fatal ingestions secondary to fluoride toxicity. Fluoride binds avidly to calcium and magnesium and may result in hypocalcemia, hypomagnesemia, QT prolongation, and fatal cardiac dysrhythmias. We describe a case of prolonged hypocalcemia in a pediatric patient refractory to multiple boluses of calcium gluconate following the ingestion of ammonium bifluoride.
Case report: A 2 year-old female ingested "a couple of fingers full" of Armour Etch® glass etching cream (21–27% ammonium bifluoride) resulting in five episodes of vomiting over the next hour. One hour after ingestion, the child's father contacted Poison Control and was instructed to call 911 for emergent transportation. Upon arrival to the Emergency Department two hours after ingestion, the patient was asymptomatic and no longer vomiting. Laboratory evaluation revealed an ionized calcium of 1.21 mmol/L and the patient was administered calcium gluconate per Poison Center recommendations. The patient remained asymptomatic but developed multiple episodes of hypocalcemia with a nadir ionized calcium of 0.94 mmol/L 24 hours after the time of ingestion despite 5 repeat boluses of calcium gluconate (see Table 1).
Case discussion: Hydrofluoric acid is a weak acid that is absorbed passively from the stomach and small intestine. When ammonium bifluoride reacts with body fluids it forms hydrofluoric acid and fluoride ion. In a fasting state, peak absorption of hydrofluoric acid occurs in 30–60 minutes and the elimination half-life is 2.4–4.3 hours. Previous reports of hypocalcemia secondary to ammonium bifluoride ingestion report calcium nadirs within 6 hours of ingestion. In our case the nadir occurred 24 hours after ingestion despite 5 boluses of calcium gluconate. The difference may be secondary to absorption kinetics. Previous reports of ammonium bifluoride toxicity were secondary to the ingestion of liquid wheel cleaner. Our case involved the ingestion of ammonium bifluoride paste. The greater viscosity of the paste form may have prolonged the gastric absorption. As such, ammonium fluoride paste ingestions may require prolonged observation and treatment compared to other fluoride products.
Conclusions: Ingestion of ammonium bifluoride paste may result in prolonged hypocalcemia greater than 24 hours after exposure requiring the frequent administration of intravenous calcium gluconate.
Keywords: Ammonium bifluoride, Hypocalcemia, Pediatric

Table 1. Data for abstract 63.

Hours after ingestion	Serum ionized calcium (mmol/L)
2.25	1.21*
5.25	1.36
9.75	1.01*
12.50	1.00*
14.75	1.10*
15.25	1.24
18.25	0.98*
20.75	1.36
23.50	0.94*
26.00	1.36
28.00	1.32
30.00	1.33

*Indicates the administration of 0.186 mEq/kg of calcium gluconate.

64. Intrathecal insult with iothalamate meglumine

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Background: Toxicity from medications inappropriately administered intrathecally occurs rarely. We present an accidental exposure of an ionic contrast material infused intrathecally that was treated successfully with benzodiazepines and cerebral spinal fluid (CSF) drainage.
Case report: A 71-year-old male with history of spinal stenosis was undergoing a myelogram of his lumbar spine when 18 mL of iothalamate meglumine was accidentally injected intrathecally for contrast enhancement. Over the course of the following nine hours, the patient developed sinus tachycardia (134 beats per minute), elevated blood pressure (183/103 mmHg), and spasms/contractions beginning in his lower extremities. His mental status remained normal sparing mild progressive drowsiness. Physical exam noted stiffness and rigidity in the patient's lower extremities greater than upper extremities. Management consisted of high doses of benzodiazepines and lumbar drain placement. Reverse Trendelenburg position was maintained at greater than 30 degrees to facilitate gravitational forces and CSF flow through the lumbar drain. He was intubated for sedation and on day 2 was noted to have spasms in the extremities with stimulation and position changes but no seizures. The creatinine phosphokinase (CPK) rose to 2800 IU/L. He developed rapid atrial fibrillation, which converted back to normal sinus rhythm with amiodarone and diltiazem. CSF drainage was maintained at roughly 10–20 mL per hour over the course of 3 days. Daily head computed tomography images demonstrated decreasing CSF attenuation of radiocontrast material until no longer visible after 3 days. Sedation was discontinued and the patient was successfully extubated. He was later transferred to inpatient physical rehabilitation for mild generalized weakness.
Case discussion: Iothalamate meglumine is an ionic hyperosmolar radiocontrast material. Intrathecal administration of these agents has been associated with significant morbidity and high mortality due to neurotoxic reactions. The ionic nature of the contrast material causes the excitatory effects, yet the hyerosmolarity may also have inhibitory effects. This results in an ascending tonic-clonic seizure syndrome and can lead to fatal complications unless treated aggressively. CSF drainage or exchange has also been reported as a method of treatment.
Conclusions: Intrathecal medication errors occur infrequently but can have deleterious consequences. We present a patient with intrathecal administration of iothalamate meglumine that was aggressively treated with benzodiazepines, intubation, and CSF drainage preventing seizures and its associated complications.
Keywords: Iothalamate meglumine, Intrathecal, CSF drainage

65. Coagulopathy after accidental pediatric rivaroxaban ingestion

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Background: Rivaroxaban is an approved oral factor Xa inhibitor for the prophylaxis of deep vein thrombosis (DVT) in recent hip- or knee-arthroplasty patients. Routine measures of coagulation parameters prothrombin time (PT), international normalized ratio (INR), and partial thromboplastin time (PTT) are unnecessary, and rivaroxaban induced coagulopathy cannot be excluded by these routine measures of coagulation. There are no definitively effective reversal agents in cases of rivaroxaban toxicity. We have found no pediatric rivaroxaban exposures documented in the literature.
Case report: A 21 month-old 12 kg female presented to the emergency department approximately 2 hours after ingesting rivaroxaban. The patient's grandfather had recently undergone total hip arthroplasty and had been prescribed 35 tablets of 10 mg ribaroxaban. The patient was found playing with an open prescription bottle. One intact tablet was removed from the patient's mouth and 2 additional tablets were unaccounted for (20 mg or 1.67 mg/kg). The patient was asymptomatic upon arrival. She was given 1 gm/kg of activated charcoal PO. Initial CBC, BMP, and PTT were unremarkable. The patient's initial INR was 2.1, and she was admitted for observation. INR peaked at 3.5 about 12 hours after ingestion. Repeat INR measurements were 2.6 at 15 hours post-ingestion and 1.7 and at 20 hours post-ingestion. The patient remained asymptomatic without signs of bleeding and was subsequently discharged home without any additional coagulation studies.
Discussion: Routine coagulation studies are not recommended in patients taking rivaroxaban for DVT prophylaxis after recent hip- or knee-arthroplasty. Furthermore, data is lacking on pediatric exposures to rivaroxaban. We report a case of an asymptomatic 21 month-old patient who experienced an elevated INR after ingesting 2 10 mg tablets of rivaroxaban. The patient's INR may have had a higher peak if activated charcoal had not been administered approximately 2 hours after ingestion.
Conclusions: It may be reasonable to obtain routine coagulation studies in pediatric patients exposed to rivaroxaban. An elevation in these parameters may warrant admission and serial laboratory measurements until coagulation studies have normalized. Given the lack of definitively effective reversal agents, activated charcoal may benefit patients who seek medical care shortly after ingestion of rivaroxaban. Further clinical description and research is warranted.
Keywords: Anticoagulant, Pediatric, Activated charcoal

66. Transient toddler trance: Acute gamma butyrolactone toxicity in a toddler ingesting nail polish remover

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Background: γ-butyrolactone (GBL) is a lactone ring that is quickly metabolized to γ-hydroxybutyrate (GHB) by peripheral lactonases, with clinical effects similar to GHB. GBL is widely used as an industrial solvent, and in residential environments may be found in acetone-free nail polish removers. Its neurodepressant effects are well studied, with greater potency and more rapidly achieved peak effect than GHB.
Case report: The mother of a 2-year old boy contacted the Poison Center shortly after her son ingested 1 ounce of liquid nail polish remover to inquire if she should induce vomiting. On reassessing the child, she found him unresponsive. With emergency medical services (EMS) en route, she was advised not to induce vomiting. A first responder confirmed the unresponsiveness of the child, who he found with eyes half-open, with gurgling respirations. The nail polish remover was identified as "Once REMOVED." An internet search revealed that the product was known to contain GBL.

The child's initial vitals revealed a heart rate of 90 bpm and blood pressure of 100/67 mmHg. En route to the accepting emergency department (ED), EMS provided supportive cares including O_2 delivery. Upon arrival to the ED the child had regained consciousness and was awake and alert. He was observed in the ED for several hours and subsequently discharged home without permanent sequelae.
Discussion: GBL is more rapidly and completely orally absorbed than GHB, with dose-dependent plasma concentrations. Once REMOVED® and similar products (Boots Conditioning Nail Polish Remover Pads®, MAVALA Extra Mild Nail Polish Remover®) have previously been implicated as high-GBL concentration products (as high as 84% GBL in some cases); a recent, less potent reformulation of Once REMOVED® is recently described in the literature. In June of 2004, several bottles of Once REMOVED® were seized in a raid of a suspected clandestine GHB laboratory in Metairie, LA. High performance liquid chromatography revealed pure or near-pure GBL. Ever-lifetime abuse of GBL is common in some groups but less so in the general population; inadvertent toxicity such as this is rare.
Conclusion: Few substances cause such rapid onset of effective comatose state and respiratory depression as GHB, GBL, and the precursor compound 1,4-butanediol. Although availability of these toxins has been reduced, recreational use continues and products containing them remain available. Ingestion should be considered in children with a rapidly decreasing level of consciousness. Poison specialists and providers who care for poisoned children should be aware that GBL toxicity is a possible complication of nail polish ingestion.
Keywords: Pediatric, Ingestion, GBL

67. Energy drinks: The wrong kind of fuel

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Background: Energy drinks represent a growing portion of the consumer beverage market accounting for 5 to 6 billion dollars in sales annually. Packaging and volumes of energy drinks vary. Some are marketed in miniature sizes (e.g. 2 ounces, 60 milliliters [mL]) and are designed to be portable and consumed all at once. Similar packaging is employed for other consumer goods such as travel-sized personal care items (e.g. shampoo, conditioner) and motor vehicle products (e.g. fuel additives). The similarities in packaging can lead to unintentional ingestion of products that may cause toxicity. We report two cases of unintentional poisoning from products which were mistaken as energy drinks.
Case reports: Case 1: A 43 year old male presented to the emergency room (ER) after ingesting 9 mL of what he thought was a small energy drink. The product he drank was similarly packaged to

an energy drink but was actually a bottle of Rush™ liquid incense, a commonly abused sexual enhancement inhalant, containing isobutyl nitrite (95 to 100%). The patient admitted to purchasing the product at an adult entertainment store. The energy drink and the Rush™ were both packaged in bright colored miniature containers. After drinking the liquid incense at 2300 hours he realized his mistake, drank milk and spontaneously vomited. He presented to the ER and the Poison Center was contacted at 0058 hours. His blood pressure and heart rate were 98/58 and 135 respectively. On follow up the patient had become cyanotic with a methemoglobin level peaking at 23.6%. He was administered methylene blue 215 milligram (2 milligrams per kilogram) intravenously at 0120 hours. The patient recovered, was observed as an inpatient for a couple of hours and subsequently discharged well.

Case 2: A 46 year male presented to the ER complaining of nausea, vomiting and anxiety 40 minutes after ingesting 30 mL of a fuel additive called Eco Fuel Saver™ (65% ethanol, 25% benzyl alcohol) which he thought was an energy drink. The packaging of the fuel additive was brightly colored and contained 60 mL, which was similar to his energy drink. Blood work revealed an anion gap of 15. The patient was treated symptomatically with benzodiazepines, pantoprazole, and dimenhydrinate, and was discharged.

Discussion: Both cases presented were precipitated by incorrect identification and subsequent ingestion of a potentially toxic product due to similar product packaging to miniature energy drinks.

Conclusions: The wide scale popularity of energy drinks and their similarities in packaging with other potentially toxic products may expose consumers to unintentional poisoning.

Keywords: Energy drinks, Ingestion, Stimulant

68. Tiki trouble: Fulminant respiratory failure and cardiac necrosis in a 22-month-old ingesting Tiki torch fuel

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Background: Hydrocarbon ingestions may progress rapidly to severe pulmonary damage and death with aspiration of even trivial amounts. Severe hydrocarbon toxicity is characterized by marked respiratory symptoms including coughing, cyanosis, respiratory distress, and vomiting.

Case report: A 22-month-old girl aspirated an unknown amount of Tiki torch fuel, a source of long chain aliphatic hydrocarbons. Immediately she began to cough, and was taken to a local emergency department (ED). There she was found to be hypotensive and in significant respiratory distress. Frothy, bloody oral secretions were noted. Initial labs were as follows:

Potassium: 8 mEq/L
Glucose: 700 mg/dL
Lactate: 12 mmol/dL
Venous blood gas:
pH: Acidotic, pH "not recordable"
pCO_2: 248 mm
pO_2: 33 mm
Solvent and volatile screens: negative

Shortly after arrival the child turned dusky and her eyes "rolled back into her head." Chest radiograph revealed complete opacification of the right lung field. She was suctioned for frothy, bloody secretions, emergently intubated and transferred to a higher level of care.

Arriving to the receiving facility, the patient had an immediate cardiac arrest. Pediatric advanced life support was undertaken for 1 hour. Oxygen saturation never surpassed 50%, and extreme hypercapnia (pCO_2 240 mm) was noted. Despite resuscitation efforts, the patient expired.

Post-mortem microscopic exam revealed extensive alveolar wall damage, neutrophiic and eosinophilic infiltrates, acute hemorrhage and extensive edema throughout. Cardiac microscopy revealed focal lymphocytic infiltrates, scant histiocytes and eosinophils, and cardiac myocyte necrosis.

Case discussion: This case illustrates an extreme case of aliphatic hydrocarbon toxicity from an unintentional ingestion in a child. Hydrocarbon aspiration is not known to cause myositis, although arrhythmogenesis secondary to myocardial catecholamine sensitization is hypothesized. A subset of hydrocarbon ingestions will require observation and supportive cares for subacute toxicity related to ingestion and delayed pneumonitis. A small subset develops fulminant respiratory symptoms, respiratory failure and death. Our case demonstrates the acute course of aspirated aliphatic hydrocarbon toxicity, with rapid deterioration and death despite maximal medical management.

Conclusion: In patients suspected of hydrocarbon aspiration, acute severe respiratory symptoms bode poorly and portend rapid deterioration. Early aggressive management is mandatory in these patients, who may expire despite aggressive efforts.

Keywords: Hydrocarbon, Pediatric, Poison center

69. Severe hyperthermia following misoprostol for postpartum hemorrhage

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Background: Misoprostol is a prostaglandin E1 derivative commonly used for its uterotonic effects in postpartum hemorrhage (PPH). Hyperthermia is a known side effect but reports of severe hyperthermia and autonomic instability are scant. We report a case of acute transient hyperthermia, delirium and tachycardia attributed to misoprostol toxicity sustained during an urban home birth.

Case: A 28-year-old woman presented to Labor and Delivery immediately following buccal administration of 1600 mg misoprostol for PPH following home delivery. Her only home medicine was Armour® natural thyroid replacement. On arrival she was alert and oriented and in no acute distress with a blood pressure of 140/80 mmHg and heart rate of 150 bpm. No initial temperature was recorded. Within one hour she became confused and agitated and was found to be oriented to self only. Her heart rate was 210 bpm, blood pressure 141/99 mmHg and oral temperature 40.7°C. She then received intravenous saline, empiric broad-spectrum antibiotics, droperidol for agitation and magnesium sulfate for eclampsia prophylaxis. She had gradual decline in temperature and heart rate with normothermia and normal heart rate achieved 5 hours after initial hyperthermia. Her mental status returned to baseline over 24 hours. Evaluation included electrocardiogram (ECG), chest x-ray,

coagulation and hemolysis studies, complete blood count, troponin I, creatinine kinase (CK), thyroid stimulating hormone and blood cultures. These data were negative except for the following: ECG with sinus tachycardia, white blood cell count of 33.1 k/cmm, hemoglobin 10.5 g/dL, troponin I of 0.779 ng/mL and CK of 3082 IU/L.

Discussion: Misoprostol is well suited for resource-poor settings such as rural locations or home births given its potent uterotonic effects and stability at ambient temperatures. Its pyrogenic effects are due to prostaglandin-mediated modulation of the hypothalamic set point. Reports of temperatures over 40°C are limited to scattered case reports and one series of 58 Ecuadorian women. The clinical course appears to be acute, transient and self-limited. This case is noteworthy for the remarkable tachycardia and delirium as well as the initial setting of an urban home birth. This is also the first report of myocardial ischemia associated with misoprostol toxicity, and likely represents myocardial supply-demand mismatch of oxygen secondary to tachycardia.

Conclusions: Clinicians should be familiar with the potential for dramatic thermoregulatory, autonomic and central nervous system effects of misoprostol toxicity in the immediate postpartum period.

Keywords: Misoprostol, Hyperthermia, Postpartum hemorrhage

70. Allopurinol overdose and associated acute renal failure

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Background: Allopurinol is a common therapy for gout. No significant effects from allopurinol overdose (OD) have been previously reported. We report an unintentional OD of allopurinol with associated hemorrhagic gastroenteritis and acute renal failure (ARF).

Case report: A 58 year old male travelled to Nigeria. His daily medications were allopurinol, furosemide, carvedilol, simvastatin, colchicine, clopidogrel, and aspirin. He normally organized his daily medications into a pill bottle. In addition he brought stock bottles due to his prolonged stay in Nigeria. Three days prior to hospital admission in the Untied States (US), he had confused his stock bottle of allopurinol (100 mg) with his daily medication bottle and ingested approximately 40 tablets. On realizing this, he sought medical treatment in Nigeria, and underwent a form of gastric lavage. He returned to the US for further treatment. In flight he reported bloody stools, lower chest pain (CP) and emesis.

Upon arrival in the Emergency Department in the US, he complained of CP and rectal bleeding. His physical exam was significant for bilateral chest wall tenderness and heme positive stool. Laboratory findings were significant for creatinine protein kinase of 552 U/l, Blood urea nitrogen of 43 mg/dl and creatinine of 3.6 mg/dl (Baseline 1.5–1.9). He was fluid resuscitated and admitted to the medical service. An allopurinol concentration on admission was undetectable and an oxypurinol level was 21 mcg/ml.

During hospitalization he received intravenous hydration and his renal function improved. Upper endoscopy revealed diffuse gastritis. Renal ultrasound showed evidence of medical renal disease and non-specific cysts. He was discharged on hospital day 4.

Discussions: Allopurinol competitively inhibits xanthine oxidase, which is responsible for the pathway of hypoxanthine to xanthine and then uric acid. Its metabolite oxypurinol is a non-competitive inhibitor of xanthine oxidase. The half-life of allopurinol is approximately 1–2 hours and 18–30 hours for oxypurinol. This is prolonged in kidney disease and in patients on furosemide. A number of cases report ARF from allopurinol hypersensitivity syndrome.

Conclusions: To our knowledge no studies correlate allopurinol OD with ARF. The secondary effect of bloody gastroenteritis has not been reported in allopurinol OD. This is a unique case of allopurinol OD with both renal and gastrointestinal consequences.

Keywords: Allopurinol, Overdose, Renal toxicity

71. Atypical antipsychotic exposures in young children

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Introduction: In 2010 there were over 3,000 exposures to atypical antipsychotics in children < 6 years reported to poison centers. Most published studies in children focus on one drug or involve a relatively small number of cases. A study comparing atypical antipsychotic exposures in young children using a single database would provide important information concerning

Table 1. Results for abstract 71.

	ARIP (N = 43)	OLAN (N = 20)	QUET (N = 33)	RISP (N = 38)	ZIPR (N = 11)	Total (%)
Disposition +						
Treated/Released ED	31 (72)	8 (40)	30 (90)	27 (71)	8 (73)	104 (71)
Admit Critical Care	1 (2)	5 (25)	1 (3)	2 (5)	1 (9)	10 (7)
Admit Non-Critical	11 (26)	5 (25)	2 (7)	7 (19)	2 (18)	27 (19)
Lost to Follow-up	0 (0)	2 (10)	0 (0)	2 (5)	0 (0)	4 (3)
Disposition Total						**145 (100)**
Outcome + +						
No Effect/Expect No Effect	14 (33)	5 (25)	17 (51)	14 (37)	6 (55)	56 (41)
Minor Effect	18 (42)	7 (35)	8 (24)	15 (39)	4 (36)	52 (38)
Moderate Effect	8 (18)	4 (20)	1 (3)	1 (3)	0 (0)	14 (10)
Minimal Effects Possible	1 (2)	3 (15)	7 (21)	5 (13)	0 (0)	16 (11)
Outcome Total*						**138 (100)**

+(comparing Treated/Released with Admission) p = 0.011; + +(comparing no effect/expect no effect with other outcomes) p = 0.239.
*7 cases with unrelated-probably not responsible were excluded.

relative toxicity. The purpose of this study is to evaluate atypical antipsychotic exposures in children < 6 years of age reported to a regional poison center and to compare toxicity, treatments required and outcomes.

Methods: A retrospective study of poison center data on atypical antipsychotic exposures in children under 6 years of age was performed. The study included cases from 2005 to 2011 involving aripiprazole (ARIP), olanzapine (OLAN), quetiapine (QUET), risperidone (RISP) or ziprasidone (ZIPR) treated in a healthcare facility (HCF). Only single substance exposures (no co-ingestants) were evaluated. Disposition and coded medical outcomes were compared.

Results: Of 254 atypical antipsychotic cases in children, 145 (57%) were managed in an HCF (olanzapine 74.1%, ziprasidone 73.3%; aripiprazole 60.6%, quetiapine 55.9%, risperidone 46.3%) and these cases comprised the study sample. The majority were treated in the emergency department and released (see Table 1). There were no major effects or deaths; 10% experienced moderate toxicity. Treatments included activated charcoal in 54 (37%) and antihistamines in 8 (6%) cases. One exposure to olanzapine required intubation.

Conclusions: Olanzapine cases were most likely to be admitted while quetiapine were most often treated/released from emergency department (ED). Coded medical outcomes were similar.

Keywords: Antipsychotic, Pediatric, Overdose

72. Compounding error leads to clonidine toxicity associated with hyperglycemia

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Background: Clonidine (C) is a centrally acting alpha 2-adrenergic agonist that is used in the treatment of hypertension, attention deficit hyperactivity disorder, and opioid withdrawal. Following overdose, CNS and respiratory depression, miosis, bradycardia, and hypotension may be observed. We present the case of a child who developed lethargy, transient apnea, bradycardia, and hyperglycemia after the administration of what was thought to be a therapeutic dose of an improperly compounded C suspension.

Case report: A 23 month-old female with a past medical history significant for autism presented to the emergency department for evaluation of lethargy and apnea. Her symptoms began 10 minutes after receiving her normal dose of 3 mL of C, her only prescribed medication, from a newly filled bottle. After arrival, she had intermittent episodes of apnea; definitive airway management was contemplated but ultimately deferred. Her vital signs at presentation were notable for a temperature of 36.2°C, pulse of 79 bpm, blood pressure of 129/66 mm Hg, respiratory rate of 24 bpm, and oxygen saturation of 95% on room air. The only notable examination finding was bradycardia. There was no history of antecedent infectious symptoms or physical evidence of trauma. Her laboratory evaluation revealed a WBC of 27.9 K/uL (70.6% neutrophils) and platelet count of 540 (K/uL). Her basic metabolic panel was notable for a glucose of 323 mg/dL, bicarbonate of 13.2 mmol/L, and an associated anion gap of 18.8. The urine analysis was notable for glucosuria but was without ketonuria or evidence of concomitant infection. Her CXR was normal. She received 0.1 mg/kg of naloxone with no observed clinical change. An aliquot of her C suspension was sent for quantification, the intended concentration was 0.5 mg/5 mL and the measured concentration was 75 mg/5 mL. Her clinical condition improved and she was discharged on hospital day #3.

Case discussion: Due to the increasing use of C in the pediatric population and available liquid preparations, significant toxic effects following inadvertent ingestions may occur in this population. Moreover, compounding errors, as highlighted by this case, may result in toxicity. The observed laboratory aberrations are noteworthy; of relevance, rat studies have demonstrated a dose-dependent hyperglycemic effect in rats receiving C. This child ultimately did well with supportive cares.

Conclusions: Healthcare providers should be cognizant of the potential for compounding errors when toxic effects are noted in children taking liquid formulations of medications therapeutically, and hyperglycemia may be observed in children presenting with clonidine toxicity.

Keywords: Clonidine, Pediatric, Adverse drug event

73. A retrospective review of supratherapeutic ADHD medication ingestions in pediatric patients

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Objective: Approximately 3% of American children receive attention deficit hyperactivity disorder (ADHD) medications, including stimulants, alpha 2 agonists, and antidepressants. Thousands of patients present to health care facilities (HCF) annually for supratherapeutic consumption of ADHD medications. The purpose of this study is to better understand the characteristics of supratherapeutic ADHD medication exposures in pediatric patients.

Methods: For this study, we queried the electronic database of a statewide poison system - for cases from Jan 2006 to Dec 2010, for patients 18 years old and under, using the following search terms: methylphenidate, dexmethylphenidate, amphetamine, lisdexamfetamine, atomaxetine, clonidine, and guanfacine. An Excel spreadsheet was used to record the following variables: age, sex, substances ingested, sustained release (SR) preparation or not, dose of exposure, co-ingestants, where the ingestion occurred, reason for exposure (taking additional dose, given additional dose, taking sibling's meds, dispensing error), symptoms, duration of symptoms, HCF evaluation, admit vs discharge, length of hospitalization, treatment, and number of follow up.

Results: 301 cases of pediatric patients who consumed supratherapeutic amounts of ADHD medications were reported. The most common age of ingestions were 6 to 10 years (N = 26–35). The top 3 reported ADHD substances ingested were methylphenidate −28 (42.5%), amphetamine/dextroamphetamine −55 (18.3%), and clonidine −52 (17.3%). 161 were SR formulations. The dose ranged from 0.5 to 10 tablets. Co-ingestion was reported in 78 patients. The ingestion occurred at home in 298 cases and school in 3 cases. Reason for exposure included taking additional dose −103 (34.2%), given additional dose −145 (15%), taking sibling's meds −28 (9.3%), dispensing error −25 (8.3%). Symptoms were

recorded in 92 (30.6%) patients with the top 3 symptoms: lethargy −39 (13%), tachycardia −21 (7%), hypotension −19 (6.3%). 120 patients (40%) were evaluated in HCF. Of these, 29 were admitted for 1 day or less. Alpha agonists result in more HCF visits and symptoms. Treatment included AC −28 (9.3%), IV fluid −9 (3%), and benzodiazepine −3 (1%).
Conclusions: The most commonly reported ADHD medications taken were methylphenidate and amphetamine/dextroamphetamine. Thirty percent of patients had reported symptoms. Alpha 2 agonists result in more HCF visits and symptoms. Most pediatric patients with supratherapeutic ingestions of ADHD, excluding alpha 2 agonists, do not require HCF visits.
Keywords: Ingestion, Pediatric, ADHD medications

74. Management of unintentional methanol ingestions; kids aren't little adults?

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Background: Methanol is not uncommonly ingested by pediatric patients. These unintentional exposures may result in utilization of antidotal therapy, admission, and transfer to higher levels of care. The aim of this study is to characterize and compare unintentional methanol ingestion in young patients vs. older pediatric and adult patients.
Methods: Utilizing Crystal Reports (Version 11.0), all methanol exposures reported to the Illinois Poison Center over a 10 year period (2002–2011) were retrospectively searched. Two groups of patients with documented methanol levels met inclusion criteria: Patients ≤6 years of age and patients >6 years of age. Only unintentional ingestions were queried for the comparison.
Results: Twenty-four patients ≤6 years of age met inclusion criteria. Age range was 14 months–5 years with an average of 28 months (54% female). Methanol concentrations were undetectable in 20 (83%) patients. The highest concentration obtained was 24 mg/dL. This patient had a second level of 16 mg/dL after treatment with fomepizole. Three other patients had detectable levels (5, 6, and 17 mg/dL). No patient was treated with ethanol or hemodialysis, however, 14 patients (58%) were treated with fomepizole. Twelve patients were admitted to the hospital for a minimum of 24 hours. By comparison, 36 patients >6 years of age met inclusion criteria. Age range was 9–68 years with an average of 37 years (69% male). Methanol concentrations were undetectable in 23 (64%) patients. The highest concentration obtained was 150 mg/dL. Three other patients had levels of 23, 35, and 47 mg/dL, with the remaining measuring between 5 and 10 mg/dL. Two patients were treated with ethanol, 21 patients (58%) were treated with fomepizole and the patient with a level of 150 mg/dL was managed with hemodialysis. Eleven patients were admitted to the hospital for at least 24 hours.
Conclusions: Unintentional methanol poisoning can be a difficult diagnostic and treatment dilemma. For pediatric methanol exposures under the age of 6, 83% of cases had undetectable levels; those with detectable levels typically had insignificant concentrations unlikely to cause harm. Comparatively, several unintentional exposures in older children and adults, resulted in elevated methanol concentrations requiring focused therapy. Given these results, it may be reasonable to observe most children ≤6 years of age while waiting for methanol concentrations or following serial chemistries without administering fomepizole empirically.
Keywords: Methanol, Pediatric, Laboratory

75. Do unintentional ethylene glycol exposures require intentional management measures?

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Background: Ethylene glycol (EG) is not uncommonly ingested unintentionally. While many exposures are merely a "lick or taste", others may include larger ingestions because of the color and palatability of many products. Cases of exposure to EG being stored in inappropriate containers may also exacerbate this problem. The aim of this study is to characterize and compare unintentional EG ingestions in patients ≤6 years of age to those >6 years of age.
Methods: Utilizing Crystal Reports (Verson 11.0), all EG cases reported to the Illinois Poison Center over a 10 year period (2002–2011) were retrospectively searched. Two groups of patients with documented EG levels met inclusion criteria: Patients ≤6 years of age and patients >6 years of age. Only unintentional ingestions were queried for the comparison.
Results: Eight patients ≤6 years of age met inclusion criteria. Age range was 11 months–5 years with an average of 24 months (50% female). EG concentrations were undetectable in 6 (75%) patients. The highest concentration obtained was 9.9 mg/dL. Only 1 other patient had a detectable concentration (2.5 mg/dL). Two patients were treated with ethanol (one of whom also received a dose of fomepizole), 3 patients were administered fomepizole alone, and none received hemodialysis. Three patients (38%) were transferred to a higher level of care for admission. By comparison, 6 patients >6 years of age met inclusion criteria. Age range was 17–45 years with an average of 30 years (100% male). EG concentrations were undetectable in 3 (50%) patients. The highest concentration obtained was 30 mg/dL. Two other patients had levels of 14.8 and 3.8 mg/dL, with the remaining concentrations measured being undetectable. Two patients were treated with ethanol, 3 patients (50%) with fomepizole, and the patient with a concentration of 14.8 mg/dL was acidemic (HCO_3 7 mEq/L) and received hemodialysis. One patient was admitted to the hospital for at least 24 hours and no patient required transfer to a higher level of care.
Conclusions: Interestingly both groups were equally benign overall. A greater number of younger patients had concentrations that were undetectable (75% vs. 50%). Additionally, the administration of alcohol dehydrogenase (ADH) inhibitors was also greater in the older group (75% vs. 83%). Out of the entire 14 patients managed, 71% received ADH inhibition (with 4 administrations of ethanol). One patient was an outlier in that he presented late with acidemia from an alleged unintentional exposure. While these data are limited, the requirement of antidotal therapy and need for hospital admission is exceedingly rare in these patients. A prospective study would help further characterize this dilemma.
Keywords: Ethylene glycol, Pediatric, Laboratory

76. Childrens' acetaminophen dosing: A new set of problems

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Background: Medication errors related to the use of pediatric formulations of acetaminophen (apap) often involve misunderstanding the dosing directions and calculation errors. Dosing errors were primarily due to the availability of two pediatric formulations, the concentrated infant's drops (80 mg/0.8 mL) and the children's suspension (160 mg/5 mL). To address these concerns, in June 2011 the FDA considered, and industry voluntarily embraced, creating a single dosage formulation that can be given to both infants and children. However, the public message announcing this formulation change may not have been optimal, leading to some of the confusion. We present a review of calls to our poison center related to pediatric apap dosing before and after the introduction of the new formulation.
Methods: We reviewed all of our Center's Toxicall data from January to December 2011 involving a therapeutic error related to apap in children under 6 years. The Managing Director and Educator identified the cause of the error and classified the findings using a blinded data abstraction sheet. Given the limited time since introduction, data from the latter time period is limited.
Results: A total of 212 cases were reviewed. The majority of calls (89%) came from parents or caregivers and 11% from the medical provider. Almost all exposures occurred in the home and were managed on site (91%). Of the total sample of 212 cases involving acetaminophen, 30% were coded as incorrect dose; 18% inadvertently took/gave medicine twice; 10% incorrect formulation given; 12% confused units of measure; 8% dispensing cup error and 5% involved doses given/taken too close together. Products were coded as original children's 160 mg/5 mL (47%), concentrated infant's 80 mg/0.8 mL (41%), or new formulation children's 160 mg/5 mL (13%). Qualitative analysis of the 26 calls after the formulation change revealed dosing concerns. Parents felt they were giving too much apap based on the less concentrated volume and pediatricians' made recommendations based on the original infant's formulation.
Conclusions: Since its release less one year ago, confusion about the new children's apap product dosage and instructions remain an issue for both parents and providers. An integrated and enhanced effort is needed to raise awareness and provide education to both groups regarding the proper use of the new formulation to reduce medication errors during this transition period.
Keywords: Acetaminophen (paracetamol), Medication errors, Dosing

77. Ten-fold enoxoparin overdoses: Two post-operative pediatric patients

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Background: Enoxaparin is a Low molecular weight heparin (LMWH) that is commonly used postoperatively in pediatrics for the prophylaxis of thromboembolic events. We report the first cases of an iatrogenic ten-fold pediatric dosing error of enoxaparin, administered twice to two children over 24 hours without significant bleeding complications.
Case reports: Patient 1, a 7 month-old male, and patient 2, a 22 month-old male, on a post-operative recovery ward, were receiving enoxaparin (Lovenox) for postsurgical venous thromboembolism prophylaxis. Each was given a tenfold dosing errors dose twice before pharmacy recognized the error. Patient 1 was accidently given 36 mg of enoxaparin subcutaneously instead of 3.6 mg and patient 2 was accidently given 47 mg instead of 4.7 mg. Both children remained asymptomatic and without evidence of bleeding at the time the error was reported. Laboratory coagulopathy evaluation at the time of error recognition was unremarkable for both patient 1 (prothrombin time = 13.7, activated partial thromboplastin time = 27.3, international normalized ratio = 1.0) and patient 2 (PT = 15.9, aPTT = 42.7, INR = 1.2). Thrombocytosis (platelets = 740 K) was identified in patient 2. Enoxaparin was discontinued for 24 hours in both cases. Subsequent coagulation studies remained unremarkable and both patients remained asymptomatic.
Case discussion: Enoxaparin is a low molecular weight heparin used for the prophylaxis and/or treatment of thromboembolic events. Although LMWH such as enoxaparin has been proven to be efficacious and safe in prophylaxis of thromboembolic events in children, overdose of LMWH has resulted in bleeding complications. We report a ten-fold dosing error that occurred in two children without any bleeding complications.
Conclusions: Despite the recognized adverse effects of LMWH overdose, we report two ten-fold dosing errors with enoxaparin without significant bleeding complications.
Keywords: Adverse drug event, Anticoagulant, Pediatric

78. Acetaminophen mis-dosing in children younger than 7 years of age

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Background: Fever is one of the most common reasons for pediatric emergency room (ER) visits. Acetaminophen is widely available over-the-counter for the treatment of fever. Pharmacodynamic efficacy studies suggests that doses between 10 to 15 mg/kg are needed for optimal antipyretic efficacy when given at 4 hours intervals. Studies have shown that 66% of patients presenting to emergency departments with fever had received inaccurate doses of acetaminophen. There is little information about the proportion of children at risk of chronic supra-therapeutic acetaminophen toxicity in this age group (> 75 mg/kg/day). In clinical practice, acetaminophen doses are based on the child's weight measured during the visit. Children weights differ across age groups. We describe local practices to develop recommendations addressing home acetaminophen dosing in children.
Objectives: To identify the proportion of patients and factors contributing to inaccurate doses of acetaminophen in children younger than 7 years.
Methods: Cross-sectional questionnaire of 230 patients, under 7 years, with Canadian Triage and Acuity Scale category 4 or 5 presenting to the emergency department with history of fever, having received acetaminophen at home.

Results: 41% of children did not receive an optimal dose of acetaminophen. Acetaminophen doses of more than 15 mg/kg were noted in 15% of our population. Acetaminophen doses less than 15 mg/kg were reported in 30% of age-base and in 12% of weight-base groups. Doses more than 15 mg/kg were reported in 19% of age-base and in 12% of weight-base groups. Inadequate doses were more often seen in children aged 5 to 7 years (52%) then in children aged 1 to 3 years (36%). Factors positively associated with accurate dosing was presence of siblings and higher parental education.
Conclusion: In our survey, 41% of children coming to our ER for fever received inappropriate acetaminophen doses as per our gold standard of 15 mg/kg/dose. The main factor found to be contributing to this is age-based calculation. Even with weight-based dosing, many are receiving inaccurate amount of acetaminophen compared to our gold standard. Although in this study we did not address specifically chronic supra-therapeutic ingestion of acetaminophen, our results suggest a potential risk of toxicity in this subset of patients. Despite limitations, we think that this issue is multifactorial but better parental education regarding appropriate acetaminophen dosing and regular kids weighing would contribute greatly to better parental satisfaction regarding acetaminophen effect at home for the treatment of fever in their children and reduce risk of supra-therapeutic dosing if given for many days.
Keywords: Acetaminophen (paracetamol), Children, Fever

79. Pediatric zinc chloride ingestion with metabolic acidosis and elevated zinc levels

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Background: Pediatric ingestions of zinc chloride (ZnCl) containing products are rarely reported. Ingestions are characterized by repeated vomiting, metabolic acidosis and corrosive gastrointestinal tract injury. Care is usually supportive. This case report documents the effects of a ZnCl ingestion including zinc and copper levels.
Case report: A 30-month old boy ingested an unknown amount of soldering flux (<60 ml) containing ZnCl. He immediately began vomiting and was taken to a local emergency department (ED) for evaluation. In the ED he had 10 + episodes of vomiting and was noted to have a venous blood pH = 7.28, Na = 139 mmol/L, K = 3.2 mmol/L, Cl = 102 mmol/L, bicarb = 14 mmol/L, glucose = 184 mg/dl, lactate = 2 mmol/L and an anion gap = 23 mmol/L. The blood zinc level was >800 ug/dl (range = 60–120). He was then transferred to a pediatric hospital for further evaluation and care. In the second ED, he was alert and awake with normal vital signs. His physical exam was unremarkable with no visible oral burns. He received ondansetron 2 mg IV and 20 ml/kg of normal saline IV. Laboratory testing: Na = 137 mmol/L, K = 4 mmol/L, Cl = 110 mmol/L, Bicarb = 15 mmol/L, and an anion gap = 12 mmol/L. At 5 hours after the ingestion, a venous blood sample showed a lactate = 1 mmol/L, pH = 7.31 and pCO_2 = 31 mmHg. The zinc level had dropped to 576 ug/dl. Six hours post ingestion, an esophogastroduodenoscopy (EGD) was performed. The examination was notable for diffuse gastropathy with exudate, gross edema and friability. The patient was admitted for observation and started on pantoprazole and sulcralfate. He was observed overnight and discharged the next day without further symptoms. Twenty-three days after the exposure, the patient had an upper gastrointestinal barium study performed which was normal. Normal blood levels of zinc, 99 ug/ml (range 60–120), copper, 130 ug/dl (range = 75–153) and magnesium, 2.2 mg/dl (range = 1.6–2.3) were also determined at this time. Follow up at 42 days post ingestion showed an asymptomatic child with no obvious sequelae.
Case discussion: ZnCl ingestions in children can produce significant injury and toxicity including nausea, vomiting, and gastric damage with strictures. Additional complications include hyperglycemia, metabolic acidosis and systemic zinc toxicity. This patient had many of these clinical symptoms, notably a metabolic acidosis, elevated zinc levels and gastric damage. Chronic but not acute zinc exposure can produce lower copper levels as this case demonstrates.
Conclusions: Any pediatric ZnCl ingestion should be carefully evaluated with endoscopy, and monitoring for the effects of elevated zinc blood levels and metabolic acidosis.
Keywords: Pediatric, Caustic, Acidosis

80. Adverse opioid analgesic exposures among the elderly

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Background: Opioid analgesic use, and its consequences, is increasing in the US. At the same time, the elderly population in the US is increasing. However, there is little information on adverse exposures to opioid analgesics among the elderly. This study describes opioid analgesic exposures among the elderly reported to poison centers.
Methods: Opioid analgesic exposures among patients age 60 years or more reported to a statewide poison center system during 2000–2011 were identified. Exposures involving additional substances and those not followed to a final outcome were included. The distribution of exposures was determined for various demographic and clinical factors.
Results: There were 4126 total exposures. The annual number of exposures increased 201% from 167 in 2000 to 502 in 2011. The most frequently reported opioids were hydrocodone (49.1%), tramadol (14.0%), propoxyphene (13.8%), oxycodone (8.3%), morphine (6.1%), codeine (5.0%), and methadone (4.0%). The patients were 53.0% in their 60 s, 29.5% in their 70 s, 14.9% in their 80s, and 2.9% in their 90s or older. The patients were 65.7% female. Ingestions were involved in 97.7% of the exposures. The circumstances of the exposures were 43.9% therapeutic error, 21.2% suspected attempted suicide, 9.6% intentional misuse or abuse, 9.2% adverse drug reaction, and 16.0% all other/unknown. The management site was 41.8% already at/en route to a healthcare facility, 45.7% managed on site, 11.5% referred to a healthcare facility, and 0.9% other/unknown. The medical outcome was 17.6% no effect, 19.0% minor effect, 14.4% moderate effect, 4.4% major effect. 0.7% death, 3.2% not followed (nontoxic), 28.5% not followed (minimal effects), 9.0% unable to follow (potentially toxic), and 3.2% unrelated effect.
Conclusions: Adverse opioid analgesic exposures among the elderly reported to this poison center system tripled during this time period. The highest proportion of the exposures involved hydrocodone and occurred by ingestion. The majority of the patients were in their 60s and female. The highest proportion of exposures was therapeutic

error, and only a fraction of the exposures were intentional misuse or abuse. Almost half were managed on site and the majority were known or expected to not result in a serious outcome. Although most of these exposures can be managed at home and probably will not result in serious effects, actions might need to be taken to attempt to reduce the number of these exposures that occur.

Keywords: Opioid, Poison center, Elderly

81. Combined synthetic cannabinoid-synthetic cathinone exposures reported to poison centers

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Background: Much has been published on the new designer drugs synthetic cannabinoids and synthetic cathinones. No literature has examined the outcomes when the two drugs are used in combination. The purpose of this study is to describe patients who have been exposed to both agents.

Methods: Synthetic cannabinoid-synthetic cathinone combination exposures reported to a statewide poison center system during 2010–2011 were identified. The distribution of exposures was determined for various demographic and clinical factors.

Results: Fourteen combination exposures were identified, representing 1.3% of total synthetic cannabinoid and 3.9% of total synthetic cathinone exposures. Two of the exposures also involved alcohol; the remaining cases reported no additional substances. The patients were 85.7% male and had a mean age of 24 years (range 16–46 years). The route of the exposure was inhalation and ingestion (57.1%), inhalation alone (28.6%), and ingestion alone (14.3%). All of the patients were at or en route to a healthcare facility when the poison center was contacted. The medical outcome was 7.1% minor effect, 85.7% moderate effect, and 7.1% major effect (compared to 13.7%, 43.7%, & 7.9% for Synth Cathinones alone and 24.4%, 41.4%, & 4% for synth cannabinoids alone). The most commonly reported adverse clinical effects were agitation (71.4%, vs. 37% with Synth Cathinones or 21.8% with Synth Cannabinoids, alone), tachycardia (35.7%), hallucinations (28.6%), hypertension (21.4%), confusion (14.3%), and mydriasis (14.3%, vs. 1.7% with Synth Cathinones or 2.8% with Synth Cannabinoids, alone). The most frequently reported treatments were administration of IV fluids (78.6%), benzodiazepines (57.1%), and oxygen (14.3%); similar to the treatment used for either agent alone.

Conclusions: Synthetic cannabinoid-synthetic cathinone combination exposures accounted for a small fraction of all exposures to these drugs reported to this poison center system. Compared to either agent alone, the combination resulted in more patients with moderate effects, agitation, and mydriasis. This finding may reflect synergistic effects. A limitation of the study was that the exposures were not verified by toxicological tests.

Keywords: Cannabinoid, Synthetic, Drug of abuse, Designer drug

82. Clonidine for cuties: The quick fix in neonatal abstinence syndrome

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Background: Maternal drug abuse results in serious health consequences for the fetus, including neonatal abstinence syndrome (NAS). Poor feeding, autonomic instability and seizures make NAS a challenging condition; narcotic therapy complicates it further with CNS and respiratory depression and the need for long methadone tapers. Clonidine, an alpha agonist, has been used in young child and adult opiate withdrawal, but little data exists on its use as a primary agent to prevent NAS.

Methods: A retrospective chart review was conducted on neonates with NAS at Evanston Hospital (EH) of the NorthShore University HealthSystem from August 2007–July 2011. The EH neonatal intensive care unit offers a NAS order set with clonidine (0.5–1 mcg/kg every 6 hours) as the primary medication. The order set lists as-needed lorazepam and phenobarbital as adjuncts. Methadone is given on an individual basis. Pharmacotherapy is provided with modified Finnegan scores of 8 or above. Thirty-one charts were reviewed; 2 were excluded due to concomitant methadone and clonidine. From the remaining charts, demographic data, dose and duration of therapy, modified Finnegan scores, and adjunct therapy were recorded. A statistician used Chi-square testing, Fisher's exact testing and Wilcoxon two sample testing for statistical analysis.

Results: Out of 29 patients, 21 were given clonidine and 8 were given methadone by physician choice. None had seizures or adverse drug events. Gender, average gestational age in days (C: 33.43 vs. M: 29.25), and average weight in kg (C: 2.32 vs. M: 2.09) were not statistically different between groups ($p > 0.05$). The duration of treatment in days was shorter with clonidine than methadone (C: 10.38 vs. M: 34.63, $p = 0.0008$). The duration of adjunct therapy in days was shorter in the clonidine group (C: 4.5 vs. M: 12, $p = 0.804$). Also, clonidine use was associated with successful extubation rather than methadone (C: 83.3% vs. M: 16.7%, $p = 0.012$). The minimum and maximum abstinence scores were higher with clonidine use (C: 2.95, 9.05 vs. M: 0.94, 6.26, $p = 0.0484$, 0.0426), yet efficacy of both treatments is evident by the virtual normalization of modified Finnegan scores by the end of treatment (C: 3.64, M: 2.44, $p = 0.270$).

Conclusions: This study demonstrates that clonidine can be safely and successfully used as a primary agent without a narcotic in the prevention of NAS. Clonidine required shorter treatment duration, as well as shorter adjunct therapy duration than methadone. Home therapy was avoided with the use of clonidine. With its use, neonates experienced a similar 60% drop in modified Finnegan scores as the methadone group. Larger studies are needed to determine whether these results are reproducible in other populations.

Keywords: Pediatric, Neonatal Abstinence Syndrome, Clonidine

83. Passive multi-state surveillance for levamisole-associated neutropenia in cocaine or heroin users

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Background/Objectives: Numerous reports describe patients with agranulocytosis after using levamisole-contaminated cocaine (LCC). We conducted surveillance to characterize the demographic, clinical and epidemiologic features of levamisole-associated neutropenia in cocaine or heroin users.
Method: Multi-state, passive surveillance was conducted between October 15, 2009 and May 31, 2010. State health departments (HDs) were recruited for participation when CDC was notified of suspected cases by a clinician, a HD official or from a poison center. A probable case was defined as an adult (>18 years) with neutropenia (absolute neutrophil count <1000 cells/mL or white blood cell count <2000 cells/mL) and either self-reported cocaine or heroin use in the past month, or detection of cocaine in urine. A confirmed case was a probable case with detection of levamisole in urine. Health department officials abstracted data on suspected cases, attempted a patient interview and submitted demographic, epidemiological and clinical data to CDC for descriptive analysis.
Results: Forty-one data abstraction forms were received by CDC; sixteen (40%) did not meet eligibility criteria and were excluded. Of the 25 remaining cases used in the analysis, 24 were probable cases and one was confirmed. These cases represented Michigan (n = 14; 56%), New Mexico (n = 10; 40%), and one from Minnesota (4%). Ten (40%) completed interviews administered by local or state HD personnel. The average age was 44.6 years and half were male (n = 13; 52%). Half presented with infectious illnesses (n = 13; 52%), and two-thirds reported active skin lesions (n = 17; 68%). Five included descriptions: necrotic (n = 3) and ulcerative (n = 2). The majority of interview respondents used cocaine >2–3 times a week (n = 9; 90%). More than half used cocaine >2 years (n = 6; 60%), smoked it (n = 7; 70%), and preferred crack cocaine (n = 6; 60%). All were unaware of exposure to LCC and of levamisole's inherent toxicity (n = 10; 100%). No unique attributes differentiating an experience with LCC from a non-LCC experience were identified through chart review or interview.
Conclusions: Levamisole-associated neutropenia from cocaine use is an emerging public health issue. Most cases reported chronic, long-term cocaine use and were unaware of levamisole exposure. Cocaine use is more prevalent among males, however, this and other studies looking at neutropenia in LCC users have found an equal or higher proportion of females suggesting females may be at higher risk. Physicians should suspect exposure to levamisole-contaminated recreational drugs when treating persons with a history of cocaine use presenting with unexplained neutropenia.
Keywords: Cocaine, Epidemiology, Surveillance

84. Adjunct ketamine use in the management of severe alcohol withdrawal: A case series

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Background: Abrupt cessation of chronic ethanol abuse can result in a prolonged hyperexcitable state due to prolonged antecedent GABA-A desentization/downregulation. Historically, patients in delirium tremens require ICU-level care, large doses of benzodiazepines, receive intubation in severe cases, and have a prolonged hospitalization with tapering doses of benzodiazepines. In addition to its GABA agonist properties, ethanol also functions as an NMDA antagonist; thus,chronic alcoholism results in concomitant upregulation of NMDA receptors. Use of an NMDA receptor antagonist offers a biologically plausible adjunctive therapy that may decrease the total GABA agonist dose utilized for treatment of chronic alcohol withdrawal.
Case series: We instituted a protocol of intravenous ketamine administration (10 mg/hour) for patients in severe alcohol withdrawal (AW) as diagnosed by a board certified medical toxicologist. The ketamine infusion was continued until delirium resolved. All patients defined as severe alcohol withdrawal required greater than 100 mg diazepam in less than 24 hours (or equivalent benzodiazepine) and ICU admission.

Our series included a total of 8 patients (7 men, 1 woman). Table 1 contains age, sex, ICU length of stay, and number of days of ketamine infusion. None of the patients required intubation.

Table 1. Patient characteristics.

Sex	Age (Years)	ICU (Days)	Ketamine (Days)
M	52	3	1.5
M	45	5	2.5
M	49	5.5	2
M	51	4	1.5
F	57	4	3
M	52	4	4
M	33	8	5
M	55	5	2

Discussion: The addition of ketamine to standard GABA agonist therapy is theoretically beneficial due to its NMDA antagonist properties. Historically, the use of ketamine is safe, well tolerated, and with minimal adverse effects. The results of this initial case series are promising for the use of adjunctive ketamine in severe AW. Overall ICU length of stay and GABA agonist requirements were less than anticipated given the severity of the patients' alcohol withdrawal (based on the judgment of experienced toxicologists).We hypothesize that the addition of an NMDA antagonist will: 1) decrease the amount of GABA agonist required for sedation, 2) decrease length of stay in the ICU, and 3) prevent intubation. Follow-up studies will be required to determine the true utility of ketamine in severe AW. These will include dose response curves, randomization of patients to treatment arms, and blinding of providers to therapies.
Conclusions: Based on this descriptive case series, one center has had good success using ketamine in cases of severe AW. Adjunctive ketamine infusions can be considered for patients in severe AW requiring large initial doses of parenteral benzodiazepines that require ICU level of care.
Keywords: Alcohol, Withdrawal, Ketamine

85. Botulism outbreak in a state prison from "pruno"

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Background: Outbreaks of botulism in the US are very rare. We report a large outbreak of botulinum poisoning in a prison population after ingestion of homemade "wine".

Methods: This is an observational case series identified by a sentinel case in an ED. Data was obtained from bedside exams and medical record reviews.
Results: The sentinel case arrived in the ED 52 hours after ingestion of a homemade alcoholic beverage (pruno) complaining of diplopia, dysarthria, fatigue, and dysphagia. At least 11 more prisoners subsequently admitted to consuming pruno. Eight patients consumed the first batch of pruno, which contained a rotten potato and four patients consumed the second batch, which did not contain a potato. Two of the 4 patients from the second batch of pruno were evaluated in the ED while the other 2 patients were evaluated by a neurologist via telemedicine. None of these prisoners were admitted to the hospital but were observed in the prison infirmary. For the 8 patients who consumed the first batch of pruno, the mean onset of symptoms after ingestion was 75.5 hours. Initial symptoms included: dysphagia (5 pts), diplopia (4 pts), dysarthria (3 pts) and general weakness (3 pts). All 8 of these patients were admitted to the ICU for monitoring and received antitoxin an average of 96 hrs after exposure. Seven of these 8 patients had positive stool samples for botulinum toxin. Three patients developed respiratory failure and were intubated. The 5 patients who did not require intubation had a mean Negative Inspiratory Force (NIF) of −58 cm H_2O (range, −50 to −60) and a FVC of 4.36 L range, 3.25–4.7). The 3 patients who required intubation had an average NIF of −35 cm H_2O (range, −15 to −60) and FVC of 3 L (range, 0.9 t°4.7). All 8 patients had an EMG, which was abnormal in 1 of 5 non-intubated patients and in 2 of 3 intubated patients. Seven of 8 hospitalized patients had persistent symptoms 2 months post-ingestion. The 3 intubated patients had a slower recovery with 2 requiring tracheostomy and PEG tube placement. These 2 patients required prolonged inpatient stay in the rehabilitation unit and developed other complications associated with the exposure. They had removal of tracheostomy and PEG at 51 and 90 days post-ingestion.
Discussion: Ingestion of "pruno" at a state prison resulted in one of the largest isolated outbreaks of botulism in the US within the last 10 years.
Keywords: Botulinum, Neurotoxicity, Food poisoning

86. The effectiveness of a state designer drug ban one year later

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Background: Cathinone is a monoamine alkaloid with stimulant and psychoactive properties found in the plant *Catha edulis*. Synthetic cathinones were first noted in the U.S. in 2010, being sold on the internet, in head shops, smoke shops and convenience stores. The products were labeled as bath salts, insect repellent, plant food, and stain remover to disguise their intended use and to skirt existing drug laws. The first synthetic cathinone calls were received by U.S. poison centers in July 2010. The first cathinone call to the Louisiana Poison Center (LPC) occurred in September 2010. The number of cases reported in our state increased dramatically over the next three months. The LPC responded to over fifty five percent of the cathinone calls reported to all U.S. poison centers in 2010. As a result of the number of calls and the severe effects exhibited by many users of these products a ban on six specific cathinones was enacted by emergency rule in Louisiana on January 6, 2011. The ban was expanded to cover additional stimulant/hallucinogen drugs during the spring legislative session and became law in July 2011.

Table 1. Results for abstract 86.

Month	Calls
September 2010	1
October 2010	4
November 2010	23
December 2010	110
January 2011	30
February 2011	6
March 2011	7
April 2011	3
May 2011	7
June 2011	4
July 2011	7
August 2011	1
September 2011	5
October 2011	2
November 2011	3
December 2011	0

Methods: The National Poison Data System (NPDS) was queried to determine the number of synthetic cathinone calls the LPC received by month from September 2010 through December 2011. The numbers of calls prior to the ban were compared to those post-ban in an attempt to gauge whether regulating these designer drugs could help quell the outbreak of calls occurring in Louisiana.
Conclusions: A ban placing six cathinones into Schedule I, making the manufacture, distribution, or possession of those substances illegal in Louisiana went into effect January 6, 2011. The number of synthetic cathinone calls decreased 94.5% from 110 in December 2010, the last month prior to the ban, to 6 in February 2011, the first full month after the ban was put in place. While the number of cases has decreased, they have not ceased. These products are still available and being abused. It appears that the ban in Louisiana helped decrease the number of calls related to these substances. Vigilance by poison center staff to detect new emerging drugs of abuse and education about the dangers of these designer drugs at all levels is critical to guard public health.
Keywords: Legislation, Designer drug, Cathinone

87. Rural vs. urban distribution of synthetic marijuana cases

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Background: Since 2010, U.S. PCCs have managed calls related to synthetic marijuana (SM) exposures. SM is sold as "legal marijuana", easily accessible from convenience stores and gas stations.

The substances bind to CB1 and CB2 receptors with an affinity 60–80 times that of THC. Known by several street names such as spice and K2 and with a price as much as 4 times that of conventional marijuana, SM has emerged as a popular drug of abuse. The study goal is to determine the distribution of SM calls based on population density in our state.
Methods: We identified 334 human exposure cases involving SM in 2011. Seven cases were excluded since caller site was unknown. The remaining 327 cases were sorted by original caller's county.

Table 1. Statewide SM cases stratified by population density.

Geographic area	# of SM cases	Population	# of SM cases/100,000
Large metropolitan	71	5,194,675	1.37
Medium metropolitan	123	3,936,816	3.12
Small metropolitan	60	1,922,605	3.12
Micropolitan/rural	73	866,051	8.43
Large + medium	194	9,131,491	2.12
Small + micro + rural	133	2,788,656	4.76

Using the guidelines listed by the U.S. census bureau the cases were further grouped into the following four categories based on number of inhabitants: Large metropolitan (> 1,000,000); medium metropolitan (250,000–999,999); small metropolitan (50,000–249,000); and micropolitan/rural (< 50,000). Adjusting for population base, we divided the number of calls per 100,000 people within that respective region.

Results: Data collected are tabulated in Table 1.

These results reflect a marked difference in the rate of SM cases reported to our PCC when compared to population density. The rate of SM cases reported per 100,000 population originating from micropolitan/rural and small/medium metro areas was over 6.1× and 2.3× greater respectively than the population corrected rate for macro/urban areas.

Conclusion: It is unknown why there are more SM exposure cases originating from rural areas compared to larger metropolitan areas when corrected for population density. Possible factors include: 1. A higher rate of synthetic drug abuse in rural populations. 2. Rural populations may be more likely to seek ER care for drug related issues. 3. Smaller rural hospitals may not have the resources to manage these cases, thus rely on PCC consultation and call more frequently. Further study as to the underlying cause of increased SM reporting rates to regional PCCs will be needed.

Keywords: Epidemiology, Substance abuse, Marijuana

88. A Comparison of synthetic cannabinoid exposures near the Mexican border vs. those distant from the border

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Synthetic cannabinoids are new substances of abuse. The 14 counties that border Mexico differ demographically from the 240 counties in the rest of the state. It is unclear if this difference may be associated with different clinical presentations. This study compared synthetic cannabinoid exposures reported in the two geographic areas.

Methods: Synthetic cannabinoid exposures reported to our poison centers during 2010–2011 were identified and grouped by whether the calls originated from border or non-border counties. Circumstances & clinical effects were compared between the two areas.

Results: There were 100 border & 937 non-border exposures, resulting in a rate per 100,000 population of 4.1 in each region. The patients in the border and non-border counties were, respectively, 51.0% & 55.5% 20 years or older and 72.0% & 75.0% male. The exposure occurred through inhalation alone in 82.0% of the border and 78.0% of the non-border cases. The border exposures, compared to the non-border exposures, had more serious outcomes (76.0% vs. 62.4%, RR 1.22, 95% CI 1.08–1.37) and more moderate (vs. minor) outcomes (67.0% vs. 41.9%, RR 1.60, 95% CI 1.37–1.87) The Table 1 includes the rates for the most common clinical effects and treatments.

Table 1. Results for abstract 88.

	Border counties (%)	Non-border counties (%)	RR, 95% CI
Clinical effects			
Tachycardia	41.0	38.8	1.06, 0.82–1.35
Agitation	23.0	22.8	1.01, 0.69–1.47
Drowsiness	27.0	18.2	1.48, 1.04–2.10
Vomiting	22.0	14.5	1.52, 1.02–2.26
Hallucinations	8.0	10.8	0.74, 0.37–1.48
Confusion	13.0	10.1	1.28, 0.75–2.20
Nausea	10.0	9.1	1.10, 0.59–2.05
Hypertension	5.0	9.8	0.51, 0.21–1.22
Chest pain	9.0	6.8	1.32, 0.68–2.57
Dizziness	7.0	6.5	1.08, 0.51–2.29
Treatments			
IV fluids	52.0	42.6	1.22, 1.00–1.50
Benzodiazepines	21.0	21.9	0.96, 0.64–1.43
Antiemetics	10.0	5.0	1.99, 1.04–3.82
Naloxone	6.0	2.0	2.96, 1.21–7.23

Conclusions: Synthetic cannabinoid exposures reported from border and non-border counties were similar with respect to exposure rate, patient age and sex, & route of exposure. However, patients from border counties had significantly higher rates of (1) potentially serious outcomes, (2) drowsiness & vomiting, & (3) administration of naloxone & antiemetics.

Keywords: Drug of abuse, Designer drug, Public health

89. Do mosquito swarms lead to increased potentially adverse insect repellent exposures?

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Background: On October 9, 2011, after a long period of drought, the Houston, Texas, area received rain. On October 24, 2011, the Houston Chronicle newspaper reported that this rain, along with warm temperatures, had led to a swarm of "floodwater mosquitoes." The newspaper suggested that the public use insect repellent when outdoors. This study examined whether this mosquito swarm affected the number of insect repellent exposures reported to state poison centers.

Methods: All exposures involving insect repellents (Generic codes 0201048, 0201049, 0218000) reported to Texas poison centers during 2011 were identified. Only those received from Public Health Region 6 (the 13 counties that comprise Houston and the surrounding area) were included in the study. The daily number of exposures during September–December 2011 and the weekly number of exposures during the entire year were determined. The daily and weekly pattern of exposures was compared to the dates of the rain and media report of the mosquito swarms.

Results: The highest weekly number of insect repellent exposures (13) was reported during October 22–28. The next highest weekly

number of exposures (4) was reported during March 12–18, October 15–21 (the week immediately before the week with the highest number of exposures), and October 29–November 4 (the week immediately following the week with the highest number of exposures). During October 21–27, insect repellent exposures were reported every single day. During September 1–October 20, exposures were reported on 9 out of 50 days (mean every 5.6 days). During October 28–November 30, exposures were reported on 7 out of 34 days (mean every 4.9 days).

Conclusions: The highest number of insect repellent exposures in the Houston area during 2011 was reported around the time the media reported a mosquito swarm. The insect repellent exposures appeared to increase in number and frequency before the media reported the swarm and recommended use of insect repellent. Although not definitive, this study suggests that increases in mosquito populations will lead to an increase in potentially adverse insect repellent exposures. This suggests that when increases in mosquito populations are known or expected to occur, public health authorities might want to advise the public to use insect repellents with care and what to do should potentially adverse exposures to the products occur. Moreover, monitoring of insect repellent exposures reported to poison centers might prove useful for surveillance of mosquito populations.

Keywords: Poison center, Pesticide, Insecticide

90. Impact of 2011 drought on snake bites reported to poison centers

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Background: In 2011, Texas experienced the hottest June-August on record for any state in the United States and the longest drought on record for the state with November 1, 2010, to October 31, 2011, being the driest one-year period in the state's history. It has been suggested that encounters with snakes increase during drought and hotter temperatures because the reptiles are searching for food or seeking different habitats. This study examined whether the extreme conditions in 2011 affected the number of snake bites reported to Texas poison centers.

Methods: All snake bites reported to Texas poison centers during 2008–2011 were identified. Snake bites reported from outside of the state were excluded from the study. The annual number of snake bites was determined for all snakes as well as non-exotic venomous snakes and the 4 major types of venomous snakes found in Texas (copperheads, rattlesnakes, cottonmouths, coral snakes). The number in 2011 was compared to that in 2008–2010. The mean and standard deviation for the snake bites reported during 2008–2010 were calculated. If the number of snake bites in 2011 was higher than this mean plus 2 standard deviations, then the 2011 number was considered to be significantly higher than expected.

Table 1. Annual number of snake bites reported to Texas poison centers.

Year	2008	2009	2010	2011
Total snakes	860	898	848	944
Non-exotic venomous snakes	475	551	474	554
Copperheads	259	274	227	313
Rattlesnakes	95	128	130	96
Cottonmouths	30	31	41	54
Coral snakes	36	43	23	29

Results: The associated Table 1 provides the annual number of snake bites. The number of snake bites in 2011 was higher than any of the previous 3 years for total snakes, non-exotic venomous snakes, copperheads, and cottonmouths, and the number was significantly higher than expected for total snakes, copperheads, and cottonmouths.

Conclusions: The number of total snake bites and bites by copperheads and cottonmouths reported to Texas poison centers were elevated during 2011 when compared to the previous 3 years. A similar pattern was not observed for rattlesnakes and coral snakes. It is unclear why all types of venomous snakes did not demonstrate an increase in reported bites in 2011, although differences in habitat and geographic distribution across the state might be involved. Although a causal relationship was not definitely demonstrated, this study suggests that conditions of drought and high temperatures might result in higher numbers of snake bites, particularly copperheads and cottonmouths, reported to poison centers.

Keywords: Snake bite, Poison center, Drought

91. The interaction between social media & a poison network during a public health event

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Background: Poison Centers have long been involved in public health event surveillance & communication. Recent examples include the Columbia shuttle disaster, H1N1 pandemic, Gulf Oil Spill, & the emergence of designer drugs. In recent years, social media has become a new communication platform for individuals & organizations in both daily life & times of disaster. This case reflects the impact of social media on a public health event & subsequent calls to the Texas Poison Center Network (TPCN).

Case report: On Oct 3, 2011 a large chemical factory located near Dallas was destroyed by a fire that started when flammable vapors ignited. The fire was contained within 8 hours; however the site smoldered for several days. Over 225,000 pounds of chemicals, including bulk pesticides & heavy metals, were consumed in the fire and $>1{,}000$ residents were evacuated.

The TPCN received 7 calls that day regarding the fire. Clinical effects included: bad taste in mouth (3), throat irritation (2), headache (1), & numb lips (1). Many members of the public used social networking to discuss the incident, including acute health effects. A community activist group started a Facebook® page dedicated to this event. Within 24 hours, there were 35 posts from the public reporting adverse health effects from the smoke including: throat irritation (17), cough (16), headache (16), shortness of breath (11), nausea (8), rash (4), fatigue (3), dizziness (1), & vomiting (1). On the Facebook® page was a Google Map® used to show the location of the posts, which were consistent with the plume map provided by the National Weather Service.

On Oct 18 the community activist group started contacting different public health agencies regarding the response to the

health concerns of the community. The group became aware of the TPCN's services and started notifying the public via Facebook® to contact the TPCN for any health concerns related to the fire. The TPCN received 9 additional cases within 24 hours of the posting. Clinical effects reported included: throat irritation (6), cough (6), headache (5), dyspnea (2), & vomiting (1).
Discussion: Based on a literature review, there are little data about the impact of social media in disasters. We could not find any reports on poison centers using social media as a public health surveillance tool during disasters. Based upon our experience, poison centers can increase their surveillance by using social networking sites.
Conclusion: In future public health events, poison centers can monitor social network sites and interact with the public via these sites. This will enable poison centers to deliver timely and correct information to the public & encourage the public to contact the center with questions.
Keywords: Public health, Poison center, Social media

92. "Unquestionable answers": A social media campaign promoting the use of poison centers by college-age adults

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Background: Young adults frequently find themselves in new and unexpected situations as they leave home for college, work or military service. Some of these situations involve potential poisonings, whether that means a venomous bite or sting, drug abuse, alcohol poisoning, or a medication mistake. According to a national survey of adults conducted by HRSA, with the exception of senior citizens, people ages 18 through 24 are:

- Less likely than other adults to be familiar with poison control centers and the 1-800-222-1222.
- Poison Help number.
- Less likely to be aware that the poison control hotline is a free service, available 24 hours a day.
- Less likely to know that poison centers take calls about adult medication mistakes.

People ages 18–24 are also much more likely than other groups to seek information on poisonings from the Internet via a search engine such as Google® or Yahoo®.

Poison prevention outreach has historically focused on the prevention of pediatric poisonings and has concentrated on reaching parents and caregivers. However, as poisoning statistics have come to be dominated by deaths in adults (including young adults), and frequently involve substances of abuse, it is clear that efforts are needed to promote the hotline in adults well before they become parents.
Study: The University of Miami hosts a 25-hour "blitz" in which teams of communications students create free, made-to-order ad campaigns for non-profit clients. The Florida Poison Information Center – Miami requested a social media awareness campaign geared toward people exactly like the team members – students aged 18–24. The final campaign, called "Unquestionable Answers," consists of 24 "e-posters" designed for sharing via Facebook. The colorful posters show an internet search box with a poison-related question typed in. Topics ranged from accidental misuse of household products, to food poisoning and popular drugs of abuse. Each poster features the tag line, "Stop searching. Just call 1-800-222-1222."
Results: The AAPCC launched the campaign on April 10, 2012 with a press release to 4173 media outlets. The release was picked up in 378 publications. As of April 16, the campaign had logged 16,949 online impressions. The materials remain available for sharing and posting from the AAPCC website.
Conclusions: The campaign has received very positive feedback and has raised awareness of poison centers among organizations promoting safety in college and university students. It also highlights that, in order to reach younger people who use the phone less frequently for interpersonal communication, new methods are needed to encourage the continued use of the phone for poison emergencies.
Keywords: Social media, Education, Young adults

93. Internet shopping: A vast resource for dangerous poisons

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Background: While the sale and distribution of most toxic substances is tightly regulated within the Unites States, highly dangerous chemicals and drugs can still be purchased with relative ease. Many antique poison containers for sale by collectors still hold their original contents. The purchase of these products may potentially place members of the general public at risk for serious poisonings if unintentional or intentional exposures occur. Our objective was to quantify the wide variety of dangerous toxins and toxicants available for sale to the general public on a popular online auction Web site.
Methods: Over a 6-month period, weekly listings on the online auction Web site eBay® were searched using the term ''poison bottles". Products advertised as containing any of their original contents were identified. Product name, formulation, toxic ingredient(s), the amount of the product in the container, and relative toxicity rating were recorded in an Excel spreadsheet. Relative toxicity ratings were determined using Clinical Toxicology of Commercial Products, by Gosselin, et al. Descriptive statistics (frequency tables, confidence intervals) were used to summarize the data.
Results: A total of 105 individual products were identified during the study period. Fifty-three (50%) were liquids, 31 (30%) were in solid/tablet form, and 20 (19%) were powders. Product containers were full for 48 items (46%) and partially full for the remaining 57. At least 12 (11%) of the containers were described by the seller as cracked or poorly sealed. Sellers were made up of both private individuals and businesses. Fifty-eight products (55%) contained 29 ingredients rated as "extremely toxic" which possess a probable oral lethal dose (POLD) of less than 50 mg/kg. Examples included arsenic, cyanide, mercury, morphine, strychnine, nicotine, pilocarpine, and phosphorus. Twenty-nine products (28%) contained ''very toxic'' ingredients with a POLD of 50–500 mg/kg and included lead, camphor, digitalis, pyrogallic acid, quinine, lindane, and warfarin. The remaining 18 products (17%) contained ingredients that were classified as ''moderately-slightly toxic" with POLDs of 500 mg/kg or greater.

Conclusions: While the vast majority of the products identified were being sold for the nostalgic appeal of their containers, there is no guarantee that purchasers of these products would not attempt to discard the contents or use them in some way. Closer scrutiny of the existing rules, regulations, and policies regarding the sale of poisonous product containers via the Internet is warranted.
Keywords: Abuse, Education, Public health

94. What do I do when my child gets into poison? Ask the internet!

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Background: Poison control centers (PCC) are a resource for parents and caregivers in accidental pediatric exposures, reducing unnecessary emergency department (ED) visits and health care costs. In the last few years, PCCs have noted a reduction in call volumes nationwide. Although the cause of this trend may be multifactorial, the increased availability of the Internet resources and portable devices, such as smart phones and tablets computers, may be contributing to the reported call volume decline. Entering "my baby ate silica gel" into a search engine yields over 80,000 hits. While the accuracy of content on these sites with respect to management and referral is highly variable, users may not validate information with a reliable source. We propose that increased Internet access and availability of Internet ready portable devices is a contributing factor to decreased call volumes. A review of the total number of exposure calls to the local PCC and annual number of hits to its website from 2007 to 2011 was used to examine this relationship.
Methods: The local PCC database was searched from January 2007 to December 2011 for all human exposures, and the total number of calls for each year was tallied. The total number of hits on the PCC's website, which contains information on household exposures, during the specified time period was also extracted.
Results: The number of annual exposure calls demonstrated a steady decline during the aforementioned timeframe from 54,596 to 47,350. In contrast, the annual number of hits to the local PCC website increased from 5,048,426 to 7,024,691. The results are summarized in Table 1.
Conclusions: The decrease in PCC exposure calls inversely correlates with hits to the same PCC's website over a 5-year period. Recent technologic advances have resulted in improved access to information, likely decreasing PCC utilization for questions regarding common household exposures. Adopting innovative ways to disseminate information to the public is likely necessary for PCCs to remain viable first-line resources in this era.
Keywords: Poison center, Call decline, Internet

Table 1. Results for abstract 94.

Year	Total exposure calls	Annual hits to local PCC website
2007	54,596	5,048,426
2008	54,693	5,076,646
2009	52,264	5,033,120
2010	49,512	5,620,455
2011	47,350	7,024,691

95. From the ED and beyond: Successful collaboration leads to a prompt response

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Background: An important function of poison centers is to utilize the information obtained to provide toxicosurveillance. By virtue of being an organization that provides 24-hour service to local populations, poison centers can play a sentinel role by alerting local health departments of unusual cases or occurrence of a common case above the threshold level for a particular time and place. We report an instance of prompt, effective collaboration with an Emergency Department (ED); Regional Poison Center and the Local Health Department after patients presented with symptoms consistent with strychnine after heroin use.
Case reports: Patient 1 presented to the ED with symptoms of tremors, muscle rigidity, high blood pressure, and increased heart rate. The patient reported that he had used heroin via insufflation earlier that evening and that the symptoms began shortly thereafter. The treating physicians rapidly recognized that the patient was experiencing symptoms not consistent with heroin toxicity and immediately considered that this may indeed be heroin contaminated with strychnine. The regional poison center was contacted who then subsequently contacted the local Health Department. The health department facilitated qualitative urine testing for strychnine. The same day, a second patient (Patient 2) presented to a different ED (in the same city) with history of heroin abuse and symptoms similar to Patient 1. Similar steps were undertaken for Patient 2 as for Patient 1. Within 24 hours, strychnine was identified in urine samples of both patients. Quantitative tests conducted later on serum and urine samples of Patient 1 showed very high but non lethal Strychnine levels. The local health commissioner alerted the State Health Department and all local Emergency Departments of possible strychnine poisoning in people abusing heroin. Law enforcement was notified of these occurrences and the dangers of strychnine poisoning.

Within four days of the first patient encounter, a health advisory was issued by the local Health Department and a press release was generated alerting of possible strychnine contamination of heroin.
Discussion: We describe a scenario where effective collaboration between healthcare facilities, poison centers and health departments resulted in a prompt public health response and likely mitigated further exposures and toxicity. Cases received by the Emergency Departments or PCCs can initiate a series of activities undertaken by other agencies such as health departments and law enforcement to stem the spread of such poisonings.
Keywords: National poison data system, Public health, Heroin

96. Call center call volume decreases differ by age group and by type of caller

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Background: Call volumes reported by most US poison centers (PC) have been decreasing for 5 years or longer, despite increased population. Historically, more than half of PC calls have concerned children < 5 years of age. The reason for this decrease in PC calls remains unknown.
Objective: To establish whether call volume changes differ by age groups and types of call, during the period 2000–2010.
Methods: Call volumes to one large PC and to one large pediatric nurse advice center (PNAC) were analyzed by age of patient, site of call, and type of call. The PC served the entire state. The PNAC, operated as a free service during 2003–2008 by the children's health care system with > 95% regional market share of pediatric inpatient and ED care, served the state's largest metropolitan area (47% of state population). PC data from 2000–2010 was utilized; PNAC data was only available for 2003–2008. US Census Bureau annual population estimates for the state by age group were used.
Results: During years 2000–2005, estimated child population < 5 yr (CL5) increased 16% while call volume about CL5 from homes to the PC was level and from healthcare increased 11%. PNAC CL5 volume decreased 6% from 2003 to 2005. During 2005–2010, CL5 population decreased 0.5% while CL5 calls to the PC from homes decreased 11.3%, and from healthcare increased 12.5%. PNAC CL5 calls decreased an additional 13% from 2005–2008.

During years 2000–2005, estimated adult population > 65 yr (AG65) increased 10% while calls about AG65 increased 37%. During 2005–2010, AG65 population increased 19% while calls about AG65 increased 6.5%.
Discussion: The trend of decreased PC volume is predominantly driven by calls from homes about children < 5 years old. The same decrease in call volume from lay callers has affected the area's largest PNAC as well. Call volume about older adults increased as did population. This trend may reflect a change in information seeking practices by young adults as compared to older adults. Most of the parents of the children < 5 years are younger adults who have been raised during an era of technological change and ascendancy of the internet and social media, and who have been shown to be more likely to have internet access. Given that the trends observed span across different types of health call centers, the etiology is not specific to PCs and the need for their services. Further study is needed about these trends.
Keywords: Poison center, Pediatric, Epidemiology

Table 1. Results for abstract 96.

	2000	2003	2005	2008	2010
Population est < 5 yr	595150	659238	692726	740521	688521
Population est > 65 yr	788885	826506	870422	981024	1036578
Calls from Residence re < 5 yr	37266	36897	36759	35871	31715
Calls from Healthcare re < 5 yr	3281	3443	3637	4024	4092
Lay Calls re > 65 yr	1533	1754	2109	2161	2286
PCNA Calls re < 5 yr		237408	221410	191149	

97. Population adjustment for US poison center call volume changes: 2000–2010

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Objectives: We examined the National Poison Data System (NPDS) of the American Association of Poison Control Centers (AAPCC) change over time (COT) in closed human exposure, non-corporate encounters for the years 2000–2010 to assess the impact of population changes to the decrease in exposure call volume since 2008.
Methods: Calls from health care facilities (HCF) have continued to increase, thus countering the overall volume trend from 2008–10, so we first identified a "signal" subset of exposures (SSE) that comprised the subset chiefly responsible for the exposure volume decline since 2008 via a stepwise subsetting of Caller Site, Reason, Outcome, and Exposure Site. Population data were available for 2000–2010 for the 7 age groups shown in the Table 1. Quadratic regression was fit to each population group, each age subset of the SSE, and the population corrected SSE groups. The slope at the year 2010 was calculated for each from the regression curve.
Results: The SSE comprised 60% of all NPDS exposures. The Table 1 shows the mean number of exposures, slope for exposure counts, slope of exposures %, population corrected exposures, population, and the % increase (adjusted to unadjusted). All Ages SSE decreased at 3.62%/year, Population increased at 0.95%/year, and Exposures/1000 population decreased at 4.45%/year. The last column shows that the 3.62% to 4.45% overall exposures declined 22.9% more rapidly than would be evident without population correction.

Table 1. Results for abstract 97.

Age group (years)	Mean exposures (/year)	Slope @ 2010 (cases/yr)	Exposures slope @ 2010 (%/year)	Exposures/1000 slope @ 2010 (%/year)	Population slope @ 2010 (%/year)	% Increase adjusted/ unadjusted
0–4 y-o	997,420	−18677	−1.87%	−2.07%	0.18%	−10.3%
5–13 y-o	118,181	−4658	−3.94%	−4.53%	0.61%	−14.9%
14–17 y-o	22,155	−2499	−11.28%	−10.04%	−1.03%	11.0%
18–24 y-o	38,762	−3450	−8.90%	−8.90%	0.50%	0.04%
25–44 y-o	96,804	−8397	−8.67%	−8.91%	0.06%	−2.75%
45–64 y-o	56,609	−3971	−7.01%	−7.99%	2.13%	−13.8%
65 and >	24,887	−1113	−4.47%	−6.77%	2.66%	−51.4%
All ages	**1,409,452**	**−51020**	**−3.62%**	**−4.45%**	**0.95%**	**−22.9%**

Conclusions: For this SSE: 1) All 7 age groups showed a strong COT signal and all 7 had a negative slope at year 2010, 2) 0–4 y-o were the largest group and the largest contributor to the decrease; 3) the population is increasing in most groups and the population correction served to increase the rate of decline for All Ages, and for 5 of the 7 age groups; 4) The oldest age group (65 and >) was most effected by the population correction.

Thus population correction tends to make the call volume "look worse" (demonstrates a more rapid decline in exposure calls at year 2010). These SSE analyses excluded HCF and other call classes which have continued to increase.

Keywords: Epidemiology, Poison control centers, Exposures

98. Are emergency department visitors receptive to obtaining information and engaging in political advocacy in support of poison control centers?

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Background: Poison control centers (PCC) throughout the country have been closing due to reduced governmental funding. This has been associated with a negative impact on the delivery of health care to the communities they service. As compared to the general lay public, visitors to the hospital may be more receptive to obtaining information and engaging in political advocacy in health care policy.

Objective: Our objective was to determine if visitors in the waiting area in hospital emergency departments (ED) were willing to receive information on PCC funding and willing to generate a letter to the Governor of New York to reinstate funding for New York PCC.

Methods: We performed a multi-center IRB-approved, convenience-sample, survey study at three hospitals across New York State from June 2010 to April 2012. All hospitals in the New York State American College of Emergency Physicians Research Consortium were invited to participate. Research interns approached adult, English-speaking visitors in the ED waiting area. In Stage 1, visitors were asked to complete an anonymous nine-question survey on health care issues. Questions were extracted from a national survey (Kaiser Family Foundation/Gallup Poll, 2007). In Stage 2, visitors were offered an informational brochure from the American Association of Poison Control Centers on the importance of poison control centers. In Stage 3, visitors were asked to sign a letter to the Governor of New York State in support of continued funding for PCC and to indicate whether they wanted their letter to be sent. Data were analyzed employing descriptive statistics.

Results: A total of 1077 subjects were approached. 705 subjects (65.5%) agreed to complete the survey in Stage 1. Of these subjects, 454 (64.4%) were willing to read about PCCs in Stage 2. Most subjects in Stage 2 also agreed to sign a letter in support of PCCs in Stage 3 (n = 359, 79.1%), and have the letter sent to the Governor (n = 343, 75.6%).

Conclusions: Over two-thirds of all visitors who were approached were willing to read information on PCC and over one-third of them were willing to send a letter of support of PCC. Hospital visitors are a willing cohort to engage in political advocacy in support of PCC.

Keywords: Poison center, Legislation, Public health

99. Time requirements for management of hospital cases by poison centers

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Background: Although the majority of calls to poison centers (PC) result in home management, a significant number of calls involve patients managed at health care facilities (HCF). PC assistance in management and documentation of HCF cases may require more time than home cases. Time-work data are needed to accurately gauge FTEs needed to handle HCF cases.

Methods: Human HCF exposure calls managed by one poison center were randomly selected for study at the time of the initial call. Specialists (SPIs) involved with each case tracked all on-and-off phone time spent managing, researching, consulting and documenting the cases using a digital clock. Times for each activity were recorded on a worksheet which was maintained until a case was closed. One researcher entered the data into an Excel database and utilized the PC medical records and phone call database to ensure all calls related to a case were included. An initial trial period for data collection was performed for three months to provide feedback and training reinforcement to staff to ensure accurate data collection. Study data was then collected in blocks of 10 cases. The end point was pre-determined to be the point after 100 cases when the median management time no longer changed with the addition of 10 cases and the mean management time changed by less than 2%.

Results: There were 140 cases included in the analysis during the 3 month study period. This represents 3.4% of all calls to the PC that were managed at a HCF during that time period. Mean total management time was 46.0 minutes. Median total management time was 29 minutes with an interquartile range of 19.2 to 53.5 minutes. 23% of cases required more than 1 hour for total management.

Conclusions: Although further work (planned) is needed to directly compare time required to manage a patient at home versus in a HCF, the amount of time required for PC management of a HCF case appears substantial. Accurate measurements of time required for total handling of the different types of calls will permit more accurate estimations of SPI FTEs required for PC operations.

Keywords: Poison center, Time, Health care facilities

100. Professional carpet cleaners do not comprehend the implications of spilled mercury in the home

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Background: Glass thermometers containing elemental mercury are a source of accidental mercury exposure because many people attempt to clean the mercury using a standard vacuum. This aerosolizes the mercury, creating the risk of inhalation exposure and potential pulmonary toxicity, especially for children.

According to the CDC, adequate ventilation and avoidance of spreading or aerosolizing the mercury are key measures to be taken during cleanup, preferably by a contractor trained in mercury removal.
Objectives: To determine how knowledgeable carpet cleaner companies are in dealing with mercury removal.

We hypothesize that less than half of the cleaning contractors contacted will answer the following question correctly, "Is there any special equipment or methods necessary to clean up a class thermometer from a carpet?"
Methods: Research Assistants identified and created a list of carpet cleaning agencies in the Philadelphia area by using telephone books and Internet search engines. Researchers posing as a concerned parent contacted the agencies via telephone interviews. Through a scripted conversation, they were asked the following questions:

- Do you need to use any kind of special vacuum or is there some special method to clean mercury?
- Is there anything I should do in the meantime?
- Can I let me 2-year old baby play there?

Results: 75 companies within the Philadelphia area were contacted. Of those, 56 companies responded to the telephone calls and were able to participate. Only 28.57% of companies (16/56) knew that there was a special method to clean up mercury; however, of these companies only 31.25% (5/16) gave a proper/safe method. Overall, only 8.93% (5/56) of companies gave a safe method of disposal.

Only 32.14% (18/56) established that there was anything that could be done in the meantime. Of this percentage, 66.67% (12/18) gave an approved technique.

Almost 95% (53/56) carpet cleaning companies indicated that it was unsafe for a child to come into contact with the soiled area.
Conclusions: Of 56 carpet cleaning companies contacted, only 28.57% indicated that there was a proper methodology to safely clean up a mercury spill. More importantly, 91.07% that indicated a specific technique gave an unsafe or inappropriate method of disposal. 95% of companies indicated that it was hazardous for children and that they should be kept out of the vicinity of the spill. However, the data collected from this preliminary study suggests that barely 9% of carpet cleaning "professionals" actually provided a safe technique, which could be detrimental to the health of the community.
Keywords: Mercury, Carpet, Clean up

101. Disaster preparedness of poison control centers in the United States

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Objective: We sought to determine changes in the demographics of the US poison control center (PCC) and PCC disaster preparedness as compared to Vilke et al. (Disaster Preparedness of Poison Control Centers in The United States, Clinical Toxicology, 1996; 34(1): 53–58). We also sought to determine the extent to which PCCs are engaged in disaster and terrorism preparedness planning and other public health roles and responsibilities.
Methods: A computerized questionnaire was sent to the managing director of the 57 member PCCs of the American Association of Poison Control Centers.
Results: A response was received from 37/57 (65%) members. PCCs serve a population greater than 1 million (65%) or greater than 5 million (35%) and have an annual call volume between 25,000 and 60,000 (45%) or greater than 60,000 (55%). A written disaster plan was reported by 100% of respondents with 65% of centers conducting drills annually to rehearse the plan. Arrangements in place for back-up coverage with another poison center was reported by 97%. Arrangements for a loss of phone coverage was reported by 95% of centers and 67% have a backup phone system in place. The presence of a backup generator (76%), uninterrupted power supply (70%), plan for loss of power (95%), physical plant damage (84%), or increased phone traffic (73%) were also reported. Fifty-four percent have been involved in a county, state, or federal declared disaster. Hurricane (27%) was the most frequent disaster. An inability to operate at some point in the previous 5 years was reported by 70% of centers with 46% reporting a loss of services between 1 and 12 hours in length. Loss of physical structure (16%) and loss of computer services (14%) were the most frequent reasons for an inability to operate. Involvement in terrorism preparedness and response policy (70%) and disaster preparedness and response policy (86%) at the local, state or federal level was reported. Involvement in public health functions such as reportable illness surveillance or answering "after hours" public health calls was reported in 92% of respondents. Comfort with the ability of the PCC to operate during a disaster and meet the public's need was reported by 90% of managing directors.
Conclusions: Despite a decrease in the number of PCCs nationwide, an increase in calls received and population served per center, US PCCs appear more prepared for disaster than previously described when measured by the presence and components of a disaster plan and regular ongoing drills. PCCs are actively involved in terrorism and disaster preparedness and response planning and traditional public health responsibilities such as illness surveillance.
Keywords: Poison center, Education, Public health

102. The use of ECMO in hydrocarbon aspiration: A case report

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Background: Hydrocarbons are a common source of accidental ingestion in the pediatric population, leading tosubsequent lung injury. The use of ECMO in the pediatric population is an underutilized and novel tool in oxygenating the ventilated hydrocarbon toxic patient. We present a case of 23 month old Male with lamp oil aspiration and subsequent pneumonitis, right pneumothorax and pneumomediastium who was successfully oxygenated with the use of ECMO.
Case: A 23 month old male presents to a community hospital with a main complaint of sleepiness, vomiting, and tachypnea, requiring intubation for airway protection. Mother states that patient ingested unknown amount of a decorative lamp oil that was at home. CXR done at the time showed aspiration pneumonitis.

Patient subsequently developed right pneumomediastinum and pneumothorax, and ARDS with subsequent chest tube placement. Patient was admitted to the PICU and had persistently elevated $PaCo_2$ despite adequate ventilation, during which time patient was switched to ECMO. Patient was subsequently extubated 2 months later with residual neurological deficits thought to be due to hypoxic encephalopathy. Patient recovered well in a rehabilitation home and was discharged home with family in good condition.

Discussion: The main hazard associated with ingestion of hydrocarbons is a chemical pneumonitis following aspiration of vomitus. Ingestion of small amounts can still result in the oil "creeping" into the lung, causing a severe chemical reaction and inflammation. In a retrospective study evaluating kerosene ingestion in Iraqi children, 90% of the children had pulmonary complaints and 43% had pneumonia. The treatment of oil lamp ingestion is mostly supportive. The use of conventional mechanical ventilation usually leads to high airway pressures and air leaks, leading at times to the need for high jet ventilation and more advanced ventilation techniques. Failure to adequately oxygenate and ventilate is an indication for ECMO, yet ECMO in the older child is controversial and its use in the hydrocarbon toxic patient has not been studied well. Rarely is the pneumonitis in hydrocarbon toxicity severe enough to need advanced oxygenation than what mechanical ventilation can provide. Several recent studies showed that the expanded application of ECMO into hydrocarbon toxicity did in fact improve survival for older children and which the above case illustrates well.

Conclusions: In the severely hydrocarbon poisoned patient, the use of ECMO can be novel and life saving when other conventional therapy fails. Only with further research on severe hydrocarbon poisoning and the criteria needed for use of ECMO can its benefit be truly put to the test.

Keywords: Hydrocarbon, Ingestion, Education

103. Your FIRST CALL in a poisoning emergency?

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Background: This Regional Poison Center (RPC) experienced an 11% decrease in exposure calls between 2001 and 2011, despite an 18.3% increase in population. Lay callers occasionally recount internet use prior to calling the PC. Residential internet access in the US has increased four-fold since 1997, with over 70% of US households being "on-line".

Objective/Methods: To ascertain the role that the internet or other resources may have on utilization of PC services, callers were asked, "Did you call anyone else or access the internet prior to calling the Poison Center?"

Results: All calls from 2/8/2012 and 4/1/2012 between 07:00 and 18:00, handled by the same CSPI, were analyzed. Of 462 calls, 6.2% of all exposure calls from the study period, 262 were eligible for inclusion. Calls from HCFs and health care workers were excluded, as were lay calls that required immediate medical attention. Sixty-two percent of the calls occurred during the week, 38% on the weekend. Mothers and fathers were 66% of callers (155 & 19 respectively), 6% grandparents. 17% called about themselves. Of the 39% who called someone else prior to calling the PC, 76% had called a health care professional; 22% had called a family member or friend; 8% had called more than one person. Of the 27% who stated they had accessed the internet, 58% did so to get the PC number. Prior to calling the PC, 37% had neither called anyone else nor accessed the internet. Of the 7115 lay calls to this RPC during the study period, only 1.93% of the callers were transferred directly by EMS or an Advice Nurse Line.

In 2011, 27% of the callers to this RPC who participated in a satisfaction survey stated they got the PC number from the internet; 29% from a HCF, Advice Nurse Line or 911. When asked, "If the Poison Center was unavailable, what would you have done?", 85% responded they would either call or go to a HCF or call 911 or an Advice Nurse Line. Only 5% would search on-line.

Discussion: It is impossible to know the actual incidence of poisonings that occur based on PC data. The number of potential callers who access the internet or some source of information in lieu of contacting a PC is likewise unknown. The exact cause of the decrease in calls to this RPC remains uncertain. The role of internet access in decreasing call volume is unknown, though the respondents in this study used the internet primarily to get the PC number. Based on the responses, the PC appears to be the "first call" for poisonings only in some cases. Continued publicity about PCs, their services, and the National 800 number is needed.

Keywords: Poison center, Health care utilization, Epidemiology

104. Overall quality of poison center data for acetaminophen-containing products is high, but coding of product-specific fields needs improvement

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Background: Research suggests that poison center (PC) data quality is suboptimal. However, no systematic quality assessment of PC data across multiple centers has been performed. Exposure information related to acetaminophen-containing products represents approximately 8% of annual PC data and provides a targeted set of data to assess for quality. We measured the accuracy of PC data collection of acetaminophen-containing products.

Methods: 12 US PCs randomly selected 100 acetaminophen-containing product exposure cases that occurred during March 2011. Audio recordings were compared to the documented case fields to assess accuracy of 18 demographic, exposure characteristics, and outcome/management fields, and 7 product-specific fields for each substance involved. Accuracy was determined based on standard field definitions provided in the NPDS reference manual. The percent correct was calculated for topical subgroups of the data (patient demographics, exposure characteristics, outcome/management information, substance information) and for overall. Mean Quality Scores (MQS) are reported for the 12 PCs combined.

Results: 1200 acetaminophen-containing product cases were reviewed. 3 cases were excluded for incomplete data, leaving 1197 cases (35,063 fields) eligible for analysis. 784 (65%) cases involved

Table 1. Results for abstract 104.

Data field category	Mean quality score (% correct)	5th–95th percentiles
Patient demographics	96.8	80.0–100
Exposure characteristics	95.1	75.0–100
Outcome/management information	97.2	75.0–100
Substance information	89.0	57.1–100
Product name/code	67.4	0–100
Generic name/code	94.1	50.0–100
Quantity	88.1	0–100
Quantity unit	91.6	0–100
Certainty	85.4	0–100
Formulation	97.4	100–100
Route	98.7	100–100
Overall score	94.0	83.3–100

1 product; 413 (35%) cases involved ≥2 products. Overall MQS were high (94.0%; 5–95 th percentile 83.3–100%). MQS by topical subgroups were highest for outcome/management fields (97.2%; 5–95 th percentile 75–100%) and lowest for substance information fields (89.0%; 5–95 th percentile 57.1–100%). Among substance fields, route (98.7%; 5–95 th percentile 100–100%) and formulation (97.4%; 5–95 th percentile 100–100%) had the highest MQS, while product name/code (67.4%; 5–95 th percentile 0–100%) had the lowest.

Conclusions: Overall accuracy of PC data collection for acetaminophen-containing product exposures is high, but opportunity for improvement is evident, especially regarding product code identification. Product identification is critical in poison center case management as well as drug safety surveillance, therefore additional efforts are needed to improve accuracy.

Keywords: Quality, Acetaminophen, Poison center

105. Temporal trends in fatal motor vehicle crashes associated with driving while intoxicated by vehicle class

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Background: Driving while intoxicated (DWI) is associated with significant morbidity and mortality due to motor vehicle crashes (MVC). Substantial public health efforts have been directed at educating commercial and non-commercial drivers with regard to the dangers of DWI. We analyzed data collected by the Federal Motor Carrier Safety Administration (FMCSA) to determine whether there has been a significant trend in the percent of fatal MVC associated with DWI.

Methods: Using annual data released by the FMCSA, we performed bivariate regression for trend on the percent of fatal MVC associated with DWI for drivers of large trucks, light trucks, passenger cars, and motorcycles for the years 1985–2008 at blood alcohol concentrations (BAC) of 0.01 g/dL and 0.08 g/dL. We also performed Analysis of Variance (ANOVA) to determine if there was a statistically significant difference between drivers of large trucks, light trucks, passenger vehicles, and motorcycles in the percent of fatal MVC in which the driver had a BAC of greater than 0.01 g/dL or 0.08 g/dL.

Results: Bivariate regression on each class of drivers at the two tested BAC levels demonstrated a quadratic regression best fit the data with a decline in the percent of fatal MVC associated with DWI from 1985 until approximately 2003 and a slight up-trend from 2003–2008, with R^2 values varying between 0.62 and 0.92 depending on the driver class and BAC level analyzed. Trends at the BAC of 0.01 and 0.08 g/dL levels paralleled each other such that the same regression line fit both data for each class of vehicles. ANOVA demonstrated that, as compared to drivers of light trucks and passenger cars, motorcyclists had a significantly higher percent of fatal MVC associated with DWI, while drivers of large trucks had a lower percent of fatal MVC associated with DWI.

Conclusions: The decrease in percent of fatal crashes associated with DWI suggests that public health and regulatory efforts directed at decreasing the mortality associated with DWI have been successful. Limitations of our analysis include the increasing important of electronic distractions, such as cell phones and texting, that may confound the temporal trends. Alcohol intoxication associated with fatal MVC is less common in commercial drivers of large trucks and more common in motorcycle drivers than in drivers of other types of vehicles, suggesting that greater public health efforts should be directed at motorcycle drivers to target this particularly at risk group.

Keywords: Occupational, Alcohol, Death

106. Improving the percentage of known outcomes: A successful quality improvement initiative

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Objective: The objective of this study is to describe a quality initiative to increase the number of follow-up calls and improve known outcomes.

Methods: This study was carried out in a regional poison control center between January 2009 and December 2011. Cases included were human exposures where the reason for exposure was unintentional or adverse reaction. The baseline percent of cases with known outcomes was 49% at the end of 2008. The staff collaborated to develop a goal to increase the percentage of cases followed to a known outcome of 55%. Known outcomes were calculated as 100% minus the percentage of cases that were coded as not followed (judged as nontoxic, minimal clinical effects possible, or potentially toxic). Monthly reports describing individual and team results were presented to all spe-

Table 1. Results for abstract 106.

	2008 (Baseline)	2011 (Final)	Yearly improvement over baseline (%)		
			2009	2010	2011
SPI 1	56%	64%	2%	8%	9%
SPI 2	48%	71%	6%	15%	23%
SPI 3	42%	44%	−1%	2%	1%
SPI 4	50%	57%	3%	6%	7%
SPI 5	53%	61%	7%	9%	8%
SPI 6	52%	66%	3%	4%	13%
SPI 7	44%	69%	12%	29%	25%
SPI 8	47%	50%	1%	3%	3%
SPI 9	42%	66%	10%	14%	24%
Center total	50%	60%	4%	9%	10%

cialists and highlighted regularly at staff meeting. As the study progressed, ongoing individual and center results were provided regularly to staff.
Results: Individual results were available from 2008 (baseline) to 2011 for 9 (6 full-time, 3 part-time) specialists (Table 1). Four additional specialists joined the staff after this project was initiated. Their data are included in the center total results but their individual improvement over baseline was not quantified. Known outcomes increased to 53.4% in 2009; 58.6% in 2010, and 59.9% in 2011. Results for the nine specialists participating during the entire study showed an average individual increase of 12.6% for known outcomes, with a range of 1 to 25% increase in known outcomes. For the year 2011, a 10% increase in known outcomes is approximately 4047 additional cases followed. Every specialist improved their individual known outcomes over the study period.
Conclusions: We describe a quality improvement initiative that was developed with staff input that lead to an increase in the number of cases followed and an increase in known outcomes.
Keywords: Poison center, Quality improvement, Known outcomes

107. Retrospective analysis of consultations data in a poison control center

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Background: Poison Control Center in China CDC opened a 24 hours hotline, and provided poison information service for the whole country in 1999. We established a database for collecting the information of poison consultations in January, 2008.
Objectve: To evaluate the epidemiologic characteristics of 24 h poisoning consultations in the last 4 years, find common and most harmful toxic substances, and provide reference for clinic treatment and poison control.
Methods: We reviewed the collected consultationS data from 2008 to 2011, and performed a descriptive analysis.
Results: A total of 15303 valid records, 49% of them occurred from May to August. 66.08% by physicians, 22.00% by relatives. Most exposures were intentional or suicide (56.94%), unintentional and accident (24.94%) was the secondary cause. The commonest substances were pesticide (86.47%, permethrin 28.23%), chemical (6.23%), medicine (3.35%) poisonous plants and animals (2.00%). The highest mortality was caused by paraquat. Sex ratio of male and female was 2.2:1 in age group (≤ 17), infant and preschool children occurred were unintentional and accident. Sex ratio of male and female was 0.83:1 in age group (≤ 60), 1.2:1 in age group (≥ 60), and occurred were intentional. There were significant differences in sex ratio among different age groups.
Conclusions: Pesticide poisoning is an important status in China today. Infant and preschool children unintentional and accidental poisoning cases occurred in increasing numbers, intentional poisoning of school-age children and teenagers are increasing. Greater national efforts should be directed to improve poison prevention and monitoring, strengthen management of toxic substances and take measures to improve health education.
Keywords: Poison center, Pesticide, Child abuse

108. The educational BfR-garden of poisonous plants – a public show of plants based on the German classification of poisonous plants

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Objectives: Plants delight us in forests and gardens, they produce oxygen, provide comfortable climatic conditions and constitute the most important fraction of human food. Findings made by mankind have resulted in the knowledge that only a defined share of the plants is inedible or poisonous. Deadly poisonous in Middle Europe are only few plants like e.g. Aconitum napellus. Most of the fatal cases refer to mistakes or misuse in adults, whereas fatalities among children are extremely rare. To get an objective assessment of plants, the BfR run a Federal research project which led to the first German Classification of Poisonous Plants (GCPP) in 2000. After a review of data, provided by German poison centres, completed by new literature and under the competence of a working group of the German "Poison Committee", we are about to publish the first review. To refresh the knowledge around poisonous plants, we are now under the construction of a public garden for poisonous plants.
Method: Following the assessment of the toxicity of chemicals in analogy to the German Regulations on Dangerous Substances, we re-classified more than 80 plants into three categories, namely plants which could lead to (+) minor poisoning, (++) moderate poisoning and (+++) severe or deadly poisoning. For all plants, the habitat (garden, park, great outdoors) and a comparable amount of a possible ingestion have been borne in mind. Based on the first GCPP, we listed plants for the garden under educational reasons into two garden beds: 1) plants with moderate poisoning and severe or deadly poisoning 2) plants belonging to the category minor poisoning.
Results: The garden is situated in a protected institute's area, but with a public access for an interested audience. The raised garden beds will have small trails and visit points, where informative signboards – close to the plants – show details about the name, family, toxic parts of the plant, the category of toxicity, etc. For the garden bed (ca. 4 times 6 meter) of the moderate, severe or deadly poisoning plants, we chose 15 plants from Aconitum napellus to Convallaria majalis. For the counterpart, the garden bed (ca. 4 times 4 meter) of the minor or non-poisoned plants, we chose another 15 plants from Cyclamen to Clematis.
Conclusions: The German Classification of Poisonous Plants contains useful information more than 150 different plant species. The final table of poisonous plants will find its counterpart in a list of more than 60 non-poisonous plants appropriate to be used in the close proximity of children's playgrounds, kindergartens, schools, public parks etc. The design of the garden is for educational purpose and will be opened in summer 2012.
Keywords: Plants, Education, Public health

109. BfR-human case report database for poisonings – the public access prototype

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Background: German Physicians and Poison Centres report human data of poisonings to the BfR Documentation and Assessment Centre for Poisonings (BfR-DocCentre). This realistic data obtained from the evaluation of cases of poisoning in humans is especially important for an well-founded assessment of risks in human health. The existing data is condensed in a harmonized and standardized data file for analysis in the BfR-Poison Information Database. In addition cases of special toxicological and scientific interest (e.g. rare poisonings, high-/low-dose exposures, cases with unexpected clinical course, substances of special interest etc.) are regular prepared for standardized case reports. To manage these case reports, the BfR developed a bilingual case report database, which has been improved step by step.
Methods: Selected cases are documented in a standardized form (accident/situation of poisoning/symptoms/signs/exposure data/clinical course/assessment/remarks), indicated by the substance/product involved and supplemented with important references. After co-checks for correctness, completeness and readability, the German text is translated into English and transferred into the database. Significant case reports from literature were transferred as pdf-files into the database as well as case reports from international scientific congresses/conferences. The recent development step was to find an easy and ergonomically retrieval procedure for the human data, compile the documents and transform the database for the www-Access via the BfR-internet portal.
Results: Based on an Informix 9.2 database (web-browser technology/user-interfaces with Java Server Pages) the BfR-Human Case Report Database is developed as a module of the datamanagement system of the BfR-DocCentre. The case reports with related images (pictures, labels etc.) are directly linked to the case report database. On selection of different items the processed cases and case reports can be retrieved in both German and English language. The selected case reports are listed with their literature or case records and a short significant abstract. The BfR-Human Case Report Database offers the documents in English or German language and additionally posters or presentations of case reports. Since July 2002 more than 750 cases have been selected, prepared and processed with additional data.
Conclusions: The BfR-case reports were written down in uniform documents, provided with keywords and additional information and can be easily retrieved in the BfR-Human Case Report Database by index words. Since April 2012 we implemented a public access. The prototype will be demonstrated via the BfR-internet portal.
Keywords: Human case reports, National poison data system, Intoxication

110. Characteristics analysis of acute poisoning from 6 hospitals in 5 provinces in China between 2009 and 2010

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Background: To analyze the characteristics (type, occupation, cause, outcome, etc,) of acute poisoning cases.
Methods: Analysis data were obtained from all acute poisoning cases between 2009 and 2010 from 6 hospitals selected from five provinces in China, analyzed statistically with SPSS18.0.
Results: Proportion between males and females was 1:1.28, mortality was 2.09%. The top three toxic materials were pesticides (42.96%), chemicals (30.55%) and medicine (20.41%); Paraquat poisoning, accounting for 83.14% of all the deteriorated cases, ranked the top. In view of season, pesticide ranked the top in summer, while carbon monoxide in winter, others had little seasonal variations; The top three occupations were farmers (60.41%), workers (14.91%) and students (5.79%); The top three poisoning cause were suicide (61.60%), accident (30.14%) and occupational exposure (5.42%), female dominate in cases of pesticide poisoning, otherwise male dominate in cases of accidents and occupational exposure.
Conclusions: Pesticide poisoning ranked the top among all the acute poisoning. Paraquat poisoning had poor prognosis. Suicide was the most common cause of poisoning. Most poisoned patients came from urban area. Pesticides and carbon monoxide poisoning occurred seasonally.
Keywords: Acute poisoning, Poisoning surveillance, Paraquat

111. Lessons learned from weather-related events and implications for poison centers

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Background: On August 27th and October 29th, 2011, a tropical storm (TS) and nor'easter (NE), caused massive power outages in our state, for up to 12 days. Each event significantly impacted our call volume and tested our emergency resources. Early on, poison specialists (SPIs) identified an influx of carbon monoxide (CO) exposures, due mainly to portable gasoline-powered generators and unsafe heat sources. They realized the immediate need to alert authorities and consumers. We evaluated our response and actions taken during and after these events and the lessons we learned.
Methods/Results: A retrospective analysis of CO cases to our center confirmed call clusters matched to event date and utility company power restoration data. The TS resulted in 14 reported CO exposures, from 8/27 to 9/2, compared to a prior 4 year historic mean of 3 reports for the same period. The NE resulted in 193 reported CO exposures, from 10/30 to 11/7 (4 year historic mean 7 exposures).
Discussion: In both events, case analysis majorly implicated generator use, while the NE included fire pits, charcoal grills and other substitute heating sources. It was apparent that lessons learned from the TS were forgotten during the NE, as desperate citizens had to cope with prolonged cold temperatures. In each event, the PC sent public health alerts about CO dangers and collaborated with the Department of Public Health (DPH) and other state agencies. Communication methods included Facebook, emailing and fax blasts to emergency departments. Radio interviews expanded messaging and callers reported listening in the dark on battery-operated radios. Of note, both storms resulted in higher call volume several days post-landfall. The added caseload limited communication among SPIs and resulted in staffing implications. The NE alone generated $3500 in overtime costs. Post-storm collaboration with the DPH led to expanded creation of multilingual CO materials including hangtags for house doors and better inclusion of the PC in their disaster infrastructure.

Conclusions: PCs play a vital role in providing emergent public awareness and education. Early identification of the problem was integral in alerting health partners and consumers. A final "Two Storm Panel" committee report to the Governor confirmed 13 fatalities and other medical issues as CO-related and implicated the use of portable generators and improper heat sources. Alternate communication mediums are essential in a crisis. Partnerships with media and public health agencies were critical and further strengthened. The integral position of PCs in state-wide emergency preparedness was demonstrated. Lastly, PCs may need to increase staffing several days after a catastrophic event.
Keywords: Carbon monoxide, Poison center, Public health

112. Taking it to the streets: toxicology training for emergency medical services professionals

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Objective: To develop a toxicology-related education program for Emergency Medical Services (EMS) personnel statewide.
Methods: An on-line needs assessment survey was conducted to evaluate toxicology/poisoning education needs and training format preferences of EMS agencies in our state. Based on survey results, we designed an educational program about toxidromes specific to pre-hospital care providers in a train-the-trainer format. The program was initially presented to EMS personnel at an annual conference for emergency medical technicians. Based on feedback, program content was modified and then offered to EMS instructors and trainers who are required to attend one of the semi-annual state EMS training conferences. Pre-and post-tests and program evaluations were completed at each class. The slide presentation and script were made available to EMS trainers on CD. A pocket-sized toxidrome reference booklet was produced for use by individual EMS providers. An additional survey was distributed electronically one year after launch of the training to evaluate its usefulness.
Results: More than 300 people, representing all 29 counties in the state, attended in-person train-the-trainer presentations taught by personnel from our poison control center (PCC). Scores on two pre- and post-test questions showed a significant increase in toxicology knowledge. The overall mean satisfaction score on evaluations from all trainings was 4.7 on a Likert Scale of 1–5. One hundred percent of participants who completed evaluations said they would recommend this training to a colleague. Fifty-three professionals, representing both rural and urban EMS agencies, completed the survey distributed one year after commencement of the training. Thirty-eight respondents (72%) found the training to be useful or very useful, and 33 (62%) reported they provided this training to additional EMS personnel (an average of 14 additional people trained per respondent). Twelve respondents (23%) said the training increased their use of the regional PCC. In addition, our state's largest EMS agency has made this training a requirement for both orientation and annual education.
Conclusions: We successfully developed a toxidrome training program for EMS providers who deemed it valuable and worth sharing with colleagues. The curriculum improved participants' toxicology knowledge, while the train-the-trainer format allowed for extensive statewide distribution and ongoing opportunities for local EMS trainers to provide the education to members of their agencies according to their specific needs.
Keywords: Emergency medical services, Toxidromes, Education

113. Acing the CSPI exam 2011 – development of an in-house CSPI exam prep series

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Background: To establish an in-house series of education sessions to prepare team members for the annual CSPI Exam that would be cost-effective, easy to attend, and productive of passing test scores.
Methods: Based on multiple requests from staff for a convenient, systematic, and gradual method of review for the CSPI Exam, an education program utilizing in-house resources was developed. The topics listed in the 'AAPCC CSPI Exam Objectives' were organized by the course master into a series of 13 individual 2-hour sessions. Each test candidate and other interested CSPI staff members were invited to prepare and present one of the sessions. The underlying premise was that preparing a session and its associated handouts would encourage in-depth study of the topics. Presenters utilized toxicology texts and journals, Micromedex, and on-line resources to research and develop an educational handout and supporting materials which would be presented orally. All handouts were reviewed for content by the medical director prior to making them available for study. Additionally the medical director was in attendance at all sessions for backup support and to facilitate the learning process through impromptu discussion. The series was open to all team members in addition to exam candidates. Sessions were offered by teleconference; these were held at a set time each week over a 13-week period beginning in January 2011 through April 2011. This allowed staff to anticipate the session and easily attend from any location with telephone access. Recordings from each session and associated handouts were promptly packaged and made available by email for later study to those unable to attend during the original presentation.
Results: Over the 13-week series there was overwhelming response with 144 attendances (representing 288 contact hours) for all sessions. Not only did all 7 candidates from our center who took the 2011 CSPI Exam receive passing scores, but additionally national exam results indicated that 6 of the top 11 scores in the country were from members of our center.
Conclusions: The development of an in–house education series is a cost effective, methodical approach to effective learning and successful test scores on the CSPI Exam. Far-reaching benefits from this endeavor were exemplary test scores and enhanced team engagement in the process of education regardless of whether or not a team member was taking the CSPI Exam. An additional follow-up benefit was staff demand for ongoing monthly education sessions with the same teleconferencing format, based on topics of interest submitted by members of the team.
Keywords: Education, CSPI certification, Teleconferencing

114. Evaluating a public health media campaign in a rural community

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Background: Educational efforts in rural areas can be challenging. As part of a media plan, cinema advertising may be effective for delivering a message to an attentive, captive audience.
Method: A movie theater advertisement was developed and then played in a small rural movie theater that serves two counties with a population of 37,500. The still-image advertisement included head-shots of four people of different ages and a cell phone with the poison center number. The key message was "We help people of all ages – in all types of situations." The goal of the advertisement was to raise awareness of poison center services. Poison center magnets and stickers were also provided in the theater lobby for customers to take home and place on or near their phones. Households in 25 towns within a 25-mile radius of the theater were selected for an IRB-approved random telephone survey. The phone numbers were purchased from a survey sampling company. Calls were made by a volunteer medical student from October 2011 to March 2012 between 8 a.m. and 8 p.m. (avoiding 6–7 p.m.). Participants needed to be at least 18 years old, give verbal consent to participate in the survey and have viewed a movie at this theater in the last six months. Participants were given a questionnaire with six questions that included demographics (age and gender), recall of key message (four choices were given) and changes in behavior. Three attempts were made to reach potential participants and all were documented.
Results: The volunteer attempted to call 329 people and reached 319 who were eligible to participate. Of the 319, 32% (n = 102) agreed to participate. 81% (n = 81) of participants had viewed a movie at the theater within the last six months. 77% (n = 62) of these remembered seeing the ad. 39% (n = 24) remembered correct message, "We help people of all ages – in all types of situations"; however none selected it as their only choice. There was an average of 1.9 messages chosen per respondent. Other response options included "store medicines up high and out of reach" (68%, n = 42), "keep medicines locked up" (65%, n = 40) and "program your cell phone" (21%, n = 13). Of the 62 that remembered seeing the ad, 42% (n = 26) took a sticker from the lobby but only 23% (n = 6) of those that took a sticker posted it on or near their phone.
Conclusions: Cinema advertising is effective for reaching a rural audience. This study reinforces the importance of field testing a message with a clear action step for a desired behavior change. The study was limited by targeting only landline phone users and those 18 and older for the survey. It was also limited by not specifying an action step for the magnets and stickers and not assuring their availability throughout the campaign (the supply was replenished only once).
Keywords: Evaluation, Media, Education

115. Enhancing first responder preparedness for hazardous chemical incidents: Training that combines high fidelity medical simulation with clinical decision-support tools

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Background: Effective preparedness for mass casualties requires rehearsal. Mental rehearsal and clinical experience (simulated or real) are effective in preparing responders for critical decision making and for taking appropriate clinical actions.
Objective: Describe a pilot training program that provides first responders with active learning and hands-on training using decision-support tools and high fidelity simulation. The intended outcomes for this 3 hour workshop are for learners to demonstrate competency to recognize important toxic syndromes associated with hazardous chemical accidents and terrorism and to take appropriate therapeutic actions.
Methods: The course consists of learning and practicing recognition of 5 important toxic syndromes applying the Chemical Hazards Emergency Medical Management Intelligent Syndromes Tool (CHEMM-IST: a National Library of Medicine on-line decision support tool, http://chemm.nlm.nih.gov/chemmist.htm) to a set of case scenarios. Five syndromes were used: 1) "knock down" (cyanide poisoning); 2) nerve agent poisoning; 3) chemical burns (hydrofluoric acid); 4) Irritant gas syndrome (chlorine gas poisoning); 5) acute solvent exposure (inhaled chlorinated hydrocarbon exposure). Using high fidelity simulation, learners then practiced a focused toxicological patient assessment looking for clues to chemical exposure leading to toxic syndrome recognition. Challenging simulated cases gave the learners an opportunity to recognize and treat chemically exposed, critically ill patients.
Results: 20 first responders demonstrated a good-to-strong ability to recognize toxic chemical syndromes and take appropriate actions in a simulated patient scenario after completing a 3 hour workshop.
Conclusions: Hands-on training combining high fidelity simulation with clinical decision-support tools enhances emergency preparedness by integrating knowledge into clinical practice. Medical simulation serves as a tool for learners to demonstrate competency in clinical practice.
Keywords: Public health, Cyanide, Organophosphate

116. 911: Interfacing with the poison center – working better together

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Background: A retrospective analysis of Toxicall data for 10 years (2002–2011) demonstrates that less than 1% of calls to the poison center (PC) are from 911. A strategic analysis shows the importance of addressing 911 systems in outreach. The goal is improving the value, reputation, and utilization of PC services in the 911 community by partnering with a range of 911 organizations to establish protocols and procedures for calling the PC.
Methods: A literature search was completed using keywords poison control center, emergency medical system, cost savings, benefit and cost effective. An inverted funnel format survey was sent electronically to 103 Public Safety Answering Point (PSAP) managers statewide. A contracted third party conducted 1 focus group with

12 PSAP managers and 5 key informant interviews with medical control directors. All were convenience samples and used open and closed questions.

Results: Forty six participants are managers (19) and dispatchers (27). Survey respondents place a high value on PC advice and have confidence in our service. 93% would be likely or highly likely to recommend the PC to a colleague. They like our 24/7 access; the ease, speed and dependability; and the knowledge and information we dispense. Dispatchers don't frequently call about carbon monoxide, illicit drugs, hazmat, overdoses, or patients who are not infants or children. About half do not have a policy or procedure for calling the PC. Participants indicated their training preferences and needs for clarification on when and how to call the PC.

Focus group and key informant interviews find the PC professional, responsive and capable. About half did not know whether PSAPs use PC services. Reasons to interface include better customer service; closely aligned missions; faster intervention; expert patient care; increasing safety; and saving money, time and resources. Barriers include resistance to policy change, fear of liability, narrow perception of poisoning, training costs, longer dispatch times, and bureaucracy.

Conclusions: Respondents were very satisfied with PC services but not comfortable with their current level of knowledge of the PC. In the 911 system the PC is underutilized and is not viewed as an emergency service. We are not part of the emergency medical dispatching, PSAP, or medical control worlds. Marketing implications point to the importance of building awareness of the full breadth and depth of PC services in the 911 community with special emphasis on the fact that the PC can help at any point during the 911 call per the PSAPs protocols. Notably, the findings shaped future communications with PSAPs and the design of a 2 hour training curriculum based on participant input.

Keywords: 911, Poison center, Utilization

117. Characterizing risk factors for pediatric lamp oil product exposures

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Optimal practices for preventing unintentional pediatric exposures to lamp oil have yet to be identified.

Objective: To identify commonalities and potential risk factors for unintentional pediatric lamp oil product (LOP) exposures.

Study design: Data collected from poison centers (PCs) and reported to the National Poison Data System (NPDS) regarding pediatric LOP exposure calls from 1/1/2000–12/31/2010 were reviewed for age, gender, state, exposure date, exposure site, and management site data. Regional penetrance was calculated by grouping states into regions and dividing number of exposure calls by pediatric population per region. Temporal analysis was performed by comparing number of exposures by season and Chanukah (a previously identified risk factor). In the second part of this project, five PCs administered a telephone-based survey to the parents of children from the 10 most recent LOP calls to their individual PC. Calls in which a parent or guardian witnessed a pediatric LOP ingestion were eligible for inclusion. Demographics, exposure information, behavioral traits, and health data were collected. Frequency and descriptive analysis, Chi-square analysis, and Poisson regression were performed.

Results: Among NPDS data, two years was the most common patient age reported and exposure calls increased in summer ($p < 0.0001$). States in the Western and Mid-western regions had the highest numbers of exposure calls compared to other regions. Most occurred at home, were managed in non-healthcare facilities, and had a "no effect" medical outcome. Of the 50 PC administered surveys to parents or guardians, most met inclusion criteria ($n = 47$; 94%). Notable findings which were most commonly associated with LOP ingestions included an age of 2, inadequate supervision, improper storage, and ineffective child resistant closures, whereas color and scent were not.

Conclusions: Prevention should focus on improving supervision, storage practices, and product closures. Targeting color and fragrance would likely not reduce exposures. Campaigns may have a greater effect in West and Mid-western regions. They should be intensified in summer and within communities routinely using LOP.

Keywords: Hydrocarbon, Public health, Pediatric

118. "What's in your bottle?" A survey of consumer knowledge of OTC analgesics

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Background: Over the counter (OTC) analgesics are among the most common pharmaceuticals found in households. Acetaminophen (APAP) is the leading cause of liver failure in the United States, with nearly half of these cases due to repeated supratherapeutic ingestions. Chronic misuse of APAP and other analgesics is more likely to occur when patients lack awareness and understanding of the active ingredients in their medications.

Methods: This was an oral survey conducted by Poison Center (PC) RN CSPIs at 4 retail locations in different neighborhoods of a single metropolitan area. Two were national discount department stores, one was the pharmacy at a regional supermarket chain, and one was a national pharmacy chain. Shoppers at these facilities were invited to participate if they spoke

Table 1. Results for abstract 118.

Drug name	% Correct	Number correct	Active ingredient
Bayer	68.0	153/225	Aspirin
Tylenol	64.9	146/225	Acetaminophen
Advil	49.3	111/225	Ibuprofen
Aleve	46.2	104/225	Naproxen
Total correct	26.7	60/225	

English and were ≥19 years of age. Subjects were asked to match containers of the OTC analgesics Tylenol®, Bayer®, Advil®, and Aleve® with placards listing the active ingredients "acetaminophen", "aspirin", "ibuprofen", and "naproxen". The active ingredients on the labels were obscured so participants were unable to read the ingredients. After completing the survey subjects were given Poison Center brochures, magnets, and an illustrated flyer describing the variety of medications that contain APAP.

Results: A total of 225 individuals completed the survey. Most (161) were women. Only 26.7% were able to match all 4 products with the correct ingredient, although 68% and 64.9% correctly identified the active ingredients in Bayer® and Tylenol® respectively. Survey responses are summarized in Table 1.

Discussion: Overall knowledge of the active ingredients in OTC analgesics was low, although the proportion of individuals aware that Tylenol® contains APAP was higher than in some previous studies. Participants were not queried on the adverse effects associated with specific ingredients. Our results suggest that consumers pay little attention to the active ingredients in OTC products. A survey containing less easily recognized brands or generic products would likely have resulted in even lower scores. OTC analgesics can result in significant poisoning morbidity and mortality when taken inappropriately. Poison Center programs focused on educating consumers to be more aware of the active ingredients in the OTC products they purchase may be cost-effective tools to reduce unintentional poisonings.

Keywords: Education, NSAID, Acetaminophen (paracetamol)

119. Improving patient ability to identify acetaminophen-containing products: Preliminary data of a brief ED educational intervention

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Background: A profound gap exists in patient recognition of prescription and over-the-counter (OTC) products containing acetaminophen (APAP). We investigate if a brief emergency department (ED) educational intervention can improve patients' ability to identify APAP-containing products.

Methods: Adult patients were recruited from the ED waiting room and included in the study if they were English-speaking, had basic reading skills, and in no apparent distress. Further categorization into control or intervention groups was based on the day of presentation. All participants were initially asked the generic name for Tylenol, after which they were told the answer. The intervention group then received a 5-minute teaching session guided by a visual aid, prior to taking both a written and interactive exam. Control groups did not receive teaching prior to exams. Written exam asked about target organ in APAP toxicity and asked patients to circle APAP-containing products from a list of product names. Interactive exam required subjects to physically sort bottles containing APAP from an array of OTC and prescription bottles. Total possible score from both the written and interactive exam was 20 points (10 points each).

Results: Preliminary data from 100 participants (target sample size 200) were demographically similar in age (average: 42 years) and race (predominantly African American). Average level of education was high school. Prior to any intervention, 12% of all participants knew the generic name. Intervention group scored 78% overall and 68% on the written portion. Control group scored 56% overall and 33% on the written exam. Both groups performed similarly in physically sorting bottles (control group: 79%; intervention group: 88%). While only 31% in the control group identified the liver as the affected organ in toxicity, 78% in the intervention group answered this question correctly.

Conclusions: The teaching session utilized improved patient ability to identify APAP-containing products. Interestingly, both groups may have performed similarly in the interactive portion of the exam merely from having been told the generic name initially and being able to physically look for the name on the bottles. Even very brief teaching opportunities may be beneficial. A future intervention may be including the visual aid employed in ED discharge instructions and seeing if it improves patient retention of this vital information. Although still preliminary, our data support the use of a brief educational intervention to increase knowledge about APAP-containing products and potential liver toxicity.

Keywords: Acetaminophen (paracetamol), Education, Intervention

120. Successful short and long-term educational outcomes in residents using internet toxidromes curriculum

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Background: Internet learning may replace or supplement live teaching, but there is little research on educational effectiveness of internet learning in medical toxicology. Many poisoned patients are treated by emergency physicians without toxicology subspecialty training. Medical Toxicology is co-sponsored as a subspecialty by the American Board of Emergency Medicine and many toxicologists gained interest in their subspecialty during EM residency training. Despite the relationship between med tox and EM, most EM residencies do not have a faculty toxicologist to teach residents. Our objective was to assess immediate and longer-term educational outcomes in residents from two different residencies completing an Internet learning module on the topic "Toxidromes."

Methods: We developed a self-directed Internet-based module on basic toxicologic syndromes ("toxidromes"). The learning objectives of the module were developed by a board-certified toxicologist. A pre-test, posttest, and a "follow-up" test were created with the assistance of an expert in educational assessment. Tests were formatted in 4-choice best answer format. To assess construct validity of the assessment questions, the questions (without the teaching component) were administered to board-certified toxicologists and compared to PGY1 residents without teaching. Residents at two different residencies were assigned to take the teaching modules. Pre-test and posttest were taken immediately before and after the module, respectively. The follow-up tests were administered to residents a minimum of three-months after taking the initial curriculum.

Results: The mean score of 9 attending toxicologists taking the assessments without teaching was 91.4% for the pre-, post- and

follow-up tests respectively. The mean score for PGY1 (without teaching) was 57.1%. Sixty-six residents took the curriculum. The mean pre-test scores for PGY1–4 residents was 65.1% (95% CI: 62.2%–70.0%). The mean posttest score (immediately after curriculum) was 98.7% (97.8%–99.7%). The mean follow-up score (minimum 3 months later) was 88.8% (85.3%–92.3%).

Conclusions: The difference in score between untrained PGY1 subjects and experts suggests that this assessment tool has some construct validity for measuring medical knowledge in "toxidromes" in this population. The improvement from pre-test to post-test and from pre-test to follow-up tests suggests that emergency medicine residents who took this module gained medical knowledge in "toxidromes" management and retained much of the knowledge 3 months later. Internet learning may replace or supplement live learning for EM resident toxicology education.

Keywords: Education, Internet, Toxidromes

121. Poison center collaboration with college of pharmacy to increase educational outreach

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Background: A Regional Poison Center (RPC) partnered with its host College of Pharmacy (COP) to not only provide poison prevention outreach to children and providers at daycare centers throughout the state, but also provide a service learning experience for first year pharmacy students (P1s).

Case report: After completion of their first year in pharmacy school, pharmacy students complete an Introductory Pharmacy Practice Experience (IPPE) in a community pharmacy setting. The RPC contacted the COP experiential department concerning a collaboration to provide students the opportunity for community outreach during their P1 IPPE. The COP had yet to develop a community outreach program for students, and considering there are two campuses 100 miles apart, it was felt the students should still be provided the same experience. The RPC developed a program for children ages 3–5 years of age for daycare facilities throughout the state. The RPC initially contacted the daycare centers for consent of the program. Pharmacy students were given a one hour orientation session which included information on poison center services and information on common poison exposures. They also received a packet of materials which included a script and activities for the children. The students were paired based on geographical location and presented the program during their IPPE.

Discussion: One hundred-seventy nine students completed the service learning project in the summer of 2011 at 95 sites. Programs were conducted in 13 out of 46 counties in the state. Seventy-seven percent of the students believed that the service learning project increased their ability to communicate with non-healthcare professionals. Evaluations from 54% of the daycares were returned, and of those, 100% reported the students were knowledgeable of the topic and 96% reported the students interacted well with the children.

Conclusions: The partnering of a RPC with a COP allowed for a large increase of poison prevention programs to be performed throughout the state. The pharmacy students were able to improve their communication skills with non-healthcare professionals while also learning about poison center services. Partnering with health profession schools allows for a RPC to increase its outreach efforts while providing awareness and education to future healthcare professionals.

Keywords: Education, Outreach, Pediatric

122. Results of a medicine safety program pilot targeting English, Spanish and Chinese speaking older adults

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Background: The Poison Center (PC) developed a medicine safety program targeting older adults. A patient education guidebook was created and field-tested for content in English, Spanish and Chinese. An instructor manual was created to accompany the medicine safety guidebook that emphasized teaching areas identified during the field test including the meanings of drug interactions, auxiliary labels and active ingredients. We report the findings of pilot programs conducted with English, Spanish and Chinese-speaking older adults.

Methods: Workshops were conducted by PC educators with native speaking older adults. Educational sessions were delivered with a target sample of 150 participants (50 in each language) using the medicine safety guidebook and instructor's manual. A 14 question pre-workshop survey asked 4 demographic, 5 knowledge-based, 2 behavior-based, 2 PC related and 1 workshop related question. A 15 question follow-up telephone survey repeated the pre-test with an additional question for PC number recall. The follow-up survey was conducted at least two weeks after the session by a PC educator in the participants' native language. Participants received a tote bag with PC information, a medicine box, and a magnet with the PC number. The pre and post data were analyzed (Chi-Square and T-test) for each language group using EpiInfo Version 3.5.3.

Results: Educational sessions were held reaching a total of 158 older adults; Chinese N = 81, English N = 32, and Spanish N = 45. Follow-up results (n = 70) showed that all languages showed a significant increase in knowledge about active ingredients. Chinese and Spanish participants reported increased knowledge of auxiliary labels and the qualifications of PC staff. In addition, both groups reported an increase in use of a medicine box after the intervention. The English group reported a significant increase in comfort with calling the PC about medicines and use of a medication list. The meaning of drug interactions remains a problem across all language groups.

Conclusions: Overall, the medicine safety program pilot was well received and showed improvements in both knowledge and self-reported medication management strategies across languages. PC

educators will continue to emphasize the meaning of drug interactions throughout the session and employ strategies that reinforce participant understanding.
Keywords: Education, Medicine safety, Older adults

123. A community based mobile video challenge

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Background: Poison exposure is now the 2nd leading cause of injury and death to children under the age of 5. Research suggests communities and individuals actively engaged in their health have better outcomes. Education and prevention are integral components of good health outcomes, but these messages do not reach many remote and at-risk communities because they lack cultural meaning and have been crafted for, rather than by, their intended audiences. The California Poison Control System is the largest provider of poison control services in the U.S. and constantly seeks to provide meaningful, actionable information that improves health and prevents injury.
Methods: Through an innovative approach to meeting the needs of disadvantaged and at-risk populations, the CPCS challenge sought to cultivate scalable poison prevention education initiatives in remote and underserved areas. Using a social entrepreneurship model, at-risk communities were spurred to create, and provided the opportunity to share with each other and the broader public, novel approaches to poisoning prevention education. Community groups were invited to concept poison prevention messages using cell phones, webcams or flip cameras as part of a statewide grassroots health challenge. Videos on poison prevention were collected and shared through YouTube.
Results: A panel of community members judged the submissions for cultural relevance, creativity and clarity of message. Three winners were selected and received cash prizes. All submissions are on the CPCS YouTube channel and have received nearly 2000 views.
Conclusions: Community based participatory education is a proven method to behavior modification and knowledge increase. The video challenge involved community partners and created innovative poison prevention education tools by and for the community. Research and/or practice implications: Low-cost and large reach education efforts using technology are needed to implement successful public health programs.
Keywords: Community based participatory education, Video, Mobile

124. Likes, tweets and follows: California poison control's obsessive use of social media

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Background: Social media is now integral to business, education, and even public health. There are hundreds of millions of users around the world accessing social media daily. Facebook has more than 850 million active users and accounts for 1 out of every 5 page views on the Internet. In the U.S., 72% of Internet users are on Facebook and share over 100 billion connections. Twitter has over half a billion registered users, 106 million of whom are in the U.S. Everyday, 175 million tweets and 600 million queries are generated. YouTube has 800 million users who watch 3 billion hours of video per month, more than anywhere else on online.
Methods: Lithium and Radian6 were used to mine social media for discussions around keywords "poison", "poisoned" and "poison control". Observations led to the development of Facebook pages in English and Spanish for the California Poison Control system with custom-built quizzes, badges, daily updates/tips, videos, and frequent "Ask an Expert" segments. "@poisoninfo" Twitter feed was also established for real-time news and answers to home and safety questions. A Poison Info YouTube channel now hosts videos on home safety in English, Spanish, and Hmong.
Results: There are more than 600 Facebook likes, over 600 Twitter followers and over 5000 "tweets", 2025 views on the Poison Info YouTube channel and a newly launched "Show us What's in Your Purse" campaign on Pinterest.
Conclusions: Social media is a valuable, influential, and cost-effective strategy to building and maintaining the CPCS brand, poison prevention education and receiving feedback from consumers in a diverse and geographically large state. It must, however, be part of an overall strategy and reflect an organization's goals to work.
Keywords: Social media, Educational strategy, Education

125. Use of mobile devices by low-income, low-English proficiency hispanic consumers

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Background: In 2011, reports from Google and the Nielson Company found 93% of U.S. Hispanics use a mobile phone regularly and 45% of users have smartphones. When compared to 34% of the general market, Hispanics have the highest rates of smartphone use among all ethnic groups, including Whites. Such data on the Hispanic population, however, includes acculturated and U.S.-born residents, whose adoption of mobile technology may be more consistent with the general population. Our qualitative study sought to discover the level of mobile phone use among low-income (at or below 125% of Federal Poverty), low-English proficiency Hispanics.
Methods: A qualitative study of over 100 low-income Hispanics was conducted across California, 31 of who were agricultural workers, to assess mobile device utilization. A total of 10 focus groups were conducted across 5 California markets: Los Angeles, Fresno, Bakersfield, Salinas and San Diego. Of the 10 groups, 6 were with consumers and 4 with promotoras/community health workers
Results: All but two respondents had mobile phones and over half had web-enabled phones. Agricultural workers ranked their mobile phone among their most important possessions and strongly preferred mobile over land phones. Respondents talked, texted, shared photos, used email, social media, calendars and alerts; watched and recorded videos; listened to music and played

games. Nearly all accessed the Internet primarily through mobile devices.
Conclusions: Receiving health information on a mobile phone was perceived as highly desirable. Respondents stated a preference for receiving such information via text message and indicated they would pay up to $5/month for personalized health information. This study also found that promotoras working with low-income Hispanics reported using mobile phones extensively in outreach and voiced a desire for mobile-based teaching tools. Low-income, low-English proficiency Hispanics are sophisticated users of mobile, which has important implications for outreach.
Keywords: Mobile devices, Health communication, Education

126. Learning how to get legally high

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Background: Various legislative actions have been taken to ban the sale of tetrahydrocannabinol (THC) homologs and synthetic designer stimulants such as "bath salt" type products. A previous metropolitan investigation confirmed that these products are still being sold in head shops. We examined the ease of availability and proximity to educational institutions in our city.
Methods: We used an Internet search engine (www.Google.com) to query for the terms 'head shop' and 'public schools'. The geographic locations from the search results were plotted on a metropolitan map. The distances between the schools and shops were calculated using an Internet geographical information system (www.Bing.com).
Results: Twenty-four head shops were located in our central metropolitan area. Fourteen of the shops were within 2500 feet of a school (Table 1).
Conclusions: The close proximity of these shops to schools affords students easy access and temptation. The prices of these products ($8–14.95/0.25 gram) are within many students' budgets. These factors create the perfect setting for experimenting with getting "legally high". This information can be used by poison centers to raise the awareness of government officials and public service agencies to create improved zoning regulations. Poison center education programs for schools can be directed to those within closest proximity to head shops.
Keywords: Bath salts, THC homolog, Education

Table 1. In our metropolitan area, 22 schools were located within 2500 feet of a head shop.

	Proximity of schools to head shops				
Type of school	500 feet	1000 feet	1500 feet	2000 feet	2500 feet
Elementary		5	3	1	1
Middle		2	1	1	2
High	1			1	2
University		2			

127. Comparison of high-fidelity medical simulation to short-answer written examination in the assessment of emergency medicine residents in medical toxicology

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Background: Simulation is a popular and accepted teaching tool for post-graduate education, but data are limited regarding the use of simulation as a tool for competency assessment.
Objectives: To compare the accuracy, precision, and satisfaction of high-fidelity medical simulation (sim) to short-answer written examination (written) in the assessment of emergency medicine residents (EMR) on a medical toxicology rotation.
Methods: Knowledge-based assessment tools using sim and written examinations were developed, piloted, and revised. From July 2011 to April 2012, all consecutive rotating EMR on a month-long toxicology rotation were eligible for participation in the study. On odd months, EMR were assessed using a sim case of an aspirin (ASA) overdose patient and a written examination of a tricyclic antidepressant (TCA) overdose patient. On even months, testing modalities were switched (TCA sim and ASA written). The assessment tools each contained ten knowledge-based questions. For each toxin (ASA and TCA), the scenario, questions, and answers were exactly the same; only the assessment tool (sim v. written) varied. At the end of the assessment, the EMR completed a survey. EMR were blinded to the purpose of the study. Scores were not used in the final rotation grade. Using a ten-point scale, the third author scored both the simulation and written tests to ensure consistency in grading.
Results: 45 EMR were enrolled [19 females, 26 males; PGY 2(2), 3(30), 4(10), and 5(3)]. Examination number, mean scores, and standard deviations were: ASA sim: N = 24, mean = 6.17 + /−2.32; ASA written: N = 21, mean = 7.19 + /−0.93; TCA sim: N = 21, mean = 6.57 + /−1.25; and TCA written: N = 24, mean = 6.92 + /−2.14. Both the written tests (42/45; 93%) and sim tests (45/45) were judged to be fair (as opposed to not fair) by EMR. However, on a three-point Likert scale, the sim tests had a higher satisfaction rate (40/45 very satisfied; 89%) than the written tests (22/45 very satisfied; 49%). Although only 5/45 EMR (11%) had been assessed using simulation previously, the majority (31/45; 69%) would prefer to see simulation incorporated into the American Board of Emergency Medicine (ABEM) certifying (oral) examination.
Conclusions: In this pilot study, assessment by sim has similar accuracy (demonstrated by the similar mean scores) and precision (demonstrated by similar standard deviations) but higher satisfaction rates when compared to a written examination. Most participants felt simulation should be incorporated into the ABEM certifying (oral) examination. Further study is warranted to see if these findings are reproducible.
Keywords: Education, Salicylate, Antidepressant

128. Does the training of 911 center personnel impact calls to a poison center from 911 centers?

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Background: In a recent survey conducted by Edelman Public Relations 59% of participants responded they would call 911 with a poisoning. The American Association of Poison Control Centers (AAPCC) estimates that 85% of calls to US Poison Centers (PC's) are managed by phone, reducing unnecessary hospitalizations. A study by Lovecchio F, et al., indicated that millions of dollars were saved by a PC's ability to prevent unnecessary hospital visits. Therefore, PC's may look to 911 training to ensure proper management of poisonings, while reducing unnecessary hospital visits. This study looks at a one PC's training model and at PC call volume from 911 Centers pre and post training.
Methodology: The PC developed methodology for PC/911 interface, offering it to all 911's in the PC's coverage area. Each 911 Center trained, reviewed and approved this policy to assure understanding. Over 14 months, training was conducted at twelve 911 Centers for 307 call-takers. Training described: accurate identification of a poisoning; appropriate call disposition based on Clausen procedure; appropriate utilization of the PC and effective interface. The impact of the curriculum was assessed through pre and post-testing.
Results: The pre and post-tests administered to trainees contained the same nine multi-part questions. McNemar's testing was applied to analyze responses. Most respondents accurately identified the routes of poisoning exposures in both the pre and post-test. However, significant change was demonstrated in the post-test on responses related to case disposition, i.e., emergency transport with/without calling PC. In comparing response change from pre to post-testing for all questions, the majority of responses demonstrated statistical significance and nearly 100% of the test-takers answered "correctly" following training.

To evaluate the significance of overall change in case referral pattern from the year prior to training and post-training, an analysis of variance (ANOVA) was conducted. As data were not available for two of the counties for the year prior, these counties were not included in the analysis. While a comparative of pre and post-testing seems to indicate the efficacy of 911 training, differences in PC call volume from the 911's over the three years were not statistically significant, $F(1.6, 9.6) = 1.43$, $p > .05$.
Conclusions: With mutual cooperation and appropriate training 911 staff can learn to effectively, efficiently and appropriately respond to the poison emergency call to 911. However, there seem to be many intervening variables that impact change in call volume to a PC from a 911 Center and training is only one variable. Further study may identify these variables.
Keywords: Education, 911 training, Public health

129. Mobile technology use by promotoras: A small qualitative study in California

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Background: The purpose of this qualitative research was to understand the extent to which promotoras and community health workers (CHWs) engaged in outreach to low-income Hispanics use mobile devices in their work and determine if mobile devices have a role as teaching tools.
Methods: Four focus groups (two of promotoras serving urban populations, two serving rural) with a total of 26 respondents (24 women, two men) were conducted in English and Spanish in the two largest Hispanic areas of California.
Results: Every promotora had a mobile phone. The majority had web-enabled phones and made extensive use of personal cell phones in outreach work. Promotoras accessed websites for clients, took pictures during home visits, texted information and showed photos and videos to teach. Although only three had used a tablet computer, all expressed excitement about tablets and educational applications, particularly games and interactive experiences.
Conclusions: Promotoras stated that digital content accessible on mobile devices is now essential, as it is a better fit for their teaching style and takes advantage of their Hispanic clients' communications preferences. Games with low or no text were most desirable. Many felt poorly served by and expressed frustration with the limited impact of print materials. In the words of two respondents, using mobile devices "reduces the amount of tools that we have to carry" and "makes it less boring". Promotoras look for tools that are fast, simple and concise and feel mobile devices fit these criteria. This has important implications for the development of education materials.
Keywords: Community health promotoras, Technology, Mobile health

130. Preparedness and poison control: Emergency readiness in families with special needs

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Background: There is an emerging body of scientific evidence that health care inequities exist for families experiencing disabilities; with particular vulnerability and potential for adverse outcomes in emergencies. Poison control centers address the core functions of public health, and opportunities exist for assessment, assurance, and outreach to families with special needs. The purpose of this study was to survey knowledge of poison control center resources, as part of an overall investigation of emergency readiness in those families with medical vulnerabilities.
Methods: Workshops and events on emergency preparedness, including poisoning emergencies and environmental disasters, were conducted in this IRB-approved study, using a convenience sample, at facilities serving families with special needs. Participants were family members and staff caring for those with medical vulnerabilities including physical, mental, and emotional needs. Pre and post workshop surveys were conducted, addressing poison center awareness and preparation for poisoning emergencies. The approach was three-tiered, addressing: Potential, Preparation, and Protection regarding emergency situations. Lecture content included potential poisoning scenarios, prevention and preparation strategies, and efforts for response offering the best protection in an emergency. All participants were given website resources and Poison Hotline information for use in an emergency; or to obtain poison prevention information.
Results: In the pre-presentation survey, 40% of participants reported being either "very" or "moderately" concerned about poisoning emergencies. However, 76% responded they did not know the Poison Center hotline number. After the workshop presentation, 72% of respondents reported that they would utilize poison

center resources to obtain updated information about a disaster or outbreak; and 80% reported that they would seek non-emergency prevention information on poisoning exposures. All participants received phone stickers and/or magnets, provided at the educational events and workshops.
Conclusions: Concern for poisonings in families with special needs is an area offering opportunities for expanded community outreach and education about poisoning emergencies and poison control center services. Education on poisoning emergencies for families with special needs can be effectively integrated with disaster preparation strategies. Future studies on poisoning could target this population with medical vulnerabilities to expand on further needs, services, and community penetrance.
Keywords: Public health, Poison center, Education

131. Successful short and long-term educational outcomes in residents using internet acetaminophen poisoning management curriculum

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Background: Internet learning may be a good supplement or substitute for live teaching, but there is little research on educational effectiveness of Internet learning in med tox. Many poisoned patients are treated by emergency physicians without toxicology subspecialty training. Medical Toxicology is co-sponsored as a subspecialty by the American Board of Emergency Medicine and many toxicologists gained interest in their subspecialty during EM residency training. Despite the relationship between med tox and EM, most EM residencies do not have a faculty toxicologist to teach residents. Our objective was to assess immediate and longer-term educational outcomes in residents from two different residencies completing an Internet learning module on the topic "acetaminophen poisoning."
Methods: We developed an Internet-based, self-directed module on acetaminophen poisoning. The learning objectives of the module were developed by a board-certified toxicologist. A pre-test, posttest, and a "follow-up" test were created with the help of an expert in educational assessment. Tests were formatted in 4-choice best answer format. To assess construct validity of the assessment questions, the questions were administered to board-certified toxicologists and compared to PGY1 residents without teaching. Residents at two different residencies were assigned to take the teaching modules. Pre-test and posttest were taken immediately before and after the module, respectively. The follow-up tests were administered to residents a minimum of three-months after taking the initial curriculum.
Results: The mean score of 9 attending toxicologists taking the assessments without teaching was 100%, 95.6%, and 97.77% for the pre-, post- and follow-up tests respectively ($p > 0.05$.) The mean score for 14 PGY-1 residents (without teaching) was 52.9%. The mean pre-test scores for 70 PGY 1–4 residents was 62.0% (95% CI: 57.3%–66.7%). The mean posttest score (immediately after curriculum) was 74.7% (70.0%–79.4%) The mean follow-up score (minimum 3 months) was 74.6% (69.2%–79.9%).
Conclusions: The large difference in score between untrained subjects and experts suggests that this assessment tool has some construct validity for measuring medical knowledge in acetaminophen poisoning in this population. The results of posttest and follow-up tests suggests that EM residents who took this module had a statistically significant increase in medical knowledge of acetaminophen poisoning management and retained the knowledge 3 months later. Internet learning may be a good substitute for or supplement to live learning for EM resident toxicology education.
Keywords: Acetaminophen (paracetamol), Education, Internet

132. RADARS system poison center intentional exposures to OxyContin: A look at the differences in rates of abuse via unintended routes before and after reformulation intervention

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Background: OxyContin was reformulated in August of 2010. The new formulation is intended to deter abuse through inhibiting use via unintended routes, such as chewing, injecting, and inhalation. The purpose of this analysis is to compare routes of administration before and after reformulation within the RADARS® System Poison Center (PC) Program.
Methods: The RADARS System PC data for route of administration in OxyContin abuse cases from October 2009–December 2011 were analyzed. Swallowed whole was considered "intended route of administration"; all other routes were considered "unintended". To adjust for changes in study coverage, rates per 100,000 population were calculated for each year/quarter using the 2000 US Census data. To adjust for changes in drug availability, rates per 1000 unique recipients of dispensed drug (URDD) were calculated for each year/quarter. Abuse rates were calculated by intended and unintended routes of administration. Abuse rates from before the reformulation (October 2009–September 2010) were compared with those after reformulation (October 2010–December 2011) using negative binomial regression.
Results: For unintended routes of administration, there was a 43% (95% CI: 24 to 57%, $p < 0.001$) decline in the average abuse rate per 100,000 population and a 36% (95% CI: 14 to 52%, $p < 0.002$) decline in the average abuse rate per 1000 URDD after the introduction of the new formulation. For intended routes of administration, there was a 29% (95% CI: 8 to 45%, $p = 0.004$) decline in the average abuse rate per 100,000 population and a 19% (95% CI: 0 to 37%, $p = 0.091$) decline in the average abuse rate per 1000 URDD after the introduction of the new formulation. All decreases were statistically significant with the exception of the decrease in URDD rates for intended routes of administration. The declines in unintended routes of administration rates were not significantly different from the declines in intended routes of administration.
Conclusions: Results suggest that overall abuse rates for OxyContin have declined since the introduction of the reformulated

product. However, the rate of change is not different between the routes of administration.
Keywords: RADARS system, Routes of administration, OxyContin reformulation

133. Systematic misclassification in product-specific coding of poison center data: The example of buprenorphine in the RADARS system poison center program

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Background: Misclassification of product-specific codes affects the accuracy of poison center (PC) data. Apparent differential misclassification associated with Specialists in Poison Information (SPI) choosing the first code option listed in Micromedex® has been described. Using the example of codes for specific buprenorphine formulations we sought to quantify the accuracy of product-specific coding within the RADARS® System Poison Center Program.
Methods: The RADARS System Poison Center Program captures information regarding drug exposures from participating PCs in the United States. SPIs use standardized electronic systems to record case data, including product codes and narrative notes. RADARS System staff then perform quality control checks to verify product coding accuracy. Exposures to Suboxone® tablets and oral film from the second and third quarters of 2011 were reviewed. A trained researcher reviewed the substance code and narrative notes for each case. Discrepancies were verified by a second researcher. When narrative notes and product codes were discrepant, the narrative notes in combination with substance formulation were considered authoritative. The totals of initial and final product code classification were compared using McNemar's test for correlated proportions to assess differential misclassification.
Results: A total of 1088 cases were reviewed. During the study period, Suboxone oral film was the first Suboxone formulation listed in Micromedex. During RADARS System review, 4.1% (16/395) of cases initially coded by SPIs to Suboxone tablets were re-coded to Suboxone oral film, and 56.3% (390/693) of cases that were initially coded to Suboxone oral film were re-coded to Suboxone tablets. Suboxone tablets were accurately coded in 95.9% of cases (379/395), while 43.7% (303/693) of Suboxone oral film cases were accurately coded. This differential misclassification was significant ($X^2 = 942$, df = 1, $p < .0001$).
Conclusions: Data from the National Poison Data System and individual PCs are frequently used to study adverse events related to product-specific medication use. The reliability of this research relies on accurate product coding. This study shows that differential misclassification may introduce systematic bias, in which PC data over-reports the first listed formulation in a product class. Quality control measures can identify and correct these errors. The accuracy of buprenorphine product-specific coding in PC data varies differentially. Until data verification steps are applied, PC data may over-report the first formulation listed in a product class.
Keywords: Poison control centers, Buprenorphine, Analgesics, Opioids

134. Opioid overdoses treated in the military community – complications, admission rates, and health care resources consumed

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Background: Opioid overdoses are the leading cause of unintentional death in the US and visits to the emergency department (ED) for opioid overdose have increased over the last 10 years in civilians. Military veterans also have an increased opioid overdose rate; however, clinical complications, health care resources consumed, and the proportion of opioid overdoses treated in the military ED has not been reported.
Methods: We performed a retrospective cohort study on cases evaluated in the ED of one military hospital. The military hospital is a tertiary referral center and Level 1 trauma center with an annual volume of 75,000 pts/year. Using a search of the ICD-9 codes, we obtained records of patients that were coded with overdose, suicide attempt, substance abuse, opioid use, intent of self-harm, and poisoning. We included patients with opioid ingestion with the intent of self-harm. Variables collected included demographics, military service, method of arrival, vital signs, clinical complications, hospital admission, naloxone use, and drug ingested. One trained abstractor abstracted each chart and used a standard data collection form. Periodic training was performed and 10% of cases were reviewed by a second author for accuracy.
Results: In 2011, we detected 114 opioid overdose cases. The mean age was 34.7 yrs (range 18–89), 44% were male, 51% active duty, 15% family members of active duty, and 40% arrived by ambulance. 25% of overdoses involved opioids. 18% of opioid cases received naloxone and one patient was intubated in the ED. No patient died or had cardiac arrest. Compared to non-opioid cases, opioid cases were more likely to arrive by ambulance (53 vs 36%, $p < 0.045$), more likely to be admitted (57 vs 41%, $p < 0.048$), more likely to be admitted to the ICU ($p < 0.047$), and had a longer hospital stay (4.4 days vs 1.8 days, $p < 0.014$). Incidence of hypoxia, hypotension, and bradycardia were similar between groups. Kappa score was 0.85 (0.75–0.98).
Conclusions: Opioid overdose comprised of 25% of all overdoses treated in a military ED and were more likely to arrive by ambulance, be admitted, be admitted to the ICU, and have a longer hospital stay. Opioid overdoses consumed greater military hospital resources than non-opioid overdoses.
Keywords: Opioid, Drug of abuse, Overdose

135. Misuse of prescribed opioids in a military population – is there a correlation with deployment, active duty, or combat illnesses or injury?

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Background: Opioid abuse and misuse has increased over the last 10 years in the civilian population. Recent military veterans have an increased opioid abuse rate. However, there are no reports about the military community (both active duty and non-active duty) reporting the incidence of opioid use/misuse, reasons for misuse, and correlation with deployment injuries and illnesses.
Methods: We conducted a prospective, anonymous survey in the emergency department waiting room of a military tertiary care hospital. The survey was approved by our IRB. We created an 11 question survey tool with fixed response (closed end) and multiple choice questions that included validated questions from the 2008 and 2010 DoD Survey of Health Related Behaviors. The survey was revised through a pilot sample and revision. A research nurse and emergency medicine resident used a standardized verbal script to greet subjects and then they offered the subjects the survey form to complete anonymously. Survey collection was conducted 9 hours a day over 3 weeks. Opioid misuse was defined as taking more than prescribed, obtaining opioids from others, and taking an opioid for a recent injury/illness that was prescribed for an old injury/illness. We expected a margin of error $<6\%$ and a response rate $>98\%$ based on our sample size of 500 subjects. Proportions were compared with chi-square and fisher's exact test.
Results: Thus far we have collected 300 completed surveys with a 99% completion rate. The mean age was 34.5 yrs (SD 13.6), 63% were male, 61% (CI 54–68%) were active duty, and 39% had deployed with an average aggregate deployment duration of 17.5 months. 15% of subjects reported a diagnosis of TBI and 19% PTSD. 36% of subjects reported a diagnosis of a physical injury in the last 5 yrs. 70% (CI 64–77%) had taken an opioid in the last 5 year and the most common opioids were hydrocodone (26%), codeine (23%), and oxycodone (14%). 10% of respondents had taken more opioids than as prescribed and 20% had consumed opioid pills for a recent injury/illness that was prescribed for an old injury/illness. Subjects that were active duty, diagnosed with PTSD, diagnosed with a physical injury, or who had deployed were more likely to have taken an opioid recently than non-active duty ($p<0.005$). These groups were not more likely to have misused or taken more than prescribed. Female subjects were more likely to have taken a previously prescribed opioid for a new illness or injury.
Conclusions: Opioid drug misuse occurs in the military community. In our survey, we did not detect a correlation between opioid misuse and a self-reported diagnosis of PTSD, TBI, history of deployment, or being on active duty.
Keywords: Drug of abuse, Opioid, Substance abuse

136. Alcohol use disorders and previous history of deliberate self-poisoning are associated with greater degree of toxicity in deliberate self-poisonings

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Background: Alcohol Use Disorders (AUDs) have previously been shown to increase the risk of self-poisoning however it is not known how AUDs, or the acute use of alcohol, influences the severity or outcome of an act of DSP. The Poisoning Severity Score is a standardized, previously validated and generally accepted way of comparing poisoning severity. The PSS grades severity as (0) none, (1) minor, (2) moderate, (3) severe, and (4) fatal poisoning. The PSS takes into account review of 12 specific organs/systems.
Methods: Retrospective descriptive case review of Toxicology bedside consult case records over a one year period (1/1/2011–12/31/2011) with analysis of variables including age, sex, history of self-injury, previous AUD, drug use disorder (DUD), or history of mental disorder as well as blood alcohol level (BAL) on Poisoning Severity Score and number of organ systems affected.
Results: 558 unique patient encounters were seen by the Toxicology Consult service from 1/1/2011–12/31/2011. These included 315 acts of DSP. 22% (69) of these directly involved alcohol. Mean age of DSP with alcohol involved was 36.7 years (STD 11.99). 46.7% were male. 29.3% had mild poisoning as identified by the PSS, 37.9% were moderate, 29.3% severe and there was one fatality. 70% had a previous history of DSP and 76.7% had an identifiable AUD. 50% had other chemical dependency or DUD. 93.3% had history of mental disorder. T-tests of variables and poison severity showed AUD (p 0.026) and history of self-injury (p 0.019) were independently associated with increased medical severity of the incident. AUD (p 0.008) and history of self-poisoning (p 0.003) were also shown to be associated with a greater number of organs affected in the DSP. Continuous variables including age and BAL were also analyzed for correlation with poisoning severity and number of organs affected in the DSP however neither was shown to be significant. BAL was positive 46 of 69 individuals with mean level of 86.45 mg/dL (STD 95.19 mg/dL). 17 patients had identified use of alcohol during DSP but had levels <10 mg/dL on presentation. 5 individuals did not have BAL obtained on presentation.
Conclusions: While AUD and history of DSP were shown to correlate with PSS and number of organs affected in DSP directly involving alcohol, other variables, including BAL, did not. BAL, however, is a limited measurement in terms of amount of alcohol consumed in DSP due to its rapid metabolism (e.g. 15 mg/dL/hour in average individuals) as well as an often delayed presentation to the hospital in many acts of DSP.
Keywords: Alcohol, Substance abuse, Overdose

137. Prescription opioid and stimulant use among pregnant women: Surveillance by poison control centers participating in the RADARS system

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Background: Concern is being raised about prescription drug use among pregnant women. The Florida Agency for Health Care

Administration recently reported a 388% increase in newborns in Florida being treated for withdrawal from prescription painkillers between 2006 and 2010. Similar trends have been reported in other states such as Maine and Tennessee. The purpose of the current study was to compare characteristics of pregnant and non-pregnant women who have a prescription opioid or stimulant exposure reported to Poison Centers (PCs) participating in the RADARS® System.

Methods: The RADARS System Poison Center Program collects drug exposure cases involving prescription opioids and/or stimulants, as well as demographics including pregnancy, from 48 of 57 PCs nationally. Exposure cases analyzed in this study were collected from 1Q2011–3Q2011 and restricted to ages 13–49 years. For continuous variables, t-tests were used to compare means for the pregnant and non-pregnant groups. For categorical variables, chi-square tests were used to compare proportions.

Results: Of the 17917 cases analyzed, 195 (1.11%) were pregnant women and 17722 (98.9%) were non-pregnant women. Pregnant women had a lower proportion of intentional exposures, including suspected suicide, misuse, abuse, withdrawal, and intentional unknown (70.3% vs. 76.8%) compared to non-pregnant women ($c2 = 14.1$, $df = 2$, $p = 0.0009$). For chronicity of exposure (acute, acute-on-chronic, chronic, and unknown), a higher proportion of pregnant women had acute exposures (74.9% vs. 64.0%) compared to non-pregnant women ($c2 = 20.1$, $df = 3$, $p = 0.0002$). On average, pregnant women were 4.3 years younger (95% CI 2.9–10.5 $p<.001$) than non-pregnant women (26.0 vs. 30.3 years). Pregnant women also had fewer moderate, major, and death medical outcomes (32.8% vs. 39.4%) ($c2 = 3.5$, $df = 1$, $p = 0.0619$). There was no significant difference of drug class between groups. For example, methadone exposures were similar in both pregnant and non-pregnant women (5.1% and 3.5%). Furthermore, the frequency of route mentions, such as crush/chew (2.1% and 2.3%), were similar between pregnant and non-pregnant women.

Conclusions: Information gathered by PCs suggest women who are pregnant may have better medical outcomes potentially due to a lower proportion of intentional exposures. However, the type of medication and route of administration suggests a level of abuse that is comparable between non-pregnant and pregnant women. Small cell sizes for pregnant women and lack of data on the babies born to these mothers limit some conclusions of this study. The public health impact on both mother and child warrant continued research.

Keywords: Prescription drugs, Pregnant women, Poison centers

138. Poison center efforts to decrease prescription drug abuse

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Background: In 2009, the state had an average of 8 deaths a day from prescription drug overdoses. Of the top 100 oxycodone prescribers in the USA, 93 were in this state. Statewide poison center calls regarding intentional misuse, abuse or suicide had increased 58% from 2003–2009. When poisoning became the second highest cause of unintentional injury death statewide, health agencies turned to the poison centers for explanations.

Objectives: Deaths classified as unintentional poisoning were actually due to intentional misuse or abuse by adults. Our center's education outreach had always focused on unintentional poisonings. In response to this crisis, we broadened our education mission to include prevention of intentional poisonings due to drug abuse and misuse. In 2009, we joined CADCA coalitions (Community Anti-Drug Coalitions of America funded by Drug-Free Communities grants) in three neighboring counties. Coalition members created Logic Models to decrease substance abuse problems. The poison center supported outreach to assist these efforts.

Methods: Poison center outreach methods included:

- Statewide presentations to increase awareness of the prescription drug crisis
- Lectures about synthetic drug toxidromes to medical and detox professionals
- Providing poison center data to partners
- Identification of state resources for drug abuse data
- Media interviews
- Participation at pill-take-backs, rallies and televised Town Hall meetings
- Creation of flyers about pill disposal, synthetic drugs and overdose risk reduction
- Creation of "Don't Let Them Sleep It Off " campaign to prevent overdose deaths.

Outcomes/results for the poison center: Drug coalitions adopted four poison center flyers and applied their logos. Poison center data was published in coalition *Profile of Alcohol and Drug Indicator Reports*. Counties instituted bans on synthetic drugs based on poison center data. Poison Center had increased media presence on drug-related stories.

Impact/results for the community: Our work with CADCA coalitions were part of the efforts that initiated the state Prescription Drug Monitoring Program, enacted the 911 Good Samaritan Bill, established permanent pill-drop boxes and banned new "pill mills."

Conclusions: Our poison center led the way in the state as the first injury prevention agency to provide education about prescription medication abuse. After two years of focused outreach with new partners, the poison center is an integral part of the substance abuse prevention community and is valued for credible data and health information related to emerging hazards.

Keywords: Drug of abuse, Education, Public health

139. MDMA in urban homicides from 2000–2010: It is not just a club drug anymore

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Background: MDMA is a phenethylamine with stimulant and entactogenic properties, commonly associated with dance club culture. A small pilot study demonstrated violent penetrating trauma deaths in the presence of MDMA. This study aims to further characterize this observation.

Methods: This is a retrospective study examining medical examiner (ME) records of an urban city from 2000–2010. Any case with the presence of MDMA in blood, urine or tissue was included in the study sample. Initial ELISA positives were confirmed

and quantitated with GC-MS. Additional case information was tabulated and further analysis was made using ME annual reports.
Results: 98 cases met inclusion criteria. 86/98 were male. The mean and median ages were 28 and 26 years, respectively. 55/98 cases were African-American (AA), 30 Caucasian, 9 Asian and 4 Hispanic. Homicide was the predominant manner of death (59/98), followed by accident (29/98), suicide (9/98), natural (2/98) and undetermined (1/98). Accident was the majority manner of death in Caucasian (67%), Asian (44%) and Hispanic (75%) cases. Homicide was the manner of death in 52/55 (95%) of AA cases. The cause of death in all AA homicide victims was gunshot wounds. MDMA blood concentrations were quantitated in 79 cases. The mean blood concentration of MDMA was 0.6 mg/L (median: 0.28 mg/L, range: 0.01–5.6 mg/L). In the homicide deaths, mean [MDMA] was 0.45 mg/L (n = 48, median: 0.29 mg/L, range: 0.01–2.1 mg/L). Accidental deaths had a mean [MDMA] of 0.77 mg/L (n = 25, median: 0.28 mg/L, range: 0.06–5.6 mg/L). Co-ingestants were present in 88/98 (90%) of cases. In the 27 accidental deaths with co-ingestants, 32 different drugs were identified. In the 53 homicide cases with co-ingestants, 8 different drugs were identified. The most commonly detected compounds were other amphetamines in 29/53 cases and cocaine in 25/53 cases. In all years, MDMA was encountered in less than 1.2% of all ME cases. MDMA accidental deaths constituted 0.3 to 1.3% of all-cause accidental deaths without any discernible trend from 2000–2009. MDMA homicide deaths steadily increased, accounting for 1.5% of all-cause homicides in 2000 to 14% in 2008, with a slight drop-off to 10% in 2009. Although only 1.5% of all homicide cases in 2000 were AA patients with detectable MDMA, this number increased to 13% in 2008. In 2008, 29% of all AA homicide victims tested positive for MDMA.
Conclusions: In this urban population, MDMA was found more often in homicide victims than accidental deaths. The majority of cases were young AA men who died due to gunshot wounds. Our data does not imply that MDMA toxicity is responsible for the violent deaths. Rather, it represents a new subset of MDMA users which must be examined in further studies.
Keywords: MDMA, Forensics, Trauma

140. Regional differences in seasonal trends in suicide exposures to prescription opioids as reported to poison centers participating in the RADARS system

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Background: Suspected suicide is the predominant reason coded for intentional prescription opioid exposures reported to poison centers in the RADARS® System. Extant literature suggests suicides follow a seasonal pattern peaking in late spring and early fall. We sought to determine if suicide attempts using prescription opioids followed similar seasonal patterns and if these patterns differed by latitude.
Method: Opioid exposures with a reason code of suicide were selected from 3-digit ZIP codes consistently covered from the 4 th quarter of 2006 to the 4 th quarter of 2011 by the Poison Center program. ZIP codes were used to classify cases in northern states, middle states, and southern states. Suicide exposure population rates were assessed in a negative binomial harmonic regression model. Suicide exposures were regressed on month, latitude (north, middle, and south), harmonic terms, and two level interactions of each covariate. A correlation structure that assumed higher correlation between points closer in time was used. Population was adjusted for average monthly growth with a linearized correction using state specific growth rates between the 2000 and 2010 Census.
Results: Suicide was the reason for exposure in 62,195 cases; 54.2% of all intentional exposure cases. The median reported age was 36 years (25th–75th percentile range 25–47). Females accounted for 61.2% of cases. Consistent with existing literature, the first and second harmonic terms were significant ($p < 0.05$), suggesting a seasonal pattern to suicide exposures to opioids detected using data from the Poison Center program. The interaction of the first harmonic and latitude was significant ($p < 0.05$), suggesting seasonal patterns differed by latitudes. The peak of the harmonics occurred in different times in the three different latitudes: April in the northern latitude, May and September in the middle latitude, and October in the southern latitude.
Conclusions: Suicide exposures detected by Poison Centers follow a seasonal pattern. This pattern varies by latitude with peaks generally occurring later in southern states. The differing patterns by latitude further support a general seasonal nature of suicide previously observed, but also suggest that this pattern could be related to different climatological factors in different latitudes such as the degree of which seasons change.
Keywords: Prescription opioids, Suicide, Seasonal trends

141. International trends in designer amphetamine abuse in UK and US, 2009–2012

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Objectives: Designer amphetamine (bath salts) exposures were identified as emerging public health threats in North America and the European Community. We compared the experience in the US via the American Association of Poison Control Centers' National Poison Data System (NPDS) and UK via the National Poisons Information Service (NPIS).
Methods: We examined the change over time (COT) in NPIS and NPDS health care facility human exposure telephone

Table 1. UK and US designer amphetamine HCF exposure encounters by month, 2009–2012.

				Encounters/million population served		
Country	Total	Maximum	Mean	Total	Maximum	Mean
UK-NPIS	870	128	22.9	16.7	2.46	0.438
US-NPDS	5227	521	145.2	16.4	1.64	0.457

encounters for designer amphetamines by month for the last 4 years (March 2009 through March 2012). NPIS calls queries were assigned to 1 of 12 classes while the NPDS data were assigned to 1 of 5 classes. Three product classifications were common to both coding systems: ivory wave, mephedone, and methylone. Because of this product code difference, encounters were aggregated and corrected for population (interpolated by month). We calculated the correlation coefficient for time shifted population corrected NPIS counts for 0 to 16 weeks versus the NPDS counts.

Results: Results are shown in the Table 1 for total number of calls, the maximum calls per month, and the monthly call volume means. The last 3 columns show the same data corrected for population. The UK peak of 2.46 calls per million population per month occurred in March 2010 while the US max of 1.64 per million population occurred in June 2011 (15 months later). The correlation coefficient for NPIS versus NPDS population corrected COT was −0.391 and reached a clear maximum of 0.775 ($p < 0.0001$, $N = 24$) with a 14 month shift of the NPIS COT.

Conclusions: While unadjusted aggregate exposure encounter counts were greater in the US than UK, population adjusted calls/month/million population served were similar for the UK and US. Peak exposures to these agents occurred more than a year earlier in the UK than the US. Global real-time data analysis and cooperation can provide early warning data of emerging trends. Our analyses revealed differing product categories underscoring the need for a common approach to products classification, especially for emerging products of abuse.

Keywords: Abuse, Bath salt, Epidemiology

142. FDA's ban on ephedra results in plummeting calls to poison centers

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Background: The sale of ephedra and ephedra containing products ceased in April 2004 when the Food and Drug Administration (FDA) implemented its rarely used capacity to remove a product from the market because of safety concerns. Ephedra had been used either alone or in combination with other sympathomimetics as an aid in weight reduction and as a "safe high". Cases of cardiovascular toxicity sometimes resulting in death were reported as a result of its use. The United States Court of Appeal upheld this ban in 2006. Ephedra can still be obtained from complementary medicine practitioners for the treatment of nasal congestion and wheezing.

Prior to its ban here was no standardization in strength or purity of ephedra products because they were regarded as dietary supplements. The 2 primary alkaloids in ephedra were ephedrine and pseudoephedrine. These alkaloids caused CNS stimulation, cardiovascular stimulation, and heat production. Ephedra was purported to enhance athletic performance and was abused by several professional athletes resulting in their deaths.

Methods: Toxic Exposure Surveillance System (TESS) data from 2001 through 2007 was examined to measure the number of cases of ephedra related calls to regional poison centers in the United States. The logs were scrutinized to determine how often ephedra's use resulted in a major effect and the number of fatalities that were recorded.

Results: The number of calls to poison centers related to the use of ephedra peaked in 2002 (10,326), the year it was banned, and steadily declined through 2007 (830). Major effects related to ephedra also peaked in 2002 (107) and only 4 major effects were reported in 2007. The number of fatalities from the use of ephedra peaked in 2004 (7). No fatalities were reported in 2006 while there was 1 fatality reported in 2007.

Conclusions: The FDA's ban on the sale of ephedra had a direct impact on ephedra related calls to poison centers. There was more than a 1200% decrease in the number of calls made, greater than a 2600% decrease in the severity of effects, and a 700% decrease in the number of deaths in the TESS data from 2004 through 2007. This ban was a very effective means to diminish ephedra's availability and therefore its potential toxicity within the United States.

Keywords: Drug of abuse, Cardiac toxicity, Stimulant

143. Intentional and unintentional prescription stimulant exposures in Italy, Germany and the United States

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Table 1. Results for abstract 143.

Country	Exposure type	Exposures/100,000 population 2007	2008	2009	2010	2011	% change 2007 to 2011
Amfetamine							
Germany	Intentional	0.023	0.015	0.015	0.030	0.023	0.0
Italy		0.010	0.007	0.007	0.005	0	−100
United States		0.678	1.455	1.683	2.033	2.168	219.9
Germany	Unintentional	0.008	0	0.015	0	0.008	0.0
Italy		0.005	0.005	0.002	0.003	0	−100
United States		1.026	2.198	2.667	3.080	2.918	184.4
Methylphenidate							
Germany	Intentional	0.311	0.288	0.371	0.333	0.409	31.7
Italy		0	0	0.005	0.002	0	ND
United States		0.429	0.921	0.915	0.988	1.020	138.1
Germany	Unintentional	0.326	0.394	0.394	0.462	0.515	58.1
Italy		0	0	0.002	0.002	0.002	ND
United States		1.165	2.259	2.380	2.428	2.371	103.5

ND = Not Defined; % change calculated from rate data prior to rounding.

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Background: Stimulant abuse in the US is reportedly rising yet abuse in other countries is not well studied. This study provides abuse rate data for two stimulants over a 5 year period in Italy, United States (US) and Northwest Germany.
Methods: Human exposures to methylphenidate and amfetamines from 2007–2011 were obtained from Milan and Göttingen PCs. US data were obtained from PCs participating in the RADARS® System. PCs in all 3 countries manage calls from healthcare providers as well as the public. Milan PC handles 65–70% of all calls in Italy. Rates are expressed as number of exposures per 100,000 population separately for intentional and unintentional exposures. Intentional exposures are those with a reason of suicide, abuse, misuse, and unknown intentional. Unintentional exposures include unintentional general, unsupervised ingestions and therapeutic errors.
Results (Table 1): In Italy, trends for amfetamines and methylphenidate for both intentional and unintentional exposures showed no clear trends over the 5 year time period. Since 2009–2010 amfetamine drugs were gradually retired as prescribed drugs in Italy. In Northwest Germany, amfetamine exposure trends were variable with no change in the rates of intentional or unintentional exposures from 2007 to 2011. For Methylphenidate intentional exposures rates for 2011 were 32% increased from 2007 levels and unintentional exposures were 58% increased from 2007 levels. In the US increasing rates are seen for both intentional and unintentional amfetamine and methylphenidate rates.
Conclusions: Amfetamine exposures reported to PCs appear to be on the rise only in US, not Italy or Northwest Germany. However, intentional and unintentional methylphenidate exposures reported to PCs appear to be on the rise in US and Northwest Germany. Stimulant abuse does not appear to be a pressing issue in Italy based upon calls to PCs. Transatlantic data collection is possible with comparably few resources.
Keywords: Poison center, Stimulant, Prescription drug abuse

144. Synthetic cannabinoid laced brownies

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Background: Synthetic cannabinoids (SC) are emerging designer drugs of abuse. Most reports on the health effects of these drugs are case reports. Marijuana has classically been used via many routes of exposure including oral, such as in brownies. We report on eleven symptomatic patients who unknowingly ingested brownies laced with analytically confirmed SC.
Case series: The toxicology service was asked to see eleven workers from a unit at a major hospital who were brought to the emergency department (ED) following ingestion of brownies brought to work by one of their colleagues. The brownies were allegedly doctored by the son of the lady that brought in the brownies. The brownies were eaten by staff during their lunch break.

All 11 employees who ate the brownies had been well until symptoms began 30–60 minutes following the ingestion and arrived to the ED within 2 hours of symptom onset. There were 5 males and 6 females; age range 20–57 years. Neuropsychiatric and cardiovascular symptoms predominated: memory impairment (91%, 10/11), inappropriate giggling (36%, 4/11). All the patients had lightheadedness, perioral and facial numbness and tingling sensation, dry mouth, difficulty focusing/blurring of vision, and sluggishness. One patient had 5 mm pupils; all the others had normal pupils. No patient had depressed consciousness. Two patients had heart rates >100 and 4 of 11 (36%) had BP $>140/90$. One patient had chest pain. None were short of breath and there were no gastrointestinal symptoms. All the symptoms were completely resolved four hours following their onset except two patients had ongoing weakness and fatigue.

All patients had negative urine drug screens, ethanol, acetaminophen and salicylate concentrations; as well as normal EKGS and basic metabolic panels. All the patients were discharged from the ED in stable condition within ten hours of the exposure.

The remaining brownie was analyzed by Department of Public Safety laboratory. Preliminary analysis showed that it contained the synthetic cannabinoid AM 2201.
Discussions: To our knowledge, this is the first case series of symptomatic ingestion of brownies laced with SC. These patients were unaware of the contents of the brownies but they all shared common clinical features, mostly neuropsychiatric and cardiovascular symptoms. These cases reflect that oral SCs' can cause adverse effects and that they do not degrade with cooking in an oven.
Conclusions: Oral exposure of eleven patients to brownies laced with analytically confirmed SC resulted in neuropsychiatric and cardiovascular symptoms. This series reflects that like Marijuana, oral exposures to SC can lead to symptoms.
Keywords: Synthetic cannabinoid, Brownies, Neuropsychiatric symptoms

145. Fatalities from acetaminophen combination products reported to poison centers

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Background: Supratherapeutic dosing or intentional overdose of acetaminophen (APAP), single or combination products, can cause hepatoxicity or death. The objectives of the study are to determine the relative contribution of APAP compared to other active drugs in fatalities involving APAP combination products and related risk factors.
Methods: A 10-year retrospective analysis of NPDS fatality abstracts of only undoubtedly responsible and probably responsible cases was conducted. Four raters independently examined the abstracts for the cause of fatality as most likely due to APAP, the other active drugs, or both.
Results: Interrater reliability was substantial with overall Kappa of 0.74. Of the 339 cases, 59.9% involved APAP-opioid, 30.0% APAP-antihistamine, 3.6% cough-and-cold/headache/other pain relief products, and 6.5% more than one APAP combination product. The raters determined that the overall fatality was caused by

Table 1. Results for abstract 145.

	Suicides (n = 231) No. (%)	Nonmedical Use (n = 65) No. (%)
Chronicity		
Acute	177 (76.6)	10 (15.4)
Acute-on-Chronic	29 (12.6)	16 (24.6)
Chronic	18 (7.8)	38 (58.5)
Unknown	7 (3.0)	1 (1.5)
Overall fatality contribution rating categories		
Category 1: Acetaminophen	125 (54.1)	57 (87.7)
Category 2: Other drugs	78 (33.8)	7 (10.8)
Category 3: Both	28 (12.1)	1 (1.5)
Category 1 products		
Opioid	46 (36.8)	43 (75.4)
Antihistamine	71 (56.8)	6 (10.5)
Cough and Cold or Headache/other pain	3 (2.4)	1 (1.8)
More than one product	5 (4.0)	7 (12.3)
Category 2 products		
Opioid	58 (74.4)	6 (85.7)
Antihistamine	10 (12.8)	–
Cough and cold or Headache/other pain	5 (6.4)	–
More than one product	5 (6.4)	1 (14.3)

APAP in 60.3%, other drugs in 29.7%, and both drugs in 10.0% of cases. APAP was responsible for death in 79.7% of chronic use cases. For acute or acute-on-chronic exposures, APAP and other drugs were responsible for deaths in 55.8% and 34.5%, respectively. Most deaths were due to suicides (68.1%) and nonmedical use (intentional misuse/abuse) (19.2%). See Table 1 for comparison of suicides and nonmedical use deaths.

Conclusions: The majority of deaths are attributed to APAP. Products containing antihistamines and opioids are most often involved in suicides and nonmedical use, respectively. The proportion of deaths from the non-APAP drugs increases when the chronicity is acute or acute-on-chronic.

Keywords: Acetaminophen (paracetamol), Overdose, Death

146. Status epilepticus after acute endosulfan poisoning: A study of 25 cases

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Background: Endosulfan, a chlorinated hydrocarbon, is a widely used insecticide with a high mortality rate. The toxicological effect mainly results from over stimulation of the Central Nervous System (CNS) by inhibiting Ca^{++} and Mg^{++} ATPase and antagonizing chloride ion transport in gamma-aminobutyric acid (GABA) receptors. Due to its easy availability, it is emerging as a common self-poisoning agent in adults.

Method: All calls to the Nepal Drug and Poison Information Center (NDPIC) involving endosulfan ingestion resulting in status epilepticus (SE) during the period of March 2011 to February 2012 were included in the study. Data entry and analysis was done using SPSS 16.00.

Results: A total of 25 exposure cases were reported to the NDPIC. There were 14 females (56%) and 11 males (44%). Ages ranged from 15 to 61 years with a mean age of 31.8 years (± 1.3 years). Time post exposure ranged from 30 to 120 minutes (median = 60 minutes). Reported dose ingested ranged from 25 to 70 grams (mean = 34.6, median = 35). Besides SE, other initial presenting symptoms were abdominal pain, vomiting, dizziness and headache. Thirteen (52%) patients developed Refractory Status Epilepticus (RSE) and, of which eight (61.5%) died. Development of RSE was significantly associated with mortality (OR: 2.6, 1.3–5.2). However, there was no significant association between sex, time post exposure and amount ingested with mortality.

Conclusions: Endosulfan poisoning continues to carry a high case-fatality rate in resource-limited settings. Patients who develop seizures should be treated aggressively in the early stages of endosulfan poisoning to prevent status epilepticus. More research is needed to identify high-risk features for patients with organochlorine pesticide poisonings.

Keywords: Insecticide, Seizure, Poison center

147. Outcome analysis of exposures to methylsalicylate-containing liniments

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Background: Practitioners appropriately regard methylsalicylic acid (MeSA), the notorious constituent of oil of wintergreen, with respect and trepidation. Topical balms such as Ben Gay™, Icy Hot™, Thera-gesic™, and Flexall™ may contain up to 30% MeSA. Just 5 ml of a 30% preparation provides as much as 150 mg/kg of salicylate to a typical toddler, so even a trivial ingestion could theoretically produce toxicity. Outcomes from human exposures to these liniments are not adequately characterized.

Methods: The National Poison Data System (NPDS) was queried for all closed human exposure cases given AAPCC generic codes 041790 (MeSA) or 008790 (camphor and MeSA in combination), between 1/1/2000 and 12/31/2011. Cases involving "Oil of Wintergreen" (98% MeSA) were excluded because of the well-described toxicity of this highly concentrated and bioavailable preparation, relative to topical liniments. Special attention was given to the analysis for the population at greatest risk for unintentional, single, acute ingestion (<6 year-old age group).

Results: 98% of all exposures in NPDS involve unintentional, predominantly pediatric, ingestions. Only 10% were evaluated in a health care facility.

Conclusions: Twelve years of NPDS data suggest that there is little, if any, risk of death after ingestion of available MeSA liniments. There appears to be extremely low risk of acidosis or other major clinical effects after ingestion. While most cases involved uninten-

Table 1. Results for abstract 147.

	# of exposures reported to NPDS	Incidence of acidosis	% coded as "Major Effect"	Deaths
All ages	133,221	13 (0.01%)	27 (0.02%)	None
<6 years old	108,462 (81%)	4 (0.004%)	9 (0.008%)	None

tional ingestion by young children, risk appears nominal in other age groups as well.

Professionals should appreciate the incomplete consistency of coding and of follow-up practices among reporting centers. And appropriate caution should be taken not to over-interpret the value of retrospective clinical (non-research) data for prospective decisions about case management. Nevertheless, routine ED referral for such exposures does not appear warranted in the vast majority of cases.

Keywords: Salicylate, Methylsalicylate, National Poison Data System

148. An 11-year retrospective comparison of dihydropyridine and non-dihydropyridine calcium channel blockers

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Background: Calcium channel blockers (CCBs) are the leading cause of fatal cardiovascular drug poisoning, representing approximately 40–50% of cardiovascular drug-related deaths reported to the American Association of Poison Control Centers (AAPCC). CCBs are categorized as dihydropyridines (amlodipine) versus non-dihydropyridines (verapamil) with the latter commonly considered responsible for most CCB-related deaths. Although many view dihydropyridine poisoning as inconsequential when compared to the non-dihydropyridines, we hypothesize that dihydropyridine CCBs are associated with substantial morbidity and mortality.

Methods: We conducted an 11-year retrospective review of data from one Poison Control Center encompassing all calls received between January 1, 2000 and December 31, 2010 for reported poisoning exposures from CCBs. Exclusion criteria included information calls, calls from outside health care facilities, non-human exposures and cases where the outcome was unknown. Remaining cases were separated into dihydropyridines versus non-dihydropyridines. Clinical effects were categorized as no effect, minor effect, moderate effect, major effect, and death based on the definitions established by the AAPCC. We dichotomized no, minor and moderate effect as good outcome and major effect/death as poor outcome. We then performed a chi square test statistical analysis.

Results: A total of 1346 cases of calcium channel blockers met inclusion criteria. There were 863 cases of dihydropyridine versus 483 cases of non-dihydropyridine CCBs. P value for chi square test was < 0.001. Table 1 summarizes our results.

Conclusions: Our 11-year retrospective study show that while non-dihydropyridine CCBs are associated with morbidity and mortality, contrary to many clinicians' belief, the dihydropyridine CCBs are also associated with morbidity and mortality. Dihydropyridine CCB poisoning should not be considered benign and should be treated appropriately.

Table 1. Results for abstract 148.

	Good outcome	Poor outcome (Deaths)	Total cases
Dihydropyridines	748	115 (14)	863
Non-dihydropyridines	341	142 (19)	483
			1346

Keywords: Calcium channel blocker, Poison center, Epidemiology

149. Fatal cesium chloride toxicity after subcutaneous administration of an oral preparation

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Background: Cesium Chloride (CsCl) is sold as a treatment for several types of cancers. The purported mechanism of action is alkalinization of relatively acidic neoplastic cells. The efficacy of CsCl has not been demonstrated in controlled experiments. Oral and IV CsCl use has been associated with seizure, cardiotoxicity, syncope, and death. However, this appears to be the first reported case of CsCl toxicity secondary to subcutaneous injection.

Case report: A 61 year old woman presented in cardiac arrest 20 hours after subcutaneous use of 9 mL of an oral CsCl preparation. The patient had been taking oral CsCl, selenium, silymarin, vitamin D, folic acid, and potassium for one year, to treat a breast mass. She was treated with ACLS and had return of pulses. She received lidocaine, amiodarone, and magnesium prior to transfer to an intensive care unit. During transfer, she suffered multiple episodes of ventricular tachycardia requiring cardioversion. On arrival to the ICU, her vital signs included HR 58 and BP 110/61. Her initial ECG showed sinus bradycardia at 45 bpm and a QT of 744 ms. She had disconjugate gaze and decorticate posturing. She was ponatremic and hypokalemic. She was treated with amiodarone, magnesium, and rapid correction of hyponatremia and hypokalemia. She was also treated with Prussian Blue 3 g TID. Debridement of the injection was recommended, but refused by her family. The patient remained in sinus rhythm for the remainder of her course except for one episode of atrial fibrillation that resolved with metoprolol. Her QT decreased to normal on day 4 after exposure. Unfortunately, she remained in a neurovegetative state and died 11 days after exposure. Serum cesium levels drawn on day 3 after exposure were: whole blood 100,000 mcg/L (ref. range < 10 mcg/L), plasma 27,000 mcg/L (ref. range < 10 mcg/L) , and urine 270,000 mcg/L (ref. range < 20 mcg/L). Her serum selenium level was 163 mcg/L (ref. range 23–190 mcg/L).

Case discussion: Cesium Chloride is widely sold as an alternative cancer treatment despite a lack of demonstrated efficacy and clear evidence of harm. Cesium causes QT prolongation and arrhythmia via by blocking potassium rectifier channels on atrial and ventricular myocytes. To our knowledge, this is the first reported case of cesium toxicity secondary to subcutaneous administration. Prussian Blue has been demonstrated to increase elimination of radioactive and non-radioactive cesium. However, this patient had already suffered anoxic brain injury and was unlikely to benefit from increased elimination. Likewise, debridement of the injection site may have decreased her exposure, but it is unlikely that she would have had any increased recovery in neurologic function.

Keywords: Fatal, Cesium, Supplement

150. Energy drink exposures in the American Association of Poison Control Centers (AAPCC) National Poison Data System (NPDS) database

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Background: Energy drink (ED) consumption is raising health concerns, particularly the rates of consumption and physiologic effects in children and adolescents. There are reports associating adverse events with ED exposure, but there are no systematic studies. In 2010, AAPCC began tracking ED exposures reported to poison centers.
Methods: Retrospective case series. We included all human exposures to EDs reported to the AAPCC NPDS database from 10/1/10–9/30/11. EDs were categorized as: Alcoholic (AED) or non-alcoholic (NAED); caffeine-containing (CC); containing caffeine from a single source (C); and containing caffeine from multiple herbal additives (C+). Cases were also categorized as single product (SP) exposures when the only substance of exposure was an ED product. Non-CC sports drinks, and ED exposures to substance of unknown formulations, were excluded.
Results: There were 4,854 total ED exposures reported to the NPDS database. Of those, 1,480 (31%) were exposures to SP, CC, NAEDs and 182 (4%) to AEDs. Compared with all human exposures in NPDS, the SP, NAED exposures were disproportionately male (61% v 48%) and also disproportionately male in preteens (71% v 58%) and teens (60% v 46%). Compared with all human exposures, there was a disproportionately greater percentage of ED exposures in pre-teens (11% v. 6%), and adolescents (26% v 7%), and also a disproportionately greater percentage of cases coded as an intentional exposure (26% v 15%). Overall, a greater proportion of patients were managed in a healthcare facility (31% v 25%) but a smaller proportion were admitted (3% v 9%) (all prior comparisons, $p < 0.0001$). There were no significant outcome differences between C and C+ exposures, suggesting that clinical effects were solely the result of caffeine content. When SP, CC, NAED outcomes were compared with the overall database, there was no difference in the proportion of major outcomes but there was an increase in the proportion of moderate outcomes. Major effects included ventricular dysrhythmias (1), other dysrhythmias (2), hypertension (1), seizures (3), and tachypnea (1). There were no deaths following a SP, ED exposure. There were two deaths in cases involving multiple substances that included EDs.
Conclusions: Energy drink exposures reported to poison centers disproportionately involve males, pre-teens and adolescents, and were disproportionally intentional, relative to their representation in the overall NPDS database. Clinical effects, some major, appear to be secondary to the caffeine content. The apparent increased incidence of ED exposures in pre-teens and adolescents relative to other exposures is a cause for concern.
Keywords: Epidemiology, Pediatric, Stimulant

151. Toxic alcohol and glycol poisoning in the UK - a 12 month prospective follow-up study

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Background: Enquiries regarding poisoning with ethylene glycol or methanol are frequently referred to a consultant clinical toxicologist in the UK, because of diagnostic difficulty, severe toxicity or inconsistent availability of specific assays and antidotes. This prospective study was undertaken to investigate the epidemiology of systemic exposures reported to the National Poisons Information Service (NPIS).
Methods: Details of exposures to products containing toxic alcohols and glycols reported to NPIS were collected from 1 January 2010 to 31st December 2010 and cases of significant systemic exposure were followed up to obtain information on antidote use and patient outcome.
Results: There were 608 enquiries about toxic alcohols and glycols exposures, involving 488 individuals over the 12 month period. 309 (63%) patients were male and 89 (18%) were under 5 years of age. Exposures were mainly by ingestion ($n = 434$; 89%) and occurred mainly at home ($n = 431$; 88%). There were 328 (67%) accidental cases, 119 (24%) intentional cases and 4 cases of recreational use. The most common products were surgical spirits, antifreeze and screenwash products with ethylene glycol identified as the most common ingredient. At the time of the enquiry 409 (84%) patients had no or minor symptoms and 71 (15%) had moderate/severe symptoms using the Poisons Severity Score. Enquiries about 238 exposures originated from hospital, of which 182 cases met the criteria for case follow up. Follow-up was not undertaken in 9 cases and was incomplete or failed in a further 51 cases. The majority ($n = 112$) of the 122 patients for whom outcome data were available made a complete recovery, 5 had residual renal impairment and there were 4 deaths. Antidote treatment was provided in 99 patients and extracorporeal treatment (haemodialysis or haemofiltration) in 33 patients.
Conclusions: Serious poisoning with toxic alcohols and glycols occurs infrequently in the UK and is associated with morbidity and mortality in a small number of cases. Exposures are predominantly acute ingestions involving ethylene glycol and occur more frequently in males. The majority of patients show few symptoms at the time of the enquiry and most make a complete recovery.
Keywords: Ethylene glycol, Methanol, Epidemiology

152. An elderly woman with trismus and painful spasms

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Background: Trismus associated with painful spasms in the setting of normal mental status has a narrow differential diagnosis of tetanus, strychnine poisoning, neuroleptic malignant syndrome, dental infection and stiff person syndrome. We present a case of a

91-year-old female who presented with left lower extremity infection, trismus and painful spasms.

Case report: A 91-year-old Hispanic female with history of hypertension and diabetes presented with a 2-day history of dysphagia and inability to open her mouth with pain and spasms of her neck and jaw. She had just come from Mexico.

Two weeks before her presentation, she cut her left leg and a scrap of metal was removed. She did not receive antibiotics or a tetanus booster. There was no history of dental infection, fever or ingestion of rat poison. She was not on antipsychotic medications. Her immunization history was unclear.

Initial vital signs were BP 212/119 mm Hg; HR 123 beats/minute; RR 14 breaths/min; temperature 35.4°C; O_2 saturation 79% on room air, 90% on a non- rebreather mask on 15L O_2. She was alert and had board-like rigidity of the neck and spine as well as intermittent spasms. Her oropharynx could not be examined due to her trismus. She had partially healed wounds on the lateral left leg.

She was intubated and transferred to the ICU. She received a provisional diagnosis of tetanus and was given tetanus immune globulin (TIG) and tetanus toxoid vaccine (Td). Her wound debrided by the surgical team and she was started on metronidazole, zosyn and vancomycin.

Blood and urine strychnine testing was negative. Her tetanus antibody concentration was 0.100 IU/mL (reference[3] 0.1500 IU/mL); creatine kinase at its highest level was 1869 units/L; and her wound culture was negative for *Clostridium tetani.* No radio-opaque foreign bodies were found on x-rays of the left leg

Her hospital course was complicated with ventilator-associated pneumonia and bacteremia. Due to her poor prognosis, her family decided to withdraw care and she died 16 days post-admission.

Discussion: Tetanus is rare in the developed world. Most of the cases in the US are in recent immigrants or visitors. It is usually a clinical diagnosis once other causes are excluded. Our patient had generalized tetanus as evidenced from her clinical presentation. Her tetanus antibody level was low suggesting lack of immunity and she had undetectable strychnine concentration in her blood and urine thereby excluding strychnine poisoning.

Conclusions: Tetanus is an uncommon toxin induced disease in the developed world that is often lethal despite therapy. Because tetanus is rare in the US, diagnosis and management are often a challenge to clinicians.

Keywords: Trismus, Spasms, Tetanus

153. TOXINZ: comparison of pharmaceutical searches by subscribing countries

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Background: TOXINZ (www.toxinz.com) is an Internet based poisons information database containing in excess of 190,000 chemicals, pharmaceuticals, plants and hazardous creatures. Used throughout Australasia and by Poisons Centres in over 20 countries, the product has recently been made available via subscription access in Canada. This has made it possible to compare the drugs that are most accessed in Australia, New Zealand and Canada to determine if there are differences.

Methods: User data collected from 1 April 2011 to 31 March 2012 from the three countries was extracted then analysed using MS Excel® and the ATC (www.whocc.no) classification system. Statics for international use were downloaded from Google Analytics (www.google.com/analytics) for the same period.

Table 1. Comparison of pharmaceutical searches by subscribing countries.

	Australia		New Zealand		Canada	
	Drug	%*	Drug	%*	Drug	%*
1	Acetaminophen	7.9	Acetaminophen	14.3	Acetaminophen	7.9
2	Quetiapine	4.7	Quetiapine	7.3	Quetiapine	2.4
3	Valproate	2.2	Zopiclone	3.6	Venlafaxine	2.0
4	Venlafaxine	1.7	Citalopram	3.5	Aspirin	1.7
5	Duloxetine	1.6	Venlafaxine	2.6	Bupropion	1.5
6	Sertraline	1.6	Codeine	2.6	Digoxin	1.5
7	Amitriptyline	1.5	Ibuprofen	2.5	Ibuprofen	1.4
8	Ibuprofen	1.5	Fluoxetine	2.2	Citalopram	1.4
9	Diazepam	1.5	Valproate	1.9	Amitriptyline	1.2
10	Lithium (Therapeutic)	1.5	Amitriptyline	1.8	Dimenhydrinate	1.2

*Percent of drug searches.

Results: During the period, TOXINZ managed 80,007 visits from 26,379 unique visitors providing 380,223 page views. In Australia, 39,365 clinical management documents were accessed with a further 25,228 and 5,352 viewed in New Zealand and Canada respectively. Pharmaceuticals accounted for 58.3% (22,961), 66.8% (16,862) and 70.1% (3751) of the total each for Australia, New Zealand and Canada. Table 1 lists the 20 top clinical monographs accessed for single pharmaceuticals in each country.

Conclusions: It has been identified that the top 20 pharmaceuticals viewed by Australia, New Zealand and Canada are broadly similar. The greatest variation was noted in Canada where the database had only been available via subscription for four months; as use increases this variance may reduce. While acetaminophen has historically been at the top of such tables, the appearance of quetiapine as the second most accessed toxicology advice document in all three countries is striking. While it should be recognised that use of this type of resource can only infer the rate of differing types of poisonings, systems such as TOXINZ have potential to provide ongoing monitoring of clinical toxicology challenges as they arise in a range of user countries (e.g. novel recreational drugs, new pharmaceutical agents, etc.).

Keywords: TOXINZ, Database, Information

154. Variation in suicide occurrence by day and during major American holidays

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Background: Because of the observed seasonality and daily variations in completed suicides, it has been suggested that environmental factors and how they relate to mood may play a role in suicide. Suicide attempts may be more influenced by impulsivity and environmental factors. The purpose of this study is to determine if particular days of the week, months, or holidays were associated with increase number of suicide attempts by poisoning.

Methods: All calls recorded in the National Poison Database System that were coded as "suspected suicide" during the period

1/1/2006–12/31/2010 were included. The Newman-Keuls test was used to determine if exposures were reported more commonly during a day of the week. The results were also stratified by age group: <13, 13–19, 20–39, 40–59, 60–79, >80. In addition, the number of exposures occurring on the following holidays: New Year, Martin Luther King Jr. Day, Valentine's Day, Easter, Mother's Day, Memorial Day, Father's Day, Independence Day, Labor Day, Thanksgiving, and Christmas, as well as the 1 day (3 days inclusive) and 3 (7 days inclusive) days before and after each holiday were compared to three control dates, March 15, June 15 and September 15.

Results: There were 1,065,067 exposures during the study period for an average of 583 per day. Deaths occurred in 3790 (0.4%). Females were 63% of the total. Sundays (617/day) and Mondays (614/day) were the most common and statistically different from the other days but not from each other. Fridays (540/day) were the least common. Sundays and Mondays remained the most common days for all age groups except for the <13 and 13–19 age group in which Mondays and Tuesdays were the most common. New Year days (668/day) were associated with higher number of exposures when compared to control days (598/day). Independence (544/d), Thanksgiving (491/d), and Christmas (456/d) days were associated with lower number of exposures. All others were not statistically different. None of the 3 or 7 day periods surrounding each holiday were associated with higher number of exposures when compared to 3 and 7 day periods around control days. Independence (3 day), MLK (7 day), Father's (7), Thanksgiving (3 and 7 day), Christmas (3 and 7 day) day periods were associated with lower number of exposures when compared to 3 and 7 day periods around control days.

Conclusions: The beginning of the week was associated with higher number of suicide attempts which has been similarly seen in studies on completed suicides. Holidays had relatively little effect on suicide attempt with more periods around holidays having lower number of attempts. This has implications for clinicians and therapists for advising patients and potential victims.

Keywords: National Poison Data System, Suicide, Overdose

155. Risk identification of eye injuries in Germany - the importance of the brandname for clear product identification

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Background: The BfR Documentation and Assessment Centre for Poisonings (BfR-DoCCentre) as part of the toxicological network with German Poison Centres is dealing in general with a broad range of acute intoxications. Eye injuries account for the foremost share of reports by physicians under §16e Chemicals Act (ChemG).

Method: A specific German analysis of products involved in eye injuries was carried out for all cases reported in 2010. The study has analysed the regular summary reports of the BfR-Product Information System (PRINS) and included medicinal products as well as chemical products. The severity of health impairment was assessed by the Poison Severity Score.

Results: For only 282 (12.4%) of the 2266 cases of eye injury reported to BfR in 2010, the product involved was specifically named and could be clear identified on a formulation level. Of the 282 products reported, 264 (93.6%) had caused minor eye injuries. Six (2.1%) cases were classified as moderate and for another six (2.1%), a degree of severity was not specified or could not be assessed. In the category of severe eye injuries, no definite specific product names were reported. The moderate eye injuries had been caused by an agricultural surface disinfectant (quicklime), an herbicide, a commercial surface/metal cleaner, a hardener and a two-component adhesive, respectively. Among the causative agents of the cases of minor severity, disinfectants (102 cases, corresponding to 38.6%) and cleaning agents (97 cases, corresponding to 36.7%) were predominant. Of the disinfectants involved, ca. 40% represented surface and hand disinfectants and 20%, instrument disinfectants. In 21 out of 264 (7.9%) cases, eye injury had been caused by medicinal products. Herbicides and pesticides were reported to have been involved in 17 (6.4%) of the cases of minor severity. The cases of eye injuries lacking data on the degree of severity had been caused by a medical sclerosing agent, a cosmetic and food additive, a commercial dishwashing detergent, an instrument disinfectant and an agricultural surface disinfectant. Other cases that were not assigned a degree of severity included three reports involving an alcoholic hand disinfectant, two involving two surface disinfectants and one involving a crack filling material used as an auxiliary product in the construction sector.

Conclusions: The BfR study on cases of eye injuries showed that only in a minor part of the reported cases, the products having caused these accidents could be systematically analysed on a formulation basis. The clear brand name of the product involved is highly important for an early recognition of risks and related preventive measures. This should be improved.

Keywords: Eye injuries, Intoxication, National Poison Data System

156. Major morbidity and death risk in older adults with suicidal intent

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Objective: To describe suicide cases in older adults reported to U.S. poison centers over a 10-year period and to estimate the relative risk of major morbidity and death for pharmaceuticals as compared to non-pharmaceuticals following single-substance and multiple-substance exposures.

Methods: Data from the National Poison Data System involving individuals >60 years with a reported exposure to a pharmaceutical or non-pharmaceutical substance and suicide as the exposure reason were analyzed. Sample demographics and medical outcomes were described. The probabilities of major morbidity and death were calculated for single-substance and multiple-substance exposures to pharmaceuticals and non-pharmaceuticals. Relative risk of major morbidity or death was estimated for the pharmaceutical compared to non-pharmaceutical substances for single-substance and multiple-substance exposures.

Results: The analytical sample included 46,494 cases. The majority involved pharmaceuticals (92.3%), single-substance exposures (53.3%), and females (62.2%). Females comprised 63.8% of pharmaceutical and 43.5% of non-pharmaceutical cases. For single-substance

pharmaceutical exposures, the probability of major morbidity (91.9/1000 exposures) was significantly lower compared to non-pharmaceuticals (170.9/1000 exposures) [RR = 0.54 (95% CI: 0.49–0.59)] as was death (12.9 vs. 52.7/1000 exposures) [RR = 0.25 (95% CI: 0.20–0.30)]. In contrast, the probability of major morbidity from pharmaceuticals (156.7/1000 exposures) was similar to that for non-pharmaceuticals (169.9/1000 exposures) [RR = 0.92 (95% CI: 0.80–1.06)] for multiple substances. The probability of death from pharmaceuticals (20.5/1000 exposures) was significantly lower compared to non-pharmaceuticals (37.1/1000 exposures) [RR = 0.55 (95% CI: 0.40–0.77)] in the multiple-substance group.

Conclusions: In poison center cases, pharmaceutical agents were more likely to be used in suicide attempts by older adults. For single-substance exposures the relative risk of major morbidity or death was lower with pharmaceuticals when compared to non-pharmaceuticals. The relative risk of death following multiple substance exposures was also lower with pharmaceuticals when compared to non-pharmaceuticals. However, for multiple-substance exposures there was no difference in the relative risk of major morbidity when pharmaceuticals and non-pharmaceuticals were compared. The use of multiple substances appears to moderate the differential risk in major morbidity and death between pharmaceuticals and non-pharmaceuticals.

Keywords: Suicide, Aged, Relative risk

157. Comparison of concurrent nonmedical use of benzodiazepines and methadone or buprenorphine

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Background: Nonmedical use of benzodiazepines is widely reported among opioid-dependent drug users, including populations in opioid maintenance treatment, where life-time concurrent use of benzodiazepines is 66–100%. Studies of these populations consistently identify that patients taking benzodiazepines concurrently experience greater levels of drug-related harm. Respiratory depression and death following nonmedical use of benzodiazepines and methadone or buprenorphine is reported. This study will compare the toxicity of benzodiazepines with co-ingestion of methadone or buprenorphine.

Methods: A retrospective analysis of buprenorphine and methadone cases reported to NPDS from November 1, 2002 to December 31, 2010. Inclusion criteria were ≥ 18 years old, nonmedical use of buprenorphine or methadone with or without benzodiazepines, and managed in the health care facility. Cases with other co-ingestants or unknown outcomes were excluded. Outcomes measures were clinical effects, treatments, disposition and final medical outcomes.

Results: Of the 764 cases, 692 were in the methadone-benzodiazepine (methadone-BZD) group and 72 were in the buprenorphine-benzodiazepine (BUP-BZD) group. Frequently reported clinical effects were lethargy 71.1% vs 59.7%, respiratory depression 29.0% vs 15.3%, and cardiac arrest 1.9% vs 0% for methadone-BZD and BUP-BZD groups, respectively. The frequency of respiratory depression was significantly higher in the methadone-BZD group (p = 0.019). Patients in the methadone-BZD group were four-times more likely to receive naloxone (60.4% vs 15.3%) or to be intubated (16.3% vs 4.2%) than BUP-BZD group. Hospitalization rates were 67.3% for methadone group and 43.3% for BUP-BZD group. Disposition and final medical outcomes can be found in the Table 1. Sixteen patients in the methadone-BZD group died vs none in the BUP-BZD group. There were significant differences in distribution of disposition and medical outcome.

Table 1. Results for abstract 157.

	Methadone-Benzodiazepine (n = 692) No. (%)	Buprenorphine-Benzodiazepine (n = 72) No. (%)
Disposition (p < 0.001)		
Admission to critical care (ICU)	360 (52.0)	17 (26.6)
Admission for medical care	106 (15.3)	12 (16.7)
Treated and released from emergency department	190 (27.5)	37 (51.4)
Medical Outcomes (p < 0.001)		
No effect	28 (4.1)	12 (16.7)
Minor	178 (25.7)	37 (51.4)
Moderate	341 (49.3)	22 (30.5)
Major	129 (18.6)	1 (1.4)
Death	16 (2.3)	0

Conclusions: Nonmedical use of benzodiazepines with methadone is associated with higher hospitalization rates, greater ICU utilization rates and considerably worse outcomes when compared to nonmedical use of benzodiazepines with buprenorphine, as reported to poison centers.

Keywords: Benzodiazepine, Buprenorphine, Methadone

158. Pesticides and suicides: Demographic disparities

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Objective: The suicide rate continues to increase across the US and varies by methods. The World Health Organization identifies pesticide ingestion as one of the leading methods of suicide. Pesticide exposures are often reported to poison centers (PC) and a significant number of these exposures are related to attempted suicide. This study aims to evaluate the prevalence of suicide by intentional pesticide exposure in a large, demographically diverse state bordering México.

Methods: Exposure cases for the most recent 12-year period (2000–2011) were retrospectively analyzed from our PC network database. Cases were evaluated for their exposure characteristics, outcomes and the relationship between suspected attempted suicides and pesticide exposures. A range of 119–139 cases of suicide by pesticides were reported each year. The study excluded patients under age twelve.

Results: Over a twelve-year period, a total of 1495 cases were reported in which patients attempted suicide by exposure to pesticides. Insecticides (46%) and rodenticides (46%) accounted for the type of pesticide used in the majority of cases. Cases were distributed by sex in a 3:2 ratio of males to females. Patient age was 20-years-old or greater in 84% of cases. These attempted suicide

exposures by pesticide occurred in the patients' residence (91%) and the route of exposure was by ingestion in 97% of cases. Analysis of the initial management site reported showed that 77% of the patients were en route or already in a healthcare facility(HCF), while 21% were referred to a HCF. Major or moderate effects were present in 18% of cases, 51% of cases had minor or no effects, 7% of cases were not followed, and 23% were not able to be followed. Death was the medical outcome in only 1% of cases. When examining demographic variations for these exposures, reported attempted suicide by pesticide exposure occurred in urban communities in 85% of cases and 15% in rural communities. Upon further examination, based upon population in rural vs urban areas the rate/100,000 population was 7.29 in rural communities and 6.85 in urban communities. The comparative exposure rate/100,000 population for cases occurring in counties that are located along the US-México border was 8.11 versus 6.79 in non-border counties.

Conclusions: Rural areas and border communities are at greater risk for attempted suicide by means of pesticide exposure. This study reveals that with a steady frequency of pesticide poisoning by suicide, there is a need to train and equip healthcare facilities to work with PCs in the evaluation and treatment of these poisoned patients. Because pesticides are easily accessible and stored without any precautions in most households, the frequency of these exposures is likely to continue.

Keywords: Pesticide, Suicides, Ingestion

159. A spoonful of cinnamon: The "cinnamon challenge" – Google Trends and the National Poison Data System

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Background: The "cinnamon challenge" is a popular dare in which a person attempts to swallow a tablespoon of ground cinnamon without water in under 60 seconds. News reports of the challenge surfaced in 2001 with popularity peaking in January 2012. This challenge is often videoed and uploaded to social media websites. Serious health consequences can result. Articles, including an AAPCC press release (28 March 2012), warning of health risks emerged in early March 2012. The National Poison Data System (NPDS) of the American Association of Poison Control Centers (AAPCC) aggregates near real-time human exposure data from the 57 US poison centers. Google Trends (GT) provides the relative frequency of topic searches over time and correlates the search frequency with news reports. Limited studies have explored the promise of internet search system results being used to predict public health events. We looked at the predictive capabilities of both systems for this popular dare.

Methods: We examined the time based (secular) trends in Cinnamon Exposures reported to NPDS and Google Trends (Cinnamon, Cinnamon Challenge) by week (4 January 2004–8 April 2012) using change over time profiles and correlation coefficients.

Results: Weekly NPDS call volumes ranged from 0–15 for the time period of 2004–2011. In 2012, the range was 4–49, with the peak of 49 occurring the week of 29 January 2012. GT queries for cinnamon and cinnamon challenge demonstrated a similar pattern over the years with GT searches peaking the same week. Activity for both NPDS and GT started to increase 33 weeks ago (21 August 2011–8 April 2012), with volumes tripling. During this time period, weekly GT volumes were each highly correlated with NPDS calls [GT Cinnamon: r-square = 0.624; GT Cinnamon Challenge: r-square = 0.820; and GT Cinnamon + Cinnamon Challenge: r-square = 0.761; with a $p < 0.0001$ and $n = 33$ for each]. Change over time profiles for NPDS calls and GT queries were similar and showed GT queries for cinnamon challenge led the NPDS calls.

Conclusions: The peak of cinnamon related NPDS exposures and GT queries predated the official release of health warnings. This phenomenon was also demonstrated in 2008 with a salmonella contamination event. The wide public access and use of internet search engines represents a largely underutilized source of early and relevant epidemiological information. Google Trends makes search information easily available and deserves evaluation by public health, including Poison Centers.

Keywords: National Poison Data System, Empidemiology, Internet

160. Comparison of exposure fatalities from the medical examiner's office and the poison center

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Introduction: The Office of the Chief Medical Examiner (OCME) play a critical role in investigating exposure deaths. In 2010, a collaboration between the OCME and the Maryland Poison Center was started. The type of fatal exposure most likely to be reported to the poison center has not been studied.

Purpose: Determine and characterize the exposure deaths investigated by the OCME and reported and not reported to the poison center.

Methodology: A query of the OCME database from January 1, 2011 to December 31, 2011 (12 months) was performed using query words that would identify all exposures fatalities. Demographic information including all zip codes, site of death, cause of death, manner of death, complete toxicologic analysis and field notes were obtained.

Table 1. Hospitalized exposure deaths captured and not captured by the poison center.

Reason for exposure	Captured cases n = 26 (%)	Noncaptured cases and causes of death n = 65 (%)
Suicide	9 (60)	6 (40): carisoprodol 1; DoA 3; metoprolol 1; APAP 1
Misuse/abuse	9 (18)	42 (82): DoA 18; liver failure 12; GI bleed 4; ICH 4; MI 2; sepsis 2; pneumonia 2; others 7
Unknown	4 (33)	8 (66): DoA 7; phenytoin 1
Adverse drug event	2 (33)	4 (66)
Environmental	0	4 (100): housefires 4
Occupational	1	0
Therapeutic error	1	0
Other	1	0

DoA: Drugs of Abuse

Results: A total of 810 poisoning fatalities were examined over a 12-month period. Four hundred ninety (61%) died pre-hospital; 320 (39%) died in hospital. Of these, 229 died in the emergency department (ED) after presenting in cardiac arrest without return of spontaneous circulation and 91 died after being admitted to the hospital. Comparison of the 91 hospitalized exposure deaths is found in Table 1. Twenty six deaths were captured and 65 were not captured by the poison center. There were 5 hospitalized fatalities in children under 18 years of age. The poison center captured 3 but did not capture one 13 yo burn victim and one 17 yo heroin overdose.
Discussion: Only 11% of exposures deaths investigated by the OCME were hospitalized before dying. These would be the cases most likley to be reported to poison centers. Hospitals reported 60% of suicides. but only 18% of misuse/abuse resulting in death. Most of the misuse/abuse cases (57%) suffered from serious other life-threatening conditions (e.g. liver failure, bleeding,...) which may have influenced the reporting. Adverse drug events and environmental exposures were not frequently reported to the poison center. Hospitals that chronically underreported were identified.
Conclusions: The majority of deaths from exposures occur in non-hospitalized settings. Hospitals were more likely to report suicides than misuse/abuse, adverse drug event or environmental exposures.
Keywords: Death, Epidemiology, Overdose

161. Variability of limb circumference measurements in a simulation of crotalid envenomated patients

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Objectives: Documenting the progression of edema resulting from Crotalid envenomation requires measurements of limb circumference. It is assumed that measurements are accurate and can influence decision-making for antivenin administration when cases are managed through poison center telephone consultation. We conducted a study designed to assess the variability between individual observers and repeatability of measurements of limb circumference in healthy, non-envenomated volunteers.
Methods: Two healthy volunteers acted as simulated snakebite patients. Two bite sites on the wrist and ankle were indicated by an indelible marker. Nurse participants received verbal instructions of the case scenario and instructed to measure limb circumference at the bite site, 10cm distal, and 10cm proximal to the bite site. Nurses repeated the procedure after 4–5 hours on the same patient. We used both variance components (mixed model ANOVA) and Bland-Altman analyses to estimate repeatability and reproducibility. Repeatability is the variation resulting from duplicated measurements taken on the same part by the same observer; reproducibility is the observer-to-observer variability. Clinically-relevant limits of agreement were estimated as 2 cm.
Results: A total of 450 observations were made by 34 observers on two simulated patients. Estimated reproducibility was 1.7 cm and repeatability was 3.1 cm. Limits of agreement range from Bland-Altman analysis was 5 cm.
Conclusions: Reproducibility (observer-to-observer variation) was within *a priori* guideline limits. However repeatability (measurements obtained by the same nurse at the same site at two separate time periods) was poor. Three participants showed significantly large deviations (>3 SD) between replicate measurements. Limitations of the study include use of simulated non-envenomated patients and a small possibly non-representative patient sample. Potential strategies to increase accuracy and precision should include greater standardization in measurement procedures and duplicate measurements per sampling period. Otherwise, our data suggest that an increase of >5 cm in limb circumference may be required to indicate clinically-significant edema.
Keywords: Envenomation, Rattlesnake, Venom

162. Review of Eastern coral snake (*Micrurus fulvius fulvius*) exposures in Florida: 1998–2010

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Background: Envenomation by the Eastern coral snake (*Micrurus fulvius fulvius*) is rare but may be associated with significant morbidity. Antivenom is effective in treating neurotoxicity in envenomated patients, but acquisition of North American Coral Snake Antivenin (NACSAV) is increasingly difficult since it is no longer produced. The purpose of this study is to characterize coral snake exposures in Florida over a twelve year period and determine the effects of varying treatment paradigms on patient outcomes.
Methods: This study is an observational case series utilizing the cases received at Florida poison centers. The study protocol was approved by the institution's institutional review board. Cases to be included were those occurring between 1/1/1998 and 10/31/2010 with the AAPCC code of 0137104 (Coral Envenomations). Excluded cases included those not related to an exposure to an Eastern coral snake or those not followed for at least twenty-four hours post envenomation.
Results: Based on above inclusion criteria, 553 cases were identified. In the final analysis, 387 cases were included based on inclusion and exclusion criteria. Males made up 85.53% of patients included. Bites to the fingers and hands involved 49.35% and 25.58% of cases, respectively. According to case comments, 56.3% of patients experienced no systemic symptoms. The most common symptoms developed were pain (40.6%), paresthesia (28.4%), nausea (12.7%), and emesis (11.4%). NACSAV was administered to 252 patients (65%). Of those patients receiving NACSAV, 18.25% experienced an adverse reaction with hives/rash/welts occurring in 12%, itching in 9%, and shortness of breath in 8% of cases. There was a significant trend in a decreasing number of patients receiving NACSAV throughout the study period ($p<0.001$). Comparisons were made between patients receiving empiric antivenom therapy with no presenting symptoms (Empiric, $n=134$) compared to those patients with no symptoms on presentation who received antivenom upon

developing signs of systemic envenomation (Withhold, n = 106). Patients in the Withhold group had significantly fewer minor, moderate, and major outcomes than patients in the Empiric group ($p < 0.001$).

Conclusions: This data represents a review of Eastern coral snake exposures occurring in Florida over the past twelve years. This data suggests that waiting to treat patients with an Eastern coral snake exposure until they develop signs of systemic envenomation is not associated with worsened patient outcomes. This strategy may result in antivenom conservation for more symptomatic patients and may help reduce the number of adverse reactions occurring secondary to antivenom administration.

Keywords: Snake bite, Antivenom, Envenomation

163. A retrospective study of scorpion stings presenting to an emergency department in Jeddah, Kingdom of Saudi Arabia over a 12-year period

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Background: Saudi Arabia is known to be the habitat for at least 23 scorpion species, the majority of which are venomous. Even though a number of studies exist in Saudi Arabia on scorpion stings, none have described the frequency and general characteristics of these envenomations in the second largest city in Saudi Arabia, Jeddah.

Methodology: Retrospective cohort study of all scorpion stings that presented to an Emergency Department in Jeddah between 1999 and 2011 and required admission. Cases were identified by searching the electronic medical record database for the ICD 85 diagnosis of scorpion sting.

Results: 82 cases were identified during the study period but 71 had complete medical records. 82% (n = 58) of patients were under the age of 14 years and 42% (n = 30) were under the age of 5. The male to female ratio was 1:1.2 and 61% (n-43) of the stings occurred at night. The most common sting site was the toe (55% n = 39), followed by the finger (20% n = 14) and unknown in 1 patient. Yellow colored scorpions (Lerius quinquestriatus) were found to be the most common identifiable type (27% n = 19) followed by black colored scorpions or Androctonus crassicauda (10% n = 7). Pain at the sting site was the most common symptom (70% n = 50), followed by vomiting (37% n = 26), nausea with no vomiting (11% n = 8), and numbness at the sting site (7% n = 5). Local erythema was the most common sign (51% n = 36), followed by anxiety (34% n = 24), local swelling (30% n = 21), diaphoresis (28% n = 20), local muscle spasms (6% n = 4), convulsions (6% n = 4) and myoclonus (3% n = 2). Serum creatine phosphokinase and amylase were both elevated in 24% (n = 17) and 11% (n = 8) of cases respectively. Polyvalent antivenin was administered in 65% (n = 46) of the cases according to the Saudi Ministry of Health guidelines. 44% (n = 31) were admitted to the Intensive Care Unit (ICU), of which 48% (n = 15) were younger than 5 years old. 80% (n = 25) of the patients who were admitted to the ICU, received the antivenin.

Conclusions: Scorpion stings are likely underestimated in Jeddah and most commonly occur in children based on our study. Our findings are compatible with other studies in Saudi Arabia. Future National studies evaluating the clinical characteristics of these stings will better define secondary morbidities and appropriate management.

Keywords: Scorpion, Stings, Jeddah

164. Non-native (exotic) snake envenomations in the U.S., 2005–2011

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Background: Non-native (exotic) snakes are a problematic source of envenomation in the U.S. A prior analysis of the database of the American Association of Poison Control Centers (AAPCC NPDS) database for the period 1995–2004 found a large number of species involved, that the majority of envenomations occurred in private residences, a sizeable percentage of pediatric bites suggesting at-risk household members, and a 60% coding error rate, with native snakebites erroneously coded as exotics and numerous case duplications, among other findings. This study was designed to determine whether the demographics of the bites or rates of coding errors had changed.

Methods: Retrospective case series in the AAPCC NPDS database from 2005 through 2011, with primary center verification of suspect coding errors.

Results: Between 2005 and 2011, there were 259 human exposures exotic snakes in the United States reported to U.S. poison centers. There were at least 68 exotic species involved, with 42% to viperid snakes and 40% to elapids. The number and spectrum of exotic species was similar to the previous study. The demographics of exposure were also similar to the previous study: Most poison center contacts occurred during the warmer months; there were similar proportions of exotic envenomations involving males (80%), occurring in the home (70%), and involving children (< 18 years, 17%; < 6 years, 9%). Of the cases followed to outcome, there was also a similar pattern of medical outcomes. There were no deaths in this series. The coding error rate for the years 2005–2008 was 56% (v 60% for 1994–2005; p = ns). The prior study was published in 2007 and POISINDEX(R) changed the way it listed non-native venomous snakes at the end of 2008. The coding error rate for 2009–2011 fell to 14% ($p < 0.0001$), with the remaining coding errors mainly being the Deinagkistrodon code being erroneously used for native copperhead or cottonmouth envenomations, and case duplications, where more than one center coded itself as primary.

Conclusions: Exotic snake envenomations occur with a consistent frequency in the United States. Numerous, diverse species are involved. There has been no significant change in the demographics or outcomes of such bites since 1994. Poison centers should be aware of the potential for such envenomations and the resources available to assist in their management. A change in the way non-native snake codes were displayed in POISINDEX(R) resulted in a significantly reduced rate of native snakebites being coded as exotic envenomations. More prominent designation of Deinagkistrodon snakes as non-native

and safeguards against duplication of cases may help to further reduce coding errors.

Keywords: Snake bite, National Poison Data System, Poison center

165. First report of envenomation by the Great Lakes Bush Viper (Atheris nitschei)

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Background: The Great Lakes Bush Viper (Atheris nitschei) is a venomous pit viper from Central Africa. No prior published reports of human envenomation by this species have been reported.

Case report: A 30 year-old male was bitten by Great Lakes Bush Viper on the third digit of his left hand. On presentation, the patient had a dramatically swelling of left upper extremity (LUE) extending above the elbow and a hematoma in the left axilla. His left hand was edematous with surrounding hemorrhagic bullae and mild bleeding from the bite site and the oral mucosa. He reported nausea but denied other symptoms. See the Table 1 for laboratory results and blood products transfused. A surgeon evaluated the patient for LUE compartment syndrome. No fasciotomy was performed. Mucosal bleeding ceased within 12 hours of admission. No further bleeding was observed. His smear demonstrated hemolysis. He was discharged from the hospital on post-envenomation day 6 and has not returned for follow up visits.

Case discussion: Envenomation by the Great Lakes Bush Viper is uncommon, even in its home range of Central Africa. The venom of other Atheris species is associated with hemolysis, coagulopathy, and renal failure. There is no Atheris specific antivenom available, although other antivenoms have been administered in Atheris sp. envenomations with an unclear impact on the natural course of these cases. Both exotic antivenoms and CroFab were considered for this patient, but neither was given. The consulting Hematologist suggested transfusing cryoprecipitate to a goal fibrinogen of >200, but this was never achieved. Plasmapheresis has been suggested in prior case reports of Atheris envenomation. This intervention was considered but deemed unnecessary as the patient demonstrated clinical improvement within the first 24 hours. The patient was discharged with improving tissue edema, paresthesias, limited range of motion in the left hand, and persistent mild hematologic abnormalities.

Conclusions: This is the first case report of an envenomation by the Great Lakes Bush Viper (Atheris nitschei). It resulted in local tissue damage and hematologic abnormalities that included hemolysis, thrombocytopenia, hypofibrinoginemia, coagulopathy, and elevations in d-dimer. He was treated without antivenom or plasmapheresis but with transfusion of multiple blood products.

Keywords: Snake bite, Envenomation, Venom

166. Sonographic signs of snakebite

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Introduction: Crotalid bites are routinely assessed with serial measurements of limb circumference and physical examination of soft tissue damage. The purpose of this pilot investigation was to develop a protocol correlating external findings of rattlesnake bites with internal anatomic changes visible on ultrasound imaging of a bitten extremity.

Methods: IRB approval was obtained to enroll snakebite victims treated at our hospital. Clinical findings were serially recorded. Then, using an 8-mHz linear-array soft tissue probe, static ultrasound images and video clips of both the bitten and contralateral (control) extremities were taken during the same assessment.

Results: Four patients were enrolled for this pilot phase of the study (see Table 1). In all cases, the ultrasound imaging of the affected extremity was consistent with gross visual inspection findings. The most common abnormal ultrasound findings were hyperechogenicity and thickening of the skin and subcutaneous layers, consistent with edema.

Other interesting sonographic findings: In two patients (A and B) with fasciculations, myokymia was apparent in all visualized striated muscle layers, and fine contractions were apparent on ultrasound even in areas with no external findings. Further, the diffuse, progressive edema in the envenomated legs of patients A and B was clearly subcutaneous, sparing the fascial planes, deeper muscle bundles, and perimysium. In two patients (C and D) with finger bites, small volar tendons and pulp spaces were more readily visualized by immersing the imaged hand in a water bath to facilitate imaging.

Discussion: This pilot study suggests that ultrasound imaging can facilitate a more complete view of the depth and severity of tissue damage after snakebite. For instance, normal deep muscle bundles and fascial integrity were seen in 2 cases with diffiuse leg swelling. Neither of these patients was clinically suspected of compartment syndrome, so the prognostic value of such isolated findings

Table 1. Data for abstract 165.

Day	Hct	Plt	INR	PT	PTT	DDimer	Fibrinogen	Cr	Transfusion
1	46	29k	>9	>120	>200	>4500	<20	0.9	Factor VIIa
	35.4	14k	>9	>120	>200	>4500	<20		2u FFP/2u Cryo
2	28.6	34k	2.8	28.6	35.6	>4500	<20	0.8	2u FFP/2u Cryo
	25.7	45k	1.8	20.2	29.5	>4500	29		1u Cryo
3	25	75k	1.6	18.3	27.1	3493	50	0.7	1u Cryo/1u PRBC
4	27.3	118k	1.4	17.3	29.7	1509	48	0.8	
5	29.1	194k	1.3	16.1	31.5	1061	78		
6	26.8	183k	1.4	16.8	30.2	990	74		

Table 1. Data for abstract 166.

Patient (age/sex)	Bite site	Edema border/clinical effects
A (48 yo M)	Medial left heel	thigh; fasciculations; bullae
B (16 yo M)	Lateral right mid-shin	knee; fasciculations
C (29 yo M)	Left middle finger	shoulder; delayed necrosis
D (55 yo M)	Right middle finger	metacarpal-phalyngeal joint; blister

is uncertain; however, these intriguing initial echoes do warrant further exploration.

Conclusions: In addition to the standard *external* assessments, sonographic examination could aid the *internal* examination of an envenomated limb. Additional experience with a greater diversity of bites and anatomic locations will fully elaborate the strengths and limitations of this technique as a diagnostic adjunct in snakebite severity assessment.

Keywords: Envenomation, Rattlesnake, Ultrasound

167. Black mamba envenomation treated with neostigmine

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Background: Black mamba (*Dendroaspis polylepis*) bites are rare in North America. Envenomation produces rapidly progressive paralysis. Management consists of prompt antivenom administration. Some authorities recommend consideration of neostigmine in neurotoxic elapid envenomation as a bridge to or adjunct with antivenom administration. We present a patient's clinical course after *Dendroaspis polylepis* envenomation treated with neostigmine.

Case report: A 22 year-old exotic snake owner presented to a rural emergency department 10 minutes after being bitten on the left forearm by a black mamba. His medical history included treatment for multiple snakebites, both crotalid and elapid, including a bite from an Egyptian cobra (*Naja haje*). On presentation, he complained of tunnel vision, nausea and craniofacial burning. He was tachycardic and hypertensive with slurred speech but had an otherwise unremarkable neurologic exam. The poison center recommended pressure immobilization and neostigmine 0.5 mg IV every 20 minutes up to four doses until appropriate antivenom was administered. At 60 and 80 minutes post-envenomation, he received neostigmine. By 90 minutes post-envenomation, the patient experienced left hand numbness, ptosis, dysarthria, "throat tightness," and difficulty breathing, prompting endotracheal intubation. The patient was transported to a regional medical center with bedside toxicology consultation and antivenom obtained from the local zoo. The patient received SAIMR polyvalent antivenom in two 4 vial (40 mL) doses 3 and 6 hours post-envenomation. 32 hours post-envenomation, he was extubated. At discharge on hospital day 3, he exhibited no neurologic sequelae.

Case discussion: Among African snakes, black mamba envenomations have the highest mortality. Death may occur despite the prompt administration of antivenom. Adjunctive carbamates appear beneficial in elapid neurotoxicity from species whose venoms contain only post-synaptic nicotinic neurotoxins. However, carbamate use in mamba envenomation is controversial as *Dendroaspis* venoms also contain dendrotoxins and fasciculins, pre-synaptic neurotoxins. Although the natural course of venom-associated neurotoxicity varies, neostigmine use in our patient did not prevent the need for endotracheal intubation and mechanical ventilation. This lack of effectiveness mirrors that of neostigmine use in envenomation from kraits, another elapid with both pre- and post-synaptic neurotoxins.

Conclusions: Adjunctive neostigmine may not obviate the need for airway management and ventilator support in patients experiencing neurotoxicity secondary to envenomation from *Dendroaspis polylepis*.

Keywords: Envenomation, Snake bite, Neurotoxicity

168. Garter snake envenomation mimicking Crotalid envenomation

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Background: Garter snakes (*Thamnophis* species) are usually considered non-venomous and bites from the species are typically associated with minimal effects. We report an eastern garter snake bite of the hand causing significant envenomation.

Case report: A 57 year-old school teacher was bitten in the right thumb while attempting to remove a snake from the school playground. The patient reported the snake chewing on her thumb for 5 minutes despite efforts to detach it. Within fifteen minutes she experienced pain and swelling at the site with development of edema, ecchymosis and discoloration progressing over the next two hours up her forearm and into the antecubital fossa. No systemic manifestations occurred. The reptile was transported in a container to her physician who emailed pictures of the snake and the affected extremity to our Regional Poison Center staff. Aided by a herpetologist, the snake was identified as an Eastern Garter snake, or Common Garter (*Thamnophis sirtalis*). No blood tests were obtained. The patient was discharged home and was examined by the physician twice during the following 4 days. Her symptoms and clinical findings were reported to improve significantly. At two month follow-up, she had no complaints and her hand function was normal.

Case discussion: Many genera of the Colubridae family such as *Thamnophis* possess Duvernoy's glands, which secrete compounds that aid consumption and digestion rather than hastening prey death. Duvernoy's gland is distinguished from the venom gland and is not found in viperidae or elapid snakes. The secretions have variable toxic effects which range from minor pain and local irritation to the presentation of this patient. Since 1981, only 3 accounts of *Thamonphis* species bites resulting in envenomation have been reported. All three cases were associated with a prolonged bite duration, mild edema, and resolution of symptoms without systemic signs of envenomation or coagulation abnormalities. In only one of these reports was the snake identified definitively.

Conclusions: This case demonstrates that patients may develop significant symptoms and findings that mimic *Crotalid* envenomation but, consistent with previous case reports, experience resolution of symptoms with only symptomatic care. Thorough descriptions

and pictures in conjunction with local herpetology resources were instrumental in avoiding unnecessary use of antivenom.
Keywords: Snake bite, Envenomation, Venom

169. Pygmy rattlesnake envenomation successfully treated with Crotalidae polyvalent immune fab antivenom

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Introduction: The pygmy rattlesnake (Sistrurus miliarius barbouri) is a pit viper native to the southeastern United States. It belongs to the subfamily Crotalidae which includes the western diamondback (Crotalus atrox), copperhead (Agkistrodon contortrix), and the more closely related massasauga rattlesnake (Sistrurus catenatus). The pygmy rattlesnake is the smallest pit viper in the US. There have been a total of three unconfirmed cases describing pygmy rattlesnake envenomation. All previous cases have described serious adverse reactions including renal failure, hypotension, and significant swelling. All three were treated with equine derived polyvalent antivenin.
Case report: We describe a 28-year-old man who presented to an outlying hospital 1 hour after being envenomated on the right hand by his friend's pet pygmy rattlesnake. On presentation to an outside hospital he was noted to have significant right hand and forearm swelling. The patient received an initial dose of 6 vials of crotaline polyvalent immune fab antivenom (CroFab®). He subsequently received a second dose of 4 units of antivenom for progression of right upper extremity swelling and then was transferred to our tertiary care center. The patient reported improvement after the loading doses and he was started on maintenance dosing of the antivenom, 2 vials every six hours for a total of 3 doses. The coagulation studies all remained normal. All electrolytes remained normal. He had both objective and subjective improvements in pain and swelling and was discharged the following day with recommendation to have repeat complete blood count (CBC), fibinogen and prothrombin time (PT) in 3–5 days. Unfortunately, the patient was lost to follow up.
Discussion: Previous analysis of dose-lethality of American pit viper venoms in mice has suggested that pygmy venom is more lethal than the venom of the southern copperhead (Agkistrodon contortrix). In vitro hemotoxicity has also been described. Crotalidae polyvalent immune fab antivenom has demonstrated efficacy against pygmy rattlesnake venom in a murine-lethality model. This case represents the first time in the documented medical literature that CroFab® has been used in the treatment of a pygmy rattlesnake envenomation.
Conclusions: Based on our patient's clinical and laboratory response to CroFab®, its use, when indicated, appears to be safe, effective, and appropriate for pygmy rattlesnake envenomation.
Keywords: Antivenom, Envenomation, Rattlesnake

170. Acute renal failure after a pigmy rattlesnake bite

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Background: Renal failure is rare following pigmy rattlesnake bites. Acute renal failure is a major cause of morbidity and mortality after exotic pit viper bites. We report a case of renal failure after a pigmy rattlesnake bite treated with CroFab.
Case report: A 42-year old male was bitten on the left little finger. He did not have a known history of renal failure. The patient identified the snake as a pigmy rattlesnake. Within 30 minutes after the bite, he experienced vomiting, diarrhea, hypotension (96/64) and tachycardia. He had pain and swelling to the knuckles. Initial labs revealed an elevated BUN (25 mg/dl), elevated creatinine (1.66 mg/dl), elevated fibrinogen (900 mg/dl), elevated INR (2.3), prolonged prothrombin time (23.7 sec) and hypokalemia (2.8 mEq/L). CroFab was started immediately. Blood pressure normalized (138/113) within an hour. The patient was transferred to a trauma center. A fluid challenge of 7 liters resulted in very little urine output and the attending physician was concerned about acute tubular necrosis.

By 29 hours post-bite, 13 vials of CroFab had been infused. Vital signs and clotting factors had stabilized. Creatinine kinase (CK) increased to 131 u/L. The patient continued to rely on opiates for pain relief and had small increases in swelling.

Two days post-bite, the fibrinogen was 502 mg/dl, platelets were 96 mm^3 and prothrombin time was 9.9 sec.

By discharge 12 days post-bite, the patient had received 6 dialysis treatments and was discharged to outpatient dialysis.
Discussion: It is possible that the patient identified the snake incorrectly. It is possible that the patient had a pre-existing renal insufficiency. However there was no previous history and the patient was relatively young. A renal insult from a brief period of hypotension and hypo-perfusion should normally have cleared with IV fluids. The patient tested positive for myoglobinuria and had 2–5 red blood cells in the urine, but his CK was not in the range to be classic rhabdomyolysis.
Conclusions: This patient exhibited renal failure which began within hours of sustaining a reported pigmy rattlesnake bite and was documented prior to administration of antivenom. To our knowledge, this is the first report of renal failure following a pigmy rattlesnake bite.
Keywords: Renal toxicity, Hemodialysis, Rattlesnake

171. Impact of a novel rapid reconstitution method on protein content for CroFab polyvalent immune fab, ovine

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Background: CroFab® has been clinically demonstrated to be most effective when administered within 6 hours of snake envenomation, and improved clinical outcomes are correlated with quicker timing of administration. A new method of preparation allows for reconstitution of CroFab® in less than 4 minutes and involves injection of an increased amount (18 mL) of 0.9% saline and manual inversion of the vial. The originally labeled method for reconstitution used 10 mL sterile water for injection and required gentle swirling for up to 20 minutes. Manual inversion may result in foaming of the product. The increased amount of injected saline was chosen to reduce reconstitution time whilst minimizing the

potential for foaming and thereby allaying concern over loss of protein content in the reconstituted product.
Methods: An analytical study (UV absorbance) was performed to compare the protein content of samples from 3 separate batches of CroFab®. Three batches (A, B, and C) were each reconstituted using the original labeled procedure (10 mL WFI with gentle swirling for 20–23 minutes) and then using a modified rapid procedure (18 mL 0.9% saline and manual inversion for ~3–4 minutes).
Results: Protein content of the batches reconstituted using the original labeled method (gentle swirling for ~23 minutes) was comparable to batches reconstituted with the new rapid method (manual inversion for ~3 minutes) using analytical methodology. The total protein (± 10% mean total protein of each batch with each reconstitution condition, measured by UV absorbance at A280 nm) in mg/vial was: for Batch A, B, and C (original method: 839, 834, 846) (new method: 848, 833, 827).
Conclusions: When comparing batches that were reconstituted using the original and new reconstitution methods, the total protein yield was demonstrated to be comparable. This suggests that any foaming that occurs in conjunction with the newer, more rapid method does not result in loss of protein.
Keywords: Antivenom, Envenomation, Reconstitution

172. Snakebite on a plane-central American coral snake

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Background: Poison Centers (PC) frequently receive telephone calls from patients outside their geographical areas. A few PCs regularly receive calls from othercountries seeking toxicology advice. We are unaware of any previous PC calls pertaining to a passenger on an international airline flight with a serious snakebite envenomation.
Case report: A caller from Texas notified a local PC that a family member was on board a flight returning from Honduras where he had been bitten by a coral snake 5 hours earlier. This 21-year-old, previously healthy man traveled as a missionary to an island off the coast of Honduras and was bitten on the right thumb. He was examined by a Honduran physician who notified the patient's family in the US. Arrangements were made for the patient to take a 3-hour commercial flight back to the US to be treated. In flight, the patient was asymptomatic except for localized hand pain and blurred vision. The PC determined that diverting the flight would not benefit the patient based on multiple factors. He arrived in Texas 8 hours post-bite, and he was taken to the hospital by helicopter. The patient had endotracheal intubation and received 6 total units of North American Coral Snake Antivenin (*Micrurus fulvius*) (Equine) in two doses. He was extubated the following day, but he had a hospital course complicated by a fever of 101.5 degrees F. This was treated with antibiotics, and he continued to improve. He was discharged home after 6 days without symptoms and given warnings about developing serum sickness.
Case discussion: Approximately 60 species of venomous coral snakes exist in Central and South America, with the greatest variety occurring from Mexico to northern South America. There are at least 5 species in Honduras, but only one genus: *Micrurus*. Our patient was treated with North American coral snake antivenom, and he did well. Others have demonstrated that this antivenom may be successfully used to treat bites from snakes not indigenous to North America. The manufacturer has stated that this antivenom will not be available in the future.
Conclusions: PC personnel should be aware that bites from coral snakes outside the US may be effectively treated with NA coral snake antivenin (equine). This treatment may be important if the specific exotic coral snake antivenom is not available. PC personnel should also be aware that they may receive calls pertaining to patients from far outside their geographic region such as from an international airline flight. Because coral snake envenomation potentially may be fatal, there is need for continuing availability of antivenom in the US.
Keywords: Central American coral snakebite, North American coral snake antivenom, International flight

173. Are coral snakes really more dangerous than rattlesnakes in the US?

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Background: Approximately 8000 venomous snakebites occur in the US annually. The most common source is the rattlesnake, but a small fraction of these are from coral snakes. Rattlesnake venom contains hemotoxins which can cause tissue necrosis, coagulopathy and death. Coral snake venom contains potent neurotoxins which can cause paraly sis and may lead to subsequent death from respiratory failure. Although death from coral snake bite is extremely rare in the US, anecdotal evidence suggests that coral snake bites have a higher morbidity than rattlesnake bites. However, no study has examined the morbidity of coral snakes bites compared to rattlesnake bites.
Objective: Our goal was to determine the morbidity and mortality of coral snake bites and rattlesnake bites in the US over a ten year period.
Methods: This study was an observational, case-control study of telephone calls to all US poison centers (National Poison Data System) for coral snake and rattlesnake bites from 2000 to 2009. Data were extracted from this database and analyzed to determine morbidity (defined as major, moderate, minor, and no effect) and mortality (death) rates for coral and rattlesnake bites. There were 64,698 total snakebites in the database for this time period.
Results: There were 672 (1.0%) coral snake bites and 9474 (14.6%) rattlesnake bites. These two groups of victims had similar average ages (33.2 and 35.1 years) and male gender (82.4% and 79.4%). Of the coral snake bite victims, 21.2% were under 18 years compared to 17.0% of rattlesnake bite victims ($p = .006$). No deaths were reported following coral snake bi tes compared to 21 (0.22%) for rattlesnake bites ($p = .44$). Major effects were seen after 4.0% of coral snake bites and 9.8% of rattlesnake bites ($p = .001$). There were 28.9% and 60.1% moderate effects ($p < .001$) and 56.9% and 26.9% minor effects ($p < .001$) in the two groups, respectively. No effect, a presumed "dry bite", was

seen in 8.6% of coral snake bites and 2.9% of rattlesnake bites ($p < .001$).
Conclusions: Victims of rattlesnake bites were significantly more likely than victims of coral snake bites to have major (life-threatening) outcomes or moderate outcomes. Victims of coral snakes were significantly more likely to have minor outcomes or dry bites. Limitations of this study include the reliance on information from callers to the poison centers and the lack of treatment information (use of antivenom, time from bite to antivenom administration, surgical treatment). The low number of dry bites suggests that these victims do not call the poison centers compared to envenomated victims. The anecdotally assumed high morbidity of coral snake bites is not supported by this data.
Keywords: Coral snake, Rattlesnake, Morbidity

174. Update on the coral snake antivenom shortage

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Background: In 2003, Wyeth Pharmaceuticals discontinued manufacturing of North American Coral Snake Antivenin®. Set to expire October 31, 2008; vials of one lot have been repeatedly extended, but that supply is nearing exhaustion. Wyeth was merged into Pfizer, and began manufacturing their product again in 2011, but new supplies are not anticipated to be available until 2013. There is still no other FDA approved product available. Because the area with the largest number of significant coral snake envenomations is in this center's region, this center has chosen to prioritize this issue and work actively to address this critical shortage.
Methods: The Poison Center developed a coral snake antivenin discontinuation task force to address the shortage. Based on the plan developed by that task force, the Center has been actively working to monitor supply using focused phone surveys of hospital pharmacies, monitor and assist in management of cases in an attempt to improve treatment while maximizing supply, educate health care providers, and work toward developing a reliable supply of antivenom. A dedicated webpage was developed and updated as needed. Contact with the antivenom manufacturer was maintained. New treatment guidelines were developed and instituted. A study was developed, and IRB approval obtained.
Results: A total of 43 coral snake envenomations were reported in the state in 2011. By fall of 2011, the supply of antivenin reached a critical level. Delays in receiving the antivenom have resulted in patients needing ventilators and expensive prolonged ICU care. Legal and financial issues have delayed the acquisition of an alternative supply. The study has been delayed by supply issues. Pfizer's new antivenom will not be available before 2013 at the earliest.
Conclusions: This poison center continues to work actively to address a significant regional shortage of an orphan drug. Outreach is now focused on minimizing transport times for significantly envenomated patients and more closely monitoring care. Requests for support have been submitted to multiple agencies to reverse this costly and life-threatening situation. The Poison Center has been instrumental in responding to this crisis.
Keywords: Antivenom, Snake bite, Surveillance

175. Clinical effects and antivenom use for snakebite victims treated at three U.S. hospitals in Afghanistan

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Background: The Afghanistan terrain harbors several venomous snakes. Annually, over 100,000 U.S. civilians and U.S. and international military members work in Afghanistan. Snake envenomations occur, but the paucity of published reports of Americans and others treated for Afghan snakebites are insufficient to support hospital antivenom (AV) treatment and stocking guidelines.
Objectives: To report the clinical effects and treatments recorded for snakebite victims presenting to 3 U.S. military hospitals in Afghanistan.
Methods: A case series of all snakebite victims presenting to three U.S. military hospital EDs between July 2010 and August 2011 in northern and southern Afghanistan using a standardized data collection tool to extract cases from ED patient logs and available medical records. Abstracted data included age, gender, bite date and anatomical location, snake description, hematological and biochemical blood test results, AV use, AV adverse effects, bite complications, procedures performed, and hospital course.
Results: 17 cases were collected and included. Median patient age was 20 years (IQR 12–30), 16 were male, and most (82%) were Afghans. All bites were to an extremity (76% lower extremity). On arrival 8 had tachycardia and none had hypotension or hypoxia. A viper was implicated in 5 cases. Median time from bite to ED care was 2.8 hours (IQR 2–5.8). Ten were treated with at least one dose of Iranian or French polyvalent AV, most commonly for coagulopathy, and no adverse effects were detected. Six received additional AV for elevated INR. Five had an INR > 10 and none developed delayed onset coagulopathy after ED presentation. Three received blood transfusions. Hospital length of stay ranged 1–4 days. All had resolution of coagulopathy and improved swelling and pain at discharge. None required vasopressor support, fasciotomy, other surgery, or died.
Conclusions: We report the largest series of Afghan snake envenomations treated by U.S. physicians. In our study, AV resolved coagulopathy and improved local symptoms without adverse effects. No patient required fasciotomy, aggressive vasoactive support, or died. The retrospective nature of much of the data collection, source record variability, and short follow-up period are notable study limitations.
Keywords: Antivenom, Snake bite, Envenomation

176. Report of a bite from a new species of the *Echis genus-Echis omanensis*

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Background: In Oman, 9% of snake envenomations are caused by the saw-scalded viper (*Echis carinatus*). This snake is distributed throughout the country, especially in rocky areas. Recently, a new species belonging to the *Echis genus*, *Echis omanensis* has been recognized in Oman. Not much is known about clinical mani festations of envenomation from its bite. Even though, past reports of *Echis omanensis* may have been falsely attributed to *Echis coloratus*, we describe the first case that is accurately linked to this newly recognized species and that responded to the Saudi polyvalent antivenom without any complications.
Case report: A 63-year old snake keeper presented to the Emergency Department shortly after being bitten by a captive specimen of the Oman carpet viper (*Echis omanensis*). The incident occurred during expression of the venom at a research center. The patient complained of severe pain and swelling of the left index finger. The swelling extended to the mid forearm within one hour. His vital signs remained stable with no evidence of systemic manifestations. The bedside clotting test after 20 minutes was normal. He was treated initially with analgesics and tetanus toxoid. Based on the concern that this snake bite could cause delayed coagulopathy and due to the rapidly progressive swelling, the Saudi National Guard polyvalent snake anti-venom was administered. This antivenom is raised from horses inoculated with the venom of 6 terrestrial snakes including *Echis carinatus* and *Echis coloratus*. As per protocol, he received 40 mL diluted in 5 mL per kilogram of body weight of normal saline, infused intravenously slowly over 30 minutes. The patient was admitted to the intensive care unit, where he received another similar dose of the antivenom because his swelling and pain did not improve after four hours. He remained hemodynamically stable, had normal serial coagulation tests, and had resolution of his swelling within 48 hours.
Conclusions: This case describes the clinical consequences of a bite from a newly recognized species of *Echis* snakes that primarily consisted of local manifestations and that responded to the Saudi polyvalent antivenom.
Keywords: Snake bite, Venom, Rattlesnake

177. Multiple organ failure following *Vespa basalis* stings & the potential benefits of plasmapheresis

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Introduction: There are more than 20 species of *Vespa* worldwide while 7 could be found in Taiwan including *Vespa basalis, affinis*, *velutina*, *ducalis*, *analis*, *manderinia* and *V. wilemani*. Among them, *V. basalis* is believed to be the most dangerous since its aggressiveness and highly toxic venom. There were several deaths caused by multiple *V. basalis* stings however the entomological details and clinical manifestations frequently were unavailable.
Case report: We present a case experienced multiple *Vespa basalis* stings, who developed hypotension, hemolysis, acute renal failure, hepatic injury, coagulopathy and myocardial dysfunction as well as acute respiratory distress syndrome. Plasmapheresis one session was conducted 19 hours post-stinging. The patient's liver function, coagulopathy and hemodynamic status improved soon after the plasmapheresis.
Discussion: In mice, the median lethal dose (LD50) of the *V. basalis* venom is 430 μg/kg (i.p.), in contrast to 2 μl/kg of *V. affinis*, 2.8 mg/kg of *V. tropica* (i.v.), and 2.5 mg/kg of *V. orientalis* (i.p.). Most of human deaths were caused by *V. affinis* & *V. tropica* in Papua New Guinea and southeast Asia, and *V. orientalis* from the middle east and India while the time to death varies from hours to 9 days. The case fatality rate was 33% or even higher when patients developed multiple organ failure, pulmonary edema or shock. Treatment is generally supportive and/or temporary hemodialysis. In the literatures however, plasmapheresis had been successfully performed in 2 cases of massive envenomation from honeybee or wasp stings in an attempt to remove the circulating venoms or inflammatory mediators. Although it is not listed as a definitive indication by the American Society for Apheresis for insect envenomations, given the fact that large molecules are removed more efficiently during plasmapheresis than dialysis, it might pose beneficial effects in the treatment of multiple organ failure caused by multiple hornet stings.
Conclusions: Multiple organ failure induced by massive envenomation of hornet stings is a life-threatening event. Plasmapheresis might have beneficial effects in addition to supportive care.
Keywords: Envenomation, Venom, Hemodialysis

178. Gila monster (*Heloderma suspectum*) envenomations reported to the American Association of Poison Control Centers' National Poison Data System

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Background: The Gila monster (*Heloderma suspectum*) is a venomous lizard native to the deserts of southwestern United States and northern Mexico. While the venom delivery system differs considerably from that of snakes, it too is capable of causing systemic envenomation. Published reports of Gila monster bites are rare and likely represent more severe cases. Commonly reported effects include pain, local edema, weakness, dizziness and nausea. Severe, life threatening symptoms have also been reported. The aim of this study was to describe calls reported to US Poison Control Centers (PCCs) concerning human *H. suspectum* exposures.
Methods: A retrospective review of PCC calls, received between 1/1/00 and 10/31/11, reported to the National Poison Data System (NPDS) regarding Gila monsters. This study was approved by our institutional review board.
Results: Of over 45 million calls reported to NPDS during this time period 319 concerned Gila monsters. A total of 197 were exposure calls, of these 92 involved animals and were excluded. There were 105 human exposures; most 79% were male. Arizona's PCCs received 69.5% of the calls; the rest were handled by 16 other PCCs. Of the human exposure calls, 71 (68%) were referred to a health care facility (HCF) and 30 (29%) were managed onsite; there were 4 unknowns. Of the 71 HCF referrals, 36 (51%) were discharged home and 17 (24%) were admitted. Most (n = 11; 65%) admissions were to an intensive care unit. A total of 18 (25.3%) patients refused referral or were lost to follow up. Limitations

included the retrospective design and reliance on the validity of the data reported to, and recorded by, PCCs.
Conclusions: Human envenomation by Gila monsters represents a very small proportion of calls to US PCCs. Of those patients with a known outcome, many (29.7%) were managed at home and most (83.2%) did not require hospitalization.
Keywords: Envenomation, Gila monster (*Heloderma suspectum*), Poison center

179. Dinosaur envenomation? A case of a Komodo dragon bite

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Background: Komodo dragons (*Varanus komodoensis*), native to the islands of Indonesia, are the world's largest lizards. It is often debated if the death of their prey is due to direct trauma from a bite, envenomation, or sepsis due to the pathogenic bacteria which are present in their saliva. There are no reports in the medical literature of a human exposure to a Komodo dragon bite.
Case report: A 34 year-old female zookeeper was bitten on the hand by a Komodo dragon during a routine feeding. The animal bit and clenched down for about 3 seconds before its bite was disengaged. She received a dose of amoxicillin/clavulanate and was transported to the emergency room. Her physical exam was notable for multiple small lacerations to the distal left 5 th digit, the largest being 3 cm overlying the distal interphalangeal (DIP) joint on the dorsal surface. She was unable to fully extend at the DIP joint, but was neurovascularly intact. The wound was irrigated and allowed to heal by secondary intention. She was discharged home on amoxicillin/clavulanate and a finger splint with the IP joints in extension. Multiple wound checks revealed a well-healing wound without signs of infection. After physical therapy and splinting for over 6 weeks, the patient did not regain the full ability to extend the finger.
Discussion: The patient's mallet finger was most likely due to a traumatic tendon injury inflicted by the bite. However, she was also at high risk for infection and complications of envenomation. A study of wild and captive Komodo dragons showed a total of 57 species of bacteria isolated from their saliva. *Escherichia coli* and *Staphylococcus* species were the most commonly isolated, however *Pasteurella multocida* has also been isolated and thought to be a cause of prey mortality (1). Fry, et al. described mandibular venom glands in Komodo dragons that deliver their complex mixture of proteins by a "grip-and-rip" mechanism, meaning the serrated teeth create wounds to allow for entry of the venom (2).
Conclusions: This patient, who sustained a Komodo dragon bite and received immediate care, did not show evidence of bacterial infection or significant envenomation. Management should be aimed at evaluation of traumatic injuries, proper wound care, healing by secondary intention, and early antibiotic therapy.
Keywords: Envenomation, Venom, Komodo dragon

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180. Pause-dependent ventricular tachycardia and torsades de pointes after ibogaine ingestion

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Background: Ibogaine, an indole alkaloid isolated from the root bark of the African shrub *Tabernanthe iboga* has been used increasingly over the last 25 years in medical and nonmedical settings, most often for opioid detoxification. It reduces opioid withdrawal symptoms and drug self-administration in humans and preclinical models, but is illegal in the US due to its classification as a hallucinogen. Although ibogaine has been associated with fatalities and cardiac arrhythmias, in most cases underlying cardiac disease, concomitant coingestants and use of alkaloid extracts or dried root bark of uncertain composition were contributing or proximal causes. Ibogaine is associated with QT prolongation presumably due to hERG channel blockade. It is also associated with bradycardia, but the mechanism remains unknown. We present the first case of well-characterized torsades de pointes (TdP) and pause-dependent ventricular tachycardia (VT) following the ingestion of ibogaine in a patient with a normal pretreatment medical evaluation.
Case report: A 63-year-old man with a history of IV heroin use and subsequent methadone maintenance presented to the emergency department (ED) due to an irregular pulse after ingesting ibogaine in a nonmedical setting. The patient had transitioned off methadone to short-acting opioids one month prior. He received 10.5 mg/kg of ibogaine HCl (85–98% purity) and 14 mg/kg *T. iboga* root bark extract (50% alkaloids) over an interval of 15 hours, with the last dose 3 hours prior to ED presentation. On arrival, ECG showed sinus bradycardia at 36 bpm with a QTc interval of 498 ms. Two episodes of TdP associated with loss of consciousness as well as multiple episodes of pause-dependent VT occurred. A temporary transvenous pacing wire was placed. The initial ED potassium (K+) level was 4.1 mEq/L, however subsequent K+ levels were as low as 2.9 mEq/L. The hypokalemia persisted despite repletion through hospital day four, which was likely secondary aggressive cathartic use prior to starting ibogaine. The magnesium level remained normal. Dysrhythmias resolved by day five, and the QTc stabilized at $\leq$450 by day six. The pretreatment evaluation showed an ECG with sinus rhythm of 56 bpm, QRS interval of 84 ms and QTc of 422 ms and normal stress echocardiography. Electrolytes were normal four days prior to admission.
Case discussion: Although previous reports indicate ibogaine is associated with bradycardia, QT prolongation and ventricular tachyarrhythmias, this is the first report of pause-dependent VT and TdP as well as a normal pretreatment screening.
Conclusions: Patients treated with ibogaine are at risk for pause-dependent VT and TdP despite pretreatment medical screening.
Keywords: Alternative medicine, Cardiac toxicity, Electrocardiogram

181. Tea tree oil ingestion treated with surfactant

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Background: Tea tree oil is an essential oil used topically for its antifungal, antiviral, and antibacterial properties. It contains a mixture of aromatic hydrocarbons sold over the counter in a variety of concentrations. We describe an 18-month old male who ingested tea tree oil, developed central nervous system (CNS) depression and respiratory distress, and received early treatment with surfactant.
Case report: A previously healthy 18-month old presented to the pediatric emergency department (ED) by ambulance immediately following the oral ingestion of 15 mL of 100% tea tree oil. On EMS arrival, he was lethargic and in respiratory arrest. Bag-valve-mask ventilation was begun and the patient regained spontaneous but inadequate respirations. Upon ED arrival he was unresponsive with a BP of 104/50, HR of 159, RR of 25, and an oxygen saturation of 98% on 10 L non-rebreather mask. His breath sounds were rhonchorous throughout. Chest x-ray revealed a left lower lobe infiltrate. He was intubated due to his altered mental status and poor oxygenation. A toxicology consult was obtained and surfactant administration was recommended. Sixty-two minutes after his ingestion, 80 mL/m^2 of surfactant was given via endotracheal tube without complication. During his PICU admission, his respiratory status and ventilator settings remained stable without changes in airway pressures or oxygenation. The following day he was extubated and promptly developed inspiratory stridor and respiratory distress, which was treated with three doses of racemic epinephrine and steroids. His stridor improved and he was discharged home in good condition on hospital day three.
Case discussion: Tea tree oil is an essential oil that can cause severe CNS depression, as well as chemical injury to the airway and lung parenchyma. Surfactant has been shown to be an effective treatment for hydrocarbon aspiration in animals, and also shown to improve oxygenation and mortality rates when used in pediatric acute lung injury patients. Human case reports have illustrated benefit with pediatric hydrocarbon aspiration, although most administered surfactant hours to days into their clinical course. In this case, surfactant was given soon after aspiration without side effects. The patient never developed any difficulties with oxygenation or increased airway pressures while intubated, and was extubated the following day. We postulate that giving surfactant early may prevent further lung injury.
Conclusions: Essential oils can cause profound CNS depression and aspiration pneumonitis. Early administration of surfactant for significant toxicity after hydrocarbon ingestion may mitigate the inflammatory effects on the lower airway and prevent the development of further lung injury.
Keywords: Essential oil, Pediatrics, Surfactant

182. Cinnamania: 15 seconds of internet fame, 3 days in the ICU

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Background: The Cinnamon Challenge is an internet-based phenomenon where participants attempt to swallow a spoonful of cinnamon then post recordings of the explosive results on YouTube. The Cinnamon Challenge has gone viral in the last year, with 50,000 videos posted on YouTube as of April 2012. Celebrities including Illinois Governor Pat Quinn, professional basketball players Nick Young and JaVale McGee of the Washington Wizards, and comedian Jimmy Kimmel have contributed to the growing cultural fascination by recording their own attempts or featuring the phenomenon in their discourse. The AAPC issued a statement in March 2012 warning of the risks of the Cinnamon Challenge associated with choking or aspiration. We present a case of a patient who became critically ill after attempting the Cinnamon Challenge.
Case report: A 43-year old female called 911 after becoming dyspneic upon attempting to complete the Cinnamon Challenge by swallowing a spoonful of cinnamon without water. She was transported to the ED where she required intubation for impending respiratory failure. She was noted to have aspiration pneumonia on chest x-ray, and secretions suctioned from the endotracheal tube were described as thin and "cinnamon-like." (Chest X-ray image available.) On hospital day (HD) 2, she developed refractory shock requiring 3 pressors with subsequent hypotension-related liver injury (AST 1724, ALT 1135), and she was treated with moxifloxacin and piperacillin/tazobactam. An echocardiogram showed decreased cardiac contractility. On HD 3, she was successfully extubated but continued to cough up brownish secretions. She was discharged on HD5.
Case discussion: The internet is teeming with videos of people, often teenagers, attempting to swallow cinnamon then exhaling large puffs of brown smoke as they cough or vomit afterwards. Although watching these videos may be entertaining, consideration of the health risks of aspiration cannot be ignored. We present the case of a patient requiring intubation, ICU admission, and suffering multi-system organ failure as a result of attempting the Cinnamon Challenge.
Conclusions: Health care providers should educate patients and parents regarding the serious health risks of completing the Cinnamon Challenge: go big, and you may go to the ICU.
Keywords: Dietary supplement, Public health, Internet

183. Purified diphtheria toxin complication in an immunized individual

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Background: Diphtheria toxin is produced by *Corynebacterium diphtheriae*, an anaerobic gram-positive bacteria. Infection with this bacteria causes diphtheria, an upper respiratory infection that can progress to multi-organ dysfunction from toxin absorption. Although diphtheria has largely been eradicated due to the administration of the diphtheria-pertussis-tetanus vaccine, we report a case of a student who was unintentionally exposed to diphtheria toxin in a laboratory.
Case: A 29 year-old diphtheria immunized graduate student presented to the ED following an unintentional stick to her finger with a needle used to transfer laboratory grade diphtheria toxin. She aggressively irrigated her finger under running water before going

to the hospital. On examination, she had normal vital signs. At the site of the injury, there was a 5 mm blister with associated tingling in the finger. The rest of her physical examination was normal. The MSDS cites a minimum lethal dose of ≤100 ng/kg. Because her immunization status was unknown initially, the treating physician called the the poison center and the diphtheria duty officer at the Centers for Disease Control (CDC). The patient received a Tdap booster and was discharged after the toxicologist and the CDC recommended outpatient observation without antitoxin administration. However, the patient developed swelling of her affected hand over several days. The edema progressed to her wrist before improving, but she never developed systemic symptoms.

Discussion: Multi-organ dysfunction can occur as a result of diphtheria toxin's ability to block protein synthesis by inhibiting elongation factor-2. Intradermal injection of diphtheria toxin is used as part of the Schick test, which was developed over 100 years ago to test immunity to diphtheria. Subjects who developed significant skin reactivity with wheals and erythema were deemed lacking immunity. Our patient's extensive hand swelling may be due to exposure to a significant amount of toxin, larger than the standard dose used in the Schick test, or it may indicate either a partial immunity or a pseudoreaction. Exposure to diphtheria toxin is potentially lethal, particularly in non-immunized patients, and antitoxin is available through the CDC for treatment in severe cases.

Conclusions: Percutaneous exposure to diphtheria toxin may cause skin swelling in individuals lacking immunity. Intramuscular and intravenous exposures are riskier and may result in systemic toxicity. While our patient's exposure occurred in a laboratory, we are reminded of diphtheria's potential for re-emergence from poor herd immunity. This case renews our awareness of the disease and the need for vigilance regarding universal immunity.

Keywords: Occupational, Diphtheria, Public health

184. Ibogaine for opioid addiction: A Deadly treatment

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Background: Ibogaine is a naturally occurring psychedelic indole alkaloid affecting dopaminergic, serotonergic, cholinergic, and GABA neuroreceptors. Since the 1960s, ibogaine has used to treat opioid addiction and is readily available on the Internet. Although there are few documented cases of lethality, it remains illegal in the United States and portions of Europe. We report the death of a 25 year old male who took 2 grams of ibogaine to self-treat heroin addiction.

Case report: 25 year old male with a past history of heroin abuse and cardiac dysrhythmias with AICD placement, was found in a PEA arrest after ingesting 2 grams of ibogaine. He was resuscitated but he remained unresponsive and intubated with a temperature of 97.9 F, pulse of 120 bpm, and BP of 131/111 mmHg. His pupils were dilated and bowel sounds were present. Initial labs were remarkable for hypokalemia, hyperglycemia, an anion gap metabolic acidosis, and lactate of 18 mg/dL. His troponin was 0.33 ng/ml. ECG showed a sinus tachycardia with a prolonged QRS of 208 msec, and QTc of 521 msec. While in intensive care, he developed seizures,hypotension and hyperthermia requiring norepinephrine, sodium bicarbonate, and phenylephrine. Over the next 8 hours, his blood pressure continued to fall. Echocardiography showed an EF of 10% and labs showed a worsening acidosis and rising anion gap, pH 7.2, pCO_2 47, lactate 9, AST 491, and ALT 1099. The patient continued to deteriorate, and the decision was made to provide comfort care followed by his death. According to the patient's AICD, the cardiac arrest was from Vtach/Vfib. GC/MS of a capsule from the same shipment provided by the family contained ibogaine and ibogoa. Post mortem analysis showed ibogaine: heart blood concentration of 2.2 mcg/ml, iliac blood 1.8 mcg/ml, vitreous 0.98 mcg/ml, and liver 4.2 mcg/g. The patient's death was attributed to ibogaine intoxication and preexisting heart condition.

Discussion: In the face of dwindling resources for opioid addiction treatment, more patients will turn to social media to find financially accessible treatment for their addictions. As ibogaine enjoys internet support for opioid addiction therapy, it may become more popular as patients look for home treatment options. Health care providers need to be aware of ibogaine's unregulated use in the community.

Keywords: Postmortem, Substance abuse, Alternative medicine

185. Nattokinase: A novel anticoagulant

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Introduction: Nattokinase (NK) is claimed to have fibrinolytic activities similar to plasmin. It is marketed as an alternative to aspirin and coumadin in the prevention and treatment of heart disease, stroke, and thromboembolic disorders. There is a paucity of published literature regarding safety, efficacy, or complications related to use. We present a case of traumatic subarachnoid hemorrhage in a patient using NK for primary stroke prevention.

Case: A 73 year-old male with history of atrial fibrillation, congestive heart failure, 5 vessel coronary bypass, and implanted cardiac defibrillator presented after a mechanical fall from standing height with a periorbital hematoma. Medications included oral NK 2000 "fibrinolytic units" twice daily, enalapril, digoxin, carvedilol, and furosemide. The patient denied aspirin or other anticoagulant ingestion or overdose of nattokinase. He had been taking NK previously for 13 months. His head CT 3 hours post injury was unremarkable. Repeat head CT at 8 hours post injury demonstrated a small subarachnoid and intraparenchymal hemorrhage. Physical examination was only significant for a left periorbital hematoma. His initial coagulation studies demonstrated an elevated PT 14.4 (9.7–12.5) INR 1.4, aPTT 35.2 (25–34), fibrinogen 282 (200–450), and Thrombin Time 15.1 (12.8–16.2). The patient had no history or signs of liver disease. The patient was discharged two days after presentation. At no time did the patient have any neurological symptoms.

Discussion: Nattokinase, a novel soybean derived nutraceutical anticoagulant, is available over-the-counter and is in use by patients in the United States. The mechanism of action has not been clearly described. Previous studies have suggested that it may decrease fibrinogen, factor VII, and Factor VIII levels.

Animal studies suggest that it may inhibit plasminogen activator inhibitor. Further studies are needed to clarify the mechanism of action, safety, and indications.
Conclusions: Despite marketing as a safe and natural alternative to aspirin and coumadin therapy, NK represents an alternative anticoagulant of concern. Routinely available clinical laboratory tests may not be sufficient to detect clinically significant anticoagulation from NK. Bleeding complications, coagulopathies, and intracranial hemorrhage may occur with use.
Keywords: Adverse drug event, Alternative medicine, Anticoagulant

186. Retrospective review of castor bean plant exposures reported to a state-wide poison control system

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Background: Castor bean plant (Ricinus communis) exposure carries the risk of toxicity from the toxalbumin ricin, a potent inhibitor of protein synthesis. Given the paucity of available data regarding human exposures to the castor bean plant, we sought to describe to the experience of a state-wide poison control system.
Methods: We reviewed a state-wide poison control system's database for exposures to castor bean plants from 2001–2011. Data collected included age, gender, circumstances surrounding exposure, number of castor beans consumed, symptoms described, laboratory values, treatments and patient outcomes.
Results: There were 115 cases identified. Seventy-six (55%) were male with an average age of 36 years. Eighty-six cases involved ingestion of castor beans. Sixty-six (77%) of these were reported as intentional ingestions. Crushing or chewing of seeds in took place in 27 cases. On average three and five beans were consumed in unintentional cases and intentional cases, respectively. Gastrointestinal symptoms predominated: vomiting (n = 37), diarrhea (n = 15) abdominal pain (n = 15), and hemotochezia (n = 1). AST/ALT values were documented in 14 (16%) cases. Only one abnormality was noted: an asymptomatic patient one week following ingestion had AST/ALT of 93 U/L and 164 U/L respectively. Ricin was confirmed in the urine of one patient who had GI symptoms but recovered uneventfully. Twenty-two (26%) received activated charcoal, 7 (8%) received intravenous fluids, and 5 (6%) received antiemetics. Twenty-two (26%) patients were admitted for an average of 2.6 days (range 1–10 days). In 25 (29%) cases the poison center advised the treating health care providers of possible delayed symptoms as per Poisindex. No delayed symptoms, serious outcomes or deaths were reported. There were 29 non-oral exposures. Cutaneous exposures to components of castor bean plants occurred in 23 (82%) patients with rash or pruritis reported in 15 of these cases. Inhalational exposure to burning or decaying plant material occurred in 5 (17%) patients with no symptoms reported. One patient reportedly injected ground castor beans intravenously and developed hematemesis and abdominal pain. All patients were managed at home with the exception of the intravenously exposed patient who was admitted for 2 days with complete resolution of symptoms. No deaths or other serious complications were reported.
Conclusions: In this retrospective review, gastrointestinal and dermal symptoms were most commonly described after reported exposures to castor bean plants. These exposures were not associated with serious morbidity, mortality, or delayed symptoms.
Keywords: Plants, Poison center, Environmental

187. Severe chronic Vitamin A toxicity in an infant receiving dietary supplements

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Background: Acute and chronic Vitamin A toxicity have been reported in children and adults with either no or mild clinical effects to hepatic effects. To our knowledge, we report the first case of an infant with severe multisystem effects after a seven-month history of ingestion of greater than 200,000 IU/day of supplemental vitamin A drops (recommended daily infant allowance 1000 IU/day).
Case report: A previously healthy 10-month old girl presented with a three week history of loss of developmental milestones, macrocephaly, and hypotonia. The mother reported that for the past seven months the child was given unpasteurized goat's milk with multiple supplements: to each eight ounce bottle of milk (at least four bottles/day) was added 10 drops of Vitamin A (each drop contains 5000 IU of Vitamin A palmitate), flax oil, molasses, cod liver oil, and an unknown quantity of vitamin E.

The physical exam was significant for an irritable child who had macrocephaly, desquamation of orange-tinted skin, and hepatomegaly. Initial abnormal laboratory findings were ionized calcium 2.06 mmol/L, calcium 14.2 mg/dl, hemoglobin 5.5 g/dL (normal MCV), INR 2.5, direct bilirubin 1.17 mg/dL, and alkaline phosphatase 513 U/L. Long bone roentgenograms showed rachitic changes. On hospital day 2 (HD#2), the retinyl palmitate was 2.71 mg/L (normal 0–0.1 mg/L), retinol was 1.48 mg/L (normal 0.2–0.5 mg/L) and alpha tocopherol was 12.0 (normal 3.5–8.0).

On HD#2 oliguria and congestive heart failure developed after the administration of plasma and packed red cells. She was intubated due to increased work of breathing. Fluid overload and hypercalcemia were corrected. Brain MRI noted macrocephaly and expanded subarachnoid spaces. Abdominal ultrasound showed nephrocalcinosis. The patient was extubated on HD#4. She was sent home on HD#10 on baby formula without any supplements. Appropriate follow-up was arranged.
Case discussion: Vitamin A, a fat soluble vitamin that is stored in the liver, is essential for visual function and cell growth. Vitamin A toxicity is rare, but affects many organs. Hypercalcemia and rachitic changes due to a direct effect of Vitamin A on osteoclasts may occur. Vitamin A toxicity may also cause irritability, pseudotumor cerebri, bone marrow suppression, and hepatic dysfunction. The present infant had hypercalcemia, macrocephaly (due to pseudotumor in the presence of non-fused skull sutures), irritability, anemia, and hepatic dysfunction due to documented Vitamin A toxicity.

Conclusions: We report a 10-month old infant with multiple severe systemic effects secondary to chronic vitamin A toxicity. A detailed history of over-the-counter and dietary supplements is essential to clinical history taking.
Keywords: Dietary supplement, Chronic overdose, Pediatric

188. Not-so-edible susumber berries

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Background: *Solanum torvum* (Susumber berries; SB) grow in tropical climates and are a popular food in the West Indies. Berries are boiled and served with salted fish. SB are normally edible but may cause toxicity under certain conditions. Solanaceous glycoalkaloids (SGAs) have been isolated from SB in previous toxic ingestions, and are associated with gastrointestinal (GI) symptoms and neurotoxicity.
Cases: A couple shared a traditional Jamaican meal of codfish and SB, which were transported from Jamaica and frozen for weeks prior to preparation. Ten hours after ingestion, the 65-year-old husband (H) and his 49-year-old wife (W) developed GI and neurological symptoms, prompting ED evaluation. H, who ate more of the berries, presented with nausea, vomiting, unsteadiness and speech difficulty. His initial vitals were BP 154/93, HR 63, RR 16, SpO_2 98%. Physical examination showed opsoclonus, ataxia, past pointing and dysarthria with profound speech difficulties. W complained of nausea, mild dysarthria, and had rotary nystagmus on exam. Her vitals were BP 182/117, HR 63, RR 20, SpO2 97%. Both patients had normal brain MRIs. Respiratory testing showed decreased vital capacity (VC) of 1.5 (H) and 1.8 L (W), and impaired negative inspiratory force (NIF) of −20 (H) and −40 (W) cm H_2O, without clinical respiratory failure. Their hospital courses were complicated by hypertension, transaminitis and rhabdomyolysis (see Table 1). H's blood pressure peaked at 219/91, and improved with calcium channel blockers.

Laboratory abnormalities improved with IV fluids and supportive care. VC increased to 2.8 (H) and 3.6 L (W), and NIF improved to −43 (H) and −89 (W) cm H_2O. All respiratory, GI and neurological symptoms resolved in 24 hours.
Discussion: Poisoning from SB is rare. While normally edible, SB growing conditions and post-harvest stress during transport or storage may contribute to toxic SGA formation and subsequent toxicity. As in previously reported cases, the affected patients displayed dose-dependent, delayed-onset GI and neurological symptoms, and developed respiratory compromise, hypertension, hepatotoxicity and rhabdomyolysis. Respiratory failure requiring mechanical ventilation has been reported.
Conclusions: Multiple patients presenting with neurological and GI symptoms should raise suspicion for a shared toxic ingestion. A history of SB exposure should be sought, especially in patients of West Indian descent. Treatment of SB toxicity is supportive, but early recognition may better anticipate delayed complications such as hepatotoxicity, rhabdomyolysis, and respiratory failure.
Keywords: Susumber berries, Food poisoning, Neurotoxicity

Table 1. Data for abstract 188.

		AST (U/L)	ALT (U/L)	CK (U/L)
H	Initial (I)	46	31	181
	Peak (P)	489	163	39,450
W	I	406	237	NA
	P	959	1205	5281

189. Toxic Squash Syndrome: A case series of diarrheal illness following ingestion of bitter squash, 1999–2011

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Background: The cucurbitacins are a class of tetracyclic triterpenoids produced by members of the Cucurbitaceae family, which includes pumpkin, zucchini, cucumbers, squash, melons, and gourds. Cucurbitacins impart a bitter taste and are thought to protect plants against insects. Cucurbitacin-containing plants have played a role in various folk and alternative medicine practices with touted anti-inflammatory, antipyretic, and antimicrobial properties. The cucurbitacins have known cytotoxic effects and have been investigated as potential chemotherapeutic agents since the '60s.
Case series: We established a case definition of Toxic Squash Syndrome that included 1) exposure to Cucurbitaceae within $<$24 hrs, 2) specific description of a bitter taste, and 3) diarrhea with abdominal pain or nausea/vomiting and no other more likely cause of the syndrome. We conducted a retrospective review of our poison centers' databases from 1999–2011 using the search criteria of human exposure with a substance code indicating an ingestion of one of the following: cucumber, zucchini, or a member of the Cucurbitaceae family. 17 cases met the case definition of Toxic Squash Syndrome. 11 (65%) were women. All cases were adults. In most cases, only 1 or 2 bites of squash had been ingested. 4 patients required hospital admission for control of their symptoms, and 2 required ICU level care for severe dehydration.
Case series discussion: Cucurbitacins occur naturally in members of the Cucurbitaceae family. Wild-growing varieties may contain high cucurbitacin levels, but those bred for human consumption are usually nontoxic. Levels may be increased, however, due to changes in growing conditions, over-ripeness, or other factors. The toxins are heat stable and will not be destroyed by cooking. They are associated with an unpleasant bitter taste that can produce significant GI distress and associated volume loss after even a single bite of affected squash. Our series is limited by our narrow search terms and by its dependence on Poison Center reporting and consistent coding by specialists. The true incidence of this syndrome is unknown.

Conclusions: This case series represents the first published case definition of a "Toxic Squash Syndrome" following ingestion of bitter tasting squash.
Keywords: Plants, Ingestion, Food poisoning

190. Characterizing herbals and dietary supplements toxicities using the Toxicology Investigators Consortium (ToxIC) registry

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Background: Toxicities resulting from herbals and dietary supplements are likely under-recognized. The ToxIC Registry is a national database of consultations provided by medical toxicologists.
Objective: To characterize herbals and dietary supplements toxicities using preliminary data from the ToxIC registry.
Methods: The ToxIC Registry began collecting data on bedside toxicology consults in January 2010. Patient demographic and healthcare data is de-identified and entered into a prepared password protected electronic database with fixed fields and is maintained by the American College of Clinical Toxicology. For this study, the database was retrospectively queried for all herbals, dietary supplementsand vitamins cases between January 15, 2010 and March 30, 2012.
Results: During period studied, the ToxIC Registry collected data on 12,125 cases. Herbals, dietary supplements and vitamins accounted for 65 (0.5%) cases. The largest age group involved in these cases was 19–65 years (35, 53.8%), while 43% (28) were 18 years of age or younger. Thirty-six patients (55%) were female. Substances ingested most commonly were multi-vitamins (9, 13.8%) followed by caffeine (8, 12.3%), iron formulations (7.7%), vitamin C (4, 6%) and echinacea (3, 4.6%). Twenty-seven cases (42%) involved co-ingestants: most commonly non-opioid analgesics (13, 20%) then sedative/hypnotics (9, 13.8%) and either EtOH, anticholinergics/antihistamines or cardiovascular medications (5, 7.7%). Nine (13.8%) patients exhibited toxidromes on presentation, including 2 anticholinergic, 5 sedative-hypnotic and 2 sympathomimetic. The most commonly associated end-organ toxicities were neurologic (27, 41.5%) followed by cardiovascular effects. Five (7.7%) patients had a metabolic acidosis and 3 (4.6%) had electrolyte abnormalities. Three (4.6%) patients developed coagulopathies. Five (7.7%) patients developed acute kidney injury. Two (3.1%) developed rhabdomyolysis. Regarding treatment, 9 patients (13.8%) received benzodiazepines, 7 (10.7%) received N-Acetylcysteine, 2 (3.1%) received activated charcoal and 2 (3.1%) underwent urinary alkalinization. Fifteen (23%) patients received IVF resuscitation and 4 (6%) required endotracheal intubation.
Conclusions: Herbals, dietary supplements and vitamins poisoning cases are a small but important proportion of cases of sufficient seriousness to require care by medical toxicologists. Although further research is needed, this initial characterization of patients poisoned by herbals, dietary supplements and vitamins may contribute to the development of educational and prevention efforts.
Keywords: Herbals, Dietary supplement, Vitamin

191. Not just for elementary school: A field trip and a new antidote solidify trainees' knowledge of amatoxin poisoning

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Background: Acute poisoning after ingestion of amatoxin-type mushrooms is an uncommon emergency department (ED) presentation. Accurate identification may prove difficult, as assistance of an experienced mycologist may not be readily available and patients may damage identifying characteristics of the mushroom when picking them or preparing them to eat. We present a case of accidental poisoning by Amanita virosa, in which a field trip to the mushroom source was the impetus for patient transfer and use of a new antidote.
Case report: A 74 y.o. female presented to an ED for evaluation of vomiting and diarrhea that started 9 hours after ingesting 2 wild mushrooms. Physical examination was remarkable only for mild tachycardia (104 bpm). Laboratory evaluation included normal chemistries, renal function, complete blood count and liver function tests (LFTs). Due to intractable vomiting and diarrhea and concern for poisoning with amatoxin, the patient was admitted.

Pictures of the mushrooms were e-mailed from the local ED to the poison center (PC), who contacted an experienced mycologist to aid in identification. Because of the picture quality and because the patient had picked them from the ground, the mycologist was unable to discern whether the mushrooms were Amanita spp. or Lepiota spp.

The medical toxicology fellow contacted the family of the patient, who gave directions to the area from which the mushrooms were picked. Survey of that area revealed numerous mushrooms. Samples were dug from the ground and taken to the PC, where the mycologist identified them as Amanita virosa.

LFTs obtained on hospital day 2 were elevated. The patient was transferred to a tertiary care center and received treatment for 5 days with intravenous silibinin as part of a multicenter clinical trial. LFTs (AST 5403 U/L) and INR (4.0) peaked on hospital day 4 and then trended down. The patient was discharged home in good condition and without permanent sequelae.
Case discussion: Intravenous silibinin is an antidote currently under investigation for amatoxin-induced hepatic failure. Because amatoxin poisoning is uncommon and because clinical and laboratory evidence of poisoning may not occur for hours, diagnosis and antidotal therapy could be delayed. In our case, notification of the PC and a field trip to identify the mushroom served as an impetus for the patient's receipt of antidote.
Conclusions: Intravenous silibinin is a promising antidote for amatoxin-induced hepatic failure. A field trip to gain hands on experience regarding features of amatoxin-containing mushrooms can positively affect patient care and help to solidify knowledge gained by medical trainees.
Keywords: Mushroom poisoning, Silibinin, Hepatotoxicity

192. Status epilepticus and acidosis after niacin misuse

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Background: Ingestion of niacin in the belief that it will defeat urine drug screens has gained popularity in recent years. We present a patient who was initially hypoglycemic, and remained in status epilepticus for hours after glucose correction.
Case presentation: A 15-year-old female with history of bipolar disorder and marijuana use was found unresponsive by her mother around noon after complaining of nausea earlier that day. Home medications were escitalopram, lamotrigine, and risperidone. There was no history of prior seizures. Point of care (POC) glucose by EMS was <20 mg/dL. Five minutes after dextrose 25 g IV, repeat POC glucose was 210 mg/dL. In the Emergency Department the patient was not hypoglycemic but had generalized seizures that recurred despite multiple lorazepam doses. Her initial vital signs were BP 117/76 mm Hg, heart rate 115/min, respirations 40/min, temperature 94.6 degrees F. She was intubated, loaded with fosphenytoin and phenobarbital, and propofol infusion was begun. Labs were notable for arterial pH 6.97, pCO_2 18 mm Hg, pO_2 216 mm Hg, serum bicarbonate 5 mEq/L, anion gap 29, lactate 17 mmol/L, and WBC 27,600/mm^3. Electolytes were otherwise normal. Urine drug of abuse screen was negative. Acetaminophen, salicylate, ethanol, ethylene glycol, and methanol were not detected. Head CT and LP results were unremarkable.

The patient was transferred to a children's hospital. Anticonvulsants were held and EEG showed no seizure activity. The patient was given pipercillin/tazobactam, and acyclovir. She was extubated the next morning and admitted to taking extended release niacin, approximately 1.5 g daily for 7–10 days to defeat an upcoming drug screen. She denied intentional self-harm and substance abuse other than smoking marijuana 2–3 weeks earlier. Acid-base status normalized within 24 h, and hypoglycemia did not recur. Serum nicotinic acid, nicotinamide, and nicotinuric acid levels were at 49, 8000, and 14 ng/mL respectively. The patient had dizziness and blurred vision that, along with leukocytosis, resolved by hospital day (HD) 4. Mild elevations of AST (156 U/L) and ALT (100 U/L) peaked on HD 4, and normalized by HD 6. Creatinine was initially normal but rose to 2.99 mg/dL on HD 6, and then improved. This may have been a complication of acyclovir given for the first 3 days.
Discussion: Large niacin ingestions have been associated with early hypoglycemia, metabolic acidosis, leukocytosis, and liver enzyme elevation. Recurrent seizure activity despite correction of hypoglycemia has not, to our knowledge, been reported following niacin misuse.
Conclusions: Although the mechanism remains unclear, status epilepticus after correction of hypoglycemia may be a previously unrecognized effect of niacin overdose
Keywords: Niacin, Drug screen, Seizure

193. Am I blue? Highest reported methemoglobin level after boiler water ingestion

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Introduction: Methemoglobinemia secondary to ingestion of boiler water is exceedingly rare and is believed due to nitrate preservatives. A case of a patient who developed profound methemoglobinemia after drinking boiler water is presented.
Case report: A 50 year old man without medical history and prescribed no medications who was living in his car entered his workplace on a cold night in order to keep warm and obtain hot liquids. He drained a boiler pipe into a cup. The first and second cups were brown so he discarded them. He mixed 2 cups of yellowish water into a bowl of noodles and drank it. He subsequently developed palpitations and a feeling of "impending doom". The patient drove himself to the ED where his skin appeared blue-gray. Vital signs included: temperature 36.4°C, heart rate 106 bpm, respiratory rate 20 breaths/min, blood pressure 105/72 mmHg, and room air oxygen saturation 82% (minimally improved with 02). His blood was dark brown, and cooximetry measured 59% methemoglobin and carboxyhemoglobin level of 2.9% (normal 0–1.5%). His hemoglobin was 15 g/dL (normal 14–17.5 g/dL). 150 mg IV methylene blue was administered; repeat cooximetry measured 12% methemoglobin. He was admitted and recovered uneventfully. UCDS (GC/MS) detected no exogenous agents. Attempts to obtain a boiler water sample for analysis were unsuccessful.
Discussion: Rare reports of inadvertent boiler water ingestion (used to make coffee in an office and soup in a school) resulting in methemoglobinemia were due to nitrates and nitrites. Commercial conditioner fluid and some coolants are known to contain nitrite and sodium metaborate. To date this is the highest reported level of methemoglobin from boiler water consumption.
Conclusions: Physicians should be aware of uncommon, yet potentially dangerous sources of oxidizing agents capable of generating life-threatening methemoglobinemia. Further, proper labeling of these potential hazards is paramount to minimize potential exposure.
Keywords: Methylene blue, Ingestion, Public health

194. Fatal restaurant carbon dioxide exposures

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Background: CO_2 is a simple asphyxiant gas not classified as toxic or harmful in accordance with Globally Harmonized System of Classification and Labeling of Chemicals. Concentrations higher than 1% (10,000 ppm) will make some people feel drowsy. Concentrations of 7% to 10% may cause suffocation, dizziness, headache and unconsciousness within a few minutes to an hour. One source of CO_2 comes from commercially available carbonation systems used to carbonate soft drinks and soda water. The purpose of this case series is to publicize this potentially toxic scenario, find any reports of CO_2 exposures, investigate the similarities of these exposures, and determine if new guidelines or measures should be taken to prevent recurrence.
Case report: Our Poison Center was recently involved in an eleven-person exposure to restaurant-grade CO_2 at a local fast-food

chain restaurant where one victim died. The CO_2 was used in the carbonation system of the soft drink machine. Upon investigation, it was discovered that a leaking hose caused release of CO_2 into restrooms adjacent to where the leak was found. Utilizing a basic internet search, we reviewed recent restaurant CO_2 exposures. In addition to the case reported to our Poison Center, two other reports were identified in other states. Poison centers and local authorities were contacted for further information on each case. The second case also involved a leaking hose, and the third case involved a leak while refilling CO_2 tanks in an enclosed area.

Case discussion: Of these known cases, those with fatalities happened at the same restaurant chain and involved the same CO_2 supply company. The leaks were due to either a faulty valve or a tear in the hose connected to the CO_2 tank. All exposures occurred in poorly-ventilated areas of the restaurants.

Conclusions: Based on the cases described here, we believe that restaurant soft drink machines and CO_2 tanks may pose a significant public health hazard. Safeguards are needed to alert management if carbonated systems installed in restaurants develop leaks. The systems need to be placed in adequately vented areas where CO_2 would escape outdoors if a leak were to occur. Lastly, inspections of food service establishments should include safety inspection of the CO_2 carbonation system to ensure they are installed properly and working correctly.

Keywords: Public health, Carbon dioxide, Restaurant

195. Detection of a DMPS-MMA(III) complex during treatment of massive arsenic ingestion

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Background: Unithiol (DMPS) has been advocated for the treatment of acute inorganic arsenic intoxication but study of its in vivo metabolism and direct chelation of arsenic is limited. We report urinary arsenic speciation during DMPS treatment of a fatal massive arsenic ingestion.

Case summary: An 85 kg 34 year old male was transported to the ED five hours after an intentional ingestion of 5 grams of arsenic trioxide. En route he became unresponsive and profoundly hypotensive. He was intubated, and dimercaprol (BAL) 300 mg IM was administered within 20 minutes. BP improved with IV fluid and norepinephrine. Initial ABG revealed pH $<6.8/pCO_2$ $84/pO_2$ 505. Lactate was 15.6. Abdominal films revealed metallic densities in the upper abdomen that persisted despite 8 hours of attempted whole bowel irrigation. Chelation was continued with dimercaprol 300 mg IM for the second dose, followed by unithiol (DMPS, Dimaval®) 250 mg IV q4 h $\times$ 9 doses. Despite initial stabilization, within 12 hours multi-organ failure ensued, characterized by severe metabolic acidosis, ARDS, oliguria, and coagulopathy. A prolonged QTc was present, although the preterminal rhythm was a monomorphic wide complex tachycardia followed by asystole 52 hours post ingestion. Autopsy revealed acute hemorrhagic enterocolitis and acute renal tubular necrosis, and were attributed to the direct toxic effects of arsenic. The associated hypovolemia and ischemia resulted in hepatic necrosis and splenic infarctions. Diffuse soft tissue edema was also noted.

Urine arsenic species were analyzed by HPLC combined with ICPMS. On day 2, urinary arsenic excretion was as high as 62,531 µg/L, consisting of As(III) 47.8%, As(V) 0.6%, MMA(V) 1.2%, and DMPS-MMA(III) complex 50.4%. No DMA(V) or DMPS-As(III) were detected.

Discussion: Multi-gram ingestions of inorganic As are often fatal despite intensive care and chelation. Prior work has identified the *in vivo* formation of a DMPS-MMA(III) complex, and this case reports observation of this complex during treatment of massive inorganic As intoxication. The presence of a large quantity of DMPS-MMA(III) combined with the relatively low concentration of MMA(V), and the absence of DMA(V) or DMPS-As(III) are consistent with DMPS exerting antidotal properties by binding in vivo to MMA(III), a highly toxic metabolite of inorganic arsenic. Although not widely available in North America, DMPS is the only arsenic chelator demonstrated to directly form an *in vivo* complex with an arsenic metabolite.

Keywords: Arsenic, Chelation, Ingestion

196. 800 Pebbles in a stream: whole bowel irrigation and colonoscopy for staggered lead shot ingestion

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Background: Acute poisoning after ingestion of lead containing foreign bodies is uncommonly reported. Various techniques for reduction of systemic lead exposure have been described; however due to the paucity of experience, optimal guidelines for management have not been established. We present a case of staggered lead shot ingestion accompanied by a rapid elevation in blood lead levels (BLL).

Case report: A 13 year old, previously healthy male presented to the emergency department after a staggered ingestion of lead shot in a suicide attempt. He reported ingesting 0.5 ounces on three occasions over 9 days, approximately 800 pellets.

Four days after the last lead intake, the patient presented for evaluation. He denied symptoms and had not seen any pellets pass in his stool. His physical examination was unremarkable. The initial abdominal x-ray (AXR) was consistent with the reported history. The patient's whole BLL was 23 ug/dL and the remainder of his laboratory studies was unremarkable.

The patient was admitted and underwent whole bowel irrigation at a rate of 1.5 L/hr for 7 days. Daily BLLs and AXRs were obtained.

On hospital day 8, the patient underwent colonoscopy, which removed a large burden of lead pellets. A post-procedure BLL was obtained and was not significantly changed from prior (23 ug/dL). The patient was sent home with close monitoring, which included frequent BLLs and clinic follow up.

An AXR at his one month clinic follow up appointment showed no residual lead pellets and his two month BLL was 10 ug/dL.

Case discussion: This case raises several issues pertinent to the management of lead-containing foreign body ingestion. First, significant elevation of whole blood lead levels can be achieved after ingestion of lead containing foreign bodies, necessitating intervention to prevent sequelae. Second, WBI may be insufficient to expel the poison from the gastrointestinal (GI) tract. In our case, due to insufficient progress with WBI after 7 days and a large burden of pellets near the cecum and fear of appendicolith formation, we attempted removal of the large peri-cecal congregation with colonoscopy, which was ultimately successful in freeing that area of lead.
Conclusions: Elevated blood lead levels can occur after ingestion of lead-containing products. When the number of ingested foreign bodies is high, WBI may be insufficient to clear the GI tract. Colonoscopy may be a safe and acceptable method for removing collections of lead pellets from intestinal tortuosities, avoiding prolonged hospitalization and laparotomy.
Keywords: Lead, Decontamination, Ingestion

197. Toxic vaults: Chloropicrin theft deterrents in a historic midwestern county courthouse

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Background: Chloropicrin is a colorless, oily liquid first synthesized in 1848 as a fungicide, insecticide, and nematocide. It was historically used in chemical warfare, and is responsible for clusters of illness outbreaks related to agricultural use. Exposure is tolerated for less than one minute at 15 ppm. Higher concentrations are associated with lacrimation and nausea, and may result in death. Limited human data suggest an LC50 of 120 ppm at 30 minutes. We present a mass chloropicrin exposure during the renovation of a historic courthouse.
Case report: The Department of Health (DOH) was notified of nine contractors remodeling a 1905 rural courthouse who presented to an emergency department (ED) after violating a theft deterrent plate containing two glass ampules behind the lock dial of a vault door. All presented with mucous membrane and eye irritation, but appropriate respiratory rates and oxygen saturations on room air. Five of nine required eye irrigation in the ED; a sixth declined. One received outpatient fluticasone for an asthma exacerbation. All nine were discharged from the ED. Local emergency providers knew of no additional sequelae.

The theft deterrent plate and two similar plates from additional vault doors were sent to DOH, a Centers for Disease Control (CDC) level 1 Laboratory Response Network laboratory. An intact vial was scored and sampled under a class III biosafety hood. Fourier transform infrared spectroscopy (FT-IR) revealed a visual spectral match with chloropicrin. Gas chromatography/mass spectroscopy revealed pure chloropicrin. Preliminary results were communicated to the treating medical team on the same evening.
Case discussion: With the advent of acetylene torches in the 1900s, irritant theft deterrent devices were installed on an unknown number of safes. Few chloropicrin exposures have resulted from violation of chemical theft deterrent devices in turn of the century safe boxes. This unexpected occupational exposure to chloropicrin resulted in minor irritant effects. The DOH laboratory worked in collaboration with county and hospital officials and the regional poison center to quickly identify the offending substance, eliminating concerns for further anticipated medical sequelae.
Conclusions: We report an unusual mass exposure of nine construction workers to chloropicrin as they remodeled a historic courthouse. Chloropicrin is a potential occupational hazard when renovating historic buildings containing vaults, and personal protection equipment should be considered in these circumstances. Close local, laboratory, and poison center collaboration were critical in the response to this mass chemical exposure.
Keywords: Occupational, Forensics, Chloropicrin

198. A comparison of occupational fatalities from inhalation injury in confined versus non-confined spaces from 1992 through 2010

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Background: Confined spaces include underground tanks, storage bins, manholes, pits, silos, process vessels, and pipelines. Confined spaces are generally considered to be the most dangerous setting for the inhalation of chemicals.
Methods: The Census of Fatal Occupational Injuries (CFOI) part of the Bureau of Labor Statistics Occupational Safety and Health Statistics program, uses diverse state, federal, and independent data sources to identify all fatal work injuries in the US. We queried the CFOI for fatalities from inhalation injury in confined spaces and non-confined spaces from 1992 through 2010. Poisson probabilities were calculated to determine the statistical significance of changes in mortality related to open versus closed work settings as reported by the CFOI.
Results: Table 1 lists fatalities for occupationally related deaths in both confined and non-confined spaces from 1992 through 2010.

Table 1. Occupational deaths in confined versus non-confined spaces.

Year	Confined spaces	Non-confined spaces	Total
1992	51	18	69
1993	43	15	58
1994	44	24	68
1995	35	21	56
1996	56	12	68
1997	25	19	44
1998	27	14	41
1999	23	23	46
2000	22	20	42
2001	25	18	43
2002	20	23	43
2003	27	32	59
2004	13	32	45
2005	21	45	66
2006	15	38	53
2007	32	32	64
2008	17	35	52
2009	14	30	44
2010	25	30	55

From 1992 through 2001 deaths in closed spaces exceeded those in non-confined spaces. From 2002 through 2010, deaths in non-confined spaces exceeded those in confined spaces. For the closed space death data set, the change appears to have occurred starting in 1997, when the fatality rate decreased by half, a change that is statistically significant at the $p < 0.001$ level. Open space deaths data apparently increased starting in 2003, which was also significant at the $p < 0.001$ level.

Conclusions: Confined spaces are generally thought to be more hazardous than non-confined spaces as confined spaces may have limited ventilation, increased escape difficulty, and increased difficulty for rescue. CFOI data reveal that fatalities due to inhalational injury became more prevalent in non-confined spaces beginning in 1997. It is likely that multiple factors including better regulatory protection and improved awareness for workers operating in closed spaces is responsible for the apparent increase in non-confined space deaths. Analysis of the raw data representing each of the deaths at issue will be required to determine the factors influencing this change. Occupational inhalational exposures in non-confined spaces represent serious risks for workers. Consequently, worker education as well as regulatory guidance should be adjusted to reflect this previously unrecognized risk for occupational death.

Keywords: Occupational, Death, Epidemiology

199. Environmental anthrax: Where the buffalo roam

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Background: Environmentally acquired inhalational anthrax caused by *Bacillus anthracis* occurs through exposure to contaminated animal products (e.g. wool, hair, or hide), direct contact with infected animals, or from soil. After inhalation, anthrax spores release lethal factor (LF) and edema factor (EF), which combine with protective antigen (PA) to cause a hemorrhagic mediastinitis and occasionally a necrotizing pneumonia. The course of disease is biphasic including a prodromal phase of myalgias and fever and a fulminant phase, characterized by worsening respiratory symptoms. Treatment includes doxycycline, ciprofloxacin, or penicillin G procain, given as a multidrug regimen. Anti-protective antigen immunoglobulin is available from the CDC as an adjunctive treatment.

Case report: A 62 year-old Male with past medical history significant for remote chemical pneumonitis from pesticide exposure presented with malaise, fever, dyspnea, and productive cough for 4 days. Blood cultures obtained revealed *Bacillus anthracis*, confirmed by the State Department of Health. An FBI investigation ruled out bioterrorism and the CDC concluded that his exposure was environmental. Possible sources of exposure for this patient included collecting rocks from dry riverbeds and contact with wild mule and bison during a three-week vacation in national parks in the Western United States.

Showing no improvement on azithromycin and ceftriaxone, he subsequently developed hypoxic respiratory failure and required mechanical ventilation. Chest computed tomography (CT) revealed extensive right lung infiltrates with bilateral effusions, which were drained by interventional radiology. The CDC supplied anti-anthrax immunoglobulin to be given with a multidrug antibiotic regimen of ciprofloxacin, clindamycin, and meropenem. Less than 20 people worldwide have received anthrax immune globulin since its development. Under this regimen, the patient sustained a full recovery and was discharged with a course of ciprofloxacin.

Case discussion: Inhalational anthrax is a rare disease. (Review of pubmed using search term "anthrax case reports" from 2004–2012, revealed only 3 published case reports of environmentally acquired inhalational anthrax). Due to successful implementation of a rapid response laboratory network, appropriate treatment for this patient was instituted in a timely manner. He survived despite progression to the fulminant phase, which has had a reported mortality rate of 97% without antibiotic treatment.

Conclusions: Environmentally acquired inhalational anthrax is rare but lethal. Mortality is improved by rapid diagnosis, early antibiotic therapy and potentially by administration of anti-anthrax immunoglobulin.

Keywords: Environmental, Public health, Anthrax

200. Consumption of pesticide-treated wheat seed by a rural population in Malawi

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Background: An outbreak of typhoid fever in rural Malawi triggered an investigation by the Malawi Ministry of Health and the Centers for Disease Control and Prevention in July 2009. During the investigation, villagers were directly consuming washed, donated, pesticide-treated wheat seed meant for planting. The objective of this study was to evaluate the potential for pesticide exposure and health risk in the outbreak community.

Methods: A sample of unwashed (1430 grams) and washed (759 grams) wheat seed donated for planting, but which would have been directly consumed, was tested for 365 pesticides. Results were compared to each other (percent change), the US Environmental Protection Agency's (EPA) health guidance values and estimated daily exposures were compared to their Reference Dose (RfD).

Results: Unwashed and washed seed samples contained (respectively): carboxin, 244 and 57 ppm; pirimiphos methyl, 8.18 and 8.56 ppm; total permethrin, 3.62 and 3.27 ppm and carbaryl, 0.057 and 0.025 ppm. Percent change calculations (unwashed to washed) were: carboxin, −76.6%; pirimiphos methyl, +4.6%; total permethrin, −9.7%; and carbaryl −56.1%. Only carboxin and total permethrin concentration among washed seed samples exceeded US EPA health guidance values (285× and seven times respectively). Adult estimated exposure scenarios (1 kg seed) exceeded the RfD for carboxin (8×) and pirimiphos methyl (12×). Adult villagers weighing 70 kg would have to consume 0.123, 0.082, 1.06, and 280 kg of washed seed daily to exceed the RfD for carboxin, pirimiphos methyl, permethrins and carbaryl, respectively.

Conclusions: Carboxin, pirimiphos methyl, permethrins and carbaryl were detected in both unwashed and washed samples of seed. Carboxin, total permethrin and carbaryl concentration were partially reduced by washing. Health risks from chronic exposure to carboxin and pirimiphos methyl in these amounts are unclear. The extent of this practice among food insecure communities receiving relief seeds meant for planting and resultant health impact needs further study.
Keywords: Pesticide, Food poisoning, Environmental

201. Chelation therapy by medical toxicologists in the U.S.

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Background: Chelation therapy remains a controversial treatment. While National Health Statistics (NHS) data (2008) reported 72,000 children (< 18 years) and 111,000 adults (≥ 18 years) were given chelating agents in 2007 the AAPCC National Poison Data System (NPDS) data showed that relatively few cases of chelation therapy are reported to a poison control center. In 2010 out of the 10,638 possible heavy metal exposure reported to the NPDS, only 358 (3.4%) were treated with chelating agents. It is unclear as to how often medical toxicologists actually prescribe chelation therapy. We investigated the Toxicologic investigators Consortium (ToxIC) Registry data of the American College of Medical Toxicologist (ACMT) to see the frequency and pattern of chelation therapy among medical toxicologists in the US.
Methods: ACMT maintains a registry of encounters seen by participating medical toxicologists at the bedside and in the clinic. A total of 34 centers throughout the U.S. regularly contribute data into the registry. Based on a 2009 survey of ACMT members who practice medical toxicology at the bedside, centers who participate in the Registry account for a majority of active medical toxicology practices. We queried the registry data for the frequency of toxic metal exposure and pattern of chelation therapy from January 1st, 2010 to December 31st, 2011 using search terms such as metals/metalloids, individual metal name and chelating agents
Results: 10,414 encounters were reported in ToxIC during the 2 year period. 328/10,414 (3.1%) were for exposure to metals. Chelating agents were used in 31 cases, 25 of which were prescribed by medical toxicologists and 6 which presented after urine chelation challenge test by naturopaths or self-chelation. During the same period 3982/10,414 (39%) received other antidotes. Chelators represent only 25/4007 (0.6%) of all antidotes used by medical toxicologists.

A chelating agent was prescribed in 25 (7.6%) of the 328 metal exposures; 18 children and 7 adults Dimercaptosuccinic acid (DMSA) was used in 18 cases, 15 of these were for lead, one was for arsenic, one for mercury and one for an unknown metal. Ethylenediaminetetraacetic acid (EDTA) was used in 5 cases, all for the treatment of lead poisoning. Deferoxamine was used in 4 cases, 2 for iron, one for aluminum and one for sucralfate. British anti Lewisite was used once to treat lead encephalopathy in combination with EDTA.
Conclusions: Medical toxicologists rarely prescribe chelation therapy. This is consistent with uncommon use of chelating agent reported to the NPDS. Medical Toxicologist's prescribing of chelating agents likely accounts for a tiny proportion (<<1%) of all use of chelating agents as reported in the NHS data.
Keywords: Chelation, Medical toxicologists, Metals

202. Accidental cartap poisoning: Our experience

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Background: Cartap Hydrochloride is a pesticide commonly used to control weevil and caterpillars. It is frequently used by farmers producing cauliflower and cabbage. Human toxicity due to this compound is uncommon. We report four cases of the poisoning managed at our centre.
Methods: Retrospective analysis of 4 cases presenting to the medical emergency at out institute over a period of 3 years was carried out. The demographic data, presenting symptoms, vitals, baseline laboratory parameters and final outcome was recorded. The patients were followed up for development of any complications daily till discharge.
Results: 4 patients, all males in the age range of 24–45 were admitted over past 3 years. All 4 patients were farmers and had accidental exposure to the compound. All of them were exposed during the dusting of crop with the pesticide. None of the patients were using protective measures while dusting. All of them presented with nausea, vomiting, and breathlessness. There was no history of excessive salivation, bronchorrhea, seizures, altered behavior, chest pain or palpitations. All the patients had a characteristic green colored staining of the hands. All of them had tachycardia and tachypnoea. Wheezing was noted in 3 patients but there were no crepititions. All the patients showed evidence of mild hypoxia on pulse oximetery but had respiratory alkalosis on arterial blood gas analysis performed after initiation of treatment. Chest X ray was normal in all. Decontamination was performed using tap water and all the patients were given oxygen supplementation along with symptomatic treatment in the form of nebulized salbuatamol and a single dose of 100 mg hydrocortisone. All of them were observed in the emergency for development of complications. No systemic complications were noted and they were discharged after 24 hours.
Conclusions: Accidental exposure to Cartap hydrochloride powder may result in mild respiratory symptoms with nausea. No severe toxicity was seen in our patients with mild exposure.
Keywords: Insecticide, Inhalant, Public health

203. Spinosad insecticide exposures reported to poison centers

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Background: Spinosad (spinosyn A and spinosyn D) is a biologically-derived insecticide created from the soil-dwelling bacteria Saccharopolyspora spinosa. It is used to control a variety of insects. Spinosad is reported to have low acute mammalian

toxicity. In 2011 the US approved spinosad for the treatment of head lice in humans. This study describes spinosad exposures reported to poison centers.
Methods: Spinosad exposures reported to a statewide poison center system during 2000–2011 were identified. This was done by searching the PoisIndex code description field and the verbatim reported substance field name of all of the records for any products known to contain spinosad. The distribution of exposures was determined for various demographic and clinical factors.
Results: Thirty-five exposures were identified during 2000–2011, of which 85.7% occurred during 2008–2011. Patients 5 years and younger accounted for 65.7% of the cases; 57.1% of the patients were male. All of the exposures were unintentional. The exposure route was ingestion alone (91.4%), inhalation alone (2.9%), dermal alone (2.9%), and ingestion and dermal (2.9%). The exposure occurred at the patient's own residence (97.1%) or at school (2.9%). All of the patients were managed on site. The medical outcome was no effect (42.9%), minor effect (8.6%), not followed but judged nontoxic (17.1%), and not followed but minimal clinical effects possible (31.4%). The adverse clinical effects, reported in 1 case each, were erythema, abdominal pain, diarrhea, vomiting, and coughing.
Conclusions: Spinosad exposures are rarely reported to poison centers, although the number may be increasing. The exposures can usually be handled on site with little or no adverse effects expected.
Keywords: Insecticide, Poison center, Spinosad

204. Poison center calls related to water treatment facilities

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Background: Water treatment facilities treat water to make it potable for public consumption or process waste water to release it back into the environment. A variety of chemicals are used in water treatment, thus creating the potential for hazardous chemical exposures. This study describes exposures involving water treatment plants reported to poison centers.
Methods: The electronic notes field of each of the exposures reported to a statewide poison center system during 2000–2011 was searched for the terms "water treatment," "water plant," "water facility," or "waste water." The records containing these terms were further reviewed to identify those exposures that occurred in or were otherwise related to a water treatment facility. The distribution of these exposures was determined for various demographic and clinical factors.
Results: A total of 55 patients were identified in 51 separate incidents. The annual number of patients ranged from 1 to 7 with no annual trend. There was no seasonal trend. One patient was age 17 months and the remaining had a mean age of 37 years (range 17–66). The patients were 87.3% male. The exposures involved inhalation (60.0%), dermal contact (25.5%), ingestion (20.0%), and ocular (16.4%), with 21.8% of the exposures involving multiple routes. All of the exposures were unintentional; 89.1% involved individuals at work, 3.6% visitors, and 7.3% people at home where the substance was brought home or was thought to have drifted from the facility. The management site was 63.6% patient already at/en route to a healthcare facility, 21.8% on site, 12.7% referred to a healthcare facility, and 1.8% unspecified. The medical outcome was 12.7% no effect, 43.6% minor effect, 18.2% moderate effect, 1.8% major effect, 16.4% not followed (minimal effects possible), and 7.3% unable to follow (potentially toxic). The most common products involved in the exposures were chlorine (52.7%), hypochlorite (9.1%), sodium hydroxide (7.3%), aluminum sulfate (5.5%), ferrous sulfate (5.5%), and sulfuric acid (5.5%).
Conclusions: In spite of the large number of water treatment facilities in the US, few incidents involving them are likely to be reported to poison centers. Individuals involved in water treatment facility-related incidents tended to be adults, male, and workers at the facilities. Most of the exposures involved inhalation of substances. The exposures that were reported were not likely to have serious outcomes and could be managed on site. Most of the exposures involved chlorine or related substances. Continued education of water treatment facility staff would be useful in preventing and managing such exposures.
Keywords: Poison center, Water treatment, Chlorine

205. White Halloween: Mass carbon monoxide poisoning following a Nor'easter

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Background: On October 29th 2011, an unusual snowstorm, caused power loss to over 900,000 residences in our state and was not restored for up to 12 days. This event led to an influx of reports of carbon monoxide (CO) poisoning. We report on the characteristics of these exposures.
Method: This is a descriptive study using data collection software (Toxicall® Aurora, Co). A scan statistic was used to identify associated call clusters matched to event date and utility company power restoration data. 193 storm-related CO exposures were identified, with all clusters occurring between 10/30/11 and 11/7/11. The prior 4 year historic mean for the same period was 7 CO exposures.
Results: 168 (87%) of the 193 involved multiple patients exposures (73% acute), patient gender was female (55%) (Pregnant N = 7 [9 to 37 weeks]); male (40%); unknown (5%); age range (26 days–89 yrs). Call site was health care facility (HCF) (78%); Exposure site was residence (99%). 88% of patients were treated in a HCF. 104 (54%) patients had carboxyhemoglobin (COHb) concentrations measured (range < 2–33%). The most common clinical effects included: headache (32%), nausea (17%), dizziness (12%), vomiting (10%); 6% of patients experienced syncope. Treatments included 100% O_2 (83.6%); HBO_2 (7.7%).
Discussion: In this large case series, we were able to capture objective measurements of CO poisoning for a large percentage of patients. Patient histories revealed that poisonings often occurred inside the home even when generators were located outdoors. Another significant cause of CO poisoning was indoor use of charcoal grills as heating sources.
Conclusions: In this case of prolonged storm-related power outages, the use of portable generators was implicated as the major source of CO poisoning. This differs from the more common

environmental occurrences of CO poisoning from faulty furnaces and water heaters.

Our analysis revealed that CO poisoning occurred even when generators were used outdoors.

Keywords: Carbon monoxide, Case series, Environmental

206. Convulsion: A rare, maybe overlooked presentation of caprolactam poisoning

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Background: Caprolactam is widely used in nylon fiber industry, not only an irritant to respiratory tract, eye, skin and mucous membrane but also a convulsant poison after extensive expose. We reported a young women exhibiting dermatitis and convulsions after exposure to caprolactam.

Case report: A 30 year-old female referred to emergent department with initial presentations of convulsion and conscious change. The patient was well in the past and had been working in the plastic recycling industry for 3 months. She experienced vomiting and lethargy while finishing works. Seizure attack was noted on the way to hospital. Physical examination revealed erythematous change and pigmentation of bilateral elbows and thighs. Laboratory tests showed leukocytosis,hypokalemia and metabolic acidosis. Electroencephalogram revealed abnormal cerebral activity in bilateral temporal area. Urine tests by GC/MS reported caprolactam. The maximum caprolactam concentration (778,189 ng/mL) was observed at 17 h after seizure attack. She recovered uneventfully after conservative treatment.

Discussion: Caprolactam demonstrated an antagonic property of GABA (gamma-aminobutyric acid) mediated inhibition in animal studies and resulted in central nervous toxicities, such as seizure, dystonia and paresthesia. Half of workers exposed to caprolactam might develop occupational dermatitis through prolonged skin contact. Coincidence of the typical skin lesion with unexplained CNS disorder in the patient strongly suggested caprolactam poisoning was the main cause of the illness.

Conclusions: Occurrence of neurological disorder of workers in the plastic industry should take consideration of caprolactam poisoning, especially when dermatological manifestations are encountered.

Keywords: Caprolactam, Neurotoxicity, Seizure

207. Baseball's deadly hazard: Brain cancer

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Background: Baseball is lauded as a game of statistics. Perhaps one of the most stunning statistics is not based on player performance but on the incidence and mortality from brain tumors. In a recent Chicago Tribune article, it was suggested that professionals in the game of baseball are more likely to develop brain tumors. We reviewed baseball players, managers, and umpires diagnosed since 1985 with primary central nervous system (CNS) tumors in comparison to the general population. Multiple hypotheses exist for the etiology of tumors and one premise is the introduction of artificial turf (AT) in 1966.

Methods: Review of baseball records reveals that 12 on-field professionals have been diagnosed and died from primary CNS tumors, 9 of which occurred after 1985 with careers that spanned 1961 to 2004. There have been 17,760 major league baseball players from 1870–2012, giving an average number of 1269 players per decade. There are currently 17 four-man umpire crews and 30 managers for an additional 98 individuals on the playing surface daily. Accounting for fluctuations in rosters, managerial staff, umpires, and expansion teams; upwards of 1500 individuals per decade (6000 between 1960 and 2000) played, managed or umpired major league baseball. Inferential statistics were performed using chi-squared test with Yates' continuity correction comparing baseball's population versus the general population of the United States.

Results: According to Central Brain Tumor Registry of the United States, the annual incidence rates of primary CNS tumors range from 7.3–19.9 cases per 100,000 individuals, with a slight male predominance. During the period AT was used, the baseball population had a significantly higher rate of developing CNS tumors than the general population (9/6000 vs. 20/100,000, $P < 0.0001$) with an odds of developing CNS tumor of 7.5 times that of the general population. Further analysis of these individuals reveals similarities all being Caucasian males that were occupationally exposed to AT for at least 2 years.

Discussion: The incidence of primary CNS tumors in baseball players, managers, and umpires is significantly higher than the average US population. Exposure to earlier generation AT could possibly explain this heightened incidence as these players were exposed for at least 2 years. The adhesive substance used to bind AT to the cement was polyvinyl chloride and in monomeric form is associated with carcinogenesis. The correlation with vinyl chloride and hepatic angiosarcoma is established, however the connection with specific brain cancers is debatable. Regardless of the etiology, the number of primary CNS tumors among the baseball community is truly alarming and requires further investigation.

Keywords: Baseball, Brain tumor, Artificial turf

208. Dimethyl sulfate exposure

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Background: Although dimethyl sulfate (DMS) is used/transported in the United States (U.S.), all peer reviewed exposures have occurred outside the U.S. (series of 62 pts in China, 9 in England, 1 in Iran). 1 victim of a multiple victim incident in the US is described.

Case report: ED called the Poison Center (PC) regarding a 36 y/o male presenting 10 hrs post-exposure to DMS. Lines containing DMS were being purged in the room in which he was working; respirators were not in use. The duration of exposure could not be

determined. He had irritated eyes/hoarseness; HR 101, RR 20, O_2 sats 94% on room air, T 36.5ºC, BP 133/77. 4 hrs later, lung sounds described as "not very good"; humidified O_2 40% v-mask started, RR 16. Methylprednisolone (MP) given per PC recommendations. In ICU, reported to be short of breath (SOB) and choking. Facial edema/erythema, nasal congestion, mucous production/post-nasal drainage reported. The uvula/oropharynx were red/edematous associated with dysphagia. CXR showed "lots" of fluid in lungs. Receiving 60 mg MP IV every 6 hrs and albuterol nebs every 4 hrs. pH 7.42, pCO_2 38, pO_2 58, HCO_3 24, O_2 sats 91% with humidified O_2 40% v-mask; unable to tolerate nasal cannula. BUN 15, SCr 1.2 mg/dL.

Day 2, productive cough persists but improved; sputum previously watery, now yellow. Now receiving MP (125 mg IV every 6 hrs), albuterol nebs, enoxaparin and pantoprazole. Hoarseness, sore throat, conjunctivitis and photophobia remained; VS: HR 105, BP 122/72, RR 12–20, O_2 sats 94–98% with humidified O_2 40% v-mask. CXR: right pleural effusion/lower lobe infiltrate; left lung was clear.

Day 3, reported to be better; O_2 sats 97% on RA. Productive cough diminishing. CXR: right lower lobe opacity but overall improvement; started incentive spirometry. Facial edema abated. Denied SOB and tolerating soft diet. MP dose was now 60 mg IV every 8 hrs and being tapered. Eyes "better" but still red. Day 4, VS: HR 66, BP 123/86, RR 14, O_2 sats 94–95% on RA. Sputum culture reported gram (+) cocci /gram (−) rods; started on levofloxacin. MP continued; no formal eye exam. Discharged that night.

Case discussion: Case demonstrated the classic presentation of delayed onset of toxicity and significant respiratory and mucous membrane involvement. Early/aggressive steroid therapy was utilized based on recommendations from the case series in China. Although this 1988 paper recommended the use of prophylactic antibiotics (abx), it was decided to await evidence of infection as part of current abx stewardship practices.

Conclusions: The potential for delayed effects following DMS exposures should not be ignored. Limited numbers of cases limit ability to prospectively evaluate the use of steroids; however, the potential benefits in these cases should be considered.

Keywords: Occupational, Dimethyl sulfate, Steroids

209. Occupational exposure of laboratory workers to drugs of abuse

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Background: The Analytical Laboratory (AL) of the Israeli Police handles investigation files suspected to involve drugs of abuse (DOA). At any given moment there are in the AL tens of kilograms of DOA, including cannabis, heroin, cocaine, amphetamines, methamphetamines and cathinone derivatives. These DOA are notorious for their cardiotoxic, neurotoxic and psychiatric effects. In recent years workers in the AL have complained of health problems such as dizziness, headache, nausea, cough, eye irritation and pruritus; most of these complaints have been reported in the literature after consumption of the above DOA. Limited information exists on the effect of occupational exposure to DOA, especially in anesthesiologists, and law enforcement personnel investigating clandestine laboratories. These studies did not use biological monitoring of DOA.

Objective: To evaluate the extent of exposure of workers in the AL of the Israeli Police to DOA and the health effects of such exposure.

Methods: A prospective open study using health and occupational questionnaires, clinical assessments and monitoring of the urinary excretion of DOA was performed. The study included three blocks of one week each. At the beginning of each week baseline assessments were done, and the same assessments were repeated at the end of three working days of that week. Demographic, clinical, occupational and laboratory data were subjected to descriptive analysis and were compared using paired Student's t-test. The urine samples were analyzed by immunochemical and chromatographic assays in another laboratory whose workers were blinded to the DOA identified by the AL.

Results: Fifty one workers aged 24–53 years old, 72.5% females, were sampled. A significant reduction in diastolic blood pressure was found post-shift compared with the beginning of the working week, 71.2 ± 11.2 mmHg and 77.2 ± 13.6 mmHg, respectively, $p < 0.0001$. Small significant decrease in spirometry parameters were found post-shift compared with baseline values: FEV1 $98.3 \pm 14.6\%$ and $100.7 \pm 12.7\%$ respectively, $p < 0.0001$, FVC $101.4 \pm 13.7\%$ and $103.7 \pm 13.9\%$, respectively, $p = 0.003$, FEF25–75 $85.7 \pm 19.4\%$ and $89.6 \pm 18.7\%$, respectively, $p = 0.006$. No changes were recorded in the ECGs. The main health complaints included headache, fatigue and dry eyes. No DOA were detected in urine samples.

Conclusions: The combination of health effects reported by the AL workers, significant post-shift decrease in diastolic blood pressure and significant minor decrease in spirometry parameters, and the lack of DOA in the workers' urine hint to an environmental problem. Assessment of the environmental protection in the AL (e.g., turnover of air, use of hoods and adequate protective masks) is warranted.

Keywords: Drug of abuse, Occupational, Biomonitoring

210. Significant hydrogen sulfide exposure from a kitchen sink

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Background: Hydrogen sulfide (HS) is a toxic gas leading to lethal outcomes. It is encountered in sewer systems, oil refineries and in the maritime, asphalt, and mining industries. We report the first case of a significant HS exposure due to working on a clogged kitchen sink.

Case report: A 55 year old plumber who was feeling well (no chest pain, shortness of breath, nausea) that day when he suddenly developed loss of consciousness while working on a clogged kitchen sink. It occurred soon after he poured concentrated sulfuric acid in the sink. He was found down by a coworker who smelled a rotten egg odor. The coworker called 911 and he started CPR on the victim till the emergency medical services (EMS) arrived. The fire department confirmed the presence of high concentrations of hydrogen sulfide at the scene. In the emergency department he was

found to have blood pressure of 180/110, tachycardia, O_2 saturation: 100% on room air. He was intubated for airway protection. The patient developed hypotension and was started on pressors. He developed acute kidney injury, mild elevation in liver enzymes and troponin elevation with an EKG with non-specific changes. He underwent a cardiac catheterization which showed coronary artery disease (CAD) but without acute occlusion. He did well and was discharged home.

Discussion: The patient's collapse was likely due to chemical asphyxia due to inhibition of the electron transport chain by the HS. HS exposure is well reported due to working with sewer systems but not from working on kitchen sinks. The scene data clearly reflected HS exposure. It is unclear what else was in the sink that combined with the sulfuric acid to result in the release of the HS.

Another possible etiology for the patient's collapse was a spontaneous cardiac arrhythmia. This is unlikely because the patient did not have cardiac symptoms prior to his collapse. His EKG on presentation did not have changes that were clearly consistent with cardiac ischemia and there were no abnormal rhythms. Also, while he did have CAD on heart catheterization, there were no acute occlusions. The hypoperfusion with the cardiac arrest is the likely cause of the troponinemia and the multiorgan failure.

Conclusions: HS poisoning is a risk for plumbers or anybody else working with sulfuric acid to unclog sinks.

Keywords: Hydrogen sulfide, Kitchen sink, Multiorgan failure

211. Propofol use for pediatric procedural sedation in a general emergency department

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Background: Propofol (Ppf) is an ultra-short non-barbiturate non-opioid sedative with amnestic and anticonvulsant properties. Many tertiary pediatric emergency departments (ED) have not instituted routine use due to its adverse effects of dose-dependent respiratory depression and hypotension. We reviewed Ppf use in the pediatric population requiring deep sedation for painful procedures. To our knowledge, this is the largest retrospective review evaluating the safety and efficacy of this drug in children in a general ED.

Methods: A 4 year retrospective medical record review from 1/1/2007–12/31/2010 in children <18 years was conducted. Patients (pt) were identified via the ED medication Pyxis system. Data collection included: age, race, sex, history, procedure, weight, dosing, vital signs, sedation level and adverse events. Chi-square or Fisher exact test and analysis of variance were used to assess differences among data parameters where appropriate.

Results: 331 pts were identified: 66% were male, 80% Caucasian. 71% underwent fracture reduction and 9% laceration repair. Mean initial bolus administered was 1.54 mg/kg (range 1–7 mg/kg) and mean total dose 239 mg (6 mg/kg) (range 144–860 mg). 96% received no adjunct analgesic medication. 12 pts (4%) received analgesic medication prior to Ppf: 9 opioids, 1 nitrous, 2 ketamine. Recovery times were: mean 18 minutes (range 12–66 minutes). 95% obtained moderate to deep sedation. Adverse events occurred in 16% (52 pts): of these 82% of these required chin lift/jaw thrust; 13% had oxygen desaturation $>5\%$ while on 3 liters/minute of oxygen, 8% apnea, 4% bag-valve-mask ventilation (BVM), 4% hypotension, 1 pt with bradycardia and ectopy. 2 procedures were aborted: 1 allergic reaction and 1 pt with severe agitation with initial bolus. 2 pts required intubation:1 for laryngospasm and 1 for hypoxia and apnea not resolved with BVM. Adverse events by age were: <6 years -15%, 6–13 years -17%, >13 years -10% ($p=0.38$). Adverse events by weight were: $<75\%$ weight for age -15%, $>75\%$ weight for age -16.5% ($p=.11$). Nursing documented adverse events in 16% of pts, physicians in 4%.

Conclusions: Ppf administered to children for painful procedures in a general ED is safe and effective. Only 3% experienced a serious adverse event. Although adverse events were higher in this study population than previously reported by others, this may be reflected in the increased dosing required to achieve deep sedation when analgesics are not co-administered.

Keywords: Pediatric, Propofol, Sedation

212. Five cases of airway angioedema after a Gila monster (*Heloderma suspectum*) bite

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Background: Gila monster (*Heloderma suspectum*) is a venomous lizard found in the southwestern United States and northern Mexico. Airway angioedema has been reported after a Gila monster bite. We report 5 cases that were reported to poison centers in Arizona between 1/1/2000 and 10/31/2011. Case 5 has been previously published.

Case 1 August, 2004

A 35 yo man was bitten while playing with a Gila monster that he found while hiking. As documented by home video, the lizard was removed from the man's finger after 42 seconds by prying its jaw open with a flat rock. Within 5 minutes, the patient became dizzy, vomited and had diarrhea. At the hospital, he was noted to have facial and tongue edema, necessitating intubation. Hypotension was treated with vasopressors. Two days later he was discharged.

Case 2 June, 2008

A 26 yo man placed a Gila monster on his shoulder; it bit him on the neck. The animal dropped to the ground. He picked it up, and placed it in a hat. It bit him again on his finger through the hat. In the ED he was found to have edema on both sides of his tongue; he had no trouble swallowing or breathing. He was admitted for observation. The following day the edema resolved and he was discharged.

Case 3 January, 2009

A 29 yo man was bitten on the arm. He developed a sensation of tongue swelling. He was transported to an ED where tongue edema was noted. After several hours of observation, he was discharged.

Case 4 May, 2009

A 49 yo man found a Gila monster at a lake. He placed the animal inside his shirt; it bit him on left upper quadrant of his abdomen. It let go immediately. Within 30 minutes he developed respiratory problems. Upon arrival to the ED he lost consciousness. Severe edema was noted of the tongue and glottis. The airway was secured by cricothyrotomy. He was discharged 5 days later.

Case 5 July, 2011
A 29 yo man was bitten on the forearm by a Gila monster that he found at a dry riverbed. The lizard made chewing movements during the several minutes that it was attached. The patient presented to an ED within an hour and later was transferred to the medical toxicology service at another hospital. About 13 hours after the bite he described a sore throat. Angioedema developed at the lower lip and uvula. The patient was discharged on hospital day 4.
Discussion: Gila monster venom contains kallikrein-like toxins that convert kininogens to bradykinin. Activation of bradykinin receptors produces vasodilation, vascular leak, inflammation, pain, and angioedema.
Conclusions: Airway angioedema, a potentially life threatening consequence of Gila monster envenomation, was noted upon presentation in most cases. In one, it developed over several hours. Angioedema was seen with brief as well as prolonged bites.
Keywords: Envenomation, *Heloderma suspectum*, Angioedema

213. Tapentadol vs tramadol: Comparative toxicity utilizing data reported to the National Poison Data System

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Background: Tapentadol (TAP) (Nucynta®) is a C-II medication approved for the treatment of moderate to severe pain in patients >18 years old. Immediate release TAP became available in June 2009 and extended release TAP was approved in August 2011. TAP is orally active, with mu-opioid receptor agonism and norepinephrine reuptake inhibition, in addition to weak serotonin reuptake inhibition. The only similar FDA approved medication indicated to treat moderate to moderately severe pain is tramadol (TRA), marketed since 1995. Because of their similar mechanisms of action, the goal of this study was to determine if there were differences in the reported toxicity of TAP and TRA.
Methods: A retrospective cohort study was conducted analyzing data from the National Poison Data System for single agent (TAP or TRA) cases reported to the American Association of Poison Control Centers (AAPCC) from June 2009 through December 2011. Excluded cases included those with multiple ingestions, non-ingestion route, outcomes that were unrelated, confirmed non-exposure, and no follow-up. Case outcomes as defined by AAPCC criteria, as well as the presence of specific symptoms were compared. Odds ratios and 95% confidence intervals were calculated between TAP and TRA for non-significant and clinically significant (moderate/major/death) adverse outcome severity and for common symptoms.
Results: There were 298 TAP and 11,699 TRA cases that met inclusion criteria. The majority of patients were female, 61.1% for TAP and 57.5% for TRA. Median age was 36 years old (0.75–92) for TAP, and 25 years old (6 days to 98) for TRA. The odds of having a clinically significant adverse outcome was significantly higher for TAP (OR = 1.317, 95% CI = 1.016–1.706). In the TAP patients, there were significantly greater odds of developing confusion, slurred speech, sedation, hallucinations, coma and respiratory depression. TRA patients had significantly greater odds of having seizures (OR = 8.879, 95% CI = 3.305–23.86).
Conclusions: In this study TAP patients had greater odds of having clinically significant outcome severity which included coma and respiratory depression, than TRA patients. This likely relates to the parent drug, TAP, being orally active. In contrast, TRA possesses weak affinity for the mu-opioid receptor. For its opioid effect TRA is bioactivated to a more potent metabolite, O-desmethyl-tramadol, which has much greater mu-opioid receptor affinity. The increased seizure liability of TRA deserves further research. The limitations of the study include the retrospective nature of the study, reporting bias, small relative number of TAP cases, and inability to confirm exposures with drug levels.
Keywords: Tapentadol, Tramadol, National Poison Data System

214. An 11 year review of oral Tilmicosin exposures using National Poison Data System (NPDS) data

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Background: Tilmicosin (TMC) is a veterinary macrolide antibiotic and its toxicity in humans is primarily cardiac, mediated via a decrease in inotropy. In parenteral human exposures, the case fatality is 10 times greater than the average mortality from all other exposures in the AAPCC database. However, effects after small oral exposures are not well described. A review of data from the NPDS is presented to augment current knowledge regarding outcomes and adverse effects following small oral exposures to TMC.
Methods: The NPDS database was searched from January 2000 to December 2010 for all human exposures to TMC. Single-substance oral exposures of taste amount were included for analysis; patient age, exposure reason, clinical effects, medical outcome, and therapy employed were abstracted. Confirmed non-exposures were excluded.
Results: A total of 247 cases satisfied inclusion criteria; there were 36 pediatric exposures (11 months–18 years) and 211 adult exposures. All pediatric exposures were unintentional. Reason for exposure in adults was coded as unintentional (209), intentional

Table 1. Data for abstract 214.

	Unknown if related	Related
Chest pain (incl. noncardiac)	3	0
Cough/choke	0	1
Dizziness/vertigo	3	3
Drowsiness/lethargy	0	1
Dyspnea	1	0
Fever/hyperthermia	1	0
Headache	5	0
Hypertension	2	0
Hypothermia	1	0
Nausea	3	5
Oral irritation	1	10
Other	17	11
Rash	2	0
Syncope	1	0
Tachycardia	2	2
Vomiting	3	1

misuse (1), and adverse drug reaction (1). 52% (129) were managed on site and 42% (104) were either referred to or already in a healthcare facility when the poison center (PC) was contacted. Medical outcome was coded as no effect (138), minor effect (47), moderate effect (4), and unrelated effect (17). Thirty-six cases were not followed, and 5 cases were considered potentially toxic but follow up was not possible due to lack of patient/care giver/hospital cooperation. No moderate effects were reported in children; the 2 minor effects in this population were oral irritation and vomiting. In adults, documented effects are summarized in Table 1. Of note, no hypotension, bradycardia, dysrrhythimas, or ECG changes were documented. No patients received GI decontamination or antidysrhythmics. Dilution/irrigation/wash was performed in 115 exposures, food/snack administered in 2, and IV fluids and oxygen administered in 1.

Conclusions: This review of 247 cases suggests that small oral exposures, unlike parenteral exposures, are not associated with serious toxicity. Limitations include lack of detailed information (no access to actual PC notes), number of cases lost to follow-up, and a small sample size. These data suggest asymptomatic patients exposed to small, unintentional taste amounts of TMC may be managed at home.

Keywords: Tilmicosin, Ingestion, Toxicity

215. Development of a droperidol application guideline: Influence of a policy on continuous droperidol usage

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Objective: Droperidol is an effective agent against headache and refractory vomiting, but also promotes QT prolongation. The black box warning of QT prolongation has led to the discontinuation of droperidol use in many hospitals. This report describes a successful collaboration between the Departments of Medical Toxicology, Emergency Medicine, Pharmacy and a hospital regulatory agency that created a policy to ensure continuous availability of droperidol for patients at our institution.

Methods: In 2008, a working committee was formed at our institution to address the consideration of retaining droperidol on our hospital formulary. Many medical centers in our state had withdrawn droperidol from their formularies under regulatory and hospital administration scrutiny at that time. Our committee conducted an intensive review of medical literature surrounding the use of droperidol, and produced a guideline addressing safe and effective droperidol use in preparation for regulatory evaluation. Within the guidelines, IV droperidol less than 2.5 mg is considered safe and requires no cardiovascular monitoring in patients less than 65 years of age. When used in higher doses, ECG monitoring for QT prolongation is required, but allows an ECG to be performed as soon as possible after droperidol administration for severely agitated patients where patient or staff welfare is at risk. Strict monitoring of droperidol use was instituted to insure guideline compliance. Cases not meeting compliance were identified and individual counseling and feedback provided to prescribers.

Results: From Jul 2010 to Dec 2011 droperidol was used 437 times at our medical center. 96.6% of droperidol was used in the ED. The average age was 40 years (range 13–89 years). 411 of providers (94%) were compliance with dosing guidelines. 26 patients (6%) received >2.5 mg IV droperidol. The indications were agitation-15, vomiting-6, and HA-5. 8 of the 26 patients had documented ECGs pre and post droperidol administration. The difference between pre and post QT durations ranged -89 to 21 ms. Post droperidol only QT duration was reported in 20 of the 26 patients, of which 505 ms was the longest. No dysrhythmias were reported. Case discussion: This illustrates the proactive role that pharmacists and physicians can take in the event a medication with adverse effects faces potential restriction. By guiding the safe and efficacious use of droperidol, its availability has been preserved at our medical center.

Conclusions: Collaboration between the toxicology physicians and the hospital regulatory agency ensure continuous availability of droperidol in the ED. Compliance with low dose droperidol administration is safe.

Keywords: Development of application guideline, Collaboration with regulatory agency, Droperidol

216. Multi-system pediatric cough/cold product surveillance methodology: Evaluation of data sources

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Background: While real-time data surveillance can be used to monitor drug safety trends after media, industry and regulatory events, quality data sources are challenging due to the uniqueness and limitations of each source. Data from an ongoing multi-system pediatric cough/cold (CC) medication safety surveillance project was used to evaluate the quality of source data that enables adverse event (AE) causality assessment and evaluation of trends over time.

Methods: Between 1Q08-4Q11 cases were obtained from 5 data sources: English language medical literature, NPDS, FDA/AERS data, manufacturer safety records, and media reports. Case inclusion criteria: age <12 y, exposure to ≥ 1 CC ingredient, ≥ 1 AE, occurred in US. The Pediatric CC Medication Safety Surveillance Team reviewed each case to reach consensus on the relationship between the drug(s) involved in the exposure to the AEs reported. Evaluable cases contained history of ingestion, drug concentration levels, and clinical course consistent with exposure as well as case notes indicating other possible contributing factors. Non-evaluable cases lacked adequate detail regarding these characteristics to reach panel consensus. For purposes of analysis, the data was limited to cases detected from only 1 source.

Results: Of 3046 single-source cases reviewed by the Team, 2677 (87.9%) were evaluable; 369 (12.1%) were non-evaluable. NPDS contributed the largest proportion of cases (80.5%), of which only 8.8% were non-evaluable. Media reports contributed the smallest proportion of cases (1.0%), with 56.5% non-evaluable. 99.7% of detected NPDS cases occurred during the surveillance period (1Q08-4Q11), while event date was either not reported or occurred prior to 2008 in all medical literature cases and 60.8% of media reports.

Table 1. Results for abstract 216.

	AERS N (%)	Manufacturer safety records N (%)	Medical literature N (%)	NPDS N (%)	Media N (%)	Total
Causality						
Evaluable	75 (69.4)	313 (75.9)	42 (84.0)	2237 (91.2)	10 (43.5)	2677
Non-Evaluable	33 (30.7)	99 (24.0)	8 (16.0)	216 (8.8)	13 (56.5)	369
Event Date						
1Q08-4Q11	79 (73.1)	209 (50.7)	0 (0.0)	2446 (99.7)	9 (39.1)	2743
Pre-2008	8 (7.4)	24 (5.8)	0 (0.0)	1 (0.0)	9 (39.1)	42
Not Reported	21 (19.4)	179 (43.4)	50 (100.0)	6 (0.2)	5 (21.7)	261
Total	108	412	50	2453	23	3046

Conclusions: Cases occurring during the detection period contributing sufficient information to evaluate causality are important for surveillance because they allow for accurate analysis of the impact of media or regulatory activities. While the utilization of multiple data sources is recommended, this study illustrates the relative quality and timeliness of data sources often used to make regulatory and clinical determinations regarding drug safety.

Keywords: Data sources, Quality, Research methodology

217. A 48 month retrospective review of pregabalin ingestions in adults

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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 regarding the toxicity in ingestions of pregabalin.

Method: A 48 month retrospective review of ingestions of pregabalin in adults reported to a regional poison center during the period of January 1, 2009 to December 31, 2011was performed. IRB approval was obtained and cases were blinded prior to analysis. Inclusion criteria were pregabalin as a single ingestant and cases followed to a known outcome. Data collected were age, sex, amount ingested, clinical symptoms, and patient outcome.

Results: A total of 147 cases of pregabalin ingestions without coingestants were identified. 48 patients (33%) were male, and 99 patients (67%) were female with a mean age of 54 years old (range 22–99 years old, SD 21.7 yrs). The mean amount ingested was 762 mg (range 25–9000 mg, SD 1366 mg. Of the 147 patients, 35 patients (24%) developed drowsiness, 14 patients (9.5%) developed dizziness, 10 patients (6.8%) developed tremors, 3 patients (2%) developed nausea/vomiting, 6 patients (4%) developed other minor side effects such as slurred speech and agitation, headache, rash, dyspnea, erythema and agitation, and mild confusion. 1 patient (0.7%) developed respiratory depression at a dose of 4500 mg. 25 patients (17%) were treated in the ED. Activated charcoal was administered to 12 (8%) of the 147 patients. All 25 patients (100%) in the ED were discharged without sequelae. Outcome: No effect in 98 patients (66.7%), minor effects in 38 patients (25.8%) drowsiness, dizziness, rash, and nausea/vomiting, moderate effect in 10 patients (6.8%) tremors, and a major effect in 1 patient (0.7%) that was reported as respiratory depression.

Conclusions: Based upon the results of this retrospective study, pregabalin toxicities usually manifest as minor effects. Moderate and major effects such as tremors and respiratory depression were also observed in doses exceeding the recommended daily dose. The results did not show a clear correlation between doses and toxicity. Pregabalin ingestions showed favorable outcomes with supportive care and gastric decontamination with activated charcoal in the ED. Continued evaluation of adult ingestions of pregabalin is essential to determine more specific thresholds for toxicity.

Keywords: Anticonvulsant, Ingestion, Poison center

218. Clinical findings of patients exposed to spice and bath salts with laboratory confirmation

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Background: Little data is known on the clinical course of patients exposed to spice and bath salts. Much of the known data is not supported by laboratory confirmation. Our hospital has had several cases of young adult patients exposed to these agents. In this retrospective chart review, we sought to quantify the signs and symptoms of those patients who have tested positive for the parent compound or metabolite of either spice or bath salts.

Methods: We retrospectively reviewed the electronic charts of patients who presented to our hospital from January 2008 to November 2011 and were found to be positive for the metabolites of the spice products JWH – 018 and JWH – 073 and the bath salts product MDPV. Our study was approved as exempt status by

Table 1. Clinical findings of patients exposed to 'spice' and 'bath salts'.

Sign/Symptom	Spice, n = 21	Bath salts, n = 6	Both, n = 4
Hypertension	14 (67%)	5 (83%)	4 (100%)
Somnolence	14 (67%)	4 (66%)	3 (75%)
Tachycardia	13 (62%)	5 (83%)	3 (75%)
Leukocytosis	10 (48%)	5 (83%)	3 (75%)
Elevated CK	6 (29%)	3 (50%)	2 (50%)
Acidosis	5 (24%)	2 (33%)	2 (50%)
Positive for other agents on urine/serum drug testing	11 (52%)	5 (83%)	1 (25%)

the Institutional Review Board. Variables collected and analyzed included age, sex, symptoms, laboratory data, and treatments.
Results: Of 25 cases reviewed, two cases were excluded for incomplete records. One patient presented twice within 24 hours and is included as a single case. The mean age was 24 years (range 20–29 years), 88% of patients were males. Of the included cases, 21 tested positive for spice, six for bath salts, and four were positive for both. In 13 cases (57%) the patient or a companion at the bedside admitted to use of one or both compounds. Treatment primarily consisted of intravenous (IV) fluids (43%) and benzodiazepines (43%) with rare cases requiring other treatments. Fourteen cases (61%) required hospital admission, including five patients admitted to the intensive care unit. Most patients had effects last less than eight hours.
Conclusions: Based on this retrospective review, patients exposed to spice often present with HTN, somnolence, and tachycardia. Those patients with bath salts exposure may have HTN, tachycardia, leukocytosis, and are more likely to test positive for coingestions. HTN occurred in all four cases exposed to both agents, and tachycardia, leukocytosis, and somnolence were documented in three of the four. Based on these findings treatment suggestions after ensuring no other causes might include benzodiazepines, IV fluids, and anti-emetics.
Keywords: Bath salt, Cannabinoid, Synthetic, Drug of abuse

219. Correlation between tachycardia and seizures in bupropion exposures

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Objectives: While creating a poison center guideline for managing bupropion exposures, a relationship between tachycardia and seizures was noted. NPDS data was searched to determine if there is a correlation between tachycardia and seizures in bupropion exposures.
Methods: AAPCC's NPDS data for July 1, 2004, through June 30, 2011, was searched for all human exposures to bupropion. All ages were included. The exposures included both single-medication and multiple-medication exposures. Each case's clinical effects were searched to determine (a) whether "tachycardia" was present and (b) whether "seizure (single)" or "seizure (multiple/discrete)" or "seizure (status)" was present. Every bupropion exposure fell into one of four categories: (1) YES tachycardia & YES seizure, (2) YES tachycardia & NO seizure, (3) NO tachycardia & YES seizure, (4) NO tachycardia & NO seizure.
Results: 24,693 bupropion exposures were identified. 4002 patients had at least one seizure, and 20,691 did not have any seizures.

Statistical analysis of the NPDS bupropion exposure data revealed the following. For those with bupropion exposure and tachycardia, the positive predictive value for having a seizure in this series was 98.655%. For those without tachycardia, the negative predictive value was 99.797%. The sensitivity of tachycardia as a predictor of seizures was 98.951%, and its specificity was 99.739%. The relative risk of having a seizure if the patient was tachycardic was 485.7.
Conclusions: As bupropion is a cathinone derivative, it is the indirect sympathomimetic effects that cause both the tachycardia and the subsequent seizures. The association between tachycardia and seizures in bupropion exposures in this data was very strong, and it is possible that tachycardia may be a harbinger of impending seizures There is no implication of causality in this data. Further research is needed to evaluate the possible clinical utility of this observed association.

The AAPCC maintains the national database of information logged by the country's 57 poison centers. Case records in this database are from self-reported calls: they reflect only information provided when the public or healthcare professionals report an actual or potential exposure to a substance, or request information/educational materials. Exposures do not necessarily represent a poisoning or overdose. The AAPCC is not able to completely verify the accuracy of every report made to member centers. Additional exposures may go unreported to PCs and data referenced from the AAPCC should not be construed to represent the complete incidence of national exposures to any substance(s).
Keywords: Bupropion, Seizure, Tachycardia

Table 1. Results for abstract 219.

	Tachycardia present	Tachycardia absent
Seizure present	3960	42
Seizure absent	54	20637

220. Hypoglycemia: Substances to consider in differential diagnosis

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Objectives: Hypoglycemia often occurs after exposure to insulin and oral diabetes medications, but other substances may also cause hypoglycemia. The objective of this study was to characterize exposures reported to US poison centers where hypoglycemia is coded as a related clinical effect.
Methods: Data from the American Association of Poison Control Center's National Poison Data System (NPDS) were obtained for exposures between January 1, 2000 and December 31, 2010 in which hypoglycemia was coded as related to the exposure. Cases with more than one substance were excluded.
Results: A total of 12794 single substance exposures with hypoglycemia coded as related were reported to NPDS. The ten most frequent causes of hypoglycemia were identified (see Table 1).

Table 1. Results for abstract 220.

		Total: 12794	
Rank	Substance category	#	%
1	Insulins	5051	39.5%
2	Sulfonylureas	4452	34.8%
3	Miscellaneous/unknown drugs	429	3.4%
4	APAP alone or in combination (including opiates)	416	3.3%
5	Biguanides	373	2.9%
6	Ethanol containing products	333	2.6%
7	Other/unknown oral hypoglycemics	324	2.5%
8	Thiazolidinediones	143	1.1%
9	Opiates alone or in combination (excluding APAP)	116	0.9%
10	Beta blockers	101	0.8%

Six of the most frequent causes of hypoglycemia are medications used to treat diabetes or miscellaneous/unknown drugs. For the remaining four substance categories of APAP, ethanol, opiates and beta blockers, we report the most frequently associated clinical effects in addition to hypoglycemia. For APAP exposures related clinical effects were elevated liver enzymes >1000 (n = 304, 73%), prolonged prothrombin time (n = 267, 64%) and acidosis (n = 251, 60%). For ethanol containing products related clinical effects included drowsiness (n = 151, 45%), acidosis (n = 91, 27%), or increased anion gap (n = 56, 17%). For opiate exposures, common related clinical effects were coma (n = 65, 56%), respiratory depression (n = 48, 41%) and hypotension (n = 45, 39%). For beta blocker exposures, related clinical effects were bradycardia (n = 50, 50%), hypotension (n = 40, 40%) and drowsiness (n = 26, 26%).
Conclusions: Exposures resulting in hypoglycemia varied and mostly resulted from diabetes medications. However, APAP, ethanol, opiates and beta blockers were also associated with hypoglycemia in addition to other related clinical effects.
Keywords: Hypoglycemic, Medical toxicology, Differential diagnosis

221. Use of ultrasound in identification and management of a first-trimester pregnant body packer

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Background: "Body packers" transport illicit drugs across international borders through internal concealment. Drug packets are generally detected using plain film radiography or computed tomography (CT). We report a case of a 27 year old pregnant, bodypacker whose drugs were detected using abdominal ultrasound.
Case report: A 27 year old woman was brought to the emergency department by US Customs Agents shortly after arriving from the Dominican Republic. At customs, she admitted to having ingested roughly 75 shotgun pellets packed with cocaine prior to her flight. She was asymptomatic at the time of presentation with normal vitals signs and a benign physical exam. Routine screening revealed that she was pregnant. In an attempt to quantify total drug burden without unnecessary fetal irradiation, as well as to verify that the patient had an intrauterine pregnancy, a complete abdominal ultrasound was performed.

Ultrasound showed numerous areas of acoustic shadowing within the bowel consistent with the stated history of ingested pellets. Urine drug screen was positive for cocaine metabolites. The patient was admitted to the medical service for five days and underwent whole bowel irrigation resulting in spontaneous passage of 78 intact pellets. The patient was asymptomatic throughout her stay and was subsequently discharged to police custody.
Case discussion: Radiographic detection of ingested packets is often made using plain film radiography (the gold standard) or CT. Recent advances in drug packaging has made xray detection increasingly difficult. Although contrast CT provides a higher sensitivity for the detection of drug packets, it has failed to detect known ingestions in the past. In addition, there are increasing reports of both children and pregnant women serving as drug transporters. In these patient populations, judicious use of radiographic imaging is required to minimize radiation exposure.

Abdominal ultrasound is a noninvasive, rapid and inexpensive method of assessing patient anatomy without ionizing radiation. The presence of drug packets appears as hyper-echoic structures with characteristic acoustic shadowing. The positive predictive value of ultrasonography for the detection of ingested drug packets has been reported as comparable to that of plain radiography and as high as at 97.6%. Abdominal ultrasound has also been used to confirm equivocal plain film findings for drug packets.
Conclusions: With continued concerns regarding excessive radiation due to repeat CT imaging particularly in at-risk patient populations, we propose the use of abdominal ultrasonography as an alternative diagnostic tool for drug packers presenting to the emergency room.
Keywords: Foreign body, Cocaine, Ingestion

222. Middle cerebral artery stroke associated with use of synthetic cannabinoid K2

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Background: There has been increased recreational misuse of synthetic cannabinoids in the United States over the past several years. In 2011 the American Association of Poison Control Centers (AAPCC) received 6959 number of calls regarding synthetic cannabinoids in comparison to 2906 in 2010. While there have been case reports of cannabis induced cerebrovascular accident (CVA), there have been no reports, to date, due to synthetic cannabinoids. We report a case of a synthetic cannabinoid induced CVA.
Case report: A 22 year-old woman with a history of bipolar disorder and ADHD was transferred to our institution for an evaluation of CVA. She had smoked K2 for the first time earlier that day and then suffered an apparent anxiety attack. She fell asleep and then awoke with left sided weakness and slurred speech. At the transferring institution, she had a normal noncontrast head CT followed by a MRI that demonstrated a large right sided stroke. Her standard urine drug of abuse screen was negative.

The patient was then transferred to our institution, where a CT angiogram of the head was performed and demonstrated an acute infarct of the right middle cerebral artery. The next day, a repeat head CT demonstrated a left parafalcine herniation. She was then started on hyperosmolar therapy with mannitol; successive imaging demonstrated small amounts of hemorrhagic transformation. A hypercoagulability workup failed to demonstrate a clotting predisposition.

Throughout the course of her hospitalization, the patient's mental status improved; however, she remained hemiplegic and was subsequently discharged to a rehabilitation facility.
Case discussion: K2 is a synthetic cannabinoid which has grown in popularity after initially having escaped attention and regulation. In March of 2011, citing increasing health concerns, the DEA exercised its emergency scheduling authority, banning the sale and possession of these substances; this ban was recently extended for another 6 months. However, despite the DEA's proactive stance, K2 remains easy to obtain and abuse.

Chest pain and myocardial infarctions have been seen with both cannabis as well as synthetic cannabinoids; the postulated

mechanism is secondary to vasospasm as these occur in young, otherwise healthy, individuals with a negative atherosclerosis work up. Synthetic cannibinoids has been reported to cause seizures and episodes of paranoia; however, this is the first reported case of a CVA secondary to K2 use.
Conclusions: Despite recognition of the danger of these substances, they remain readily available. We recommend the consideration of synthetic cannabinoids in the differential diagnosis for CVA in the young patient.
Keywords: Cannabinoid, Synthetic, Stroke, Substance abuse

223. Medical toxicology referrals in the acute care setting

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Background: Medical Toxicologists (MT) evaluate patients in several different acute care settings. The types of providers whom request such services have not been defined. Knowledge of who requests our services, and why, may enable MT to better market themselves. The Toxicology Investigators Consortium (ToxIC) is a registry that was created 2 yrs ago by the American College of Medical Toxicology. De-identified, descriptive data obtained by MT during bedside encounters or office evaluations is entered into the database. We queried the database to better understand the source of toxicology referrals.
Methods: The ToxIC database was queried according to the source of referral using the advanced search option. The entire database was searched to obtain the total number of encounters in the registry. To determine the total number of referrals from each source of referral, multiple searches of the entire database were done with each source of referral as the main search term. Patients seen in the outpatient setting were subtracted from those numbers so only patients seen in the acute care setting were included. The referral sources included the emergency department (ED), other hospital services (OHS), self-referral, outside transfer, poison center (PC), and primary care provider (PCP). The sources of referral were stratified according to the type of encounter. Data was compared to similar data available from the registry one year ago.
Results: The registry included 12,511 patient encounters. Of the 12,511 encounters, fifty-seven percent were from the ED and 18% from OHS. Compared to 1 year ago, fewer referrals were from the ED and more were from OHS when the percentages were 69% and 13%, respectively. Transfers were responsible for 14% of referrals, constant from a year ago. Few referrals were from the PC (5%) or a PCP (3%).

Nearly 50% of referrals from the ED were for intentional overdoses. Adverse drug events and reactions represented $<1\%$ of ED referrals. There were 825 ED referrals (6.6%) for patients in withdrawal.
Discussion: The ED is the source of referral for most acute care consultations. MT are infrequently consulted by other services. The number of referrals initiated by the ED may be misleading as ED physicians might order the consultation at the request of the inpatient service. However, this may reflect that MT need to better market themselves to other providers. Most ED consultations were for management of drug ingestions. Only a few were for patients actively withdrawing or for adverse drug events or reactions. This could be evidence that MT can improve their marketing.
Conclusions: The ED is the source of referral for most acute care toxicology consultations. MT may want to improve marketing of their services to hospital providers.
Keywords: Medical toxicologists, ToxIC, Consultations

224. Propofol for procedural sedation in obese children in the emergency department

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Background: Propofol (Ppf), a short-acting non-narcotic non-barbiturate sedative with amnestic properties is used for procedural sedation in the emergency department (ED). Typically, dosing is weight-based with titration starting at 1–2 mg/kg followed by 0.5 mg/kg infusions as needed to achieve or maintain sedation. Limited data exist examining Ppf use and adverse effects in the obese pediatric population. We describe the safety and efficacy of Ppf in obese children requiring moderate to deep sedation for painful procedures in a general ED.
Methods: A 4 year retrospective medical record review was conducted from 1/1/2007–12/31/2010 in obese children <18 years of age who received Ppf for procedural sedation in the ED. Patients (pt) were identified via the ED medication Pyxis system. Data obtained included age, weight, vital signs, medication dosing, sedation level, and adverse events. Using guidelines from the Centers for Disease Control of pediatric growth charts for weight by age (WBA), pts were grouped into 5 levels: Group 1: $<25\%$ WBA, Group 2: 26–50% WBA, Group 3: 51–75% WBA, Group 4: 76–95% WBA and Group 5: $>95\%$ WBA. Obesity was defined as weight $>95\%$ (Group 5). To assess differences in complications and Ppf dosing by weight group, chi-square tests and analysis of covariance adjusted for age was used.
Results: 331 pts charts were reviewed. 66% were male and 80% Caucasian. 92 pts (28%) were in Group 5. The majority of patients underwent fracture reduction (71%) followed by laceration repair (9%). 96% of all pts received no adjunct analgesic agents. Initial mean bolus of Ppf administered to Group 5 was 1.4 mg/kg, with a mean total dose of 5.5 mg/kg compared with 1.6 mg/kg induction bolus and total dose of 6 mg/kg for pts in Groups 1–3 ($p>.05$ for both). Adequate sedation was achieved in 95% of all pts. Mean recovery and total procedure time in Group 5 was 16 minutes (range 2–66) and 42 minutes (range 5–130) respectively, compared to 23 and 46 minutes in Group 1. In Group 5, adverse events occurred in 17% of pts. Of these 80% were minor, requiring chin lift/jaw thrusts. Additionally, 27% had decreased oxygen saturations $>5\%$ while receiving oxygen at 3 Liters/minute. One pt required intubation for hypoxia and apnea not resolving with assisted ventilation. Adverse events for pts in Groups 1–3 were 15% vs pts in Groups 4–5 of 16.5% ($p=.11$).
Conclusions: Weight-based Ppf is a safe and effective medication for obese children requiring sedation for painful procedures in the ED. No increase in complications or dose of Ppf by weight was found in this study population.
Keywords: Pediatric, Obese, Propofol

225. Serotonin toxicity: Case characteristics and associated medications/drugs

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Background: Serotonin toxicity is a common cause of drug-induced altered mental status in hospitals nationwide. Unfortunately, the initial description of serotonin toxicity was non-specific and may have led to misdiagnoses in early reports. It remains unclear which patient characteristics are associated with serotonin toxicity and which medications are responsible. In the past several years, a more specific definition has emerged and a national database of prospectively collected data on patients who are diagnosed by a bedside medical toxicologist has become available. We sought to determine the characteristics of patients who develop serotonin toxicity as well as the agents that are associated with its development.
Methods: We accessed the ToxIC registry database and searched for "serotonin syndrome" in the "Syndromes, symptoms and signs" section for the 2 y period between 1/1/2010–1/1/2012. ToxIC is a registry of cases that are seen and diagnosed by medical toxicologists throughout the country. Data is entered prospectively by over 30 geographically-diverse toxicology services.
Results: Our database search identified 209 cases of serotonin syndrome diagnosed by medical toxicologists. Patients were predominantly adults(1% <6 yo; 1% 7–12 yo; 20% 13–18 yo; 76% 19–65 yo; 3% >65 yo) and were mostly female (60%). 56% were seen in an ED, 44% were seen in a hospital ICU or ward (total of 176 reported location). 68% of cases were reported after an intentional overdose, 7% after unintentional overdoses, 15% were adverse effects, and 24% were drug abuse. Of those cases reporting symptoms/effects (200/209), 60% reported hyperreflexia/myoclonus/clonus, 45% delirium, 41% agitation, 40% tachycardia, 39% hypertension, 18% mydriasis, 14% hyperthermia, 19% developed rhabdomyolysis, 16% required intubation and 3% had ventricular dysrhythmias. Most cases were treated with benzodiazepines (52%) and 11% were treated with cyproheptadine. There were 51 cases where a single drug was noted. The drugs noted in single-drug cases of serotonin toxicity are listed in Table 1.
Conclusions: We describe the clinical characteristics of, and the drugs associated with, serotonin toxicity as described by medical toxicologists nationwide over a 2 y period.
Keywords: Serotonin syndrome, Epidemiology, Medical toxicology

Table 1. Medications noted in single-drug cases of serotonin syndrome (# mentions/51 cases).

Citalopram (9)
Dextromethorphan (7)
Bupropion (4)
Fentanyl/sufentanil (4)
Paroxetine (2)
Sertraline (2)
Tramadol (2)
Valproate (2)
Venlafaxine (2)
Medphedrone (2)
1 each of:
Adderall
Clomipramine
Cocaine
Diphenhydramine
Duloxetine
Fluoxetine
Lithium
MDMA
MDPV (bath salts)
Methamphetamine
Mirtazapine
Nortriptyline
Quetiapine

226. Ketamine: An alternative agent for the management of agitated delirium

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Background: Ketamine is well known for its use in procedural sedation and general anesthesia. As a noncompetitive N-methyl-D-aspartic acid (NMDA) antagonist, ketamine is an effective agent for achieving a dissociative analgesic state. While ketamine has been reported to control agitation in traumatic injury, it has not yet been reported for the management of an agitated delirium due to poisoning. We report a case of ketamine administration for sedation of a toxicologic-induced agitation following unsuccessful management with intravenous benzodiazepines.
Case report: A 15-year-old male presented to the emergency department in an agitated state with complaints of visual hallucinations. He reportedly ingested four tabs of "acid" two hours prior to arrival. Vital signs were normal except for a sinus tachycardia of 107 beats per minute. Laboratory studies were unremarkable except for a positive cannabinoids test on urine drug screening. Intravenous (IV) benzodiazepines (BZDs) were administered as a first-line agent to control agitation, but were ineffective. The patient required physical restraints for control of agitation, despite receiving 24 mg of lorazepam, 80 mg of diazepam, and 8 mg of midazolam over eight hours. Adequate sedation was achieved only after administration of IV ketamine (1 mg/kg). Chemical sedation allowed for a complete physical evaluation, diagnostic testing, and observation.
Discussion: Lysergic Acid Diethylamide (LSD) or "acid" is a serotonin agonist that has been associated with anxiety, paranoia, visual hallucinations and agitation. Uncontrolled agitation is a dangerous clinical situation that places patients and medical providers at risk for serious harm. Patients may develop rhabdomyolysis, metabolic derangements or sustain physical injuries. Gamma-aminobutyric acid (GABA) agonists, like BZDs, are appropriate first-line sedative agents for agitated patients regardless of the ingested toxic agent. Alternative agents must be considered when there are shortages of IV BZDs or when BZDs fail to achieve adequate sedation. Using alternative agents that are also sedating may result in respiratory depression necessitating endotracheal intubation. Ketamine, an NMDA antagonist, preserves airway reflexes obviating the need for endotracheal intubation. Like BZDs, intravenous and intramuscular ketamine has rapid onset to peak effect and is titratable.
Conclusions: Ketamine may be considered as an alternative agent for sedation of the toxicologic-induced agitated patient.
Keywords: Sedation, Drug of abuse, Ketamine

227. Drug-induced seizures in children presenting to the emergency department

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Background: Seizures are a worrisome, though not uncommon in children presenting to the emergency department (ED). Seizures may be drug-induced, and can be associated with exposure to a wide range of medications and non-pharmaceutical agents. This etiology should be considered in any new-onset seizure in a child, especially in an afebrile child. Many drugs are classified as having the potential to provoke seizures, however, it is unknown which drugs are commonly responsible for this acute pediatric presentation in real-life context. Using the Toxicology Investigators Consortium (ToxIC) Case Registry as a toxico-surveillance tool, we aimed to describe common agents responsible for pediatric drug-induced seizures in the United States.

Methods: In 2010, the American College of Medical Toxicology (ACMT) established a nationwide database, the Toxicology Investigators Consortium (ToxIC) Case Registry, exclusively cataloging all cases in which bedside consultation by a medical toxicologist was provided in any of the 31 American registry sites, in a prospective manner. Using the ToxIC registry database, we identified all children consulted by a medical toxicologist for drug-induced seizures over a two-year period between April 1, 2010 and March 31, 2012. Demographic and clinical parameters were collected and analyzed.

Results: We identified 143 drug-induced seizure cases reported to the ToxIC registry in patients 18 years or younger (57% male). The majority of cases (75%) occurred in children aged 13–18 years, 13% were in children less than 2 years, and 12% in those between 2–12 years. Forty-three percent were consulted while in the Emergency Department. The most common drug classes involved in seizure activity were antidepressants (42%), anticholinergic/antihistamines (22%) and non-opioid analgesics (15%). Thirty-nine percent of cases involved ingestion of multiple agents. The majority of seizure episodes (84; 59%) resulted from intentional overdoses, all of which occurred within the 13 to 18 year age group (i.e., 79% intentional in this group). Of the intentional overdoses leading to seizures in children, 61% involved antidepressants.

Conclusions: Antidepressants are the most common drug class leading to pediatric drug-induced seizures in the United States, more so in intentional overdose. In the context of adolescents presenting with new-onset seizures of unknown etiology, the possibility of deliberate self-poisoning should be explored, since the majority of drug-induced seizures in this age group are intentional.

Keywords: Pediatric, Seizure, Adverse drug event

228. Medical toxicologists (MTs) involvement in medication management

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Background/Objective: MTs are being increasing looked to as experts regarding adverse drug reactions, medication errors, formulary decision-making, and other medication-related concerns. The extent and scope of such involvement is unknown. We sought to determine the roles of MTs in their institution's medication management programs.

Methods: An anonymous 12-question survey was distributed via email with reminders to all American College of Medical Toxicology (ACMT) members using an electronic internet-based survey tool. Questions, which were both keyed and open ended, regarded their involvement in medication management issues in their institutions. Topics included: role on different medication management committees, participation in medication guidelines/policies formulation, and evaluation of medication errors and adverse drug events. The survey was not designed for quantitative analysis and only those participating in these efforts were asked to respond.

Results: There were 84 respondents though not all respondents answered all questions. See Table 1 for summary data. Range of time served on such committees was less than 1 year (15); 1–5 years (63); 5–10 years (23); 10–15 years (19); greater than 15 years (13). MTs contributed to the development of guidelines related to acute pain treatment (39), alcohol withdrawal management (45), antidote usage (76), sedation for agitated patients (32), procedural sedation (29), rapid medical response team (9) and anticoagulation/reversal of anticoagulation (5). Respondents also report involvement in administrative aspects of medication management including: quality assurance (52), regulatory/disciplinary (32), and root cause analysis (44).

Conclusions: Based on a limited sample we have identified a wide range of involvement of MT in their institution's medication management process. Many have important administrative and leadership roles. MT are ideal candidates for these positions and responsibilities given the broad nature of the specialty and expertise developed through training and practice.

Keywords: Medical toxicology, Medication management, Formulary and therapeutics

Table 1. Results for abstract 228.

Committee	Chair/Co-chair	Assistant chair	Current member	Alternate member	Past member
Pharmacy and Therapeutics (or Equivalent)	10	3	42	1	1
Medication Safety	7	0	13	2	1
Adverse Drug Reaction/Event	10	1	8	0	0
Other (e.g., medication use, high alert drugs, anticoagulation, antihypertensive therapy, pediatrics, oncology, critical care)	10	0	27	0	0

229. Caustic esophagitis: A porcine model

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Introduction: Caustic ingestions are uncommon in the United States, but have potentially devastating consequences. Evaluation and treatment is primarily aimed at esophageal injury and the complications associated therewith. Treatment with dilution or neutralization to minimize esophageal injury is theoretically beneficial, but not usually recommended because of the lack of safety and efficacy data. Small animal data, such as ex vivo rat esophagi undergoing caustic injury, have demonstrated benefit with these treatments. However, it is difficult to extrapolate this experiment to humans because the conditions are very dissimilar to true ingestion, and because the rat esophagus is very different than human. We describe a new porcine model of caustic esophageal injury and pilot a study to determine efficacy of milk dilution after sodium hydroxide induced esophageal injury.
Methods: Five, 60–80 lb, Yorkshire pigs underwent anesthesia and endotracheal intubation. Subsequently, an orogastric (OG) tube was placed into the hypopharynx. Four out of 5 pigs received 25 mL of a 50% NaOH solution into the esophagus, one subject was uninjured to serve as a control. Two subjects then received 75 mL of 2% milk via OG tube. Flexible endoscopy was then performed at 1, 3 and 6 hours after the injury. The endoscopist was blinded. Injury severity was determined by the endoscopist using a standard grading scale. Animals that survived to the end of the protocol were euthanized.
Results: Two of the four injured animals died proximal to the first endoscopy one hour after injury. One subject that survived the protocol and did not receive milk dilution had a severe, grade 3b injury, while the one subject that survived the protocol and did receive milk therapy had a slightly less severe, grade 3a injury on final endoscopy. The control pig had no injury on edoscopy.
Limitations: The two early deaths were believed to be due to early esophageal perforation and subsequent bilateral pneumothorax. This was likely due to misplacement of the OG tube.
Conclusions: We describe a new model for caustic esophagitis that more reasonably approximates human experience. While strong conclusions cannot be drawn from such a small sample size, we believe that there is some benefit to milk dilution after caustic ingestion. Additionally, we believe that this model can be used to better determine the effects of emergent treatment of caustic ingestions and has advantages over previously described animal models.
Keywords: Caustic, Ingestion, Decontamination

230. The role of early notification from a social networking site in intentional cyanide ingestion

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Background: Cyanide is a potent and rapid acting chemical asphyxiant with varied industrial and laboratory uses. We report a case of intentional cyanide overdose by a university student laboratory worker who was successfully resuscitated due to prompt recognition of his ingestion on Facebook.
Case report: A 21 year-old college student notified friends through a Facebook conversation of his plans to ingest cyanide while alone in his dormitory. Campus police arrived minutes later and he was drowsy with slurred speech. He arrived to the hospital comatose and erythematous. He was tachycardic at 113 beats per minute, blood pressure 109/70 mmHg, and had Kussmaul respirations at a rate >30 breaths per minute. He was promptly paralyzed, sedated, intubated, and placed on a ventilator with a minute ventilation of 20 L/min. He was given 5 g of hydroxocobalamin and 50 g of activated charcoal followed by 12.5 g of sodium thiosulfate. Initial pH was 7.04, and lactate returned at 20.5 mmol/L. Two and a half hours later his lactate was 5.2 mmol/L and pH was 7.54. The patient was extubated the following day with a full neurologic recovery. A blood cyanide concentration ultimately returned at 4.990 mg/L. Death has been associated with concentrations above 3 mg/L.
Case discussion: Cyanide is a potent inhibitor of mitochondrial cytochrome oxidase and is often fatal due to profound cellular hypoxia. Exposure is possible through oral, inhalational, and dermal routes. Clinical presentation includes rapid onset of nausea, confusion, syncope, coma, seizure, with acidosis and eventual cardiovascular collapse. The presentation and serum cyanide concentration are consistent with an expected fatal outcome. Aggressive supportive care and rapid administration of antidotal therapy likely contributed to a favorable outcome in this case. Social networking media has the ability to rapidly disseminate information and was instrumental in the positive outcome of this case. Networking sites, like Facebook, are aware of this phenomenon and have material to assist members with appropriate responses when suicidal gestures are presented to the general network community. Only one indexed article in the literature addresses the importance of social networking sites with regard to suicidality. In this case, rapid acquisition of information assisted in the appropriate treatment with lifesaving benefits.
Conclusions: There is a paucity of literature on medical implications of social networking sites. This case highlights how such sites may help healthcare workers identify and treat patients with otherwise unknown exposures in a timely fashion.
Keywords: Cyanide, Overdose, Social networking

231. Medication and drug use in early pregnancy

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Background: The identification of prescription pharmaceutical and illicit drug misuse among pregnant women is complicated by concerns about stigmatization, custody issues, criminal prosecution, and forced rehabilitation which may compel pregnant women to conceal or minimize their substance abuse in interviews with medical providers. Urine drug testing has been established as an adjunct in the evaluation of drug use during pregnancy. The goal of this study is to identify the prevalence of medication and drug use in our prenatal clinic population, utilizing comprehensive urine drug testing methodologies.

Methods: After IRB approval, anonymous urine residuals were collected during first prenatal visits to our women's primary care clinic in a tertiary care, university hospital in Providence, RI. In cases where subjects were unable to void, an empty specimen cup was collected Specimens were stored frozen until urine drug testing was performed for pharmaceutical and illicit drugs. Samples were screened qualitatively for amphetamines, benzodiazepines, buprenorphine, cocaine, ethanol, methadone, opioids, oxycodone using an EMIT immunoassay. In addition, all specimens were subjected to single-step liquid-liquid extraction with gas chromatography mass spectrometry analysis. Drug confirmation was conducted using multiple analytic methods for specimens that screened positive.
Results: During the study period, there were 560 first pre-natal visits. Clinic staff collected 245 urine specimens, including 45 empty cups, leading to a capture rate of 47.5%. In this population, 64% of specimens tested positive for caffeine, and 17% for cotinine. Antibiotic use was identified in 2.5%, including TMP-SMX, metronidazole, and fluconazole, Anticonvulsant use was found in 1.5%. SSRIs were found in 1%. Ethanol was found in 0.5%, and cocaine metabolites in 1%. THC was identified in 13%. Opioids, methadone, and buprenorphine were found in 2%, 2.5% and 1%, respectively. Illicit drugs were identified in 14% of all specimens, and in 40% of methadone users.
Discussion: In this group, caffeine, cotinine and THC were the most commonly detected substances. We found a low prevalence of benzodiazepines and opioids in the tested specimens. A high rate of concurrent substance abuse was found in the small population of patients who tested positive for methadone exposure. Maternal substance abuse remains the leading preventable cause of fetal morbidity. Identification of the prevalence of drug abuse in a target population using anonymous specimen collection with urine drug testing methods may help guide substance-specific interventions.
Keywords: Substance abuse, Epidemiology, Surveillance

232. 5-oxoprolinemia may not be the only cause of undifferentiated anion gap metabolic acidosis in the setting of acetaminophen use

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Background: Case reports suggest that high levels of 5-oxoproline is responsible for an elevated anion gap (AG) metabolic acidosis in both acetaminophen (APAP) overdose and therapeutic dosing in the absence of liver injury. 5-oxoproline (pyroglutamic acid) is an organic acid intermediate produced during the gamma-glutamyl cycle and as an intermediary during glutathione synthesis. In the setting of glutathione deficiency, increased production and decreased elimination of 5-oxoproline results in its accumulation and subsequent acidosis. We present a case of a massive APAP ingestion with profound metabolic acidosis without predominantly elevated 5-oxoproline levels or liver injury.
Case report: A 69-year-old female with a history of chronic osteomyelitis presented to an outside emergency department (ED) for shortness of breath. The patient admitted to taking a total of 9 grams of APAP over the prior 24 hours. She was found to be profoundly acidemic with a pH of 6.8 and bicarbonate of 3 mEq/L. Laboratory results showed: BUN 42 mg/dL, creatinine 2.19 mg/dL, AST 21 IU/L, and APAP 177 µg/mL. Ethanol and salicylate levels were undetectable. The patient was treated with sodium bicarbonate, N-acetylcysteine (NAC), and fomepizole. Upon arrival at our ED, the patient was alert but hyperpneic. Initial venous blood gas was: pH 7.01, pCO_2 18 mmHg, bicarbonate 4 mEq/L, lactate 3.4 mMol/L. Ibuprofen, ethylene glycol and methanol levels were undetectable. The urine drug of abuse screen was negative. Urinary organic acid evaluation revealed lactate, beta-hydroxybutyrate, succinate, hippuric acid, and only trace amounts of 5-oxoproline. She was treated with IV fluids, sodium bicarbonate and NAC and admitted to the ICU. She had complete recovery over the next 3 days. Her transaminases never elevated. She was discharged to a psychiatric facility on hospital day 7.
Discussion: The patient's clinical presentation was consistent with previously described cases of 5-oxoprolinemia. However, evaluation of the patient's urine identified many different organic acids with a relatively small amount of 5-oxoproline. The predominant organic acid in our case was succinate. Previous reports have suggested that severe acidosis in the setting of APAP ingestion is due to electron transport chain uncoupling or inhibition of essential mitochondrial enzymes. The lack of significant 5-oxoproline urine levels in this patient suggests this as an alternative etiology of her acidosis.
Conclusions: Succinate and not 5-oxoprolinemia was the predominant cause of undifferentiated AG metabolic acidosis in our case of a massive APAP ingestion without signs of acute liver injury.
Keywords: Acetaminophen (paracetamol), Acidosis, Laboratory

233. Using analysis of pooled urine samples from stand-alone urinals (pissoirs) in London to confirm significant use of novel psychoactive substances

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Background: Information on the pattern of use of classical recreational drugs and novel psychoactive substances is typically based on self-reported user surveys. However, this can be unreliable as a number of studies have shown that the content of both classical and novel recreational drugs is variable and therefore users may not be aware of what they are using. There is increasing interest in the use of waste water analysis, typically at the level of water treatment plants, to understand population/sub-population patterns of drug use. This is limited to classical drugs such as MDMA and cocaine, because of poor understanding of the metabolism and stability of novel psychoactive substances. There is the potential to use standalone urinals (also known as pissoirs) to collect a pooled urine sample closer to the point of elimination and therefore detect novel psychoactive substances.
Methods: Urine samples were collected from 12 different stand alone urinals deposited around the City of Westminster area in London, UK (SoHo, West End, Westminster areas) for use by men over a single weekend night in March 2012. After collection, each of the 12 pooled urine samples were stored at 4°C and subsequently analysed using liquid chromatography tandem mass spectrometry (LCMSMS). The study was discussed with the local IRB and deemed not to require formal approval.
Results: All urine samples were positive for nicotine and/or its metabolite cotinine. The following drugs were detected (with

the frequency of detection): 2-aminoindane (2-AI) (11 urinals), cocaine and metabolites (11), amphetamine (10), MDMA and metabolites (8), methylhexaneamine (1,3-dimethylamylamine, DMAA) (7), ketamine and metabolites (6), 4-methylmethcathinone (4-MMC) (5), methamphetamine and metabolites (4), cathine (2), methiopropamine (2), methoxetamine and metabolites (1) and 4-methylamphetamine (1).
Conclusions: Analysis of pooled urine samples from stand-alone urinals in a large inner city can detect the use of both classical recreational drugs and novel psychoactive substances. This study shows a high detection rate of novel psychoactive substances including 2-aminoindane, methylhexaneamine and methiopropamine, which are currently uncontrolled in our country. There is the potential to expand this study to provide analytical confirmation of the actual novel psychoactive substances being used and look at trends both over time and between different geographical regions to allow legislative authorities to consider whether they should be controlled.
Keywords: Substance abuse, Surveillance, Bath salt

234. Pediatric marijuana exposures in medical marijuana legal state

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Background/Objectives: Sixteen states and Washington DC have enacted laws to legalize medical marijuana. Until October 2009, the use of medical marijuana was limited by concerns about enforcement of federal drug laws. In October 2009, federal prosecutors were instructed not to arrest medical marijuana users and suppliers who comply with state laws. The objective of this study is to determine if the change in federal policy was associated with an increase in pediatric accidental marijuana exposures evaluated at our children's hospital.
Methods: A retrospective cohort study of patients evaluated from January 2005 through December 2011 at our children's hospital. All patients younger than 12 years with emergency department (ED) visits for ICD-9 codes representing accidental ingestions or poisonings by psychedelics/hallucinogens were identified and reviewed. The following information was abstracted for cases identified as marijuana exposure: demographics, product ingested (medical vs nonmedical), clinical effects, disposition, and testing. The monthly rate of visits before and after the change in policy was calculated using a Poisson regression.
Results: Between January 2005 and October 2009, there were no patients younger than 12 years evaluated in the ED for marijuana ingestion. Between October 2009 and December 2011, there were 14 patients evaluated for marijuana ingestion. The median age of cases was 3 years, with a range from 8 months to 12 years and 65% were male. Nine patients (64%) were documented exposures to medical marijuana (8 as a food product); in 5 cases the product was not documented as medical or non-medical. One patient presented with a history of a minimal exposure and had no symptoms or workup. The remaining 13 had central nervous system effects such as lethargy and ataxia; one case developed respiratory insufficiency. All symptomatic patients had marijuana detected on urine testing. Other evaluations included: laboratory tests (13), CT (8), and lumbar puncture (2). Five patients (36%) were observed in the ED and eventually discharged; 8 were admitted (57%), 2 (14%) of which to the intensive care unit. The change in federal policy was associated with an increase in the incidence of cases from 0 (95% CI 0 to 0.06) to 0.5 (0.3 to 0.9) per month.
Conclusions: The increased availability of medical marijuana in our community was associated with an increase in visits to the emergency department for marijuana exposures in pediatric patients <12 years of age after October 2009. The majority of patients were younger than 6, ingested a food product containing medical marijuana, had CNS effects, received extensive testing and were ultimately admitted.
Keywords: Marijuana, Pediatric, Medical marijuana

235. Injectable tanning toxicity: More than skin deep

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Background: The plethora of recreational and designer drugs available through the Internet can baffle even the most highly trained physicians. These substances may be used to achieve states of euphoria, to avoid detection of the law, and to enhance physical performance and appearance.
Case report: A 39-year-old male stated that he injected 6 mg of Melanotan subcutaneously in an effort to darken his skin during wintertime. This was his first use of this product and was a 6-fold dose injection. He denied any other drugs or intent of self-harm but did take a "pain pill" earlier in the day. In the emergency department 2 hours post injection, he complained of diffuse body aches and anxiety. Vital signs included blood pressure of 151/85 mmHg, heart rate of 130 beats per minute, and a temperature of 97.8 °F. Physical exam was significant for an anxious and restless appearing male with mydriasis (7 mm), diaphoresis, diffusely flushed skin, tachycardia, and fine muscle tremors. Significant lab results included new elevation of creatinine to 2.25 mg/dL, creatine phosphokinase (CPK) to 1760 IU/L, troponin to 0.23 ng/mL, leukocytosis to 19.1 THO/mm^3. Urinalysis revealed red cell casts and 3+ blood with 0–2 RBC and a qualitative urine drug screen was negative for metabolites of cocaine or amphetamines but positive for opiates. The patient was given benzodiazepines for agitation and anxiety with improvement of symptoms and was admitted to the intensive care unit. CPK peaked at 17773 IU/L twelve hours after hospitalization but clinical symptoms improved with normalization of heart rate and resolution of diaphoresis and tremulousness. He continued to receive intravenous fluids for rhabdomyolysis and the CPK decreased to 2622 IU/L and creatinine improved to 1.23 mg/dL. The patient was then discharged after hospital day 3. The substance, which the patient provided and stated that he injected, was analyzed via mass spectrometry and was confirmed to be melanotan II when compared with a standard sample.
Case discussion: Melanotan induces melanocyte production thus enhance darkening of the skin and create sunless tanning. Additionally, it is marketed as a supplement to treat sexual dysfunction in men and women. It is easily purchased over the Internet, and law does not regulate delivery to consumer's residence. To our knowledge, this is the first reported case of systemic toxicity from melanotan usage in the medical literature.

Conclusions: This case highlights an unregulated substance available over the Internet and its potential for systemic toxicity. Medical toxicologists must maintain a high index of suspicion for these new Internet based drugs. Reporting these exposures and related toxicities will aide in the management of these emerging hazards.
Keywords: Melanotan, Adverse drug event, Dietary supplement

236. Surveillance for radiation exposures using the National Poison Data System

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Background: Since 2010, the Centers for Disease Control and Prevention (CDC) and the American Association of Poison Control Centers (AAPCC) have conducted surveillance for exposures to radiation and radioactive materials reported from all 57 US Poison Centers (PCs) using the National Poison Data System (NPDS). CDC, AAPCC and Poisindex® Thomson Reuters, recently implemented improvements to the NPDS radiation coding structure to enhance its utility for public health surveillance. Our objective is to describe results from this surveillance and associated radiation incidents during a one-year period.
Methods: We analyzed all PC calls from September 1, 2010 to September 1, 2011 that met our case definition: human exposure calls with any of the following NPDS codes: "miscellaneous non-radiopharmaceutical isotopes", "miscellaneous radiopharmaceuticals", or "ionizing radiation". Exclusion criteria were: exposure calls about substances such as "radon", "radon gas", "non-ionizing radiation", "smoke detectors" or calls with outcomes of "confirmed non-exposure" or "adverse reaction". Using NPDS data, CDC staff reviewed each call meeting this definition. CDC and AAPCC staff also contacted the regional PC to obtain further information and confirm exposures. When multiple reported exposures clustered in space and time were detected, news stories in the public media were also monitored for associated radiation incidents.
Results: During the study period, a total of 186 calls met the case definition. 51 of these were associated with 3 radiological incidents reported in the public media and one anti-terrorism exercise. The incidents included a radiation exposure from industrial radiography which led to the temporary closure of a hospital (n = 4; 8%), a transportation accident involving potential radioactive contamination to several medical personnel (n = 11; 22%), the Fukushima Daiichi Japan nuclear reactor disaster (n = 10; 20%) and a regional radiation anti-terrorism exercise involving a cesium radiological dispersal device (n = 26; 50%). The remaining 135 calls were single person exposures and were not followed by the regional PC.
Conclusions: During a one-year period, our case definition detected 51 calls associated with four radiation incidents. These incidents were of potential public health significance because they involved multiple persons and had the potential to cause widespread public concern. Similar surveillance strategies for detecting and tracking future radiological incidents using NPDS may be helpful to regional PCs and their public health partners.
Keywords: Radiation, National Poison Data System, Surveillance

237. Intentional and Unintentional Prescription Opioid Exposures in Italy, Germany and the United States

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Table 1. International Prescription Opioid Exposures Reported to Poison Centers from 2007–2011.

Country	Exposure type	Exposures/100,000 population					% change
		2007	2008	2009	2010	2011	2007 to 2011
Oxycodone							
Germany	Intentional	0.152	0.205	0.212	0.288	0.288	90.0
Italy		0.003	0.003	0.029	0.033	0.025	614.7
United States		3.238	3.634	4.122	4.496	4.344	34.2
Germany	Unintentional	0.030	0.114	0.045	0.061	0.174	475.0
Italy		0.005	0.003	0.010	0.010	0.018	249.4
United States		1.507	1.696	1.947	1.997	1.758	13.8
Buprenorphine							
Germany	Intentional	0.098	0.098	0.121	0.106	0.129	30.8
Italy		0.033	0.045	0.021	0.014	0.011	−64.9
United States		0.290	0.438	0.535	0.610	0.589	103.4
Germany	Unintentional	0.061	0.023	0.030	0.008	0.061	0.0
Italy		0.005	0.009	0.012	0.010	0.008	58.8
United States		0.225	0.397	0.593	0.659	0.483	114.6
Methadone							
Germany	Intentional	0.295	0.250	0.295	0.326	0.417	41.0
Italy		0.076	0.072	0.069	0.084	0.074	−2.5
United States		1.277	1.170	1.270	1.286	1.191	−6.7
Germany	Unintentional	0.076	0.083	0.098	0.121	0.106	40.0
Italy		0.021	0.028	0.022	0.029	0.023	11.2
United States		0.354	0.322	0.373	0.337	0.280	−21.1

% change calculated from rate data prior to rounding.

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Background: Prescription opioid abuse in the US has been deemed epidemic. However, abuse in other countries is not well studied. This study provides abuse rate data for three common opioids over a 5 year period in Italy, US, and Northwest Germany.
Methods: Human exposures to oxycodone, buprenorphine, and methadone reported from 2007–2011 were obtained from Milan and Göttingen Poison Centers (PCs). US data were obtained from PCs participating in the RADARS® System. PCs in all 3 countries manage calls from healthcare providers and the public. Milan PC handles 65–70% of all calls in Italy. Rates are expressed as number of exposures/100,000 population separately for intentional (IE) and unintentional (UE) exposures. IEs are those with a reason of suicide, abuse, misuse, and unknown intentional. UEs include unintentional general, unsupervised ingestions or therapeutic errors.
Result (Table 1): From 2007 to 2011, Italy PC rates of IE to oxycodone increased dramatically while methadone remained stable and buprenorphine decreased. UE rates increased for all 3 drugs. In Northwest Germany IE and UE rates for all 3 drugs increased with the exception of UE to buprenorphine. In the US, methadone is the only drug that had decreased exposure rates.
Conclusions: Rising rates of opioid exposures are not unique to US. Perhaps the most concerning is the dramatic rise in unintentional exposures since these are most often accidental unsupervised ingestions in children which are life-threatening. Transatlantic data collection is possible with comparably few resources.
Keywords: Poison center, Stimulant, Prescription drug abuse

238. Assessing the prevalence of pancreatitis following resuscitative use of intravenous lipid emulsion

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Background: Intravenous (IV) lipid emulsion has long been part of total parenteral nutrition (TPN) regimens. Recently, the use of IV lipid emulsion (ILE) therapy for drug toxicity has become popular. The amount of lipid given per unit time is typically much greater in antidotal use than in TPN. Complications from use of ILE as an antidote, including pancreatitis (PANC), have only recently been described. The purpose of this pilot study is to determine the frequency by which patients develop pancreatitis following ILE therapy.
Methods: A retrospective review of all patients admitted between 1/1/2005 to 12/31/2011 and treated with ILE at 2 in-patient toxicology services. Patients who expired within 24 hours of admission were excluded. Data were abstracted using standardized chart review methods. Descriptive analysis was performed. PANC was defined a priori as an elevated lipase with symptoms of epigastric pain or nausea/vomiting.
Results: Seven patients received ILE. One patient was excluded (expired < 24 hours). Mean age was 27.4 years (range 13–48); 50% were male. Five patients received ILE for dysrhythmias (WCT/VT = 2, asystole = 2), while 1 each received ILE for refractory hypotension or seizures. Three patients developed PANC. The 1st case was a 13 y.o. treated for amitriptyline toxicity with 2 boluses of ILE (1.5 mL/kg each), followed by 0.25 mL/kg/min for 30 minutes. Lipase and triglycerides peaked at 1849 IU/L and 8611 IU/L, respectively. The 2nd case was a 20 year old with doxepin toxicity treated with 2 doses of ILE 2hapart (each dose 1.5 mL/kg, followed by 0.25 mL/kg/min for 30 minutes). Lipase and triglycerides peaked at 2951 IU/L and 3648 IU/L, respectively. The 3rd case involved a 20 year old with bupivacaine toxicity who received 1.7 mL/kg bolus, followed by 0.26 mL/kg/min for 3h. The patient complained of epigastric pain and vomiting following resuscitation. A lipase was checked on hospital day 13, as the patient's symptoms were improving, and was 185 IU/L. Two of the 3 patients who developed pancreatitis received 2 boluses and one received a prolonged infusion. The 3 patients who did not develop PANC had 1 bolus each; 2 had no maintenance infusion and 1 had a 60 min infusion.
Conclusions: PANC is common among patients who are treated with antidotal doses of ILE and survive their drug toxicity. PANC appears more common in those who received multiple doses or prolonged infusion of ILE. Due to the small number of patients included, firm conclusions should not be drawn from these data. However, this pilot study suggests toxicity is possible from antidotal use of ILE. Until further information is available, it is prudent to restrict use of ILE rescue to life-threatening toxicity.
Keywords: Lipid therapy, Pancreatitis, Antidote

239. Outpatient experience of hip arthroplasty metal ion evaluations

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Background: Last year, we presented our outpatient clinic experience of the first twenty of patient evaluations for elevated blood metal ion levels from metal-on-metal (MoM) hip arthroplasties. We now present additional data for 26 months involving 32 patients.
Methods: This was a consecutive series of patients presenting to two outpatient medical toxicology clinics from January 1, 2010–March 1, 2012 with history of having undergone total hip arthroplasty. 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 32 patients were analyzed. The average age was 55.2 years (range 28 to 86 years). There were 10 males and 22 females. Ten patients exhibited no symptoms and sought evaluation for gsymptom (n = 7) while 2 other patients had been previously diagnosed with fibromyalgia. Twenty-five (78%) had pain described as hip or thigh pain, clicking sound with ambulation and/or grinding with activity. Two (7%) patients had ataxia and later diagnosed with demyelination neuropathy with one patient demonstrating marked improvement after revision. Patients with MoM arthroplasties generally exhibit a tenfold increase in metal ion levels than traditional arthroplasties (Cobalt level 22.1 mg/mL IQR .9–59 in MoM vs. 1.25 ng/mL IQR 0.9 to 2.6 in non-MoM). Finally, 17 (58.6%) patients had replacement or revision of their hip implant. Of these patients, 10 (34.5%) had pre- and post-procedure cobalt and chromium concentrations. The median pre- and

post-procedure cobalt concentrations were 44 ng/mL (IQR 5–80) and 2.65 ng/mL (IQR 1.2–4.1) respectively. The median pre- and post-procedure chromium concentrations were 31 ng/mL (IQR 3.8–91) and 3.45 ng/mL (IQR 1–8) respectively. No patients underwent chelation.
Discussion: A significant number of individuals who seek toxicology consultation will either have minimal symptoms (fatigue, muscle aches) or be essentially asymptomatic (n = 19 or 59%). Ion levels alone are not diagnostic for systemic toxicity. Documented neurotoxicity is extremely uncommon and, does not appear to be a dose-related phenomenon. The decision for hip revision solely for toxicologic reasons is exceptionally rare and usually involves a multidisciplinary approach. This series suggests that MoM arthroplasty can result in elevated cobalt and chromium concentrations. Most patients seeking toxicologic referral may be minimally symptomatic and seek guidance regarding elevated blood/serum metal ions; however, toxicologic based interventions are unusual.
Keywords: Neurotoxicity, Foreign body, Heavy metals

240. Exposures to direct thrombin inhibitors and factor Xa inhibitors reported to United States poison control centers

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Objective: The objective of this study was to characterize exposures to direct thrombin inhibitors (e.g. dabigatran) or Factor Xa inhibitors (e.g. rivaroxaban) reported to US poison centers.
Methods: Data from the American Association of Poison Control Center's National Poison Data System (NPDS) for exposures to direct thrombin inhibitors and factor Xa inhibitors between January 1, 2009 and December 31, 2011 were obtained. Cases were excluded if the number of substances was greater than one.
Results: A total of 929 exposures to direct thrombin inhibitors were reported to NPDS, of which 376 were single substance exposures. *Reason for Exposure:* 309 (82%) of exposures were unintentional, 6 (2%) were intentional, 57 (15%) were adverse reaction and 4 (1%) had an unknown reason. The most common reason for exposure was unintentional-therapeutic error, which occurred in 262 cases (70%). The associated scenario was most often that medication was inadvertently given twice (180 cases, or 69% of therapeutic errors) or doses given too closely together (49 or 19% of therapeutic errors). *Age:* Children 6 years and younger were 25 (7%) exposures, no exposures for ages 7 to 20 years, and 11 (3%) exposures occurred in patients between 20 and 49 years. The majority of patients were over age 50 (306 patients or 81%), with the largest proportions between 70 and 79 years (109 or 29%) and 80–89 years (96 or 25%). *Management Site:* Although most cases were managed on site (255 cases or 68%), 31 (8%) patients were referred to a health care facility, and 81 (21%) patients were already in or en route to a health care facility when the call was taken. *Clinical Effects:* Effects occurred in 56 (15%) exposures. The most frequently reported clinical effects included bleeding (16 patients or 4%), PT prolongation (18 patients or 5%), other coagulopathy (16 patients or 4%), and hematemesis (9 patients or 2%). *Outcomes:* There were 6 deaths (1.6%), 11 exposures with major effects (3%), 32 exposures with moderate effects (8%), 7 exposures with minor effects (2%), and 80 exposures with no effects (21%) coded. Most cases (214 or 57%) were not followed, judged to be either nontoxic or minimal clinical effects possible.
Conclusions: Exposures to direct thrombin inhibitors and factor Xa inhibitors were primarily accidental and occurred in older adults. At least 15% of exposures resulted in symptoms, although most cases were not followed to known outcomes.
Keywords: Anticoagulant, National Poison Data System, Direct thrombin inhibitors/factor Xa inhibitors

241. Case series of patients presenting with warfarin overdose

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Background: Warfarin is a vitamin K antagonist commonly used in the prophylaxis and treatment of venothromboembolic disease. Guidelines for the management of patients on warfarin who present with supratherapeutic international normalized ratios (INRs) exist. However, these guidelines are not designed for managing acute warfarin overdose. We sought to describe the clinical presentation of patients with warfarin overdose and the use of vitamin K in these patients.
Methods: Patients who were admitted between 1 January 1987 and 31 March 2012 with diagnosis of acute warfarin overdose at 2 teaching hospitals were identified. Charts were reviewed and data were extracted, for the following: demographic information; reported dose of warfarin ingested; reason for anticoagulation; initial and peak prothrombin time (PT)/INR; doses, amount, and route of vitamin K administered; time to medical clearance; and bleeding or thrombotic complications. For cases whose PT/INR were > 100/10, a PT/INR of 100/10 were used respectively for calculations.
Results: 20 patients were identified with a mean (SD) age of 44 (+/−16.7; range = 2–60) years; 65% were men. The warfarin belonged to the patient in 17/20 (85%) of cases. The most common reason for anticoagulation was venothromboembolic disease. The amount ingested was reported in 14/20 (70%) cases. The median (IQR) amount of warfarin ingested was 106 (90–265) mg. 5 patients had a peak PT/INR that exceeded the lab's limit of quantification. The median (IQR) initial and maximal PT/INR were 19.5 (14–40) seconds/1.7 (1.1–3.9) and 58.6 (33.3–100) seconds/6.3 (3.3–10), respectively. 2 patients had epistaxis on admission. vitamin K was administered to 15/20 (75%) patients. Among those patients who received vitamin K, the median total (IQR) amount of Vitamin K administered was 10 (10–55) mg, and the median (IQR) number of doses was 3 (2–4). 2 patients had hemorrhagic complications; epistaxis which was present in each on admission, and a retroperitoneal hematoma that developed during the hospitalization in 1 of the patients with epistaxis. No patient had subsequent thrombotic complications from over-correction during their hospitalization stay. The vitamin K was administered orally in 8 cases, intravenously in 3 cases, and via multiple routes in 4 cases. The mean (range) time to medical clearance was 5 (2–25) days.
Conclusions: In this series of patients with acute warfarin overdose, patients received higher doses of vitamin K than would have

been given based on published guidelines for management of warfarin over-anticoagulation. Published guidelines for management of patients on warfarin who present with supratherapeutic INRs may not be applicable in the setting of acute warfarin overdose.
Keywords: Anticoagulant, Warfarin, Vitamin K

242. Verification of vitamin-k dependent factor activity for marker of brodifacoum poisoning

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Background: Brodifacoum, a readily available rodenticide, is a four-hydrooxycoumarin derivative that is long-acting and 100 times more potent than warfarin. Ingestion causes life-threatening coagulopathy via inhibition of vitamin K-2,3-epoxide reductase and reduced production of the vitamin-K dependent clotting factors II, VII, IX and X. Treatment of brodifacoum often requires large amounts of vitamin K for weeks to months to reverse the associated coagulopathy. Timely identification of brodifacoum and other "superwarfarin" anticoagulant rodenticides is limited by access to appropriate testing facilities. We present a case that demonstrates the use of vitamin-K dependent factor activity as a surrogate for the diagnosis of superwarfarin anticoagulant rodenticide poisoning.
Case report: A 49 year-old female with a history of recurrent GI bleeds, uterine cancer, radiation proctitis and partial colectomy, and recurrent hematochezia, presented to an outside hospital with hematochezia. Labs demonstrated a prothrombin time (PT) of 163 sec (11.7–15.3), INR 16.7 (0.8–1.2), partial thromboplastin time (PTT) of 130 sec (22.7–35.6) and a hemoglobin was 7.3 gm/dL. The patient received fresh frozen plasma, packed red blood cells and was transferred to our facility for further management. She denied warfarin ingestion as well as any rodenticide exposures. PT and PTT mixing studies were normal. Factor V (97%; range 70–150), factor VIII (133%; range 60–150) and thrombin time (16.7 sec; range 16–22) were normal. Vitamin K-dependent clotting factors II (40%; range 70–150), VII (6%; range 70–160), IX (7%; range 60–150) and X (29%; range 70–150) were all found to be low. These findings were highly suggestive of a vitamin-k inhibition coagulopathy. A brodifacoum drug level was subsequently found to be positive via high performance liquid chromatography/tandem mass spectrometry. These findings contributed to transfer of the patient to a local psychiatric hospital after medical optimization on 60 mg of vitamin K daily.
Discussion: Brodifacoum induced vitamin K-2,3-epoxide reductase inhibition will demonstrate low II, VII, IX and X coagulation factor activity, whereas non-vitamin K dependent factors VIII, V and thrombin time will remain normal.
Conclusions: This case suggests the utility of measuring and comparing vitamin K dependent clotting factor activity to non-vitamin K dependent factor activity as a surrogate marker of brodifacoum poisoning. Superwarfarin levels generally take several days for results and may delay appropriate follow up and treatment. In facilities that can obtain clotting factors, this may be a more expeditious way to make a superwarfarin diagnosis.
Keywords: Anticoagulant, Laboratory, Rodenticide

243. Melanotan II: An unusual cause of drug-induced priapism

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Background: The synthetic melanotropic peptides, Melanotan I (afamelanotide) and Melanotan II (bremelanotide), are non-selective melanocortin receptor agonists originally developed as tanning agents. A chance discovery revealed melanotan II could induce penile erection. Benefit for patients with erectile dysfunction has been investigated. Only mild adverse events have been reported.
Case report: A 60 year-old man presented to a urologist with refractory priapism after self-injecting an indeterminate dose of Melanotan II, obtained from an online source, into the subcutaneous (SC) tissue of his left lower abdomen. Within 30 minutes of injection, he developed a painful erection. Temporary detumescence was achieved after 0.25 mg SC terbutaline, corporal aspiration, and irrigation with a vasoconstrictor. He developed diaphoresis, tachycardia, and rigors, prompting transfer to the ED. Vital signs signs were normal except HR 113 and BP 142/81. He was intensely diaphoretic, complaining of 10/10 penile pain, nausea, and a "need to stretch." He exhibited odd voluntary back-arching behavior. He had WBC 12.4, 10% bandemia, anion gap 13, and creatinine 1.35 mg/dL. His tachycardia, diaphoresis, hypertension, and back-arching abated with repeated doses of benzodiazepines. Priapism recurred requiring Winter's shunting by a urologist. The patient was admitted for 24 hours with continued benzodiazepine treatment and recovered completely.
Case discussion: The prevalence of bremelanotide use is unknown. Online communities of users exist, the most popular of which report more than 5000 members. In addition to concerns about potential impurity or contamination of unregulated products, adverse effects of these compounds are minimized on websites. Published accounts of adverse events in the medical literature include nausea and vomiting, flushing, headache, hypertension, taste disturbance, stretching and yawning syndromes, darkening or eruption of melanocytic nevi, and melanoma. In addition to previously published side effects of bremelanotide, our patient presented with, tachycardia, diaphoresis, back-arching, and recalcitrant priapism, requiring surgical shunting. Medication review did not reveal an alternative explanation for his priapism.
Conclusions: Melanotan II is an unregulated xenobiotic used for tanning, enhanced penile erection, and increased libido. The medical literature reports only minor adverse effects which are minimized by the procurement websites. We report a case of bremelanotide-associated recalcitrant priapism, tachycardia, and leukocytotic effects. To our knowledge, these are the most severe adverse effects yet reported with melanotan II use.
Keywords: Adverse drug event, Dietary supplement, Internet

244. Multisystem organ failure following lipodissolve injections

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Background: Lipodissolve, also known as injection lipolysis, is a procedure marketed as a means to rid the body of unwanted pockets of fat. The process entails a series of injections of phosphatidylcholine and deoxycholate; however, other substances such as vitamins, herbal extracts and drugs are sometimes added. This product has not been approved by the FDA, yet is available and sometimes used without any credible scientific evidence that supports its safety or efficacy.
Case report: A 63-year-old woman, on lisinopril, citalopram, solifenacin, herbal medications (raspberry ketone, acai, cranberry) and naturopathic vitamins, underwent her first lipodissolve procedure at a home spa run by a nurse. She received 27 subcutaneous injections in her abdomen, back, buttocks, and thighs, which were reported to contain a total of 135 mg lidocaine, 27 mL of a phosphatidylcholine & deoxycholate mixture (provided by a compounding pharmacy), and 284 mL of 0.9% NaCl. Immediately following the injections she became dizzy. Vital signs (VS) documented by the RN were BP 107/64 mmHg and HR 48 bpm. Vomiting and back pain developed; she was sent home, but persistent vomiting prompted her to go to an ED, where VS were normal and she was afebrile. Labs 6 hr after the procedure revealed AST 6992 IU/L, ALT 3467 IU/L, TBili 1.7 mg/dL, PT 17.2 sec, CPK 8 IU/L, BUN 27 mg/dL, Cr 0.81 mg/dL, lipase 38 IU/L, and normal electrolytes. WBC was 5.8 K/mm^3, Hgb 13.3 g/dL, plt 287 K/mm^3. APAP and ASA were negative. ECG, CXR and abdominal US were normal. Over the next 6 hr AST and ALT climbed to 22,560 IU/L and 12,032 IU/L, and Cr to 1.75 mg/dL. Hepatitis work-up was negative. LFTs 3 months earlier had been normal. Liver function recovered over days, but renal failure progressed, with Cr = 9.9 mg/dL on day 5. Hemodialysis was begun. Renal biopsy showed ATN. GCMS analysis of the phosphatidylcholine & deoxycholate solution revealed lidocaine and benzyl alcohol, and sterility testing was negative. The patient's urine showed only lidocaine and meds she was given in the ED.
Discussion: Lipodissolve is continuing to be marketed as a fat reduction technique despite FDA warnings that there is no evidence to show it is safe or effective. This report highlights a severe complication following this injection therapy. The exact cause of ATN in this patient is unknown. It is possible the patient had undocumented hypotension prior to ED presentation or that the product contained an unidentified nephrotoxic agent.
Conclusions: Lipodissolve treatments may cause severe complications such as acute hepatitis and renal failure.
Keywords: Alternative medicine, Hepatotoxicity, Renal toxicity

245. Pancreatitis following treatment with intravenous lipid emulsion therapy for severe TCA toxicity

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Background: Intravenous lipid emulsion (ILE) therapy is advocated as an antidote for life-threatening toxicity due to lipophilic drugs, including tricyclic antidepressants. Complications from ILE are rarely described. We describe a case of ILE-induced pancreatitis.
Case report: A 20 yo man was found unresponsive 75 min after last being seen well and 3 days after release from inpatient psychiatric care. The patient had known access to doxepin (>3 g), levetiracetam, and citalopram (1.2 g). EMS reported a HR of 130 and SBP of 87. He was intubated without medications and experienced a brief, generalized tonic-clonic seizure prior to arrival in the ED. Upon arrival, his BP was 68/32 with a HR of 150. Initial ED treatment included lorazepam and fosphenytoin, followed by sodium bicarbonate 150 mEq IV for a prolonged QRS (126 msec). He developed brady-asystolic arrest and was resuscitated with additional bicarbonate and epinephrine. After a second brady-asystolic arrest, a 1.5 mL/kg of 20% ILE bolus, followed by 0.25 mL/kg/min for 30 minutes was given. The patient was transferred to our ICU with a HR of 131 and SBP of 100. Upon arrival to the ICU, he became bradycardic with a wide QRS. 2 hours after the first dose of ILE was administered, a second bolus and infusion of ILE were given. In total, the patient received 1550 mEq of sodium bicarbonate and 1210 cc of 20% ILE. Norepinephrine and amiodarone infusions were utilized.

Labs on arrival to the ED were pH 6.66, CO2 15 mmol/L, and arterial lactate 14.9 mmol/L. Labs on arrival in the ICU were total tricyclic 2368 ng/mL, levetiracetam 57.2 mcg/mL, and lipase 32 IU/L. Urine drug testing (GCMS) revealed doxepin, phenytoin, citalopram, and levetiracetam. No evidence of shock liver occurred. Triglycerides peaked at 3648 mg/dL, when tested hours after ILE, but fell to 85 mg/dL the following day. Bilirubin began to rise on HD#3 and peaked on HD#4 at 2.9 mg/dL. Lipase level began to rise on HD#5 and peaked on HD#6 at 2951 IU/L.

The patient made a full recovery. He was transferred to inpatient psychiatry on HD#8, with a lipase of 2942 IU/L, no abdominal pain, and tolerating a low-fat diet. The patient was readmitted to a medical bed when he began vomiting and complaining of epigastric pain after eating on HD#9. CT scan on HD#11 revealed pancreatitis, no pseudocyst or hemorrhage, and a normal gallbladder. His lipase was 456 IU/L, 24 days after ILE.
Discussion: Pancreatitis may follow ILE. Hyperlipidemia and hypertriglyceridemia are expected after ILE. Despite their known association with pancreatic disease, pancreatitis following ILE therapy for overdose has been reported only once previously. We recommend serial assessments for pancreatitis follow ILE rescue.
Keywords: Lipid therapy, Antidepressant, pancreatitis

246. Exposures to newer injectable diabetes medications reported to United States (US) poison control centers

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Objectives: The objective of this study was to characterize exposures to glucagon-like peptide-1 (GLP-1) receptor agonists (exenatide or liraglutide) and human amylin analogs (pramlintide) reported to US poison centers.
Methods: Data from the American Association of Poison Control Center's National Poison Data System (NPDS) for exposures to GLP-1 receptor agonists and amylin analogs between January 1, 2000 and December 31, 2010 was obtained. Cases were excluded from analysis if the number of substances was greater than one.
Results: Nine-hundred nineteen exposures to GLP-1 receptor agonists (exenatide and liraglutide) and 205 exposures to

pramlintide were reported. Most (n = 819, 89%) exposures to GLP-1 receptor agonists were unintentional, with 723 therapeutic errors (79%). The reason was adverse reaction in 70 exposures (8%), intentional in 21 exposures (2%), and 5 exposures (1%) were other-contaminant or tampering exposures. Most exposures (n = 203, 99%) to pramlintide were unintentional, with 191 therapeutic errors (93%), and the remaining 2 (1%) exposures were adverse reactions. GLP-1 receptor agonist and pramlintide exposures occurred mostly in adults in their 50s (n = 221, 25% and n = 75, 37% respectively) and 60s (n = 207, 23% and n = 50, 24% respectively), with only 18 (2%) GLP-1 receptor agonist and 4 (2%) pramlintide exposures in children 6 years and under. Most exposures to GLP-1 receptor agonists and pramlintide were managed on site (n = 703, 76% and n = 147, 72%, respectively). The most commonly reported clinical effect was nausea for both GLP-1 receptor agonists and pramlintide (n = 158, 17% and n = 18, 9% respectively). GLP-1 receptor agonist exposures also resulted in vomiting (n = 87, 9%) and hypoglycemia (n = 76, 8%). There were no deaths in either group. GLP-1 receptor agonist exposures resulted in 5 major effects (1%), 97 moderate effects (11%), 144 minor effects (16%) and 245 exposures with no effects (27%). Most GLP-1 agonist receptor cases (n = 353, 37%) were not followed. Pramlintide exposures resulted in moderate effects for 7 patients (3%), minor effects in 21 patients (10%), and no effects in 84 patients (41%). Sixty-eight pramlintide cases were not followed (33%).

Conclusions: Exposures to GLP-1 receptor agonists and human amylin analogs were primarily therapeutic errors and occurred in adults 50 years of age and older. Clinical effects were noted in more than 25% of GLP-1 exposures, although most cases were not followed to known outcomes. Clinical effects were less common in the pramlintide group.

Keywords: GLP-1 receptor agonists, Injectable diabetes medications, National Poison Data System

247. Cyproheptadine withdrawal with features of serotonin toxicity

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Background: Cyproheptadine is an antihistamine with serotonin (5-HT) antagonist properties used to treat Serotonin Syndrome. Sparse literature describes its use for pediatric migraine. We describe a case of discontinuation of high-dose cyproheptadine that resulted in features of serotonin toxicity.

Case report: An 8 year old male without previous psychiatric history had been taking cyproheptadine 4 mg PO BID for headaches for 2 months. The medication was abruptly discontinued when his local pharmacy ran out of its supply. Approximately 36 hours after his last pill, he developed agitation and diaphoresis. He presented to the ED, received lorazepam 1 mg PO, and was discharged. Later that evening, he returned to the ED after becoming progressively agitated and was described as unable to sit still, inconsolable, and undirectable. Physical exam was notable for tachycardia and psychomotor agitation with no tremor or clonus. Basic labs were normal; UDS was positive for benzodiazepines. He received midazolam 2 mg IV, lorazepam 2 mg IV, and was treated with cyproheptadine 4 mg PO; he subsequently slept comfortably and was discharged the next morning with plans to resume a 4 mg PO BID regimen. Shortly after returning home, he again became agitated; on return to the ER, he required 5-point restraints, olanzapine 5 mg IM, and cyproheptadine 8 mg PO prior to calming. After discussing his dosing with his mother, it became clear he had been taking a higher dose of 8 mg PO BID for 2 weeks prior to drug discontinuation. He was discharged the next morning on 8 mg PO BID with plans for a prolonged taper. Comprehensive serum drug screening sent at the time of his second ED presentation was positive only for caffeine, theobromine, and ibuprofen.

Case discussion: There is minimal published data regarding adverse outcomes from long term use of cyproheptadine. This case describes a discontinuation syndrome exhibiting features of serotonin toxicity (mental status changes, neuromuscular hyperactivity, autonomic instability). We postulate that our patient became hypersensitized to 5-HT in the setting of chronic 5-HT antagonism and that abrupt removal of the antagonist resulted in functional serotonin overstimulation.

Conclusions: This is the first published account of cyproheptadine withdrawal with features of serotonin toxicity.

Keywords: Adverse drug event, Serotonin syndrome, Pediatric

248. Methemoglobinemia and carboxyhemoglobinemia associated with Zosyn-induced bite cell hemolytic anemia

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Background: Bite cell hemolytic anemia, AKA drug-induced immune hemolytic anemia (DIIHA), is a rare adverse drug effect. Piperacillin is the third leading cause of DIIHA. Although methemoglobinemia (MetHb) has been observed in DIIHA from known MetHb-inducers, MetHb and carboxyhemoglobinemia (COHb) have never been described in DIIHA secondary to non-MetHb inducing xenobiotics. We describe a case of MetHb and COHb associated with Zosyn administration.

Case report: A 37 year-old male with cardiomyopathy and heart failure was admitted for abdominal pain. His medication regimen did not include known MetHb-inducers. Diagnostic CT resulted in contrast nephropathy and subsequent urinary tract infection. On hospital day (HD) 3, he was started on Zosyn. On HD 6, his hemoglobin dropped from 12.2 to 6.1 mg/dL, he desaturated to 85%, and co-oximetry indicated MetHb of 9.5% and COHb of 4.7%, while schistocytes and bite cells appeared on his peripheral smear. G6PD activity was 4.90 U/G Hb (range 4.60–13.50). His haptoglobin level was < 30 mg/dL, consistent with hemolysis. Ascorbic acid was initiated while transfusing packed RBCs. A Coombs test revealed positive direct antibody testing (DAT+) and negative eluate testing, consistent with piperacillin-induced immune hemolytic anemia. On HD 9, Zosyn was discontinued, followed by gradual resolution of the patient's hemolysis, MetHb, and COHb. Unfortunately, the patient died on HD 43 secondary to a pulmonary embolism.

Case discussion: Piperacillin and tazobactam are not considered MetHb-inducers. Zosyn-induced DIIHA is associated with IgG-coated RBCs, leading to opsonized RBC destruction by the

reticuloendothelial system. This makes direct xenobiotic-mediated oxidant stress an unlikely cause of our patient's MetHb. Although RBCs are opsonized with IgG in piperacillin-associated DIIHA, the antibodies do not react during eluate testing. This produces a characteristic Coombs DAT+ and eluate negative test pattern. COHb up to 9.7% is reported with antibiotic-associated DIIHA. The suggested mechanism is inability to clear endogenous COHb. Similarly, our patient's uremia and low G6PD activity may have contributed to endogenous MetHb formation. Hemolyzed RBCs liberate heme and free iron. This labile iron pool is a known oxidant stress. Further, phagocytic cells possess NADPH oxidase and release superoxides during DIIHA cell lysis. Therefore, our patient may have been unable to cope with his multifactorial oxidative stress, resulting in elevated MetHb levels. The temporal link suggests Zosyn is the inciting agent.

Conclusions: We present the first case of combined MetHb and COHb secondary to Zosyn-associated DIIHA.

Keywords: Adverse drug event, Antibiotic, Hemolysis

249. A poison center retrospective review of duloxetine-exposed patients

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Background: Duloxetine is a serotonin norepinephrine reuptake inhibitor (SNRI) approved in the US for the treatment of major depression, generalized anxiety, fibromyalgia, diabetic peripheral neuropathy, and chronic musculoskeletal pain. Given the limited published information regarding human overdoses to this medication, our goal was to characterize such exposures.

Methods: We retrospectively reviewed a state poison system's database for all single agent exposures to duloxetine from 2004–2011. Data collected included age, gender, circumstances surrounding exposure, dose taken, symptoms, outcome, medical management and patient disposition.

Results: There were 159 cases identified. After exclusion for errors in coding, non recorded data, and loss to follow-up, 118 patients were included for review. Of the 61 pediatric and adolescent (0–19 years old) identified, 8 cases involved intentional overdose with a dose of 80–1200 mg when known. All intentional ingestions were seen in the emergency department with 7 cases coded as no effect, one minor effects and no patients requiring admission. Of the remaining 53 unintentional ingestions, 17 were seen in the emergency department with no hospitalizations required. All 53 patients had no effect recorded as outcome. See Table 1 for details.

Of 57 adult cases, 33 involved intentional ingestions with a dose of 100–2000 mg when known. Four of these patients were admitted to the hospital for an average of one day. There were 16 patients with no effect, 15 minor outcomes, 1 moderate outcome, and 1 major outcome. The remaining unintentional ingestions (24 patients) represented an exposure of 20–300 mg when known, 4 patients were seen in the ED with one hospitalization. Outcomes included 2 no effect, 22 minor effect. There was one death following unintentional ingestion of 60 mg however this was felt unlikely due to duloxetine ingestion as the patient had concomitant renal failure, recurrent episodes of hypoglycemia and elevated temperature combined with a relatively low ingested dose. See Table 1 for details.

Conclusions: Serious effects were rare following duloxetine ingestions of supratherapeutic doses in this case series. Gastrointestinal and mild neurological symptoms were the most frequent adverse effects seen.

Keywords: Antidepressant, Overdose, Duloxetine

Table 1. Results for abstract 249.

	Pediatric intentional (N = 8)	Pediatric unintentional (N = 53)	Adult intentional (N = 33)	Adult unintentional (N = 24)
Nausea	0	1	3	4
Vomitting	0	0	4	3
Abdominal pain	0	0	2	0
Diarrhea	0	0	1	2
Drowsiness	1	1	9	3
Agitation	1	0	1	4
Confusion	0	0	2	1
Dizziness	0	0	0	3
Seizure	0	0	2	0
Tremor	0	0	0	5
Headache	1	0	1	4
Hypertension (SBP > 150)	1	0	6	0
Hypotension (SBP < 90)	0	0	0	0
Tachycardia (HR > 110)	1	0	3	1
Bradycardia (HR < 60)	0	0	0	0
Diaphoresis	1	1	0	1
EKG Changes	0	0	0	0

250. The potential use of poison centre data for the vigilance of herbal medicinal products in Canada

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Background: Herbal medicinal products (HMPs) are widely used by consumers, and monitoring adverse reactions (ARs) to health products is key in their safety monitoring. Side effects to HMPs are not always reported to regulatory authorities, including Health Canada (HC). Poison centre (PC) data may complement the AR information currently submitted to HC. A collaborative study was conducted to investigate the nature and amount of data on HMPs collected by a Canadian PC and the potential usefulness of this information in the vigilance of these products.

Methods: The Canadian PC's electronic database was searched retrospectively from 2005–2009 for finished commercial HMPs, using American Association of Poison Control Center (AAPCC) codes. Symptomatic exposures, which included adverse reactions, were retrieved and analyzed. A comparison was also made between the type of information collected by the PC and that which is important for regulatory safety assessment.

Results: Of the total number of exposure calls received by the Canadian PC (58–76 calls per year), 20 were defined as "adverse reactions" according to AAPCC criteria (definition excludes cases of misuse/abuse). However, 70% of all symptomatic exposure calls met the minimum criteria for an AR report to HC (a patient, a reporter, a prod-

uct, and symptoms) and included reactions associated with abuse and misuse (included in the HC definition of "AR"). In addition, the call records contained information useful for safety/causality assessment. Specific product, ingredient, and manufacturer identification was not always possible; this could theoretically be improved through the collection of HC authorisation numbers for HMPs.

In total, 176 different HMPs were associated with symptomatic exposures to the Canadian PC between 2005–2009. Many products (43%) were associated with only a single exposure during this time period, while 15 products (9%) were associated with five or more. In some cases, the PC data confirmed known adverse reactions.

Conclusions: The data held in one Canadian PC database contains information from symptomatic exposure calls related to HMPs, including useful information for safety/causality assessment. Many of the symptomatic exposures meet the minimum AR reporting criteria for HC. Some gaps exist between what is collected for PC purposes, and what is ideal for regulatory assessment. These gaps are largely seen as resolvable; however discussions are required with PCs to determine the feasibility of collecting additional data given their primary role is the management of the poisoned patient.

Keywords: Adverse reactions, Herbal, Poison center

251. Boozy liver: A therapeutic misadventure with alcohol sclerotherapy

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Background: Symptomatic simple hepatic cysts are rare; however, if treatment is indicated, alcohol sclerotherapy has been reported to be beneficial. With this technique, the cyst is completely aspirated, and a volume of 95–99% ethanol 25–40% that of the liver cyst is instilled. The dwell time in a single session is typically between 20 and 60 minutes but large cysts may require more than one cycle. Complications of this procedure are usually minor and may include pain, nausea and vomiting, and transient increases in basal temperature. Elevated blood alcohol concentration (BAC) secondary to systemic absorption is another concern but is not often described. We report a case of a patient who became clinically intoxicated after receiving alcohol sclerotherapy.

Case report: A 79-year-old woman with a history of multiple large hepatic cysts associated with right upper quadrant pain underwent alcohol sclerotherapy. Using ultrasound guidance, the interventional radiologist drained 1500 mL of serous fluid from a cyst in the right hepatic lobe and subsequently instilled 100 mL of absolute ethanol. The procedure was complicated by the presence of an external leak in the drainage catheter. After the catheter was replaced, attempts to aspirate the instilled ethanol were unsuccessful because no fluid was found. A computed tomography (CT) scan of the abdomen was performed and revealed a small sliver of free fluid around the dome of the liver, and 10–15 mL of fluid was aspirated. While preparing to discharge the patient, she was noted to become lethargic and hypoxemic and developed slurred speech. Due to concern for airway compromise, she required close monitoring in the intensive care unit. Her BACs obtained 4.5, 6.5, and 17.5 hours post procedure were 187, 172, and less than 10 mg/dL respectively. She was discharged home the following day.

Case discussion: Because alcohol sclerotherapy is a relatively rare procedure, potential complications and defined observation periods are not well established. Clinical intoxication is infrequently anticipated because the instilled ethanol is aspirated after sufficient contact time with the cyst wall. However, systemic effects, including CNS and respiratory depression requiring intervention, may occur with this therapeutic modality. We postulate the delayed absorption and intoxication is secondary to intraperitoneal and/or transperitoneal absorption.

Conclusions: Healthcare providers should be cognizant of the potential systemic effects of alcohol sclerotherapy and ensure an adequate period of monitoring following the procedure.

Keywords: Ethanol, Intoxication, Hepatic cyst

252. Propofol extravasation: Tissue necrosis requiring muscle flaps and skin grafts

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Background: Cases of propofol extravasation are rarely reported. A PubMed search reveals 3 previously reported cases in 19 years. In one case, propofol leaked into the subcutaneous (SQ) tissues of a hand, with pain and swelling, but eventual full recovery, reported. Another case reports SQ extravasation of clear propofol, with erythema, severe pain, and hand swelling ensuing. This hand became tense and cyanotic. Incision, flushing, and drainage were instituted. This patient recovered without complication. Another case reports dorsal foot extravasation of propofol in an infant, associated with blister formation and tissue necrosis by day 2. Early wound debridement and delayed skin grafting were utilized. This was the only previous case to note tissue necrosis and the need for skin grafting. We report more severe injury.

Case report: A 62 yo man presented for scheduled hip surgery. Propofol (150 mg), fentanyl, midazolam and rocuronium where injected into a left hand IV. Induction failed to occur. Extravasation of medications was suspected. The hand was dressed. A new IV was placed. The surgical case was complicated by hemorrhagic shock. Postoperatively, his SBP was in the 70s. Blood, norepinephrine, and IV fluids were given.

The left hand dressing was removed 48 hours later. Deep-purple discoloration of the radial and ulnar hand was noted. (See photos) Areas of discoloration were in dependent areas, lateral to the IV site, not in the fingertips. Bleb formation was seen 72 hours after extravasation. The patient complained of pain with movement of the hand, but the hand was not tense. Necrosis of the underlying tissues was suspected. A hand surgeon was consulted.

Hand debridement was done 14 days later. Two areas were debrided (ulnar and radial hand). The first webspace and ulnar aspect of left hand were incised and drained. Dense, deep tissue necrosis was seen. Skin, SQ tissues, and muscle were debrided. An intrinsic muscle was freed and placed over an exposed 2nd metacarpal bone. A skin graft was placed. Next, the 5th metacarpal was exposed via debridement. A 5th dorsal metacarpal artery flap was harvested and placed. A skin graft was placed.

The next day, his BP fell to 69/48 mmHg. Dopamine was started. His cumulative problems included acute kidney injury,

shock liver, anasarca, metabolic acidosis, and encephalopathy. He was transferred to hospice for comfort care.
Discussion: Most previous reports and reviews suggest that propofol extravasation produces minimal tissue damage. Our case demonstrates otherwise.
Conclusions: Propofol extravasation may produce severe tissue necrosis requiring muscle flaps and skin grafting.
Keywords: Propofol, Extravasation, Tissue necrosis

253. Transdermal clonidine toxicity from a compounded cream

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Background: Clonidine is an α2-adrenergic and imidazoline receptor agonist used for chronic pain management. It is administered with local anesthetics, opioids, and other agents by a variety of methods including intrathecally, epidurally, and transdermally.
Case report: The PC was consulted about a 23 year-old man with altered mental status, bradycardia, and hypotension after he reportedly applied an unknown amount of his compounded pain cream to his entire body. The cream was reported to contain clonidine 0.2%, ketamine 10%, gabapentin 6%, imipramine 3%, lidocaine 2%, and mefenamic acid 1% (weight/weight) with 1 pump equaling 1 g of a 120 g container. Vital signs were HR 40s, BP unobtainable, RR 15, SpO2 99% on room air, and rectal temperature 95 F. He responded only to pain, with mydriasis and a rash on the neck and chest. ECG showed sinus bradycardia with HR 40, QRS 104 ms, QTc 481 ms. Electrolytes, renal and liver function tests were normal. Salicylate, APAP, and ethanol levels were undetectable. Urine drug screen was positive for TCAs, THC, and amphetamines. Recommendations included skin decontamination, fluid resuscitation, warming, atropine, and serum alkalinization. CT head revealed a moderate sized subarachnoid hemorrhage. He was intubated and transferred to a tertiary care hospital. No surgical interventions were performed. He made a full recovery. Initial serum had a clonidine level of 5200 ng/mL (therapeutic range 0.5–4.6 ng/mL), imipramine and desipramine 13 ng/mL (reference range 150–300 ng/mL), and undetectable lidocaine and monoethylglycinexylidide.
Case discussion: Clonidine is a centrally acting antihypertensive agent that decreases sympathetic outflow from the central nervous system to peripheral tissues. Toxicity may produce depressed mental status, hypotension or hypertension, bradycardia, respiratory depression, miosis, and hypothermia. Clonidine pharmacokinetic studies have demonstrated dose linearity with increased topical application. Our patient covered his entire body with a cream containing 0.2% clonidine and presented a few hours later with altered mental status, bradycardia, and hypotension. His serum clonidine level, 5200 ng/mL, is the highest published level by any route of exposure. A case involving a 3 year-old with ingestion of a clonidine suspension resulted in a serum level of 300 ng/mL 18 hours after exposure; an error in compounding was suspected to contribute to the toxicity. In this case, the cream was not available for testing so a compounding error cannot be ruled out.
Conclusions: Topically applied clonidine can result in serious systemic toxicity. It is unclear whether a compounding error, in addition to excessive use, resulted in this patient's toxicity.
Keywords: Clonidine, Dermal toxicity, Laboratory

254. Paraspinal compartment syndrome and acute kidney injury complicating MDPV ingestion

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A 37 year-old, 85 kg male with a history of a right nephrectomy due to trauma ingested an unknown quantity of "bath salts" approximately four hours prior to seeking medical care. Per the patient's report, he had no prior history of underlying abnormal renal function, although he had not been evaluated by a physician in several years. Following the ingestion, he described feeling "hot" and subsequently sat in a bathtub for approximately four hours. He was found agitated, combative, and brought to an emergency department (ED) where he was tachycardic (HR 153 beats per minute), tachypneic (RR 50 breaths per minute) and febrile (T39oC). Given the agitation, he was intubated to facilitate evaluation and for airway protection prior to transfer to a tertiary care medical center. Initial laboratory studies in the ED were notable for a sodium 165 mmol/L, chloride 114 mmol/L, CO2 18 mmol/L, BUN 49 mg/dL, creatinine 6.2 mg/dL (new), and an AST/ALT 863/514 IU/L. No CK was performed.

Upon arrival in the ICU, the patient's CK was 90,168 IU/L, with a troponin-I of 10.9 ng/mL. The patient was extubated, and complained of mild diffuse myalgias, but had soft thigh, calf, forearm, and paraspinal muscle compartments. Approximately 12 hours later, the CK was $>$350,000 IU/L, creatinine 5.1 mg/dL, and troponin-I 14.1 ng/mL. He had received 8545 cc of fluids, with a urine output of 345 cc over the preceding 24 hours (0.17 cc/kg/hr). His exam was notable for tenderness and firmness in the lumbar paraspinal region. He was started on hemodialysis, and was taken to the operating room (OR) where the deep paraspinous compartments of lumbar spine were noted to be tight. Necrotic muscle from the deep erectors were resected.

The patient was taken back to the OR two days later for re-exploration, where additional necrotic muscle was resected, and surgical bleeding was controlled. A comprehensive urine drug screen via GC-MS, which was obtained from the initial urine on admission, which confirmed the presence of caffeine, hydrocodone, methylenedioxypyrovalerone (MDPV), and propofol. Serum MDPV (via LC-MS/MS) 7 hours after first seeking medical care was 120 ng/mL, and 89 ng/mL 10 hours after seeking medical care.

Five months later, the patient remained in renal failure on hemodialysis.
Keywords: Bath salt, Compartment syndrome, Rhabdomyolysis

255. Tipsy toddler: A case of metronidazole-induced peripheral neuropathy

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Background: Metronidazole, a protozacide and bactericidal agent, is used in the treatment of inflammatory conditions, Clostridium difficile colitis (CDC), and bacterial vaginosis. Despite a relatively safe therapeutic profile and widespread clinical use, case reports documenting peripheral neuropathy after exposure to metronidazole exist. In most instances, the neuropathy develops after prolonged use and high doses. We present the case of a child who developed a painful peripheral neuropathy after receiving supratherapeutic doses of metronidazole for 1 week.
Case report: A 20 month-old girl presented to clinic for evaluation of painful hands and feet and an abnormal gait. Her symptoms began after she completed a 7-day course of metronidazole for treatment of CDC that occurred after receiving antibiotic therapy for streptococcal pharyngitis. Due to an iatrogenic dosing error using an electronic prescription writer, she received 120 mg/kg daily instead of the intended dose of 30 mg/kg/day. While receiving treatment, she experienced nausea and vomiting; however, these symptoms had resolved by the time of presentation. She subsequently developed an unsteady gait and would consistently point to her distal extremities and proclaim "owie." Treatment with folic acid was initiated (150 mcg/day). Over the next 2 weeks, her neuropathy resolved and she returned to baseline. At home, she had a mechanical fall, attributed to her abnormal gait, and sustained a head injury prompting an Emergency Department evaluation. No head imaging was obtained, and she suffered no long-term sequelae.
Case discussion: Due to the increasing use of electronic prescription writers, there is potential for iatrogenic errors. In this case, the prescribing physician intended to write for 30 mg/kg/day of metronidazole in 4 divided doses but inadvertently wrote for it to be administered 4 times daily. This child developed peripheral neuropathy after a relatively short course of treatment; this is in contrast to the majority of published case reports. It is suspected that peripheral axonal degeneration is the etiology, and while it is usually reversible with drug cessation, the administration of folic acid may shorten the time to resolution.
Conclusions: Healthcare providers should be aware of the potential for iatrogenic dosing errors when using electronic prescription writers and the development of peripheral neuropathy in patients receiving short courses of metronidazole. The mainstay of management is drug cessation but folic acid supplementation should be strongly considered.
Keywords: Antibiotic, Neuropathy, Pediatric

256. A case of Charles Bonnet syndrome possibly induced by esomeprazole

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Background: Psychiatrist Charles Bonnet reported visual hallucinations described by his blind grandfather in 1769. Patients with Charles Bonnet Syndrome (CBS) have normal consciousness and insight and their hallucination is not accompanied by sound. CBS is commonly associated with senile macular degeneration and glaucoma.
Case report: 84 yo male presented to the ED complaining of vivid visual hallucinations that started suddenly while riding in a taxi. He described a blue beard growing from the ED staffs' faces and flowers silently blooming out of the air. He was aware that all his bizarre images were not real. The patient was not confused or disoriented.His wife denied noticing any change in his personality. The patient had recently been hospitalized for bleeding esophagitis and GE junction ulcers that required transfusion, discontinuation of warfarin and initiation of esomeprazole while in the hospital. Esomerazole was changed from IV to oral formulation. The hallucinations started in the taxi ride home from the hospital. PMHx includes macular degeneration, Afib and atrial and mitral valve replacements. In the ED, the patient had normal vital signs. PE was insignificant except irregular cardiac rhythm noted to be atrial fibrillation at a controlled rate. He was alert and oriented, with no focal neurological deficit. Brain CT showed no disease and basic labs were unchanged from the time of previous discharge. The patient was re-admitted for observation. Neurology and psychiatry were consulted and psychiatry suggested the diagnosis of Charles Bonnet syndrome. Esomeprazole was replaced by pantoprazole and the hallucinations gradually subsided and disappeared. Patient was readmitted a few more times for the next several months for his cardiac conditions, but hallucination was never reported.
Case discussion: In 2009 an Italian pharmacovigilance system reported 'psychiatric adverse reactions' related to proton pump inhibitor (PPI) medications. Esomeprazole was the most common PPI associated with those cases. Administration of PPIs increases serum gastrin level. Gastrin-induced peptide receptors exist in the brain and can be affected by PPIs to cause neuropsychiatric symptoms. Our patient demonstrated many of the classic characteristics of Charles Bonnet Syndrome including macular degeneration, absence of any previous history of hallucination, rapid onset of hallucination temporally related to exposure to esomeprazole and remission of the hallucinations with discontinuation of esomeprazole. It is plausible to associate his hallucinations with his exposure to esomeprazole.
Conclusions: This is a case report of Charles Bonnet syndrome possibly associated with the use of esomeprazole.
Keywords: Adverse drug event, Charles Bonnet syndrome, Esomeprazole

257. Adverse events from misuse of clenbuterol for weight loss and body building

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Background: Clenbuterol is a b2-adrenergic agonist approved in the US for veterinary use in non-food animals. In addition clenbuterol may have anabolic and lipolytic activity. Animal studies have attributed these effects to activity at b3-receptors. A trend of clenbuterol use is emerging among body builders and fitness enthusiasts attracted to the hypertrophic and lipolytic effects.
Methods: Retrospective chart review of clenbuterol exposures reported to the Kentucky Regional Poison Control Center involving clenbuterol. Clenbuterol was analyzed via tandem HPLC/MS
Results: Misuse was reported in 5 of 7 clenbuterol users: all male, mean age 25.2 years. Two remaining exposures were accidental: during veterinary use and an infant's ingestion of a mother's clenbuterol obtained for weight loss. Clenbuterol source among misusers, when known, was from internet purchase (n = 2). Weight loss/body image was cited as reason of the 5 misuse patients.

Ingested dose (n = 3) ranged from 300–4500 mcg [mean = 2600 mcg]. Reported clinical effects included tachycardia [n = 4, max HR 118–170], widened pulse pressure [n = 4, 65–77 mmHg], tachypnea [n = 3, 20–44 bpm], hypokalemia [n = 2, 2.7 mEq/L], hyperglycemia [n = 3, max BG 159–334 mg/dL], ST changes on EKG (n = 2), elevated troponin [n = 1, 10.4], elevated CPK [n = 2, 256–1549 IU/L], palpitations (n = 2), chest pain (n = 1), and tremor (n = 1). Measured serum clenbuterol concentration (n = 1) was 2983 pg/ml post 4.5 mg ingestion. Coingestants (n = 2) included T3 (7.5 pg/mL) and anabolic steroids Two patients were treated and released from the ED while three were admitted to inpatient service. Treatments included activated charcoal (n = 1), benzodiazepines (n = 2), beta-blockers (n = 3), potassium replacement (n = 2), and IV fluid (n = 3). Duration of clinical effect exceeded 24 hours in 3 of 5 patients.

Discussion: There is an increasing use of the internet for illicit drug use for body-building and weight loss purposes, with widespread use of these drugs within the body building community without full understanding of the risks. These patients may not view use as drug abuse or present as the stereotype of illicit drug abusers, but may present as healthy athletic low-risk patients. Clinical effects persisted greater than 24-hours with evidence of myocardial injury in 2 patients. Ingested doses may be enormous and vary widely based upon anecdotal advice within the body building community. Co-abused drugs may include anabolic steroids, estrogen antagonists, thyroid hormones, and caffeine.

Conclusions: Clenbuterol is increasingly being abused within the body-building subculture. An increasing number of patients are presenting to emergency departments with adverse or overdose effects

Keywords: Abuse, Adverse drug event, Beta agonist

258. Corticosteroids continue to be inappropriately used in the management of NAC related anaphylactoid reactions

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Background: Intravenous (IV) N-acetylcysteine (NAC) is the antidote of choice for acetaminophen (paracetamol) poisoning in the UK and recently has been more widely accepted in IV form in the US. Previous reports have suggested anaphylactoid-like adverse reactions (ADR) develop in 3–50% of patients who receive IV NAC. Risk factors identified include past history of allergy/asthma and low paracetamol concentration. There is controversy over the appropriate management of these reactions particularly over the role of steroids and histamine-2 receptor antagonists.

Methods: We conducted a retrospective review of all cases where there was documentation of an ADR to IV NAC in patients where it was being used for acetaminophen poisoning (February 2005–June 2011). Data was extracted from hospital records on the timing of the reaction in relation to NAC treatment, clinical features seen and management.

Results: A total of 82 patients identified. 59 patients had complete case records available for review, of which 34 (58%) were female, 25 (42%) were male. 12 (20%) had a history of asthma. The ADR occurred during the 15 minute (150 mg/kg) infusion in 36 (61%) cases, 22 (37%) in the 4 hour (50 mg/kg) infusion and 1 (2%) in the 16 hour (100 mg/kg) infusion. The time from NAC initiation to ADR onset was 0 to 122 minutes (median 32.5 minutes). Clinical features seen included: nausea (n = 19, 32.2%), vomiting (23, 39.0%), abdominal pain (2, 3.4%), flushing (15, 25.4%), pruritis (12, 20.3%), urticaria (16, 27.1%), angioedema (7, 11.9%) breathlessness (14, 23.7%), respiratory distress (1, 1.7%), bronchospasm (6, 10.2%), hypotension (1, 1.7%), tachycardia (6, 10.2%), chest pain (7, 11.9%), headache (1, 1.7%), and paraesthesiae (3, 5.1%). Management included administration of anti-emetics (n = 36, 61%), histamine H-1 antagonists (26, 44%), corticosteroids (16, 27%), inhaled beta-2 agonists (6, 10%) and adrenaline (3, 5%); no patients were treated with an histamine H-2 antagonist.

Conclusions: The clinical features seen in this series were similar to those in previously published series. However, management varied widely – this is likely to be due to poor understanding of the mechanisms involved. In particular 27% of cases were treated with corticosteroids which have no role in the management of NAC ADRs. Clinical Toxicologists need to ensure that ED physicians are aware of the mechanisms and management of NAC ADRs.

Keywords: Acetaminophen (paracetamol), Adverse drug event, N-acetylcysteine

259. Muscle relaxant adverse effects seen in medical toxicology practices – Data from the ToxIC Case Registry

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Background: Muscle relaxants are primarily centrally acting medications used to treat muscle spasm. The two most popular muscle relaxants are cyclobenzaprine and carisoprodol, with approximately 22 and 11 million prescriptions dispensed in 2011, respectively. While these muscle relaxants pose a health risk, a description of their use and side effects have not been previously evaluated and systematically reported by toxicologists. Objectives: This study compares reported use and adverse effects of different muscle relaxants as reported by consulting medical toxicologists.

Methods: The American College of Medical Toxicology ToxIC Case Registry, from its inception in 2010, was reviewed for cases involving muscle relaxants. Descriptive statistics were used to evaluate single exposure cases involving this class of medications.

Results: Of the 275 ToxIC Case Registry entries involving muscle relaxants, 62 were for single exposures, most frequently involving cases for carisoprodol (52%) or cyclobenzaprine (39%). The genders were equally represented with 52% of cases involving females. The majority of exposures were in adults (82%), with less minors (16%), and few adults over age 65 (2%). Most cases were due to intentional pharmaceutical overdoses (74%), while only 13% of cases were for unintentional pharmaceutical overdoses. Among serious adverse events was coma/CNS depression, which was reported to have occurred in 32% of cases, due to cyclobenzaprine and carisoprodol equally. Respiratory failure requiring mechanical ventilation was noted in twice as many cases for carisoprodol than cyclobenzaprine. Both medications were associated with rhabdomyolysis and delirium. However, although rare, acute coronary syndrome, aspiration pneumonitis, and death were only reported with cyclobenzaprine.

Conclusions: Although cyclobenzaprine was prescribed significantly more often than carisoprodol, certain side effects were reported more frequently with carisoprodol. Similarly, the American Association of Poison Control Centers' National Poison Data System Annual Report for 2010 shows 35% more single exposures for cyclobenzaprine then carisoprodol but 3% fewer Major Outcomes. This raises concern for greater toxicity of carisoprodol. This is evident in the DEA's recent change of carisoprodol to a schedule IV drug in February 2012. However, cyclobenzaprine also has potential for adverse effects and should not be looked upon as a harmless alternative to carisoprodol, especially in light of widespread usage of the drug.
Keywords: Muscle relaxant, Carisoprodol, Cyclobenzaprine

260. Profound hypothermia induced by paliperidone

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Background: Hyperthermia related to antipsychotic drug use is a well-known adverse effect, especially in relation with neuroleptic malignant syndrome. A lesser known adverse effect, hypothermia, may also occur. We present a case of profound hypothermia induced twice following administration of the appropriate dose of paliperidone.
Case report: A 48 year old female with a history of ruptured cerebral arteriovenous malformation presented with new onset altered mental status. She developed increasing lethargy over the previous 2 weeks, which culminated in sleep during 30 continuous hours and inability to attend her activities of daily living. She presented alert only to person with incoherent speech and miotic pupils. Vitals: pulse 46/min; BP 86/51 mmHg; resp 12/min; temp 29.9ºC via bladder probe. She had no exposure to cold. Abnormal laboratory data: platelets 53,000/mm^3, PTT 55.6 s, alk phos 206 U/L, AST 168 U/L, ALT 357 U/L. EKG had sinus bradycardia with rate of 40/min and Osborn waves. Head CT and brain MRI showed no acute changes. Endocrinology studies were significant only for hyperprolactinemia of 155.7 ng/mL. Further history revealed that paliperidone had been introduced for behavioral disturbances 9 weeks prior and increased from 3 to 6 mg daily, three weeks after initiation. Patient was passively re-warmed and by day 10 of hospitalization, the temperature was stable and thrombocytopenia and transaminitis had resolved. She was discharged to a nursing facility on day 11, but, mistakenly, paliperidone was restarted. Patient presented 2 days later with altered mental status with pulse 59/min, BP 90/55 mmHg, resp 14/min and temp 34.3ºC via bladder probe. Laboratory data: AST 75 U/L, ALT 122 U/L, alk phos 226 U/L. Patient was passively re-warmed and discharged from hospital 4 days later with return to baseline function.
Discussion: The Naranjo Causality Scale was used to estimate the probability that paliperidone caused the adverse clinical event of hypothermia in this case, with a total score of 7 indicating probable adverse drug reaction. Review of the literature revealed no case of paliperidone-induced hypothermia. However numerous reports are attributed to risperidone; paliperidone is the primary active metabolite of risperidone. Proposed mechanisms for hypothermia include 5-HT2A receptor antagonism and peripheral alpha2-adrenergic blockade. There is an increased susceptibility to hypothermic effects induced by the atypical antipsychotics in those with pre-existing brain damage.
Conclusions: Paliperidone can induce a hypothermic crisis. Recognition of antipsychotic drugs that may cause hypothermia is essential, as management of the condition requires a prompt discontinuation of the offending agent.
Keywords: Hypothermia, Antipsychotic, Adverse drug event

261. Effect of acute aluminum phosphide exposure on rats – a histological and biochemical correlation

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Background: Aluminum phosphide (ALP), a widely used fumigant and rodenticide leads to high mortality if ingested. Its toxicity is due to phosphine which is liberated when it comes in contact with moisture. The exact mechanism of action of phosphine is not known, though, it is widely believed that it inhibits mitochondrial oxidative phosphorylation.
Methods: In this study male albino Wistar rats, weighing 150–200 g were procured from the institute animal house and divided into two groups (ALP group and Control). Animals were housed in polypropylene cages and fed on a standard diet and water ad libitum. Ethical clearance for killing of animals was duly obtained from the institute's animal ethics committee. The animals were subjected to overnight fast. On the day of experiment, ALP group - received a single LD100 dose of ALP (20 mg/kg) powdered, weighed and stuffed inside moist wheat flour pellets, orally. Control group - received an equal volume of plain wheat flour pellet. After administration, animals were observed for a period of 12 hours or till death, whichever occurs earlier. Control animals were sacrificed by cervical dislocation. Basic serum biochemical parameters, activity of mitochondrial complexes, antioxidant enzymes and parameters of oxidative stress, individual mitochondrial cytochrome levels were measured along with tissue histopathology and immunostaining for cytochrome c.
Results: The serum levels of creatinine kinase-MB and lactate dehydrogenase were higher whereas as the activities of mitochondrial complexes I, II, IV were observed to be significantly decreased in liver tissue in ALP group rats. The antioxidant enzyme activity of catalase was lower with a significant increase in lipid peroxidation whereas superoxide dismutase and glutathione peroxidase were unaffected in them. There was also a significant increase in tissue lactate dehydrogenase activity. Histopathology revealed congestion in most organs with centrizonal hemorrhagic necrosis in liver. There was a significant decrease in all the cytochromes in brain and liver tissues with the exception of cytochrome b in brain, the levels of which remained same. There was marked reduction in the cytochrome c staining in ALP group as compared to the controls.
Conclusions: Following ALP exposure, not only cytochrome c oxidase but other complexes involved in mitochondrial electron transport and enzymes are also inhibited, in particular the heme based ones.
Keywords: Medical toxicology, Rodenticide, Cardiac toxicity

262. Evaluation of crotalidae polyvalent fab antivenom for the treatment of Middle Eastern and South Asian Viperidae envenomation in a murine model (Mus musculus)

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Background: Worldwide, snakebite is the single most important cause of human injury from venomous and poisonous animals, with an estimated 2.5 million bites per year and approximately 125,000 deaths each year. With ongoing conflicts throughout the Middle East involving hundreds of thousands of US forces, our military population is at greater risk for exotic snakebite due to frequent worldwide deployments and harsh living conditions. Although foreign antivenom exists, it is expensive, difficult for rapidly deploying units to acquire, they lack documented safety profiles and military providers are generally unfamiliar with their use. Based on in-vitro and anecdotal evidence suggesting that cross-neutralization between Viperidae and Crotalidae antivenoms exists, we performed a blinded, randomized, placebo controlled study comparing the ability of Croatlidae polyvalent immune Fab Antivenom (CrofabTM) to decrease lethality, from intraperitoneal injection of preincubated Viperidae venom and CrofabTM, by 50%, compared to controls injected with preincubated venom and saline only, in a murine model.
Methods: LD50 (median lethal dose) concentrations for vemon from the following snake species were determined using a standard method:

Gloydius halys caucasicus
Macrovipera lebetina cernovi
Cerastes cerastes cerastes
Pseudocerastes persicus
Echis carinatus sochureki

The lethality of each venom was investigated using sham control (antivenom only), lethal control (venom only) and study (venom + antivenom) groups for each venom. Venom (2LD50) and/or antivenin (enough to neutralize 10LD50) were pre-incubated with enough normal saline to equal a total of 1.5 milliliter injection volume. Mixtures were warmed to 37 degrees Celcius then injected intraperitoneally. Mice were observed 24 hours for survival.
Results: See Table 1.
Conclusions: The lethality of simulated envenomation using intraperitoneal injection, for 4 Middle Eastern viper venoms, was significantly reduced using species non-specific antivenom (CrofabTM) in this murine model. We suspect our LD50 determination for the Pseudocerastes venom was inaccurate. Future research using a similar design comparing species nonspecific Crotalidae-derived venom to species-specific Viperidae-derived venom is underway.
Keywords: Antivenom, Snake bite, Venom

Table 1. Results for abstract 262.

	#	Died	Lived	Lethality
G halys				
AV only	6	0	6	0%
venom only	22	15	7	68%
AV + venom	22	0	22	0%
M lebetina				
AV only	6	0	6	0%
venom only	22	16	6	73%
AV + venom	22	4	18	18%
C cerastes				
AV only	6	0	6	0%
venom only	22	21	1	95%
AV + venom	22	1	21	5%
P persicus				
AV only	4	0	4	0%
venom only	22	0	22	0%
AV + venom	22	0	22	0%
E carinatus				
AV only	0	0	0	0%
venom only	22	21	1	95%
AV + venom	22	2	20	9%

263. Polyethylene glycosylation prolongs the stability of recombinant human paraoxonase-1

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Paraoxonase-1 (PON1) is a native enzyme that is synthesized in the liver and is capable of hydrolyzing organophosphates (OPs). It is regarded as part of a promising approach for the pretreatment and therapy of OP poisoning. Previous experiments with purified rabbit serum PON1 have established that it can protect rats against many OP exposures. In the current paper, we described a preparation of active recombinant human PON1 (rHuPON1) by engineering an Escherichia coli expression system. Recombinant HuPON1 was purified by Ni-NTA affinity chromatography followed by DEAE sepharose fast-flow chromatography. After purification, rHuPON1 was chemically modified with polyethyleneglycol (PEG)-20K. Recombinant HuPON1 exhibited a mean residence time (MRT) of 8.9 h, which was threefold shorter than that of native HuPON1 in rats. However, rHuPON1 chemically modified with PEG-20K displayed an MRT of 19.5 h, suggesting that PEG modification can prolong the circulatory stability of rHuPON1. PEG-rHuPON1 had a catalytic efficiency sufficient in protecting rats against OP poisoning, as measured by acetylcholinesterase activity in tissues and signs after poisoning.
Keywords: Organophosphate, Pharmacokinetics, Environmental

264. Metabolites and potential metabolic pathways for the novel psychoactive substance methoxetamine

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Background: Methoxetamine is a ketamine analogue increasingly being used as a recreational drug in Europe and the US during 2011/2. Small case series suggest acute methoxetamine toxicity includes cerebellar and sympathomimetic features in addition to ketamine-like hallucinogenic and dissociative features. Currently there is no published data on the pharmacology of methoxetamine.

The aim of this study was to determine potential methoxetamine metabolites in urine samples from 3 patients with analytically confirmed lone acute methoxetamine toxicity.

Methods: Patient urine samples and blank urine were diluted with 1:4 with ultrapure water. These were analysed on an Accela UPLC system interfaced to a LTQ Orbitrap Discovery operating 30,000 mass resolution. Chromatographic separation was performed on a Waters Atlantis T3 C18 column using a water/acetonitrile 0.1% acetic acid gradient. Data were acquired in full scan mode over a mass range of 50–650 Da. Components identified by accurate mass as possible metabolites were investigated further by positive-ion LCMS/MS using the LTQ and Orbitrap. Postulated metabolites were based on the known phase 1 metabolism of ketamine. Accurate masses for these possible methoxetamine metabolites were generated and used for data interpretation.

Results: The presence of methoxetamine was confirmed and responses for all postulated metabolites were seen: N-Desethyl (nor), Dehydro, Dehydro-nor, Hydroxy, Hydroxy-nor, Hydroxy-dehydro and Hydroxy-nor- dehydro metabolites. Greatest responses were seen for the N-Desethyl (nor) and Hydroxy-nor metablites (response relative to methoxetamine of 38.3 and 13.3%). Further data interrogation using data file comparison software highlighted additional metabolites (e.g. O-desmethyl, Dihydro-nor, O-desmethyl-hydroxy-nor and O-desmethyl-dehydro metabolites) produced through O demethylation and hydrogenation of the ketone function.

Conclusions: In this analysis of urine specimens from three patients with acute methoxetamine toxicity we determined the presence of a number of Phase I metabolites of methoxetamine. In addition to expected metabolites based on the metabolism of ketamine, we detected additional metabolites produced through O-demethylation and hydrogenation. Further work is required to determine the potential for both acute and chronic toxicity associated with these methoxetamine metabolites.

Keywords: Substance abuse, Pharmacokinetics, methoxetamine

265. Ammonium nitrate cold pack ingestion presenting with a negative anion gap

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Background: Large, negative anion gaps are uncommonly encountered in clinical practice. Laboratory error is the most frequent cause of a negative anion gap; other causes include bromide or iodide intoxication, and multiple myeloma. Ammonium nitrate cold pack ingestion is an infrequent human poisoning, with one prior case series of five patients published.

Case report: A 45 year-old man presented to the emergency department after a suicide attempt in which he ingested the contents of a Medline cold pack containing ammonium nitrate crystals. He also ingested an unknown amount of spray deodorant containing 10% aluminum chlorohydrate. He vomited a short time after the ingestion. Initial vital signs were T 97.8 F, heart rate 97, blood pressure 147/102, respiratory rate 16, and oxygen saturation of 96% on room air. Serial serum chemistries were obtained using a Siemens Advia 1800 analyzer (Table 1). Serum bromide and iodide were undetectable by ion chromatography and total protein was 7.2 g/dL (normal 6–8.5 g/dL). Serum nitrate by ion chromatography was 1100 mcg/mL (normal 0.6–5.7 mcg/mL). The patient was treated with intravenous fluids, electrolyte replacement and bicarbonate infusion. He was discharged on day 4 with a normal chloride of 110 mmol/L.

Table 1. Data for abstract 265.

Time	Na (mmol/L)	Cl (mmol/L)	HCO_3 (mmol/L)	Anion gap
Day 1 17:05	141	154	16	−29
Day 1 19:30	141	164	14	−37
Day 2 03:20	141	157	11	−27
Day 2 11:00	140	153	16	−29
Day 3 07:00	140	118	26	−4

Case discussion: A prior case series of ammonium nitrate cold pack ingestion describes findings that include gastrointestinal (GI) upset, metabolic acidosis, diuresis, vasodilation, and methemoglobinemia. Our patient had vomiting, metabolic acidosis, and a large negative anion gap. His acidosis likely resulted from ammonium ion conversion to urea in the liver, a process that generates hydrogen ions and dissipates bicarbonate. The Siemens chemistry analyzer uses a chloride ion-selective electrode that is known to have interferences from halide ions, however our patient had negative halide ion assays. Aluminum chlorohydrate is a polymer and unlikely to be well absorbed from the GI tract, hence it probably contributed little to elevated chloride levels. Therefore, we suggest the nitrate ions caused interference resulting in a falsely elevated chloride level with corresponding negative anion gap.

Conclusions: This case of ammonium nitrate ingestion resulting in a large negative anion gap suggests possible nitrate interference on an automated chloride ion-selective electrode-based chemistry analyzer. If a negative anion gap is encountered in the setting of ingestion and a high chloride concentration, halide and nitrate interference with chemistry analyzers should be considered.

Keywords: Laboratory, Ammonium nitrate, Negative anion gap

266. Analytical interferences resulting from intravenous lipid emulsion

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Objectives: Lipid resuscitation therapy (LRT) using intravenous lipid emulsion (IVLE) for drug overdoses has gained widespread use. However, there is little information regarding its adverse effects. Dosing protocols supported by the American College of Medical Toxicology call for 1.5 mL/kg of IVLE as a bolus (repeatable), followed by infusion of 0.25 mL/kg/min. For a 70kg individual, this would be in excess of 230 g of lipids over the course of 1 hour (compared to typical infusions of 4 g/hr in parenteral nutrition therapy). Kinetic studies of lipid clearance have described a maximal clearance of 6 mmol/L/hr. With a typical plasma volume of 3 L, we would expect triglyceride (TG) levels of approximately 80 mmol/L after 1 hour of treatment. Lipemic interference indexes generally go up to 4 g/L of IVLE , far below the levels attained in LRT. We performed lipemic interference studies on typical automated platforms to investigate the potential of LRT to interfere with the reliability and turn around time of analytes that would

be of interest in acute intoxications. We also tested methods to minimize interferences.

Methods: Serum pools were spiked with increasing concentrations of Intralipid-20% (0–30%). Analyses were performed on Beckman-Coulter DXC800 and DXI and Roche Modular-P. Analytes demonstrating $>5\%$ interference were re-measured after dilution or centrifugation (14000×g for 10 minutes).

Results: TG and glycerol-blanked TG concentrations were similar in IVLE-free samples. However, with addition of IVLE, concentrations were markedly different (139 vs. 76 mmol/L).

The following demonstrated significant interference: albumin (150% of expected), lipase (162%) and Mg (344%). Amylase, creatinine, PO4, total protein, ALT, CK and bilirubin became unmeasurable in IVLE-spiked samples.

There was no appreciable interference on the troponin-I, Na, K, Cl, Ca, CO_2, urea or CRP assays.

Whereas glucose measurement by potentiometry was free of interference, colorimetric methodology was error prone (500%).

Diluting with saline or water was of no benefit. Centrifugation removed $>90\%$ of glycerol-blanked TG (max = 5.8 mmol/L), dramatically reducing lipid interferences.

Conclusions: IVLE results in appreciable analytical interferences at concentrations demonstrated in LRT. Of particular concern is the marked interference on glucose and Mg, which may result in disastrous interventions. These interferences can be minimized by brief centrifugation at relatively low speeds on equipment readily available in most core labs. Of note, Intralipid contains 2.25% glycerol, and will cause falsely elevated TG results if a glycerol-blanking method is not used.

Keywords: Laboratory, Lipid therapy, Interference

267. Chronic methoxetamine exposure in a mouse model demonstrates that methoxetamine is not a "bladder friendly" alternative to ketamine

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Background: There is increasing evidence that chronic ketamine use is associated with significant lower urinary tract symptoms including increased frequency of small volume micturition, dysuria, supra-pubic pain and painful micturition; bladder biospies demonstrate cystitis with urothelial ulceration and eosinophilic ulceration. The novel psychoactive substance Methoxetamine, an arylcyclohexylamine derivative of ketamine, is currently marketed to recreational drug users as a "bladder friendly alternative to ketamine". There is currently no evidence to support this statement.

Methods: A validated mouse model used by our group to demonstrate chronic lower urinary tract pathology associated with chronic ketamine exposure was used. Two month old Institute of Cancer Research (ICR) mice were administered either 30 mg/kg of methoxetamine per day (5 mice) or saline control (7 mice) by intraperitoneal injection for a period of three months. On completion of the three months, the animals were sacrificed and the structures of the urinary tract were removed for macroscopic examination and histological analysis. This study was reviewed and approved by the local equivalent of an institutional review board (IRB).

Results: *Kidney:* There was hydropic degeneration in both the proximal and distal convoluted tubules and spotty infiltration with inflammatory cells in the kidneys of all of the five mice exposed to methoxetamine. In addition, there was glomerular atrophy in three of the methoxetamine treated mice. *Bladder:* All of the methoxetamine treated mice had random infiltration of mononuclear inflammatory cells in the submucosal layer and in the muscle layer of bladder. Animals treated with saline control showed none of the kidney or bladder features described above.

Conclusions: This mouse model of chronic methoxetamine exposure has demonstrated significant histological changes in both the kidney and bladder developing after three months of daily exposure to methoxetamine. This suggests that chronic use of methoxetamine in humans is likely to be associated with the same lower urinary tract symptoms that have been described for chronic ketamine use. It is important that animal models such as this are developed to allow rapid screening of novel psychoactive substances in an attempt to determine the potential for chronic toxicity associated with the use of these substances.

Keywords: Substance abuse, Renal toxicity, Methoxetamine

268. The effects of L-Carnitine in a murine model with a higher degree of verapamil toxicity

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Objective: L-Carnitine (CAR) is an amino-acid derivative that plays a key role in the energy utilization of free fatty acids by cardiac and skeletal muscle. In the setting of shock states such as verapamil (VER) toxicity, it has been theorized that the heart shifts away from fatty acid utilization for energy. Our prior work showed that CAR improved survival and hemodynamic parameters in a murine model of VER toxicity. Following up on that, we wished to determine if CAR was beneficial in a model with a higher degree of VER toxicity. The primary objective was to determine if CAR increases survival in a model with a higher degree of VER toxicity. The secondary objectives were to determine CAR's effect on hemodynamic parameters.

Methods: This was a controlled, blinded, randomized animal study utilizing 20 male Sprague-Dawley rats. Each animal received general anesthesia, active ventilation and instrumentation to allow for recording of heart rate (HR) and mean arterial pressures (MAP). For the testing phase animals were divided into two groups, CAR and normal saline. To induce toxicity all animals received a constant infusion of 10 mg/kg/hr of VER at time zero. Five minutes after commencement of the initial infusion, animals were randomized to receive either 50 mg/kg CAR or an equal volume of normal saline (NS). The experiment concluded with the development of pulseless electrical activity or asystole or at the end of a 150 minute observation period. Data was analyzed using Kaplan Meier survival analysis and ANOVA.

Results: The median survival for the CAR group was 70.64 minutes (95% CI 45.83–95.47) while the median survival for the NS group was 71.90 minutes (95% CI 47.16–96.64), (log rank $p = 0.767$). One animal in the CAR group and 2 animals in the NS group survived to the end of the 150 minute observation period. There were no significant differences in HR or MAP at measured time points up to 25 minutes after the start of the verapamil infusion.

Conclusions: Carnitine did not improve survival or hemodynamics in this model with a higher degree of verapamil toxicity.

Keywords: Carnitine, Calcium channel blocker, Verapamil

269. Contribution of serum ethanol concentration to the osmol gap: A prospective volunteer study

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Background: The contribution of ethanol (EtOH) to the osmol gap (OG) is commonly described by [EtOH (mg/dL)]/*k*, where *k* is assumed to be 4.6 (one-tenth of its molecular weight) if ethanol behaves ideally in solution. However, several studies on convenience samples of intoxicated patients presenting to the emergency department suggest that ethanol does not behave ideally and that *k* may be as low as 3.7.
Objectives: To determine prospectively the relationship between serum ethanol concentration and total serum osmolality in a group of healthy volunteers.
Methods: A total of 10 volunteers (5 men and 5 women) participated in the study. Eight subjects (4 men, 4 women) ingested 20 mL of 100% ethanol diluted in sugar-free soda at a rate of 1 drink every 10 minutes, up to a maximum of 8 drinks. The 2 controls ingested 20 mL of water diluted in sugar-free soda at the same rate. Blood samples were obtained at baseline and then every 20 minutes for 180 minutes to measure serum [EtOH], electrolytes, glucose, and osmolality (via freezing-point depression). Predicted osmolality was calculated using the formula: 2*[Na] + [BUN]/2.8 + [Glu]/18. OG was determined by subtracting calculated osmolality from measured osmolality. The OG was divided by [EtOH] to determine the coefficient of ethanol's contribution to total serum osmolality.
Results: Baseline OG among all subjects was variable (mean 2.84, median 4.71, IQR 0.47–5.38). OG also varied within the 2 controls over 180 minutes (range, −0.2 to 10.4). Mean peak [EtOH] was 229 mg/dL (median 223.5 mg/dL, IQR 171–273 mg/dL). There was a linear relationship between [EtOH] and OG (Pearson coefficient of 0.99). Assuming a baseline osmol gap of zero, *k* was calculated to be 3.98 (95% CI, 3.89–4.06) averaged over all participants.
Conclusions: Our data demonstrate that the coefficient describing the contribution of ethanol to serum osmolality (*k*) is 3.98. This indicates that ethanol contributes more to total serum osmolality than predicted for an ideal solute.
Keywords: Alcohol, Laboratory, Osmol gap

270. CYP drug interactions in overdose: How simulation software helps to determine the severity of poisoning

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Background: CYP enzymes play a major role in the phase I metabolism of many xenobiotics. Their contribution to adverse events and interactions in therapeutic drug use has been extensively studied, but their significance in drug interactions in overdose, and to what extent they influence the severity of poisoning, is largely unknown. Antidepressants and antipsychotics are often involved in multiple drug overdose situations, and many of these drugs are metabolized by the same CYP enzymes. In this study the frequency of such multi-drug intoxications is analyzed. In addition, simulations of drug interactions involving several antidepressants metabolized through CYP2D6 (as an example), are carried out to quantify the changes in blood concentrations of these drugs.
Methods: From 2009–2011 all multi-drug overdoses reported to the Poisons Information Center were analyzed. All consecutive cases with at least one antidepressant or antipsychotic agent in combination with other drugs metabolized by CYP enzymes were included. The ingested compounds were categorized as substrate, inhibitor and/or inducer for the CYP enzymes. Simulations were performed using the SIMCYP software in a normal adult population. The simulated ingested doses were according to dose ranges that would cause a mild, moderate or severe poisoning in a single drug overdose.
Results: In 3 years time, a total of 56.657 patients with a drug overdose were identified; 9.456 patients ingested an antidepressant, and 7.082 patients an antipsychotic drug. Among those patients, 5.868 in the antidepressant group and 4.521 in the antipsychotic group ingested multiple drugs (N = 10.389, overlap in 2.011 patients). Interactions on CYP level were recorded in 4.671 out of 8.378 patients (56%). The order of involvement of CYP enzymes was CYP3A4 (45%), CYP2D6 (34%), CYP2C19 (13%), CYP1A2 (8%), CYP2C9 (3%), CYP2E1 (3%), CYP2B6 (0.1%), CYP2C8 (0.01%). Simulations show that drug-drug interactions can be significant. For instance, the peak concentration of imipramine (single dose 5 mg/kg) is increased by 63% when combined with fluoxetine (relative strong inhibitor, single dose, 1.4 mg/kg). With an increase in the fluoxetine dose to 25 mg/kg, the peak serum concentration of imipramine is increased by 146%.
Conclusions: The number of drug interactions through CYP enzymes in overdose is potentially high. The use of dose-effect relations for individual drugs may well lead to underestimation of the severity of the poisoning. Population-based simulators like SIMCYP are useful to simulate interactions in case of multi-drug intoxications, particularly in situations in which several metabolism pathways exist.
Keywords: Drug interactions, CYP enzymes, Antidepressant

271. Detection of acetaminophen-protein adducts in decedents with opiate-acetaminophen combination product overdose

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Background/Objectives: A biomarker of acetaminophen-protein adducts (APAP-CYS) is measurable in serum from patients taking acetaminophen (APAP). Serum APAP-CYS concentrations >1 μM have been associated with APAP toxicity. This serum biomarker assay

was used to determine if persons who die of suspected opiate-APAP combination product overdose have measurable serum concentrations of the APAP-CYS biomarker and whether there are differences in serum concentration between heart and femoral samples.
Methods: The State Medical Examiner's office collected liver tissue and heart and femoral blood samples at autopsy from decedents suspected of opiate-APAP combination product overdose. Samples from 22 decedents were collected over a 4-month period. Serum samples were processed and the biomarker APAP-CYS was quantified using previously validated liquid chromatography-tandem mass spectrometry (LC-MS/MS) procedures. Histology was performed on liver samples.
Results: Heart and femoral blood samples were unusable from 2 decedents, so 20 decedents were included for analysis. The biomarker APAP-CYS was measurable in samples that were not degraded or hemolyzed. Three decedents did not have measurable APAP-CYS in either heart or femoral serum (lower limit of quantification 0.01 μM or 2.7 ng/mL). APAP-CYS was measurable in either heart or femoral serum in samples from 17 decedents. Heart serum APAP-CYS concentrations ranged from 0.04 μM to 28.9 μM (11.7 ng/mL to 7817 ng/mL). Femoral serum APAP-CYS concentrations ranged from 0.03 μM to 9.2 μM (8.7 ng/mL to 2477 ng/mL). In 12 decedents, APAP-CYS was quantifiable in both serum samples and in 11/12 of these decedents, heart serum APAP-CYS concentrations were the same or greater than femoral serum APAP-CYS concentrations. Opiates available in combination with APAP (hydrocodone, oxycodone, tramadol) were detected at autopsy for 18/20 decedents. Liver histology did not suggest significant hepatic injury or necrosis even in decedents with high serum APAP-CYS concentrations.
Conclusions: Postmortem APAP-CYS concentrations tended to be higher in heart samples than in femoral samples. In the absence of hepatotoxicity, postmortem serum APAP-CYS concentrations can be elevated into the range associated with hepatic necrosis in living patients with APAP toxicity. These results suggest postmortem release of APAP-CYS adducts into blood with postmortem redistribution. Future directions include quantification of acetaminophen and acetaminophen metabolites in these samples and the quantification of the biomarker APAP-CYS in liver samples.
Keywords: Acetaminophen (paracetamol), Laboratory, Postmortem

272. Trends in nicotine content and relationship to smoke nicotine yield among popular Japanese cigarette brands

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Background: Accidental cigarette ingestion by children is the most frequent occurrence in Japan. To estimate amount of nicotine ingested, we measured the actual nicotine content of tobacco in 33 popular cigarette brands (Vet Human Toxicol, 1997). In 2007, the US study confirms increased machine-measured levels of smoke nicotine over the period 1998–2005 as a result of increased nicotine in the tobacco rod. We sought to determine whether nicotine content would show an overall increase over time and would associate with smoke nicotine yield in Japanese cigarette brands.
Methods: Samples; 36 filtered and one non-filtered brands. Analytical method; The papers and filters were removed and the tobacco weighed. After crushing in a blender for 60 sec, the contents were quantitatively suspended in 100 ml of methanol:0.1 N NaOH (1:1) solution, ultrasonically vibrated for 60 min, and filtrated through polytetrafluoroethylene filters (pore size 0.45 μrn). Then the filtrate was diluted 50 times with a 0.067 M phosphate buffer (pH 7.0) and 20 μl were injected into the HPLC. Apparatus; Hitachi L-7100 pump, L-7400 UV detector set at 262 nm, and Hitachi D-7500 data integrator. The column; Inertsil ODS-3 (150 × 4.6 mm, 5 μm). Mobile phase; 0.067 M phosphate buffer (pH 7.0): acetonitril (88:12 v/v), containing 2 mM sodium hexanesulfonate. Flow rate; 1.0 ml/min. Retention time for nicotine; 9.4 min. Three cigarettes from each pack were measured. The correlation between these values and the nicotine yield on the label was measured by linear regression.
Results: The 37 brands sampled account for more than 50% share of the domestic sales in fiscal 2009. Average nicotine content and smoke nicotine yield were as follows: 14.18 mg ± 2.60 (range: 9.03–28.31 mg) and 0.62 mg ± 0.52 (0.1–2.3 mg), respectively. Compared to the average value, nicotine content increased by 18.9% over the period 1997–2010 (1997: 12.09 mg ± 3.09), the same trend was seen with the United States. On the other hand, nicotine yield has been reduced 21.5% (1997: 0.79 mg ± 0.56).
Conclusions: Nicotine yield is not correlated with nicotine content ($r^2 = 0.39$). Therefore when based on the guide (yield) number on the label, there is a risk of low intake estimate in cigarette ingestion. From the results of this study, the ratio of the yield and content from that it has expanded to 23-fold from about 15 times, that risk was further increased in 13 years.
Keywords: Nicotine contents, Cigarettes, Tobacco poisoning

273. The liability of opioid likeability

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Despite the availability of medicinal oral forms of morphine for nearly a century, morphine abuse is not a significant national concern. The alteration of morphine to create semisynthetic derivatives, such as heroin and the prescription opioids (PO), has been associated with widespread opioid misuse. PO abuse has become a public health epidemic with over 15,000 annual deaths. Although it is recognized that POs have sufficient reinforcing properties to sustain their misuse, we sought to evaluate the supporting likability science.
Methods: We obtained the English-language articles identified on a PubMed search for "opioid abuse liability" from 2001–2011. Additional articles were derived from their references. Articles were selected that discussed comparative likability of the POs hydrocodone, oxycodone, and morphine. Because design and terminology varied among studies, those directly comparing abuse liability of two or more POs in individual subjects were considered as reflecting likability.
Results: 17 studies were found that met search criteria and on further review 6 were deemed relevant. The majority of study subjects were current or former opioid abusers. Funding for most was provided by NIDA. Details are in the Table 1.
Conclusions: Oxycodone demonstrates a higher likability, and greater abuse potential, than morphine or hydrocodone across the

Table 1. Results for abstract 273.

Study	Design	Outcome measures	Likability
Comer SD. Neuropsychopharmacol 2008; 33:1179–91.	DBPC crossover: IV fentanyl, oxycodone, morphine, buprenorphine, heroin	VAS, Opioid Symptom Checklist, Subjective Opioid Withdrawal Scale, Drug Effect Questionnaire, Drug vs. Money Breakpoint Comparison	OXY > MOR
Stoops WW. Psychopharmacol 2010; 212:193–203.	DBPC crossover IV oxycodone, morphine, and hydrocodone, placebo	VAS, ARCI, 17 Item Adjective Checklist	MOR = HYD = OXY
Zachny JP. Psychopharmacol 2003; 170:242–54.	DBRPC crossover PO oxycodone, morphine, lorazepam, placebo	VAS, ARCI, Adjective Rating Scale (ARS), Drug Liking Questionnaire (DLQ), 24-hr post-session sequelae questionnaire	OXY > MOR
Zachny JP. Psychopharmacol 2008; 196:105–16.	DBRPC crossover PO oxycodone, morphine, placebo	VAS, ARCI, ARS, questionnaire drug liking/drug effect/take again, 24-hr post-session sequelae questionnaire	OXY > MOR
Zachny JP. Psychopharmacol 2003; 165:146–56.	DBPC cross over study PO hydrocodone, morphine, placebo	VAS, ARCI, ARS, DLQ, 24-hr DLQ	HYD = MOR
Zachny JP. Drug Alcohol Depend 2005; 78:243–52.	DBRPC crossover PO hydrocodone, morphine, placebo	VAS, ARCI, ARS, DLQ, 24-hr DLQ	HYD = MOR

majority of studies assessed. Abuse potential, as a component of safety, should be more closely scrutinized, and the risk benefit profile should be considered when prescribing opioids.
Keywords: Opioid, Abuse, Substance abuse

274. The novel development of an experimental model of dihydropyridine calcium channel blocker poisoning using intravenous amlodipine

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Background: Cardiovascular drug overdoses are among the leading causes of poisoning fatality reported to the American Association of Poison Control Centers (AAPCC). Within this class, calcium channel blockers (CCBs) account between 50–60% of deaths. CCBs are characterized as dihydropyridines or non-dihydropyridines. The majority of experimental animal models utilize non-dihydropyridines (verapamil) to study CCB poisoning. Since dihydropyridines are physiologically different from non-dihydropyridines and do account for morbidity and mortality we sought to establish an experimental model of dihydropyridine poisoning.
Methods: Healthy Sprague-Dawley rats weighing 400–500 g were anesthetized with isoflurane via tracheostomy. Intravenous access was established in the left femoral vein. Arterial blood pressure was measured through the right carotid artery. ECG was recorded continuously using 3 skin electrodes.

1. Normal saline, tween, and DMSO solvents were used to attempt to dissolve amlodipine.
2. Successful solvents were tested for cardiovascular toxicity.
3. LD50 was estimated using the up-and-down method as described by Dixon, which uses an iterative dose-selection algorithm. Starting with an initial exposure of 25 mg/kg, each subsequent dosage was raised or lowered based on the survival of the preceding animal. The maximum likelihood estimate for LD50 with SE was established using the following equation: LD50 = average (Xi) + d/N (A + C), where average (Xi) is the average test level (in mL/kg) for the last n trials, N is the nominal number of samples or total number of samples, minus 1 less than the number of identical samples at the beginning of the trial, A and C values are acquired from Dixon's tables after the series of experiments are performed, and d is the distance between data points.

Results:

1. We were able to dissolve amlodipine in 40% DMSO.
2. No changes in hemodynamics with DMSO concentrations as high as 40% after two hours of observation (which is our established period of observation).
3. The majority of animals died from PEA arrest about one hour after administration of amlodipine.
4. The Dixon up-and-down method for N > 6 gave a maximum likelihood estimate for the LD50 of 8.65 mg/kg (SE, +/− 2.67 mg/kg) mg/kg. The distance, d, between data points was 5 mg/kg. The total number of samples was 7.

Conclusions: We describe a novel preparation and (LD50) dose of IV amlodipine in a rat model of amlodipine poisoning. The applications are wide and can be used to study various treatment options such as high-insulin-euglyemic therapy.
Keywords: Calcium channel blocker, Shock, Cardiac toxicity

275. Characterizing methanol elimination in toxic overdose

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Background: Review of common toxicology textbooks has shown that there is limited information concerning the characteristics of toxic alcohol elimination. Published reports state that methanol is removed by zero order kinetics via alcohol dehydrogenase (ADH). However, when treatment with ethanol (EtOH) or fomepizole is initiated and ADH is blocked, elimination changes to first order before reverting to zero order when treatment effect decreases. This duality has caused confusion and resulted in reported summative, zero order clearances ranging from 0.88 to 20 mg/dL/hr. These

Table 1. Results for abstract 275.

	N	Half life (hrs)	Clearance (mg/dL/h)
Published: No treatment	6	7.1–24.3	0.93–3.33
Published: EtOH	2	12.9–33.5	4.32–7.33
Poison Center: EtOH	2	16–28	0.2–1.05
Published: Fomepizole	2	32.2–39.8	0.54–0.98
Poison Center: Fomepizole	7	8.9–70	0.5–1.94
Published: HD	6	2.2–34.3	1.82–20.59
Poison Center: HD	11	3.8–18	0.79–20.52

calculations may be inappropriate as the properties of methanol elimination at different methanol concentrations has yet to be elucidated. Clarification of these characteristics may aid in determining the need and duration of fomepizole, ethanol, and hemodialysis (HD) treatment. Methanol was chosen due to its longer half-life allowing easier assessment of its elimination characteristics.

Method: Data collected from case reports included age, sex, amount of methanol ingested, coingestants, pertinent treatment, and blood methanol levels. Of 1189 methanol poison center reports from 2001–2009, 35 contained 3 or more methanol levels. However, 17 were excluded due to poor documentation. In addition, using predefined search criteria, over 140 abstracts were reviewed using MEDLINE. This resulted in 7 overdose reports containing 14 patients with 3 or more methanol levels.

Results: Published case reports and local poison center data were separated into four treatment groups: no treatment, EtOH, fomepizole, and HD.

Surprisingly, a mixture of zero and first order kinetics was found in each treatment group in both the published and poison center data. This may suggest inadequate characterization or a mixed order elimination process.

Conclusions: This is the largest retrospective review of published and local poison center data on methanol overdoses to date. The result supports the ambiguity of current toxicology textbooks, but is hampered by the small number of patients. Due to the inability to characterize methanol elimination, regardless of treatment modality, caution should be used when interpreting pharmacokinetic data in the overdose setting. Methanol levels should be drawn frequently to best characterize patient specific elimination, as assumptions made using the current pharmacokinetic model may be inaccurate.

Keywords: Methanol, Pharmacokinetics, Clearance

276. Survival following methanol poisoning with initial serum pH <6.5

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Background: Methanol (MeOH) is metabolized to formic acid causing ocular, central nervous system, and renal toxicity. Lower arterial pH at presentation correlates with increasing mortality in MeOH poisoning. We present the first reported case of survival from severe MeOH poisoning with initial arterial pH less than 6.5.

Case report: A 50 year-old man presented to an ED after being found altered. On arrival, the patient (pt) had fixed and dilated pupils, localized to noxious stimuli, and had unintelligible speech. HR was 106 bpm, respiratory rate 18 breaths/min, and BP 126/79 mmHg. The patient was rapidly intubated.

Arterial blood gas (ABG) results, using a Siemens Rapid Point 405 blood gas analyzer were: pO_2 218 mmHg and pH <6.5. On initial labs, sodium was 153 mmol/L, potassium 5.9 mmol/L, CO_2 <5 mmol/L, creatinine 2.4 mg/dL, ammonia 341 umol/L, lactate 12.8 mmol/L, and ethanol was 18 mg/dL. CT of the brain showed old right frontal infarct. EKG demonstrated sinus tachycardia with qrs prolonged at 145 msec. The patient was given 2 liters of normal saline, 100 mEq of sodium bicarbonate, and 15 mg/kg of fomepizole (4-MP) IV. He became hypotensive and norepinephrine infusion was started. The pt was transferred to our medical toxicology service.

On arrival, the pt was comatose, without sedation. Temperature was 37.8°C, HR 154 bpm (atrial fibrillation), BP 91/53 on 30 mcg/min norepinephrine infusion, ventilated at 20 breaths/min. ABG pH was 6.915, pCO_2 38 mmHg, pO_2 358 mmHg. CO_2 was 9 mmol/L, creatinine 1.96 mg/dL, ammonia 236 umol/L, phosphorus 11.1 mg/dL, and lactate 8.7 mmol/L. Acetaminophen, salicylate, valproate, and ethylene glycol levels were undetectable. Serum MeOH level was 51 mg/dL.

Continuous veno-venous hemodiafiltration (CVVHDF) was initiated. 4-MP was dosed every 4 hours during CVVHDF.

Serial MeOH levels were followed down to 14 mg/dL. The pt's neurologic examination improved, and he was off vasopressors, alert, and successfully extubated on hospital day (HD) 3. Ophthalmologic consultation found no evidence of retinal toxicity. The pt made a full recovery and was transferred to an inpatient psychiatric facility on HD 8.

Case discussion: This pt presented after MeOH ingestion with profound hyperammonemia and hyperphosphatemia likely due to dehydration and severe acidosis. Metabolism of MeOH was blocked by administration of the competitive alcohol dehydrogenase inhibitor 4-MP. Rapid initiation of CVVHDF corrected the pt's acidosis and metabolic disarray as well as enhancing clearance of MeOH.

Conclusions: Survival without sequelae from MeOH poisoning despite initial arterial pH <6.5 is possible with early initiation of renal replacement and maximal intensive supportive care.

Keywords: Methanol, Acidosis, Fomepizole

277. Missed isoniazid exposure secondary to inappropriate serum testing leads to repeat overdose

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Background: Isoniazid (INH) overdose is a well-known cause of seizures and lactic acidosis. We report a patient with repeat INH overdoses over 13 months, initially undiagnosed secondary to inappropriate serum testing.

Case report: A 14 year-old female presented to the emergency department (ED) on Christmas Day 2010 with seizures, vomiting, and altered mental status. She received 8 mg midazolam en route. Initial testing revealed a metabolic acidosis with a pH of 6.9 and a lactate of 60.8 mg/dl. She was currently taking 300 mg/day of INH for latent tuberculosis. Given the history of recent INH use, 3500 mg of pyridoxine was administered empirically along with intravenous fluids. The patient denied intentional ingestions and an inpatient workup did

not reveal an etiology of her seizures. A serum INH level, obtained 36 hours after admission, was <0.1 mcg/mL. She was discharged with rectal diazepam and instructed to continue INH.

The patient returned to the ED in January 2012 in status epilepticus. She received midazolam en route and after arrival was treated with phosphenytoin and bicarbonate. The patient was found to have a mild acidosis with a pH of 7.29 and lactate of 11.2 mg/dl. Again, she was empirically treated with 4130 mg of pyridoxine. A serum INH level obtained 24 hours after presentation was 1.8 mcg/dL. After stabilization, the patient admitted to intentional INH overdoses, precipitating both presentations.

Case discussion: Seizures and metabolic acidosis complicate INH overdose. INH reaches a peak serum concentration in 1–2 hours and is metabolized by acetylation and hydrolysis, with an elimination half-life of 70 minutes in normal acetlyators and 180 minutes in slow acetylators. Approximately 75–95% of metabolites are excreted renally within 24 hours. Detection of urine metabolites is used in monitoring INH compliance and has a 96–99% sensitivity at 24 hours, and 76% at 48 hours. However, serum levels obtained >24 hours may be misleading.

Conclusions: An understanding of INH pharmacokinetics dictates the likelihood of INH exposure confirmation with either serum or urine testing. To confirm an INH exposure serum levels should be obtained within 24 hours of ingestion. Clinicians caring for patients presenting with status epilepticus without a clear etiology or suspected of INH toxicity should consider the timing of specimen collection in order to determine the most appropriate diagnostic strategy.

Keywords: Overdose, Seizure, Laboratory

278. Severe lamotrigine toxicity treated with intralipid emulsion therapy

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Background: Lamotrigine (LTG) is a phenyltriazine anti-epileptic compound that inhibits voltage-gated sodium channels, presynaptic release of the excitatory neurotransmitters glutamate and aspartate, as well as serotonin reuptake. To date, the highest reported premortem LTG level was 74.7 mcg/ml in a patient who expired on hospital day (HD) 4 after experiencing seizures, widened QRS and cardiovascular collapse. This case describes a LTG poisoned patient with a LTG serum level of 90 mcg/mL measured 16 hours post ingestion.

Case report: A 23 year-old male was found shaking and foaming at the mouth with empty bottles of LTG and fludricortisone. Initial vitals included BP 141/58 mmHg, respirations 24/min, and normal oxygen saturation. The patient was agitated and delirious, with generalized myoclonic activity. Laboratory data showed bicarbonate 17 mg/dl with anion gap 18, and lactate 6.5. ECG demonstrated a QRS of 117 with terminal R-wave of 4 mm in lead aVR and S-wave in lead I and aVL. The patient was transferred to a tertiary toxicology management facility where he continued to exhibit intermittent generalized myoclonic restlessness and agitation. On HD 3, his LTG level resulted at 90 mg/L (collected and sent out at 16 hours post-ingestion). Intralipid emulsion (ILE) therapy was administered because of the continued agitation. Serial measurements of the LTG level demonstrated an apparent half-life of 43 hrs. The clearance of LTG appears to increase after ILE was administered and toxicity resolved on HD 6.

Case discussion: Case reports have associated LTG toxicity with hyperkinetic and hypokinetic movement disorders, seizures, confusion, agitation, hyperthermia, tachycardia, hypertension, hyperreflexia, clonus, prolonged QRS, and elevated CK. These reports often include coningestants which may confound the presentation. We present a less confounded LTG ingestion (assuming fludricortisone has low toxicity) with a markedly elevated LTG blood level. This is a unique opportunity to observe LTG kinetics and symptomatology before and after the administration of ILE therapy.

Conclusions:

1. LTG toxicity is associated with movement disorders characterized by intermittent myoclonic restlessness and agitated delirium, prolonged QRS, likely the result of sodium channel blockade on electrocardiogram and possibly seizures.
2. The apparent half life of LTG is 43 hours, however the clearance appeared to increase at 120 hours post ingestion.
3. Intralipid therapy may hasten the recovery from lamotrigine toxicity as decreased agitation and myoclonic restlessness was noticed in our patient shortly after treatment with this modality.

Keywords: Lamotrigine, Intralipid emulsion, Pharmacokinetics

279. Method validation of a tricyclic antidepressant drug panel in urine by LC-MS/MS

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Background: Tricyclic antidepressants (TCA) can cause serious cardiac complications and can be lethal if misused at high doses; as a result, TCA should be monitored. To monitor patient compliance to therapy, urine specimens may be preferred since the collection is non-invasive and the specimen can provide a longer detection window. TCA are frequently monitored by immunoassay; however false positives and cross reactivity to other cyclic compounds may compromise results. The purpose for this study was to develop a confirmation method by liquid chromatography-tandem mass spectrometry (LC-MS/MS) to determine TCA in urine specimens. This LC-MS/MS method can quantify amitriptyline, nortriptyline, doxepin, desmethyldoxepin, desipramine, imipramine, clomipramine, desmethylclomipramine and protriptyline.

Methods: Standards were obtained by Cerilliant and prepared in drug-free urine. The internal standard which includes: nortriptyline-D3, doxepin-D3, desipramine-D3, imipramine-D3, clomipramine-D3, protriptyline-D3 were added to the patient urine, standards, and controls in microcentrifuge tubes. Protein precipitation was performed with a methanol/acetonitrile solution, then samples were centrifuged to pellet the proteins. The supernatant was then injected into a LC-MS/MS (Waters Acquity TQD). The concentration of analyte(s) was calculated from the calibration curve and ion ratios between the analyte(s) and the internal standards.

Results: The calibration curves obtained with human urine were linear with a correlation coefficient of over 0.99 in the range of 100–2000 ng/mL. The coefficient variation for inter- and intra-day precision was within 12% for each analyte at five different concentrations: 100, 300, 1000, 2500 and 5000 ng/mL. There was no carryover observed in this assay, with the high concentration of 5000 ng/mL. To evaluate accuracy, drug-free urine was pooled and used a matrix to spike 22 samples with TCA at different concentrations. An additional 33 patient samples were collected and sent to an alternative laboratory for split sample comparison by gas chromatography-mass spectrometry (GC/MS). In a total of 55 results, there were three false negative results for nortriptyline and two false negative results for amitriptyline were observed when comparing to GC/MS method. The false negative results could be due to the difference in sample preparation, and positive cut-off concentrations, between the two laboratories. No false positive result was observed.
Conclusions: Urine TCA testing can be a useful approach in identifying patterns of compliance, misuse, and abuse. We provide an option to confirm and quantify the TCA concentration in urine by LC-MS/MS.
Keywords: Antidepressant, LC-MS/MS, Urine

280. Ethylene glycol poisoning and hypocalcemia; Is there a correlation?

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Background: Ethylene glycol (EG) gets metabolized to oxalate which subsequently combines with calcium to form calcium oxalate crystals. Several medical toxicology texts advocate close monitoring of calcium levels and warn of the dangers and morbidity associated with hypocalcemia in the context of EG poisoning. The aim of this study is to characterize this relationship.
Methods: Utilizing Crystal Reports (Version 11.0), all EG cases reported to the Illinois Poison Center over a 10 year period (2002–2011) were retrospectively queried. Those cases where an EG concentration was reported >/ = 20 mg/dL, and a serum calcium level was measured and recorded, were included. Hypocalcemia was defined as a serum level less than 8.5 mg/dL. Any hypocalcemia related morbidity (QTc changes on EKG, muscle spasms or cramps, etc.) or requirement for calcium supplementation was also recorded.
Results: A total of 589 cases of EG were managed over the 10 year period. Thirty cases had documented EG concentrations >/ = 20 mg/dL and a recorded calcium level. Within this group, EG concentrations ranged from 23–575 mg/dL (average 170 mg/dL). Serum calcium levels ranged from 7.1–10.4 mg/dL (average 8.9 mg/dL). No ionized calcium levels were reported. Five patients (17%), by definition, were classified as hypocalcemic (range 7.1–8.1 mg/dL; average 7.6 mg/dL) with corresponding EG levels (range 28–575 mg/dL; average 222 mg/dL). The patient with the highest EG concentration (575 mg/dL) had a calcium level of 7.7 mg/dL, and the patient with the lowest calcium level (7.1 mg/dL) had an EG of 28.7 mg/dL. No morbidity related to hypocalcemia was reported and no calcium supplementation was required in any of the 30 patients.
Conclusions: Contemporary medical toxicology textbooks recommend serum calcium levels in patients exposed to EG. While measuring oxalate concentrations may best determine if a relationship between EG poisoning and hypocalcemia exists, this was impossible with these retrospective data. These data additionally depend on the requirement of an EG concentration and calcium level to have been recorded within the poison center record. The timing of these laboratory values was also impossible to address. A minority of patients in this series was considered hypocalcemic. No direct trend between EG concentration and serum calcium level was appreciated. No patient experienced hypocalcemic related morbidity nor did any patient require calcium supplementation. A prospective study would help better define a relationship between EG exposure and the potential for hypocalcemia and the existing prescription for obtaining calcium levels in EG poisoned patients.
Keywords: Ethylene glycol, Hypocalcemia, Laboratory

281. Correlation of osmolar gap and ethylene glycol concentration during dialysis

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Background: Ethylene glycol (EG) is the chief ingredient in antifreeze solutions. EG ingestion may lead to seizures, coma, acidosis, renal failure and death. Hemodialysis (HD) removes EG, toxic metabolites and corrects acidosis. The use of osmolar gap as a surrogate for serum EG concentration could improve upon currently varied practices and HD run times especially in situations where EG concentrations cannot readily be obtained. We present a case that demonstrates correlation between EG concentration and osmolar gap (OG).
Case report: 47 year-old woman presented to the ED after being found obtunded. She arrived with a pulse of 115 beats/minute, blood pressure 115/81 mmHg, O_2 saturation of 100% and Kussmaul respirations. She was intubated and given IV saline. Laboratory values were significant for pH 7.19, anion gap 33 mmol/L, serum osmolality 373 mosm/kg, and a calculated osmolarity of 299 mosm/L. She was given 15 mg/kg fomepizole; her EG level returned at 341 mg/dl. She was placed on HD for 6 hours with resolution of metabolic acidosis and normalization of osmolar and anion gaps. Four EG concentrations were drawn during HD and correlated to the osmolar gaps. The patient was extubated the next day. Her creatinine peaked at 5.3 mg/dL; she required intermittent HD for 10 days and ultimately recovered renal function.
Discussion: EG elimination by HD is described by the formula $t = [-V \ln(5/A)/0.06\,k]$; t is time, V is the Watson estimate of total body water, A is the initial toxin concentration in mmol/L, and k is 80% of the manufacturer specified dialyzer urea clearance. EG concentrations as high as 905 mg/dL have been published making a wide potential range of needed time on HD. It is prudent to continue HD until the anion gap metabolic acidosis resolves and EG concentration is <20 mg/dl. Practice varies greatly with regard to serial EG concentrations during HD. While the availability of measuring serum EG concentration is limited, the OG is rapidly obtainable at most centers. A published case series highlighting

the correlation of OG and serum methanol concentration during HD proposed that using OG to guide HD duration could have reduced HD times. Similar correlation with EG may help prevent over or under utilization of HD and lab resources. A best-fit trend line applied to a scatter plot of the four values each of EG concentrations and OG from the above case correlated with an R^2 value of 0.995.

Conclusions: We present an EG intoxication where there is strong correlation between osmolar gap and serum EG concentrations during HD. Further study is warranted as use of osmolar gap rather than frequent EG concentrations has the potential to optimize resource utilization and minimize risk to the patient during HD.

Keywords: Ethylene glycol, Hemodialysis, Osmolar gap

282. On the whole, kinetics of rectal vicodin are unreliable

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Background: Analgesics, including acetaminophen (APAP) containing products, are consistently the leading exposure substances according to the National Poison Data System. Apap-containing products accounted for at least 13% of Poison Center deaths in 2010. While the pharmacokinetic profile of orally ingested APAP is well known, and the Rumack-Matthew Nomogram is widely used to determine potential toxicity, information regarding other routes of exposure is lacking. APAP may be administered orally or rectally, and the dosing and frequency is equivalent. We report a case of unexpected pharmacokinetics after rectal administration of an APAP-containing product.

Case report: A 42 year old man presented with lethargy 5 hours (h) after the ingestion and rectal administration of a mixture containing an unspecified Raid product and eliminator weed and grass killer and the insertion of an unknown quantity of Vicodin rectally. His APAP level was 50 mcg/ml. The patient was admitted to psychiatry. He became tachycardic and diaphoretic 10.5 hours post-ingestion, and his APAP level measured 95 mcg/ml. Treatment with N-acetylcysteine was initiated. Subsequent APAP levels were 132 mcg/ml, 84 mcg/ml, and 30 mcg/ml at 16.5 h, 22 h, and 27 h post-ingestion respectively. His APAP level was undetectable 24 h after presentation. At 39 h post exposure, his AST and ALT peaked at 74 and 44, and his INR peaked at 1.23 on hospital day four.

Discussion: The recommended dose of APAP is 10–15 mg/kg every 4–6 hours irrespective of route. Rectal suppositories contain glycerin to facilitate absorption. The bioavailability of APAP is between 60% and 98% with an estimated time to peak of 30–120 minutes for oral preparations. In this case, the patient administered an oral preparation rectally; the anticipated pharmacokinetic profile following this exposure is largely unknown. In general, however, rectal absorption is slower and the peak serum concentration lower. In a study with 8 children, a 13 mg/kg APAP dose resulted in a maximum concentration of 7.7 mg/L at 1.6 h and 4.9 mg/L at 2.0 h following oral and rectal administration [1]. This data is based on formulations intended for rectal use. In this case, the peak serum concentration occurred between 5 and 16 hours and the elimination half-life was prolonged.

Conclusions: Providers should be aware of the altered kinetics of rectally administered acetaminophen containing products and consider obtaining serial APAP levels.

Reference

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Keywords: Acetaminophen (paracetamol), Pharmacokinetics, Rectal

283. The effect of pre-hospital nebulized naloxone on suspected heroin-induced bronchospasm

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Background: Snorting or smoking heroin is a known trigger of acute exacerbations of asthma in addition to usual factors (tobacco smoke, upper respiratory infections weather changes, etc.). In fact, heroin abuse may be a risk factor for more severe asthma exacerbations, as these users have a higher rate of intubation compared to nonusers. Research has demonstrated that heroin, and other opioids, provoke pulmonary bronchoconstriction. Investigations in the basic science literature show that naloxone may play a role in decreasing opioid-induced bronchospasm. There are no known clinical cases where the effect of naloxone on opioid-induced bronchospasm has been described.

Methods: This is an observational study in which nebulized naloxone was administered to patients with suspected heroin-induced bronchospasm. In 2010, the Chicago Region XI EMS system added a new route of naloxone administration for the treatment of patients with suspected opioid intoxication or undifferentiated altered mental status. Patients with spontaneous respirations were administered 2 mg of naloxone with 3 ml of normal saline by nebulization. While the majority of cases reviewed were administered nebulized naloxone for depressed respirations or altered mental status, here we describe a case series of administrations for suspected heroin-induced bronchospasm.

Results: Out of 129 cases of nebulized naloxone administered over a 6-month period (January 1st-June 30th 2010), there were 21 administrations to patients with suspected heroin-induced bronchospasm. Of these, 19 patients had a clinical response to treatment documented. 13 patients displayed clinical improvement (68%), 4 patients had no improvement (21%), and 2 patients worsened (10%). Of the two patients that had clinical decline, none required intubation. Of the patients that improved, 1 patient received only nebulized naloxone and one patient received naloxone and albuterol together. Seven patients showed clinical improvement after the administration of albuterol, atrovent, and naloxone together as a combination. Four patients showed additional improvement when the naloxone was administered after the albuterol and atrovent combination.

Conclusions: This case series indicates that naloxone may play a role in reducing acute opioid-induced bronchoconstriction, either alone or in combination with albuterol. Future controlled

studies should be conducted to determine if the addition of naloxone to standard treatment improves bronchospasm without causing adverse effects.
Keywords: Naloxone, Opioid, Asthma

284. Case series of 25I-NBOMe exposures with laboratory confirmation

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Background: 25I-NBOMe is a derivative of the phenethylamine hallucinogen 2C-I. We report a case series of four patients with adverse effects from 25I-NBOMe exposure. Three cases of exposure are supported by laboratory confirmation.
Case series: Four males between the ages of 18 and 19 simultaneously presented to the emergency department after recreational exposure to 25I-NBOMe. They purchased the drug from a dealer who obtained it through the internet. The substance was either snorted or ingested orally. Upon arrival, all patients were tachycardic and displayed varying levels of psychomotor agitation. None were capable of providing a clear history. Three patients experienced prolonged seizure activity which required pharmacologic therapy, intubation, and mechanical ventilation. Patient D developed rhabdomyolysis and renal failure requiring hemodialysis. Urine specimens were analyzed from patients A, C, and D. Using a sample of 25I-NBOMe that was obtained by law enforcement as a standard, our laboratory was able to use liquid chromatography-mass spectrometry (LC-MS) to confirm and quantify the presence of 25I-NBOMe in the biologic specimens. Pertinent data for the four patients recorded at the time of presentation is summarized in Table 1.
Discussion: This is the first reported series of symptomatic patients after 25I-NBOMe exposure with quantitative urine confirmation. 25I-NBOMe is a ring substituted phenylethylamine derivative of 2C-I with potent agonist activity at $5HT_{2A}$ receptors. Like 2C-I and other phenylethylamines, use of this emerging "legal high" can result in serotonergic and sympathomimetic toxicity including hypertension, tachycardia, psychomotor agitation, hyperglycemia, seizures, and rhabdomyolysis. Especially concerning with these exposures was the development of protracted seizures that occurred in 3 of 4 patients and necessitated aggressive pharmacologic therapy and supportive measures.
Conclusions: The recreational use of 25I-NBOMe can result in significant adverse effects including prolonged seizures. This "legal high" can be detected in the urine of users with LC-MS. Clinicians should be cognizant of this drug and its potential for toxicity in order to provide appropriate care.
Keywords: Bath salt, Seizure, Designer drug

285. Fatalities following parenteral injection of MDPV sold as "hookah cleaner"

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Background: Designer stimulants sold as "bath salts" have proliferated in recent years. We report two fatalities following parenteral administration of a purported water pipe cleaner with documented serum methylenedioxypyrovalerone (MDPV) levels and detection in the product.
Case 1: A 43-year-old man was found dead by police at the edge of a lake. He was not brought to the Emergency Department (ED). His girlfriend told police they arrived the previous night to go camping. He was "acting weird" so she locked herself in a vehicle and found him the next morning. Earlier that day he had self-injected glass cleaner obtained at a "head shop." Police reviewed security video that showed him purchasing "hookah cleaner." The shop owner told police its active ingredient was MDPV.

Multiple small abrasions and contusions were seen at autopsy. There was no evidence of drowning or major traumatic injury, but single vessel coronary disease was noted. Urine was negative for drugs of abuse (DOA). Blood ethanol was <10 mg/dL. Serum MDPV was 160 ng/mL.
Case 2: EMS responded to the home of a 37-year-old man when a neighbor reported he was naked and screaming. He was found apneic and pulseless. EMS initiated CPR and intubated the patient. Bedside glucose was 236 mg/dL. Naloxone 1 mg IV and multiple doses of epinephrine were given. Initial cardiac rhythm was asystole. Paramedics found needles and drug paraphernalia at the scene. They worked on the patient for 45 minutes in the field. Resuscitation was terminated in the ED 10 minutes after arrival.

The patient had a history of drug abuse. His wife told police that when his chronic pain was not relieved by tramadol, he self-treated by injecting "Crystal Clean Hookah & Pipe Cleaner" obtained from a head shop.

Autopsy revealed 3 vessel coronary stenosis and rib fractures consistent with CPR. Urine was negative for DOA. Blood ethanol was <10 mg/dL. MDPV, tramadol, and caffeine were detected in the blood. Serum MDPV was 340 ng/mL.

Table 1. Data for abstract 284.

Patient	Heart rate (bpm)	BP (mmHg)	Serum CPK (U/L)	Serum HCO_3 (mmol/L)	Serum glucose (mg/dL)	Seizure activity	Intubated	Urine [25I-NBOMe] (ng/mL)	Urine drug screen
A	122	121/56	n/a	22	239	no	no	2	caffeine
B	108	140/60	826	22	484*	yes	yes	n/a	n/a
C	153	148/49	292	13	286	yes	yes	36	caffeine
D	184	107/82	30000	11	79	yes	yes	28	caffeine nicotine

n/a = specimen not available or test not performed.
* = Patient B has known diagnosis of type 1 diabetes mellitus.

MDPV was detected in the "Crystal Clean" product found at his home.

Discussion: Excited delirium and death have been reported following parenteral abuse of MDPV-containing bath salts. These decedents injected a product labeled as "hookah cleaner" according to histories obtained by police. Both had documented serum MDPV levels. Detection in the original product supports this being the source. While the immediate cause of death is unclear, MDPV toxicity is associated with tachycardia, and may have precipitated dysrhythmias. The presence of coronary disease could have been a predisposing factor in both cases.

Conclusions: As public awareness of bath salt abuse increases, the active ingredients may be sold in other forms that are less recognizable.

Keywords: Abuse, Bath salt, MDPV

286. Normal MRI with symptoms and diagnosis of subacute combined degeneration from nitrous oxide abuse

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Background: Subacute combined degeneration (SCD) refers to the degeneration of the posterior and lateral columns of the spinal cord caused by a deficiency in vitamin B12. SCD has been described both with the use of nitrous oxide for anesthesia and with abuse of nitrous oxide using whipped cream propellant canisters ("whippits"). Nitrous oxide causes a functional B12 deficiency via oxidation of the cobalt in the corrin ring, rendering it inactive. In SCD from nitrous oxide, B12 levels may be low or normal, and other sequelae of B12 deficiency may be present. Treatment involves cessation of nitrous oxide abuse and B12 supplementation. We describe a case of symptomatic SCD of the spinal cord caused by abuse of nitrous oxide with an initially normal MRI.

Case report: A 31 year-old woman presented with a one week history of lower extremity weakness and paresthesias in both her legs up to her mid back and hands. Upon exam, she had 3/5 strength in her lower extremities with hyperreflexia, decreased vibratory and light touch sensation up to the mid abdomen, and decreased proprioception in her toes. An initial MRI of the spine was normal and the cause of her symptoms was unclear. She was discharged with neurosurgery follow up scheduled. Her symptoms worsened and a repeat MRI one month later revealed extensive enhancement and edema of the central and dorsal aspects of the spinal cord from the craniocervical junction to the level of the T8 vertebral body with scattered areas of sparing. Only after a large number of nitrous oxide canisters were found in her purse was the diagnosis of SCD made. She was started on B12 shots, but was lost to follow up.

Discussion: It is known that patients with all-cause SCD may have persistent symptoms despite normal MRI studies; however, this is the first known case of symptomatic nitrous oxide-induced SCD in the setting of an initially normal MRI. The neuropathologic changes seen with SCD exist along a spectrum, from initial edema and irregularity of the myelin sheaths, to eventual demyelination of the axons and cell death. The first MRI in this case was likely due to the patient presenting early in the disease process, as spinal cord edema and demyelination are later findings. Additionally, our patient was not forthcoming with her drug abuse history, further confounding her diagnosis in the setting of normal imaging.

Conclusions: This case is the first to demonstrate that symptomatic SCD from nitrous oxide abuse may present with a normal MRI. Consequently, normal imaging should not preclude diagnosis of this rare disease. If clinical suspicion is high, treatment with B12 supplementation should not be delayed, even if a history of nitrous oxide abuse is not forthcoming.

Keywords: Inhalant, Abuse, Neurotoxicity

287. Sudden death following confirmed phencyclidine overdose: Toxicologic and pathologic analysis

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Background: Sudden death due to phencyclidine (PCP) toxicity is rare and is typically associated with medical comorbidities or physical restraint. It is usually attributed to excited delirium, stress cardiomyopathy, or catecholamine-induced arrhythmia. However, establishing a causal relationship is difficult given low incidence. We present a case of a minimally-restrained, 35 year old man who died suddenly following PCP intoxication and arrest.

Case report: A 35 year old man died in police custody following arrest for bizarre behavior at a bar. During arrest observers noted that he had strength well beyond expected and Taser deployments had minimal effect. Eventually he was handcuffed, taken to jail, and placed in a cell. Handcuffs were removed and the subject's only restraint was a shackle and chain attached to a wall. The subject paced, danced, and yelled for approximately one half hour, after which he appeared to be breathing heavily. Shortly thereafter officers found him pulseless and apneic. CPR was immediately initiated and an AED was attached. The AED did not advise shock. EMS personnel provided CPR for approximately 20 minutes before death was pronounced. Autopsy revealed abrasions and contusions but no major injuries. Internal examination demonstrated pulmonary vascular congestion, congested lung parenchyma, and pulmonary edema. Histologic evaluation confirmed pulmonary edema, intraalveolar hemorrhage, and hepatic sinusoidal congestion. The cardiovascular, central nervous system, and renal systems were grossly and microscopically unremarkable. Toxicologic analysis included GC/MS blood screen which identified PCP, nevirapine, and caffeine. Blood PCP concentration was quantified at 710 ng/mL.

Discussion: Sudden death from PCP intoxication is very rare. Reported cases of sudden death attributed to PCP intoxication almost universally include four-point or 'hog-tie' restraints. However, this subject was only minimally restrained prior to death. He did not appear to have any comorbid medical conditions, though the presence of the antiretroviral nevirapine may indicate HIV infection. Postmortem findings suggest sudden death due to acute cardiac failure. The AED did not recommend shock, suggesting that neither ventricular fibrillation nor ventricular tachycardia were present. His sudden death is consistent with acute stress

cardiomyopathy secondary to high serum PCP or endogenous catecholamine concentration.
Conclusions: Unexpected cardiopulmonary collapse of this individual with high-level PCP intoxication illustrates the need for close monitoring by EMS and law enforcement personnel in cases of suspected PCP intoxication.
Keywords: Phencyclidine, Death, Substance abuse

288. Psychosis from a bath salt product containing flephedrone and MDPV with serum, urine, and product quantification

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Background: The use of designer drugs commonly marketed as bath salts or plant food has risen dramatically in recent years. Several different synthetic cathinones have been indentified in these products, including 3,4-methylenedioxypyrovalerone (MDPV) and 4-fluoromethcathinone (flephedrone). We report a case of bath salt intoxication with quantitative MDPV and flephedrone levels in a patient's serum and urine, and from the bath salt product.
Case report: A 23 year old male with a prior psychiatric history arrived via EMS for bizarre behavior, suicidality, and hallucinations after reportedly insufflating a bath salt. Serum, urine, and the bath salt product were sent for testing using liquid chromatography-time-of-flight mass spectrometry (LC-TOF/MS) (TOF 6230, LC 1260, Agilent). He was found to have MDPV levels of 186 ng/mL and 136 ng/ml in his serum and urine, respectively, and flephedrone levels of 346 ng/mL and 257 ng/mL in the serum and urine, respectively. A comprehensive LC-TOF/MS screen for 39 other cathinones and 309 other drugs was negative. The white powder in question was found to contain 143 mcg MDPV and 142 mcg flephedrone per mg powder. His psychosis and agitation resolved with lorazepam, droperidol, and observation in the emergency department.
Case discussion: Agitation, psychosis, movement disorders, tachycardia, and hypertension have all been attributed to the use of MDPV; there are no prior reports detailing clinical experience with flephedrone. Considering that our patient's serum flephedrone levels were two-fold higher than his MDPV level, it is likely flephedrone contributed to his clinical toxicity. Halogenation of phenylethylamines at the para-position, such as with flephedrone, prevents metabolism via para-hydroxylation and potentially increasing clinical effects.
Conclusions: This case suggests the possibility that fluorinated cathinones, such as flephedrone, may have altered metabolism and/or elimination which may affect their course of clinical toxicity. This case also highlights the evolving composition of synthetic cathinones found in bath salt products.
Keywords: Designer drug, Drug of abuse, Laboratory

289. Sudden cardiac death associated with methylone use

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Background: Methylone is a synthetic cathinone derivative that, along with other structurally related substances, can be found in so-called "bath salt" products. Toxicity due to these substances has increasingly been reported in the medical literature in recent years. There have been few reports of severe toxicity due to methylone. We report a case of sudden cardiac death associated with the use of methylone.
Case report: A previously healthy 19-year-old male without known medical problems collapsed suddenly while jogging. Witnesses noted him to be pulseless and apneic and immediately started CPR and activated the emergency medical services system. Paramedics arrived within 5 minutes and cardiac monitoring revealed pulseless electrical activity. He was transported to a nearby emergency department where ACLS efforts were continued, but he was pronounced dead soon after arrival. An autopsy was performed and showed no anatomic cause to explain the patient's sudden death. Comprehensive toxicological analysis for drugs of abuse was performed on the patient's urine and central blood within 24 hours of death using GC/MS and detected methylone in the urine at a concentration of 70 mcg/dL. No other drugs were detected in urine or blood including pseudoephedrine, ephedrine, amphetamine, methamphetamine, MDMA, MDA, or cocaine or its metabolites. Analysis was also negative for other bath salt components including flephedrone, n-ethylcathinone, mephedrone, methedrone, ethylone, butylone, MDPV, and naphyrone.
Discussion: Methylone toxicity has rarely been described in the medical literature. Only one other methylone-associated fatality has been reported, in a 24-year-old female who developed serotonin syndrome and disseminated intravascular coagulation after ingesting a combination of methylone and butylone. No cases of sudden cardiac death associated with methylone use have been described previously. While a definitive cause-and-effect relationship cannot be established in this case, the lack of anatomic abnormalities found on autopsy and the failure to detect other cardioactive agents in the patient's blood or urine suggests strongly that methylone contributed to the patient's death.
Conclusions: Methylone has the potential to result in severe toxicity including sudden cardiac death. Specific testing for methylone and other bath salt components should be considered in such cases, particularly when other causes for sudden cardiac death are not found.
Keywords: Bath salt, Death, Forensics

290. Clinical presentations and medical complications after exposure to substances labeled as "bath salts": A ToxIC preliminary report

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Background: The increasing usage of synthetic cathinones, sold as bath salts, has resulted in reported medical complications. Most reports involve small numbers of patients. We are conducting a multi-center study of clinical presentations, complications, and dispositions in patients presenting to Emergency Departments after reported bath salt exposure. Although a preliminary report, our data represents the largest cases series to date.
Methods: Bath salt cases were identified from the ToxIC Network Database using the search terms: bath salt, methylenedioxypyrovalerone, MDPV, mephedrone, methylone, or dimethoxyphenethylamine. Centers reporting cases were contacted and, after IRB approval, reviewed patient medical records and filled out a uniform data abstraction form for each identified case. The data were consolidated and summarized using descriptive statistics.
Results: 126 cases were identified in ToxIC. At the time of this writing, 40 cases from 6 sites have been received. The majority of cases involved young (median age 29) men (75%). Most patients had a prior history of mental illness (57.5%). 75% of patients used bath salts recreationally. Insufflation or unknown (both 40%) were the most frequent routes of exposure. 40% of cases involved a single drug exposure; the most commonly reported co-ingestant was a synthetic cannabinoid (17.5%). Tachycardia (HR > 100 bpm) was present in 70% of cases. Other sympathomimetic signs included systolic hypertension (SBP > 140 mmHg, 35%) and hyperthermia (Temp > 100.1°F, 15%). Agitation was reported in 57.5% of the cases. The majority of initial basic metabolic panels were normal, abnormal findings included hypokalemia (K < 3.5 mmol/L, 27.5%) and acidemia (HCO_3 < 22 mmol/L, 37.5%). 57.5% of cases had confirmatory testing with MDPV identified in 78% of these cases. The majority of patients had normal EKGs. Most patients were treated with benzodiazepines (72.5%) with a smaller percentage receiving an antipsychotic (30%). 22.5% of patients were intubated with agitation cited as the most common reason (67%). Most patients were admitted to the hospital (85%) with the mean length of stay being 5.1 days (± 8.6, Stdev); of those admitted 85% were admitted to an ICU. Patient dispositions were most commonly to home (57.5%) or to a psychiatric unit (37.5%). There was one death reported.
Conclusions: Tachycardia and agitation were the most common abnormalities reported after exposure to bath salts. The majority of patients required benzodiazepines and hospitalization. Although still legal in some states, drugs commonly sold as bath salts may represent a particularly toxic form of synthetic cathinone.
Keywords: Bath salt, Stimulant, Drug of abuse

291. Pneumorachis, pneumomediastinum, and subcutaneous emphysema after synthetic cannabinoid use

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Background: Pulmonary complications including pneumothorax and pneumomediastinum are well reported after the inhalational use of illicit drugs. Pneumorachis (epidural pneumatosis) has been reported after thoracic trauma, lumbar puncture, weight lifting, and epidural injection. There are 3 medical case reports of pneumorachis after the inhalational use of illicit drugs. This is the first reported case of pneumorachis after synthetic cannabinoid use.
Case report: A 16 year old previously healthy male presents with throat pain, chest pain and upper body swelling that began the morning prior to presentation. His pain is worse with coughing and with movement. He reports 1 day of cough and high-pitched voice. He smoked 1 cigar of "spice" yesterday. He does not report any recent physical trauma or vigorous physical activity.

On exam his temperature is 97.8°F, pulse is 72 bpm, blood pressure is 136/72 mmHg, respiratory rate is 14/min., and oxygen saturation is 98% on room air. The patient is awake and alert and in no distress. His exam is notable for slight decrease in breath sounds at the lung bases bilaterally and for crepitus on his neck, shoulders and upper chest wall.

Chest x-ray shows bilateral neck and upper chest subcutaneous emphysema and pneumomediastinum. Chest CT shows subcutaneous emphysema in the soft tissue of the neck, chest, and abdomen as well as pneumorachis. A barium esophagram is normal.

The patient is hospitalized for 48 hours. During the hospitalization he is stable on room air with resolution of his pain, cough, and high-pitched voice. His crepitus over his neck is still palpable and his pulmonay exam is normal at the time of discharge. His repeat chest x-ray on the day of discharge shows decrease in his subcutaneous emphysema and pneumomediastinum.
Case discussion: This patient developed subcutaneous emphysema, pneumomediastinum, and pneumorachis after inhalational use of a synthetic cannabinoid. Although the exact mechanism for these findings is unclear, 2 plausible explanations exist: excessive coughing leading to barotrauma or forced inspiration against resistance leading to barotrauma.
Case conclusions: Barotrauma associated with inhalational use of synthetic cannabinoids can result in pneumorachis, pneumomediastinum, and subcutaneous emphysema. Our patient had spontaneous improvement in symptoms and radiographic findings with supportive care.
Keywords: Cannabinoid, Synthetic, Drug of abuse, Pneumorachis

292. Comparison of ingested versus inhaled synthetic cathinone exposures

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Background: Among the novel classes of synthetic "designer" drugs that have gained popularity in recent years are synthetic cathinones ("bath salts"). Synthetic cathinones (SC) are used by various routes. It is unclear if the clinical outcomes and adverse events are affected by the route of exposure. The purpose of this study was to compare ingested and inhaled SC exposures.
Methods: SC exposures reported to a statewide poison center system during 2010–2011 were identified and grouped by whether the exposure was by ingestion alone or inhalation alone. Exposures by other routes or involving other substances were excluded. The distribution of exposures was determined for various demographic and clinical factors and comparisons made between the two routes.
Results: There were 80 SC ingestions and 155 inhalations. The ingestion and inhalation patients were 88.8% and 86.5% 20 years or older and 73.8% and 74.2% male, respectively. The patient was already at or en route to a healthcare facility when the poison center was contacted in 66.3% of the ingestions and 72.3% of the inhalations. A potentially serious outcome was reported in 72.5% of

Table 1. Results for abstract 292.

	Ingestions (%)	Inhalations (%)
Clinical effects		
Tachycardia	43.8	45.8
Agitation	33.8	36.8
Hypertension	20.0	19.4
Hallucinations	16.3	17.4
Confusion	13.8	11.6
Chest pain	7.5	6.5
Fever	8.8	6.5
Drowsiness	6.3	6.5
Treatments		
IV fluids	40.0	52.3
Benzodiazepines	26.3	42.6
Oxygen	3.8	9.7
Other sedation	3.8	6.5

the ingestions and 70.3% of the inhalations. The Table 1 provides the rates for the most common clinical effects and treatments. The only comparison with a significant difference was that those who inhaled the SC were more likely to be treated with benzodiazepines (42.6% vs. 26.3%, RR = 1.62, 95% CI 1.07–2.44).

Conclusions: There are no prior data that examined SC exposures based upon the route of exposure. Synthetic cathinone ingestion and inhalation patients were both likely to be adults and male. The adverse clinical effects were similar between the two routes. However, benzodiazepines were more likely to be administered in inhalations. The reason for this difference is unclear.

Keywords: Bath salt, Drug of abuse, Stimulant

293. Comparison of synthetic cathinone and methylenedioxymethamphetamine (MDMA) exposures

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Background: Among the novel classes of "designer" drugs are synthetic cathinones ("bath salts"). Synthetic cathinones (SC) purportedly produce pharmacological effects similar to methylenedioxymethamphetamine (MDMA). This study compared demographics, adverse effects, treatment, and clinical outcomes of SC and MDMA exposures reported to poison centers.

Methods: SC and MDMA exposures reported to a statewide poison center network during 2010–2011 were identified. Exposures involving only SC or MDMA were included; any with additional substances were excluded. The distribution of exposures was determined for various demographic and clinical factors and comparisons made between the two drugs [with relative risk (RR) and 95% Confidence Intervals (CI)].

Results: There were 292 SC and 265 MDMA exposures. The SC and MDMA patients were, respectively, 86.6% and 50.9% 20 years or older and 75.0% and 63.0% male. The most common route of exposure was inhalation for SC (57.2%) and ingestion for MDMA (89.1%). The patient was already at or en route to a healthcare facility when the poison center was contacted in 71% of the SC and 60% of the MDMA exposures. A potentially serious outcome occurred in 73.3% of the SC and 64.5% of the MDMA exposures (RR 1.13, 95% CI 1.01–1.27). The accompanying Table 1 provides the rates for the most common clinical effects and treatments.

Conclusions: Compared to MDMA, SC exposure patients were more likely to be adults and male. SC was most likely to have been inhaled while MDMA was most likely to be ingested. Since SC and MDMA are both phenylethylamines, one would expect similar side effects. The data support this hypothesis. However, the data also show that SC were significantly more associated with adverse effects and serious outcomes.

Keywords: Bath salt, MDMA, Drug of abuse

Table 1. Results for abstract 293.

	Synthetic cathinones (%)	MDMA (%)	RR, 95% CI
Tachycardia	45.5	27.2	1.67, 1.3–2.1
Agitation	37.3	18.1	2.06, 1.53–2.77
Hypertension	19.2	9.4	2.03, 1.30–3.16
Hallucinations	17.8	9.4	1.88, 1.20–2.95
Confusion	12.3	3.0	4.10, 1.93–8.62
Vomiting	9.4	4.1	2.30, 1.18–4.48
Chest pain	7.5	7.9	1.05, 0.59–1.87
Dizziness	6.8	2.7	2.48, 1.1–5.6
Dyspnea	6.4	2.7	2.34, 1.03–5.34
Treatments			
IV fluids	50.0	34.0	1.47, 1.2–1.8
Benzodiazepines	38.4	21.5	1.78, 1.35–2.34
Oxygen	9.6	4.2	2.31, 1.17–4.54
Other sedation	6.8	1.9	3.63, 1.38–9.53

294. Comparison of synthetic cannabinoid and methylenedioxymethamphetamine (MDMA) exposures

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Background: Synthetic cannabinoids have become a popular substance of abuse over the last several years. Synthetic cannabinoids purportedly produce pharmacological effects similar to methylenedioxymethamphetamine (MDMA). This study compared synthetic cannabinoid and MDMA exposures reported to poison centers.

Methods: Synthetic cannabinoid and MDMA exposures reported to a statewide poison center network during 2010–2011 were identified. Exposures involving only synthetic cannabinoids or MDMA were included; any with additional substances were excluded. The distribution of exposures was determined for various demographic and clinical factors and comparisons were made between the two drugs [with relative risk (RR) and 95% Confidence Intervals (CI)].

Results: There were 952 synthetic cannabinoid and 265 MDMA exposures. The synthetic cannabinoid and MDMA patients were, respectively, 55.0% and 50.9% 20 years or older and 75.3% and 63.0% male. The most common route of exposure was inhalation for synthetic cannabinoids (88.4%) and ingestion for MDMA (89.1%). The patient was already at or en route to a healthcare facility when the poison center was contacted in 73.9% of the synthetic cannabinoid and 59.6% of the MDMA exposures. A potentially serious outcome was reported in 62.4% of the synthetic cannabinoid and

Table 1. Results for abstract 294.

	Synthetic cannabinoids (%)	MDMA (%)	RR, 95% CI
Clinical effects			
Tachycardia	37.5	27.2	1.38, 1.11–1.7
Agitation	21.8	18.1	1.2, 0.9–1.6
Drowsiness	17.9	8.7	2.05, 1.36–3.11
Vomiting	15.4	9.4	1.63, 1.09–2.44
Hallucinations	9.9	9.4	1.04, 0.68–1.6
Nausea	9.8	8.7	1.12, 0.72–1.74
Confusion	9	3	2.99, 1.46–6.09
Hypertension	8.5	9.4	0.9, 0.59–1.38
Treatments			
IV fluids	38.9	34	1.14, 0.95–1.37
Benzodiazepines	20.1	21.5	0.93, 0.7–1.2
Oxygen	9.1	4.2	2.2, 1.19–4.0

64.5% of the MDMA exposures. The accompanying Table 1 provides the rates for the most common clinical effects and treatments.

Conclusions: There are no prior data comparing these agents. Compared to MDMA, patients who used synthetic cannabinoids were more likely to be male. Synthetic cannabinoids were most likely to have been inhaled while MDMA was most likely to be ingested. Synthetic cannabinoids were more likely to have hemodynamic and neurological effects. The treatments for both exposures appear to be similar.

Keywords: Cannabinoid, Synthetic, MDMA, Abuse

295. Prolonged, severe agitation and rhabdomyolysis after intravenous injection of a synthetic cannabinoid

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Background: Use of synthetic cannabinoids in the US is common. However, most abusers pyrolyze or orally ingest synthetic cannabinoids, and intravenous use is rare. We report a case of prolonged, severe agitation and rhabdomyolysis after the intravenous injection of a synthetic cannabinoid.

Case report: A 24 year-old female with a prior history of polysubstance abuse purchased incense containing a synthetic cannabinoid from a "head shop" in Illinois. She mixed the incense with water and self-administered the solution three times intravenously via insulin syringes. Shortly thereafter, she became very agitated and ran through a nearby cornfield. The patient sought help from a neighbor who called EMS. Upon arrival to the emergency department, the patient was extremely agitated requiring several doses of benzodiazepines. Initial vitals signs were remarkable for: T = 99.7, BP = 127/58, HR = 140, RR = 30, and O_2 = 98%RA. Initial labs were significant for a CPK of 1842. EtOH and UDS were negative. The patient was admitted to the intensive care unit and required intermittent doses of benzodiazepines for agitation over 48 hours. CPK peaked at 68,744 on hospital day #2. AST/ALT also increased to 572 and 154 respectively: hepatic synthetic function remained normal. The patient did well with supportive care, and all symptoms and laboratory abnormalities resolved by hospital day #3. The patient was later discharged to a psychiatric facility.

Discussion: Synthetic cannabinoids are known to cause agitation and rhabdomyolysis. However, we report a patient with a prolonged course of severe agitation and significant rhabdomyolysis after intravenous injection of a synthetic cannabinoid. Intravenous injection of a synthetic cannabinoid may make titration of the drug more difficult when compared to the typical abuser who pyrolyzes the drug. Intravenous injection also bypasses first-pass hepatic metabolism compared to patients who may orally ingest cannabinoids. Injection of pyrogens may also complicate this route of use.

Conclusions: Agitation and rhabdomyolysis may be more pronounced in patients who parenterally abuse synthetic cannabinoids. Further clinical description and research is warranted.

Keywords: Cannabinoid, Synthetic, Delirium, Drug of abuse

296. Severe poisoning following self-reported use of 25-I, a novel substituted amphetamine

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Background: Abuse of synthetic amphetamine and cathinone derivatives resulting in significant toxicity and occasional fatality is being increasingly reported by poison centers. Chemically unique molecules are developed as soon as existing ones become illegal. Haphazard manufacturing and lack of testing complicate toxicologic assessment. We describe a series of self-reported exposures to 25-I, a novel amphetamine derivative allegedly made in a local makeshift laboratory.

Case series: Ten patients with an average age of 17 yrs (range 14–20 years) presented to local emergency departments in our community after ingestion and/or insufflation of a drug referred to as "25-I". Six of 10 reported 25-I alone; other substances admitted to by the other 4 included ethanol, 2-CE, THC and ketamine. Most common effects included tachycardia (90%), hypertension (70%), agitation (60%) and hallucinations (50%). The average heart rate was 123 beats per minute (range: 78–mid 150s). Two patients were found in status epilepticus and another was found unresponsive. One of the patients who had a seizure was found to have multiple, discrete intraparenchymal hemorrhages and acute kidney injury. Hyperthermia was not documented in any case. In eight patients with available CBC results, the average WBC was 19,913 (range 10,700–29,400). Sixty percent of patients were admitted to the ICU, two were treated in the ED and released, and one each was admitted to psychiatry or managed in a clinical decision unit and subsequently discharged. Three patients required emergent intubation, and all admitted patients (7/10) were given intravenous benzodiazepines for sedation. All patients were discharged in good condition once symptoms resolved. Urine and blood specimens were obtained from several patients are being analyzed for synthetic phenylethylamines and cathinones.

Case discussion: 25-I is thought to represent 25I-NBOMe, which is a (n-benzyl) phenylethylamine in the 2C "family". Although synthesized for research several years ago, there are no published reports of its use as a drug of abuse. In addition to sympathomimetic effects from the phenylethylamine backbone, methoxy and other substituent groups impart serotonergic effects resulting in

hallucinogenic properties. 25-I appears to be potent with a reported "dose" of 500 mcg and can result in greater potential for inadvertent overdose.
Conclusions: This case series describes significant morbidity following self-reported use of 25-I, a newly identified drug of abuse in a cluster of young patients.
Keywords: Amphetamine, Designer drug, Drug of abuse

297. Adverse effects from pediatric exposures to spice (cannabinoid agonists)

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Background: A new and potentially harmful intoxicant known as "Spice" has emerged in recent years. Spice contains plant material mixtures, sold as "herbal incense," and is smoked for potential euphoric effect. Marketed under a variety of names, it is readily available to children despite trends toward legal prohibitions. These plant blends contain compounds that function as potent cannabinoid receptor agonists. Limited data on the clinical effects associated with Spice is available, particularly in children.
Objective: To describe the demographics, clinical effects and medical interventions associated with pediatric/adolescent exposures to Spice, and to determine whether a state law making Spice illegal is associated with changes in reported complications from such exposures.
Methods: This is an observational case series. A retrospective chart review was conducted of all intentional Spice (THC analogues) exposures in patients 0–19 years of age reported to a single US poison center from 11/09–03/12. Data collected and reviewed included age, sex, evaluation at a health care facility (HCF), clinical effects, medical interventions, outcomes and disposition.
Results: 67 reports of pediatric Spice exposures occurred during the study period. Mean age was 17 years (range 11–19), and 66% (n = 44) were male. The most commonly reported adverse effects were: lethargy 28% (n = 19), anxiety/agitation 25% (n = 17), nausea/vomiting 21% (n = 14), confusion 16% (n = 11), chest pain 13% (n = 9), and tremor 12% (n = 8). 15% (n = 10) suffered seizure and 9% (n = 6) had hallucinations. Of the 46 patients (69%) evaluated at a HCF, 74% (n = 34) had tachycardia, mean heart rate 128 (range 96–220), and 7% (n = 3) had hypokalemia. There were single cases of pneumomediastinum, atrial fibrillation, and rhabdomyolysis reported. 30% (n = 14) of HCF cases received medical interventions including naloxone, benzodiazepines, supplemental potassium, oxygen, antiarrhythmic medication, alkalinization of urine and intravenous fluids; and 22% (n = 10) were admitted to the hospital. In the 16 months proceeding the substance being made illegal in this state, an average of 3.19 pediatric exposures were reported to the poison center per month, and in the 13 months after the law was passed, 1.23 per month were reported (rate ratio 2.59; 95% CI: 1.48–4.54). There was a marked reduction in the 4 months immediately after the law was passed, with 0.5 exposures per month reported (0 in the first 2 months).
Conclusions: Children and adolescents exposed to Spice show a broad range of adverse effects. Many require emergency medical interventions and hospitalization. Fewer exposures and adverse events were reported to the poison center after Spice was made illegal.
Keywords: Pediatric, Drug of abuse, Poison center

298. Status epilepticus following use of synthetic marijuana

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Background: Synthetic cannabinoids are sold as incense in convenience stores and on the internet. These products are smoked as an alternative to marijuana. They appear to have far more dangerous side effects than their illicit counterpart.
Case report: EMS responded to a call for a 46-year-old man who had experienced a seizure at home. Prehospital personnel noticed no evidence of trauma, overdose, or environmental abnormalities. During transport to our ED, he experienced two more seizures without a lucid interval, which were aborted by administration of midazolam, 4 mg (total) IV. His blood glucose concentration was normal.

In the ED, two more tonic-clonic seizures occurred, again, with no lucid interval. The seizure activity ceased after administration of diazepam, 10 mg IV. Vital signs: temperature, 36.8 C; pulse, 140 beats/min and regular; respiratory rate, 10 breaths/min; blood pressure, 177/119 mmHg. Rapid-sequence endotracheal intubation was performed for airway protection and management of status epilepticus (SE) (etomidate, 20 mg, and rocuronium, 100 mg). Fosphenytoin, 1 gm IV, was administered. Propofol was used for continuous sedation and to protect against subsequent seizure activity.

A family member reported that the patient had been "smoking incense" earlier in the day and presented a packet labeled "BLACKJACK WILD, 1+ Grams potpourri, legal 50 states."

A chest film showed aspiration in the left lower lung; head CT scan was negative. Laboratory revealed normal electrolyte concentrations and metabolic acidosis, attributed to the seizure activity. Urine toxicology screen was positive for benzodiazepines. Common causes of SE (e.g., infection, endocrine and metabolic abnormalities, structural deformities, illicit drugs) were considered but not suspected. SE has not been reported with synthetic marijuana. The patient exhibited no further seizure activity, was admitted to the medical ICU, and discharged the next day with a diagnosis of SE secondary to use of synthetic marijuana.
Case discussion: Synthetic cannabinoids, e.g., "Spice" and "K2," give users a marijuana-like high. Physical signs of their abuse are consistent with sympathomimetic toxicity. The mechanism involves interaction with the CB1 and CB2 receptors. Synthetic compounds have a 3-fold higher affinity for CB1 than CB2 and a 4-fold higher affinity for CB1 than THC, which may explain the seizure threshold alteration. Synthetic cannabinoids are not regulated drugs in most states and do not test positive on routine toxicology screens. This makes them popular among patients subjected to random drug screens.
Conclusions: Emergency physicians and toxicologists should be aware of these substances and the potential side effects, including SE.
Keywords: Marijuana, Substance abuse, Abuse

299. Self-medication with methoxetamine as an analgesic resulting in significant toxicity

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Background: Methoxetamine (MXE) is a non-scheduled arylcyclohexylamine drug of abuse with clinical effects similar to ketamine or phencyclidine. We report a case of MXE toxicity after self-medicating for treatment of chronic foot pain.
Case: A 29 year old man was brought to the emergency department (ED) for altered mental status. He was apprehended by police after running into the street and lying down in front of oncoming traffic. The patient admitted to sampling a new bag of MXE an hour earlier by dipping a wet finger in the bag and licking it. A plastic bag containing white powder, labeled MXE with CAS number 1239943-76-0 was found in his pocket. On presentation to the ED vital signs included a HR of 117, BP 140/81, RR 28, and Temp of 37.1. He was agitated, combative, and noted to have vertical nystagmus. Neurologic symptoms included: dysphoria, psychomotor agitation, labile mood, and dissociative confusion, including partial amnesia to the preceding events. The patient was given 10 mg IV diazepam in the ED and was admitted for monitoring and was back to baseline by 24 hours. Laboratory analysis for confirmation is pending. He reported using MXE for its analgesic effects to treat chronic foot pain. He had ingested 5–10 mg every four hours × 5 days the week prior for analgesia and a one-time recreational insufflation of 100 mg two weeks before. With all previous use, he denies ever having a dysphoric reaction.
Discussion: MXE is a dissociative anesthetic that has a similar chemical structure and effects as ketamine, including euphoria, dysphoria and analgesia. Recreational use of MXE has been reported and appears to be relatively uncommon. A presumably small dose of MXE taken for chronic pain resulted in toxicity similar to previously described recreational use. This highlights the potential for dosing errors in non-standardized designer drug manufacturing.
Conclusions: This was a case of significant MXE toxicity from self-medicating as an analgesic. Clinical effects were similar to previous case reports from recreational use.
Keywords: Neurotoxicity, Abuse, Designer drug

300. Internet snapshot survey to understand the availability of alpha-methyltryptamine (AMT) in the UK

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Background: Alpha-methyltryptamine (AMT) was investigated in the 1960s as an antidepressant, but psychedelic, stimulant and entactogenic effects limited further development. It is a Schedule I controlled substance in the US, but is legal in the UK as it is not covered by generic tryptamine control. Novel psychoactive substances are often sold from Internet suppliers. Using established European Monitoring Centre for Drugs and Drug Addiction (EMCDDA) Internet snapshot methodology we investigated UK Internet availability of AMT.
Methods: The Internet search engine "google.co.uk" was searched (March 2012) for AMT Internet suppliers. Data was extracted on the amount (mass of powder, number of capsules/pellets) and cost of purchase. Sampling was undertaken to exhaustion: the first 100 Internet sites were reviewed in full then sampling continued until 20 successive unrelated sites were identified.
Results: A total of 44 Internet sites sold AMT: 30 powder, 10 pellets, 4 capsules; 41 quoted prices in UK£, 3 in US$; none sold in Euros. One site sold combined AMT and MDAI (5,6-Methylenedioxy-2-aminoindane) powder (£121 for 10 g).
AMT powder: The majority (30/44) of sites sold AMT powder (29 UK£, 1 US$), 7 sites sold bulk quantities ≥ 100 g. Price per gram of AMT powder reduced from £75–249.90 (100 mg purchase) to £30–80 (500 mg) to £20–64 (1 g) to £15–45 (5 g) to £13–40 (10 g). Bulk purchases (100–250 g) were £8.50–£15 per gram. On the site selling in US$, AMT was $119.90 per gram (100 mg purchase).
AMT pellets: 10/44 sites sold AMT in pellets (9 UK£, 1 US$), 3 sites sold bulk quantities ≥ 1000 pellets. Price per AMT pellet reduced from £6.50 (1 pellet) to £3.50–£5.00 (10) to £2.00–£4.00 (100). Bulk purchases (1000 pellets) were £1.50–£3.00 per pellet. The site selling in US$, price reduced from $7.20 (2 pellets) to $4 (100); bulk purchases (1000 pellets) were $2.40 per pellet. There was no indication on the quantity of AMT per pellet on any site.
AMT capsules: 4/44 sites sold AMT in pellets (3 UK£, 1 US$). Price per capsule reduced from £8.49 (1 capsule) to £2.33 per capsule (15) to £2.00 per capsule (40) to £1.67 per capsule (150). On the site selling in US$, AMT was $11.90 for a single capsule. 2/4 sites provided information on capsule content(30–34 mg AMT/capsule).
Discussion: This study showed that AMT was widely available in powder, capsule and pellet from in the UK in March 2012 from Internet sites selling in both UK£ and US$, with decreasing price with increasing purchase size. Users report desired effects with 20–30 mg of AMT, therefore it appears AMT is sold in both user and dealer quantities. Use of Internet snapshot survey methodology can be useful in determining availability of novel psychoactive substances.
Keywords: Substance abuse, Designer drug, Alpha-methyltryptamine

301. Pseudo-ergotism resulting in quadruple amputation after DOI abuse

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Background: 2,5-dimethoxy-4-iodoamphetamine (DOI) is a weak serotonin agonist predominately used in neuropsychiatric research. Because of its serotonergic properties, this drug has abuse potential as a hallucinogenic agent. Currently, DOI is not regulated in the United States and is occasionally sold as a substitute for lysergic acid diethylamide (LSD). To date, there are no published reports of DOI abuse. We report a case of a patient who underwent quadruple limb amputation after DOI abuse.
Case report: A 21-year-old male presented to a suburban community hospital after reportedly ingesting, then injecting DOI intramuscularly then intravenously. Shortly thereafter, he sought evaluation for worsening paresthesias in his upper and lower extremities. Reportedly, he injected the drug because he was not getting the desired hallucinogenic effect.

Our regional poison center was consulted five days into the patient's hospital course, after having developed myocardial ischemia with elevated troponins and a reduced ejection fraction. He also had progressive cyanosis in all four extremities and a creatine kinase of 38,000. The patient was subsequently transferred to a tertiary care facility, where he received NTG paste to the extremities, heparin, dobutamine and dextran. Additional vasodilator therapy with a dihydropyridine calcium channel blocker was further recommended upon consultation with the on-call toxicologist.

Despite aggressive support and peripheral vasodilating agents, the patient developed ischemia in all his extremities. After demarcation of the ischemic injury, he underwent surgical amputation of four extremities below the knees and elbows.

Discussion: Ergotamines and ergot derivatives agonize alpha-adrenergic and serotonin receptors. Serotonergic agonists are believed to induce vasoconstriction through direct activation on smooth muscle, activation of alpha-adrenoceptors, and activation of additional vasoconstrictors such as norepinephrine. Resulting vasospasm has been associated with cardiac ischemia, as well as extremity paresthesias with peripheral artery constriction progressing to gangrene. DOI, like an ergot, is a serotonergic agent which structurally resembles 4-bromo-2,5-dimethoxyamphetamine (DOB) and Bromo-dragonfly, both of which have been reported to cause vascular insufficiency and subsequent bilateral amputation of the lower extremities.

Conclusions: This is the first reported case of DOI induced ergotism resulting in extremity necrosis and subsequent quadruple amputation. Treatment options may include intra-arterial vasodilators, dihydropyridine calcium channel blockers, anticoagulants, hyperbaric oxygen and surgical sympathectomy.

Keywords: Abuse, DOI, Hallucinogen

302. A retrospective study of cases of 'bodypackers' and 'bodystuffers' admitted to the Cardiff Poisons Unit from 2001 to 2009

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Background: The Cardiff Poisons Unit is a ward dedicated to the treatment of adult poisoned patients. The management of individuals who swallow illicit drugs for concealment is difficult, with risk of package rupture leading to a large quantity of drugs being rapidly released into the body. Such patients are often admitted whilst in police custody and may not give a reliable history.

We aim to study patients admitted to the poisons ward from 2001 to 2009 suspected of concealing drugs within the body.

Method: The admissions database was searched for all patients classified as bodypackers or body stuffers. Patient notes were retrieved and details regarding treatment and outcome recorded.

Results: Sixty cases (49 male and 11 female) recorded as body packers or stuffers were identified for this period. Of patients questioned regarding current drug use, 37 admitted to heroin use, 20 regularly used cannabis and 12 used cocaine; 5 did not use drugs. The majority of patients (n = 33) were suspected of swallowing packages to avoid detection by police (stuffers), 16 had inserted drugs per rectum and 6 had swallowed drugs packaged for smuggling purposes (packers).

Ten patients refused imaging investigations, 20 did not receive imaging for unspecified reasons. Fifteen chest x-rays were negative for packages. Of 20 abdominal x-rays performed 4 were positive and 2 undecided. Two patients received computed tomography, one negative and the other showing multiple packets. A urine toxscreen was performed in 26 of the patients: 11 were positive for benzodiazepines; 9 for cocaine and opiates; 8 for cannabis; 3 for methadone and 1 each for amphetamine and barbiturates.

Five of the patients refused treatment. A laxative was given to 31 patients. One patient underwent emergency surgery secondary to package rupture and did not survive. Three individuals were treated for opiate withdrawal whilst admitted.

In 18 cases packages were retrieved, in the majority of cases (n = 9) just one package. The largest number of packages retrieved was 50 (in the patient who died).

Conclusions: Other cases may have been admitted but not coded as 'packers' or 'stuffers'. As 92% of cases admitted to some recreational drug use, urine screening is often not of great value in assessing package rupture. Many patients are drug users and may suffer withdrawal symptoms whilst in hospital and so may require treatment unrelated to the actual drug concealment. Lack of patient cooperation or reliable medical history adds to the difficulty of managing patients to a standard protocol.

Keywords: Drug of abuse, Bodypacker, Foreign body

303. Nasopharyngeal necrosis after chronic opioid (oxycodone/acetaminophen) insufflation

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Background: Nasopharyngeal necrosis results from narcotic insufflation, but cocaine use is more commonly associated with this pathology than opioid abuse. Physical exam findings associated with severe tissue destruction are not routinely seen on physical examination or available in the medical literature. We present a case of chronic oxycodone/aceteminophen insufflation and images of a severely damaged soft palate.

Case report: A 25 year-old female presented to the Emergency Department (ED), with stable vital signs, complaining of a headache. She stated that her primary care doctor, working in concert with an otolaryngologist, instructed her to return to the ED any time she had a significant headache. Her past medical history was significant only for crushing and insufflating diverted Percocet ® tablets for many years. She states that she has been sober for almost a year and denies taking any prescription or illicit drugs during that time. She denies insufflating any other medications or illicit drugs besides Percocet ®. On physical exam, she has hypernasal speech (i.e. cleft palate-like misarticulations), no visible external facial deformities, and no skin changes. Visual inspection of her oropharynx can be seen in the attached images. She denies any complaints other than a mild general headache.

Case discussion: The attached images demonstrate soft palate necrosis, with no appreciable damage to the hard palate. Her soft palate destruction was a continuation of the cartilaginous destruction evidenced by her obliterated nasal septum. Despite her young age, this patient had a significant opioid addiction, and her only route of administration was insufflation over > 10 years. Due to an increased risk of infection (resulting from loss of facial structures), the patient was started on oral clindamycin and instructed

to follow up with outpatient otolaryngology consultation. Per the patient, there were plans for her to have a corrective surgery in the near future. This patient gave consent for her images to be used for publication and education.
Conclusions: Without a patient's specific description of drug diversion and insufflation, it is possible to miss the physical exam findings of nasal septum destruction or soft palate necrosis. Performing a careful physical exam of the nasal turbinates and the oropharynx may help the treating physician to better assess the patient's risk factors for infection. This patient had hypernasal speech, no visible nasal septum, and a defect in her soft palate. Treating physicians should be aware of the risk for infection, need for possible antibiotics and neuro-imaging, and be in contact with otolaryngology or corrective surgical specialty.
Keywords: Abuse, Opioid, Drug of abuse

304. Significant hand sanitizer intoxication following crude extraction method with in vitro ethanol concentration analysis

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Background: Alcohol-based hand sanitizers are recommended by many public health authorities as an alternative to washing for hand hygiene. Containing ethanol and isopropanol, these products provide a source of intoxication in otherwise controlled settings such as prison or hospital. We describe a case of an incarcerated psychiatric patient who consumed sanitizer and became significantly intoxicated. Anaylsis of the ethanol and isopropanol content of the product following the separation method described by the patient was performed.
Case report: A 40 year-old patient in a locked psychiatric facility was found minimally responsive in her room along with two empty 1 L bags of hand sanitizer. She was intubated for respiratory depression. Vital signs were normal. Toxicology studies revealed a blood alcohol concentration of 382 mg/dL. Renal and liver function, chemistry panel, blood count, blood gas analysis and a head CT were unremarkable. The patient was extubated after approximately 24 hours of supportive care and reported mixing the contents of hand sanitizer gel with table salt, then running the solution through a sock to obtain a consumable liquid. She denied any other ingestions and had no further adverse effects.
Methods: 2 TBS of Table salt were added to 2 TBS of hand sanitizer (labeled 62% ethyl alcohol), and then filtered through a cotton sock. Both the gel and resulting liquid were sent for determination of ethanol and isopropanol concentration by two different Methods: NADH generating enzymatic (EM) and headspace gas chromatography (H-GC).
Results: Prior to separation, the ethanol content was 4084 mg/dL by EM and 4380 mg/dL by H-GC. After separation, the ethanol content via EM was 5278 mg/dL and 4200 mg/dL via H-GC.
Discussion: Previously published reports detail intentional hand sanitizer ingestions and subsequent intoxications in a variety of at-risk populations; however none have included psychiatric patients in locked facilities. When tested, the parent hand sanitizer contained less than the stated 62% ethanol by volume via both methods and was 29% more concentrated with EM and slightly less concentrated with H-GC.
Conclusions: Despite the conflicting results from our in vitro analysis following a crude extraction method, alcohol-based hand sanitizer ingestions continue to represent a source of significant intoxication for patients in otherwise controlled settings. Tamper-resistant packaging should be employed when utilizing these products with at-risk populations.
Keywords: Alcohol, Drug of abuse, Ethanol

305. Death from ingestion of an ethanol-based hand sanitizer in the emergency department waiting room

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Background: Alcohol-containing hand sanitizers (HS) are present throughout hospitals and ingestions have caused significant intoxication. One report details a death from a non-ETOH-containing HS. We report the first death from the ingestion of an ETOH-based HS.
Case report: 36-yr-old presented with ETOH intoxication. 3 days prior he was administered 50 mg chlordiazepoxide for ETOH WD and DC/ed without prescription. Meds included aripiprazole and quetiapine for schizophrenia (compliance unknown). Vital signs: T-98.2°F, P-106, BP-114/76, RA pulse ox 98%. He was alert, uncooperative, with slurred speech. Blood glucose was 131 mg/dL and a *breathalyzer registered 278 mg/dL.* 4.5 hrs after ETOH measurement he was calm, cooperative, and left the ED with a steady gait. 30 minutes later he was found unresponsive, pulseless, and asystolic in the ED waiting room bathroom adjacent to an empty container (1000 mL) of HS (Purell® Instant HS) removed from the wall (unknown pre-existing quantity). The label describes 62% ETOH, with isopropanol and propylene glycol as other ingredients. No other ETOH-containing products were found. Soon after CPR, ET intubation, and IV administration of epi, vasopressin, and Na bicarb he regained circulation. Initial ECG revealed: sinus tachycardia (137), normal QRS interval, QTc 555 ms, no ischemic changes. Abnl labs soon after resusc: pH-7.05, pCO_2-85; K-3.1, bicarb-19, Ca-1.02 mmol/L (1.13–1.32), ALT-1037 U/L, lactate-101 mg/dL (4.5–19.8). Na, Cr, INR/PTT, bili, trop, CK, Mg, and CBC were nl. Serum ETOH was 526 mg/dL; salicylates and APAP were not detected. Serum ketones were positive at 1:2 (osmolarity, isopropanol not measured). Brain/cervical spine CT, and chest xry were nl. Echocardiogram day 2 was unremarkable, trop (1.58 ng/mL) and liver enzymes peaked day 2 and were attributed to initial hypoperfusion. Day 3 an MRI revealed findings consistent with anoxic brain injury. Care was withdrawn day 7 and he expired. UDS following resusc was + for benzos only and LC-MS/MS revealed nordiazepam (69 ng/mL), and oxazepam (393 ng/mL); chlordiazepoxide metabolites and concentrations consistent with therapeutic use.
Case discussion: The pt appeared to suffer a respiratory arrest following the acute ingestion of an ETOH-containing HS. It seems likely that individuals who ingest HS may not anticipate the potential for rapid absorption of a large quantity of ETOH. This appears to be the first death reported from the ingestion of an ETOH-containing HS.

Conclusions: The case highlights the potential for harm after the intentional ingestion of a HS easily accessible in a hospital. Health care facilities may consider preventive steps to limit the access of HS to patients.
Keywords: Ethanol, Alcohol, Hand sanitizer

306. Neuro-psychiatric features of acute withdrawal from Gamma-hydroxybutyrate and its analogues

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Background: Regular use of gamma-hydroxybutyrate (GHB) or its analogues gamma-butyrolactone (GBL) and 1,4-butanediol (1,4BD) is associated with physical dependency and withdrawal on cessation of use. We describe here the neuro-psychiatric features seen in a large cohort of individuals with presentations to hospital with acute GHB/analogue withdrawal.
Methods: Patients presenting to our large inner city hospital with acute GHB or analogue withdrawal between May 2008 and September 2011 were identified retrospectively from our prospective clinical toxicology database. The following data was then extracted from the medical records of these presentations: age, sex, dose of GHB/GBL/1,4-BD used per day, concurrent use of alcohol/other drugs, any neuro-psychiatric symptoms and their management.
Results: There were 31 acute GHB or analogue withdrawal presentations in 20 different patients; 17 (85%) male and 3 (15%) female. Mean $\pm$ SD age was 27.5 $\pm$ 4.3 years. The median (inter-quartile range) amount of GHB/analogue used per day was 27 (18–37) ml. 22 (71%) reported use of other drugs including methamphetamine (32.2% of individuals), cocaine (22.5%), alcohol (19.4%), MDMA (9.7%), mephedrone (6.5%), ketamine (3.2%) and cannabis (3.2%). 17 (54.8%) presentations were reviewed by liaison psychiatry; 29.4% of those required review by an attending level doctor. The commonest neuro-psychiatric symptoms during withdrawal were anxiety and agitation in 19 of presentations (61.3%) and 15 (48.4%) respectively; other neuro-psychiatric symptoms recorded included tremor (38.7%), hallucinations (35.5%), paranoia (19.4%), confusion (16.1%), amnesia (12.9%), delusions (12.9%) and sexual dis-inhibition (9.7%). Five individuals required one-to-one observation by a mental health nurse due to their symptoms during their withdrawal. In addition to our standard use of benzodiazepines and/or baclofen for GHB/analogue withdrawal, 2 individuals required treatment with anti-psychotics (olanzapine, quetiapine). 7 (22.6%) were admitted to the Intensive Care Unit due to the severity of their symptoms and 3 required sedation and endotracheal intubation for ongoing withdrawal management.
Conclusions: Acute GHB or its analogue withdrawal can be associated with severe neuro-psychiatric symptoms, which may require high-levels of liaison psychiatry intervention, use of anti-psychotic medication and/or admission to an intensive care unit for symptom control. Clinical toxicologist involvement to improve GHB/analogue withdrawal management pathways may help to reduce the severity and/or occurrence of these neuro-psychiatric symptoms.
Keywords: Substance abuse, Delirium, Withdrawal

307. Acute cardiomyopathy in a teenager using "Molly's Mosquito Cap" identified as fluoroamphetamine and prescription modafanil

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Background: Recently there has been a surge of cleverly disguised substituted amphetamines, with unpredictable clinical responses. Anecdotal reporting by patients and healthcare professionals without positive identification has only increased the uncertainty and inaccurate speculation about the clinical effects and epidemiology. We present a case of an 18 year old female who developed life threatening cardiomyopathy after ingesting fluoroamphetamine and prescription modafanil.
Case report: An 18 year old female reportedly drank two capfuls of "Molly's Mosquito Cap," insufflated 110 mg of methylphenidate and ingested 800 mg of modafinil with recreational intent. Once the euphoric effects resolved she experienced headache, nausea, vomiting, weakness and diaphoresis. Her presenting vital signs were BP 95/65 mmHg, HR 110–124 bpm, T 97.7°F. Relevant diagnostic testing included: ECG sinus tachycardia 115 bpm, no ischemic changes, QRS 82 ms, QTc 470 ms; serum bicarbonate 20 mg/dL, AG 17, glucose 166 mg/dL, urine drug screen positive for amphetamines and THC. Troponin I increased from 0.15 ng/mL to 5.4 ng/mL in five hours and decreased to 5.36 ng/mL 10.5 hours after the initial troponin. The CK increased from 74 mg/dL to 335 mg/dL over the same time period. An echocardiogram $>$ 36 hours from exposure revealed EF of 15–20%, dilation of the left ventricle, markedly reduced systolic function, diffuse hypokinesis and mild mitral regurgitation. Chest CT revealed no pulmonary embolism, but diffuse extensive ground glass opacities greatest in lower lobes. She was treated with benzodiazepines and supplemental oxygen and she recovered in four days. The urine was tested by GCMS and fluoroamphetamine and modafinil were detected, but methyphenidate was not.
Case discussion: Cardiomyopathy has been reported with MDMA, ephedrine and methlyphenidate, even with therapeutic doses. The mechanisms of amphetamine-induced cardiomyopathy are multifactorial and may include supply-demand ischemia and myocyte cell death leading to global dysfunction of the left ventricle. It is unclear if the presence of the modafanil represented a therapeutic or toxic amount. The presumptively positive urine amphetamine screen was related to the fluoroamphetamine because the modafinil will not trigger a positive finding, and no other amphetamines were detected on GC/MS. Both subtances in this case may have contributed to her cardiomyopathy.
Conclusions: Acute dilated cardiomyopathy occurred in a young female exposed to "Molly's Mosquito Cap" identified as fluoroamphetamine and prescription modafanil.
Keywords: Amphetamine, Cardiac toxicity, Designer drug

308. What? Did you say opioid induced hearing loss?

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Table 1. Data for abstract 308.

Cases	28 yo male altered with history of oxycontin abuse	14 yo female lethargic after spending the night at a friend's home	18 yo male unarousable after snorting two lines of crushed morphine tablets	31 yo male unresponsive heroin and alprazolam intoxication
Hearing loss	+	+	+	+
Altered mental status	+	+	+	+
Narcan given/effect	Given, no effect	Not given	Given, no effect	Unknown
Tachycardia	+	+	+	+
Hypotension	–	–	+	+
Negative apap/aspirin levels	+	+	+	+
Renal dysfunction	+	+	+	+
Elevated troponins	+	+	+	+
Elevated transaminases	+	+	+	+
UDS positive	Opioids, benzodiazepines, cannabis	Opioids-oxymorphone	Opioids-morphine	Opioids, benzodiazepines
Follow up	Unable to obtain	Resolved	Resolved	Unable to obtain

Background: Opioid use and misuse is increasingly prevalent and misuse of prescription opioids has been associated with specific complications that may contribute to the diagnosis of opioid poisoning. Between 1999 and 2006, the number of fatal poisonings associated with opioid use tripled. In 2009, there were 475,000 emergency department visits secondary to abuse of opioid analgesics, a number that has doubled from 2004. Multiple drugs including aminoglycosides, macrolides, vancomycin, loop diuretics, cisplatin, quinine, and salicylates are associated with ototoxicity. Chronic abuse of opioids has been shown to be ototoxic. However, acute hearing loss after acute opioid poisoning has been poorly characterized. We describe four patients who presented with altered mental status and acute hearing loss; opioid exposures were suspected and later confirmed.
Case reports: These four cases have multiple similarities despite the various type of opioid exposure. They have been summarized in the following Table 1.
Case discussion: These four cases demonstrate acute hearing loss following various acute opioid exposures. This complication is being reported more commonly, but may be attributed to the increased prescribing and misuse of opioids. There are multiple suggested mechanisms for the hearing loss including temporal lobe ischemia, genetic polymorphisms of drug-metabolizing enzymes, and cochlear ischemia. Although not well elucidated, the mechanism may be related to cerebral hypoperfusion as many of the cases demonstrated other end organ toxic effects. It appears to occur secondary to a wide range of opioid use including heroin, morphine, oxycodone, and methadone.
Conclusions: With the increasing prescribing of opioids it is important for healthcare providers to be aware of the association between opioid poisoning and hearing loss. Opioid toxicity should be considered in any patient with acute hearing loss after poisoning.
Keywords: Opioid, Overdose, Drug of abuse

309. Oxymorphone insufflation associated with acute sensorineural hearing loss

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Background: Acute bilateral hearing loss is an uncommon presentation to the ED. Many xenobiotics have been associated with acute sensorineural hearing loss, but opioids are rarely the cause. Case reports of heroin, codeine, hydrocodone, methadone, and propoxyphene are linked with opioid associated hearing loss (OAHL), but no case reports associate oxymorphone with OAHL. Oxymorphone is a prescribed oral opioid that is increasingly being abused via crushing tablets for insufflation.
Case report: A 37 year-old male, found in the basement of his mother's home, had miosis and sonorous respirations. Naloxone produced rapid improvement of mental status and hypopnea. On arrival to the ED, the patient's vital signs were stable, and he was alert and oriented to person and place. He admitted to drinking alcohol earlier in the day ([EtOH] 113 mg/dL) and then snorting oxymophone shortly before EMS arrived. He complained only of hearing loss, stating that he felt like he was "in a tunnel." He denied trauma, headache, vision changes, ataxia, or tinnitus. On physical exam, he had no neurologic deficit other than bilateral hearing loss that spontaneously resolved after 5 hours. The patient's urine oxymorphone and oxycodone concentrations on arrival were 1274 ng/mL and 1055 ng/mL respectively. After 22 hours in the hospital, those levels dropped to 34 ng/mL and 16 ng/mL respectively. Urine toxicology testing confirmed the presence of the following compounds: nicotine, diphenhydramine, ibuprofen, citalopram, oxcarbazepine, and cannabinoids. Other than marijuana, the patient stated that he was prescribed the remaining medications by his primary care doctor.
Case discussion: The presence of oxymorphone solely as a metabolite of oxycodone is unlikely, given the initial ratio of oxymorphone to oxycodone. It is possible that oxymorphone, as an oxycodone metabolite, contributed to the elevated oxymorphone level. The patient admitted to taking Percocet ® as prescribed by a doctor, and his non-detectable acetaminophen levels argue against acute Percocet ® exposure. He admitted to purchasing oxymorphone illegally from the Internet. The patient did not know if he purchased oxymorphone IR or ER. He was therefore admitted for 24 hours of observation. We could not obtain a sample from the Internet for forensic analysis. He experienced no recurrent hearing loss while in the hospital.
Conclusions: Oxymorphone insufflation appears to cause abrupt sensorineural hearing loss. Neither the mechanism of action, nor the association between timing of opioid use and OAHL have been established. Assuming zero order kinetics for oxymorphone elimination, the patient's hearing recovered at a urine [oxymorphone] ~992 ng/mL.
Keywords: Opioid, Overdose, Abuse

310. Review of intentional abuse of quetiapine exposures

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Introduction: There are several case reports of abuse of quetiapine, but no studies specifically addressing disposition and outcomes from intentional quetiapine abuse/misuse. In addition, most literature on the population that abuses quetiapine reports an older age group with previous substance abuse history, many of whom are in jail. Documenting nonmedical use of quetiapine using a single database would provide important information concerning toxicity in a typical poison center population. The purpose of this study is to evaluate quetiapine exposures involving misuse/abuse reported to a regional poison center.
Methods: A retrospective study of poison center data on single substance quetiapine exposures was performed. The study included cases from 2005 to 2011 with reason coded as intentional abuse or intentional misuse. Data were evaluated for age, disposition [e.g. on-site, emergency department (ED), health care facility (HCF) admission], treatments performed and coded final outcomes.
Results: There were 95 cases meeting inclusion criteria, of which reason was misuse in 71 and abuse in 24. Age breakdown for misuse and abuse were 15 and 10 cases between 6 and 12 years; 50 and 13 cases between 13 and 19 years; and 6 and 1 cases >19 years, respectively. No major effects or deaths were documented; 9.5% experienced moderate toxicity. Disposition and medical outcomes are displayed in the Table 1. Activated charcoal was administered to 9, benzodiazepines/other sedation to 3 and antihistamines to one patient.
Conclusions: The at-risk age group is adolescents who accounted for 66% of cases. Disposition and coded outcomes indicate that most quetiapine misuse and abuse cases do well.
Keywords: Antipsychotic, Abuse, Poison center

Table 1. Results for abstract 310.

	Misuse (%)	Abuse (%)	Total (%)
Disposition			
On-site non HCF	17 (23.9)	3 (12.5)	20 (21.1)
Managed in HCF			
Treated/Released ED	28 (39.4)	14 (58.3)	42 (44.2)
Admit critical care	2 (2.8)	3 (12.5)	5 (5.3)
Admit non-critical care	5 (7.0)	1 (4.2)	6 (6.3)
Admitted to psych	10 (14.1)	1 (4.2)	11 (11.5)
Lost to follow-up	3 (4.2)	2 (8.3)	5 (5.3)
Refused referral/other	6 (8.4)	0	6 (6.3)
Disposition total	71 (100)	24 (100)	**95**
Outcome			
None	2 (2.8)	1 (4.5)	3 (3.2)
Minor	34 (47.9)	13 (59.1)	47 (49.5)
Moderate	6 (8.5)	3 (13.6)	9 (9.5)
Minimal effects possible*	25 (35.2)	4 (18.2)	29 (30.5)
Unable to follow-pot. toxic	4 (5.6)	1 (4.5)	5 (5.3)
Outcome total	71 (100)	22 (100)	**93****

*Coded as 'not followed, minimal clinical effects possible'.
**2 cases with unrelated effect (exposure probably not responsible) were excluded.

311. Emergency department alcohol intoxicated patient resource utilization & length of stays

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Background: Alcohol contributes to many medicinal and surgical problems both in the short and long term. Its use contributes to a significant morbidity and mortality to the American population. Intoxicated patients require a significant amount of emergency department resources and stay in the emergency department for prolonged periods. The purpose of our study was to quantify the amount of emergency department resources utilized. Further, we looked to see if there was a trend for increased resource utilization and length of stay that was dependent on the level of intoxication.
Methods: Our study was a retrospective chart review of all emergency department visits between April 6, 2005 to December 31, 2009 with a primary or secondary diagnosis of alcohol intoxication. We randomly selected 125 patients from these dates. Data obtained included patient demographics (age, sex, occupation, insurance status), triage level, mode of arrival, length of stay, initial breathalyzer levels, initial and discharge vital signs, use of physical or chemical restraints or a sitter, use of labs, use of diagnostic imaging, presence of trauma or agitation, need for psychiatry evaluation, and evaluation by HPA (Health Project Assert). Subjects were then divided into quartiles based on breathalyzer levels for data analysis.
Results: 115 subjects had complete data. The average breathalyzer was 0.174 with an average length of stay of 388.62 minutes. 73% of our patients were male. We were unable to predict length of stay based on breathalyzer levels. We found that with increasing levels of intoxication, patients were more likely to be older, require physical restraints, require a sitter and were more likely to be agitated. There was no correlation between intoxication and pharmacological restraints, evidence of trauma, lab or diagnostic imaging utilization, need for psychiatry evaluation, request for detoxification, or evaluation by HPA.
Conclusions: Alcohol intoxication is a diagnostic dilemma for many clinicians. Intoxicated patients experience prolonged length of stays in our emergency departments waiting for sobriety and repeat evaluations to assess safety. They utilize a significant number of emergency department resources in what is an already scarce resource environment.
Keywords: Alcohol intoxication, Resource utilization, Emergency department

312. Plastered on pruno: Severe intoxication from prison hooch

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Background: Ethanol is one of the oldest and most widely used drugs in the world and is often used to alter consciousness. Produced by the fermentation of carbohydrates by yeast, ethanol can be made from a wide variety of sugar-containing products including fruits and vegetables. Pruno, also known as "prison wine" or

"hooch," is an alcoholic beverage made from easily obtainable ingredients such as ketchup, apples, oranges, potatoes, and sugar. Instructions for brewing pruno can be readily found on the internet, and pruno has long been reported to be made in prison.

Case report: An otherwise healthy 22-year-old male presented to an emergency department (ED) from a military prison (brig) with acutely altered mental status. He had been found down in his cell with a depressed level of consciousness, lying next to a bottle of mouthwash. In the ED he was uncooperative and became increasingly agitated, requiring sedation with droperidol and midazolam. The mouthwash bottle was noted to be filled with a foul-smelling, thick brown liquid containing particulate matter. The patient's vital signs were normal with the exception of tachycardia (111 beats/min). Physical exam was remarkable only for agitation. Laboratory analysis revealed a serum ethanol level of 279 mg/dL. Chemistries, glucose, renal and liver functions were normal. Toxic alcohol screening was negative for methanol, ethylene glycol, and isopropanol. Urine drug immunoassay and noncontrast head CT were negative. The patient was admitted and his mental status returned to baseline within 12 hours; he denied ingestion of other substances besides pruno. He was discharged to the brig in his usual state of health.

Discussion: Multiple recipes for pruno can be found online. Typically, sugar-containing products such as fruit, potatoes, ketchup, or table sugar are allowed to ferment in garbage bags or barrels for up to 7–10 days. There is a general paucity of published literature on pruno and other alternative ethanol sources in the incarcerated patient population. In addition to awareness of pruno as a readily available source of ethanol in prison, clinicians should be aware of other potentially serious complications from pruno ingestion. In 2004, several California prison inmates suffered from botulism and required prolonged intubation after ingesting a potato-based pruno product.

Conclusions: Pruno represents a potential source of ethanol for incarcerated individuals seeking to alter their consciousness and should be considered in the care and treatment of the prisoner with altered mental status. In addition to ethanol intoxication, unsanitary brewing conditions may contribute to other complications from pruno ingestion, including previously reported cases of botulism.

Keywords: Internet, Ethanol, Drug of abuse

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