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Abstracts of the 2003 North American Congress of Clinical Toxicology Annual Meeting

1. FOXY METHOXY: A NEW DRUG OF ABUSE

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Background: In 1999, a new synthetic tryptamine, 5-MeO-DIPT, became known as a street drug, with the street name of "Foxy" or "Foxy Methoxy." By February 2003, DEA reported law enforcement seizures and/or reports of abuse in 12 states. We report the first published case of toxicity. Methods: After an index case, AAPCC TESS data were examined to determine trends and extent of abuse of this agent. Case Report: A 19 year-old, multiple-pierced, male was brought to the emergency department following ingestion of "Foxy." Upon arrival, he displayed hallucinations, hypertension, tachycardia, mydriasis, and catalepsy, inability to answer questions, and staring into space with eyes open. His extremities displayed a waxy plasticity, without rigidity, but remaining in whatever position the examiner placed them. Laboratory values showed hyperglycemia, increased white count with left shift, glycosuria, and UDS positive for cocaine and phencyclidine. Symptoms resolved within several hours after administration of lorazepam, and he recovered uneventfully. Once awake, he denied use of cocaine and explained that he had never taken so much "Foxy," which he described as a research chemical used at work. Results: The AAPCC TESS database contained 27 exposures to "Foxy" between April, 2002 and February, 2003; 21 had moderate or major effects, indicating this drug has significant toxic potential. The most common finding was hallucinations (13 cases) and agitation (15 cases). Tremor and dystonia were also reported. The geographic area expanded since first appearance to involve three states by July 2002, 7 by October 2002, and 12 by December 2002. Conclusion: Given the expanding use of this and other club drugs, the spectrum of toxicity from this new agent will continue to be elucidated.

2. SEROTONIN SYNDROME FOLLOWING INITIATION OF ESCITALOPRAM MONOTHERAPY FOR DEPRESSION

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<u>Background</u>: Serotonin syndrome (SS) is an adverse effect of serotinergic medications. We present the first case-report, to our knowledge, of SS precipitated by Escitalopram monotherapy. <u>Case Report</u>: A 75 year-old Cuban-American male with a history depression presented with a one-day history of altered mental status, fever, and new onset bilateral upper extremity tremor. The day prior the patient had been prescribed escitalopram for depression. He had taken a 10 mg dose each morning for 2 days. The patient had not been on an SSRI previously nor was he taking any other serotinergic medications. Initial exam demonstrated: temperature of 103.3, blood pressure 219/82, and heart rate 95. Neurologic exam was remarkable for an ataxic gait, rigidity of the upper and lower extremities with a bilateral, symmetric tremor present with movement and at rest. Clonus was present as well. All laboratory tests and imaging studies were unremarkable. Urine toxicology demonstrated Citalopram. With observation the patient rapidly defervesced, his vital

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signs returned to normal limits later that evening and his mental status and tremor improved by 48 hours. He was discharged on Hospital day 3 with complete resolution of his symptoms. Escitalopram had been stopped at admission. A presumptive diagnosis of SS, based on criteria set forth in the medical literature, was made. <u>Conclusion</u>: Serotonin syndrome may be seen with SSRI monotherapy. Diagnosis is based on medication history, toxicoligic analysis and physical exam. A careful medication history looking for other serotineric agents as well as advising patients of the possibility for this adverse effect should be done when initiating SSRI therapy.

3. PEDIATRIC OVERDOSE OF DESMOPRESSIN ACETATE

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Background: Desmopressin acetate (DDAVP[®]) is a synthetic analogue of antidiuretic hormone used primarily for the treatment of central diabetes insipidus and primary nocturnal enuresis. To date, there have been no reported human acute overdoses of desmopressin. Profound hyponatremia associated with seizures are documented as serious adverse reactions reported with therapeutic use. Case Report: A 7 year-old, 38 kg female, with a medical history significant only for ADD and nocturnal enuresis, ingested 40-50 0.1 mg desmopressin tablets. She presented to the ED 2 hours after ingestion and received 30 g of activated charcoal. An IV of 0.9% sodium chloride was started at a maintenance rate. Routine toxicology labs were all negative, and complete blood count, urinalysis and liver enzymes were within normal limits. Her blood chemistry, 90 min post ingestion, showed: Na = 139, K = 3.8, Cl = 106, HCO₃ = 24, SCr = 0.7, BUN = 14, Glucose = 81, and serum osmolality = 288. Oral intake of free water was restricted for 8 hours. Serial specific gravities of her urine were obtained and ranged from a presenting 1.030-1.010 24 hours later. The patient's vital signs were stable throughout the entire hospital admission. Fifteen hours post exposure, her chemistry was: Na = 140, K = 4.2, Cl = 107, HCO₃ = 24, BUN = 11, SCr = 0.9, Glu = 91, and serum osmol = 291. She was hospitalized for 36 hours and never experienced headache, nausea, vomiting, or any other symptoms reported to precede hyponatremia and seizures. Conclusion: No significant toxicity was noted in this pediatric ingestion of 4–5 mg of desmopressin. Therapy consisted of conservative measures such as free water restriction, activated charcoal, IV normal saline and monitoring. The absence of toxicity following an acute oral overdose of desmopressin may be related to proteolytic degredation in the GI tract responsible for a low bioavailability (0.15%), vs. nasal (5%) and IV (100%).

4. CONSTRUCTION OF A NOVEL PREDICTOR OF HEPATOTOXICITY FOLLOWING ACETAMINO-PHEN OVERDOSE: BEYOND THE NOMOGRAM

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Background: Although highly sensitive, the Rumack-Matthew nomogram is at best a crude predictor of hepatotoxicity following acetaminophen (APAP) overdose. The nomogram broadly classifies patients into discrete risk zones based on predicted [APAP]_{4h}, thereby diluting information inherent to this continuous parameter. It also fails to incorporate the delay to initiation of rescue *N*-acetylcysteine therapy, t_{NAC} . <u>Objective</u>: To construct from first principles a single, continuous measure of exposure following acute APAP overdose, represented by ψ . <u>Methods</u>: Using an area-under-the-curve approach, the specific burden of free NAPQI was modeled from pharmacokinetic first principles. The following global parameters were defined: t_{lag} , an interval during which existing glutathione reserves are sufficient to extinguish NAPQI, and [APAP]_{threshold} (obtained at $t_{threshold}$) where NAPQI formation matches the maximal rate of glutathione regeneration. <u>Results</u>: Conceptually, ψ is the non-negative area between [APAP]_{4h}e^{-k(t-4hr)} and [APAP]_{threshold} where $k = \ln(2)/4$ hr. Solving the integral yields $\psi = ([APAP]@t_{lag} - max([APAP]@t_{NAC}, [APAP]_{threshold})/k - [APAP]_{threshold} × [<math>-t_{lag} + \min(t_{NAC}, t_{threshold})$]. This quantity is directly proportional to the weight-adjusted quantity of injured hepatocytes. <u>Discussion</u>: The clinically intuitive parameter ψ combines information available at patient presentation into a single, continuous measure of exposure following APAP overdose. This parameter will permit the generation of a dose-response curve for hepatotoxicity in humans in the post-*N*-AC era. It also enhances the power to test hypotheses regarding other risk factors such as ethanol, and regarding optimal antidotal therapy.

5. DELAYED PRESENTATION OF METHANOL INGESTION LEADING TO CEREBRAL HERNIATION AND FATALITY IN THREE OF FOUR SEVERE CASES

Stremski E, Kostecki E, Gummin D. Children's Hospital of WI Poison Center, Milwaukee, Wisconsin, USA.

<u>Background</u>: Methanol toxicity produces severe adverse neurologic effects and metabolic acidosis. We present four cases of severe methanol toxicity that lead to cerebral herniation in three cases. Continuous veno-venous Hemodialysis (CVVH) used in a 4th case made a full recovery. <u>Case Series</u>: In unrelated cases, four adult males presented with coma and acidosis following unknown quantity ingestion of methanol. Methanol poisoning was not recognized in case 1.

- 1. 19 Y presented comatose, pH 6.84, at 10 hrs after noticed to be somnolent. Suspected illness was sepsis. Despite aggressive care he arrested within 12 hrs of presentation. Post mortem findings included tonsilar herniation, blood methanol 362 mg/dL, blood formic acid 1000 mcg/cc.
- 2. 45 Y drinking windshield fluid for 2 days presented comatose, pH 6.9. Started on 10% ETOH IV and immediately had 1 run Hemodialysis (HD). Methanol level = 141 mg/dL. Fomepizole started at 7 hours. Developed clinical signs of cerebral herniation with flat EEG at 24 hrs.
- 3. 27 Y found unresponsive in jail cell. Presented comatose, pH 6.7, 10 hrs after unknown ingestion. Methanol = 136 mg/dL. Immediate IV ETOH and 1 run HD. Flat EEG within 24 hrs.
- 4. 37 Y coma, pH 7.15, 1 day after drinking wiper fluid. Methanol 240 mg/dL. Immediate fomepizole, and 1 run HD followed by CVVH for 48 hours. Survived without neuro sequelae.

<u>Conclusion</u>: CNS herniation occurred despite alcohol dehydrogenase inhibition and single run HD, as was also seen in one case without these interventions. Survival occurred in the one case where CVVH was used after single run HD. Despite delayed initiation of therapy, less severe acidosis at presentation coupled to continual extra corporeal blood filtration may both have been important factors that resulted in a favorable outcome from severe methanol toxicity.

6. A CASE SERIES OF DERMAL ACETONITRILE EXPOSURES

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<u>Background</u>: Acetonitrile fatalities have been reported following inhalation and oral exposures. Dermal absorption is described in one animal study but there are no human case reports of dermal exposure. This is the first reported human case series involving acetonitrile dermal exposure. <u>Case Series: Patient 1</u>: A 27 year-old male spilled an estimated 1 mL of 100% acetonitrile on his hand which was not washed immediately. He presented to the hospital 6 hours later complaining of feeling tremulous. Electrolytes were normal. The tremor resolved without treatment and he remained asymptomatic at discharge 10 hours post exposure. <u>Patient 2</u>: A 22 year-old male spilled approximately 50–100 mL of 100% acetonitrile on his stomach, arms, and hands. He irrigated his hands and arms after a 5-min delay, but his stomach was not washed. The patient did not develop any complications and remained asymptomatic at 24 hours post exposure. <u>Patient 3</u>: A 23 year-old male was splashed with 100% acetonitrile on his face, arms, neck, and back. His face was promptly irrigated, but his arms, neck, and back were not. Two hours later he presented to an urgent care clinic with shortness of breath and heaviness in his chest upon deep inspiration. Arterial blood gases and vital signs were normal. There cases of dermal acetonitrile exposure are presented, two with extensive exposure who did not receive prompt decontamination. None of the patients developed serious complications of cyanide toxicity or required any medical intervention. Small to moderate acetonitrile dermal exposure is unlikely to result in life threatening cyanide toxicity.

7. MEDICATION ERRORS IN CHILDREN AGE 5 YEARS AND YOUNGER

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Background: Numerous factors have been identified in being causative in producing an unintentional medication error. Most literature examines the factors leading to hospital-based errors. Our intent was to describe the circumstances of



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hospital and home exposure that lead to critical care unit (CCU) admission of children age 5 Y and under due to a medication error. <u>Methods</u>: 2 Y retrospective review of one Poison Center's cases to describe substances, type of error, and effects when CCU was required due to an unintentional med error in a child \leq 5 Y. <u>Results</u>: 1852 total medication errors, 1813 exposures occurred in the home, 15 exposures in hospitals. 1779 onsite managed, 72 (13%) managed in a hospital. 15 hospital admits, where 9 required CCU, 3 due to parent errors, 6 due to health care professional errors. Case summaries:

2 Y, ethsuximide, parent gave 15 cc instead of 3 cc, lethargy-altered mental status (AMS)

5 Y, carbamazepine, parent gave 22.5 cc instead of 7.5 cc, AMS, serum level 19 mcg/cc

3 Y, chlorpheniramine, 2 parents each gave one 20 cc dose over 6 hours, AMS and hallucinations

3 Y, carbamazepine, excessive dose for weight, possible prescribing error, AMS, level 24 mcg/cc

15 D, lorazepam, $10 \times$ IV dose error given during trauma resuscitation, coma

1 M, choral hydrate, $10 \times$ PR dose error given pre-MRI for sedation, apnea and coma

1 D, methergine given IM in newborn nursery instead of Vitamin K, seizures and coma

5 Y, risperdone, pharmacy dispensed 5mg tablets instead of 0.5 mg tablets, AMS

1 Y, acetaminophen, pharmacy clerk gave 650 mg instead of 120 mg suppository, liver failure

<u>Conclusion</u>: Rarely did medication error result in CCU admission. In all but one case, medications involved were CNS depressants. Errors made by parents and health care staff included inaccurate measurement of the dose, 10 fold decimal errors, and dispensing of the wrong medication.

8. ANILINE AND METHANOL TOXICITY AFTER SHOE DYE INGESTION

Katz K, Ruha AM, Curry S, Schwaner R. Good Samaritan Regional Medical Center, Phoenix, Arizona, USA.

<u>Background</u>: Methanol and aniline independently cause toxicity. We report a combined methanol and aniline poisoning producing severe methemoglobinemia (MHB), sulfhemoglobinemia, hemolysis, coma, and metabolic acidosis. <u>Case Report</u>: A healthy 39 year-old woman presented to an ED after ingesting 125 mL of AmberesTM Mexican shoe dye. Initial physical exam, as well as CBC, chemistries, ASA, APAP, ETOH, urine EMIT, and ECG, were unremarkable except for glucose of 267 mg/dL and CO₂ of 17 mmol/L. Serum methanol, ethylene glycol, and osmolality were not available. Within 2 hours the patient became unresponsive, requiring intubation and transfer to our toxicology center. Upon arrival, the patient was diffusely cyanotic. Vital signs were normal except for a Pulse Ox of 88% (FIO2 100%). Labs included: HGB 14.4 g/dL, PT 16.2 sec, Cr 0.6 mg/dL, Na⁺ 141 mmol/L, K⁺ 7.9 mmol/L, Cl⁻ 112 mol/L, CO₂ 13 mmol/L, AST 139 IU/L, ALT 95 IU/L, methanol 89 mg/dL, and MetHb level of 72%. The patient improved, but required a continuous MB infusion for recurrent MHB, which resolved by hospital day 5. A decline in HGB to 7.7 g/dL with RBC fragments and spherocytes occurred, requiring transfusion on day 6. A sulfhemoglobin level of 5.1% was measured on day 7. The patient was discharged on day 9. GC/MS analysis of the dye revealed aniline, and urine GC/MS detected acetanilide, aniline and APAP, metabolites of aniline. <u>Conclusions</u>: We describe a unique case of severe combined methanol and aniline toxicity after shoe dye ingestion. Clinicians should be aware of both toxicities in patients ingesting similar products.

9. DELAYS TO ACTIVATED CHARCOAL IN THE ED: WHY BOTHER?

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<u>Background</u>: Activated charcoal therapy (AC) loses efficacy with time after overdose. Recent practice guidelines suggest attempting to administer AC within 1 h of overdose. We evaluated the time interval from triage to administration of AC in acutely poisoned patients presenting to a busy University/County Emergency Department as a prelude to instituting a protocol for giving AC at triage. <u>Methods</u>: The study was embedded in a sham study evaluating side effects of activated charcoal therapy in order to minimize the Hawthorne effect. Adult acutely poisoned patients who presented to triage were

eligible for inclusion. Data sheets were recorded with time of ingestion, triage time and time to start and completion of AC. <u>Results</u>: 107 patients were enrolled. Mean time from ingestion to triage was 202.28 min (95% confidence interval (CI) 148.8–255.7). Mean time from triage to start of AC was 80.7 min (CI 57.9–103.5). The time from start to of AC to completion was 12.63 min (CI 8.7–16.5). There were no differences in times comparing patients arriving by private vehicle and those arriving by ambulance, in contrast to a similar previous study at this institution (Ambulance 71.2 min (56.8–85.5) POV 104.7 (29.2–180.1). <u>Conclusion</u>: Acutely poisoned adult patients often arrive at the ED after the traditional time window of GID efficacy has passed. The additional delay to AC in the ED may make the efficacy of AC questionable in most acutely poisoned patients. In cases where AC is indicated, efforts must be made to decrease the "door to charcoal window."

10. GHB-ASSOCIATED VENTRICULAR TACHYCARDIA AND QT PROLONGATION

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<u>Background</u>: GHB intoxication rarely causes life-threatening cardiovascular symptoms. The most common cardiovascular manifestations of GHB toxicity are bradycardia and mild hypotension, often not requiring specific therapy. Reports of other cardiac arrhythmias or conduction blocks from GHB are rare. We report a case of GHB intoxication associated with ventricular tachycardia and QT interval prolongation. <u>Case Report</u>: Paramedics brought a 22 year-old woman to the ED following ingestion of ethanol and GHB with her boyfriend at a sporting event. The patient arrived comatose (GCS = 7), with a pulse in the low 50 s/min (intermittently as low as 37/min), blood pressure 110/70 mmHg, respiratory rate 4/min. Oral suctioning aroused the patient enough to protect her own airway, and she was observed for resolution of symptoms. Thirty minutes after arrival, two brief episodes (<3 sec) of a wide-complex tachycardia were noted on the patient's cardiac monitor. The electrolyte profile was within normal limits, and a subsequent 12-lead ECG revealed sinus bradycardia with a QT interval of 528 m sec (QTc = 491). The serum ethanol level was 54 mg/dL; urine screening by EMIT, TLC, and GC-MS did not reveal the presence of any other drugs. The urine GHB level 90 min after ED arrival was 2173 μ g/mL. The patient was admitted for further cardiac monitoring and observation. Serial ECGs showed normalization of the QT interval to 398 m sec over 8 hrs, and no further arrhythmias were recorded. The patient had a normal echocardiogram, ruled-out for myocardial infarction, and was discharged in stable condition on hospital day 3. <u>Conclusion</u>: This case demonstrates reversible QT interval prolongation and non-sustained ventricular tachycardia associated with acute GHB intoxication.

11. A 2 YEAR REVIEW OF PIOGLITAZONE AND ROSIGLITAZONE INGESTIONS

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Background: Pioglitazone (PioG) and rosiglitazone (RosiG) are thiazolidinedione antidiabetic medications. Since FDA approval in 1999, no reports of acute overdose have been published. Methods: A 2 year retrospective review of human PioG and RosiG ingestions reported to a regional poison control center during 2001–2002 was conducted. Cases involving co-ingestants, chronic ingestion, or those cases lacking follow-up (for clinical status and/or serum glucose readings) past the drug's therapeutic peak effect were excluded. Results: 210 cases were reported; 48 met the inclusion criteria. Of these, 25 (52%) patients ingested PioG and 23 (48%) patients ingested RosiG. The mean PioG dose was 54 mg (range 15–90 mg) and the mean RosiG dose was 15 mg (range 4–24 mg). The mean age was 20 years: 29 patients (60%) were under 7 years. Reason for ingestion: unintentional 27 (56%), therapeutic error 10 (21%), accidental doubledose 7 (15%), and suicide attempt 4 (8%). Activated charcoal or ipecac was given to 18 (38%) patients and 30 (62%) patients were observed or given food. Disposition: 21 (44%) home-managed cases, 22 (46%) ED-managed cases, and 5 (10%) admitted cases. Of the total 48 patients, one pediatric patient developed borderline hypoglycemia with a blood glucose of 59 mg/dL but no clinical symptoms. The patient did not receive IV glucose and it was unclear if the patient received any food/drink during the 4-hour hospital observation. Another patient developed mild diarrhea. The other 46 patients did not experience any effects. Conclusion: No evidence of serious toxicity was noted in our study. Although small to moderate accidental ingestions can probably be safely observed at home for clinical symptoms, further studies are needed to confirm our findings.

12. AN OUTBREAK OF SEVERE RODENTICIDE POISONING IN NORTH VIETNAM CAUSED BY ILLEGAL FLUOROACETATE

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<u>Background:</u> Since January 2002 well over 100 cases of rodenticide poisoning have been admitted to the Clinical Toxicology Unit of the PCC in Hanoi. The responsible product is brought illegally from China, where it is illegally produced. It has been suspected to contain some organic fluorine compound. <u>Methods:</u> NMR spectroscopy, using standards of fluoroacetamide and sodium fluoroacetate as references, was used to analyze four liquid samples of the type of rodenticide ingested. The clinical findings in three typical cases are summarised below:

Case series	Initial GI symptoms	Hyperreflexia, rigidity, S-CPK ↑	Recurrent seizures	ECG findings	Hospital duration/ outcome
8 years, F accidental	Yes	Yes	Yes	T inversions and supravent. tachy.	11 days/survival
17 years, F suicidal	Yes	Yes	Yes	Prolonged QT; SVT	7 days/survival
21 years, F suicidal	Yes	Yes	Yes	Prolonged QT; VT; ventric. fibr.	1 day/fatal

<u>Results:</u> The presence of sodium fluoroacetate was demonstrated in all four rodenticide samples analysed. <u>Conclusion:</u> Although the extremely toxic substance sodium fluoroacetate has been banned as a rodenticide in China and Vietnam for many years, extensive illegal use is apparent and severe cases of intoxication are numerous.

13. XYLAZINE INJECTION IN MAN

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Background: Xylazine is an alpha-2 agonist used as an animal tranquilizer; the LD50 in dogs is 47 mg/kg. Case Report: A 52 y. o. 97 Kg male was brought to the ED at 02:00 h after IM injection of 2.5 g xylazine, (25.8 mg/kg) (Rompum[®] Bayer) and ingestion of the balance (2.5 g) of the 50 mL vial in a suicide attempt. The patient had a history of depression and was prescribed buproprion. He was disoriented and semiconscious on presentation but aroused to verbal stimuli. He was breathing (rate = 22/min) but had appeic periods of 15 to 20 seconds; pulse oximetry was 89–97%; pulse 74; BP 140/102. Lavage was performed and 100 g charcoal with sorbitol were given at 02:30. Gastric contents were clear, and a pink substance around the mouth was noted. Abnormal laboratory values included: glucose 234 mg/dL, BUN 21 mg/dL, calcium 8.0 mg/dL, WBC 13.4. A screen for drugs of abuse in plasma was negative for amphetamines, barbiturates, benzodiazepines, cannabinoid, cocaine, opiates, and phencyclidine. A sample of blood drawn at 03:00 h did contain xylazine 2 mg/L. Blood glucose remained elevated for 24 h. He recovered without further incident and was discharged 36 hours after admission. Conclusion: Three other cases of self injection of large amounts (>1 g) of xylazine are reported: a 27 years old male injected 1.5 g (13 mg/kg) IM causing a plasma concentration of 4.6 mg/L (the highest value reported in a human), a 37 years old female injected 2.4 g (22 mg/kg) IM, and a 34 years old male injected 1 g (15 mg/kg) IM. Like our patient, all three patients were hyperglycemic (177, 176, 196 mg/dL). Although the toxidrome "should" include hypotension, our patient and two of the three above patients were hypertensive upon presentation (180/100, 166/70). Recovery was complete in all cases.

14. PHYSOSTIGMINE ADMINISTRATION FOR QUETIAPINE TOXICITY

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Background: Quetiapine is an atypical antipsychotic which has been reported to possess minimal affinity for muscarinic cholinergic receptors. Case reports describing quetiapine overdose make no specific mention of clinical

anticholinergic toxicity. We present a case of a woman who had clinical reversal of symptoms from a quetiapine overdose immediately following physostigmine administration. <u>Case Report</u>: A 15 y. o. woman with history of manic-depression taking no current medications presented to the ED after an overdose of her mother's quetiapine tablets. On presentation she was confused and agitated with vital signs of T 97.2, HR 106, RR 14, BP 133/96, and POx100%. The patient was admitted to the ICU where she was administered midazolam for agitation. Initial routine blood work and EKG were normal. The confirmatory GC/MS showed the presence of only quetiapine and caffeine. The patient's serum quetiapine level 24 hours after ingestion was 804 ng/mL (Therapeutic 40.2 ng/mL). The patient remained in the ICU with altered mental status, mumbled speech, normal pupils, dry axilla, hallucinations, and mild tachycardia. Sixty hours after her ingestion the patient remained agitated. At that time 3 mg of IV physostigmine was administered for suspected antimuscarinic manifestations. The patient's speech immediately became clear, her sensorium returned to baseline and she became aware of her current situation. Sixty hours after the ingestion her quetiapine level was 164 ng/mL. She remained stable, and the following day she was admitted to psychiatric floor. <u>Conclusion:</u> This is the first report of clinical anticholinergic symptoms from quetiapine overdose and the first report of anticholinergic delirium from quetiapine with resolution after physostigmine administration.

15. ACUTE RENAL FAILURE, NEUROPATHY, AND MYOPATHY AFTER INGESTION OF DIPRO-PYLENE GLYCOL-CONTAINING FANTASIATM FOG SOLUTION

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Background: Dipropylene glycol is used in several industrial products including cosmetics, emulsifiers, solvents and as a fog solution for dance club special effects, etc. Animal studies suggest that dipropylene glycol has minimal toxicity. We present a case of a 32 year old man who ingested one pint of dipropylene glycol-containing fog solution and subsequently developed acute renal failure, neuropathy and myopathy. Case Report: A 32 year old healthy man presented with abdominal pain and flank pain after ingesting one pint of FantasiaTM fog solution, 4 oz of NyquilTM and an unknown number of naproxen sodium tablets 3 days after ingestion. His vital signs and physical examination were normal. The patient's BUN = 53 mg/dL [range: 8–25], and his creatine (Cr) = 6.4 mg/dL [range: 0.6–1.5]. Initial blood gases, electrolytes, liver function tests were normal. Comprehensive urine drug screen using gas spectrometry and thin layer chromatography, acetaminophen, salicylate, ethylene glycol, methanol, and ethanol levels were negative. The patient became oliguric and received hemodialysis (HD). The BUN and Cr peaked on day 6 at 171 mg/dL and 17.7 mg/dL, respectively. A renal biopsy performed on day 10 revealed cortical necrosis. His physical exam revealed a left facial palsy and proximal muscle weakness. A muscle biopsy showed inflammatory myositis and an EMG was consistent with peripheral neuropathy. An MRI revealed abnormal enhancement of V, VII, IX, and X cranial nerves. The patient required a tracheotomy, PEG tube and was discharged to a long-term care facility still requiring HD at 6 month follow-up. Conclusion: Acute renal failure, neuropathy and myopathy are associated with acute ingestion of one pint of dipropylene glycol ingestion.

16. ARIPIPRAZOLE (ABILIFYTM) OVERDOSE IN A 2.5 YEAR-OLD

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<u>Background</u>: Aripiprazole is a new antipsychotic drug. It is metabolized by CYP3A4 and CYP2D6. The mean elimination half-life is about 75 hours in fast- and 146 hours in slow-CYP2D6 metabolizers. Pediatric and overdose experience is limited. <u>Case Report</u>: A 2.5 year-old female ingested 13, 15 mg tablets (17.1 mg/kg) of aripiprazole. She had a spontaneous emesis and became lethargic within an hour. The patient received 10 g activated charcoal, 3 hours post ingestion. She progressed to unconsciousness, but did not require airway support. On admission, P 110, BP 106/60, RR 20, T 98.3, POX 98 (RA); PERRL; disconjugate gaze; neurologic exam was non-focal, with 1+ and symmetric DTRs, downgoing toes and appropriate withdrawal to noxious stimulae; skin was pink, warm and dry; and bowel sounds were present but decreased. The 12-lead ECG rate was sinus at 110, QRSD 70 m sec, and QTc 446 m sec. <u>Course</u>: Vital signs remained stable and she awakened gradually over 24 hours. On day 2, she continued to sleep for long periods and had truncal ataxia while sitting. On day 3, she continued with ataxia during standing and ambulation. On day 4, the main



residual effect was tremulousness of fine motor hand movements and she was discharged. Symptoms progressively resolved over the next 7 days. There have been no sequelae. Serum was subsequently sent to the manufacturer for determination of aripiprazole (A) and its major active metabolite, dehydro-aripiprazole (DA), concentrations. Ten hours post ingestion, A + DA = 1873 ng/mL (after a maximum therapeutic dose of 0.43 mg/kg, $C_{max} = \sim 120 \text{ ng/mL}$). The half-life of elimination of aripiprazole, between 10 and 60 hours, was 23 hours. <u>Conclusion</u>: A massive ingestion of aripiprazole produced significant CNS depression, but no cardiovascular instability, respiratory compromise, or ECG effects. Clinical effects persisted for more than a week.

17. MASSIVE VERAPAMIL PHARMACOBEZOAR RESULTING IN ESOPHAGEAL PERFORATION

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<u>Background</u>: In previous reports, sustained release calcium channel blocker (CCB) pharmacobezoars have been anatomically located in the stomach or found in polyethylene glycol rectal effluent. We report the first case of a CCB esophageal pharmcobezoar. <u>Case Report</u>: A 61 year old female overdosed on 100 verapamil SR 240 mg tablets. EMS personnel performed endotracheal intubation and transported the patient to the ED. Initial BP was 55 by palpation and the HR was 40. The ECG showed a QRS of 158 m sec, peaked T waves, and a RBBB. The Georgia Poison Control Center was contacted. Recommendations included calcium, glucagon, vasopressors, and whole bowel irrigation. Several attempts to pass an NG tube were unsuccessful. Air insufflation could not be auscultated over the stomach, but a KUB demonstrated the NG tube below the diaphragm. Direct esophagoscopy showed an obstructing mass of pills. Thoracoabdominal computed tomography (CT) scan revealed that the esophagus was completely casted with debris. The NG tube was identified within the mediastinum with associated mediastinal emphysema, and free contrast material was seen within the pelvis indicating peritoneal perforation. The patient was taken to the OR where opening of the esophagus revealed a massive bezoar of verapamil tablets which was manually disimpacted, and an esophageal perforation was repaired. The patient recovered fully. <u>Conclusion</u>: We report the first case of esophageal CCB pharmacobezoar complicated by NG tube perforation of the esophagus.

18. ATROPINE OVERDOSE FROM A SUPPOSITORY COMPOUNDING ERROR

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Background: Medication compounding has resulted in dose errors. Case Report: A 72 year-old female used pharmacycompounded suppositories containing ergotamine, phenobarbital, atropine, and caffeine for headaches. She had been taking this medication for years without adverse effects. Her other medications were Elavil[®], Percocet[®], Celebrex[®], Premarin[®], levothyroxin, and an unspecified blood pressure medication. With the first dose of a new batch of compounded suppositories, she rapidly developed altered consciousness, hallucinoses, garbled speech, dystonia, dilated pupils, dry mucous membranes, and urinary retention. The bedside impression of a medical toxicologist was of an anticholinergic toxidrome. The patient's daughter took one-half of a suppository from the same batch with similar, but milder, symptoms and was not medically evaluated. The patient had a urine toxicology screen that was positive for triclyclic antidepressants at 20 mcg/mL, barbiturates, and opioids. QRS and QTc durations were normal. A CT scan showed no acute findings. Her symptoms gradually improved and she was discharged on day 4, but mydriasis and intermittent hallucinoses did not completely resolve until day 25. The suppositories were analyzed by methanol extraction and liquid chromatography-tandem mass spectrometry, and found to contain approximately ten times the concentration of atropine as an older suppository. We notified the pharmacy of our suspicions at the initial patient contact and suppositories from the same production batch were destroyed. The pharmacy later confirmed a 10-fold atropine calculation error. Conclusion: A suppository compounding error resulted in a ten-fold overdose of atropine, producing an anticholinergic syndrome, with effects persisting nearly a month. Early feedback to the pharmacy likely prevented additional poisonings.

19. INCIDENCE OF GRANDPARENT'S ORAL HYPOGLYCEMIC MEDICATIONS AS A SOURCE OF PEDIATRIC INGESTIONS

Alsop JA, Welch RA. California Poison Control System—Sacramento, Sacramento, California, USA.

Background: The incidence of diabetes is 12.1% in people aged 45–64 yrs and 21.6% in ages 65–74 yrs. Diabetic control is maintained with oral medications in 49% of patients. We observed that many pediatric calls about exposures to oral hypoglycemic agents involved children having access to grandparent's medications. We selected to study the frequency of this occurrence. Method: All ingestions of oral hypoglycemics agents in children under age 6 years reported to CPCS in the year 2001 were examined to determine the source of the exposure. Results: A total of 335 pediatric oral hypoglycemic ingestions were reported. A grandparent's medication was mentioned in 155 cases (46.3%). Demographics: male 55%, female 45%, average age 20 mos. The primary oral hypoglycemic agents involved were glyburide 26%, metformin 22.6%, glipizide 18.6%. Exposure scenarios included children opening the original medication containers 53.6%, opening days-of-the-week pill dispensers 18.7%, meds left in reach on counter or table 13.5%, tablets that had fallen to the floor 12.3%, tablets stored in a purse 1.3%, or tablets stored in a baggie 0.6%. In 45% of cases, the exposure involved multiple medications that were sometimes more dangerous than the oral hypoglycemic agent. Exposures were treated at a HCF 78%, at home 17%, and unknown 5%. Cases followed to a known outcome: no effect 58.7%, minor 5.8%, moderate 14.2%, unrelated 3.9%. Conclusions: In this review, pediatric ingestions of oral hypoglycemic medications were a result of children obtaining their grandparent's medications in 46.3% of cases. In 45% of those cases, children were also exposed to multiple other potentially dangerous medications. Proper storage of grandparent's medications may prevent some pediatric poisonings.

20. UNEXPLAINED PROLONGED INRS IN A COLLEGE STUDENT

Alsop JA, Tegzes JM, Ferguson TJ. California Poison Control System—Sacramento, UC Davis Student Health, California Animal Health & Food Safety Lab., Davis and Sacramento, California, USA.

Objective: We report a case of a college student with increased INR and PTT levels of unknown origin. Case Report: A 21 year old female had repeat visits to Student Health with various ailments of vague GI symptoms and HA. She returned after hitting her head and the wound continued to bleed. Labs: INR 6.21, PTT 49.4. She denied psychological problems and denied access to anticoagulants or rat poisons. She was started on Vitamin K_1 and a hematological workup was done. She had lowered Factors II, VII, X, IX, with an INR >14.7, PTT 63.6. She received 6 units fresh frozen plasma and her INR corrected to 2.38. Over the spring her INR continued to fluctuate despite increasing doses of SQ and oral Vitamin K₁. She never developed frank bleeding. She believed someone at school was poisoning her. Several serum brodifacoum levels were drawn. Over a month her brodifacoum levels were 300, 290, 290, 260, 200, and 270 ng/mL. Her family physician did not believe that she was suicidal. When the police questioned her, she confessed that she was selfadministering rat poison. She was followed for 2 months. When school was out for the year, she moved and follow up was lost but her last INR was 3.2. Conclusion: Long acting anticoagulants inhibit the K 1-2,3-epoxide reductase enzyme that block Vitamin K dependent clotting factor synthesis. Brodifacoum is a long-acting anticoagulant rat poison with a $T\frac{1}{2}$ of 120 days in dogs. In one human case, an elimination $T\frac{1}{2}$ of 24 days was reported. Lethal human brodifacoum levels range from 160 to 786 ng/mL. In cases of prolonged INR and PTT levels, surreptitious ingestion of long acting anticoagulants should be suspected. Diagnosis can be made by measuring serum brodifacoum levels. This patient probably did well because she was compliant with clinic visits, lab draws and administration of Vitamin K₁.

21. SUICIDE FROM TILMICOSIN INJECTION: CASE REPORT AND BLOOD LEVELS

Mueller C, Bottei E. Iowa Statewide Poison Control Center, Sioux City, Iowa, USA.

Background: Tilmicosin (Mycotil 300[®]) is a macrolide antibiotic used to treat respiratory diseases in domestic livestock. Mycotil 300 (tilmicosin 300 mg/mL and propylene glycol 250 mg/mL) is known to exert potent negative ionotropic properties in several animal species. Product information warns of its potential lethality if injected into humans.

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<u>Case Report</u>: An 18 year old female was found down in full arrest in the barn of the dairy farm on which she lived. A syringe and an empty vial of tilmicosin were found next to her. Despite CPR, IV fluids, and pressors, the patient could not be resuscitated. An autopsy was preformed and revealed no gross abnormalities. Forensic drug screen analysis was negative for all drugs except tilmicosin. The tilmicosin blood level was 15 mcg/mL while the gastric contents had a level of 3.1 mcg/g. The only other report documenting a tilmicosin blood level in a human found a level of 30 mcg/mL in a 25 year old male who also committed suicide. Peak blood levels in cattle treated with the recommended 10 mg/kg subcutaneous dose are usually less than 1 mcg/mL. Tilmicosin toxicity has been noted in pigs and cows with blood levels ranging of 3–7 mcg/mL. A tilmicosin-related death in a calf found a blood level of 10 mcg/mL. <u>Conclusion</u>: While the exact mechanism by which tilmicosin exerts its cardiotoxicity is uncertain, this case report documents human lethality at blood levels one-half that previously reported. Whether it is the macrolide itself or the propylene glycol that causes the cardiovascular collapse, treatment is aggressive supportive care with positive ionotropes and cardiac monitoring.

22. TREATMENT OF DERMAL EXPOSURE TO COMMON HOUSEHOLD ITEMS

Gunia P, Gray J, Gottsch S, Kalin L, Ringling S, Bottei E. Iowa Statewide Poison Control Center, Sioux City, Iowa, USA.

<u>Background</u>: Poison centers routinely receive calls from people wanting to know the best way to remove substances that are stuck to their skin. Lack of standardized treatment has lead to the use of folklore and home remedies with variable results. A literature review found no comprehensive studies comparing the efficacy of various cleaners at removing substances stuck to the skin. <u>Methods</u>: Various household products were applied to clean dry skin. After adherence of the product, various household cleaners were applied to the stain and the time until the stain was removed from the skin was recorded. <u>Results</u>: There were no adverse reactions to either the products or the cleaners.

	Acetone	Non-acetone	GooGone®	Lotion	Soap and water	Baby oil
Spray paint	Immediate	Immediate	Immediate	30 min	Ineffective	30 min
Super glue	9 min	11 min	13 min	9 min	5 min	12 min
Polyurethane	Immediate	Immediate	Immediate	15 min	15 min	Immediate
Wite out	Immediate	Immediate	Immediate	12 min	11 min	4 min
Tar/asphalt	21 min	28 min	Immediate	28 min	28 min	Immediate
Foam insulation	11 min	11 min	Immediate	30 min	30 min	30 min
Carpet adhesive	15 min	Immediate	15 min	Immediate	Immediate	15 min

<u>Conclusion</u>: GooGone and non-acetone nail polish remover provided the best results with little if any tissue irritation. This data can now provide poison centers with better recommendations for what to use to remove substances that are stuck to the skin.

23. DELAYED ONSET OF SEIZURE IN A BODY STUFFER

Yao IC, Mazor SS, O'Koren, K, Guffey, MA, Aks SE, Leikin JB. The Toxikon Consortium, Illinois Poison Center, Department of Emergency Medicine, Cook County Hospital, ENH Omega, Chicago, Illinois, USA.

<u>Background</u>: A body stuffer is one who hastily ingests poorly wrapped packets of narcotics in order to conceal evidence. Previously reported series have not defined the optimal time of observation for these patients. One paper concludes that patients with significant toxicity exhibit symptoms upon presentation or shortly afterwards, and recommends discharging the asymptomatic body stuffer after 6 hours of observation. We report a case with delayed onset of toxicity in a cocaine body stuffer. <u>Case Report</u>: A 26 year-old male was brought to the emergency room 90 min after ingestion of 5–8 packets of rock cocaine. The patient's vital signs upon presentation were: blood pressure 154/76 mmHg, heart rate 96 beats/min, respiratory rate 24 breaths/min, and temperature 96.4°F. The patient was treated with activated charcoal followed by

polyethylene glycol electrolyte lavage solution. During the patient's initial course in the emergency department, he remained asymptomatic. However, 7½ hours post-ingestion the patient exhibited signs of toxicity in the form of seizures, hypertension, and tachycardia. <u>Conclusion</u>: This case demonstrates that patients may exhibit significant toxicity beyond the recommended six-hour window post-ingestion. Therefore, it may be prudent to observe the asymptomatic body stuffer in a 23-hour observation unit for delayed onset of symptoms.

24. SHOULD ALL PATIENTS WITH DRUG OVERDOSE HAVE A SALICYLATE LEVEL?

Wood DM, Dargan PI, Jones AL. National Poisons Information Service (London), Guy's & St. Thomas' NHS Trust, London, UK.

Background: Many clinicians take a salicylate levels in all patients presenting after drug overdose regardless of the history. There have been a few studies performed to evaluate this practice in countries where salicylate poisoning is common (e.g., Hong Kong) but none in Europe where the prevalence of salicylate poisoning is much lower. Methods: We performed a retrospective survey (1st February 2001-31st January 2002) of all patients attending the ED of one hospital. The chemical pathology computer was used to identify patients who had a salicylate level taken and their hospital records were reviewed and the following data collected: drugs ingested, clinical features, investigations, and treatment. Results: Notes were available for 612 (82.7%) of the 737 patients who had a salicylate level measured; 16 had salicylate levels taken inappropriately and so were excluded. Fourty-seven (7.7%) had a detectable salicylate level (range 15-428 mg/L). None of these patients required specific management for their salicylate intoxication. A history of salicylate ingestion had a sensitivity of 81% and a positive predictive value of 79%. Nine of those with a detectable salicylate level gave a negative history of salicylate ingestion; the maximum concentration in these patients was 298 mg/L (this patient was asymptomatic). Of the 550 patients with undetectable salicylate, 11 gave a history of salicylate ingestion; the specificity of the history was 98%. Conclusion: A negative history for salicylate ingestion has a high specificity and sensitivity. On the basis of this and previously published studies, there is no justification for salicylate measurements in patients presenting with self-poisoning with a negative history for salicylate ingestion, unless there are clinical features of salicylate poisoning or a reduced level of consciousness which limits the validity of the history.

25. POISONED PATIENTS AS POTENTIAL ORGAN DONORS, A POSTAL SURVEY OF TRANSPLANT CENTRES AND INTENSIVE CARE UNITS

Wood DM, Dargan PI, Jones AL. National Poisons Information Service (London), Guy's & St. Thomas' NHS Trust, London, UK.

Background: The number of patients awaiting allograft transplantation in most countries exceeds the number of organs offered for transplantation. Most organ donors are young, fit and healthy individuals who die because of trauma or sudden cardiac arrest. Patients dying from poisoning are often of similar characteristics, but are less frequently offered as organ donors. Methods: A postal questionnaire survey of 67 physicians in all 30 UK transplantation centres and an equal number of ICU physicians was undertaken. The questionnaire consisted of four case scenarios: methanol ingestion, cocaine-related cardiac arrest, domestic carbon monoxide inhalation, and industrial cyanide exposure; in each case respondents were asked whether they would accept or offer kidneys, heart, lungs, liver or pancreas for donation. Results: 70% of transplantation physicians and 50% of ICU directors responded. Over 80% of organs would be offered and discussed with transplant coordinators by ICU directors in all of the scenarios; many also stated that they would seek the advice of a toxicologist. More than 70% of the transplant physicians would consider or accept organs from patients who had been poisoned with methanol, cyanide or carbon monoxide; however, only about 50% would consider or accept organs from patients who had been poisoned with cocaine. Conclusions: This postal survey showed that most transplantation physicians and ICU directors would consider poisoned patients as potential organ donors. Poisoned patients are another pool of organ donors, who at present are underused by transplantation services. Clinical toxicologists could play a role in increasing awareness of the potential for organ transplantation in poisoned patients with brain stem death.



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26. UNINTENTIONAL ACUTE CATAPRES[®] PATCH INGESTION IN AN ADULT

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<u>Background</u>: Unintentional ingestion of Catapres[®] patches is a known problem in the pediatric population due to the oral behavior of toddlers. Toxic effects generally occur within 30 min to 4 hours post-ingestion and then resolve within 24–72 hours. We present an unusual ingestion of a Catapres[®] patch in an adult who developed symptoms at 20 hours post-ingestion and persisted for 48 hours. <u>Case Report</u>: A 37-year-old woman ingested a 7.5 mg Catapres[®] patch (delivers 0.3 mg/day) which had inadvertently been tossed into her beverage by the mentally disabled adult to whom she provided care. The caretaker initially disregarded the exposure since the patch was already used. The following morning the caretaker was prompted to call the PCC with reports of dizziness, drowsiness, and headache, questioning if these might be related to the patch ingestion the previous afternoon. The patient was immediately referred by the PCC to the ED where she received IV fluids, WBI, and cardiac monitoring. On arrival, the initial BP was 80/50 and HR was 55. Maximal drops in BP and HR occurred on the 2nd hospital day with BP readings of 80/40 s and a sinus bradycardia of mid-40 s. The patient was not recovered and was assumed overlooked in the stools. <u>Conclusion</u>: This unusual exposure to a Catapres[®] patch illustrates that ingestion of a used patch represents a significant health risk for the adult population. An interesting feature of this case is the 20-hour delay in onset of symptoms, which may be a function of the position of the patch in relation to the gastric or intestinal mucosa.

27. TRANSAMINASE ELEVATION IN PYRETHROID INTOXICATIONS

Naumovski J,¹ Krenzelok EP,² Bozinovska C,¹ Pereska Z,¹ Kovkarova E.¹ ¹*Clinic of Toxicology, Skopje, Macedonia;* ²*Pittsburgh Poison Center, Pittsburgh, Pennsylvania, USA.*

<u>Objective</u>: To determine if acute ingestion of synthetic pyrethroid insecticide produces hepatotoxicity. <u>Methods</u>: The medical records of patients poisoned with synthetic pyrethroid were reviewed. Elevations in aspartate aminotransferases (AST) and alanine aminotransferases (ALT) were used to indicate pyrethroid—induced hepatotoxicity. Their ratio, AST/ALT, known as the DeRitis Index (DRI), was also calculated. Results were compared statistically using the Colgomorov-Smirnow test. <u>Results</u>: In a group of suicidal 28 patients, 17 (60.7%) showed an increase of AST (124.44 \pm 39.8; 70–365 U/L) and ALT (78.27 \pm 22.65; 57–138 U/L). The calculated DRI was greater than 1 (1.82; 1.5–3.9) indirectly demonstrating necrosis. <u>Discussion</u>: The combined presence of potentially toxic solvents such as aromatic, halogenated or cyclic hydrocarbons with pyrethroid insecticides is why an additional effort was undertaken to determine the influence of the solvent on the development of hepatotoxicity. The statistical analyses confirmed the influence of the solvent and/or adjuvants on this process. <u>Conclusion</u>: Synthetic pyrethroids are not associated commonly with clinically significant hepatotoxicity, but we identified liver enzyme elevation in a significant number of pyrethroid poisoned patients. Analysis of medical records indicates that the presence of solvents and other "inert" ingredients is probably contributory in the development of hepatotoxicity.

28. ESTIMATING POPULATION PHARMACOKINETIC PARAMETERS WHEN DOSE AND DOSE-TIME ARE NOT KNOWN ACCURATELY

Duffull SB,¹ Isbister GK,² Dawson AH,² Hackett LP,³ Whyte IM.^{1,2} ¹University of Queensland, Australia; ²University of Newcastle and Newcastle Mater Hospital, Australia; ³Western Australian Centre for Pathology and Medical Research, Australia.

<u>Background</u>: Accurate information on dose and time of ingestion is often missing in deliberate self-poisoning. Conventional pharmacokinetic modelling is therefore not possible or very difficult. Bayesian analysis with prior estimates of dose and time may improve the predictive ability of the pharmacokinetic model. <u>Methods</u>: Eighty nine plasma citalopram concentrations were available from 29 patients. Data were modeled by means of a Monte Carlo

Markov chain method using WinBUGS (ver. 1.3). A one-compartment model with first-order input and first-order elimination was used. Prior distribution for *CL*, *V*, and *Ka* were set to 30 L/h, 900 L, and 0.5 h^{-1} . Between subject variability was assumed to be log normal with low information priors for all parameter values. The fractional dose taken and lag-time were estimated. Both were assumed to be normally distributed (mean of 1 and 0 respectively). Precision was indexed to the veracity (reported on a four point ordinal scale) of the knowledge of dose and dose-time. <u>Results</u>: The posterior mean of *CL* was 29 L/h (between-subject variability = 41%), and *V* was 760 L (51%). Estimated actual dose ingested was 0.29–1.40 times the nominal dose recorded. Estimated actual dose-time was a few minutes to 1.6 hours before the nominal dose-time. Inclusion of informative priors improved overall model fit, decreasing between subject variability in *CL* by 43%. <u>Conclusion</u>: The use of informative priors indexed to clinical findings, within the framework of a fully Bayesian analysis, improved the predictive ability of a model developed from pharmacokinetic data arising from citalopram overdoses.

29. A CASE OF TYPE F BOTULISM IN SOUTHERN CALIFORNIA

Richardson WH, Frei SS, Williams SR. Division of Medical Toxicology, University of California San Diego (UCSD), San Diego, California, USA.

<u>Background</u>: Botulism caused by Type F botulinum toxin accounts for less than 0.1% of all human botulism cases and is rarely reported in the literature. <u>Case Report</u>: A 45 year-old woman presented to an emergency department (ED) complaining of blurred vision, difficulty focusing, and dysphagia. The treating physician initially considered the possibility of paralytic shellfish poisoning due to a report of shellfish ingestion, later determined to be frozen shrimp and a can of tuna, but no gastroenteritis or paresthesias were present. During ED observation, the patient developed respiratory distress with hypercapnea and required intubation and mechanical ventilation. Within hours, ptosis, mydriasis, and weakness in the arms and legs developed. Bivalent (A, B) botulinum antitoxin was administered approximately 24 hours from the onset of initial symptoms, but over the next 2 days complete paralysis progressed to the upper and lower extremities. Shortly thereafter a stool toxin assay demonstrated the presence of Type F botulinum toxin. The patient subsequently received an experimental heptavalent botulinum antitoxin obtained from the Department of Health on hospital day 4 but paralysis was already complete. Her 3-week hospital course was complicated by nosocomial pneumonia, but she gradually improved and was discharged to a rehabilitation facility. Anaerobic cultures and toxin assays have yet to elucidate the exact source of exposure. <u>Conclusion</u>: We report a rare case of Type F botulism believed to be food-borne in etiology. Administration of bivalent antitoxin did not halt progression of paralysis.

30. ACCIDENTAL INGESTION OF CAYENNE PEPPER SAUCE REQUIRING PROLONGED VENTILA-TORY SUPPORT

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<u>Background</u>: Prolonged ventilatory support after an ingestion of cayenne pepper sauce has not previously been reported in the literature. <u>Case Report</u>: A currently healthy 10-month old male presented to the ED after ingesting approximately four ounces of Frank's^(B) RedHot^(B) cayenne pepper sauce. His mother immediately tried to suction the hot sauce from his mouth and called 911. His past medical history was significant for premature delivery at 35 weeks gestation and hospitalization after birth for respiratory distress complicated by pneumothorax. His initial vital signs were: T, 36.6°C; P, 165/min; BP, 110/51 mm/Hg; RR, 44/min; and O₂ saturation on room air, 93%. His physical examination demonstrated expiratory wheezing, stridor, and use of accessory respiratory muscles. Intraoral erythema and excessive drooling were absent. An initial chest radiograph was normal and a nasopharyngeal wash for RSV was negative. Serial nebulized albuterol and racemic epinephrine were unsuccessful in improving the patient's condition. Laryngoscopy and bronchoscopy demonstrated significant subglottic erythema and edema, mild edema of the larynx and arytenoids, and tracheomalacia. An esophagogastroduodenoscopy was normal. After these procedures the child was admitted to the



pediatric intensive care unit and required intubation for respiratory failure. He developed bilateral perihilar infiltrates and was treated with intravenous solumedrol. The patient was extubated after 5 days and was subsequently treated for 3 days with heliox for continued stridor. The patient went on to make a complete recovery. <u>Conclusion</u>: Ingestion of cayenne pepper sauce can result in airway edema and pneumonitis necessitating ventilatory support.

31. INHALATION ABUSE OF METHANOL-CONTAINING CARBURETOR CLEANERS

Schaeffer S, McGoodwin L, Riley M. Oklahoma Poison Control Center, University of Oklahoma College of Pharmacy, Oklahoma City, Oklahoma, USA.

<u>Background</u>: A previously presented case series described clinical improvement, with minimal intervention required for most patients who had abused methanol-containing carburetor cleaners (MCC). These results do not match our experience with this type of exposure. <u>Case Series</u>: All cases involving intentional inhalation abuse of methanolcontaining automotive products reported to our center from 2001 to present were selected for review. Therapeutic interventions and clinical effects judged related to the exposure were examined. Thirteen cases meeting criteria for inclusion were identified. All patients were over twenty years of age. Six patients experienced visual disturbances and five patients developed acidosis. Eleven out of thirteen patients in our case series required aggressive therapies. Treatment modalities are summarized as follows.

Treatment	Number of patients
Ethanol only	4
Ethanol and hemodialysis	2
Fomepizole only	2
Fomepizole and hemodialysis	2
Hemodialysis only	1

<u>Conclusion</u>: Poison center specialists need to be aware that intentional inhalation of methanol-containing carburetor cleaners may result in significant toxicity and patients may require more than supportive care.

32. OUTCOME OF HEROIN BODY STUFFERS: A CASE SERIES

Jordan MT, Bryant SM, Aks SE, Wahl M. University of Illinois at Chicago; Toxikon Consortium-Cook County Hospital; Advocate Illinois Masonic Medical Center; Illinois Poison Center, USA.

<u>Background</u>: Cases of heroin body stuffers have rarely been reported. The aim of this study is to describe the clinical course of these patients. We present the largest series to date of heroin body stuffers presenting to regional emergency departments (EDs). <u>Materials and Methods</u>: A retrospective chart analysis was performed on all cases of heroin body stuffers received by a metropolitan poison control center and associated toxicology service from July 2000 to December 2002. We defined a heroin body stuffer as anyone who admitted to or was strongly suspected of ingesting heroin as a means of evading the authorities. Those using heroin for recreational use or for transport across borders to later sell (body packers) were excluded. <u>Results</u>: We identified 39 patients classified as heroin body stuffers. Most of these cases were middle-aged males with average age of 34 years (median 32). Quantity of heroin ingested ranged from one to sixteen containers, most commonly wrapped in plastic bags. Only three patients (7.6%) developed symptoms of opiate intoxication; all of which began within the hour of ingestion. Only one patient (2.5%) developed the need for naloxone. The average length of observation was 20 hours (median 22). <u>Conclusions</u>: Opiate intoxication secondary to heroin stuffing is uncommon. Those patients that developed symptoms of toxicity did so early in their clinical course. This retrospective data indicates a rather benign course in this patient population, albeit further prospective studies are needed to validate a short ED observation of heroin stuffers.

33. SEVERE NAPROXEN OVERDOSE WITH ELEVATED SERUM LEVELS

Mullen WM,¹ Meier KM,¹ Hagar SM,² Olson KR.¹ ¹California Poison Control System, San Francisco Division, Department of Clinical Pharmacy, UC San Francisco; ²Natividad Medical Center, Salinas, California, USA.

Background: Despite widespread use, published reports of naproxen overdose resulting in severe toxicity are rare. Metabolic acidosis, renal impairment, hypoprothrombinemia, and encephalopathy have been reported. We report a case of a patient who developed severe symptoms and the first documented human serum levels after naproxen overdose. Case Report: A 21 y. o. woman reportedly took 120 naproxen 500 mg. One hour after ingestion, she presented to the ED obtunded, hypotensive and had two grand mal seizures. She was intubated, resuscitated with fluids, and loaded with fosphenytoin. Her serum pH was 7.15 and serum HCO3 6 mmol/L. At 8 hours post-ingestion her serum lactate was 4.4 mmol/L. Twenty hours post-ingestion she was agitated and unresponsive. She had further episodes of hypotension with systolic BP in the 70s that responded to fluids. On the 2nd day post-ingestion she was lethargic but stable. Her WBC was 3.7 (12 initially) and platelets 97,000 (216,000 initially.) The 3rd day post-ingestion her WBC was 3.9 and platelets 206,000. She was discharged in good condition on the 4th day post-ingestion. Her naproxen level one-hour postingestion was 840 mcg/mL (therapeutic range 30-90 mcg/mL) and 640 mcg/mL at 7.5 hours post-ingestion Conclusion: We present the first case report of naproxen overdose with elevated serum levels. Further data points are needed to fully elucidate naproxen toxicokinetics.

34. "ECSTASY" AND METHAMPHETAMINE RELATED HYPERTHERMIA

Smolinske S, Baltarowich L, Thomas R. Children's Hospital of Michigan Regional Poison Control Center, Henry Ford Hospital, Detroit, Michigan, USA.

Objective: Hyperthermia is suggested to be associated with severe outcomes and fatalities following amphetamine abuse, including MDMA and analogs. We sought to determine the overall incidence of fever/hyperthermia in amphetamine (A), hallucinogenic amphetamines (E), and methamphetamine (M) intentional exposures. Methods: The AAPCC TESS database was searched, years 2000-2001, for all human intentional exposures to these amphetamine groups, excluding methylphenidate. Each fatality abstract was reviewed by two of the authors, who assessed the degree to which the death was related to the amphetamine compound. Cases were divided into (1) related primarily to A, E, or M; (2) related primarily to another toxin; or (3) related primarily to an indirect cause. Results: During the study years, there were 2789 total single-substance exposures to A, 2535 to E, and 1997 to M. In non-fatal cases, fever/hyperthermia was reported in 49/2786 (1.8%) of A, 93/2518 (3.7%) of E, and 84/1984 (4.2%) of M cases. In fatal single-substance cases, fever/hyperthermia was present in 2/3 (66%) of A, 8/17 (47%) of E, and 7/13 (54%) of M cases. The peak temperature was recorded in 29 fatalities, with a mean of 106.8° F ± 2.15 SD. There was no significant difference in peak temperature between any of the groups. If fatal cases with co-ingestants are included, fever/hyperthermia was present in 34% of A, 42.5% of E, and 43% of M. Overall, fever/hyperthermia was present in 29/68 (42.6%) fatal cases directly attributed to amphetamines and 5/36 (13.9%) in cases more likely related to another toxin. Complications of hyperthermia in fatal cases included rhabdomyolysis (12) and DIC (7). Conclusion: This study confirms that hyperthermia and its complications significantly contribute to fatalities from amphetamines.

35. A PROSPECTIVE POISON CENTER EXPERIENCE OF SUSTAINED-RELEASE BUPROPION OVER **40-MONTHS IN CHILDREN**

LoVecchio F, Hilder R, Ruha AM. Good Samaritan Regional Poison Center, Phoenix, Arizona, USA.

Background or Objective: The incidence of seizures following accidental bupropion sustained release (Bup-SR) ingestion in children is unknown. We conducted a prospective poison center survey of in-patient monitoring in children \leq 3 years old with presumed isolated Bup-SR ingestion during 40 months. Methods: Inclusion criteria: (1) isolated

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Bup-SR ingestion; (2) ≤ 3 years old. We recommended a minimum of 24 hours of in-patient observation. Adverse events (neurological events, etc.) were documented. <u>Results</u>: 71 patients fulfilled the inclusion criteria with five patients refusing admission. All patients, including patients whom remained at home were followed for at least 24 hours. Of the 71 admitted children the mean age was 18 [Range: 10–36] months with a range of amount ingested from unknown to 1200 mg. No seizures, coma or short-term adverse outcomes were documented in the 71 admitted or 5 non-admitted patients within 24 hours. <u>Conclusions</u>: We did not detect any adverse events following isolated Bup-SR ingestion in children ≤ 3 years old. The major limitation to this study is the small sample size.

36. DIPHENHYDRAMINE-INDUCED WIDE COMPLEX TACHYCARDIA SHOWN BY EXERCISE TREADMILL TESTING NOT TO BE ENTIRELY RATE RELATED

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<u>Background</u>: Diphenhydramine (DPH) has antimuscarinic and quinidine-like properties. The antimuscarinic property of the drug in poisoning often leads to tachycardia. The quinidine-like property of the drug has been used to explain the wide complex tachycardias occasionally reported in severe poisoning. However, tachycardia alone is known to induce rate-related bundle branch blocks in certain individuals. The question arises whether some of the wide complex tachycardias associated with DPH poisoning are purely a rate-related phenomenon. <u>Case Report</u>: A 44 year-old male acutely ingested 1.6 g of DPH in a suicide attempt. Following a witnessed generalized seizure he had a persistent altered mental status. Exam revealed BP of 151/94 mmHg, HR of 166 bpm, RR 22/min., T 99.5° F, mumbling speech, dry mouth, mydriasis, decreased bowel sounds, and dry skin. ECG revealed sinus tachycardia with QRS complex width of 146 ms. Three successive 50 mL boluses of 8.4% NaHCO₃ had no effect on QRS width. The patient's mental status, tachycardia, and QRS prolongation resolved over 12 hours with supportive care alone. The patient reported ingesting only DPH and comprehensive laboratory testing confirmed this and ruled out other drugs. Two months after the poisoning, the patient underwent exercise treadmill testing. During the testing his QRS width remained baseline at 82 ms despite achieving a maximum HR of 184 beats/min, well above the rate present during his acute DPH poisoning. <u>Conclusion</u>: Exercise treadmill testing confirmed that the wide complex tachycardia observed in this case of DPH poisoning.

37. ACUTE CERAMIC GLAZE INGESTION RESULTING IN LEAD POISONING

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<u>Background</u>: Acute ceramic glaze ingestions have resulted in significantly elevated blood lead levels. Even small amounts ingested can lead to significant toxicity. The majority of cases occur in elderly patients with dementia. We report a case of lead poisoning following acute ceramic glaze ingestion in an autistic child. <u>Case Report</u>: A 4 year-old female, with a history of developmental delay and autism, swallowed 1 ounce of ceramic glaze containing 6 mg of lead silicate. The patient was seen in the ED 1/2 hr after the ingestion. No emesis had occurred and the patient was asymptomatic. Vital signs were normal. Physical exam revealed a well appearing 4-year-old with white liquid around her lips. Her mental status was baseline according to her parents. Whole bowel irrigation (WBI) with PEG solution was initiated approximately 5 hours post-ingestion. An abdominal x-ray revealed opacities in the duodenum, which cleared after several hours of WBI. Whole blood lead levels were 83, 57, and 49 mcg/dL on days 1, 2, and 3, respectively. The patient was then treated with IV CaNa2EDTA for 5 days. She remained asymptomatic and was discharged on succimer in good health. <u>Conclusion</u>: Children with developmental delay are at significant risk for toxic ingestions. This is the first detailed report of acute lead toxicity after ceramic glaze ingestion in a pediatric patient. Immediate absorption of ceramic glaze resulted in early rises in blood lead levels in this patient. Consideration should be given to early WBI and chelation therapy after ceramic glaze ingestions, even prior to obtaining blood lead levels.

38. HYPERPHOSPHATEMIA AND CARDIAC ARREST FOLLOWING INHALATION OF A DRY CHEMI-CAL FIRE EXTINGUISHER

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<u>Background</u>: No systemic toxicity is reported from inhalation of dry chemical fire extinguisher. We report the first case of life-threatening hyperphosphatemia, hypocalcemia and cardiac arrest following exposure to a fire extinguisher. <u>Case Report</u>: A 46 y. o. woman with a history of depression and asthma inhaled the fumes of Ansul Foray[®] dry chemical extinguishing agent (containing ammonium phosphate monobasic) in a suicidal attempt. She presented in extremis, pulse rate 147/min, BP 119/65 mmHg, RR 32-36/min, Pulse Oximeter (RA) 74%, and T 98°F. ABG (100%): 7.07/59/162, electrolytes were normal except HCO3 20 mEq/L and anion gap 24. Approximately 4 hours later, she had a cardiac arrest and required intubation and ventilation. Abnormal labs were: HCO3 13 mEq/L (21–32), calcium < 5.0 mg/dL (8.4–10.0), phosphate 9.8 mg/dL (2.5–4.9). Osmol gap, salicylates, APAP, ethylene glycol, methanol, and ethanol were negative. She subsequently experienced three episodes of ventricular fibrillation requiring defibrillation. Vasopressors, sodium bicarbonate, calcium chloride, and hemodialysis were initiated. She completely recovered. <u>Conclusion</u>: Significant hyperphosphatemia and hypocalcemia may occur after inhalation of ammonium phosphate monobasic and may result in cardiac arrest. Hemodialysis is effective at correcting these life-threatening electrolyte abnormalities and is a valid therapeutic option.

39. EVALUATION OF DISEASE SEVERITY FOLLOWING ACUTE PARAQUAT POISONING BY APACHE II SCORES

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<u>Objective</u>: To study the usefulness of Acute Physiology And Chronic Health Evaluation(APACHE) II scores in risk stratification for patients with acute paraquat poisoning. <u>Methods</u>: The medical records of 58 adults with acute paraquat poisoning in our hospital between 1990 and 2000 were reviewed retrospectively. Demographic data, necessity of mechanical ventilation, duration of hemoperfusion, and APACHE II scores were collected for further analysis. <u>Results</u>: The mean age at the time of diagnosis was 40.5 ± 16.0 years old (ranging from 17 to 78) with 46 men and 12 women. The overall mortality rate was 72.4% (42/58). The non-survivors (n = 42) had higher APACHE II score (23.9 ± 12.6) than the survivors (n = 16) (7.0 ± 4.3) (p < 0.001). The ingesting amount of paraquat, plasma paraquat concentration, peak of fractional inhaled oxygen (FiO2), GPT, GOT, and serum creatinine, were significantly higher in non-survivors than those in the survivors (p < 0.05 in all comparisons). For the linear regression analysis, there were significant correlation between APACHE II score and ingesting amount of paraquat, plasma paraquat concentration, FiO2, and GOT. (p < 0.008 in all comparisons). All patients whose APACHE II score over 20 died before discharge. <u>Conclusions</u>: APACHE II score is a useful index to evaluate the severity and in-hospital mortality in paraquat poisoning.

40. METHYLPHENIDATE INGESTION IN PRE-SCHOOL CHILDREN: A CASE SERIES

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Introduction: Given the number of methylphenidate (M) prescriptions that are on the increase, the opportunity for younger siblings unintentional exposure is also on the rise. Symptoms associated with M ingestion in pre-school children have not been previously defined. Methods: We enrolled and followed by telephone children aged <6 years with M ingestion at time of first call to the poison control centre (PCC) over a 3 year period. Results: A total of 49 children aged 30 ± 11 months were included. Patients ingested a median of 1 tablet (range 0.25-10) for a total median dose of 0.9 mg/kg (range 0.26-12). Twenty-one patients were referred to a health care facility (HCF) and the PCC recommended

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activated charcoal (AC) for 12 of them. Patients referred to a HCF ingested more M: median 1.8 vs. 0.7 mg/kg (p < 0.001). Patients for whom AC was recommended ingested more M: median 3.5 vs. 0.8 mg/kg (p < 0.001). Twenty-four patients developed symptoms: agitation or irritability was present in 17, somnolence in 5, vomiting in 2, abdominal pain in 2. Two patients became tachycardic. Patients who ingested an extended release preparation had similar rate of symptoms as those who ingested a regular preparation, 7/9 vs. 17/40 (p = 0.07), while having ingested similar amounts, median 1.6 vs. 0.8 mg/kg (p = 0.08). Patients who ingested ≤ 1 tablet had a similar rate of symptoms as those who remained asymptomatic: median 1.2 (range 0.26–12) vs. 0.8 mg/kg (range 0.26–3.8) (p = 0.08). Conclusion: Pre-school children ingesting less than 1 mg/kg of methylphenidate can be safely managed at home in spite of the fact that some will develop minor symptoms.

41. ESCITALOPRAM, A REVIEW OF ADVERSE EFFECTS IN OVERDOSE REPORTED TO SELECT REGIONAL POISON CENTERS

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<u>Background</u>: Escitalopram is the *S*-enantiomer of the racemic SSRI antidepressant citalopram. While more potent than citalopram, escitalopram was developed to reduce the side effects seen with citalopram use. Escitalopram has been available for less than a year and the adverse effect profile seen in overdose has not been fully established. <u>Case Series</u>: A non-randomized retrospective observational study was conducted using data collected from three regional poison centers. Data were abstracted from Toxicall[®] records for analysis and included patient age, gender, drug dose, clinical effects, therapies, and clinical outcomes. Of the 82 ingestions identified, only 14 cases (ages 16 to 44, mean 25.0; 43% male) had isolated escitalopram ingestions of greater than 100 mg with documented follow-up. In these abstracted cases, doses ranged from 100 to 600 mg (mean 235.7 mg) and all but one case was thought to be the result of attempted suicide. Four out of 14 (29%) patients had drowsiness/lethargy, 2/14 (14%) agitation/irritability and one patient (7%) had tachycardia. These symptoms did not appear to be dose related. Activated charcoal was given in 9/14 (64%) cases while the remaining 5/14 (36%) were treated with observation only. Escitalopram overdose was judged to have had no effect in half (7/14) of the patients and a minor effect in the remaining half (7/14). These outcomes did not appear to be dose-related. No seizures were reported and QT intervals were not specifically tracked. <u>Conclusion</u>: Isolated ingestion of escitalopram up to 600 mg appears to be associated with only minor clinicaleffects.

42. NOT NICE TO LICE^(R)—NOT SO NICE TO EYES

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<u>Background:</u> Not Nice To Lice[®] is labeled a non-toxic, pesticide-free shampoo for head lice and nits. Directions suggest a towel for eye protection but no other warnings. Ocular exposure has caused injury ranging from irritation to visual defect. <u>Case Series</u>: Review of 12 cases reported to a poison center found 83% managed in a HCF and 50% of patients had moderate effects. A 2 year old presented with irritation, lid and facial erythema and edema, and temporary vision loss. Ophthalmology found 80% and 60% corneal burns. Two patients reported permanent worsening of pre-existing eye conditions. TESS data for the same time had 123 cases. Symptoms included: irritation/pain (n = 105) corneal abrasions (19), blurred vision (17), lacrimation (14), and visual defects (2). (multiple symptoms in some patients). <u>Conclusion:</u> Not Nice to Lice[®] contains "protease" and other enzymes and has a pH of 3.5–4.2. The product may cause significant eye injury which may be delayed. Jointly with the AAPCC we have notified the FDA to review the warning label and product advertising.

43. A REVIEW OF SEIZURES REPORTED TO THE FDA IN ASSOCIATION WITH USE OF DIETARY SUPPLEMENTS

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<u>Background</u>: Seizures in patients using dietary supplements have been reported to the FDA Med Watch system but not formally evaluated. <u>Methods</u>: Through the Freedom of Information Act, we requested all dietary supplement cases in which seizures were reported. Using a previously established framework assessing probability of causation, three reviewers independently evaluated each case based on three criteria: temporal relationship, biological plausibility and underlying risk factors. A consensus score was reached among the reviewers, determining each case to be unrelated, possibly related, or likely related event. <u>Results</u>: We evaluated 65 reports made to the FDA from 12/30/90 to 3/3/99. Our consensus score 21 as likely related, 13 possibly related, and 9 unrelated; 5 cases were judged as not seizures, and 17 cases had insufficient information for evaluation. In the 21 "likely related" cases, 20 involved ephedra; 15 herbal caffeine, and in 1 case, the supplement had no herbal constituents but an array of elemental salts. The most common ingredient implicated in the 13 "possibly related" cases was ephedra (n = 8); caffeine-containing herbs were in 5 of those products. Creatine was the only implicated ingredient in 2 "possibly related" seizure events. Seizures were likely or possibly secondary to hypoglycemia in 3 cases and cardiovascular events in 2 cases. Weight loss (44%) and athletic performance enhancement (32%) were the most common reasons for supplement use. <u>Conclusions</u>: Ephedra was the most common ingredient associated with dietary supplement-induced seizures reported to the FDA MedWatch program. Other implicated substances included caffeine and creatine.

44. MASSIVE VENLAFAXINE OVERDOSE RESULTING IN ARRHYTHMOGENIC DEATH

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<u>Background</u>: The new SSRIs are generally safer in overdose than the older TCAs, however, bicyclic, venlafaxine has been reported to cause seizures and cardiac toxicity in people, and sodium channel blockade in animals. <u>Case Report</u>: A 43-year-old female ingested 22.5 g, 1–2 hours prior to arrival. She complained of nausea and abdominal discomfort, and had stable vital signs. She was given activated charcoal, and airlifted to a tertiary center. Upon arriving, the patient had a tonic clonic seizure that lasted less than a minute. Her heart rate was 40 bpm and systolic BP 80 mmHg. The trachea was intubated and she received atropine and dopamine IV. The electrocardiogram showed 110 bpm, QRS 100 ms, and QTc 510 ms. The patient had multiple tonic clonic seizures, which were refractory to dilantin 1 g given twice and multiple benzodiazepine administrations. Despite three vasopressors, the arterial pressure continued to decline to 43/22 mmHg. The QRS complex continued to increase in length, despite a serum pH of 7.53, after seven ampules of sodium bicarbonate. The cardiac rhythm decompensated into a lethal ventricular tachycardia and asystole. The patient died roughly 10 hours post ingestion. <u>Conclusion</u>: Previous case reports have shown that venlafaxine can have arrhythmogenic properties in overdose. The prolonged QTc interval and QRS complex in this case demonstrates venlafaxine may possess both type I and type III antidysrhythmic properties in a large overdose.

45. TREATMENT OF MODERATE TO SEVERE PARAQUAT POISONING WITH VINCRISTINE AND DEXAMETHASONE

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<u>Background</u>: Universally, the mortality rate of poisoning by paraquat, a bipyridyl herbicide, is high and there is yet no proven effective treatment. At Siriraj Hospital, the largest tertiary care hospital in Thailand, a regimen consisting of intravenous vincristine and intravenous dexamethasone has been recommended for treatment of the moderate-to-severe and the acute-fulminant paraquat poisoning groups. The rationale relies on the suppression of pulmonary inflammatory reaction, that is the main cause of fatality, by using a medication with less adverse effects than the traditional use of cyclophosphamide. We are reporting the outcome of moderate to severe paraquat poisonings treated with vincristine and dexamethasone. <u>Method</u>: Medical records of patients presented to Siriraj Hospital, whose urine dithionite tests for paraquat were positive and who were



treated with vincristine and dexamethasone, were reviewed. <u>Results</u>: Of the 26 medical records reviewed, 20 had clinical courses compatible with moderate to severe paraquat poisoning. All developed acute renal failure and the survival rate was 70%. Fatalities were due to progressive respiratory failure. In the surviving patients, no adverse effects from treatment were observed. The other 6 out of 26 patients developed fatal acute-fulminant poisonings. The outcome of treatment in this series is comparable to those previously reported in cases treated with cyclophosphamide-containing regimens. <u>Conclusion</u>: We report a treatment of moderate to severe paraquat poisoning with vincristine and dexamethasone.

46. PEDIATRIC TRIMEDOXIME (TMB4) AND ATROPINE POISONING

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<u>Objective</u>: To describe the effects of combined TMB4 and atropine poisoning in children after accidental injection with automatic injectors (AI) found in the personal chemical warfare defense kits (PCWDK) distributed to the Israeli population during the recent war in Iraq. Lack of life-threatening adverse effects in 268 cases of pediatric atropine only injections during the 1991 crisis was described elsewhere. Since then, the atropine only AI was replaced by an AI containing 0.5–2.0 mg of atropine and 20–80 mg of TMB4 (dose according to the age of the person owning the PCWDK). There are no previous reports of accidental TMB4 exposure. <u>Methods</u>: Records of all pediatric patients (0–18 years) presented to two pediatric emergency departments during January–April 2003 after and injection of AI were reviewed. <u>Results</u>: 15 children 1–15 years of age presented to the pediatric emergency room 30–120 min after an accidental injection was mostly upper extremity. There were no side effects characteristic to oximes—no patient demonstrated drowsiness, dizziness, nausea or muscular weakness. There were only few side effects attributable to atropine: tachycardia (range of maximal HR: 67–164), dryness of mucous membranes (5/15). There were minor local complications—pain and local swelling. No side effect required any specific medical intervention, and all the patients were discharged from the emergency room within 4–6 hours after the injection. <u>Conclusions</u>: In this series, accidental pediatric atropine and TMB4 injection, even an adult dose in a small child, was not associated with significant side effects.

47. SERUM ENZYMES ACTIVITY IN CASES OF CORROSIVE POISONINGS (CP)

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<u>Objective</u>: We wanted to study if an activity of blood serum enzymes is an informative feature for diagnosing corrosive poisoning (CP). <u>Methods and Materials</u>: In the study we included 101 patient (69 males; 32 females) poisoned by 70% and 9% acetic acid (n = 48), mineral acids (n = 17), alkali (n = 10), bleaches (n = 13), and unknown CP (n = 13). Activity of serum enzymes was determined on days 1, 4, 6, 8, etc since the day of hospitalization. <u>Results</u>: Maximal elevation of the transaminase activity—AST was registered on the 3rd day (114.2 ± 80.8 U/L), ALT up to 62.8 ± 14.2 U/L on the 1st day, becoming normal on the 3rd day. The activity of creatine kinase (CK) was maximal on the 3rd day (1079.3 ± 582.1 U/L) in cases of acetic acid poisoning. Values lactic dehydrogenase stayed increased till up to 7–10 days of the poisoning (530.5 ± 116.4 U/L), while the alkaline phosphatasa was characterized by elevation (up to 125.2 ± 13.2 U/L). We found the increase of activity of serum enzymes to be directly proportional to the severity of CP (see Table 1).

Table 1.	Activity	of	enzymes	among	\mathbf{CP}	group	with	different	degrees	of
severity by	Persson	et	al. (1998)							

Agent	Activity of enzymes (U/L)					
	ALT	AST	СК			
All CP $(n = 101)$	71.6 ± 18.9	94.8 ± 25.7	351.3 ± 72.4			
I degree	35.9 ± 8.2	32.5 ± 4.9	123.9 ± 24.3			
II degree	28.9 ± 3.5	35.4 ± 4.6	229.9 ± 70.3			
III degree	119.6 ± 52.8	136.7 ± 56.9	438.7 ± 166.3			
IV degree	146.3 ± 47.8	329.0 ± 164.3	663.6 ± 205.4			

The measure of informativeness by Kullback of serum enzymes activity for diagnosing the outcome of CP was high: ALT (J = 1.42), AST (J = 1.34), and CK (J = 1.82). <u>Conclusions</u>: Activity of serum enzymes reflects the severity of CP and is a feature of its outcome.

48. RETROPERITONEAL HEMORRHAGE AND KIDNEY DAMAGE DUE TO TRAUMA CAUSED BY PLAY-FIGHTING DURING A COCAINE HIGH

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<u>Background</u>: Although the acute manifestations of cocaine toxicity are well known, bizarre trauma related to high-risk behavior during cocaine use is largely unreported. <u>Case Report</u>: A 23 year old male with a history of daily alcohol and occasional cocaine use was admitted to our hospital after a Christmas party, because of acute abdominal pain. He was otherwise healthy, apart from asthma that was occasionally treated with salbutamol. On arrival, he complained of pain in his left lower quadrant, had a peritoneal reaction, and was pale and sweaty. There were no visible hematomas. Urine was positive for cocaine. Abdominal x-ray was negative, but an ultrasound showed intraperitoneal free fluid. Due to a metabolic acidosis (pH 7.28, BE -5.2, HCO₃ 20 mmol/L) intestinal ischemia was suspected. Emergency explorative laparotomy found a large retroperitoneal hematoma but no cause of bleeding, despite assistance from vascular surgeons. Postoperatively, because of a falling hemoglobin (from 8.4 to 4.9 mmol/L) and persistent macroscopic hematuria, transfusion was required. A subsequent abdominal CT scan showed rupture of the lower pole of his left kidney. The patient later admitted to trauma caused by a competition while high on cocaine. The goal was to determine who could take the hardest blows to the body. He recovered uneventfully, apart from a prolonged infection. <u>Conclusion</u>: Impaired judgement combined with the anaesthetic effects of cocaine may result in injuries that mimic manifestations of toxicity. Clinicians should always suspect occult trauma in patients who use mind-altering substances.

49. THE MYSTERY ABOUT THE GAS USED FOR THE RELEASE OF THE HOSTAGES IN THE MOSCOW MUSICAL THEATER

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Introduction: Two German hostages were transferred from Moscow to our hospital short after their release from the Moscow musical theater where they were narcotized by an unknown gas. <u>Objective</u>: The question arose by which agent this could have been achieved. <u>Case Reports</u>: 1. A 18 year old girl lost her consciousness very suddenly. She only remembered a smell. The girl was admitted to a children's hospital in a critical state GCS 3 points bradycardia, brachypnoea, BP: 70/30. She was put on artificial ventilation. She was flown out to Munich where she arrived 26 hours after she had inhaled the gas. 2. A 47 year old man lost consciousness and woke up 5 hours later in Botkin hospital. He had many abrasions in the face and over the hip. He arrived in good condition in Munich. <u>Results</u>: Results of the toxicological analysis: A broad immunological analysis (CEDIA) in the urine revealed benzodiazepines in the girl. In the HPLC we found Metoclopramid. No drugs could be detected in the man's urine or blood. GC/MS/SIM performed in the blood and urine of the girl showed halothane in both materials. The sample were analyzed for fentanyl using MTPA which could not be found. The urine of the male victim showed halothane, no fentanyl was detected. <u>Conclusion</u>: The gas used for the liberation of the hostages in Moscow contained halothane. The clinical signs and symptoms point to an opioide. Fentanyl could not be found. It may have been too late to detect it at the arrival in Munich or it may have been a derivate of fentanyl.

50. THE USE OF TABLES FOR PROGNOSIS OF LIFE-THREATENING STATES REQUIRING ICU HOSPITALIZATION IN CASES OF ACETIC ACID POISONINGS (AAP)

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<u>Background</u>: Traditionally patients with life-threatening states and with a complicated development of the disease require resuscitation. Making use of non-homogeneous consecutive procedure (a modification of consecutive Wald's analysis)



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we propose a diagnostic table, which on the basis of 13 features is used for prognosis of development of life-threatening states in cases of acetic acid poisonings (AAP) (Yamanaeva and Sarmanaev, 2001). In making the prognosis, the following possible outcomes are considered: favorable, unfavorable, doubtful. <u>Aim</u>: Approbation of the prognostic tables in cases of AAP during clinical practice, estimation of its sensitivity and specificity. <u>Materials and Methods</u>: The state of 102 patients with AAP, hospitalized at the Toxicological Center of Ufa was estimated with the help of the prognostic table. An independent practitioner made the decision as to the conduction of resuscitation. To determine the informative value of the table-based method, ROC-analysis was used. <u>Results</u>: A correct prognosis was made in 75.5% of all cases, wrong prognoses made up 2.9%, the prognosis was doubtful in 21.6% of the cases. The prognostic value of the positive result of the test amounted to 94.4%, that of the negative result was 91.9% (excluding doubtful cases). The area under the ROC curve made up 0.84 \pm 0.05. The most optimal "cut off" was estimated to be "+5" (the sensitivity was 97.2% and the specificity was 70.0%). <u>Conclusions</u>: 1. The elaborated table-based method is able to correctly estimate the future development of life-threatening states. 2. The proposed way of making a decision as to carrying out resuscitation on the basis of the prognosis of the development of life-threatening states. 2. The proposed way of making a decision as to carrying out resuscitation on the basis of the prognosis of the development of life-threatening states helps to determine the groups of patients for preventive therapy in cases of AAP. <u>References</u>: Yamanaeva IE, Sarmanaev SKh. Use of Wald's sequential test for grading the severity of acetic acid poisoning (AAP) and for predicting its outcome. Toxicol Clin Toxicol 2001; 39:509.

51. UNINTENTIONAL PEDIATRIC VITAMIN D INTOXICATION

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Background: Vitamin D (vit D) is a hormone that has a lower therapeutic index than many other vitamins. We report an unintentional overdose of an imported high dose vit D supplement with resultant resistant hypercalcemia. Case Report: A 2 year-old previously healthy boy was brought to the ED complaining of 3 days of constipation and lethargy. His only medication was Raquiferol[®] (calciferol), an imported Latin American vit D supplement. VS: BP, 139/98 mmHg; P, 88/min; R, 16/min; T, 97.6°F. The child was somnolent and had a nonfocal neurologic examination. His abdomen was nontender and bowel sounds were normal. Laboratory investigation revealed a serum Ca^{2+} of 14.4 mEq/L with a corresponding vit D concentration of 106 ng/mL (normal 10-68 ng/mL, measuring combined cholecalciferol and 25hvdroxvergocalciferol). ECG was normal sinus rhythm with normal intervals. Despite therapy with intravenous D51/2NS, furosemide, calcitonin, and hydrocortisone, the Ca²⁺ increased to 15.0 mEq/L on the 2nd hospital day and did not decrease until the 4th hospital day to 13.9 mEq/L. The vit D concentration peaked to 470 ng/mL on hospital day 3. There were no complications during the 2-week hospitalization and the vit D concentration on discharge was still elevated at 389 ng/mL. The instructions on the supplement stated that two drops from the ampule should be administered daily. Each drop has 2500 IU of vit D2 (calciferol) and each ampule has 15 mg or 600,000 IU. The mother innocently administered an entire ampule daily for 4 days (total 3.6 million IU) to the child and the last dose was 8 days before presentation. The US RDA of vit D is 200-400 IU, 10 fold lower than that obtained with the administration of this supplement. Conclusion: Vit D overdose in this case caused prolonged hypercalcemia and hypertension. This case highlights the risk of parental dispensing errors and unregulated imported products.

52. CONSERVATIVE MANAGEMENT OF ELEMENTAL MERCURY SEQUESTRATION IN THE APPENDIX

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<u>Background</u>: Few cases of mercury sequestration in the appendix appear in the literature. Based on these cases appendectomy is often recommended. We report a case of conservative management of retained gastrointestinal elemental mercury resulting in no evidence of mercurialism or appendicitis two months after ingestion. <u>Case Report</u>: A 42 year old white male ingested approximately 42 mL of elemental mercury after an argument with his wife. He presented to the emergency department within 2 hours of ingestion, asymptomatic and with a normal physical exam and vital signs. An abdominal radiograph series showed mercury contained in the pylorus of the stomach. Initial laboratory testing included a normal complete blood count, electrolytes, renal function and liver function studies. A follow-up

radiograph at 48 hours showed mercury localized to the appendix, and heavy metal testing at that time revealed a 24-hour urine mercury level of 15 mcg/L (reference range < 20 mcg/L) and blood mercury level of 68 mcg/L (reference range < 10 mcg/L). Thirty-two days after the initial ingestion, the patient remained asymptomatic with a normal physical examination. Repeat abdominal x-rays still showed mercury sequestered in his appendix. A repeat blood mercury level at that time was 8 mcg/L. The patient was reexamined several times in the intervening month and remains asymptomatic at the time of this writing. <u>Conclusion</u>: Asymptomatic patients with retained gastrointestinal elemental mercury may be conservatively managed without appendectomy.

53. THIORIDAZINE INDUCED TORSADES DE POINTES TREATED WITH SODIUM BICARBONATE AND TRANSVENOUS PACING

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Background: Thioridazine (TDZ), and its metabolite mesoridazine (MDZ), are used for the treatment of psychosis. Overdose with either can result in toxicity characterized by depressed mentation, seizures, and torsade de pointes (TDP) similar to cyclic antidepressants (CAs). Five cases are reported in the literature using overdrive transvenous pacing (TVP). Experimental models suggest sodium bicarbonate (NaHCO₃) may be a suitable alternative. In this case both modalities were used based on different physician's familiarity with treating TDZ induced TDP. Case Report: A 71 yearold female presented to the ED via EMS. The pt went to sleep in the early evening and was found obtunded 4 hrs later, with 33, TDZ (200 mg) tablets missing. PMHx: hypertension, anxiety. Other meds: alprazolam, triazolam, hydrochlorothiazide. ED arrival VS: T, 96.9; P, 85; RR, 14; BP, 112/75; Pulse. Ox, 95% room air. Pts exam was unremarkable except a GCS 5. ECG done at exam revealed NSR, rate 84, QRS 97 ms, QTc 479 ms, QRS axis of -31. Pt was intubated, lavaged, given 50 g activated charcoal. Labs: Na⁺, 119 mmol/L; K⁺, 3.2 mmol/L; Mg²⁺, 2.2 mg/dL. UDS: positive for benzodiazepines, negative for all others including CAs. While in the ED, the pt developed intermittent TDP, which did not respond to 2 g of MgSO₄ or 100 mg lidocaine, but did resolve with 1 amp of NaHCO₃ on two occasions. In the ICU, the pt suffered frequent TDP. The cardiologist effectively used TVP, in lieu NaHCO₃, to treat pts TDP. The pt fully recovered to baseline in 8 days and was discharged to psychiatry. Serum TDZ and MDZ levels obtained 72 hrs after admission indicated 1136 and 1146 ng/mL respectively. These levels were in the middle and upper reference ranges respectively. Conclusion: Thioridazine induced TDP can be treated by NaHCO₃ and TVP. Choice of therapeutic modality may be based on the physician's preference and experience.

54. CLONIDINE INGESTION IN CHILDREN

Spiller HA, Colvin JM, Villalobos D, Johnson PB, Anderson DL, Klein-Schwartz W. Kentucky Regional Poison Center and the Clonidine Study Group.

Background: 84% of pediatric clonidine cases reported to TESS are evaluated in a HCF. However 77% of these children experience minor or no effects. We performed a prospective case series to evaluate if there were dosage or toxicodynamic parameters to better separate those cases that need direct medical evaluation from those that can be observed at home. Method: All clonidine ingestions in children ≤ 12 years old reported to six poison centers were followed for a minimum of 24 hours. Exclusion criterion was polydrug ingestion. Results: The study included 109 patients of whom 63 were male. Mean age was 3.8 years (S.D. \pm 2.4), with a range of 9 months to 11 years. Ninety-three patients (85%) were evaluated in a HCF, of which 59 were admitted for medical care or observation. Clinical effects were common and included: drowsiness (n = 77), bradycardia (n = 11), hypotension (n = 9), hypertension (n = 4), coma (n = 6), respiratory depression requiring intubation and ventilation (RD) (n = 7), and hypothermia (n = 1). Dose ingested was known for 88 patients (81%). Fifty-seven of 88 (65%) children ingested < 0.3 mg with 0 experiencing coma, RD or hypotension. The lowest dose ingested by history with coma and RD was 0.3 mg (0.015 mg/kg). This occurred in a 4 year-old 20 kg patient with no previous exposure and a history of cerebral palsy. Prior clonidine therapy did not effect outcome, incidence of coma, hypotension, or respiratory depression (p > 0.05). Onset of full symptomatology in all cases was complete within 4 hours of ingestion with a mean time to maximum level of CNS depression of 1.7 hours (range of 1–3 hrs). Conclusion: Ingestion of >0.2 mg may warrant direct medical evaluation. Observation for 4 hours may be sufficient to detect patients who will experience coma, hypotension, bradycardia, or respiratory depression.



55. RISK FACTORS OF PNEUMONIA DEVELOPMENT IN CASES OF POISONINGS BY ACETIC ACID (AAP)

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<u>Background</u>: The frequency of pneumonia during acute poisonings makes up 20.1–41.1% of all cases. Despite recent successes in treatment, lethality in the case of pneumonia development reaches 42–59%. The aim of the present work was to study the frequency of pneumonia developments and determine its risk factors in cases of acetic acid poisoning (AAP). <u>Methods and Materials</u>: The criteria of including a case in the study were AAP and the presence of the analyzed characteristics. Pneumonia was verified by means of chest x-ray control or post-mortem examination. The following factors were analyzed: the severity degree by IPCS/EC/EAPCCT PSS (Persson et al., 1998), age, alcohol abuse, amount of drunk liquid, exposition, aspiration, dysphony, breath frequency, degree of consciousness depression, hospitalization in ICU, hemolysis, oliguria, leucocytosis, bilirubinemia, gastro-intestinal bleeding, DIC, shock, glucocorticoid dosage. The study of the risk factors was carried out by means of a multiple regression analysis in the group of 385 patients. <u>Results</u>: The frequency of pneumonia cases amounted to 7.74% (34 patients). Lethality is the case of pneumonia development was 50%. A regression model was built for prognosis of pneumonia development. Most informative features were found to be: aspiration, DIC, dysphony, dosage, oliguria, the degree of severity, exposition, depression of consciousness, hospitalization in ICU, leucocytosis. <u>Conclusions</u>: The development of pneumonia in cases of AAP can be predicted using factors: anamnesis data, degree of severity and the fact of hospitalization in ICU.

56. TIZANIDINE (ZANAFLEX[®]) EXPOSURE

Adamson LA, Spiller HA, Bosse GM. Kentucky Regional Poison Center, Department of Emergency Medicine, University of Louisville, Louisville, Kentucky, USA.

Background: Tizanidine is a centrally acting muscle relaxant structurally related to clonidine. There are no large case series of tizanidine exposure. Methods: Retrospective review of all ingestions involving tizanidine reported to a poison control center from January 2000 through February 2003. Exclusion criteria were polydrug ingestion, no follow-up or lost to follow-up. Results: There were 121 cases of which 45 patients met entrance criteria. Mean age was 32 years (range 1 to 80). Thirty-seven patients were evaluated in a health care facility of which 27 were admitted for medical care. Clinical effects included lethargy (n = 38), bradycardia (n = 14), hypotension (n = 8), agitation (n = 7), confusion (n=5), vomiting (n=3), and coma (n=2). Mean dose ingested by history was 72 mg (S.D. \pm 86). The lowest dose associated with hypotension was 28 mg, which occurred in a 63 year-old female with a BP of 88/52 and a HR of 54. The lowest dose associated with coma was 120 mg, which occurred in a 30 year-old female with a HR of 30 and BP of 81/48. There were six patients <6 yrs (two 1 year-old patients and four 2 year-old patients). The lowest dose with bradycardia and drowsiness was 16 mg in a 2 year-old (weight unknown). All other cases in children <6 yrs involved ingestion of a single tablet (2 or 4 mg) with only mild drowsiness reported. Therapy in this series was primarily supportive and included pressors in three cases and intubation in three cases. Naloxone was administered to seven patients. There was no response to naloxone in five patients, poor documentation of response in one, and arousal in one patient. All patients recovered without residual complications. Conclusion: Clinical manifestations of tizanidine overdose include alterations of mental status, bradycardia, and hypotension. In this series, outcome was good with supportive therapy.

57. FOMEPIZOLE IS NOT SUBSTANTIALLY ELIMINATED BY CONTINUOUS ARTERIOVENOUS HEMODIALYSIS (CAVHD)

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Introduction: Fomepizole elimination by continuous arteriovenous hemodialysis (CAVHD) is largely unstudied. We report a case in which serial serum fomepizole levels obtained from the arterial and venous dialysis lines were measured and the amount of drug eliminated during CAVHD calculated. Case Report: A 31-year-old female who drank an unknown

amount of a de-icing agent containing 100% methanol developed a severe anion gap acidosis with a serum pH of 7.06. She was started on intravenous fomepizole and CAVHD simultaneously until hemodialysis could be arranged. She received approximately 8 hours of CAVHD followed by 4 hours of hemodialysis and then another 8 hours of CAVHD. During the second episode of CAVHD two separate and simultaneous blood samples were taken from both the arterial and venous dialysis lines of the CAVHD unit on two separate occasions 4 hours apart. Mean fomepizole concentrations for the two sampling times were (mean arterial/mean venous): 323/305.5 and $192.5/189 \,\mu$ mol/L. Calculated fomepizole extraction ratios were 5.4% and 1.9% for the first and second sets respectively. The reported extraction ratio of fomepizole during hemodialysis is 75%. During CAVHD, total fomepizole elimination ranged between 29.1 and 32.6 μ mol/L/hr. Endogenous fomepizole elimination is reported to be zero order at these concentrations and ranges from 13 to $16 \,\mu$ mol/L/hr. Conclusion: Although CAVHD doubled total elimination, relatively low levels of fomepizole are needed to inhibit formate production ($10 \,\mu$ mol/L), which suggest that no additional dosing is necessary during CAVHD. The low extraction ratio of fomepizole during CAVHD also suggests that it is not substantially eliminated.

58. EARLY HEMODIALYSIS IN ACUTE FORMALIN INGESTION

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<u>Background</u>: Large ingestions of formalin often result in serious toxicity and death. A prior report described fatalities in association with initial plasma formic acid levels >200 µg/mL. The use of early hemodialysis after an ingestion of formalin has not previously been described in the literature. <u>Case Report</u>: A 41 year-old female presented to the ED 1 hour after an intentional ingestion of approximately 180 mL of formalin (5% methanol). She had persistent vomiting and complained of burning in her throat and abdomen. Her initial vitals signs were: T, 37.3° C; P, 111/min; BP, 92/52 mm/Hg; RR, 18/min; and O₂ saturation on room air, 95%. Physical exam demonstrated moderate oropharyngeal erythema, moderate epigastric pain to palpation and a normal neurologic exam. Her initial laboratory studies included CO₂ 13 mmol/L, anion gap 18, methanol level 32 mg/dL, and a serum formic acid level >390 µg/mL. Other laboratory studies revealed undetectable amounts of acetaminophen, salicylate, ethanol, ethylene glycol, acetone, isopropanol, and a negative urine drug screen. Fomepizole and folate were administered and the patient underwent emergent hemodialysis 3 hours post-ingestion. Laboratory studies after dialysis revealed resolution of her metabolic acidosis and a methanol concentration of 5 mg/dL. An esophagogastroduodenoscopy demonstrated inflammation of the esophagus, stomach, and duodenum. The patient went on to make a complete recovery. <u>Conclusion</u>: In our patient, the early use of hemodialysis after acute formalin ingestion appears to have prevented significant systemic formic acid toxicity and the adverse sequelae associated with it.

59. EPIDEMY OF ACUTE RESPIRATORY ILLNESS LINKED TO USE OF WATERPROOFING TEXTILE AND LEATHER SPRAY

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<u>Background</u>: Acute respiratory symptoms after use of aerosol leather conditioner or textile waterproofing sprays have been reported in the literature on several occasions. These cases occurred after the sprays had been reformulated. The pneumotoxic effects have been attributed in part to the waterproofing fluorocarbon polymers and the solvents, with the mist particle size being the critical parameter for toxicity. <u>Case Series</u>: Our poison centre has become aware of a sharp increase of cases of respiratory symptoms after use of two recently reformulated textile and leather sprays during the last few weeks of 2002. Whereas the average annual number of similar cases was 5 to 10 during the last 7 years, it totalled to 45 in 2002 and to 108 during the first 3 months of 2003. Symptoms involved cough, shortness of breath, inability to smoke, and tachycardia. Some of the patients were admitted to hospital with fever, malaise, decreased arterial oxygen saturation, and pulmonary infiltrates. All patients recovered, although a few stayed in the hospital for up to 8 days. The sprays involved were withdrawn from the market. The particular reasons for this overt toxicity are not yet clear and are currently under investigation. <u>Conclusion</u>: These observations and the previously reported epidemics demonstrate that reformulation of aerosol waterproofing sprays may lead to a marked increase of respiratory toxicity, most probably



due to a decrease in mist particle size. Poison centres are particularly able to detect such effects at an early stage and warn the population and the authorities. We suggest that determination of particle size in such sprays becomes a mandatory feature in product regulation.

60. USE OF FLUMAZENIL FOR LORAZEPAM-INDUCED PARADOXICAL REACTIONS IN CHILDREN

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Background: Paradoxical reactions (irritability, agitation, and disinhibition) have been reported in up to 30% of children treated with benzodiazepines. Flumazenil has been reported to successfully reverse such reactions induced by therapeutic use of midazolam. We report two pediatric cases of lorazepam-induced paradoxical reactions that were successfully reversed with flumazenil. <u>Case Reports</u>: Patient 1: A 23-month-old child ingested an unknown amount of lorazepam and was brought into the hospital 45 min later with mild CNS depression. By 60 min post ingestion, the child was agitated and screaming. Symptoms persisted for 3.5 hrs at which time flumazenil 0.01 mg/kg IV was given and the child fell into a restful sleep. Approximately 1 hr later the agitation returned and flumazenil was repeated with similar results. The child received six doses of flumazenil over 6 hrs, each resulting in the child falling asleep restfully. By 14 hrs post ingestion the child had fully recovered and had no further sequelae. Patient 2: A 6-year-old developed apparent hallucinations but no agitation following two doses of lorazepam administered 4 hrs apart to treat insomnia. Flumazenil was administered, the hallucinations completely resolved within 15–20 min and the child was discharged after 1 hour with no return of symptoms. <u>Conclusions</u>: Flumazenil has successfully reversed lorazepam-induced paradoxical reactions in two young children.

61. INTENTIONAL CARDIOACTIVE STEROID POISONING FROM KYUSHIN, A TRADITIONAL JAPANESE MEDICATION

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<u>Background</u>: Cardioactive steroid (CAS) poisoning has resulted from the use of traditional medications. Although specific identification is difficult, many of these compounds can be detected with polyclonal digoxin assays because of cross-reactivity. We describe the first suicidal overdose of Kyushin, and its laboratory analysis. <u>Case Report</u>: A 54 year-old woman with a history of depression, taking Zoloft, presented to the ED after a suicidal overdose of Kyushin, a traditional Japanese preparation marketed for "anxiety and anti-fast heart beat" labeled as containing "toad venom." She was obtunded and required intubation. Her pulse was 29/min and regular and her BP was 126/68 mmHg. The ECG revealed a junctional rhythm at 41/min with prominent PVCs and delayed after-depolarizations. Her serum potassium was 5.7 mEq/L. After 0.5 mg of atropine IV her pulse increased to 98/min. A digoxin serum concentration (MEIA; Abbott AxSym[®]) was 0.7 ng/mL. Ten vials of digoxin-specific Fab were administered. Her ECG promptly normalized, and she remained hemodynamically stable without recurrent bradycardia or the need for recurrent therapy. A sample of Kyushin was obtained from the same store where the patient purchased it. Thin-layer chromatography (TLC) revealed prominent spots with the same R_f and staining as cinobufatalin, cinobufagin, bufalin, three CAS derived from *Bufo* spp. <u>Conclusion</u>: Bradycardia, hyperkalemia and PVCs are suggestive of CAS poisoning. Confirmation was obtained both by cross reactivity on digoxin assay and TLC. Digoxin-specific Fab can be used therapeutically, although dosing should not be based on levels.

62. HOMEMADE PLAY DOUGH TOXICOSES IN DOGS: 14 CASES (1998-2001)

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<u>Objective</u>: Homemade play dough often contains substantial amounts of sodium chloride. The purpose of this study was to determine a minimum toxic dose of sodium chloride in dogs following ingestion of homemade play dough, and to

correlate the dosages of sodium chloride and serum sodium levels to the clinical signs. <u>Methods</u>: Fourteen cases of dogs ingesting homemade play dough from 1998 to 2001 were evaluated. The medical records were examined for the animal's body weight, the amount of play dough ingested, the sodium chloride dosage, clinical signs, serum sodium levels, and outcome. <u>Results</u>: Twelve of 14 dogs (86%) that ingested homemade play dough showed clinical signs of hypernatremia (serum sodium > 160 mEq/L). Vomiting (9 of 14, 64%), polydipsia, and seizures (3 of 14 each, 29%) were the most common signs followed by polyuria, tremors (3 of 14 each, 21%), and hyperthermia (2 of 14, 14%). The onset of clinical signs was within the first 3 hours in 9 of 14 dogs (64%). The lowest reported dosage of sodium chloride associated with clinical signs was 1.9 g/kg. Seizures were seen in all animals with serum sodium levels greater than 180 mEq/L. No signs were seen with dosages less than 0.7 g/kg. Four of the 14 dogs had a full recovery, one was euthanized, one died, and the outcome was unknown in eight dogs. <u>Conclusion</u>: Ingestion of 1.9 g/kg of sodium chloride from homemade play dough in dogs can lead to rapid development of gastrointestinal or neurologic signs, hypernateremia, or death.

63. TWO CASES OF SEVERE "BRAKE FLUID" POISONING IN THE "MOTOR CITY"

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<u>Background</u>: "Brake Fluid" (BF) is a mixture of glycols (G) and glycol derivatives (GD) which does not contain ethylene glycol (EG). These less known glycols can produce serious or fatal toxicity. Management of BF ingestions is based on a limited number of case reports and on the similarities of all G to their related compound EG. <u>Case Reports</u>: Case 1: 33 yr/o M arrived to ED in cardiac arrest following ingestion of 12 oz of BF: polyalkylene glycols and diethylene glycol (DG) <20%. After ACLS resuscitation he had return of circulation. Gastric lavage removed a large amount of fluid consistent with BF. He had a lactic acidosis = 16, anion gap (AG) = 31, osmolar gap (OG) = 24. Volatile alcohols (VA) and EG were not detected. Fomepizole (F) was started. Hemodialysis (HD) was performed for 6 hrs while he was on vasopressors. He expired 18 hrs after arrival with refractory acidosis and shock. Case 2: 38 yr/o M brought to ED unresponsive and hypotensive after ingesting two 12 oz cans of BF: DG 13–18%, tri, tetra, penta EG and G ethers. He was intubated. Labs showed pH = 7.34, AG = 10, OG = 7, ethanol = 91 mg/dL; VA and EG were not detected. Fomepizole was started. He remained in coma with persistent hypotension. Hemodialysis was started 8 hrs after arrival and continued for 6 hrs. He was extubated next day. Renal function was normal at 72 hrs. <u>Conclusion</u>: Brake fluid ingestions can result in significant acute toxicity: coma, metabolic acidosis and shock. Serum levels are not available. An OG is not consistently present. Management with F alone, HD alone, or both is not well established. Hemodialysis should remove BF glycols with MW <500 d. Early HD plus F may prevent a major or fatal outcome.

64. IMPORTANCE OF THE EARLY PROGNOSIS OF RESPIRATORY DYSFUNCTION INDUCED BY ACUTE POISONINGS

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<u>Background</u>: The goal of the present study was to estimate the factors determining lethality in critically ill patients presenting with acute poisoning from barbiturates, neuroleptics, opioids, organophosphates, and toxic alcohols. <u>Methods</u>: Pulmonary gas exchange, cardiac output, and neutrophil granulocyte activation (lysozomal activity) were measured in 137 artificially ventilated patients. <u>Results</u>: There were 67 survivors and 70 deaths. The mean severity of poisoning was determined for each group using the APACHE-III scale on day-1 post-poisoning (score range 75–112). On day-1 there was a significant difference in arterial hypoxemia due to increased lung shunting (Qs/Qt) and the functional dead space (Vd/Vt) when comparing survivors to non-survivors. Cardiac output and arterial pressure were not different between groups. A pulmonary prognostic index (PPI) was calculated on day-1 post-poisoning to predict the outcome using the formula PPI = 81 - 131Vd/Vt - 71Qs/Qt. Of 68 patients with a negative PPI, 66 died (prognostic efficiency 97%). In non-survivors, with negative PPI, the severity of poisoning correlated with the degree of neutrophil granulocyte activation (r = 0.87). This also made it possible to predict the time of death. In 69 patients with positive PPI, 65 survived and four died, (prognostic efficiency 94%). <u>Conclusion</u>: The cause of death was acute respiratory distress resulting from systemic inflammatory response to hypoxic damage. Pulmonary prognostic index and neutrophil granulocyte activation may have prognostic value in these critically ill poisoned patients.

65. TREATMENT OF ANTICHOLINERGIC DRUG INDUCED COGNITIVE DISORDERS: CLINICAL EFFECTS AND PROGNOSIS

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<u>Background</u>: Acute 3-quinuclidylbenzylat administration to dogs has resulted in disturbance of complex conditioned reflexes for 1.5-2 months. A reversible cholinesterase inhibitor aminostigmine (AS) used for 7 days normalized disturbed reflexes whereas a single administration of the drug had no effect. The combination of pyracetam with AS appeared to be more efficient than therapy with AS alone. We therefore investigated the clinical effects of AS with pyracetam in the treatment of anticholinergic drug-induced cognitive disorders. <u>Methods</u>: Heroin addicts (45 males under 30 years with the body weight of about 75 kg, 15 days after last drug use) received the *m*-acetylcholine (Ach) receptor antagonist dexbenzetimide (DB) in a single dose of 0.5 mg i.m. In patients of the control group (n = 10) who did not receive subsequent treatment with AS, signs of cognitive disorders appeared within 7 days. In group 1 (n = 15), AS (4 mg i.m.) was administered 4 hours after DB. In group 2 (n = 10), 4 mg of AS was used on the first day and 2 mg per day was given during the next 6 days. In group 3 (n = 10), pyracetam (4 g/day) was added to the 7 days of AS therapy. Quantitative EEG and indices of memory and attention scales were recorded. <u>Results</u>: In patients of the control group and group 1 the memory and attention indices did not return to initial values. In group 3 they returned to the initial values. Positive EEG dynamics were noted in patients of groups 2 and 3. <u>Conclusion</u>: Ach-receptor degradation results in a decrease in cognitive function. Aminostigmine and pyracetam may be effective in reversing anti-cholinergic drug induced cognitive dysfunction, possibly by activation of reserve (silent) Ach-receptors, or by increasing Ach synthesis, respectively.

66. RELIABILITY OF THE GLASGOW COMA SCALE (GCS) FOR POISONING PATIENTS

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<u>Background</u>: The glasgow coma scale (GCS) was developed initially for use in neurotrauma but is now widely used to monitor changes in mental status for acutely ill patients. The reliability of this scale for use in poisoning patients is not well studied. <u>Purpose</u>: To evaluate the inter-rater reliability of the GCS for poisoning patients. <u>Methods</u>: A prospective observational study of a convenience sample of poisoning patients in the ED of two urban teaching hospitals. Informed consent was waived by the local IRB. For each patient, two care providers scored the GCS using a check off sheet listing the criteria for each score. Each provider independently scored the patient for each domain of the GCS. Not more than 5 min elapsed between assessments and no interventions took place while the assessments were performed. Specific scoring instructions were given on the sheet for intubated patients. Clinical information was also collected. Agreement was assessed using a weighted Kappa score. <u>Results</u>: 39 patients were enrolled. The mean age was 31.2 years (range 13–51); 41% were male. Physicians performed 44 evaluations, nurses performed 16 evaluations; 18 evaluators were not recorded. Glasgow coma scale ranged from 3–15; 50% of scores were 15. Overall, inter-rater agreement was excellent (Kappa = 0.84, 95% CI 0.72–0.95). <u>Conclusion</u>: The GCS is a reliable rating system for poisoning patients in the ED.

67. EPHEDRINE-INDUCED TOURETTE SYNDROME

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<u>Background</u>: The development of Gilles de la Tourette syndrome has been described in patients receiving stimulant therapy for attention deficit disorder. Presumably these patients are thought to be predisposed to Tourette syndrome. We report a case of ephedrine-induced Tourette syndrome in a previous healthy young adult. <u>Case Report</u>: A 25 year-old

Egyptian male with no previous medical history was brought to the hospital after he developed an altered mental status. The family reported that the patient had been somewhat depressed and spent most of his day swimming. The patient's only medication was an ephedra containing dietary supplement he had recently started taking for weight loss. The patient had no other medications available to him and the pill count of the dietary supplement was congruent with therapeutic usage. On presentation the patient was non-verbal with his eyes closed. Frequent facial tics were noted. Any stimulation of the patient (verbal or tactile) resulted in the patient opening his eyes and yelping (vocal tic). The patient was not combative or otherwise agitated, but was unable to offer any complaints. His vital signs were BP = 140/90 mmHg, P = 114 bpm, $T = 36.0^{\circ}$ C, RR = 14 rpm, the remainder of his examination was normal. The patient was given 10 mg of diazepam intravenously. CT of the brain and laboratory analysis were normal, including a negative toxicology screen for cocaine, opiates, and amphetamines. The patient awoke 5 hours later and had a completely normal mental status. He offered no additional information and denied overmedication. <u>Conclusions</u>: The symptoms exhibited by this patient are consistent with Tourette syndrome. This was either induced by ephedrine or unmasked by it. Physicians should be aware of yet another untoward effect of ephedrine and patients that develop these symptoms should be followed closely.

68. USE OF OCTREOTIDE IN SULFONYLUREA POISONING IN A CHILD

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<u>Background</u>: Sulfonylureas stimulate insulin release from β -cells resulting in hypoglycemia. Treatment of sulfonylurea poisoning includes IV dextrose 10%, frequent blood glucose monitoring, and supplemental dextrose 50% bolus doses as required. However, dextrose also stimulates insulin release and can produce rebound hypoglycemia. Octreotide inhibits insulin release from β -cells and has been used to treat hypoglycemia following sulfonylurea poisoning in adults. There is one previous case report in a child. We report an additional case of octreotide for pediatric sulfonylurea poisoning. Case Report: A 16 month-old child presented to hospital within 1 hr of ingesting an unknown amount of glyburide. His initial blood glucose was 3.8 mmol/L (68 mg/dL). IV dextrose 50% was given and his blood glucose rose to 4.3 mmol/L (77 mg/dL) but dropped to 2.5 mmol/L (45 mg/dL) within 30 min. Despite treatment with dextrose 10% infusion and four dextrose 50% boluses he continued to have rebound hypoglycemia. Approximately 5 hrs post-ingestion he was given IV octreotide 10 µg over 15 min. Dextrose 10% infusion was continued and blood glucose was given 8 hrs later and dextrose 10% infusion continued for another 5 hrs. Blood glucose was monitored for 6 hrs after IV dextrose was discontinued; glucose remained above 4.4 mmol/L (79 mg/dL). Child was discharged home 24 hrs post-ingestion with no sequelae. Conclusion: As in adults, octreotide is effective in treating rebound hypoglycemia following pediatric sulfonylurea poisoning.

69. A FOUR YEAR REVIEW OF PEDIATRIC LORATADINE INGESTIONS; IMPLICATIONS FOR POISON CENTER REFERRAL GUIDELINES

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<u>Background</u>: The purpose of this study was to determine the clinical effects and medical management of loratadine in unintentional pediatric ingestions reported to a poison center. <u>Methods</u>: We conducted a retrospective review of pediatric loratadine exposures (age 5 years and under) reported to 4 poison centers from 1/1/1999 through 12/31/2002. Patient age, gender, weight (where available), suspected amount ingested, treatment, symptomology, disposition, and outcome were evaluated. Claritin-D cases, multi-agent ingestions, and cases that were not followed after the initial consult were excluded. <u>Results</u>: 547 cases met the inclusion criteria (from a total of 1288 cases), 346 and 200 were managed in the home and emergency department (ED) respectively. Three hundred and ninety-seven patients (72.6%) only required observation. GI decontamination was performed by ipecac (14 cases) and ED administered activated charcoal (136 cases). No patient suffered significant sequelae and only 23 (4.2%) developed outcomes beyond minor effect (transient CNS and cardiovascular effects that did not require specific intervention). While the average suspected dose ingested by

history of the cases with moderate effect was higher (115 mg vs. 52 mg for no or minor effect), the range overlapped with an upper range of 300 mg and 310 mg for each group respectively. Eight percent of cases with no or minor effect and 35% with moderate effect had suspected doses greater than 120 mg. <u>Conclusion</u>: Unintentional pediatric loratadine ingestions reported to a poison center were rarely associated with significant outcomes. It is uncertain that these ingestions require referral to an ED based on ingestion histories alone vs. symptom-based triage criteria. To more clearly determine the toxic dose for pediatric loratadine ingestions we propose a prospective study to validate our conclusion.

70. A SURVEY OF US POISON CENTER DIRECTORS ON THE TREATMENT OF TRICYCLIC ANTI-DEPRESSANT OVERDOSE WITH CARDIOTOXICITY

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<u>Background</u>: It is well established that NaHCO₃ is effective in reducing QRS prolongation, increasing blood pressure, and suppressing ventricular dysrhythmias caused by tricyclic antidepressant overdose. Controversy lies in the optimal dosing and mode of administration of NaHCO₃ treatment in TCA overdose with cardiotoxicity. <u>Methods</u>: The directors of the 50 certified poison centers in the United States were surveyed. We obtained responses from 36 (72%) of the poison center directors. We asked the following questions: When you treat a tricyclic overdose with cardiotoxicity, do you recommend a NaHCO₃ IV bolus with repeat bolus as needed or NaHCO₃ bolus followed by continuous infusion? On a scale of 0–10 with 0 being very weak and 10 being very strong, how strong is the evidence for your decision based upon available data? <u>Results</u>: 18 directors recommended bolus and repeat bolus as needed and 18 directors recommended bolus followed by continuous infusion. On a scale of 0–10, the evidence for their decisions based upon available data was rated an average of 4.9 with a standard deviation of 1.9. The range was 0–8.5. <u>Conclusion</u>: 50% of directors of the certified poison centers recommend bolus followed by continuous infusion of 1.9. The range was 0–8.5. <u>Conclusion</u>: 50% of directors of the certified poison centers recommend bolus followed by continuous infusion of 1.9. The range was 0–8.5. <u>Conclusion</u>: 50% of directors of the certified poison centers recommend bolus followed by continuous infusion of 1.9. There are bolus as needed for the treatment of tricyclic overdose with cardiotoxicity and 50% recommend bolus followed by continuous infusion. There was no difference between the two groups. There is no consensus how to treat this condition based upon available data and the existing evidence to support directors' decisions was rated as 4.5. Further studies are necessary in order to answer this question.

71. ATYPICAL PRESENTATION INCLUDING DECEREBRATE POSTURING FROM DIPHENHYDRA-MINE (DPH) TOXICITY IN A 23 YR-OLD

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<u>Background</u>: Atypical presentations of diphenhydramine (DPH) exposures are not well described in the toxicological literature. A patient who presents to an ED in a comatose state with decerebrate posturing and horizontal nystagmus is not typical for an OD of DPH. We are presenting a patient who presented with an atypical presentation of DPH toxicity. <u>Case Report</u>: A 23 yr-old male presented to an ED unresponsive, tachycardic, tachypneic, and non-diaphoretic. On PE he was described as an unresponsive male with horizontal nystagmus having decerebrate posturing, mydriasis, dry skin, and hypoactive bowel sounds. Serum chemistry and urinalysis was significant for an anion-gap metabolic acidosis with appropriate respiratory compensation and microscopic hematuria. Transient elevation of serum creatinine was also noted. IV naloxone, thiamine, and dextrose were administered without any clinical improvement. Supportive care was provided with resolution of symptoms over the next 48 hrs. On day 2 of admission, the patient admitted to ingesting approximately 30 capsules of Unisom[®] sleep aid (1.5 g of DPH) about 3 hrs prior to his presentation to the hospital. Tox testing confirmed an elevated blood DPH level drawn on day 2 post admission of 560 ng/mL (toxicity > 100 ng/mL) and a (-) tox screen. <u>Conclusion</u>: This young man presented with acute toxicity of DPH. The presence of decerebrate posturing has not been described in this toxidrome. Microscopic hematuria has been described in only one case of DPH ingestion by Park-Matsumoto, J Neurol 1999; 162:108, along with a transient elevation in serum creatinine.

72. ANAPHYLAXIS DURING CROFABTM ADMINISTRATION

Meier KH,¹ Tsukaoka BT,¹ Dudyala V.² ¹California Poison Control System, SF Division, Department of Clinical Pharmacy, University of California, San Francisco, California, USA; ²Peninsula Hospital, Burlingame, California, USA.

<u>Background</u>: Crotalidae polyvalent immune Fab (ovine) (CrofabTM) (FabAV), has been available since October 2000. Only one case of anaphylaxis has been reported in the medical literature to date. We report a case of anaphylaxis after FabAV administration. <u>Case Report</u>: A 42 year old female was bitten on the right index finger by a rattlesnake, presumed to be Crotalus Viridis Oreganus. Initial assessment showed swelling to the wrist, laboratory values were WNL. The patient was admitted for observation and given supportive care. By the next morning, the distal part of the finger was blackened, swelling had progressed up the forearm and the patient had pain radiating to the axilla. Coagulation studies showed PLTs = 231, D-dimer = 456, INR = 1.1. Due to progressive symptoms, it was decided to administer crotalid antivenin. Previous skin testing had shown this patient to be allergic to sheep. Only FabAV was available from the pharmacy. The patient was premedicated with diphenhydramine 1 mg/kg and raniditine 75 mg IV. Two vials of FabAV were reconstituted in 500 mL NS and the infusion started at 50 mL/hr. Within 20 min, the patient developed tracheal swelling, facial flushing, and hypotension. FabAV was discontinued; solumedrol 100 mg IV and an epinephrine bolus were given. An epinephrine drip was required for 7 hours. The pharmacy was unable to locate equine polyvalent crotalidae antivenin, so it was decided to observe the patient and she was discharged on day 4. <u>Conclusion</u>: This is the second reported case of FabAV anaphylaxis.

73. SEVERE LACTIC ACIDOSIS: DO NOT FORGET PHENFORMIN

Criaco C, Bacis G, Farina ML. Bergamo Poison Control Center, Ospedali Riuniti, Bergamo, Italy.

Introduction: Although removed from the American market, phenformin is still used in several European States for the treatment of NID diabetes mellitus. Within only 1 year, in our hospital we diagnosed and treated three patients with severe lactic acidosis induced by phenformin. Case Reports: I case: A 72 year old woman, with pre-existing myocardial infarction and chronic renal failure, was admitted to our hospital with hypotension, hypoglycemia, severe lactic acidosis (pH 6.86, BE –29 mEq/L, lactic acid 29.2 mmol/L), and anuria (blood urea 152 mg/mL, serum creatinine 7.6 mg/mL). Hemodialysis was instituted with progressive reduction of lactate and after 3 hours she was hemodynamically stable and alert, but 5 days after the patient died for cardiogenic shock. II case: A 80 year old woman, with chronic renal failure, hypertension, Parkinson's, arrived in our hospital unconscious, hypotensive with severe metabolic acidosis (pH 7.03, BE -25.6 mEq/L) and high level of lactate (24 mmol/L), and mild renal insufficiency (serum creatinine 2.6 mg/dL; BUN 96 mg/L). Hemodialysis was immediately performed and she was hemodynamically stable and alert after few hours, but also this patient died of cardiogenic shock 7 days after. III case: A 78 year old woman, with duodenal ulcer, emphysema was admitted to our hospital with vomiting, excitement, hypotension, and coma. She developed severe lactic acidosis (pH 6.71, BE -31.8 mEq/L, lactic acid 24.6 mmol/L) and acute renal failure (blood urea 279 mg/dL, serum creatinine 12.3 mg/dL). Hemodialysis was performed and also thiamine was added to the standard therapy. She was discharged with good renal function 11 days after admission. Conclusion: Even though the patients were aggressively treated with hemodialysis, our case-series reaffirm the high mortality of phenformin-induced lactic acidosis.

74. INTRACTABLE PRIAPISM ASSOCIATED WITH HERBAL STIMULANTS

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<u>Background</u>: A wide variety of herbal supplements are available to the public over-the-counter without any restrictions and are often advertised as a safe "natural" alternative to prescription medications. <u>Case Report</u>: A 43 y/o white male without any past medical history presented to the ED complaining of approximately 60 hours of sustained erection. He had used an over-the-counter sexual enhancement drug with the trade name "All Night Long" which contain 16 ingredients including niacin and horny goat weed standardized extract. He took no prescription medications but was

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taking niacin and ginseng supplements. After taking the supplements, he achieved an erection, but the erection failed to subside and became associated with significant pain. In the ED he received morphine and terbutaline 0.25 mg sc without improvement. Fifty cubic centimeter of blood was aspirated from the cavernosa sinuses, 10 mg neosynephrine in 500 cc NS was irrigated into the cavernosa sinuses and two separate injections of 500 mcg phenylephrine were given but the patient remained without improvement. A Winter's shunt was performed on each side with partial improvement. He received three doses of 200 mg ketaconazole for hormonal ablation while in the hospital and was discharged the next day after complete resolution. <u>Conclusion</u>: Herbal supplements are provided over-the-counter without restriction and are thought by many people to be without risk. However they can be associated with significant negative effects and morbidity.

75. NEAR FATAL ACCIDENTAL TRANSDERMAL OVERDOSE OF COMPOUNDED KETAMINE, BACLOFEN, AMITRIPTYLINE, LIDOCAINE, AND KETOPROFEN: A CASE REPORT

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<u>Background</u>: Use of transdermal compounds containing analgesics, tricyclic antidepressants, and neuroinhibitors for the management of chronic pain is an emerging practice. Reports of overdose of these agents by the enteral and parenteral routes are abundant. Overdose by transdermal absorption has not previously been described. <u>Case Report</u>: A 35 year old male presented in an apparent post-ictal state after suffering a seizure. Over the ensuing 2 hours, his level of consciousness deteriorated to a GCS of 3, and he lost all brainstem reflexes. He required intubation and mechanical ventilation for 2¹/₂ days. CT scan of the brain was normal. EEG demonstrated a burst suppression pattern, previously reported in baclofen overdose. Urine drug screen for drugs of abuse revealed the presence of benzodiazepenes and tricyclic antidepressants. Routine cerebrospinal fluid analyses were normal. Later, it was revealed that the patient had applied an excessive amount of a compounded cream prescribed for relief of chronic pain. He received a total dose of ketamine 900 mg, baclofen 900 mg, amitriptyline 360 mg, lidocaine 900 mg, ketoprofen 1800 mg. Ketamine was detected in the CSF by gas chromatography and mass spectrometry. He was discharged neurologically intact after 4 days. <u>Conclusion</u>: Transdermal absorption of ketamine, baclofen, amitriptyline, lidocaine, and ketoprofen resulted in a prolonged, profound coma.

76. COMPLETION OF WHOLE BOWEL IRRIGATION IN PCC OVERDOSE PATIENTS

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<u>Background</u>: Whole bowel irrigation (WBI) for toxicologic patients has a number of specific indications. The utility of this gastric decontamination method is time and resource consuming. The objective of this study is to assess the ability to complete the WBI process when it is recommended by a poison control center for a toxicologic indication. <u>Methods</u>: This was a prospective, IRB approved, observational study. The inclusion criteria were overdose PCC pts that met an AACT WBI position paper indication for the use of WBI. Upon study enrollment, WBI instructions were explained by phone and faxed (each case offered this) to health care providers. Data was collected at enrollment and 4 hour intervals. Data collected included pt demographics, overdose information and WBI information. The main study endpoint was the successful completion of WBI as defined by a clear rectal effluent. <u>Results</u>: 22 patients were enrolled. 73% were male and mean age of 28.8 years. Whole bowel irrigation indications were: substance poorly adsorbed by charcoal—41%; enteric-coated or sustained-release drug—32%; foreign body—27%; and a massive overdose—0%. 95% of cases involved intentional ingestions. Of the 22 pts enrolled, 5 (23%) completed the WBI. The mean time to clear rectal effluent was 34.2 hours. Only one case completed WBI in less than 15 hours. <u>Conclusion</u>: Whole bowel irrigation is an uncommonly used and labor intensive procedure recommended in specific toxicologic patients. When recommended for use by a PCC, the majority of patients do not complete therapy adequately. Further studies need to address the reasons for these findings.

77. INTRACEREBRAL HEMORRHAGE ASSOCIATED WITH INGESTION OF TOBACCO SNUFF

Dahl B, Caravati EM. Utah Poison Control Center, University of Utah, Salt Lake City, Utah, USA.

<u>Background</u>: Ingestion of tobacco commonly causes illness but rarely life-threatening toxicity. We report a case of intracerebral hemorrhage associated with the intentional ingestion of snuff, a preparation of pulverized tobacco. <u>Case Report</u>: A 28-year-old woman with a prior history of hypertension and pseudotumor cerebri engaged in a substance eating contest and ingested the entire contents of 1 can (36 g) of SkoalTM smokeless tobacco. She vomited 15–30 min later and then became somnolent and combative. Upon arrival at the hospital, her BP was 200/110 mmHg. She had a left facial droop and left hemiparesis. A CT scan showed right putamenal hemorrhage with extension into the frontal lobe, deep basal ganglia, and right lateral ventricle. Her hypertension was initially controlled with nitroprusside and metoprolol, followed by hydralazine, diuretics, and ACE inhibitors. A MRA revealed right middle cerebral artery hemorrhage without evidence of aneurysm or AVM. She was monitored in the ICU for 5 days, and then admitted to a rehabilitation unit for 17 days. Upon discharge she was normotensive (BP 98/57 mmHg). She had a slight left facial droop and light touch in the left upper extremity. Finger-nose testing was impaired on the left. <u>Discussion</u>: This patient had rapid onset of severe hypertension, altered mental status, and intracerebral hemorrhage after ingesting a large amount (36 g) of snuff tobacco. The total nicotine dose was about 500 mg. Vomiting probably reduced nicotine bioavailability and serum nicotine levels were not obtained. <u>Conclusion</u>: Ingestion of large amounts of tobacco. She vere hypertension and intraceranial hemorrhage.

78. THE SERIOUS SIDE OF OLANZAPINE OVERDOSE

Rottinghaus D, Bryant S, Harchelroad F. Medical Toxicology Treatment Center, Allegheny General Hospital, Pittsburgh, Pennsylvania, USA.

<u>Background or Objective</u>: To determine the safety profile of frequently used anti-psychotic medication in overdosed patients. <u>Methods</u>: A three year retrospective chart review of intentional anti-psychotic drug overdose was performed at a Regional Medical Toxicology Treatment Center. Included were patients greater than 12 years of age who overdosed on either haloperidol (H), risperidone (R), or olanzapine (O). Excluded were patients who may have attempted suicide in any manner other than oral drug ingestion. <u>Results</u>: Twenty-four patients were identified. Two patients (8%) poisoned themselves with (H); one ingested only (H); the other was a poly-pharmacy ingestion. Four patients (17%) overdosed with (R); three ingested only (R). Eighteen patients (75%) overdosed with (O); 14 ingested only (O); the other 4 patients ingested a mean of four other drugs in their suicide attempt. Of the 14 who ingested only (O), 12 required endotracheal intubation and mechanical ventilation for greater than 24 hours. <u>Conclusions</u>: A preponderance of patients cared for at this Regional Medical Toxicology Treatment Center who had poisoned themselves with an anti-psychotic medication did so by overdosing on olanzapine. Serious adverse effects were noted in those patients overdosing only on olanzapine.

79. ACCIDENTAL INTRAVENOUS BARIUM SULFATE INFUSION

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<u>Background</u>: Barium sulfate is an unabsorbable radioopaque compound used for imaging of the gastrointestinal (GI) tract. Accidental intravasation of barium sulfate into the venous circulation is a rare adverse event during GI radiological studies requiring prompt recognition and resuscitation. We report a case of acute barium toxicity after an episode of accidental intravenous (IV) infusion of oral barium sulfate. <u>Case Report</u>: A 57 year-old female with metastatic gastric adenocarcinoma was about to undergo a computed tomography scan of the abdomen. Approximately 50–75 mL of oral barium sulfate contrast were accidentally administered IV through a central venous catheter located in the right subclavian vein. Immediately, the patient developed respiratory distress and hypoxemia, requiring endotracheal intubation and mechanical ventilation. After extubation the next day, shortness of breath and hypoxemia continued requiring a non-rebreather mask and non-invasive ventilation. The patient vomited and aspirated requiring re-intubation

and antibiotics. Because of the patient's terminal condition and deteriorating respiratory status, life support was withdrawn 21 days later. Autopsy revealed bilateral pulmonary edema and pleural effusions. There was no evidence of gross pulmonary emboli or microthromboemboli on pathologic examination. <u>Conclusions</u>: Barium sulfate is generally not considered to cause acute barium toxicity. Rapid onset of hypotension, respiratory distress, and death are reported consequences of acute barium toxicity if venous intravasation occurs. This is the first case to describe acute barium toxicity due to barium sulfate after an accidental IV infusion.

80. SURVIVAL AFTER ETHYLENE GLYCOL OVERDOSE WITH BLOOD pH OF 6.54

Rottinghaus D, Bryant S, Harchelroad FP. Medical Toxicology Center, Allegheny General Hospital, Pittsburgh, Pennsylvania, USA.

<u>Background</u>: Survival from any illness with a blood pH of less than 6.8 is rare. We describe a case of a patient surviving with full recovery after ingestion of ethylene glycol and initial blood pH of 6.54. <u>Case Report</u>: A 47 year-old female presented with coma. Her family found her unresponsive on the garage floor 8 hours after suspected consumption of a toxic alcohol. She was intubated at the scene and presented with an initial arterial pH of 6.54, PaCO₂ of 8. Osmolal gap was calculated to be 109 mos/kg. Core temperature was 29.1°C. Treatment included: sodium bicarbonate, fluid resuscitation, fomepizole, emergent hemodialysis, rewarming, and general critical care management. Normal core temperature and blood pH were achieved. The following day the patient was extubated and had no neurologic deficits. Blood creatinine peaked at 1.3 mg/dL. Her blood ethylene glycol level returned at 5895 mcg/mL, with methanol and ethanol concentrations of 0. After 6 hours of hemodialysis, the patient's osmolal gap remained normal. She was discharged to the psychiatry service on hospital day 2, at which time she admitted to consuming several swallows of antifreeze in a suicide attempt. <u>Conclusion</u>: Focused Aggressive Toxicologic Management (FATMAN) resulted in an excellent outcome in this critically ill overdose patient.

81. LACK OF TOXIC EFFECTS FOLLOWING ACUTE OVERDOSE OF MYCOPHENOLATE

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<u>Background</u>: Mycophenolate is an immunosuppressive used for solid organ transplant and lupus nephritis. While chronic toxicity including bone marrow suppression, hepatic injury, and renal toxicity have been reported, the course of acute overdose has not been described. <u>Case Report</u>: Twenty four year-old female with a history of lupus, complicated by lupus nephritis, overdosed acutely on 10 g of mycophenolate. She arrived 4 hours after ingestion and complained of mild abdominal pain. She received charcoal and intravenous fluids in the emergency department and was admitted to the hospital. The patient remained normotensive throughout her course. She did not develop and leukopenia, thrombocytopenia, anemia, vomiting, peripheral edema, elevated transaminases, or worsened renal function. Her mycophenolate levels were 44.1 mcg/mL (reference range 1–10 mcg/mL) at 5 hours post-ingestion, 5.6 mcg/mL at 10 hours, and 0.3 at 20 hours. She was followed up 3 weeks and 2 months post ingestion and did not develop any renal or hepatic dysfunction or bone marrow suppression. <u>Conclusion</u>: This report describes the first case of an acute overdose of mycophenolate. In an overdose of 5 times the daily dose in a patient with lupus nephritis, no significant adverse effects developed over a 2-month follow-up.

82. LACK OF TOXICITY OR SIGNIFICANTLY ELEVATED FORMATE LEVELS FOLLOWING INHALATIONAL ABUSE OF METHANOL CONTAINING SOLVENTS

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Background: Inhalational abuse of solvents containing methanol (MeOH) is common. The potential for systemic absorption of MeOH and conversion to formic acid, the toxic metabolite, has been recognized. It is not clear if this type

of poisoning will cause significant MeOH toxicity. We report a series of subjects, including MeOH and formate levels, who did not develop MeOH toxicity from inhalant abuse. <u>Case Series</u>: Five patients that presented to the ED following deliberate solvent inhalation were evaluated for elevated methanol levels. All were abusing the solvent for 6–12 hours prior to arrival and were chronic abusers of this product. The initial mean MeOH level was 19.3 mg/dL (range 10–26). The initial serum bicarbonate ranged from 13 to 20 mmol/L and the initial anion gap ranged from 13 to 31. The mean serum formate level was 31.7 mcg/mL (range 3.6–64) with a reference range of 0–12. All had an undetectable ethanol level. No patient had visual complaints and all had normal visual acuity. No patients were treated with an alcohol dehydrogenase inhibitor or dialysis, though all received intravenous folate. All had improved acidosis within 4 hours of arrival to the ED. <u>Conclusion</u>: A transient acidosis was the only toxic effect observed in this cohort. Notably, visual symptoms or findings were absent. No patient received treatment with an alcohol dehydrogenase inhibitor. Formate levels were lower than those associated with toxicity following oral ingestions. This preliminary data suggests that inhalant abusers using methanol containing products are at low risk to develop significant methanol toxicity.

83. COMPARISON OF PEDIATRIC USE OF IV AND ORAL N-ACETYLCYSTEINE

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Background: Use of intravenous N-acetylcysteine (IV NAC) occurs in a minority of acute acetaminophen (APAP) poisonings treated in the US. There is little published US pediatric safety data regarding IV NAC. Objectives: To compare the incidence of adverse reactions, length of hospitalization, and total hospital charges associated with PO-NAC vs. IV NAC in children treated for acute APAP poisoning. Methods: We retrospectively reviewed charts of patients admitted to a tertiary care children's hospital with APAP poisoning (ICD Code 965.4). We recorded the route of NAC administration, documented adverse reactions, subsequent treatments to ameliorate adverse reactions, length of stay (LOS), cost of hospitalization, and outcome. Results: We identified 89 cases of APAP poisoning resulting from single acute ingestions. Of these, 72 received PO-NAC and 17 patients received IV NAC, including 2 who changed from PO to IV NAC. Forty PO NAC patients (56%) had adverse events including vomiting in 29 patients (42%), nausea alone in 10 patients (14%), and abdominal pain in 2 patients (3%). Thirty-five PO NAC patients (49%) received anti-emetics, 2 received NG tubes for repeated PO NAC, and 2 patients were switched to IV NAC without further adverse reactions. Six IV NAC patients (35%) had adverse events including 2 each (12%) with nausea alone, vomiting, and rash. Four IV NAC patients received anti-emetics (24%), and 2 received diphenhydramine (12%). The mean LOS for patients treated with IV NAC was 1.9 days compared to 3.2 days with PO NAC (p = 0.0003) with similar total hospital charges (PO \$5261.54, IV \$5634.63). All patients recovered. Conclusion: The use of IV NAC was associated shorter hospitalizations with a trend toward fewer side effects, decreased use of antiemetics, but similar total hospital charges.

84. INADVERTENT INJECTION OF BOTULINUM TOXIN A (BOTOXTM) INTO THE INFRAORBITAL AND MENTAL NERVES RESULTING IN DYSPHAGIA

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<u>Background</u>: Botulinum toxin type-A (BTX-A) is used to treat a variety of disorders involving abnormal muscle contractions and for cosmetic purposes. The most common adverse effects involve excessive weakness of the treated muscle or the local diffusion of BTX-A into adjacent muscles causing unwanted weakness. Reported is a case of inadvertent injection of BTX-A into the infraorbital and mental nerves resulting in transient unilateral facial droop, dysarthria, and dysphagia. <u>Case Report</u>: A 39 year old female was seen for cosmetic collagen treatment of the nasolabial folds and BTX-A injections into the forehead for treatment of her migraine headaches. In preparation for collagen, injections of local anesthetic were made into the infraorbital and mental nerve bundles. Immediately after the injections it was discovered that BTX-A 7.5 U/injection was injected instead of the intended lidocaine. The patient also received injections of BTX-A into her forehead. She was referred to the Emergency Department at a local hospital. After discussion with the poison center and state health officials, botulinum antitoxin was not administered. Over the next several days she developed dysphagia with uvula deviation, and weakness of the lips and face resulting in a noticeable



unilateral facial droop. The patient had 90% improvement of her symptoms within 1 month. <u>Conclusion</u>: Inadvertent injection of BTX-A into the infraorbital and mental foraminae resulted in transient facial muscle weakness and dysphagia. Whether botulinum antitoxin would have been effective in preventing her symptoms is unknown.

85. DELAYED ONSET OF SEIZURES FOLLOWING WELLBUTRIN SR[®] OVERDOSE

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<u>Background</u>: Bupropion is an atypical antidepressant used in the treatment of depression, smoking cessation, and attention deficit hyperactivity disorders. Early and recurrent seizures have been reported following overdose with both the immediate and sustained release (SR) forms. The SR form has been available in the United States since 1996 and in Canada since 1998. Although the time to peak plasma concentration for Wellbutrin SR[®] is documented as 3 hours (vs. 2 hours for immediate release), areas under the curve are similar. We present four patients with delayed seizures (sz) following Wellbutrin SR[®] overdose; two were discharged prematurely after a period of observation following their first sz. <u>Case Series</u>:

Age/sex	Dose (gm)	1st sz (hrs)	Other agents/features	Last sz (hrs)	SZ	Bupropion (mcg/L)
23 y/f	9	12–18	None	~24	3	148 initial; 120 36h out
20 y/f	2	5	Ambien/ritalin; sz d/o	13	2	170
37 y/f	23	~38	EtOH; ED/ICU sedation/paralysis	~42	Status	—
36 y/f	$\sim \! 40$	8	Etoh/cocaine	13	2	—

<u>Conclusion</u>: The average time from ingestion to first seizure following overdose of the SR form of bupropion was 17 hours with the final seizure occurring within another 8–12 hours. This may represent prolonged absorption secondary to tablet coating and/or bezoar formation or the therapeutic effect of sedatives, particularly in the patient with the most prolonged sz onset. The usual practice of a single dose of activated charcoal and several hours of observation is not adequate treatment for these patients. The early use of whole bowel irrigation and a prolonged period of observation should be considered in patients with large ingestions of sustained release bupropion.

86. CITALOPRAM OVERDOSE: LATE PRESENTATION OF TORSADES DE POINTES (TDP) WITH CARDIAC ARREST

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<u>Background</u>: Citalopram in overdose can cause QT_c prolongation that predisposes to torsades de pointes (TDP). Cardiac conduction delays are expected within 24 hours of exposure and may be due to the metabolite didesmethylcitalopram. We report a first case of delayed citalopram induced TDP with documented serum levels and recorded ECGs. <u>Case Report</u>: A 36 year old woman with a history of multiple suicide attempts, depression, alcoholism, anorexia, and psoriasis, presented 36 hours after ingesting 1000 mg of citalopram with 2 quarts of wine. Ingestion occurred in the PM on day 1. During day 2, she felt nauseated, weak, and lethargic. She visited her parents in the afternoon, but returned home early and went to the bed. On the morning of day 3, she had palpitations and numbness in both the arms that prompted her visit to the ED. Vital signs were: BP, 84/44 mmHg; T, 99.3°F; P, 102–160/min; RR, 17/min; O₂, Sat 99% RA. An ECG had intermittent runs of wide complex tachycardia with a QT_c of 600 msc. Her Mg²⁺ level was 2.5 mg/dL. The patient was started on O₂, IV NaCl and was given MgSO₄ (2 g IV). She developed bigeminy, and despite IV lidocaine intermittent TDP occurred. An isoproterenol infusion was started and she converted to sinus rhythm. Additionally, she received KCl, K₃PO₄, and a transvenous pacemaker. 24 hours later her ECG showed normal sinus rhythm with a QT_c of 529 msec. The QT_c interval narrowed to 442 msec at 48 hours. A citalopram level was 477 ng/mL (therapeutic: 40–110 ng/mL) and desmethylcitalopram was 123.2 ng/mL (therapeutic: 14–40 ng/mL). <u>Conclusion</u>: Clinicians should be aware of the possibility for delayed and prolonged cardiac toxicity of citalopram.
87. HYPERGLYCEMIA IN A PEDIATRIC CARBAMAZEPINE OVERDOSE

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<u>Background</u>: Hyperglycemia has been reported in carbamazepine poisoned rats, but according to a medline search has never been reported in humans. This is the first case report to our knowledge, of a non-diabetic patient with hyperglycemia secondary to a severe carbamazepine (CBZ) overdose. <u>Case Report</u>: A 4 year-old male with a history of attention deficit hyperactivity disorder was found to be lethargic by his grandmother. He had been normal 2 hours previously. His medications included dextroamphetamine, mirtazapine, and an over-the-counter cold preparation that contained pseudophedrine and antihistamines. His physical examination was significant for pale skin, coma (he was unresponsive to the IV insertion), and flaccidity. His laboratory testing showed hypokalemia (2.7 mEq/L) and hyperglycemia (314 mg/dL), and a normal sodium of 139 mg/L. The patient was transferred to a comprehensive pediatric center; upon arrival at the medical center the child was intubated. Further history revealed the patient's brother was on CBZ, and a serum CBZ level was sent and revealed a level of 60 mcg/mL. The child's mental status returned to baseline 24 hours later; he received supportive care only. The glucose level also returned to normal during the convalescent phase. It was later discovered the grandmother intentionally administered the CBZ to the child to make it sleep. <u>Conclusion</u>: A previous animal study supports the possibility of CBZ causing hyperglycemia. This may be the first reported case of hyperglycemia secondary to severe carbamazepine overdose in a human, although the mechanism is unknown.

88. EFFECTIVENESS OF OBIDOXIME IN ORGANOPHOSPHATE POISONING

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<u>Background</u>: The assessment of clinical effectiveness of oximes in organophosphate (OP) poisoning is hampered by problems to measure therapeutic effects of oximes in unconcious, artificially ventilated and atropinized patients. <u>Methods</u>: In clinical trial, OP-poisoned patients needing artificial ventilation received atropine as required and obidoxime as early as possible, 250 mg bolus, followed by 750 mg/24 hours as long as reactivability of inhibited acetylcholinesterase (AChE) was anticipated. Cholinesterase status (AChE-activity of red blood cells (RBC), reactivatability of patient's RBC-AChE, plasma cholinesterase activity) plasma level of obidoxime and neuromuscular function were measured ro assess oxime efficacy. <u>Results</u>: In patients requiring artificial ventilation, RBC-AChE activity was usually inhibited by more than 90% of normal. At the above obidoxime treatment regimen a plasma concentration of about 10–20 µmol/L was measured and sufficient reactivation of non aged RBC-AChE was obtained, if the poison load was not too high. Reactivation of RBC-AChE to more than 30% of normal resulted in normalisation of neuromuscular function indicating therapeutic effectiveness. <u>Conclusions</u>: Therapeutic benefit of oximes may be anticipated when effective doses are adminstered early and long enough. Excessive poison load and rapid ageing are the main obstacles limiting net reactivation.

89. USE PATTERNS FOR A UNIVERSITY HOSPITAL-BASED MEDICAL TOXICOLOGY SERVICE

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<u>Background</u>: Clinical medical toxicology services are relatively new and uncommon at most hospitals, even university medical centers. A study was performed to determine the main reasons for using a university hospital-based medical toxicology consult service. The medical toxicology service has been functioning for the past six years. <u>Methods</u>: Records were queried from a database for medical toxicology consults between 2000 and 2003. The main information obtained from each record was the primary reason for each consult and the site of the consult. Reasons were categorized into drug classes.



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<u>Results</u>: Over this three-year period there were 342 consultations. Eighty-seven percent of the consultations originated from the adult emergency department. The other 13% were evenly distributed from the pediatric emergency department, adult and pediatric ICUs, and hospital inpatient wards. The main agents associated with consultations were: acetaminophen and aspirin—19%, psychiatric medications—17%, snakebites—17%, requests to evaluate medical conditions for possible drug etiologies—9%, toxic alcohols and acetone—5%, antiepileptic medications—4%, acid and base agents—4%, illicit stimulants—3%, cardiovascular agents—3%, metals—2%, carbon monoxide—1%, and other agents—13%. <u>Conclusions</u>: The most common reasons for using the consult service involved drugs or envenomations that can potentially be treated with antidotes and psychiatric medications, primarily antidepressants and antipsychotics. These patterns, particularly the snakebites may be unique to this location, which is a rural southern state.

90. CRITICALLY ILL NIACIN OVERDOSE

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<u>Background</u>: We present a case of massive niacin overdose taken as a therapeutic misadventure. This case is the highest reported dose, and the only niacin overdose requiring ICU management. <u>Case Report</u>: The patient was a 56 yo male who presented to the ED with burning in his abdomen and chest after ingesting niacin the night prior. Twenty hours prior to admission (PTA), he reported taking 5–6 tabs of 500 mg niacin with no adverse effect. Again 11 hours PTA, he consumed 16,500 mg niacin. He awoke 2 hours PTA with pain. The patient noted an Internet resource that recommended high dose niacin for schizophrenia as the reason for his ingestion. Past history: schizophrenia, and remote B12 deficiency. Current medications: B12, zinc 120 mg, and B6 600–700 mg per day. He stopped his psychiatric medications the several weeks PTA. He denied illicit drug or ETOH use. Upon arrival the patient was alert; temperature of 36.0 rectal; BP 92/41, HR 68, RR 16, SA0² 98% on RA. His BP dropped to 58/40. The rest of the physical exam was unremarkable, with no signs of allergic reaction. After NS bolus his SBP was 62–96 mmHg. He required dopamine for 12 hours. Extensive evaluation for other etiologies of hypotension was negative. Serum niacin levels were 8.2 µg/mL and 5.6 µg/mL at 48 and 96 hours post-ingestion respectively, giving an apparent T¹/₂ of 87 hours. <u>Conclusion</u>: Massive overdose of niacin appears to be capable of causing severe, persistent hypotension. In this case the ingestion of a dietary supplement based on Internet information led to a severe adverse reaction.

91. FACTITIOUS DIGOXIN TOXICITY FROM INTERFERENCE WITH AN AUTOMATED IMMUNOASSAY

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<u>Background</u>: Common immunosuppressive and anticancer therapies may lead to unexpected laboratory interference. We report a case where rapid recognition of lab inconsistencies prevented digoxin Fab administration. <u>Case Report</u>: A 69 y/o female with metastatic breast cancer present with complaints of fatigue, nausea, and SOB. Medications were digoxin, coumadin, and cancer chemotherapy including OKT3 bispecific antibodies and trastuzumab. Interleukin-2 was recently added for salvage therapy. A digoxin level 8 hours post-dose was reported as a panic value of 16 ng/mL which was 15.2 ng/mL on repeat. ECG showed atrial fibrillation at 120 bpm. Electrolytes did not show hyperkalemia. The patient appeared comfortable. Since her presentation was inconsistent with the lab value, a "free digoxin" level of <0.05 ng/mL was obtained on the previous specimen by centrifugal filtering. She had no known indications for digoxin-like immunoreactive substances. A literature search suggested monoclonal murine antibodies might interfere with digoxin immunoassays. The assay's manufacturer was unable to provide information related to digoxin elevation with monoclonal murine antibodies. <u>Conclusion</u>: An elevated digoxin level by immunoassay may occur in patients receiving immunosuppressants or anticancer therapies because of antibody interference. Lab values inconsistent the patient presentation should prompt further investigation.

92. NASAL BUTTON BATTERY IMPACTION AS CAUSE OF PERIORBITAL CELLULITIS AND CORROSSIVE TISSUE INJURY

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<u>Background</u>: Button batteries as nasal foreign bodies have caused nasal perforation, saddle deformity, and nasal turbinate destruction. Tissue injury can occur from leakage of potassium hydroxide, external current, or pressure necrosis. Periorbital cellulitis is an uncommon result of nasal foreign bodies and has not been reported with button batteries. <u>Case Report</u>: Four-year old male presented to ED with fever, rhinorrhea, and nausea. The treating physician inquired about trauma because of the presence of blood in his right nare. The parents were unaware of any trauma. He was treated symptomatically for presumed "viral sinusitis." Two days later he returned to the ED with headaches, fever (101.6°F), and a swollen, red right eye (periorbital, malar, and nasolabial fold swelling). White blood count was 17,500. Periorbital cellulitis was suspected leading to a head CT with facial cuts that revealed a metallic right nasal foreign body in addition to preseptal inflammatory changes. A 1.4 cm button battery with "obvious surface corrosion" was removed in the OR. The surgeon noted extensive liquefaction necrosis of the floor of the entire right nasal cavity that extended from the septal wall to the middle turbinate. A small perforation was noted in the septum. The left nasal septum also showed tissue necrosis. The battery was identified as Vinnic L1115 containing manganese oxide and potassium hydroxide. After foreign body removal and intravenous antibiotics, his periorbital cellulitis resolved. His tissue injury required repeat debridement and packing to avoid complications. <u>Conclusion</u>: An impacted, leaking button battery nasal foreign body can cause corrosive tissue injury and be misdiagnosed as periorbital cellulitis.

93. AMANTADINE TOXICITY IN A RENAL TRANSPLANT PATIENT

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Background: Amantadine is used as an antiviral agent, for treatment of Parkinson's disease, and as a stimulant after brain injury. Impaired renal function increases the risk of amantadine toxicity since 90% is excreted unchanged in urine. With amantadine levels >1 mg/L, confusion, hallucinations, and other CNS toxicity develops. Case Report: A 27-year-old woman with IDDM, ESRD status post transplant, and anoxic brain injury (due to hypoglycemic event) was treated with amantadine for psychostimulation. Her medications included TMP-SMZ, furosemide, bupropion, metoclopramide, acyclovir and many others. Nearly 1 year after transplant, she developed ataxia, hallucinations, and agitation consistent with amantadine toxicity. Her amantadine dose had been increased to 400 mg/d 5 days prior. She also had developed acute renal failure (Cr 3.3 mg/dL) of uncertain etiology (biopsy without acute cellular rejection). Benzodiazepines controlled her symptoms. Amantadine, TMP-SMZ, acyclovir, bupropion, and metoclopramide were stopped. Amantadine levels were 1.9 mg/L (day 2) and 1.3 mg/L (23.5 h later); calculated half-life was 42.9 h (avg 12 h in healthy adults). Symptoms resolved by hospital day 5 (estimated amantadine level 0.65 mg/L). Bupropion, metoclopramide, and TMP-SMZ were restarted without adverse effect. Conclusions: Care of renal transplant patients requires many drugs that potentially interact; renal function in such patients often fluctuates. This is the first reported case of amantadine toxicity in a renal transplant patient. Many factors likely contributed, including the high dose and acute renal failure. Urinary retention (from anticholinergic effects of amantadine) resulting in renal dysfunction may have contributed. In addition, TMP and amantadine both undergo tubular secretion; their combined use may have altered amantadine clearance. Amantadine dose adjustment to account for all these factors is essential to avoid toxicity.

94. INAPPROPRIATE LAUGHTER: AN ISONIAZID ADVERSE DRUG EVENT

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Introduction: Certain drugs induce mood-disorders as an undesired side effect of therapy. Although isoniazid therapy has been previously associated with mania, obsessive-compulsive neurosis, and psychosis we report the first case of

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isoniazid-associated inappropriate laughter. <u>Case Report</u>: A 21-year-old female with no past medical or psychiatric history presented to the ED with inappropriate and uncontrollable laughter. Her roommate brought her to the ED because she had been acting "bizarre" over the past few weeks. Her only medications included daily isoniazid (300 mg) and pyridoxine (50 mg) given for a positive tuberculin purified-protein derivative test, which were started approximately 1 month earlier. Her vital signs were: blood pressure, 136/66 mmHg; pulse, 78 beats/minute; respirations, 16 breaths/ minute, and temperature, 97.6°F. She was noted to have inappropriate, uncontrollable laughter and an elated mood. Her physical and neurological exams were otherwise normal. No flight of ideas or pressured speech was noted. She was admitted to the hospital and the isoniazid was discontinued. She was placed on oral lorazepam three times a day (0.5 mg) along with a one-time dose of oral pyridoxine (5 g). Routine serum laboratories and a computerized tomography scan of her head were normal. Over the next 12 hours her symptoms rapidly improved and she was discharged the next day. She was seen the following week in the behavioral, psychiatric, and toxicology clinics and continues to do well. <u>Conclusion</u>: Drug-induced mood disorders may occur with certain pharmacological agents. Isoniazid administration may be associated with inappropriate and uncontrollable laughter.

95. CONFIRMED ISOLATED BUPROPION OD PRODUCING QRS WIDENING

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<u>Background</u>: Few reports describe long QRS after bupropion (BUP) OD, but [BUP]s have not always been measured, and comprehensive tox testing did not exclude other agents. We report a patient with serial plasma [BUP]s and ECGs, and comprehensive screening ruling out other drugs. <u>Case Report</u>: A 30 y.o. man chronically on bupropion ingested approximately 30 g BUP SR about 3 hr before arrival. He received charcoal. Findings were agitation, choreoathetosis, and tachycardia. Urinary incontinence suggested a prior seizure. EMIT, GC-MS, and TLC urine screens were positive only for bupropion. He recovered completely over 4 days with supportive care. QT_c prolongation mainly reflected QRS widening.

Hrs post ingestion	Plasma [BUP] µg/L	Heart rate	QRS (ms)	PR (ms)	QT _c (ms)
4		128	100	138	470
8.75	987	140	120	122	468
13	821	114	120	162	493
19.2	604				
25	555	114	120	114	493
33.3		119	112	160	472
39.5	353	113	102	172	455
45	373	100	106	150	479
61.7	120	120	100	160	480
71	64	75	98	138	402
76.8		75	96	132	410

Conclusion: Confirmed isolated toxic bupropion ingestions can produce QRS widening.

96. PILL IDENTIFICATION: RETHINKING THE QPOISON CENTER'S ROLE

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<u>Background</u>: In anticipation of the HIPPA privacy regulations and in rethinking the scope of practice for poison control centers (PCCs), our PCC instituted a policy in February 2002 for pill identification (PI). No formal policy existed and SPIs provided PIs unless a drug abuse situation was suspected. Methods: This policy allows for routine

PIs for law enforcement and healthcare professionals only. Medications for the public are not routinely identified to maintain the privacy of patients (adult and adolescent) as well as to encourage potential drug abusers to seek medical evaluation. If parents of an adolescent wants their child's pills identified, a referral to ER or PMD is made. The PI would then be received through a physician and appropriate treatment and/or counseling can be done. If a toddler possibly ingested unknown pills, PI is made at the discretion of the specialist with a medical referral if needed. Results: After this policy was in effect for 1-year, the percentage of PI calls went from 4.76% (January 01–January 02) to 5.12% (February 02–February 03), the percentage of PI calls from the public on drugs known to be abused went from 16.4% (January 01–January 02) to 14.6% (February 02–February 03), and the percentage of PI calls from the public (vs. those from law enforcement and health care professionals) went from 45.7% (January 01–January 02) to 33.5% (February 02–February 03). Conclusion: The new policy did not eliminate PIs from our scope of practice. It allowed our PCC to focus on providing this service to those with a legitimate need and to encourage potential drug abusers to seek medical evaluation.

97. IMPACT OF BIOLOGICAL/CHEMICAL TERRORISM ON POISON CENTERS

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Background: The potential impact on poison control center (PCC) services in the event of a domestic biological/ chemical terrorism (BCT) attack is difficult to assess. During and immediately following the anthrax attacks in the eastern United States in 2001, a group of western PCCs received a large number of calls regarding BCT. This report demonstrates how PCCs are considered a primary resource by the public with regards to BCT. Methods: A retrospective review of all calls received by four PCCs in the western United States from the period of 9/11/01 through 12/31/01 was conducted. Search criteria for cases included: AAPCC designated BCT-related generic substance codes, free text substance fields containing "unknown powder," "powder unk," "anthrax," "white powder," and "unk powder." Results: The total number of BCT-related calls reported during this time was 905, with 272 coded as exposures and 633 coded as information calls. From 9/11/01 until 9/30/01, only 19 BCT-related calls were received, with 3 coded as exposures. However, from 10/1/01 until 10/31/01 when the anthrax attacks were occurring, 585 BCT-related calls were received, with 157 cases coded as exposures and 428 information calls. From 11/01/01 until 11/30/01, 249 BCT-related calls were received, with 93 cases coded as exposures and 156 information calls. December data showed 19 cases coded as exposures and 33 information calls. Conclusion: These totals reflect that the public turns to PCCs for assistance during perceived BCT-related crises. Perceived expertise in the field of BCT coupled with a 24 hour, toll-free phone number makes PCCs an attractive primary resource for information. Had the attacks been of a chemical nature or had they occurred within our region, the totals would have likely been substantially higher.

98. COST-EFFECTIVENESS OF PROMOTION BY POISON CENTERS OF ACTIVATED CHARCOAL IN THE HOME

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<u>Objective</u>: Currently there is little consensus on what position Poison Centers should take on promoting or discouraging the use of activated charcoal in the home. We conducted a cost-effectiveness analysis comparing promotion vs. non-promotion by poison centers of the use of home activated charcoal. <u>Method</u>: We use a decision-analytic technique and societal approach. We use poison center survey data, census data, data from our own surveys, and literature data for probabilities. We used HCUP KIDS data base, CPT codes, AWPs, and literature sources for costs. The main outcome is cost per time at risk avoided. <u>Results</u>: The no/low charcoal promotion policy dominated the medium/high promotion policy and is the preferred option at our baseline values. The med/high promotion policy

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resulted in slightly higher per person minutes at risk (0.5) than the low/no policy, and also had higher costs per person (\$18.50). The model is most sensitive to the number who call the poison centers, and the number who receive the charcoal. As the number who call rises, high promotion becomes the more cost-effective option. Other factors, such as number who aspirate, or costs of spills of charcoal are only moderately sensitive factors. <u>Conclusion</u>: These results lead to a recommendation for non-promotion of home use of activated charcoal. Increasing the number of poison center callers is the most important factor in decreasing risk time, and if promoted along with charcoal, then promotion could be the more cost-effective option. More research is needed on adverse events of home use of activated charcoal.

99. POLICIES AND EXPERIENCE OF US POISON CENTERS WITH HOME ADMINISTRATION OF SINGLE-DOSE ACTIVATED CHARCOAL

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Objective: There is currently no consensus on the best position for poison control centers to take on home use of activated charcoal. The current outcomes and safety literature on home charcoal use may be inadequate to lead to definite policies. Our objective was to survey US Poison Center Directors on their policies and their center's experience with the use and appropriateness of home administration of single-dose activated charcoal for unintentional, ingestions, in children, less than 6 years old. Methods: A semi-structured telephone survey was designed, pre-tested, and conducted with 58 of the 61 poison center directors. The survey focused on: reasons for home use of activated charcoal, factors influencing home use, recommendations and policies for home use of charcoal, and adverse events following charcoal use in the home. Directors were also asked to estimate numbers of unintentional ingestions, in children <6 years, and cases for which activated charcoal was recommended at home or at other sites. Results: Main reasons for home use of activated charcoal were: time saved (31%), clinical effectiveness (24%), and cost (14%). Forty percent of directors would not recommend home activated charcoal, while 40% would, and the remaining were undecided. Only 19% of poison centers currently recommend households have activated charcoal; and mainly only through their public information materials. The others either do not actively recommend, have not decided, or have no policy on or currently do not recommend charcoal use in the home. This is in contrast to syrup of ipecac where 66% currently recommend its home use. Most (60%) poison centers had never managed a child at home with charcoal and all (34%) of those who had, felt that no adverse events followed. The majority of centers (41%) who do not recommend charcoal, felt it would be difficult to do so given the lack of charcoal in pharmacies and need for large education programs to implement such a program. Others did not recommend charcoal because of aspiration risks, absence of an official policy, or lack of data to recommend. Conclusion: Poison centers are split on whether they would recommend activated charcoal in the home but most currently do not actually do so. A consensus policy on activated charcoal use in the home by poison centers would be helpful.

100. RESULTS OF A POISON CENTER SURVEY REGARDING INFORMATION PROVIDED BY HOSPI-TALS TO SPECIALISTS IN POISON INFORMATION

Stremski E, Powers M. Children's Hospital of Wisconsin Poison Center, Milwaukee, Wisconsin, USA.

<u>Background</u>: Poison center (PC) staff may be refused patient information varying from total refusal of information to partial information disclosure, due to concerns of hospital staff regarding patient privacy. <u>Methods</u>: A survey was sent to hospital's designated HIPAA coordinator and Emergency Department directors. Responders were asked to define their role as Administrative only (ADM) or ADM and Clinical (CLN). Questions focused on the need for patient authorization, and disclosing of 7 TESS data elements. Yes responses were tabulated as the responder being ADM vs. CLN and analyzed via Chi-square, where p < 0.05 is significant. <u>Results</u>: 93 replies, 27% ADM role. Table shows: percent Yes as ADM, percent Yes as CLN, *p*-value. Conclusion: Both groups show similar reluctance to disclose

Patient authorization to consult PC needed if Life threatening.	16%	18%	p = 0.85
Patient authorization to consult PC needed if Non-emergent.	56%	44%	p = 0.42
Hospital staff can give PC: Patient name.	36%	45%	p = 0.79
Hospital staff can give PC: Patient ZIP code.	72%	65%	p = 0.62
Hospital staff can give PC: Patient's symptoms.	96%	100%	
Hospital staff can give PC: Patient's medical treatments.	96%	100%	
Hospital staff can give PC: Patient's disposition.	88%	94%	p = 0.32
Hospital staff can disclose to PC if suicidal intent.	64%	54%	p = 0.54
Hospital staff can disclose to PC if drug abuse intent.	64%	54%	p = 0.54
Hospital staff can disclose to PC if unintentional exposure.	76%	59%	p = 0.19

private patient information. The need and use of patient data by a PC must remain an important topic in professional outreach.

101. VALIDATION OF A METFORMIN POISON CENTER PROTOCOL: A 48-MONTH EXPERIENCE IN TODDLERS

LoVecchio F, Klemens J, Curry SC, Wallace KW. Good Samaritan Regional Poison Center, Phoenix, Arizona, USA.

<u>Background or Objective</u>: The incidence of hypoglycemia following accidental metformin ingestion in non-diabetics is unknown. We conducted a prospective poison center survey of serial glucose monitoring in children ≤ 3 in presumed isolated metformin ingestion. <u>Methods</u>: Inclusion criteria: (1) isolated metformin ingestion and (2) ≤ 3 years old. We recommended a minimum of 24 hours of in-patient observation with serial glucose monitoring. Adverse events (neurological sequela, etc.), hypoglycemia, and elevated lactic levels if obtained were documented. Hypoglycemia was defined as glucose <50 mg/dL and symptoms consistent with hypoglycemia, and hyperlactatemia was defined as plasma lactate >2 mmol/L. <u>Results</u>: 81 patients fulfilled the inclusion criteria with 29 patients refusing admission. Of the 51 admitted children the mean age was 19 [Range: 11–36] months with a range of ingestion of unknown to 3500 mg. The incidence of hypoglycemia was 0/32 and hyperlactatemia was 2/14. Lactic acid levels returned to normal within 8 hours in all patients. No long-term adverse outcomes were documented 0/61 (0%) [95% CI 0-5]. Of the 29 children followed at home for ≥ 24 hours none, developed any symptoms. <u>Conclusions</u>: We did not detect any hypoglycemia following isolated metformin ingestion in children ≤ 3 years old. The major limitations to this study is the small total number.

102. A PROSPECTIVE POISON CENTER EXPERIENCE OF SUSTAINED-RELEASE BUPROPION OVER **40-MONTHS IN CHILDREN**

LoVecchio F, Hilder R, Ruha AM. Good Samaritan Regional Poison Center, Phoenix, Arizona, USA.

<u>Background or Objective</u>: The incidence of seizures following accidental bupropion sustained release (Bup-SR) ingestion in children is unknown. We conducted a prospective poison center survey of in-patient monitoring in children ≤ 3 years old with presumed isolated Bup-SR ingestion during 40 months. <u>Methods</u>: Inclusion criteria: (1) isolated Bup-SR ingestion and (2) ≤ 3 years old. We recommended a minimum of 24 hours of in-patient observation. Adverse events (neurological events, etc.) were documented. <u>Results</u>: 71 patients fulfilled the inclusion criteria with five patients refusing admission. All patients, including patients whom remained at home were followed for at least 24 hours. Of the 71 admitted children the mean age was 18 [Range: 10–36] months with a range of amount ingested from unknown to 1200 mg. No seizures, coma, or short-term adverse outcomes were documented in the 71 admitted or 5 non-admitted patients within 24 hours. <u>Conclusions</u>: We did not detect any adverse events following isolated Bup-SR ingestion in children ≤ 3 years old. The major limitation to this study is the small sample size.



103. FOUR-YEAR EXPERIENCE WITH METHOTREXATE EXPOSURES

LoVecchio F, Watts D, Katz K. Good Samaritan Regional Medical & Poison Center, Phoenix, Arizona, USA.

Background: Methotrexate (MTX), a folic acid antagonist, is used in the treatment of cancer and autoimmune disease. Although the toxicities of MTX during standard clinical use are well-described, there is a dearth of information regarding either accidental oral ingestion or overdose. Methods: We conducted a retrospective chart review of all human exposure calls (>150,000 charts) for MTX ingestions reported to our Poison Center during 2000–2003. Results: 13 patients met the criteria. The average amount of MTX ingested was 13.03 mg (data from 7 cases), and the average patient age was 43 years (20 months to 80 years). The most common formulation ingested was the 2.5 mg tablet. Eleven patients accidentally ingested MTX, and two admitted suicidal MTX ingestion. These two suicidal patients also had benign co-ingestions. The two suicidal patients were admitted with one receiving leucovorin, and each had MTX levels drawn $(0.09 \text{ and } 0.47 \,\mu\text{mol/L}, \text{ therapeutic } <10 \,\mu\text{mol/L})$. Both were discharged without demonstrable hepatic or hematologic toxicity. 10 of the 11 accidental MTX ingestions were observed at home with PCC follow-up for 24 hours, all with good outcomes. One patient with accidental MTX ingestion was observed in the ED and was later discharged without incident. The four pediatric patients (three accidental, one suicidal) all did well. Only one patient received leucovorin rescue (one suicidal), and only two had MTX levels drawn (two suicidal). Discussion: Although MTX toxicity can be severe and, perhaps, life-threatening, this does not appear to be a phenomenon associated with either accidental or suicidal oral ingestion. This may be related to the significantly lower dosages associated with therapeutic misadventure or overdose, especially in the outpatient setting.

104. HELP, I'M PERPLEXED ABOUT APPLICABILITY—ASSESSMENT OF A POISON CENTER'S APPROACH TO MEETING THE HIPAA PRIVACY RULE

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<u>Background</u>: As health care providers, poison centers may have to meet the Privacy Rules of the Health Insurance Portability and Accountability Act of 1996 (HIPAA). We present an analysis of one poison center's attempt to address the Privacy Rule. <u>Methods</u>: Poison specialists were instructed to tell all lay callers involving a human exposure or information call, "The Federal Government has put in place new rules regarding patient privacy. We respect your privacy. You can review our privacy policy on our website" and the site name was given. 870 sequential charts were reviewed; 159 involved lay callers about human exposures in which the statement was made. <u>Results</u>: 92 surveys (58%) were completed within 1–7 days of the initial call. Patient's ages ranged from 3 months to 80 years; 75 (82%) were <6 years of age. Mothers were 55/92 (60%) of the callers, 10 were fathers, and 10 grandparents or other relatives. Only 29/92 (32%) were familiar with HIPAA. Out of 92, 74 (80%) remembered being told the privacy statement. Out of 74, 57 (77%) understood why they were told. Out of 92, 73 (83%) reported no prior confidentiality concerns. Out of 72, 50 (69%) reported no effect of the statement on their sense of confidentiality, 21 felt better being told, and one was more concerned. Out of 74, 55 (74%) remembered how to access the policy; out of 92, 81 (88%) had access to the internet. <u>Conclusion</u>: An abbreviated verbal privacy statement resulted in appropriate retention and raised no new concerns about poison center data handling. This approach seems to be an unobtrusive method of addressing the Privacy Rule.

105. COMPUTERIZED CUSTOMER SATISFACTION SURVEY

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<u>Objective</u>: To develop an automated, electronic survey to provide staff member (SPI) specific and case specific feedback on services provided by the Poison Control Center (PCC). <u>Methods</u>: A team of MBA students was hired to develop a telephone survey database. Sample customer satisfaction surveys were obtained from other poison centers and a survey of PCC needs was completed. The telephone survey was then designed using Microsoft Access[®] which linked directly to

the PCC SQL Database. Queries automatically selected random cases for specific date ranges and filtered for other parameters such as a full 10-digit telephone number and specific caller site. The survey script was programmed into a computerized form. This allowed the surveyor to read the paperless script from the computer screen, enter the responses directly into the form, and automatically store the responses in the database. Custom reports were created to provide summary data for each question for a given time period. All survey responses were associated with the original case number and thus to the original SPI. Reports were created to provide direct feedback to the individual SPI who took the original call. <u>Results</u>: Since this survey tool was developed, the PCC has completed three quarters with a minimal staff commitment of 12 hours a month. The flexibility of the survey tool allowed for quick modification or addition of questions for emergent need. A question was added asking what callers would do if the PCC was not available which provided timely and helpful information during the current budget crisis. <u>Conclusion</u>: An automated customer satisfaction survey is a valuable instrument to provide staff feedback, monitor satisfaction and gather timely information on emergent issues.

106. MEDICAL ERROR IN POISON CENTER PRACTICE: A PRELIMINARY STUDY

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<u>Objectives</u>: This was a preliminary study to determine the incidence, types, and results of medical error at a regional poison control center (PCC). <u>Methods</u>: Charts from a six year period (1/1/96–12/31/01) from a single poison center were contemporaneously, retrospectively reviewed by a certified poison information specialist (CSPI) and a second, non-blinded CSPI if a possible error was detected, as part of a quality assurance (QA) review. Error type, toxicity estimation, case management, transmitted information accuracy, and patient outcomes were evaluated. <u>Results</u>: Of 206,134 cases reviewed, 378 medical errors were detected in 365 cases (0.18%). Four percent of error cases had more than one error. Calculation errors occurred in 71% and management errors in 32% of error cases. Of the management errors, 81% involved follow-up failure. Overall, toxicity was underestimated in 41% and overestimated in 30% of error cases, leading to 6% that were judged undermanaged and 3% that were judged overmanaged. As a result of error, inaccurate information was communicated in 7% of error cases (0.01% of all cases). There were no known adverse outcomes as a result of error. <u>Conclusion</u>: The observed overall error rate, employing a routine QA methodology, is low compared with error rates reported in other medical settings and using other methodologies. Errors most commonly involved math calculations and case follow-up. The results are limited by the retrospective, and single site, design. A multicenter, prospective study is needed.

107. POISON CENTER MEDICAL ERROR DETECTION AND PREVENTION

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<u>Objectives</u>: This was Phase 1 of a 2-phase, multi-center study to detect, characterize, and reduce the incidence of medical error (ME) at poison centers (PC). <u>Methods</u>: In Phase 1, a representative sample of human exposure charts over a 1-year period from two regional PCs were retrospectively reviewed by two certified specialists in poison information (SPIs) for ME and local variance from protocol (VFP). A physician panel subsequently reviewed potential ME cases. <u>Results</u>: Of 3000 cases reviewed, 111 MEs were detected in 103 cases (3.43%). Medical errors were categorized by process step. Overall, data acquisition errors comprised 14%, information processing errors 15%, incorrect recommendations 30%, follow-up errors 27%, documentation errors 9%, and 5% other. 59% of MEs were considered preventable. Minor adverse outcomes occurred in 2 cases (0.07%) as a result of ME. Potential ME risk to patients was judged as 14% no, 41% minimal, and 45% moderate. No MEs were judged as serious or extreme risks. ME rates and distributions were not significantly different between the PCs. Cases with ME involved older patients, were more likely to be intentional exposures, and to have been coded with an effect outcome vs. a "not followed" code. Specialists in poison information with >10 years' experience made fewer MEs. variance from protocol occurred in 53% of cases. There was no statistical



relationship between ME and VFP. Non-PC-related MEs were identified in 1.1% of cases. <u>Discussion</u>: The observed error rate is low compared with other medical settings. Detection of error was increased >10-fold over a preliminary study using a single initial reviewer. Medical errors most commonly involved case management recommendations and follow-up process steps. Phase 2 will evaluate medical error reduction efforts. This work supported by DHHS/HRSA/MCHB Grant 1 H4B MC00041-01.

108. PAEDIATRIC LIDOCAINE (LIGNOCAINE) TOXICITY

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Background: The toxic dose for oral ingestion of lidocaine is not well documented. In Australia, a product marketed for teething was produced in an identical container to an acetaminophen preparation. This product contained lidocaine and chlorhexidine. This resulted in large number of therapeutic errors in which the teething preparation was given in error for acetaminophen and hence exact doses of ingested lidocaine were available for analysis. Methods: Calls reported to a state poison information centre were followed up. Information was collected regarding demographics (age, gender, and weight), type of exposure (accidental, therapeutic error) and details of the exposure (time of ingestion, ingested dose, and adverse effects. Results: Over a 12-month period there were 33 calls regarding this product and 24 cases with complete follow up. In 19 children the product was given as therapeutic errors (9 females and 10 males; median age 11 months (Range: 7 months-4 years). The mean ingested dose for lidocaine was 2.7 mg/kg (SD 1.3 mg) and for chlorhexidine was 0.06 mg/kg (SD 0.03 mg). The largest ingested lidocaine dose was 5.9 mg/kg. Two children developed minor symptoms. One child vomited twice following ingestion of 3.3 mg/kg lidocaine. The second was reported to have increased salivation and difficulty with solid food for 20 min following ingestion of 4.1 mg/kg. No other adverse effects occurred. There were five accidental ingestions not included in the analysis. Conclusion: The range of toxicity is often hard to determine in children because reported doses are inaccurate. Our study provided a unique opportunity because exact doses were measured. It would appear to be safe for ingestions of lidocaine less than 6 mg/kg to be observed at home.

109. REALTIME TESTING OF A RPICs DISASTER PLAN

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Background: When a mass contamination occurs, the poison center's responsibilities both internally and externally should be well defined. Not only is it important to delineate these tasks but it is imperative to test the protocols in a real time situation. A RPIC participated in a county wide drill involving a mass contamination. Methods: A RPIC representative was involved in the planning process of a region wide disaster drill. Critique criteria included communication and notification processes, a RPICs ability to rapidly assess victim symptomatology and identify possible contaminants, utilization of the RPIC by area health care professionals and call volume surge capacity. The RPIC staff was aware a drill was to occur but were not informed of its nature or evaluation process. All calls were to be documented in the normal fashion although cases were to be coded as information calls to avoid data errors. Results: 50% of the involved HCFs contacted the RPIC for treatment recommendations. Internal communications including notification of the RPIC director and medical director were appropriate. Reported symptoms were rapidly identified as being consistent with a nerve agent and/or a vesicant. A fact sheet was prepared by the director and, utilizing the RPICs pre-existing hospital facsimile program, this fact sheet was faxed to all regional hospitals. This drill identified a number of communications problems both within the RPIC hospital network as well as with government and public health agencies. Conclusion: The RPIC functioned efficiently during the drill process although communications was identified as a problem. Modifications were made and will be further tested in 2003 when a 13 county region wide drill is planned involving 72 hospitals. All PCs should utilize regional disaster drills to test their internal and external disaster plans.

110. UTILIZING POISON CENTER DATA FOR PUBLIC HEALTH SURVEILLANCE

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Background: Poison Control Centers (PCC) collect data on all exposure calls. Historically, data analysis has occurred retrospectively. The State Department of Health (DOH), using health maintenance organization data, has developed an auto regression analysis and cumulative sum (CUSUM) analysis for detection of influenza-like illnesses. This analysis method can be applied to PCC data. Methods: The Certified Regional Poison Center serves the entire State and received 45,756 unintentional exposure calls in 2002. Fourteen exposure categories from the Toxic Exposure Surveillance System (TESS) were chosen and grouped together for analysis. These categories limit the analysis to calls based on unintentional exposures, adverse reactions and unknown reasons. Toxic Exposure Surveillance System data is collected using Toxicall[®] software. Data is automatically extracted without any personal identifiers, and sent in real time to a secure server at the DOH. The data is continually transmitted to the DOH via automated FTP (file transfer protocol). The data file is automatically appended to 3 years of poison center historical call data and analyzed daily using an outbreak detection algorithm designed specifically by the DOH for surveillance and detection of a covert bioterrorism or chemical release. The algorithm normalizes the data using a regression model adjusted for day-of-the-week and holidays and CUSUM analysis to detect unexpected increases in call volume. The detection system triggers an "alarm" if the call volume exceeds an acceptable threshold. Results: The system has been in place since February 2003 and has triggered no alarms. Conclusion: Poison Control Centers collect valuable public health data that can be used to provide rapid, realtime surveillance for covert biological and chemical events. This system was developed quickly and inexpensively and could be replicated in other US PCC.

111. IMPLEMENTATION OF A NOVEL BACKUP SYSTEM FOR POISON CENTERS: PC-IN-A-BOX

Anderson BD. Maryland Poison Center, University of Maryland School of Pharmacy, Baltimore, Maryland, USA.

<u>Background</u>: Poison centers rely on computer networks to function. Few reports describe efforts made by centers to build redundant computer systems. <u>Methods</u>: A redundant, portable computer system was implemented utilizing four laptop PCs, a network switch, printer, several network cables, and a portable case to house/transport the equipment. One of the laptops was configured as a server. Data collection tools plus Micromedex[®] were installed on the server. Three laptops were configured as poison center workstations. <u>Results</u>: This system was deployed emergently this year. On January 25, 2003, the Sapphire/Slammer worm was released. Within 10 min, the worm had infected an estimated 75,000 hosts. This flood of information shut down servers worldwide. Network traffic was so great that local IT administrators decided to shut down main routers on our campus. Without routers functioning, computers in the poison center were unable to communicate with any servers. Poison specialists lost access to all on-line resources including Micromedex, data collection tools, email, and internet access. The PC-in-a-box was deployed. Data collection and Micromedex[®] access were quickly re-established. <u>Conclusions</u>: The PC-in-a-box provides effective redundancy of vital computer network functions.

112. TOXBASE $^{\textcircled{R}}$ —MEETING THE INFORMATION REQUIREMENTS OF AN INCREASINGLY DIVERSE USER BASE

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<u>Background</u>: The UK National Poisons Information Service (NPIS) provides expert telephone advice to health professionals. Since 1983 it has also provided free on-line poisons information (TOXBASE[®]). In 1999 a policy decision made TOXBASE[®] both Internet based and the suggested first point of access. This has resulted in a 6-fold increase in product accesses and >4-fold increase in users with different interests and needs. We report usage patterns to illustrate the scope of such systems. <u>Results</u>: Registered users increased from 775 at 12/99 [Accident & Emergency



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(A&E) 25% users, 91% of product enquiries] to 3489 at 12/02 [A&E 12% users, 49% of product enquiries]. Thirty two percentage of product enquiries in 2002 were from the 38 NHS public access telephone health information services (NHS Direct/24). Product accesses increased from 92,769 in 1999 to 552,877 in 2002. Top topics accessed were from: A&E—pharmaceuticals; NHS Direct/24—dosage calculator, household and acetaminophen; pharmacists—teratology information, pediatrics and antidotes; laboratories—laboratory services; public health emergency responders—deliberate release of chemicals and biologicals, decontamination procedures, and antidotes. <u>Conclusions</u>: Rapidly updateable Internet poisons information has been very popular in the UK. Usage patterns indicate different professional needs that Web systems can address speedily and economically.

113. ACCURACY OF CAREGIVERS IN ASSESSING LIQUID MEDICATION SPILLS—EXPANDED INVESTIGATION

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<u>Background</u>: A pilot study indicated caregivers (CG) overestimated liquid medication spills by more than 40%. This suggests that amounts ingested by children may be greater, putting them at increased risk of toxicity. To further investigate this concern, the authors expanded the pilot study to determine if the results could be replicated in a more representative population. <u>Methods</u>: 114 CG of children ages 14 months through 3 years who shopped at a large supermarket were enrolled. An aliquot of 15 mL of Guaifenesin Syrup, USP was poured on a t-shirt and a floor tile. Caregivers were asked to estimate the amount spilled on both. A clinically significant difference was defined as >15 mL. <u>Results</u>: Seven subjects were excluded because of incomplete or ambiguous data, leaving 107 subjects. Caregivers overestimated t-shirt spills by a mean of 25.8 mL (p < 0.05, range 1 mL to 120 mL). Eighty three (78%) were able to assess the spill within 15 mL; 24 (22%) overestimated by 30 mL or more. Caregivers overestimated tile spills by a mean of 31.4 mL (p < 0.05, range 3–135 mL). Of the 107 included in the tile spill segment, 75 (70%) were accurate within 15 mL; 31 (29%) overestimated the spill by 30 mL or more. There was no statistically significant difference between CG with one child and CG with more than one child in both segments. <u>Conclusion</u>: Most CG do not estimate spills on clothing and on floor tiles accurately, as assessed in this larger study. A clinically significant number of CG (>20%) were found to overestimate spills in amounts that may put children at increased risk of toxicity.

114. SPACE SHUTTLE COLUMBIA DISASTER: UTILIZATION OF POISON CENTERS

Keyes DC,¹ Shepherd G,¹ Borys D,² Ellis M,³ Ryan M,⁴ Watson WA.⁵ Texas Poison Center Network, ¹Dallas, ²Temple, ³Galveston, Texas, USA; ⁴Louisiana Drug and Poison Information Center, Monroe, Louisiana, USA; ⁵American Association of Poison Control Centers, Washington, District of Columbia, USA.

<u>Background</u>: On February 1, 2003 at 8:00 AM, CDT, the space shuttle Columbia broke up during re-entry. The event was visible and audible from the Dallas, TX area to eastern Louisiana. By 9:15 AM National Aeronautics and Space Administration (NASA) advised the public that the shuttle's propellants were a potential health risk and contact with the debris should be avoided. Within minutes, poison centers in the area began receiving requests for information regarding the nature and extent of toxicity expected. Many requests were from hospitals wanting guidance in anticipation of patients. Officials at NASA identified the potentially toxic substances as anhydrous hydrazine, monomethyl hydrazine, nitrogen tetroxide, ammonia, and americium isotopes. <u>Case Series</u>: In Texas and Louisiana there was an increase of 102 information calls over the median number expected for a weekend day. Texas poison centers received calls about 18 human exposures to shuttle debris all within the subsequent 9 hours. Thirteen were greater than 20 years of age and five were 6–12 years of age. Seven patients reported signs or symptoms; two were thermal burns to the hand. The remaining effects of contact with debris were mild and resolved quickly without specific management. <u>Conclusions</u>: The public health impact of space shuttle Columbia's debris was minimal. Information exchanged among poison centers and public health officials throughout the day supported the provision of accurate and timely information to health care providers, the media, and the public at large. Since poison centers inevitably receive calls about many health emergencies, it is important that public health agencies and poison centers communicate and coordinate their activities and messages.

115. WATER YO-YO EXPOSURES: AN UNUSUAL TEST OF TESS SURVEILLANCE

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Objective: Real-time surveillance of TESS data can potentially find small emerging trends in PCC case patterns among the 9562 calls documented daily (2002). We hypothesized that exposure calls would increase following a press release on the dangers of water yo-yos even though they were minimal to non-toxic and did not have a unique code in PoisIndex. The following evaluation was performed to determine if TESS surveillance could detect these calls. Methods: It was assumed that most calls would be coded as toys, which has a specific generic code in TESS. After the press release, an email sent to all PCCs centers asked that subsequent cases be coded as toys. Human exposures to cases with the toy code were evaluated using toy exposures from 1/1/2000 to 4/20/2003 as a baseline. TESS cases coded as toy exposures for 9 days prior to the press release (4/1-4/9/03) and exposures received for the day of, and 10 days after the press release (4/10-4/20/03) were evaluated. Data from 6/7 PCCs in the area most likely impacted by the press release were used to evaluate: (1) whether toy exposures cases were actually water yo-yos and (2) whether additional water yo-yo exposures were received but not coded as toys and therefore missed by TESS surveillance. Both evaluations required a manual search of verbatim data entries and all toy exposures. Results: Compared with baseline data, toy exposures for the sample centers showed a 3.9-fold increase following the press release. On 1 day the volume was greater than 3 standard deviations above the mean, and therefore easily identifiable. Only 7/71 cases after 4/1/2003 were not coded as toys. Conclusions: Because of the robust nature of the database small variations in exposures can be found over existing baselines. The utility of the system is dependent on accurate and consistent coding.

116. BEWARE OF CANINE GORILLA GLUETM INGESTIONS—A FIRST REPORT

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<u>Background</u>: Household adhesive ingestions by animals are considered relatively non-toxic. Gorilla GlueTM is a household glue containing a urethane polymer and a polymeric isocyanate liquid compound and is available in container sizes ranging from 2 to 18 ounces. When applied, the glue will expand 3–4 times its original size. We report the ingestion of Gorilla GlueTM by two canines that caused an obstructive mass which required surgical intervention. <u>Case Reports</u>: *Case 1*. A 35 pound dog chewed a container of Gorilla GlueTM and was asymptomatic 30 min post-exposure. Dilution was advised. He began vomiting 1 hour post-exposure and exhibited lethargy, vomiting, and appetite loss. He was examined by a veterinarian three times over a 20 day period with no specific physical findings. On the 20th day, he was found to have an abdominal mass and surgical removal revealed a large "mesh-like ball of glue." *Case 2*. Twelve hours prior to poison center consultation, a dog ingested an unknown amount of Gorilla GlueTM. The dog was vomiting and lethargic and was referred to a veterinarian for evaluation. Physical findings included a distended abdomen. During a 1 hour observation period, the dog's abdomen continued to expand and surgery was performed to remove a large ball of glue described as "resembling expandable insulation." <u>Conclusion</u>: This common household glue has been shown to produce delayed symptoms in two dogs that required surgical intervention. Dogs with a history of Gorilla GlueTM ingestion should be monitored closely by their owners and a veterinary referral should be made if any signs of gastrointestinal distress develop.

117. PERFORMANCE IMPROVEMENT INTERVENTIONS ON THE RATES OF GASTRIC DECONTA-MINATION RECOMMENDED BY A POISON CENTER

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Background: Although the use of gastric decontamination in the treatment of poisoning by poison centers has been declining for several years, a center's use may be higher than national rates and may benefit from a change in policy.



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<u>Methods</u>: The rates of gastric decontamination recommended by Poison Information Specialists were monitored through an electronic case record system. In February 2002, a policy that narrowed the use of gastric decontamination was implemented. A combination of staff education, provider education, specific treatment guidelines, medical consultation for exceptional cases, and monthly monitoring reports was undertaken as a performance improvement plan. The rates of recommendations during the year that preceded the interventions were compared with those of 12 months following a 2-month transition for all human exposures. Chi-square and Bayesian statistics were used to compare rates during the two periods. <u>Results</u>: Recommendations for the use of ipecac syrup declined from 1.50% to 0.02% (OR; 95% CI = 0.02; 0.01, 0.03), single-dose activated charcoal declined from 5.39% to 1.38% (0.25; 0.22, 0.28), gastric lavage declined from 4.19% to 0.22% (0.05; 0.04, 0.06), and a cathartic declined from 1.48% to 0.13% (0.08; 0.06, 0.12). The declines were significant (P < 0.001) and consistent for all forms of gastric decontamination. The proportions of patients managed at the scene of the poisoning were unchanged (1.04; 0.99, 1.09) before (67.64%) and after (68.50%) the start of the intervention as were those for referral to a health care facility (20.57% and 21.42% respectively, 1.05; 1.00, 1.11). <u>Conclusion</u>: Recommendations on gastric decontamination can be effectively modified with no change in patient disposition.

118. IMPLEMENTATION OF THE HEALTH INSURANCE PORTABILITY AND ACCOUNTABILITY ACT (HIPAA) OF 1996 AND THE EFFECTS ON POISON CENTER OPERATIONS.

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<u>Background</u>: The Health Insurance Portability and Accountability Act of 1996 (HIPAA) provides for the protection of certain health information and was implemented on April 14, 2003. In the months prior to HIPAA implementation, the reluctance of hospitals to provide follow-up information to poison centers seemed to increase. Specialists in poison information were concerned that once HIPAA guidelines were enacted officially, the poison center would have difficulty obtaining patient follow-up information. <u>Methods</u>: A retrospective review of all RPIC medical records involving patients who were treated in a hospital from April 14, 2003 until April 28, 2003 was completed. <u>Results</u>: Two hundred and fifty hospitalized patients were managed by the RPIC during this 2-week period. Only two hospitals failed to provide total disclosure of patient information. One hospital provided all pertinent information regarding the overdose information or the history and stated that they just wanted a medication identified that they suspected was ingested by the patient. There were no other reports in this 2-week period post-HIPAA implementation period when hospitals refused to provide information revealed that hospitals were cooperating with the RPIC in providing the necessary data in poisonings. To properly determine whether HIPAA will ultimately affect poison center medical information acquisition will require ongoing surveillance.

119. DETERMINING THE FEASIBILITY OF CREATING AND MAINTAINING A CENTRALIZED ANTIDOTE REGISTRY

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<u>Background</u>: With the threat of domestic bio/chemical terrorism at an all time high and with periodic shortages of much needed antidotes, a method in which to quickly locate given antidotes could avoid delays in therapy and decrease the risk of morbidity and mortality. This study was designed to determine the feasibility of creating and maintaining a centralized registry for hospital antidote stocking in a large metropolitan area. <u>Methods</u>: Twenty hospitals in a large metropolitan county were surveyed for current stocking levels of specific antidotes. An antidote chart with blank quantities was either mailed, email or faxed to each institution's pharmacy director and pharmacy buyer on three separate occasions over a two-month period. <u>Results</u>: 8 out of 20 hospitals responded within 3 weeks after an initial mailing to pharmacy directors. Four hospitals responded within the next 2.5 weeks after an email was sent to each pharmacy buyer. Four more responses were received within the next 2.5 weeks secondary to verbal communications followed by a fax. The information

gathered revealed: 8/16 (50%) hospitals stocked 1 or less cyanide antidote kits, 4/16 (25%) did not stock pralidoxime, 7/16 (44%) did not stock enough crotalidae antivenom to treat more than a single, minor envenomation, and 8/16 (50%) did not stock either IV ethanol or fomepazole. <u>Conclusions</u>: A response rate of only 75% was achieved despite multiple solicitations for information. Based on this, creating and maintaining an up-to-date centralized antidote registry, even for a single county, could be extremely difficult.

120. MANAGEMENT OF ACUTE PEDIATRIC LEAD PAINT CHIP INGESTION

Goldstein RA, McKay CA, Burke E, Roche K, Bayer MJ. CPCC, UConn, Farmington, Connecticut, USA.

Background: We have reviewed regional poison control center (PCC) protocols for lead paint chip ingestion (LPCI) and found a wide variation in management (with 60 of 67 PCCs responding). We reviewed our LPCI outcomes to determine an optimum management strategy. Methods: A retrospective review of all LPCI managed by our PCC was conducted between January 2000 and June 2002. Follow-up telephone calls were conducted for patients referred for care. Results: The PCC received 179 paint chip ingestion calls over this 18-month period, 86 of which were possibly pediatric LPCI. Following our protocol, the PCC advised 37 callers to use a home lead test kit (4 tested positive; follow-up was incomplete on 23). Thirty-five cases were referred to pediatrician's offices for blood lead determinations, and 14 were referred to ED for X-ray. Six of the ED referrals were positive for radio-opaque substances throughout the GI tract. Of 22 completed telephone follow-ups, there were only four with complete data who allowed release of their records. All 4 of these patients had positive X-rays. The maximum lead determination in these four patients was 16.4 mcg/dL. Three of 4 had blood lead <10 mcg/dL when rechecked within 1 month. Conclusion: High blood lead determinations were not commonly found in our review; however, the optimum management strategy cannot yet be determined secondary to variable compliance with recommendations and incomplete data. Nearly one-half (6 of 14) of children referred into the ED after recognized LPCI had widely distributed lead in the GI tract, supporting the contention that these calls are actually the acute recognition of a chronic ingestion. Poison control center protocols for the management of LPCI need to recognize this concept.

121. FASCIOTOMY WORSENS MYONECROSIS AND HEMORRHAGE IN A PORCIONE MODEL OF INTRAMUSCULAR INJECTION OF CROTALINE VENOM

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Objective: To evaluate the efficacy of fasciotomy (FAS) in decreasing myonecrosis (MYO) and hemorrhage (HEM) after intramuscular crotaline envenomation. Methods: 20 anesthetized swine were injected IM in the anterior tibiales of both hindlimbs with 6 mg/kg of C. Atrox venom (total 12 mg/kg venom per animal). Immediately following venom injection, one of the envenomated hindlimbs underwent FAS. Muscle biopsies (BX) were obtained from the fasciotomized hindlimb at 0, 4, and eight hours and from the unfasciotomized hindlimb at the conclusion of the study (time 8 hr). One hour post envenomation animals received IV either 8 vials of reconstituted Crotaline Fab antivenom (FabAV) or an equal volume of saline. BX were analyzed by a pathologist blinded to the study. The histologic degrees of MYO and HEM in the BX were scored as follows: 0 = none, 1 = mild ($\leq 10\%$), 2 = moderate (11–30%), 3 = severe (31–70%), 4 = extensive (>70%). Statistical analysis was performed using rank tests as indicated. Only animals surviving to the conclusion of the study were included. Results: 16 animals survived to the conclusion of the study. BX from hindlimbs that underwent FAS revealed a progressive increase in both the amount of MYO and HEM over time following venom injection (MYO means at 0, 4, 8 hr: 0, 1.8, 1.9; p < 0.001, HEM at 0, 4, 8 hr: 0, 2.8, 3.3; p < 0.001; Friedman test). Comparison of the amount of MYO and HEM of BX at 8 hr revealed that limbs that underwent FAS had significantly more MYO and HEM than limbs that did not undergo FAS (MYO: 1.9 vs. 1.1; p < 0.05, HEM: 3.3 vs. 2.0; p = 0.004, signed-rank test). In subgroup analysis, FabAV did not significantly reduce MYO in either hindlimb (FAS or no FAS) at 8 hours (1.3 vs. 1.7; p = 0.250, rank-sum test), but did reduce the amount of HEM present (2.3 vs. 3.3; p = 0.008). Conclusions: FAS significantly worsened the amount of both MYO and HEM seen on BX in a porcine model of intramuscular injection of crotaline venom. Fab antivenom treatment significantly reduced the amount of HEM in this model.



122. CLINICAL EFFECTS OF WOLF SPIDER BITES IN AUSTRALIAN

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Background: Necrotic arachnidism continues to be attributed to wolf spider bites. This study investigates the clinical effects of bites by wolf spiders (family Lycosidae) and compares to other spider bites. Methods: Subjects were recruited prospectively from February 1999-April 2001 from participating emergency departments or state poison information centers. Subjects were included if they had a definite bite by a wolf spider, collected the spider and it was identified by an arachnologist. All subjects were followed up. Results: Of 1474 suspected spider bites, 46 were definite wolf spiders bites (23 males and 23 females; age range 1-69, median age 28 years). Bites were from seven different genera, including "Lycosa" (10), Venatrix (8), Venator (8), Hogna (7), and "Allocosa" (8). Bites occurred throughout Australia, 8 bites (17%) in late autumn and winter and 29 bites during daylight hours. Twenty-two bites (48%) occurred indoors. Seventy-two percentage of bites occurred on the hand or foot. Pain occurred in all bites, was severe in 11 (24%), with a median duration of 5 min (IQR: 0.5–56 min). Other effects included: puncture marks/bleeding (35%), swelling (20%), and redness (65%). Systemic effects occurred in three patients (7%), nausea (2), headache (1), and malaise (1). There were no cases of necrotic ulcers [0%; 97.5% CI 0–8%]. There was no difference in clinical effects between lycosid genera. Conclusion: Wolf spider bites cause minor effects and not necrotic ulcers. The effects are likely to be due to a mechanical injury rather than venominduced. There were no major differences between clinical effects of different genera. Clinical effects of wolf spider bites were similar to bites by huntsman spiders (Sparassidae), another group of large spiders, but were different to the medically significant group of widow spiders in which venom induced effects cause prolonged pain and systemic effects.

123. SUBCUTANEOUS INJECTION OF CROTALINE FAB ANTIVENOM FOR THE TREATMENT OF RATTLESNAKE ENVENOMATION IN A PORCINE MODEL

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<u>Background</u>: Intravenous crotaline Fab antivenom (FAB) is the current standard of care for the treatment of rattlesnake envenomation. Due to its high potency, low frequency of allergic reactions, and small molecular size this antivenom may be well suited to local injection and venom neutralization at the envenomation site. <u>Methods</u>: We tested the effects of local, subcutaneous antivenom injection on limb swelling in a porcine model. Each animal was anesthetized, intubated, and maintained on mechanical ventilation. *C. atrox* venom was then injected subcutaneously at the hock of the right hind leg. Animals were randomized to subcutaneous antivenom (SQ), intravenous antivenom (IV), or control arms. Interventions were made 2 min after venom injection. SQ animals received two vials of Fab subcutaneously at the envenomation site and two vials intravenously. IV animals received four vials of Fab intravenously. Limb circumference was measured hourly for 8 hours. Limb volumes were measured at 4 and 8 hours by water displacement. <u>Results</u>: 26 animals were randomized to three treatment groups. The SQ and IV groups included 9 animals each. There were 8 controls. In the SQ group, 2 animals died suddenly within minutes of Fab injection. There were no deaths in the IV or control arms. Neither limb circumferences nor volumes were different between control and either treatment arms. <u>Conclusion</u>: Treatment of rattlesnake envenomation with SQ antivenom injection was associated with mortality not seen in other treatment groups. Neither SQ nor IV antivenom therapy was associated with less limb edema when compared to control.

124. A DOSE-RESPONSE EVALUATION OF THE GHB PRECURSOR, TETRAHYDROFURAN

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<u>Background</u>: Since the regulation of illicit GHB as a federal schedule I drug, GHB has been substituted with its chemical precursors GBL, 1,4-BD, and most recently tetrahydrofuran (THF). Case reports of THF overdoses have described coma, hypotonia, and respiratory depression. However, its neurotoxicity as a GHB precursor relative to GBL and 1,4-BD are not known. Objective: To conduct murine dose-response evaluations of THF neurotoxicity, determine its Toxic Dose-50 (TD₅₀),

and compare its TD_{50} to those of GBL and 1,4-BD previously established in our lab. Methods: 100 CD-1 mice were administered THF 300–1000 mg/kg i.p. and evaluated for neurotoxicity by the righting reflex and rotarod test (N=10 for each dose). The TD_{50} (with 95% confidence intervals) of THF for the righting reflex and rotarod test was calculated by the Litchfield-Wilcoxon Test and compared by the *Z*-statistic to those of GBL and 1,4-BD previously established in our lab. <u>Results</u>: The TD_{50} of THF for the righting reflex and rotarod test was 691.7 (587.2–814.8) and 487.8 (436.9–544.6) mg/kg, respectively. These results were statistically significant (P < 0.05) when compared to the righting reflex and rotarod TD_{50} of GBL [366.2 (273.6–490.2) and 99.0 (60.0–163.2) mg/kg, respectively] and 1,4-BD [567.4 (505.8–636.5) and 163.9 (153.5–175.0) mg/kg, respectively]. Furthermore, THF toxicity for the rotarod test was significantly reduced by the GABA_B receptor antagonist SCH 50911 (30 mg/kg i.p.), which we previously demonstrated in our lab with GBL and 1,4-BD. <u>Conclusion</u>: THF is less neurotoxic than the GHB precursors GBL and 1,4-BD. Like GBL and 1,4-BD, THF neurotoxicity appears to be mediated by GABA_B receptors, which was reduced with the GABA_B receptor antagonist SCH 50911.

125. MYOCARDIAL HYPERTROPHY IN USERS OF METHYLENEDIOXYMETHAMPHETAMINE MDMA

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<u>Background</u>: Myocardial hypertrophy is a well-recognized complication of stimulant abuse and is a strong, independent risk factor for sudden death, myocardial infarction, and congestive heart failure. We sought to determine if use of MDMA is associated with myocardial hypertrophy at death. <u>Methods</u>: A matched, retrospective study using medical examiner (ME) death reports. Consecutive MDMA positive (+) and MDMA negative (-) deaths identified from MEs in 10 states and a local county, respectively. Five MDMA(-) cases were matched to each MDMA(+) case for age, gender, and ethnicity. Methylenedioxymethamphetamine(+) cases were confirmed using GC/MS and other drugs of abuse (e.g., cocaine and amphetamine) were absent. Matched MDMA(-) cases were trauma fatalities with intact hearts and blood negative for all illicit stimulants. Cardiac weights were compared between the two groups. <u>Analysis</u>: Two-sample *t*-test and conditional logistic regression using SAS v 8.02. <u>Results</u>: 27 MDMA(+) deaths and 135 matched MDMA(-) deaths were enrolled. Mean age was 20 (range 16–33); 44% were female. 70.4% were Caucasian, 14.8% African-American, 11.1% Asian, and 3.7% Hispanic. Mean heart weight of MDMA(+) fatalities was 315.7 g and 277.2 g for MDMA(-) fatalities (Diff = 38.5; 95% CI = 43.7–54.4). Multivariate analysis revealed that MDMA(+) fatalities were more likely to have enlarged heart (OR = 6.5; 95% CI = ; p < 0.0002). <u>Conclusion</u>: The findings of this study suggest that MDMA users might also be at risk for cardiac toxicity, similar to other stimulants.

126. DETERMINATION OF GAMMA HYDROXY BUTYRATE IN STRIATAL DIALYSATES OF RATS TREATED WITH 1,4-BUTANEDIOL

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<u>Background</u>: Gamma-hydroxybutyrate (GHB), a metabolite of gamma-aminobutyric acid (GABA), has been proposed as a putative neurotransmitter/neuromodulator. 1,4-Butanediol (1,4-BD), which is metabolized in the body by alcohol dehydrogenase (ADH) to GHB, and gamma-butyrolactone are drugs of abuse that have been gaining popularity in the United States as club drugs as a substitute for GHB after its listing as a Sch. 1 drug by the FDA. In the present study, we investigated the production of GHB in striatal dialysates after administration of 1,4-BD. <u>Methods</u>: After an initial collection of baseline samples for 2 hours, 1,4-BD (500 mg/kg, i.p.) was administered to awake, freely moving Sprague– Dawley rats undergoing microdialysis within the striatum. Samples were collected thereafter every 20 min and analyzed for GHB by HPLC-UV at 220 nm wavelength. <u>Results</u>: Acute administration of 1,4-BD (500 mg/kg) produced a gradual rise in dialysate GHB levels over time, the peak effect ($14.07 \pm 9.47 \mu g/mL$) observed 1 hr 40 min after 1,4-BD. This rise in GHB levels was prevented by pretreatment with 4-methyl pyrazole, an ADH inhibitor. Further, there appeared to be a correlation between the presence of GHB in the dialysates and the "sleep" induced by 1,4-BD. <u>Conclusion</u>: This data confirms that GHB is most likely the active metabolite involved in the production of "sleep" and possibly the other rewarding effects induced by 1,4-BD. Also, this is the first report to demonstrate the presence of GHB in microdialysates analyzed by a HPLC-UV technique, illustrating its utility in studying other probable GHB precursors.

127. VASOPRESSIN TREATMENT OF VERAPAMIL TOXICITY IN THE PORCINE MODEL

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<u>Objective</u>: Vasopressin is a novel vasopressor agent that is used for intractable hypotension. There is little data available on its use in the poisoned patient. We performed a randomized, controlled, blinded trial in a porcine model to study the effects of vasopressin infusion on mean arterial pressure after verapamil poisoning. <u>Methods</u>: Seventeen immature anesthetized monitored swine received a verapamil infusion at 1 mg/kg/hr until the mean arterial pressure (MAP) had decreased to 70% of baseline. At this time, a continuous infusion of either vasopressin (0.1 U/kg/min) or an equal volume of normal saline was initiated. Swine were monitored for 60 min after initiation of study infusion. Primary outcomes were survival and MAP. <u>Results</u>: Animals treated with vasopressin were less likely to survive than those treated with saline. One half (four of eight) of the animals in the vasopressin group died, compared with 22% (two of nine) of those in the saline group. Although not statistically significant, there was a trend towards increased MAP in animals treated with vasopressin infusion of treatment. <u>Conclusion</u>: Vasopressin infusion decreased the survival of verapamil poisoned swine when compared to those treated with saline alone.

128. THE CHARACTERIZATION AND DEVELOPMENT OF MICROSTRUCTURED CARBONS FOR THE TREATMENT OF DRUG OVERDOSE

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<u>Background</u>: Many drugs (e.g., amitriptyline) are hydrated in the stomach and therefore may be comparable or larger in size than the micropores (<20 Å) in current activated charcoal (AC) preparations. Mesoporous (20–500 Å) carbons may facilitate access of such drugs to the internal surface area of the carbon, increasing drug adsorption capacity and adsorption kinetics. <u>Methods</u>: Adsorption characteristics of three specially prepared mesoporous carbons (MC1, MC2, and MC3) were compared with AC preparations used in poisoned patients [Charcodote (CD), actidose aqua advance (AAA)]. Pore size distribution and surface area of the carbons were determined from adsorption–desorption isotherms of nitrogen at 77 K. Equilibrium adsorption isotherms and kinetic studies were carried out with amitriptyline (0–800 mg/L) in simulated gastric fluid (pH 1.2) at 37°C. <u>Results</u>: Charcodote displayed some mesoporosity (20–50 Å), AAA was predominantly microporous (<30 Å), MC1 was mesoporous (20–200 Å) and MC2 showed larger pores (100–1000 Å); MC3 had a similar pore structure to MC2 but had been oxidised and had better surface wetting characteristics. Surface area of the carbons: AAA 1243 m²/g, CD 1444 m²/g, MC1 2280 m²/g, MC2, and MC3 550 m²/g. Maximum amitriptyline equilibrium uptake: AAA 450 mg/g, CD 470 mg/g, MC1 > 750 mg/g, MC2 < 50 mg/g, and MC3 200 mg/g. <u>Conclusions</u>: Surface area contribution from mesopores enhances the adsorption capacity of carbons for the removal of amitriptyline. Tailoring the pore structure of carbons used in oral adsorbents could reduce dosage requirements and due to the lower solids fraction improve the palatability of the carbon slurry.

129. INHIBITION OF CELLULAR TOXICITY OF OXALATE BY EDTA AND CITRATE

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<u>Background</u>: A key feature of ethylene glycol (EG) poisoning is acute renal failure. Renal toxicity results from EG metabolism and can be prevented by fomepizole to inhibit formation of toxic metabolites. Precipitation of calcium oxalate (COM) crystals have been proposed as the etiology for the organ injury, leading to acute tubular necrosis.

In patients where metabolism has already occurred, adjunctive therapy may be applied to minimize the cellular toxicity of oxalate in the kidney. The mechanisms for cellular effects of oxalate have been studied in cultured kidney cells and in erythrocytes, as a model for cell membrane damage, to design, and test preventative agents. <u>Methods</u>: Normal human proximal tubule (HPT) cells in culture were exposed to COM or oxalate, with and without EDTA or aluminum citrate (AlC). Cytotoxicity was assessed by LDH leakage and by ethidium homodimer uptake. Suspensions (0.5%) of rat red blood cells in PBS were treated with COM or oxalate, with and without EDTA or AlC. The hemolytic rate was measured at 540 nm. <u>Results</u>: EDTA, at 4 mM, reverses the cytotoxicity of COM in HPT cells and blocks COM-induced hemolysis. The mechanism for EDTA is to complex calcium and thus to liberate free oxalate ion, which itself does not induce cell membrane damage. AlC, at 0.2 mM, also prevents renal cytotoxicity of COM and blocks COM-induced hemolysis. Its appears to alter the physico-chemistry of the crystal surface, inhibiting its association with and damage to the membrane damage, leading to leakage and cell death. Toxicity can be reduced by treatment with EDTA to solubilize the crystals or with AlC to minimize crystal aggregation or precipitation. Treatments that block oxalate membrane damage might reduce the renal toxicity of EG.

130. THE EFFECT OF AMIODARONE ON AMITRIPTYLINE POISONED MICE

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Objective: Amiodarone, a class III antidysrhythmic agent, blocks Na⁺, Ca²⁺, K⁺ channels as well as the β -adrenergic receptor. Despite the paradigm shift toward amiodarone in the setting of wide-complex tachycardia, it has not been investigated for tricyclic antidepressant-induced dysrhythmias. Because there is concern that amiodarone may have adverse effects in this setting we studied lethality in a mouse model. The null hypothesis was that amiodarone would not alter the LD50 of amitriptyline in mice. Methods: Based on the Litchfield and Wilcoxon mathematical method and previous mouse studies, we confirmed the LD50 of amitriptyline by giving 100 mg/kg of intraperitoneal (IP) amitriptyline to 40 mice. Similarly, the safety of the treatment dose of amiodarone alone (control) was confirmed by giving 50 mg/kg amiodarone IP to 10 mice. One hundred and nine mice were randomized to receive pretreatment with 50 mg/kg IP amiodarone (n = 55) or an equal volume of a placebo control (n = 54). 30 minutes later, the mice received amitriptyline 100 mg/kg IP. Outcome was defined as death or survival 3 hours after amitriptyline injection. No mice died after 3 hours. Results: When confirming the LD50 of 100 mg/kg of amitriptyline, 25/40 mice died (62.5%). No mice in the amiodarone control died (n = 10). In the control + amitriptyline arm, 36/54 died (66.7%) compared with 39/55 mice died (70.9%) in the amiodarone + amitriptyline arm. Using X^2 analysis, p = 0.663. A power analysis demonstrated a 90% chance of finding a 28% difference. Conclusions: Pretreatment with amiodarone does not appear to alter lethality from amitriptyline poisoning in mice. Given the inability to monitor the ECG in a small animal model, further investigation in a larger animal is required.

131. A NOVEL SELF-CONTAINED HEMOPERFUSION DEVICE FOR THE TREATMENT OF THEO-PHYLLINE OVERDOSE IN A SWINE MODEL

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<u>Objective</u>: To evaluate the efficacy and safety of a new hemoperfusion (HP) device in the clearance of theophylline (theo) following intravenous overdose. <u>Methods</u>: Three animals were administered aminophylline (40 mg/kg theo equivalent) over 30 min, with determination of endogenous clearance. Subsequently, a cross-over extracorporeal drug removal was performed by hemodialysis (HD) or HP with a wash-out period of at least 24 hours between procedures. Hemodialysis was performed using a Fresenius 2008H machine $(1.5 \text{ m}^2 \text{ polysulfon membrane F160NR})$ with a blood flow rate of 300 mL/min and a dialysate flow of 500 mL/min. Hemoperfusion used a prototype device consisting of a polysulfone hollow-fiber membrane cartridge (0.55 m^2) , activated charcoal sorbent, and peristaltic pumps for generating blood and dialysate flows. Serial theo serum concentrations were obtained, along with measurements of blood chemistries and indicators of hemolysis. <u>Results</u>: Theophylline clearances were compared between control, HD and HP runs.



Group	Theophylline half-life (T ¹ / ₂) (min) \pm SD	$T^{1/2}$ per m ² membrane area (min) \pm SD
Control	730 ± 122	_
HD	86 ± 12	57 ± 8
HP	201 ± 100	37 ± 18

<u>Conclusion</u>: Hemoperfusion resulted in significant theo clearance. There were no solute shifts or hemolysis as routinely seen using other HP-based systems.

132. PILOT STUDY: USE OF CALCIUM CHLORIDE TO TREAT HYPERKALEMIA DUE TO ACUTE DIGOXIN TOXICITY IN A SWINE MODEL

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<u>Background</u>: At toxic levels, digoxin inhibition at the sodium–potassium ATPase is exaggerated, resulting in significant increases in intracellular calcium within myocytes and systemic hyperkalemia. Early studies suggest an additive relationship to explain enhanced toxicity seen with calcium administration in cardiac glycoside toxic models, and the clinical entity of "stone heart" has been described. IV calcium treatment for hyperkalemia in this setting might be harmful, and should only be used if it shows clear benefit. The treatment of hyperkalemia with IV calcium in the setting of digoxin toxicity in the porcine model has never been previously reported. The purpose of this study was to evaluate this interaction in a porcine model. <u>Methods</u>: Anesthetized pigs were given IV digoxin in doses of 0.25 mg/kg. Once the pigs became hyperkalemic, they were treated with either an IV calcium chloride bolus of 10 mg/kg (Group I) or saline (Group II) and the effects were observed for 3 hours. <u>Results</u>: All of the pigs died. There was a longer survival time in Group II (49.5 min) vs. Group I (23 min). The administration of calcium chloride to the hyperkalemic pigs in Group I did not terminate ectopy or arrhythmias, and may have shortened their time to death when compared with Group II. <u>Conclusion</u>: In this pilot study though this study did not have the power to demonstrate statistically significance with calcium administration, our findings seem to support that calcium should not be used to treat hyperkalemia in the setting of acute digoxin toxicity.

133. HOUSEHOLD MERCURY (Hg) SPILLS: VARIATION IN POISON CENTERS (PC): HAS ANYTHING CHANGED?

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Background: Poison centers commonly provide cleanup and disposal advice for small Hg household spills, especially from broken thermometers. Our previous survey (1997) of north American PC identified significant inconsistencies and inaccuracies in PC recommendations. We sought to identify current PC practices in order to determine what changes have occurred. Method: All PCs (78) called in 1997 were asked the same questions by phone: (1) What clean-up/disposal advice does your PC provide in event of small (<5 mL) Hg spill at home? on hard surface? on carpet? (2) Do you have written protocol? If not, what information do you give on vacuuming? 2002 responses were compared with 1997 answers. Results: 70 PC responded (7 US and 1 Canadian PC had closed/merged since 1997). Positive changes identified include: (1) 23 (33%) with no previous written protocol in 1997 now use one; (2) more accurate hard surface clean-up advice: 46% increase in use of stiff paper, 26% increase in use of tape, 8% decrease in use of broom, 15% increase in use of wet paper towel; (3) improved rug cleanup advice: 27% decrease in recommending vacuum use, 7% increase in discarding vacuum bag if used prior to call; (4) 32% decrease in disposing in household garbage; and (5) 33% increase in PC referral to Hazmat agencies or state health department for disposal instructions. Negative changes include: (1) 26 (36%) still have no written protocols, 6 (8.5%) with initial protocols no longer use written protocols. Other areas of concern include (1) 6 (8.5%) still recommend vacuuming; (2) only a 8% increase in warning about aerosolization risks; (3) not asking about central vacuum use; and (4) no change in recommendations for double bag and seal disposal. Conclusion: Although the overall trend is positive, there remain a few areas of concern with room for ongoing improvement.

134. CHARACTERIZING THE INFORMATION SPECIALIST (IS)-MEDICAL TOXICOLOGIST (MT) CONSULTATION INTERACTION

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<u>Background</u>: Optimal care of the seriously poisoned patient requires the IS and MT to function together in partnership to ensure accurate clinical judgement and the delivery of appropriate current management advice. <u>Method</u>: We define four types of IS–MT consultation interactions: (1) Validation: The case is within the previous experience/training of the IS, and is developed by the IS through to recommended management. The MT confirms management can proceed as proposed. (2) Analysis: Case assessment indicates the situation is outside of IS range of understanding or how to proceed. The IS identifies alternatives and indicates where their uncertainty lies. The MT directs specific phases of the case review and management process such that the IS, based on guidance from the MT, can complete the case. (3) Direction: The IS requires specific instructions on how to proceed because the situation is outside IS understanding/experience/training or because the intensity and complexity of the case requires MT direction. While all phases of the process are directed by the MT, the IS may be required to deliver the treatment plan. (4) Control: The case requires the practice of medicine, requires physician to physician discussion or is outside of IS experience or comfort. Information specialist collects and correlates data so that the case can be analyzed by the MT. Medical toxicologist completes analysis, directs research, with the IS assisting as directed and delivers the treatment plan. Information specialist continues case followup as required by the case and MT direction. <u>Conclusion</u>: Formalizing these interactions both clarifies specific expectations during the consultation and provides a foundation for a developmental feedback plan for IS.

135. MULTIPLE COMMUNICATIONS FOR POISON CENTERS DURING MASS CHEMICAL EXPOSURE

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<u>Background</u>: Flow of accurate, timely information is problematic in mass disasters because usual methods of communication may be disrupted. Community emergency response agencies must develop redundant back-up systems to maintain vital communications networks during disasters. We describe one poison center's use of redundant communications systems to maintain routine and emergency response services during disasters. <u>Methods</u>: Our Poison Center's role in public health emergencies is to provide human health information to the public, health care providers, emergency responders, public health staff and emergency operations center staff. Our plan uses redundant information exchange. All phones can be diverted to a remote site, should evacuation be necessary. Two-way pagers are used for medical back-up. Broadcast FAX is used for notification and general information dissemination to all 50 hospitals. Portable laptop computers can access servers from remote sites by virtual private network. Key personnel are equipped with satellite phones and datalinks for remote server access. Nextel^(IIII) cellular phone access with direct connect feature allows contact with others during cellular and landline failures. Our most important communication link is with the National Radio Amateur Civil Emergency Services (RACES). Our center has handheld and base station HAM radio capabilities and some of our staff recently obtained amateur radio technicians licensing. <u>Conclusion</u>: Poison centers play a key role as information resources during disasters. Back-up systems for maintaining this communication network are essential.

136. SURVEY OF FIRST RESPONDER AND HEALTH DEPARTMENT OFFICIAL'S KNOWLEDGE OF POISON CENTER SERVICES

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Background: Few studies have been performed to evaluate paramedics (EMS), police (PO), and health department official's (HD) knowledge of poison center (PC) services. This pilot study was performed to survey the familiarity of



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these three groups with their local PC services. <u>Methods</u>: A survey was administered to EMS, PO, and HD personnel. On a scale from 1–4 (1 = strongly disagree, 2 = disagree, 3 = agree, and 4 = strongly agree), each person surveyed was asked if they were aware that the local PC provided the following services: poison information service (PIS), poison treatment advice (PTA), pill identification (PI), public/professional education programs (EP), terrorism expertise (TE), and hazardous materials expertise (HME). <u>Results</u>: A total of 111 completed surveys were obtained with results noted in the table below:

	PIS	PTA	PI	EP	TE	HME
EMS	3.8	3.7	3.4	3.2	2.9	2.9
PO	3.4	3.4	3.3	3.1	2.6	2.7
HD	3.7	3.7	3.2	3.1	2.6	2.8

<u>Conclusion</u>: Knowledge of local PC services is good in the traditional areas of poison information services and treatment advice. Local PC should continue to educate EMS, PO, and HD personnel of PC services provided in areas of terrorism and hazardous materials expertise.

137. EVALUATION OF A TV AD CAMPAIGN PROMOTING POISON CENTER (PC) AWARENESS

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<u>Background</u>: Our PC carried out a TV ad campaign in two phases of 7 (fall) and 4 (winter) weeks, respectively, to promote increased awareness of the PC as a resource for poisoning exposures. Two TV ads were developed and run: "Washroom" ad and "Strip" ad both targeting adult poisoning scenarios. <u>Method</u>: A telephone survey was conducted by a professional survey company. This consisted of a telephone survey of the residents of the large metropolitan area targeted in the fall campaign. Four hundred telephone interviews were conducted 1 week before the final week of air time. For each ad, questions were asked about unaided recall, proven recall, aided recall, sponsorship recall, main message recall, and advertising effectivness. <u>Results</u>: There was 33% unaided recall proven largely by respondents' unaided descriptions of the ads. Proven recall for the Washroom ad was higher. There was 67% aided recall, higher with the washroom ad (56%) than with the Strip ad (44%). Forty percent could correctly identify the sponsor but 49% could not identify any sponsor. The main message recalled was related to the 1–800 number and proper use of chemicals. Only 13% could not recall any message. Both ads showed 80% effectiveness as defined by: informative (36%), memorable (15%), gets your attention (13%), tells you a number to cal (12%), humorou (10%), good messag (6%). <u>Conclusion</u>: The TV ads achieved a broad reach. They were more effective in promoting the messages than the sponsor. While 33% unaided recall for TV ads is encouraging, aiming for a 60% recall would permit employing other media strategies that build on this initial awareness message.

138. THE EFFICACY OF ANTIVENIN FOR RUSSELL'S VIPER SNAKEBITE

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<u>Objective</u>: Formosan Russell's viper (*Daboia russelli siamensis*) is the sixth most frequent cause of snake bites in Taiwan. This viper's snakebite is rare and thus scarcely subjected to systemic studies. Specific antivenin was available only at few medical centers before, and lead to induce intoxication with severe degree of acute renal failure, incoagulable blood and hemolysis in patients of systemic envenoming. <u>Methods</u>: Prospectively, we performed a clinical trial from June of 1999 to December of 2001 to test the efficacy of antivenin for Formosan Russell's viper snakebite. We delivered the antivenin to the teaching hospitals that are near to the areas of *D. r. siamensis* distributed to treat snakebite patients as soon as possible. We also developed an enzyme-linked immunosorbent assay (ELISA) to diagnose the Russell's viper snakebites. <u>Results</u>: In ELISA study, the standard dose response curve of viper venom in calf serum showed a good linearity between 1 and 100 ng/mL of venom concentrations. In clinical study, 13 cases of *D. r. siamensis* snakebite were

collected totally. Coagulopathy with bleeding tendency and acute renal failure occurred early and were the two most important clinical features. Early antivenin treatment, 3–6 hours after systemic envenoming, would restore the coagulation function in 1–2 days and seemed to be effective in reducing the severity of renal damage and preventing from the necessary of hemodialysis therapy. Specific antivenin therapy also played a significant role in reducing the occurrence of other organs complications. Two to four vials of specific antivenin were needed to blocking the Russell's viper envenoming in Taiwan. <u>Conclusion</u>: The monovalent equine antivenin raised against local Russell's viper were proved to be effective. The antivenin should be administrated as early as possible to prevent from organs dysfunction.

139. LONG-TERM OUTCOMES FOLLOWING RATTLESNAKE ENVENOMATIONS: A PRELIMINARY STUDY

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<u>Background</u>: Long-term outcome following rattlesnake bite (RSB) is poorly described. <u>Methods</u>: We conducted a prospective, poison center (PC) based study on all patients with RSB during 3/02-5/02. Inclusion was RSB referred to our PC. Exclusion criteria was a dry bite. Demographic data was obtained (age, site of envenomation, extent of swelling, serial laboratory data (platelets, fibrinogen, and prothrombin time), type of treatment received (WyethTM vs. CroFabTM Antivenin vs. none) and length of stay (LOS). Snakebite Severity Score (SSS: previously validated by Dart RC) was used. A SSS of 1–2 correlates with mild envenomation and 6–8 is considered severe. The main outcomes were subjective days to return to full grip strength (upper extremity bites) and ability to weight bear (lower extremity bites). <u>Results</u>: 23 patients were envenomated during the study period with 1 patient excluded. Of the 22 patients, mean age 35.8 [range 3–79] years, 14 involved the upper extremity and 8 involved the lower extremity. Eleven received CroFab (SSS = 5–8), 4 received Wyeth (SSS = 5–8), and 8 received no antivenin (grades 1–3). Mean time to return of full grip strength was 29 days in the CroFab group (N=4; 7 lost to follow-up), 17 days in the Wyeth group (N=3; none lost to follow-up), and 33 days (N=5; 3 lost to follow-up) in the no treatment group. Weight bearing returned at a mean of 38 days (N=2; cone lost to follow-up) in the no treatment group. Conclusions: The time for RSB patients to return to a functional baseline is ~1 month. This study is limited by the small total patients and the number lost to follow-up.

140. PROSPECTIVE STUDY OF CENTIPEDE STINGS IN AUSTRALIA

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<u>Background</u>: Centipedes are readily identifiable arthropods that occur worldwide. There are limited reports of definite bites by centipedes with expert identification, which are required for attribution of particular clinical effects to different species. <u>Methods</u>: Subjects were recruited prospectively from December 2000 to March 2002 from calls to a state poisons information center. Subjects were included if they had a definite bite. Collected centipedes were identified by an expert. All subjects were followed until clinical effects had resolved. <u>Results</u>: There were 48 centipede exposures; 3 were centipede ingestions and no adverse effects were reported from these. Of 45 definite centipede bites, the centipede was obtained and formally identified in 15 cases. Identified centipedes were from the genera *Scolopendra* (5), *Cormocephalus* (7), and *Ethmostigmus* (3). Of these 15 bites, 14 occurred distally (hands or feet). One patient was bitten in the axillary region. Pain occurred in 14 cases and was severe in 5 patients. Redness/red mark occurred in 53%, swelling/raised area in 47%, and itchiness in 20%. No systemic effects were reported. More severe effects were reported with bites by *Ethmostigmus* spp. and *Scolopendra* spp. 60% of bites occurred indoors and 53% occurred at night. Treatment consisted of supportive measures such as ice pack and simple analgesia. Four patients reported relief of pain with immersion of the bite area in hot water. Similar effects were reported in 30 definite centipede bites where centipede was not collected for identification. <u>Conclusions</u>: Australian centipede bites. The genus *Scolopendra* occurs worldwide and the results may have international applicability.

141. SUNBURST TARANTULA (PTERINOCHILUS MURINUS) ENVENOMATION

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Case Report: The patient was a 29 year-old male who was bitten by his pet Sunburst Tarantula (Pterinochilus murinus) on the second finger of his left hand 28 hours prior to arrival at an emergency department. The bite had been forceful and the tarantula attached for at least a full second. He complained of numbress and aching in his arm, body aches, and leg cramping. No puncture wound was seen and there was no erythema or edema. The patient was admitted for 24 hours and treated with IV fluids, benzodiazepines and opioids with good relief and was discharged on oral opioids. One-week post bite, he developed increasing dysesthesia, pain, and swelling in the bitten hand, without erythema, or heat. He also developed worsening generalized myalgias and arthralgias. There was no rash or fever. These symptoms were selfmedicated with oxycontin and Ativan[®] and resolved over 2 weeks. Discussion: The Sunburst Tarantula (*P. murinus*) is a venomous, east-central African variety. They are very fast moving when attacking. Its venom has greater mammalian toxicity than North American Tarantulas. The venom contains multiple components, the most toxic of which is a polypeptide of molecular weight 10,500 Daltons that has a subcutaneous LD50 in mice of 0.1 mg/kg, with death due to respiratory paralysis. Typical symptoms of *P. murinus* envenomation in humans are local numbress and pain, that usually resolve within 24-48 hours but may persist for up to 2-3 weeks. There are no reported fatalities. Treatment is symptomatic and supportive. There is no antivenom. Conclusion: This patient had recurrent and prolonged symptoms from a *P. murinus* envenomation, which may have been secondary to prolonged venom effects or to a Type III hypersensitivity ("serum sickness") reaction.

142. NEUROTOXIC-INDUCED RESPIRATORY DETERIORATION REFRACTORY TO CROFAB AFTER RATTLESNAKE ENVENOMATION

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<u>Background</u>: Neurotoxicity from rattlesnake envenomation is reported in certain Crotaline species. In many instances, antivenom therapy that halts progression of cytotoxicity and hemotoxicity also effectively treats neurotoxic venom effects. <u>Case Report</u>: A 50 year-old male presented 45 min after a rattlesnake envenomation to the left ring finger. Upon presentation, he had swelling and pain to the hand and complained of dyspnea and dysphonia. Fasciculations were seen in the face, tongue, neck, trunk, and arms. The patient initially received treatment with six vials of crotaline Fab (CroFab) but continued to develop respiratory difficulty necessitating endotracheal intubation. His respiratory insufficiency appeared related to respiratory muscle incoordination instead of pure weakness since extremity motor function remained intact. Due to progression of local symptoms and fasciculations, an additional 14 vials of CroFab were administered over the next 12 hours. Local edema progression halted at the level of the mid-bicep, hematologic parameters stabilized, but diffuse fasciculations involving the entire body worsened. Although no evidence of tissue necrosis or compartment syndrome were ever present, CPK rose to 19,415 IU/L requiring vecuronium to stop fasciculations. <u>Conclusion</u>: We report a case of severe fasciculations following rattlesnake envenomation necessitating mechanical ventilation and subsequent neuromuscular blockade. Although treatment with CroFab adequately halted progression of local swelling, the neurotoxic venom effects remained refractory to antivenom therapy.

143. RATTLESNAKE ENVENOMATION TO THE FACE OF AN INFANT

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<u>Background</u>: Mortality from rattlesnake envenomation in the United States is rare. Despite approximately 8000 crotaline (pit vipers) bites per year, it is estimated that only 10–15 deaths occur. In addition to direct intravascular envenomation and anaphylaxis, bites to the head and neck likely account for these rare fatalities. We report a pediatric case of severe facial envenomation requiring emergent intubation and antivenom administration. Fortunately, no long-term clinical or cosmetic sequelae occurred. Case Report: A 14 month-old female was envenomated by a Southern Pacific rattlesnake

(*Crotalus viridis helleri*) above the right upper lip two centimeters lateral to the philtrum while playing in her backyard. Rapid swelling and ecchymosis developed, and the patient was airlifted to the nearest pediatric tertiary care hospital. Within three hours stridorous respirations complicated by significant facial and oropharyngeal edema necessitated emergent orotracheal intubation. A total of fourteen vials of CroFab (Crotalidae Polyvalent Immune Fab) were administered over the next 24 hours. The child gradually improved and was successfully extubated five days later. A three-month follow-up demonstrated no significant facial abnormalities. <u>Conclusion</u>: Crotaline bites to the head and neck have the potential for significant swelling and airway compromise. Facial bites, anaphylaxis, or rare intravascular envenomation likely account for the vast majority of fatalities from rattlesnake envenomation. As evidenced by this case, early intubation may be required to maintain airway patency.

144. INTRAVENOUS INJECTION OF RATTLESNAKE VENOM

Blair HW, Ramsey RP, Morgan DL. Central Texas Poison Center, Scott & White Hospital, A&M University Health Science Center, Temple, Texas, USA.

<u>Background</u>: Intentional intravenous (IV) injection of various substances such as lamp oil, mercury, cyanide, arsenic, sublimate solution, and bleach have been reported to the Toxic Exposure Surveillance System. A thorough literature search revealed three reported cases of suspected self-injected IV rattlesnake envenomation, however we report the only known case of intentional IV injection. <u>Case Report</u>: A 14 year-old male found vomiting and confused on a roadway stated to EMS that he "injected snake into myself." It was later determined he had milked a rattlesnake and then injected the contents with a syringe into his right antecubital vein in a suicide attempt. Intense pain immediately followed, and patient sought help. His right antecubital fossa had an area of erythema with multiple pinpoint petechiae. He was confused, tachycardic, unable to protect his airway, and a blood pressure could not be obtained. He was intubated, begun on a dopamine drip, and initially received 4 vials of CroFab[®] antivenom. Initial laboratory tests showed an elevated white blood cell count, elevated INR, and decreased platelets. The patient's hospital course was complicated by a gastrointestinal bleed. He received a total of 22 vials of CroFab, 2 units of fresh frozen plasma, 6 units of platelets, IV fluids, and supportive care. The laboratory values returned to near normal within a few days. The patient was discharged on the 5th day with a normal physical examination. <u>Conclusion</u>: Although other reports of suspected IV rattlesnake envenomation were found in the literature, we report the first known self-inflicted injection. Despite the rapid onset of life-threatening symptoms, this patient was managed successfully with antivenin and supportive care.

145. REPTILE ENVENOMATION 20-YEAR MORTALITY AS REPORTED BY US MEDICAL EXAMINERS

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Introduction: Accurate reptile envenomation mortality data in the United States is unknown, as reporting of envenomations is not mandatory to public health authorities. However, states do require completion of death certificates. Using data from the National Vital Statistics System (NVSS), a cooperative agreement between the National Center for Health Statistics and all 50 states and the District of Columbia in which fatalities are reported in a standard format, NVSS deaths from 1979 to 1998 were analyzed. Methods: Records of deaths from reptile envenomation were selected using ICD-9 code E905.0. Rates were calculated using United States Census population estimates (1978–1997) and age adjusted rates based on the 2000 US standard population. Results: During the reporting period, NVSS identified 97 deaths from venomous reptiles. Texas (17), Florida (14), and Georgia (12) reported the most fatalities (44% of fatalities, 16% of US 2000 population). No deaths were reported from 24 states or the District of Columbia. Of the 97 decedents, 88 (91%) were White (74 males and 14 females), and 7 (7%) were Black (1 male and 6 females). One male and one female were categorized as "Other" race. White males had the highest incidence of death by venomous reptiles, accounting for 76% of all fatalities while comprising only 41% of the US population. The age category comprising "Men 25-34 years old" had the most deaths, 19 of the 97 fatalities (19.6% of deaths but 7.1% of the US 2000 population). Conclusion: According to NVSS data during 1979–1998, <100 deaths by venomous reptile bites were reported nationwide. White male Southerners appear to be at greatest risk. This finding of a specific high risk population may require further investigation and public health intervention.



146. EFFECTIVENESS OF DELAYED USE OF CROTALIDAE POLYVALENT IMMUNE FAB (OVINE) ANTIVENOM

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<u>Background</u>: Crotalidae polyvalent immune Fab (ovine) (FabAV, CroFab) is approved for mild and moderate Crotalinae snake envenomation. The manufacturer recommends use within six hours of envenomation. Published post-marketing retrospective reports describe its use up to nine hours after envenomation. We describe a case of effective use of FabAV 52 hours after envenomation with resultant dramatic correction of coagulopathy and improvement of local symptoms. <u>Case Report</u>: A 45-year-old male was envenomated to his right hand by a Midget Faded rattlesnake (*C. concolor*). He developed vomiting and mild hypotension, followed by progressive local edema, ecchymoses and coagulopathy. At 48 h post-envenomation, his INR was 2.4, fibrinogen <50 mg/dL, and fibrin split products (FSP) >150 mcg/mL. He also had marked edema, pain, and ecchymoses extending from his hand to his chest and upper back. He received the initial six vials of FabAV at 52 hours post-envenomation, followed by two vials every 6 hours for a total of 12 vials. His coagulopathy improved significantly shortly after initiation of the infusion (INR 1.1, fibrinogen 139, and FSP 61). At discharge, 9 days later, his PT, INR, and fibrinogen were normal. In addition, he described his pain as decreased, had much improved edema and pain after FabAV infusion and had no further progression of his local symptoms. <u>Conclusion</u>: The prolonged progressive nature of this envenomation, which then promptly improved following FabAV administration suggests FabAV is effective after the 6 hour period recommended by the manufacturer.

147. PEDIATRIC RATTLESNAKE ENVENOMATION WITH NEUROTOXICITY REFRACTORY TO TREATMENT WITH CROTALINE FAB ANTIVENOM

Goto CS, Gutglass DJ, Richardson WH, Ly BT, Offerman SR, Clark RF. Division of Medical Toxicology, University of California San Diego; Children's Hospital and Health Center, USA.

<u>Background</u>: Successful treatment of crotaline-induced neurotoxicity with crotaline Fab antivenom (CroFab) has been previously reported. However, the neurotoxicity of certain rattlesnake species may be refractory to treatment with CroFab, which consists of ovine Fab fragments produced against the venom of four crotaline species: *Crotalus scutulatus scutulatus* (Mojave rattlesnake), *C. atrox* (Western diamondback), *C. adamanteus* (Eastern diamondback), and *Agkistrodon piscivorus* (Cottonmouth). <u>Case Report</u>: A 9-year-old boy was envenomated in the left hand by a rattlesnake. He presented to the emergency department within 1 hour and was noted to have pain and swelling to the mid-forearm with fasciculations of the tongue, face, and upper extremities. The initial platelet count was 35,000/mm³. The patient was treated with six vials of CroFab over one hour IV. The local edema continued to advance above the elbow although at a slower rate of progression, the platelet count increased to 170,000/mm³, but the fasciculations persisted. The patient received four more vials of CroFab. Local edema progression halted at the level of the mid-bicep, and the hematologic parameters remained normal. No additional antivenom was administered. Fasciculations persisted for 2 days then resolved spontaneously. <u>Conclusion</u>: We report a case of pediatric crotaline envenomation in which the hematologic and local tissue toxicity were effectively treated with CroFab, but the neurotoxic effects remained refractory. Although the rattlesnake in this case report was not identified, the most common envenomation in this area is *C. viridis helleri* (Southern Pacific rattlesnake). The neurotoxicity of some crotaline species may not respond to treatment with CroFab.

148. HOUSEHOLD PRODUCT LABELING—INADEQUACIES ABOUND

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<u>Background</u>: Current commercial household product labeling lacks consistency. Some products contain questionable or incomplete first-aid advice, while others fail to list ingredients. This observational study was designed to examine the quality of common household product labeling. <u>Methods</u>: A random sampling of commercial household products was gathered from two home improvement and two grocery stores. 118 unique products were identified and consisted of:

automotive, paint/finishing products, pesticides/herbicides and cleaners/disinfectants. Their labels were examined for: listed ingredients, route-specific first-aid advice, product use and formulation type. <u>Results</u>: 16 products failed to list any ingredients, while 10 lacked any first-aid advice. Specific advice for ingestions (with number of instances): contact poison center/physician 56, dilute 55, no vomiting 47, seek medical attention 14, induce vomiting 6 (with finger 4), milk of magnesia 3, egg whites 3, gelatin 2, vegetable oil 1. For ocular exposures, 90 products advised flushing with water, while the remaining 28 listed no advice. Of the latter, 17 were in a liquid, aerosol or pump spray form. For dermal exposures, 65 products advised washing the affected area, while 53 lacked any advice. Of the latter, 4 contained strong corrosives and 3 petroleum distillates. For inhalation exposures, 22 products listed advice, while 96 did not. Of the 22 that did, 2 products were pellets and 2 were granules. Of the 96 that did not, 11 were aerosols. <u>Conclusion</u>: The Federal Hazardous Substance Act is not detailed enough to ensure consistency and accuracy in the labeling of commercial household products. Further evaluation into this area should be considered in order to improve consumer safety.

149. THERAPEUTIC ERRORS WITH CALCIUM CHANNEL BLOCKERS. WHO? WHAT? WHERE? AND WHY?

Manoguerra AS, Cantrell FL. California Poison Control System, San Diego, California, USA.

<u>Objective</u>: Calcium channel blocking drugs (CCB) are well known to have a narrow therapeutic index and therapeutic errors can produce serious and potentially fatal outcomes. An understanding of the circumstances in which these errors occur could be helpful in attempting to minimize them. <u>Methods</u>: A retrospective review of therapeutic errors with CCB reported to our poison system from 1999 to 2001 was conducted to identify the circumstances in which these errors occur. <u>Results</u>: 877 cases were identified. In 704 cases, the person took their own CCB and in 123 cases the CCB belonged to another person. Except for 14 cases, three with the victim's own CCB and 11 with another person's CCB, home was the site of the exposure (813 cases). The victim was female in 596 cases and male in 231. The ingestion of an excessive amount of the victim's own CCB occurred most commonly in the 70th decade of age while the ingestion of another's CCB occurred most frequently in the 50th decade. In 540 cases, the victim took a double dose of their own CCB. In 53 cases, more than double their prescribed dose was taken. Other common errors included taking a spouse's medications by mistake (72 cases) and mistaking the CCB for another medication (65 cases). Therapeutic errors with CCB resulted in 276 emergency department (ED) visits. <u>Conclusions</u>: Calcium channel blocking drugs are commonly involved in therapeutic errors and result in a large number of ED visits. National efforts to reduce medication errors have focused on errors that occur in health facilities. This review demonstrates that in order to control medication errors with CCB, there must also be substantial efforts to reduce those that occur in the home.

150. PHARMACY STUDENTS AS POISON PREVENTION EDUCATORS

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<u>Objective</u>: To increase outreach education activities with current education staff and educate future pharmacists. <u>Methods</u>: An elective course was created for pharmacy students to teach poison prevention education, to promote poison prevention strategies and to conduct poison prevention programs for the public. The class was offered during fall and spring semesters. The format was 3 two-hour classroom sessions followed by 10 or 20 hours of community activity for one or two credit hours of outreach education. The first classroom session was devoted to an overview of poisonings, the role of poison centers and poison prevention strategies and the second session to the poison center's "Train-the-Trainer" program. During the third classroom session students were required to give a 10 min presentation using education concepts learned in the previous two sessions. Lesson plans were provided to students. The class was limited to 20 participants. <u>Results</u>: Twenty students registered for the elective the first semester it was offered. The majority of students (75%) were in their first year of the pharmacy program and took the class for two credits (85%). During the first semester, the students attended 29 health fairs and gave 31 presentations. Over 56,000 individuals attended these events. This compares to 16 health fairs, three presentations and just under 22,000 attendees during the same 3-month period (September, October, November) one year before the elective was offered. Additional resources required to support the class included approximately 100 hours of faculty time and \$150 for materials each semester. <u>Conclusion</u>: An elective for pharmacy students allowed the PCC to almost double its outreach education without increasing staff while educating future pharmacists.



151. IS MASS-MAILING AN EFFECTIVE FORM OF PASSIVE POISON CENTER AWARENESS ENHANCEMENT?

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<u>Background</u>: Poison information center awareness may be facilitated actively via formal programs that target specific audiences or passively through the dissemination of information such as stickers and brochures. The purpose of this project was to determine whether the massive distribution of poison center telephone number (Poison Help 800-222-1222) awareness stickers via a direct mail campaign, as a passive education technique, enhanced poison center awareness and was cost-effective. <u>Methods</u>: A regional ambulance service conducts an annual membership renewal/solicitation drive via the mail to all residents within its service area. A sheet of Poison Help stickers was inserted in each ambulance service recruitment envelope and mass-mailed in three separate mailings over a four week period. Call volumes from all zip codes that received the mailing were compared to an identical benchmark time period from the previous year. <u>Results</u>: Poison Help stickers were mailed to 51% of households of a single county with a population of 368,983 at a direct expense of \$4477, which included the cost of the stickers and the fee charged by the mail-order fulfillment company to insert the stickers into each envelope. Analysis of call volume data over the study period revealed that call volume decreased by 1.3% during the study period. <u>Conclusion</u>: A mass-mail campaign to enhance poison center awareness failed to increase poison center call volume from the targeted county and therefore, cannot be construed as being cost-effective.

152. ANIMAL EXPOSURES AND THEIR IMPLICATIONS FOR ONE POISON CENTER

Tarantino ML, Vajani M, Parramore CS. Georgia Poison Center, Atlanta, Georgia, USA.

<u>Objective</u>: To describe reported animal poisonings and evaluate the need for more comprehensive services to the region. <u>Background</u>: 45% of American households now have one or more pets. The reports of human exposures received by this regional PC from 1/1/92 and 12/31/2002 has nearly doubled. Reports of animal exposures increased threefold for the same time period, from 2559 to 8359 cases. <u>Methods</u>: Animal exposure data (N=8359) from 1/1/02 to 12/31/02 were analyzed to identify reason for exposure, animal species, caller's relationship, outcome and case fatality rate. <u>Results</u>: The majority, 84.9% of the cases involved dogs, 13.2% cats, and 1.9% others, including birds and livestock. The most common reasons for the animal exposure cases are unintentional general (87.6%), unintentional environmental (2.7%) and others (9.7%) including unintentional bite/sting and adverse reactions. Death occurred in 47 of 8359 cases reported (case fatality rate = 5.6/1000) considerably higher than the human case fatality rate of 0.47/1000. This may reflect a reporting bias toward the most severe cases. Pet owners accounted for more than 75% of the calls and the remainder were others including veterinarians. <u>Conclusion</u>: PCs may be increasingly called upon to provide traditional PC service to animal poisonings wictims. Veterinary toxicology should be a part of the PC continuing education programs. Case fatality rates reflect the need for preventive measures, early intervention and treatment. Determining the nature and extent of unreported animal poisonings might help in developing appropriate prevention strategies. Identifying additional monetary and educational resources, and raising awareness of PC services are essential elements of this growing issue.

153. EFFECT OF EDUCATION ON CHILDREN AGE 0–6 IN A SMALL MIDWESTERN CITY: MORE CALLS TO REGIONAL POISON CENTER; FEWER CALLS ORIGINATING FROM HEALTH CARE FACILITIES (HCF)

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Background: The decrease in the number of poison centers over the past several years has caused the surviving centers to serve larger geographic areas. It is not feasible for PCC educators to travel the long-distances needed to cover the remote

areas. A Midwestern PCC implemented a network of satellite centers throughout the state, with subcontracted hospitalbased educators providing poison prevention education in the counties surrounding their institutions. This has allowed for poison prevention education in areas of the state that previously have not had an organized education effort. <u>Methods</u>: A satellite center was started at a hospital serving a county with a population of 188,951. In 2002, 87 outreach events were conducted, reaching 5601 people. The call data for children age 0–6 was compared for the year before the implementation of the satellite center (2001) and after the first year of operation (2002). <u>Results</u>: Total exposure calls increased from 760 to 913 (+20%); exposure calls managed at home increased from 683 to 840 (+22%); and calls originating from HCF decreased from 47 to 31 (-34%). Referrals from the PCC to the HCF increased from 29 to 35; however, there was no change in percentage from the previous year as the number of home calls increased substantially. <u>Conclusion</u>: Satellite education centers providing focused education can substantially raise awareness of poison center services. This may lead to increased calls to the poison center and decreased utilization of emergency services for patients in the 0–6 age group.

154. PARACETAMOL (ACETAMINOPHEN) OVERDOSE IN THE UNITED KINGDOM; WHERE DO PATIENTS OBTAIN THE TABLETS?

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Background: Paracetamol (Acetaminophen) is the most common drug ingested in overdose (OD) in the UK. In 1998 government legislation was passed limiting the number of 500 mg paracetamol tablets legally able to be sold over the counter to 32 in a pharmacy and to 16 in non-pharmacy outlets (e.g., general store, gas station, etc.). Methods: A prospective observational study of adult patients presenting to the Emergency Department (ED) following drug OD. In addition to baseline patient data, the source of paracetamol (pharmacy, non-pharmacy outlets, prescription), amount of paracetamol obtained, and the reason for obtaining paracetamol (medicinal or OD) were recorded. Results: 107 patients ingested some form of paracetamol, of these 77 (72%) had ingested more than 16 paracetamol (500 mg) tablets, a potentially lethal dose. 45% (35) of the patients with a potentially lethal ingestion purchased the tablets specifically for the OD on that day, 31% (24) had the tablets at home from previous purchases or prescriptions, and 24% (18) had obtained the tablets via a recent (less than 1 month) physician's prescription. Of those who had specifically purchased more than 16 tablets for the OD (n = 35), 15 purchased them from multiple non-pharmacy outlets on that day, 15 from a single non-pharmacy outlet (which the 1998 legislation had made illegal), and 5 from a pharmacy (one pharmacy supplied more than 32 tablets). Conclusion: Despite the legislation, 72% of patients with paracetamol ODs still ingest more than 16 tablets. They are able to do so either because they have more than 16 tablets in the home, they bypass the legislative controls by purchasing the tablets from multiple outlets, or in a significant number of cases the legislation itself is ignored.

155. HIGH RISK OF PARAFFIN EXPOSURE IN ORTHODOX JEWISH CHILDREN

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<u>Background</u>: In observance of the Sabbath and other religious holidays many Orthodox Jews maintain a burning lamp using paraffin lamp oil as fuel. Unintentional pediatric exposure to this hydrocarbon, typically by ingestion, carries risk of aspiration with subsequent pneumonitis. This investigation was prompted by an apparent increase of paraffin lamp oil exposures during the Jewish Sabbath, from sunset Friday until sunset Saturday, noted by the staff of our regional poison control center. <u>Methods</u>: In this IRB-exempted investigation, we retrospectively reviewed all exposures to paraffin lamp oil occurring in our large urban city in children under 18 years old reported to our regional poison control center between January 1, 2000 and February 1, 2003. Reports were investigated to ascertain the frequency of occurrence on the Jewish Sabbath and religious holidays. Caregivers of involved children were surveyed by telephone to determine the childs religion and circumstances of exposure. Results: During these 25 months, 45 cases met inclusion criteria, and all were



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ingestions. Orthodox Jews accounted for 32 cases (71%), four cases (9%) occurred in children who were not Orthodox Jews, and demographic data was unavailable in nine cases (20%). Twenty-four cases (53%) occurred within 10 hours prior to or during the Jewish Sabbath or religious holidays. <u>Conclusion</u>: The relative risk of Orthodox Jewish children to ingest paraffin lamp oil, calculated using census data, is 829 times that of other children. Public health authorities and caregivers of Orthodox Jewish children should be cognoscente of this phenomenon. Educational efforts directed toward these preventable poison exposures are warranted.

156. MEDICATION ERROR LEADS TO PSEUDOTUBERCULOSIS EPIDEMIC

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<u>Background</u>: National attention on medication errors has lead to increased investigations into processes and common sources of error to prevent recurrence. We report the discovery of a widespread medication error which went unnoticed until adverse events occurred in 30 health care workers (HCW). <u>Methods</u>: An occupational health nurse screened 30 HCWs who had been exposed to a hospitalized patient with tuberculosis. Each HCW was advised of the usual post exposure screening protocol: intradermal injection with 0.1 cc (5TU) Tubersol purified protein derivative (PPD). <u>Results</u>: All 30 HCWs reported a large, tender, and pruritic induration and strongly "positive" (>10 mm induration) when read at 48–72 hours. Because of the perceived high conversion rate, the occupational health nurse re-examined the "PPD" bottle but found it contained tetanus toxoid (Td). The vials were both kept in the refrigerator, were the same size, with similar labeling and made by the same manufacturer. Several HCWs continued to report painful induration, pruritis and hyperpigmentation at the site of injection and, in some cases, up to 6 months later. Subsequent PPD testing in all 30 HCWs were negative. In contrast to the relatively benign effects of inadvertent intradermal tetanus toxoid, similar error mechanisms have lead to fatalities when pancuronium was injected instead of influenza vaccine. <u>Conclusion</u>: Medication errors with look-a-like drug names or medication bottle labels will continue to occur. A Failure Mode Evaluation Analysis (FMEA) was performed to identify and implement prevention measures for further similar occurrences in this institution. Increased education and heightened awareness of this potential pitfall may decrease the incidence of such errors.

157. HOSPITAL ANTIDOTE STOCKING: ARE WE READY?

Ryan ML, Arnold TC. Louisiana Drug and Poison Information Center, University of Louisiana, Monroe, Louisiana, USA.

Background: It has been shown that hospital antidote stocking is often inadequate in that supplies of a particular antidote are not sufficient to treat even one severely poisoned patient. We compared antidote stocking in hospitals in our state before and after the terrorist attacks of 9/11. Our aim was to ascertain whether the current state of heightened emergency preparedness had affected hospital stockings of certain antidotes which might be used to treat victims of a terrorist attack. Methods: The Louisiana Drug and Poison Information Center conducts an annual survey of antidote stocking in all hospitals in the state with emergency departments. This survey includes antidotes that would be needed to manage patients exposed in a chemical terrorism event. Our goal was to determine if hospitals had increased antidote stocking in response to national bioterrorism awareness and federal requests to do so. The standard survey instrument which had been used for the past three years was faxed to the hospital pharmacy department after an initial call explaining the reason for the inquiry and requesting participation. Results: Eighty seven hospitals returning surveys in both July 2001 and April 2003 were included. In the 2001 survey, 48 hospitals (55%) reported having no pralidoxime on hand. In the 2003 survey, 44 hospitals (50.5%) reported no pralidoxime on hand. In 2001, twenty-nine hospitals (33%) had no cyanide antidote kits stocked. In 2003, 27 hospitals (31%) reported the same. In 2001, all hospitals stocked atropine with a range of 25-500 mg total on hand. In 2003, again all hospitals reported atropine on hand with a range of 10-652 mg. Conclusion: Our findings reveal that hospitals have not significantly increased the amounts of antidotes which would be necessary in the event of a chemical terrorism event in our state.

158. A NATION-WIDE CONSULTATIVE NETWORK BETWEEN MEDICAL TOXICOLOGY FELLOW-SHIP PROGRAMS AND ATSDR REGIONAL OFFICES

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Background: The Agency for Toxic Substances and Disease Registry (ATSDR) is a US federal agency whose mission statement is "To Protect America's Health from Toxic Exposures." Since 1999 the American College of Medical Toxicology (ACMT) and ATSDR have had a cooperative agreement to promote environmental toxicology education for medical toxicologists. American College of Medical Toxicology has recently developed a network that would provide the 10 ATSDR regional offices with consultation from medical toxicology fellowships. Methods: Eight medical toxicologists serving as regional directors have met with ATSDR regional staff to develop a communications infrastructure between the ATSDR regional offices and medical toxicology fellowship programs. Results: A network linking the 21 ACGME approved toxicology fellowships and the 10 ATSDR regional offices has been established. ATSDR regional staff can now utilize the network to have medical toxicologists assist in telephone consultations to other health care providers, review biomonitoring data, provide chemical specific consultative advice, and participate in emergency response to acute environmental chemical exposures. Medical toxicologists will also respond to ATSDRs request for educational outreach, public information session support, and terrorism preparedness training. A database will record all the activity of this consultative network. Conclusion: A unique network connecting medical toxicology fellowship training programs to a federal agency that responds to environmental chemical exposures has been developed. This network will enhance the training of medical toxicologists in environmental health issues, and may increase the future role of medical toxicology in the federal public health infrastructure.

159. OVER-THE-COUNTER SALES OF ANTIBIOTICS IN ETHNIC STORES—A CAUTION FOR ANTIBIOTIC MISUSE IN ASIAN POPULATION

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<u>Background</u>: Amid the growing availability of Asia imported products in the United States, the unmonitored sales and misuse of antibiotics may threaten infection control in Asian communities. This pilot study is an effort to investigate the widespread unauthorized sale and potential misuse of antibiotics in Asian communities. An assessment of the variety and availability of OTC antibiotics in Asian communities was undertaken. <u>Method</u>: A surveillance study was conducted at Asian food markets in various Chinese communities in the New York area. Samples of antibiotic products were noted and collected. A list consists of product name, strength, package size, indication, directions for use, and price was compiled for quick reference. <u>Results</u>: Over 15 specific types of Asia imported antibiotic products were noted on open shelves and purchased without a prescription. Various brands of oral, topical and ophthalmic preparations available included penicillins, cephalosporins, tetracyclines, macrolides, quinolones, sulfa drugs and various others not available in the United States. Average cost per multi-dose package ranges from \$1.50 to \$8.00. Most of these products listed indications and directions for use only in the Chinese language. <u>Conclusion</u>: Misuse of antibiotic may lead to a decline in effective infection control. Illegal sales and misuse of imported antibiotics that lacks quality assurance, threatens not only the Asian community, but other ethnic communities as well. In addition to more stringent regulatory efforts, cross-cultural education is urgently needed to address issues concerning indiscretionary use of antibiotics.

160. HERBAL CONTENTS IN HIP DRINKS NOT SO SOFT

Kang-Yum E, Yang D, Chiang W, McGuigan M, Caraccio T. HerbWatch—Long Island Regional Poison and Drug Information Center, Mineola, New York, USA.

<u>Background</u>: As herbalism takes on a global trend, marketers are aggressively targeting health conscious consumers. Despite the natural sources of herbs, misuse and overuse can lead to potential health risks. This study is an effort to raise the awareness of health care professionals and consumers of the potential health risks associated with excessive consumption



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of soft drinks that contains certain herbs. The study will assess the variety of common herbal drinks and its herbal contents available in retail stores. <u>Methods</u>: A surveillance study was conducted at retail stores for soft drinks that contains herbs. A list consisting of product name, herbal contents, potential effects, and possible drug-herb interactions were compiled. It will be presented in a handy reference form. <u>Results</u>: A variety of Snapple, Arizona, Fusion herbal soft drink were noted to be more enticing in their packaging and labeling than their non-herbal brands. Most products have labels specifying the quantity of their herbal contents, but no cautionary statements were included. Some of the more common herbs listed in products were: Siberian ginseng, American ginseng, Gingko biloba, Echinacea, Guarana, Chamomile, Yerba, Gotu Kola, Licorice, Damiana, and Yohimbe. <u>Conclusions</u>: Tasty soft drinks with added herbs are becoming popular and are marketed as healthy alternatives. Although the herbal contents in a single serving of these soft drinks may not pose a problem, several servings per day typically represent the daily-recommended dosage for these herbs. Potential adverse effects and drug-herb interactions may be expected with frequent consumptions of certain herbal drinks.

161. COMMUNICATIONS STRATEGY FOR HEALTH EDUCATORS WORKING WITH LOW-ENGLISH PROFICIENCY LOW-INCOME HISPANIC CONSUMERS

Simeonov IM, Heard SE. California Poison Control System, University of California, San Francisco, California, USA.

<u>Objective</u>: Employ generative and evaluative research to aid health educators in expressing the critical attributes and perceptions of PCC services. Use knowledge gained to guide the development and design framework for new targeted consumer products that address the communication needs of low-English proficiency and low-income Hispanic parents regarding poison exposure and treatment. Develop consumer-focused PCC messages and products. <u>Methods</u>: Projective tools, such as image collage, were employed to aid educators in expressing critical attributes and perceptions of PCC services. Demographic information and research of successful business benchmarks yielded insight on Hispanic consumers. Knowledge gained guided development of new content and product design. New concepts were developed, tested and refined with Hispanic consumers over 4 months. Qualitative (two focus groups, 16 participants) and quantitative (128 face-to-face interviews) were conducted with low-income monolingual Hispanic parents. <u>Results</u>: Anxiety and concern of medical service with no direct examination and difficulties communicating in English precluded use. After detailed service description, participants indicated they considered PCCs a valuable and useful service, but required a higher degree of detail and reassurance about the call-in process. Feedback guided content and design for three Spanish-language products. Attitudinal and behavioral information on poison exposure, actual and potential PCC use provided baseline for evaluating effectiveness.

162. A DOCUMENTED ASSOCIATION OF POISON CONTROL CENTER MEDIA INTERACTIONS AND CARBON MONOXIDE CALLS

Bayer M, Hanoian A, Caperino Crean L. Connecticut Poison Control Center, University of Connecticut Health Center, Farmington, Connecticut, USA.

<u>Background</u>: This study examines the relationship between carbon monoxide (CO) calls to our poison center and media interactions. CSPIs noted increased calls to the PC regarding CO and increased media interactions regarding CO in 2002–2003 as compared to 2001–2002. <u>Methods</u>: A time frame of October (10/26) through March (3/13) was chosen based on dates of media interactions and peak seasonal CO exposure time. Media interactions included newspaper articles and radio and television interviews where the topic was CO. Cases were queried from TOXICALL. <u>Results</u>: 3 media interactions focusing on CO occurred between 10/26/01 and 3/13/02. One hundred and thirty-two total cases were recorded (130 exposures and 2 informational calls) during that time frame. The following year, 13 media interactions involving CO occurred between 10/26/02 and 3/13/03. One hundred and ninty nine total cases were recorded (195 exposures and 4 informational calls) during the same period of time. <u>Conclusion</u>: The poison center is a valuable resource for predicting trends and raising public awareness about seasonal topics. Reporting of CO cases increased during the 2002–2003 CO season as compared to the 2001–2002 CO season. The increase in CO cases occurred in association with a marked increase in media information on CO generated by our poison center. The association demonstrates that the expertise offered by the poison center plays an important role in the public's awareness of the dangers of CO poisoning.

163. CAN AAPCC TESS BE USED TO DETERMINE THE IMPACT OF FOCUSED PUBLIC POISON EDUCATION EFFORTS?

Krenzelok EP, Watson WA. Pittsburgh Poison Center, Children's Hospital of Pittsburgh, Schools of Pharmacy and Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania, USA; American Association of Poison Control Centers, Washington, District of Columbia, USA.

<u>Objective</u>: Poison prevention education is one of the poison center's primary missions. A continuing question is the validation of poison prevention activities. The purpose of this project was to determine if AAPCC TESS data could be used to determine the impact of poison prevention education endeavors by examining parameters such as call volume and patient outcome. <u>Methods</u>: AAPCC TESS was queried electronically for a 10 year period (1993–2002) to identify all unintentional human exposures to carbon monoxide in a single populous county. Exposures to CO were corrected in concordance with changes in total human exposure call volume. Data were compared prior to, during and after implementation of an aggressive CO poisoning prevention campaign that included public service announcements, media engagements and the dissemination of brochures. Outcomes were measured by comparing the sum of "no effect" and "minor effect" outcomes. <u>Results</u>: A mean of 261.2 (SD ± 81.4) CO exposures occurred during each of the 10 years. These exposures represented a mean of 1.39% (SD ± 0.24%) of all human exposures. A mean of 39.58% (SD ± 9.79%) of the patients experienced a "no effect" or "minor effect" outcome. There were no impact correlations between call volume and patient outcomes when compared to the poison education activities. <u>Conclusion</u>: Using limited measurement parameters, AAPCC TESS data failed to demonstrate any impact of the CO poisoning prevention educational program.

164. PATIENT SAFETY—FROM ADVERSE DRUG EVENTS TO HOSPITAL POLICY CHANGE

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<u>Background or Objective</u>: Despite the increased reporting of adverse drug events (ADE) altering medical outcomes within the United States, it is difficult to alter health care policy. Just as early goal-directed therapy (EGDT) has been shown beneficial in the management of acute illnesses, so can goal-directed therapy be utilized in changing hospital policy to help prevent adverse outcomes from ADE. This EGDT is reviewed in an algorithmic fashion from identification of a single case, to change in hospital policy, and education of the stakeholders. <u>Case Report</u>: A 25-year-old male presented after acute head injury that required emergency endotracheal intubation. Because of agitation related to the head injury, sedation was achieved with propofol in doses up to 280 mcg/kg/min. Patient developed clinical evidence of propofol infusion syndrome and died several days later. Initial investigation of this adverse drug event was made by the pharmacy clinical specialist. This was forwarded to the Pharmacy and Therapeutics Committee as a high alert event. Parallel courses were taken through the hospital's Adverse Event Reporting System and medical staff committees to propose specific guidelines utilized at both the physician and nursing levels. This limits propofol infusions to not more than 85 mcg/kg/min. Within 90 days from the ADE, hospital and medical staff committees had agreed to the utilization of specific propofol usage guidelines, and educational efforts were completed. <u>Conclusions</u>: With more emphasis on medication errors by third parties investigating the process of medicine, it is imperative that rapid change can occur following identification of specific ADE that can be utilized for increasing patient safety.

165. TIME SAVING ARCHIVE FOR EDUCATORS

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A primary responsibility of poison center educators is to perform poison education and prevention outreach programs in their service populations. Through presentations, articles, public service announcements and news releases, educators teach both the general public and health care professionals. A prototype web-based archiving system for educational materials was created with upload and download capabilities. Entry into the site is password protected. It currently resides on a poison center website and the ease of use has been demonstrated. The web-based archive for poison center

educators is operational and allows easy access to files on poison related topics. Any educator with access can download an educational file and adapt that file to their audience or upload a file for other educators to use. This online database would allow significant collaboration on a national level, reduce duplication of effort, save money and encourage unified messages.

166. PATIENT SAFETY ISSUES—UTILIZATION OF PHARMACY SATELLITES TO DELINEATE ADVERSE DRUG EVENTS

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<u>Background or Objective</u>: The Institute of Medicine has noted that medication errors are a significant patient safety issue. This study was designed to better define the types of adverse drug events (ADE) occurring in a teaching hospital where close review of physicians' orders is available by multiple pharmacy satellites. <u>Methods</u>: All medication orders submitted to six satellite pharmacies in a single hospital over a 2 week period of time were audited, and orders requiring clarification were categorized into type of event occurrence: appropriate agent (AA); allergy (A); dose (D); interval (I); non-formulary agent (NFA); route (R); and other (O), as well as the location of event occurrence, and drug-type. Frequency of potential ADE was categorized by severity (1–5). <u>Results</u>: A total of 173 medication orders required clarification prior to administration: AA, 10.9%; A, 4.9%; D, 36.4%; I, 4.3%; NFA, 29.9%; R, 6.5%; O, 7.1%. The trauma surgical unit accounted for 45% of the clarifications. Antibiotics accounted for 10.4% of the clarifications. Type I categorization (occurrence would not have resulted in patient harm) was found in 65%. <u>Conclusions</u>: Constant vigilance by satellite pharmacists regarding order clarification will result in numerous orders requiring changes; however, in this small sampling, the vast majority of potential ADEs would not have resulted in patient harm. Further investigation into this patient safety issue should be made.

167. EFFECT OF A CARBON MONOXIDE ALARM REGULATION ON CO POISONING

Tomaszewski C, Lavonas E, Kerns R, Rouse A. Carolinas Medical Center, Charlotte, North Carolina, USA.

<u>Background</u>: CO alarms are postulated to be protective from poisoning. On 1/1/2001, a countywide (700,000 pop.) regulation went into effect requiring CO alarms in all residences, exempting all-electric homes (35%) without an attached garage. Surrounding counties had no such regulation. We studied the effect of the law on accidental CO poisoning cases. <u>Methods</u>: Our hyperbaric oxygen (HBO) treatment center accepted referrals 24-hours daily for the index and 10 surrounding counties. We compared accidental CO poisoning cases referred for HBO from the index county vs. surrounding counties for 4 years before and 2 years after regulation enactment. Poison center (PC) calls for CO exposure and coroner reported deaths for the index county were also collated. <u>Results</u>: The number of annual cases of serious CO cases requiring HBO treatment was:

	1997–2000	2001-2002	P value
HBO cases—index county (mean/yr \pm SD)	4.3 ± 2.9	2.0 ± 0.0	NS (t-test)
HBO cases—surrounding counties (mean/yr \pm SD)	8.0 ± 2.6	14 ± 2.8	NS (t-test)
Index/total HBO cases (CO) during period	16/48	4/32	<0.05 (chi ² = 4.4)
PC CO calls—index county (mean/yr \pm SD)	71 ± 11	67 ± 5	NS (t-test)
CO deaths—index county (mean/yr \pm SD)	1.5 ± 1.9	0.5 ± 0.7	NS (t-test)

<u>Conclusion</u>: During the study period, the incidence of total exposures, serious poisoning and deaths from CO did not change. However, the proportion of CO cases requiring HBO originating from the index county decreased after the regulation took effect. A CO alarm requirement reduced the incidence of severe CO poisoning relative to surrounding counties without such a regulation.

168. PREVALENCE OF POISONING IN CHILDREN 6 AND UNDER: A QUANTITATIVE SURVEY OF ONE STATE

Simeonov IM, Heard SE. California Poison Control System, University of California, San Francisco, California, USA.

<u>Objective</u>: Measure the actual rate of poison exposure in children 6 years old and under. Compare data to cases managed by the PCC. <u>Methods</u>: Quantitative research conducted over 2 months throughout the state with parents of children 6 and under (n = 428 with a standard error of $\pm 4.9\%$). Brief (15–20 min) face-to-face interviews were conducted with parents in English and Spanish. Survey instrument measured incidence of common poison exposures reported annually to PCCs with parent's self-reported survey responses. Data was analyzed and compared to annual rate of exposures as reported statewide to the PCC. <u>Results</u>: 48% of respondents reported at least one poison incident in the past year. Responses varied according to demographic groups with medium/high income (\$30,000–\$74,999) mothers reporting a slightly higher rate of exposure and low-income (at or below Federal poverty guidelines) reporting the lowest rate. Top two answers across all demographic groups were ingestion of a medicine or household cleaning product. <u>Conclusion</u>: Children under 6 account for 7% of the state's population, or 2.5 M. Annually, PCC-managed cases within this demographic number far fewer. Comparing both sets of data, the actual rate of poison exposure to children under 6 is significantly greater than as reported to the PCC.

169. TESTING SPANISH LANGUAGE MATERIALS WITH LOW-INCOME PARENTS: WHAT WORKS?

Giraldo GP, Simeonov IM, Heard SE. University of California, San Francisco, California, USA.

<u>Background</u>: Market research and ethnographic findings led the development of two types of PCC health education materials for Spanish-speaking parents. A brochure and two fotonovelas, one photographic, one illustrated, were tested to determine target audience acceptance of culturally tailored message and format. <u>Methods</u>: 121 face-to-face interviews in 4 different locations gauged public acceptance, comprehension and preference. Interviewees were lowincome, Spanish-speaking parents of children \leq 5. 47% had lived in the United States <10 years, 46% 10–20 years. 60% had elementary education or less, 32% had graduated HS and 8% had some education beyond HS. <u>Results</u>: Cross-tabulation of education achievement and preferred format reveals respondents with less education ranked materials as follows: (1) brochure with photographs (46%); (2) fotonovela with photographs (25%); and (3) illustrated brochure (24%). Respondents with high school education ranked as follows: (1) brochure with pictures (61.5%); (2) fotonovela with pictures (26%); and (3) brochure with graphics (5.1%). The illustrated fotonovela was the least preferred among all groups (7%). <u>Conclusion</u>: Consumer perceptions are critical and market testing of materials can limit risk and ensure effectiveness. Spanish language health education materials need to address literacy, educational level and visual language. All strategic health education communications must address explicit consumer perceptions through format, visual content and core messaging.

170. EPIDEMIC CARBON MONOXIDE POISONING DESPITE A CO ALARM LAW

Lavonas E, Tomaszewski C, Kerns W, Blackwell T. Carolinas Medical Center, Charlotte, North Carolina, USA.

<u>Background</u>: Epidemic carbon monoxide (CO) poisoning during winter storms has been well described. Carbon monoxide alarms have been advocated as a method to prevent CO poisoning. We report a CO poisoning outbreak that occurred during a power outage in a community with a law requiring a CO alarm in every residence except for all-electric homes (35.4% exempt). <u>Methods</u>: The population was all residents or visitors in Mecklenburg County, NC, from December 4–13, 2002. Structured chart review was performed for all emergency department (ED), hyperbaric oxygen (HBO) unit, EMS, fire department (FD), gas company, and medical examiner records. Relative risk was determined by comparison with county demographics. <u>Results</u>: 367 FD responses, 34 EMS responses, and 149 ED cases were identified. The median ambient CO measurement in the home of poisoned patients was 238 ppm (range: 128–836 ppm). Most CO came from portable sources: charcoal (55%), generators (15%), or propane grills (13%). The median exposure duration was 9 hrs. Severe CO poisoning (syncope, altered mental status, ataxia, hypotension, or cardiac ischemia)



occurred in 22% of ED pts. Most pts were treated with oxygen (median: 3 hrs) and released. 24 pts (16%) received HBO₂ therapy. One person died. The relative risk of CO poisoning was increased in Hispanic (RR: 5.6), Asian (RR: 3.2), and East African (RR: 4.2) residents and in those who did not speak English (48% of ED pts; RR: 3.6). Although 25% of homes in the community have CO alarms, no pt with severe poisoning had a functioning alarm. <u>Conclusion</u>: A law requiring CO alarms in residences mitigated but did not eliminate a CO poisoning outbreak during a winter storm. Risk factors included immigrant status and lack of proficiency in English. Exempting all-electric homes may have limited the value of the ordinance. The presence of a functioning CO alarm was protective against severe poisoning.

171. A PROFILE OF CALLS TO A POISON INFORMATION CENTER REGARDING OLDER ADULTS

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<u>Objective</u>: Calls to poison information centers regarding older adults are significantly fewer than for children and younger adults; however, the mortality rate among older adults from poisoning is greater. We sought to examine the nature of calls to poison information centers by adults aged 50+ using data from the TESS and a CRPIC. <u>Methods</u>: We conducted a retrospective review of all cases (aged 50+) reported to a CRPIC in 1998 and 1999 (N=6365). We examined eight variables of interest: (1) patient gender; (2) age; (3) urban/rural residence; (4) acuity; (5) known outcomes; (6) reason; (7) exposure level; and (8) general categories. <u>Results</u>: The overall results indicated that therapeutic error and adverse drug reaction calls for this region were three and two times the national rates respectively and that females were more likely than males to be poison information center patients. Furthermore, females' poison experiences were more likely to be the result of therapeutic error, adverse drug reactions, ingestions, and of the acute on chronic class. The data also indicated a number of age-related differences, most notably that older age is inversely associated with acute class, suspected suicide, food poisoning, and inhalation and dermal exposures. <u>Conclusion</u>: Poisoning and subsequent mortality among older adults may become an increasing problem as our population ages. Older women may be at especially high risk because of their high prevalence of drug utilization. Results from this study substantiate the needs for both further investigation into regional poisoning trends among older adults and for poison information centers to target older adults in their education campaigns.

172. PAROXETINE EXPOSURES: FIVE-YEAR ANALYSIS OF UNINTENTIONAL INGESTIONS IN THE PEDIATRIC POPULATION REPORTED TO THE TEXAS POISON CENTER NETWORK

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<u>Objectives</u>: Although it is generally believed that unintentional ingestions of paroxetine by children are relatively safe, a MEDLINE search found no published studies of large pediatric population supporting this concept. Our goal is to determine the symptoms of children 6 years old or younger who have ingested paroxetine. <u>Methods</u>: Data was retrieved from the Texas Poison Center Network—Toxic Exposure Surveillance System database. Inclusion criteria included paroxetine exposures: between January 1, 1998 and December 31, 2002, pediatric patients ≤ 6 years old, known amount ingested, single substance ingestions, 20 mg or more ingested, and follow-up done to determine outcome. <u>Results</u>: One hundred and forty-nine cases met all of the inclusion criteria. The average age was 29 months ± 12 months. The median amount of paroxetine ingested was 30 mg but the range was 20–240 mg. Mild symptoms (vomiting, sedation, hyperactivity, and mydriasis) were noted in 12 patients or 8.0%. No major symptoms were reported. In fifty-four cases (36%) the patient's weight was also documented and we were able to calculate a mg/kg dose ingested; the median dose of paroxetine ingested was 80 mg or below. 92% of the patients remained asymptomatic on follow-up call. This retrospective study provides some evidence that up to 80 mg of paroxetine may be safe in children 6 years old or younger. These children can be left at home with only minimal effects expected, if any. This information may be helpful when triaging patients.
173. EPIDEMIOLOGY OF CARBON MONOXIDE (CM) POISONINGS IN UFA

Sarmanaev SKh, Samolova RG, Aidarova LF. Toxicological Center, Ufa, Russia.

Tabla 1

Carbon monooxide poisonings remain a serious problem of clinical toxicology. They make up 1.5-2.5% of all hospitalizations, with the lethality rate being 5.7-21.9%; mortality reaches 55-90%. <u>Aim</u>: Study of hospitalization and lethality of patients poisoned by CM in Ufa (population: 1, 2 mln). <u>Materials and Methods</u>: A retrospective analysis of medical cards of 162 patients, hospitalized in Ufa Toxicological Center with the CM poisoning diagnosis in 1998–2002. <u>Results</u>: Patients with CM poisoning comprise 1.6% of all patients, hospitalized with acute poisonings (see Table 1). Among them males are prevalent (72.2%), the mean age is 42.1 ± 1.2 Года, the mean hospitalization period is 4.6 ± 0.3 days. Lethality in 1998–2002 in cases of CM poisoning amounted to $5.4 \pm 2.1\%$, all of the deceased were males, capable of active professional employment.

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	19	98	199	99	20	00	200	01	200)2
	Abs	%	Abs.	%	Abs.	%	Abs.	%	Abs.	%
Hospitalization Lethality	32 2	1.7 6.3	40 2	2 5.0	24 3	1.2 12.5	31 1	1.5 3.2	35 0	1.6 0

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<u>Conclusion</u>: Hospitalization in cases of CM poisoning is characterized by a stable and high level and does not show any tendency for declining; lethality amounted to 5.4%.

174. DANGEROUS DRUGS—AN ANALYSIS OF 142 FATALITIES DUE TO POISONING IN NORTHERN GERMANY

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<u>Objective</u>: The knowledge of the substances that cause death in intoxicated patients are of medical, legal, or political relevance. A harmonized data documentation, like TESS, does not exist in Germany yet. GIZ-Nord poison center issues an annual report including fatalities. Aim of the study was to obtain an overview of the substances resulting in fatal poisoning. <u>Methods</u>: In a retrospective study all cases of fatalities by poisoning from January 1996 until March 2003 were analyzed. <u>Results</u>: From 1996–2003 GIZ-Nord was consulted in 170,000 cases. There were 142 fatalities due to poisoning (0.08% of all consultations). In 80 cases (57%) the lethal substance was a medical drug (see Table), in 62 cases (43%) chemicals were responsible for the decease. The vast majority of the fatalities due to drugs was intentional. In the group of psychotropic substances all 36 cases (45% of all drug induced fatalities) were suicidal.

Drugs specified	Number (suicidal)	%
Psychotropic	36 (36)	45
Cardiovascular	13 (10)	16
Aspirine	5 (5)	6
Miscellaneous	26 (11)	33
Σ	80 (62)	100

<u>Conclusion</u>: Suicidal patients are at risk by their regular psychiatric medication, furthermore cardiovascular drugs and aspirine enhance the danger.

175. RESPONSE TO A SUSPECTED WEAPON OF MASS DESTRUCTION VICTIM

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<u>Background</u>: The presentation of a sentinel victim of a weapon of mass destruction event (WMD) to the ED, directly or through contact with a poison center (PC), might be difficult to recognize. <u>Case Report</u>: A middle aged man presented to the ED at an urban trauma center with the complaint of acute, painless disintegration of the tissue of both hands. He worked as a janitor in a chemical and physical sciences building on a state university (SU) campus. He recalled cleaning up an unusual gray powder in a laboratory-classroom. The next day he noted severe swelling of both hands, with rapid progression over 1 hour to painless tissue disappearance and bone denudation. At the hospital, the patient was decontaminated and isolated. The toxicology service was consulted. State University authorities indicated the building housed Biological Hazard Level 4 (BL-4) laboratories for chemistry and radiological work. Diagnostic considerations included: extremely high dose focused ionizing or neutron radiation, direct contact with a radiation source, exposure to phenol or highly concentrated chemotherapy agent, and severe instantaneous frostbite from a super-cooled liquid. A hazardous materials team responded to the ED. A radiation team and toxicologists surveyed the ED and campus building with detection instruments. The state health department, FBI, and other federal agencies initiated a month-long investigation, without detection of a WMD hazard. The patient ultimately underwent resection of eight fingers. The investigation process will be presented. <u>Conclusion</u>: Medical toxicologists should be aware of the unusual resources at the local, state, and federal levels needed to investigate a suspected WMD event.

176. EXCESS FATALITY FROM DESIPRAMINE AND DOSAGE RECOMMENDATIONS

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<u>Background</u>: Desipramine (DMI) has higher fatal toxicity index (FTI) than other tricyclic antidepressants (TCA). Six of the 8 unexpected deaths in children treated with *therapeutic* doses of TCA were from DMI. Based on pharmacokinetic (PK) data we have explained this high FTI; DMI and nortriptyline (NT) are metabolites of imipramine (IMI) and amitriptyline (AT), respectively, and have similar PK profiles: i.e., higher VD, rbc/plasma ratio, and free fraction of the drug relative to IMI and AT. Thus DMI and NT would have higher tissue levels than IMI and AT, for a given plasma level. Thus, the recommended therapeutic plasma levels for NT, its daily dose and max. pill strength are half of that of AT. In 1994 we have suggested to do similar changes for DMI (limit the max. pill strength, reduce therapeutic plasma levels and dose). These adjustments were not done. <u>Methods</u>: Evaluation of the FTI of DMI and other TCA, from the toxic exposure surveillance system (TESS) of the AAPCC, <u>Results</u>: Summarized in Table 1.

Table 1.	FTI of TCA	during the years	1983-2001.
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	1983–1988	1989–1994	1995–2001	Total for 1983–2001
Amitriptyline	121/17,302	256/38,347	387/56,100	764/111,749
	(0.70)	(0.67)	(0.69)	(0.68)
Imipramine	68/99,596	110/19,961	85/15,678	263/45,235
	(0.71)	(0.55)	(0.54)	(0.58)
Nortriptyline	28/2,904	110/15,173	58/11,485	196/29,562
	(0.96)	(0.72)	(0.50)	(0.66)
Desipramine	83/4,113	143/9,869	53/4,050	279/18,032
	(2.02)*	(1.44)**	(1.31)***	(1.55)****
$Chi^2 = (df = 3)$	*70 <i>p</i> < 0.001	**79 <i>p</i> < 0.001	***34 <i>p</i> < 0.001	****183 <i>p</i> < 0.001

Note: Fatal toxicity index (FTI): No. of death/No. of exposures (%).

<u>Conclusion</u>: Fatal toxicity index of DMI exceeds FTI of AT, IMI, and NT by 2.28, 2.67, and 2.35 respectively, resulting in significant excess mortality. Revision of DMI dose should improve its safety.

177. ALPRAZOLAM IS RELATIVELY MORE TOXIC THAN OTHER BENZODIAZEPINES IN OVERDOSE

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Background: This study aimed to describe the epidemiology of alprazolam poisoning, its spectrum of toxicity and relative toxicity compared to other benzodiazepines. Methods: A prospective database of poisoning admissions to a regional toxicology service was searched to identify consecutive benzodiazepine deliberate self poisonings, coded as alprazolam, diazepam or other benzodiazepine. Major outcomes were length of stay (LOS), ICU admission, GCS < 9, flumazenil administration and requirement for ventilation. Prescription data were obtained for benzodiazepines for the study period. Results: There were 2063 single benzodiazepine overdoses: 131 alprazolam, 823 diazepam and 1109 other benzodiazepine. The median LOS for alprazolam overdoses was 19.4 hours (IQR: 13, 39) which was 1.27 (95% CI: 1.04, 1.54) times longer than other benzodiazepines by multiple linear regression. For alprazolam overdoses, 22% were admitted to ICU which was 2.06 (95% CI: 1.27, 3.33) times more likely than other benzodiazepines after multivariate analysis adjusting for age, dose, gender, time to ingestion and co-ingested drugs. Flumazenil was administered to 14% of patients and 16% were ventilated, which was significantly more than for other benzodiazepine overdoses. Twelve percent of alprazolam overdoses had GCS < 9. From benzodiazepine prescription data, alprazolam prescriptions increased from 0.13 million in 1992 to 0.41 million in 2001. Eighty five percent of prescriptions were for panic disorder, anxiety, depression, or mixed anxiety/depression. Conclusion: Alprazolam was significantly more toxic than other benzodiazepines. The increased prescription of alprazolam to groups with an increased risk of deliberate self poisoning, is concerning and needs review.

178. COMPARISON OF PRE-9/11 AND POST-9/11 RATES OF NERVE AGENT ANTIDOTE STOCKING IN METROPOLITAN HOSPITALS

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<u>Objective</u>: To determine any difference in amounts of nerve agent antidotes, atropine sulfate (AS) and pralidoxime Cl (2-PAM), stocked in hospitals prior to and after the 9/11/01 terrorist attack. <u>Methods</u>: Hospital pharmacies were surveyed following two disaster drills involving Sarin. On 06/08/00 (pre-9/11), 154 volunteer victims were sent to 14 hospitals following a simulated attack at a baseball game. On 09/29/02 (post-9/11) 68 volunteer victims were sent to 8 hospitals following a simulated attack at a mental health institution. Following each drill, all pharmacy departments were asked: (1) How many one-gram vials of 2-PAM were in the hospital's inventory? and (2) What is the best estimate of the total number of milligrams of AS available? <u>Results</u>: The following table summarizes data demonstrating an overall increase in the median and mean amounts of antidotes during the post-9/11 drill.

Quantity	Pre-9/11 (14 hospitals)	Post-9/11 (8 hospitals)	% change
2-PAM (median gms)	5.5 (range 0–20)	11 (range 4–54)	+100
2-PAM (mean gms)	8.4	14.9	+81
Atropine (median mgs)	275 (range 30-2800)	525 (range 110-3000)	+91
Atropine (mean mgs)	602	873	+47
Hospitals without 2-PAM	4 (29%)	0 (0%)	-29
Hospitals with <100 mg AS	4 (29%)	0 (0%)	-29

Note: The participating hospitals were different for each drill.

<u>Conclusion</u>: Comparing pre-9/11 and post-9/11 drills, we observe higher rates of nerve agent antidote stocking. This is most likely due to enhanced preparedness for possible WMD attacks.

179. NEWSPAPER IMPACT ON POISON CENTER EXPOSURE CALL VOLUME

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<u>Background</u>: News media plays an important role in disseminating health information. Although media influence on consumer behavior is well documented, news media effect on poison center (PC) call volume is not well studied despite the suggestion of some authors that an "epidemic of anxiety" naturally follows extensive news coverage of toxicologic issues. The recent surge in news coverage of ephedra-containing supplements (ECS) provided an opportunity for a pilot examination of a regional poison center's ECS exposure volume. <u>Methods</u>: The electronic archives of two nationally read newspapers, the New York Times (NYT) and USA Today (USA), and a regionally read newspaper, Chicago Tribune (CT), were searched for published stories where ECS was the subject. Phone calls to a regional PC (annual call volume >90,000) were examined for all ECS exposures. The study period was divided into two intervals: 6 weeks before and 6 weeks after the highly publicized sudden death of a major league baseball player from an ECS on 2/17/2003. <u>Results</u>: ECS was the subject of five news reports (NYT 4, USA 0, CT 1) in the 6-week interval prior to 2/17/2003; ECS was the subject of 98 reports (NYT 54, USA 22, CT 22) in the following 6-week interval. Two ECS exposures were reported to the PC in the 6-week interval prior to 2/17/2003; *no* ECS exposures were reported to the PC in the following 6-week interval. Conclusion: Despite a surge in newspaper coverage detailing the hazards of ECS following the death of a high profile athlete, there was no associated surge in reported ECS exposures to a regional PC during that same time interval. Further scrutiny of this apparent lack of newspaper effect is warranted.

180. A RETROSPECTIVE ANALYSIS OF CARDIOACTIVE STEROID POISONING

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Background: Cardioactive steroids (CAS) have been found in plants, animals, and even insects around the world. A 5- (mostly plant derived) or 6-membered (mostly animal derived) lactone is substituted at C17 of the steroid backbone and attenuates the CASs affinity for the Na^+-K^+ ATPase. No previous study has attempted to categorize CAS poisoning based on the type of lactone at C17. Digoxin-spec Fab (Fab) is known to bind many CAS in vitro but its clinical benefit is currently undefined. Methods: MEDLINE was searched from 1982 forward, so that supportive care was similar and Fab use existed, using all reported names of CAS. Foreign language papers were translated. Identified reports of CAS poisoning were hand searched to exclude licensed pharmaceuticals. The references were similarly reviewed for any further cases. Inclusion criteria included three of the following: hyperkalemia, GI symptoms, ECG evidence of digitalis toxicity, digoxin serum concentration, or history of exposure to cardioactive steroid containing substance. Demographic and clinical data was collected including treatment with Fab and outcome. Results: 59 relevant articles were identified, with a total of 924 patients. Eight hundred and ninety-seven (97%) patients ingested 5-member lactone CAS with a mortality of 6% (n = 54). Twentyseven (2.9%) ingested 6-member lactone CAS with a mortality of 29.6% (n = 8). The 6-member lactone rings with the highest mortality also had the highest number of nonsuicidal exposures. The difference in mortality between 5- and 6-member lactone CAS was statistically significant with p < 0.001, (X²). Insufficient information was available to determine the benefit of Fab. Conclusions: Mortality appears more likely when a 6-member CAS is involved. Given the presumed benefit of Fab, more aggressive treatment may be warranted in those patients exposed to 6-member lactone ring CAS.

181. A STUDY OF THE GENETIC POLYMORPHISM OF DELTA AMINOLEVULINIC DEHYDRATSE (ALAD) IN THAI LEAD EXPOSURE WORKERS

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Background: Delta Aminolevulinic acid dehydratase (ALAD) is a polymorphic enzyme, and ALAD polymorphism might alter lead kinetic. The polymorphism might also be a genetic factor to influence human susceptibility to lead

poisoning. Previous studies indicated the frequencies of ALAD gene were different among the ethnic groups. The objectives of this study were to determine the frequency of ALAD genotype and investigate the relationship between ALAD polymorphism and blood lead level in the Thai workers. <u>Methods</u>: Workers in a battery factory were included in this study. The genotype of the Msp 1 restriction site of ALAD polymorphism was investigated by PCR-RFLP technique. <u>Results</u>: The total of 389 lead exposed workers engaged in the study. The allele frequencies were 98.3% and 1.67% for ALAD-1 and ALAD-2. The genotype frequencies of ALAD1-1, ALAD1-2, and ALAD2-2 were 97.2%, 2.3%, and 0.5% respectively. The median blood lead levels were 35.3 mcg/L in ALAD1-1 (N= 378) and 38.5 mcg/L in ALAD1-2 and ALAD2-2 genotype group (N=11). <u>Conclusion</u>: The frequency of ALAD-2 in Thais is lower than in Caucasians. No relationship between ALAD polymorphism and blood lead level was found because the number of ALAD-2 allele was too small.

182. MEDICINAL USE OF COCAINE: A SHIFTING PARADIGM OVER 25 YEARS

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Background: Cocaine remains a licensed pharmaceutical despite safer alternatives. Human cocaine research is often predicated on its "safe" use in otolaryngology, citing a single study (Johns ME, et al. Trans Am Acad Ophthalmol Otolaryngol 1977; 84:969) which antedates both the epidemic of recreational cocaine use and the first cocaine-induced myocardial infarction. Our objective was to reassess the epidemiology and toxicity of medicinal cocaine use. Methods: Using the same methodology as Johns et al., an anonymous survey was mailed to active members of the American Academy of Otolaryngology-Head and Neck Surgery. The survey used a closed question format asking about the use of cocaine, safety measures taken, and adverse outcomes, and included information about practice type and location. Results were compared to the earlier work using a chi-square test with p < 0.05 considered significant. Results: 7815 surveys were mailed in batches; data reported are from the first 2011 surveys, with a 33% response rate. Currently, a similar proportion of physicians report having used cocaine at sometime in their practice (90%) as compared to 92% 25 years ago (p = 0.2). However, only 54% of respondents currently use cocaine vs. 92% previously (p < 0.001, 95% CI 8.1–12.5). Also, more respondents in the current survey reported adverse reactions (30% vs. 22%; p < 0.001, 95% CI 0.57–0.86). Although tachycardia was the most commonly reported reaction, significant events included myocardial infarction, seizure, and stroke. Two cardiac arrests were resuscitated and no deaths were reported. Conclusions: The decline in the use of cocaine may reflect a better understanding of its potential toxicities and the availability of alternative medications. The justification for using typical ENT doses of cocaine in human research should be reconsidered because cocaine is used less commonly and adverse outcomes occur frequently even at these doses.

183. ACETAMINOPHEN POISONING: AN INTERNATIONAL COMPARISON

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<u>Background</u>: Serious acetaminophen poisoning is a more common problem in the UK. We therefore set out to compare the epidemiology of acetaminophen poisoning in three countries to explore whether baseline differences at presentation may explain the differences in poisoning severity. <u>Methods</u>: Starting 10th March 2003, the following data was collected on all calls regarding acetaminophen ingestion reported to the following PICs: New South Wales (Australia), London (UK), and New Zealand: type of exposure, amount ingested, co-ingestants, time from ingestion to call, callers background, presence of risk factors, and treatment recommended. <u>Results</u>: To date there have been 80 calls to NZ, 723 to London and 258 to NSW relating to acetaminophen ingestion. Of these, intentional ingestions generated 26 calls for NZ (33%), 569 for London (79%), and 130 for NSW (50%). The remaining calls related to accidental poisoning or therapeutic errors. Data for intentional ingestions are presented here. Median ingested dose was 10 g for NZ, 8 g for London, and 6 g for NSW (P = 0.0001). Median time from ingestion to phoning the PIC was 2.4 hours for NZ, 2.8 hours for London, and 1 hour for NSW (P = 0.0001). There was no significant difference in incidence of risk factors.

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<u>Conclusion</u>: International collaboration provides an opportunity to compare differences in the epidemiology of poisoning. For intentional acetaminophen poisoning differences included ingested dose and time to phoning the PIC. The delay in time of call to the PIC and in the initiation of appropriate investigations and treatment may be one potential explanation for the increased incidence of serious acetaminophen poisoning in the UK. Data collection continues and further analysis will be carried out.

184. A CHARACTERIZATION OF POISONINGS IN THE ELDERLY

Vo ML, Caraccio TR, Mofenson HC. Long Island Regional Poison and Drug Information Center, Winthrop University Hospital, Mineola, New York, USA.

Background: Although the elderly constitute 18–20% of poisoning related deaths, only 3% of exposures (exp) are reported to Regional Poison Centers (RPC) in this age group. Current trends of exposures in the elderly population are lacking. Our purpose is to describe the trends of exp in elderly pts vs. younger adults of the various types of substances and hospitalization rates reported by a Regional Poison Center (RPC) and to TESS. Methods: A 4-yr review of exp reported to a RPC was utilized to identify prevalence rates. Elderly patients were classified as >65 yrs and younger adults between 20-64 yrs. Parameters evaluated included substances, medical outcomes, type of medical management, therapeutic intervention, and reason of exposure. A parallel review was conducted of WebTESS for a 2-yr period to account for regional variations. Results: Medications were most commonly associated with hospital admission in the elderly and included benzodiazepines, cardiac glycosides, acetaminophen, aspirin, and SSRI (30%). A higher frequency rate of elderly patients required hospital admissions to CCU/Non-CCU from the RPC (44%) and in TESS [31%] vs. younger adults 38% (RPC); [28%] TESS. Elderly patients were more likely to die vs. younger from the RPC (16.3%) and in TESS [13.7%]. Although, the elderly had a corresponding lower frequency rate of ED visits by the RPC of 25% compared to 33% in younger patients, TESS showed identical frequency rates of ED visits in elderly and younger pts [45%]. Conclusion: The elderly are more likely to be hospitalized and die as a result of toxic exposures. Greater educational efforts need to be devoted for this high risk population who take more medications than their younger counterparts.

185. ANALYSIS OF ADVERSE DRUG REACTIONS (ADR) REPORTED TO A REGIONAL POISON CENTER (RPC)

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Background: Recently, 1 out of 4 patients seen in an ambulatory site experienced an adverse drug reactions (ADR) (NEJM 2003; 348:1556). In 1994, it was estimated >1 million people were hospitalized because of ADR (JAMA 1998;289:1200). Although ADR 2^0 to dietary supplements was recently reported by regional poison center (RPC) (Lancet 2003; 361: 101), analysis of ADR due to other medications has not been well elucidated by RPC. <u>Methods</u>: This study delineates the type of ADR reported to a RPC over a 1 yr period. Cases coded as an ADR were extracted from the 2002 database with the following parameters: age, drug class, clinical effects (CE), treatment (tx) provided, disposition, and medical outcomes. <u>Results</u>: 246/27,310 cases were classified as ADR (0.9%). Age analysis 12 (4.1%) were ≤ 5 yrs, 38 (14.6%) between 6–19 yrs; and 196 (79.7%) >19 yrs old. Top 10 drugs with ADR were: (1) Analgesics 16.5%; (2) Antimicrobials 11.7%; (3) Dietary supplements 8.9%; (4) Antidepressants 8.6%; (5) Cough/cold products 5.8%; (6) CV agents 4.8%; (7) Hormones 4.5%; (8) Sedative hypnotics 4.5%; (9) GI drugs 4.1%; and (10) Stimulants 3.5%. Clinical effects by organ systems: Neurol: 33.7%, Dermat: 16%, GI: 16%, and Resp 3.2%. Tx: 14.4% IV fluids, antihistamines 5.2%, benzodiazepines 2.4%, O₂ 2.4%, hemodialysis 1.4%. Disposition: Home 133 (27 < 20 yrs; 106 adults). Recommended to HCF: 111 (23 < 20 yr; 77 adults) [11 refused]. Outcomes: None 4.4%, Min 43.9%, Mod 21.9%, Maj 5.6%. Unk toxic 13.4%, and unrelated 10.5%. <u>Conclusion</u>: A collaborative effort needs to be established with FDA/HHS to encourage reporting of ADR to RPC.

186. HOSPITAL ANTIDOTE STOCKING SUBSEQUENT TO THE 2001 TERROR ACTS

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Background: We performed a 10 year retrospective survey of hospital pharmacy antidote stocking trends in 1999, and demonstrated an increase in pralidoxime stocking, beginning in 1995. We hypothesized that this trend was initiated in response to the Tokyo sarin attacks. We question whether the stocking of antidotes by CT hospital pharmacies changed following the 2001 US terrorist attacks. Methods: A supplemental survey was faxed to pharmacy directors of all CT hospitals in February 2002. They were asked: "Have you adjusted your antidote stock as a result of the terrorist act of this fall? If so, please indicate what changes were made (i.e., survey-listed antidotes or others)." Results: Responses were obtained from 29 of 31 facilities (94%). Nearly half of the hospitals (14 of 29) made adjustments in their stocking following 9/11. Of these 14 hospitals, 12 specified the agents they had increased. The antidote stocking increase in these 12 hospitals was: doxycycline (6 of 12; 50%), ciprofloxacin or "quinolones" (7 of 12; 58%), atropine (5 of 12; 42%), potassium iodide (KI) and pralidoxime (2 of 12; 17%). Increased stocking of Lugol's solution, sodium nitrite, and cyanide antidote kit were each listed by one hospital (1 of 12; 8%). Geographically, most of the hospitals in proximity to NYC and the CT nuclear power plants documented increases in antibiotics, atropine, and KI (7 of 9; 78%). Conclusion: Hospital pharmacy antidote stocking did increase after the terror attacks of 2001. The increased stocking seemed to be targeted to the demonstrated threat of anthrax and in response to potential terror target proximity, rather than a general increase in all countermeasures to the CDCs list of high-likelihood/high effect agents. Stocking of pralidoxime was limited due to federal government diversion. Potassium iodide stocking may have been affected by the state government's developing distribution plan.

187. OUTCOMES OF A TV AD CAMPAIGN PROMOTING POISON CENTRE (PC) AWARENESS

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<u>Background</u>: We sought to evaluate the impact on call volumes to our PC from the two large metropolitan areas targeted during separate TV ad campaigns in the fall of 2002 and winter of 2003. Both these areas had shown a significant trend of decreasing call volumes over the previous 2 years. The TV ads were designed to promote increased awareness of the PC as a resource for poisoning exposures. No other PC education or outreach efforts were conducted in these areas during the TV ad campaigns. <u>Method</u>: Call volumes to the PC were tracked on a monthly basis. These were further characterized by geographic location, exposure (general and human) and information as well as by caller (public and health professional). <u>Results</u>: The decreasing trends in calls was reversed coinciding with the onset of the first set of TV ads. There was an overall decrease of 6.3% in call volume in the first half of the year (precampaign) over the equivalent time period the previous year. There was an increase of 12.3% in call volumes in the second half of the year (during and post campaign). Call volumes in the fall campaign metropolitan area increased by 18.3% during and post awareness vs. a decreasing call volume of 9.7% precampaign. The increase appeared sustained for several months even after the campaign ended. The metropolitan area targeted in the winter campaign showed a 15% increase in calls post campaign vs. a 9% decrease in the 9 months precampaign. The increase in both cities was primarily due to an increase in public human exposure calls since health professional call volume was not impacted by the campaigns. <u>Conclusion</u>: The TV ads were effective in increasing calls to the PC for exposures.

188. A NEW MEASUREMENT TOOL FOR EVALUATION OF PC FUNCTION

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<u>Background</u>: Standard PC evaluation measurements include call volumes and penetrance rates. Both these modalities do not measure poisonings not reported to a PC. <u>Methods</u>: PC data was integrated and compared with regional utilization data obtained from health authority health records and population data from the vital statistics department. Emergency visits (which include hospital admissions) were extracted for poisoning ICD-9 codes. Data was available for some regions since 1995. An incidence rate was calculated: the sum of ED visits for poisoning + the number of poisonings

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managed by the PC at home per 1000 population. Seven year trends incidence rates were compared to penetrance rates for two large metropolitan regions for all age groups, children <5, ages 5–14, 15–29, 30–59, and >60. <u>Results</u>: While the penetrance rates in children <5 has declined, so has the incidence of poisoning. In one region, the incidence and penetrance trends were virtually superimposable. For all other age groups, there is a minimal to absent change in incidence with a significant and increasing gap between incidence and penetrance rates. <u>Conclusion</u>: Incidence rates can be a valuable metric to determine a target penetrance rate for a particular region. In addition, the incidence rate over time can serve as a more accurate metric for poison prevention and education initiatives than call volumes or penetrance rates. The gap between incidence and penetrance can measure awareness and can provide a target for awareness. A limitation of the methodology for calculating incidence is that it does not take into account office visits for poisoning and deaths reported by the coroner/medical examiner.

189. AMINOTRANSFERASES AS MARKERS IN AMANITA PHALLOIDES POISONINGS

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<u>Background</u>: Toxic hepatitis is the major toxic effect in Amanita phalloides mushroom poisonings. Elevated aspartate (AST) and alanine aminotransferase (ALT) are very sensitive markers for hepatocellular damage. We examined the relationship between aminotransferases (transaminases) levels during hospital treatment, and the severity of poisoning and clinical outcome, in phalloid intoxications. <u>Methods</u>: We retrospectively evaluated the medical records of 14 inpatients with phalloid poisoning, with emphasis on their aminotransferases levels, clinical course, and outcome. Patients were divided into two groups: four (28.5%) patients with lethal outcome, and 10 (71.5%) who survived. <u>Results</u>: Increased aminotransferases levels were generally noted on the second day following ingestion. After this initial elevation they rose to their highest average level on the 5th day. In the lethal outcome group the mean of the highest AST values was 335 (22–661), while that of ALT was 812 (22–2421). <u>Conclusion</u>: Aminotransferases are important and suitable markers for the clinical course and outcome in Amanita phalloides poisonings, just like they are in poisonings with other toxic agents. They are particularly good markers for hepatotoxicity in phalloid poisonings and should be used in conjunction with other clinico-biochemical parameters when assessing intoxication severity and classifying cases.

190. COCOA BEAN MULCH AS A CAUSE OF METHYLXANTHINE TOXICOSIS IN DOGS

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Background: Cocoa bean shells, a by-product of chocolate production, are sold as mulch for landscaping. Homeowners find cocoa mulch desirable because it degrades into an organic fertilizer and provides an attractive color and odor. Unprocessed beans, derived from the Theobroma cacao plant, contain 1-4% theobromine/0.07-0.36% caffeine, whereas, cocoa bean mulch contains 0.19-2.98% theobromine. Some dogs find the mulch attractive and eat small to large quantities. Case Series: In response to increasing reports of dogs eating cocoa bean mulch used in landscaping, a retrospective case study was conducted to further define this unique phenomena. Sixteen cases of cocoa mulch ingestion by dogs were managed between January 2002 and April 2003. Of these, six cases were selected for analysis because the final outcome was known, there was evidence/observation of ingestion, and the managing veterinarian assessed the causality relationship as medium or higher. In 50% of the cases vomiting was reported, 33% involved tremors, and in 17% tachycardia, hyperactivity or diarrhea was reported. In 33% of cases no clinical signs developed. In the cases in which tremors were observed, the amount ingested was described as large or significant. California accounted for 67% of cases. Conclusion: Dogs consuming cocoa bean mulch may develop methylxanthine toxicosis. Retrospective case data suggests clinical signs following ingestion include vomiting and muscle tremors. Although oral doses could not be quantitatively determined, clinical severity increased with increasing qualitative dose descriptions. Therefore, treatment should be directed at controlling clinical signs until recovery and preventing further exposure. Pet owners should avoid use of cocoa bean mulch in landscaping around dogs with indiscriminate eating habits.

191. PONTINE STROKE AFTER ACUTE WHITE HELLEBORE POISONING

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Background: Hellebore and other plants containing veratrum alkaloids are known to cause hypotension, bradycardia, and cholinergic symptoms. These substances have been studied in the past as potential therapeutic agents for these effects in addition to causing vasodilation. We present a case of hellebore ingestion complicated by development of a large pontine CVA. Case Report: A 59 year-old male, an experienced forager for many years, picked and boiled what he identified as "skunk weed," later verified as white hellebore. Within half an hour of ingesting the plant, he developed severe vomiting, diarrhea, copious frothy sputum, and weakness. His son transported him to the emergency department where his initial BP was 50/p, HR 40, sinus bradycardia. He was given 1 mg Atropine IV, 2 L crystalloid, intubated and started on dopamine. The patient rapidly improved and dopamine was weaned off in under 12 hours. He became aphasic and developed right hemiplegia on hospital day 1. This progressed to quadriparesis and a "locked-in" state on hospital day 2. An MRI/MRA showed basilar artery thrombosis and extensive infarction of the pons. His clinical condition did not improve and at the time of discharge he had extraocular muscle function and slight movement of the left foot. Interestingly, the patient was found to have a Lupus inhibitor and hypercoagulable state. Neurology and hematology services did not believe this to be the etiology of his CVA. Conclusions: Accidental ingestion of veratrum alkaloids can occur even among experienced foragers and be mistaken for other plants, causing poisoning. Hemiparesis and aphasia have been described after hellebore poisoning. We describe an extensive pontine stroke after veratrum alkaloid poisoning.

192. WITHDRAWAL SYMPTOMS AFTER VALERIAN CESSATION

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<u>Background</u>: Plants of the genus *Valeriana* (Valerianacea), commonly used as an herbal sedative or sleep aid, contain a volatile oil with constituents including monoterpine bornyl acetate and sesquiterpene valerenic acid. Although these compounds have GABAergic effects mediated through inhibition of GABA breakdown and stimulation of GABA release, withdrawal symptoms from cessation of these products have not been described. We report a case of Valerian withdrawal. <u>Case Report</u>: A 41 year-old woman without significant past medical or psychiatric history presented with agitation, visual hallucinations, and tinnitus. According to a friend, she had been using Valerian regularly as well as acetaminophen/hydrocodone, and she had no history of ethanol abuse. She stopped taking all medications 4 days prior to presentation. Initial vital signs were: blood pressure, 122/74 mmHg; pulse 110–120 min⁻¹; respirations, 18 min⁻¹; afebrile. Physical examination was significant for tremulousness and nystagmus. There was no piloerection, lacrimation or yawning and her pupils were of normal size. The ECG was normal and her laboratory evaluation was surremarkable. A urine toxicology screen was positive for cannabinoids only. Prior to consultation, the patient was sedated with lorazepam, sodium amytal, haloperidol, and risperidone. Further sedation as necessary with benzodiazepines only was recommended. Over the following 24 hours, the patient was treated with lorazepam as needed and gradually returned to her baseline mental status. <u>Conclusion</u>: Patients may develop a withdrawal syndrome after cessation of Valerian use that is similar to withdrawal from other GABAergic agents.

193. EFFICACY OF DMSA AND CaNa₂EDTA VERSUS BAL AND CaNa₂EDTA IN ASYMPTOMATIC CHILDREN WITH LEAD POISONING

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<u>Objective</u>: To determine equivalence in the reduction of mean blood Pb (BPb) levels between two inpatient chelation protocols (one historic and one newly approved). Mean urinary Pb (UPb) excretion levels and side effects were also

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compared between groups. <u>Methods</u>: Medical records of children with BPb 40–70 mcg/dL treated with a 5-day course of chelation with either protocol were retrospectively reviewed from 3/2000 to 12/2002. An equivalence test was used to determine if po DMSA + iv CaNa₂EDTA (new protocol) was therapeutically equivalent to im BAL + iv CaNa₂EDTA (historic protocol) in reducing BPb (mcg/dL) by an apriori amount of 6.5. Appropriate statistics were used to determine differences between groups. <u>Results</u>: 22 and 24 children received BAL + CaNa₂EDTA and DMSA + CaNa₂EDTA, respectively. Pretreatment BPb (mean ± SD) did not differ by group (DMSA + CaNa₂EDTA: EDTA: 47.5 ± 5.6 vs. BAL + CaNa₂EDTA: 48.1 ± 7.7). Blood Pb at day 14 post-chelation decreased significantly within each group (DMSA + CaNa₂EDTA: -17.8, p < 0.001; BAL + CaNa₂EDTA: -16.5, p < 0.001). Mean BPb were comparable and met the defined criteria for equivalence (90% CI -2.7 to 6.5). Mean day 14 post-chelation BPb and day 5 post-chelation UPb levels were not significantly different between groups. Vomiting during BAL + CaNa₂EDTA therapy was observed more frequently compared with the DMSA + CaNa₂EDTA group (p < 0.01). <u>Conclusion</u>: Treatment with po DMSA + iv CaNa₂EDTA is comparable to im BAL + iv CaNa₂EDTA in reducing BPb levels.

194. CROSS-SECTIONAL EXPOSURE ASSESSMENT OF ENVIRONMENTAL CONTAMINANTS IN CHURCHILL COUNTY, NEVADA

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<u>Background</u>: Having identified an increased number of childhood leukemia cases within Churchill County, Nevada, the Nevada State Health Division requested that CDC conduct a cross-sectional exposure assessment to identify contaminants unique to that community. <u>Methods</u>: 14 children with ALL or AML, and 55 age and gender matched controls were enrolled. Questionnaire data and biologic samples were collected from the cases, controls, and their immediate family. Environmental samples from current and previous homes within the county were collected. All samples were tested for metals, volatile organic compounds, PCBs, and pesticides. <u>Results</u>: 205 participants were enrolled. No positive association was found between the analytes measured and the case families. Tungsten urine levels were significantly greater in study participants (mean = $1.19 \,\mu g/L$, 95% CI 0.89–1.59) than in the US population (mean = $0.08 \,\mu g/L$). The median level of urine arsenic was $37.40 \,\mu g/L$ (range $0-1180.40 \,\mu g/L$) with 34% of the participants above the normal threshold (50.0 $\mu g/L$). Tungsten and arsenic were also detected in most of the tap water samples collected. Five nonpersistent and one persistent pesticide were elevated when compared with the US population. <u>Conclusion</u>: This investigation identified several environmental exposures of concern among Churchill County residents. Steps are being taken to further define the health implications have been made to decrease personal exposure.

195. TRANSDERMAL PENETRATION OF THE HERBICIDE 2,4-D IS ENHANCED BY UV ABSORBERS FOUND IN COMMERCIAL SUNSCREENS

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<u>Background</u>: Agricultural workers are encouraged to use sunscreen to decrease the risk of UV related skin cancer. Previously, we have shown that some commercial sunscreens enhance the dermal penetration of 2,4-D and that the active ingredients in sunscreen formulations (i.e., UV absorbing components and DEET) also act as dermal penetration enhancers for this herbicide. Most commercial sunscreen formulations use multiple UV absorbers to provide broad spectrum coverage. The purpose of this work was to determine whether these combinations exhibit synergistic or antagonistic penetration enhancement effects. <u>Methods</u>: The active ingredients of the commercial products originally tested were determined and representative formulations were made in 80% ethanol. Hairless mouse skin was placed in an in vitro diffusion chamber. The epidermal side was pretreated for 30 min with the sunscreen formulations and then exposed to ¹⁴C 2,4-D for 24 hours. The penetrant was collected in 90 min fractions, assayed, and the cumulative

percent of 2,4-D absorbed and lag time were then determined. <u>Results</u>: All active ingredient combinations significantly increased the total percentage of 2,4-D crossing the skin in 24 hours when compared to the commercial formulation. Coppertone to Go (75.7 ± 5.5 vs. 56.8 ± 6.6 , p < 0.05), Coppertone Sport (80.2 ± 4.0 vs. 48.1 ± 6.3 , p < 0.001), Neutrogena Sunless (84.2 ± 1.8 vs. 59.2 ± 4.9 , p < 0.01), Neutrogena Oil Free (85.4 ± 3.3 vs. 65.1 ± 15.9 , p < 0.05), Banana Boat (85.1 ± 2.0 vs. 39.6 ± 3.4 , p < 0.001), and No Ad (80.2 ± 4.0 vs. 57.6 ± 1.3 , p < 0.05). This trend also held for the two DEET containing formulations, OFF Skintastic (83.7 ± 2.3 vs. 46.8 ± 2.6 , p < 0.001) and Deep Woods OFF (97.8 ± 0.5 vs. 62.4 ± 5.1 , p < 0.001). <u>Conclusions</u>: All combinations of UV absorbers increased the transdermal absorption of the herbicide 2,4D. Proper selection of inert ingredients is crucial to reducing this penetration enhancement.

196. WHOLE BLOOD AND HAIR MERCURY CONCENTRATIONS IN UPPER GREAT LAKES FISH-EATERS: TOTAL AND INORGANIC MERCURY

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<u>Background</u>: We have concluded 10 years of data collection and exposure assessment for approximately 800 tribal participants from the upper Great Lakes region who completed fish consumption questionnaires and most provided hair and/or blood for mercury and PCB contaminant analyses. Although we will concentrate on the mercury results, 90% of the participants had less than 3.8 parts per billion (ppb) total PCBs and 2.6 ppb total blood mercury. Compared to other studies of subsistence fishing populations these exposures are only moderately elevated and not high enough to warrant widespread restrictions to diets. We will concentrate this report on 16 individuals at higher risk due to mercury exposures. Case Series: There were 16 participants who had hair mercury concentrations that were considered elevated (above $2.0 \ \mu g/g$ total hair mercury) and had sufficient remaining hair sample material for reanalysis. Those participants' were reanalyzed at a second laboratory, including mercury speciation for inorganic and total mercury, to determine if the mercury was methylmercury. <u>Results</u>: The highest total hair mercury value determined for this subset was $32.3 \ \mu g/g$ and the lowest was $1.8 \ \mu g/g$ with an average for the 16 participants of $12.6 \ \mu g/g$. The speciated results indicate that methylmercury is the predominant form (93%). <u>Conclusion</u>: For these cases, we will summarize their fish eating habits (temporal, geographical, and demographic characteristics) and provide health risk educational outreach in their community.

197. A STUDY TO ESTABLISH AN EFFICIENT MEANS FOR DELIVERING ANTIDOTAL THERAPY AT NERVE AGENT DESTRUCTION FACILITIES

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<u>Background</u>: An important component of medical care at facilities for the destruction of chemical weapons is providing antidotes for the treatment of nerve agent (NA) exposures. The current antidotal standard is the muscarinic acetylcholine blocker Atropine (atropine sulfate 0.1%—1.0 mL) and the cholinesterase reactivator Carboxime (15% solution for injection, 1.0 mL ampules). Methods: The antidotal efficiency of administering Atropine (10 mg/kg IM) and Carboxime (30 mg/kg IM) was studied in male white rats poisoned with sarin, soman, and VX. After LD₅₀s were established for each NA, Protection Indexes (PI) were calculated: PI = (LD₅₀ of experimental group/LD₅₀ of control group). The Carboxime reactivating effect was determined by measuring cholinesterase activity in peripheral blood and brain tissue of animals, and compared against TMB-4 and HI-6, alternative cholinesterase reactivators. All data was subjected to statistical analysis. <u>Results</u>: Carboxime was shown to reactivate NA-inhibited cholinesterase (including brain tissue) better than TMB-4, but not as well as HI-6. The combination of Atropine and Carboxime may be an effective than any of the reactivators studied, alone. <u>Conclusion</u>: The combination of Atropine and Carboxime may be an effective antidotal treatment for NA poisoning at facilities where NA are being destroyed. Carboxime essentially surpassed the effectiveness of cholinesterase reactivators such as TMB-4, 2-PAM chloride, and Obidoxime. It is not as effective as HI-6, but is more convenient to administer by injection.



198. A MURINE MODEL FOR IN VIVO NEUROTOXICITY OF GAMMA-HYDROXYBUTYRATE (GHB) AND 3,4-METHYLENEDIOXYMETHAMPHETAMINE (MDMA; ECSTASY)

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Background: Gamm Hyrdroxy Butyrate (GHB) and 3,4-Methylenedioxymethamphetamine (MDMA) are popular drugs of abuse despite their federal schedule I status. Per data from the Drug Abuse Warning Network (DAWN) 2000 Report, 15% of total and 21% of polydrug GHB use involved MDMA, second only to alcohol as a co-ingestant. However, there are no published studies examining the combined effects of MDMA, a known psychostimulant, and the CNS depressant, GHB. Objective: To examine the combined neurotoxic effects of GHB and MDMA using an in vivo murine model. Methods: Thirty male CD-1 mice received ip injections of published murine toxic doses of MDMA and GHB: MDMA 20 mg/kg (N=5), GHB 500 mg/kg (N=10), MDMA 20 mg/kg + GHB 500 mg/kg (N=5), MDMA 40 mg/kg(N=5), and MDMA 40 mg/kg + GHB 500 mg/kg (N=5). All mice were evaluated every 15 min for 180 min for neurotoxicity using righting reflex (RR), grip strength (GS), and rotorod (RoRo) testing. The proportion of mice failing these tests in the single-drug groups were compared to the combined-drug groups using the two-tailed Fisher Exact Test at 15, 30, and 60 min (statistical significance at p < 0.05). Results: Mice receiving 500 mg/kg GHB failed all tests at 15 min and RoRo and GS at 30 and 60 min. No mice receiving only MDMA failed any tests. The mice at the tested combined doses failed all tests at 15 min and RoRo and GS at 30 and 60 min (p < 0.05 compared to MDMA only). Behaviorally, the mice on GHB alone remained sedated and unsteady, while those on only MDMA were hyperactive. The combined-drug mice exhibited initial GHB symptoms then, after a brief mixed-symptom period with continued failure of RoRo and GS, rapidly transitioned to MDMA hyperactive behavior with regained ability to pass all three tests. Conclusions: In combined dosing of GHB and MDMA, the CNS depressant effect of GHB initially predominates over the stimulatory effect of MDMA. The neuropharmacological effects of this combination are complex and warrant a complete dose-response evaluation, including the addition of open field locomotion testing to objectively record and validate these behavioral observations. Disclosure: Quang L acknowledges two NIH grants: #1RO3DA15951-01 and #1T32HD40128-01

199. PREDICTING HEPATOTOXICITY FOLLOWING ACETAMINOPHEN OVERDOSE: A NOMOGRAM FOR THE POST-N-AC ERA

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Background: Despite enormous clinical experience with acetaminophen (APAP) in overdose, fundamental questions remain unanswered, in part due to limited ability to predict hepatotoxicity following overdose. We have recently derived a new predictor, ψ , which combines the Rumack-Matthew nomogram approach with delay to N-acetylcysteine (N-AC) therapy. Methods: Data were obtained from a structured, explicit chart review of patients admitted for APAP overdose. Subjects were included for acute overdose (over $\leq 6 \text{ hr}$), at least one [APAP] at 3.5–24 hr, and at least one transaminase (AST or ALT) beyond 24 hr, and excluded for pre-existing transaminase elevation or late presentation. Logistic regression (probit) was used on the primary outcome of hepatotoxicity (transaminase $\geq 1000 \text{ IU/L}$). Results: Of 3520 admissions identified at six hospitals from 1980 to 2002, 1270 met inclusion/exclusion criteria (31% male, median [IQR] age 22.1 [17.2, 31.9] years, [APAP]_{4h} 1410 [992, 2180] μ mol/L). The final model using log₁₀ ψ predicted hepatotoxicity very well (concordant pairs 92.2%, Hosmer-Lemeshow p = 0.98), with ethanol co-ingestion an important covariate. The ED₅₀ for hepatotoxicity was 23.7 mM hr, and 30.3 mM hr in the presence of ethanol (adjusted OR 43.0 [22.4, 82.6] for each $\log_{10} \psi$, and 0.41 [0.78, 0.22] for ethanol co-ingestion). Age, gender, and self-reported alcoholism were not significant independent predictors. Discussion: ψ is an efficient predictor of hepatotoxicity, as evidenced by the quantification of a substantial protective effect of co-ingested ethanol. Ultimately, accurate risk-stratification could be used to individualize therapy, including ultra-short courses of N-AC for low-risk patients, and adjuvant therapy with CYP2E1 inhibitors for high-risk patients.

200. LATE ACTIVATED CHARCOAL USE IN ACETAMINOPHEN OVERDOSE

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<u>Background</u>: Previous reports have suggested activated charcoal (AC) may have an impact on outcome in acetaminophen (APAP) overdose beyond interfering with initial absorption. <u>Method</u>: Prospective case series of all APAP overdoses. Entrance criteria were: toxic APAP blood level according to the Rumack-Mathews nomogram and any/all therapies including AC and *N*-acetylcysteine (NAC) initiated \geq 4 hours post ingestion. All patients received NAC therapy. Exclusion criterion was polydrug ingestion. Cases were divided into three groups: group A = NAC begun 4 to 8 hours post ingestion, group B = NAC begun 9 to 16 hours post ingestion, and group C = NAC begun > 16 hours post ingestion. <u>Results</u>: 136 cases were collected. Sixty-one patients received AC (45%). Two of 61 (3%) who received AC had an AST or ALT > 1000, while 36 of 75 (48%) who did not receive AC had an AST or ALT > 1000 (p < 0.001).

Receive charcoal	Mean APAP level \pm SD	APAP level drawn mean hours post- ingestion \pm SD	NAC initiated mean hours post- ingestion \pm SD	AST or ALT > 1,000	Chi-square
Group A Yes $(n = 25)$	222.6 ± 81.2	4.7 ± 0.9	6.0 ± 0.9	0 of 25	p = 0.055
Group A No $(n = 14)$	192.5 ± 73.7	5.4 ± 0.9	6.4 ± 0.9	2 of 14	p = 0.055
Group B Yes $(n = 31)$	131.2 ± 68.4	8.7 ± 2.4	11.2 ± 2.0	1 of 31	p < 0.001
Group B No $(n = 33)$	124.5 ± 71.1	11.1 ± 2.6	12.4 ± 2.4	17 of 33	p < 0.001
Group C Yes $(n = 5)$	74.9 ± 48.9	12.9 ± 3.4	31.2 ± 23.4	1 of 5	p = 0.097
Group C No $(n=28)$	48.8 ± 39.7	23.3 ± 10.1	24.7 ± 9.5	17 of 28	p = 0.097

ANOVA showed no difference (in the full group and within Groups A and B) in those with and without AC in: time to initiation of NAC, APAP level or hours post ingestion of APAP level. <u>Conclusion</u>: Late administration of activated charcoal (>4 hours post ingestion) in this series of patients with toxic acetaminophen levels treated with NAC was associated with reduced incidence of liver damage, as measured by elevated serum transaminases.

201. ACETAMINOPHEN HALF-LIFE AS A REFERENCE IN CHOOSING SHORTER ORAL *N*-ACETYLCYSTEINE THERAPY FOR THE OVERDOSED PATIENTS

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<u>Objective</u>: To investigate if half-life was a safe and effective reference in choosing shorter *N*-acetylcysteine (NAC) regimen for the treatment of acute acetaminophen (APAP) overdose. <u>Methods</u>: We conducted a prospective study. Oral NAC was discontinued when APAP level became undetectable with normal aminotransferases. Of 105 patients, 22 met the inclusion criteria: an acute overdose ingestion, toxic APAP concentration, repeated APAP level obtained, and oral NAC treatment initiated within 24 hours. We compared the time to start NAC therapy (<8 hours vs. \geq 8 hours) with APAP half-life determination (<4 hours vs. \geq 4 hours) in predicting hepatotoxicity. <u>Results</u>: The mean (±SD) duration of oral NAC treatment was 31.6 ± 19.3 hours. The time to NAC therapy in predicting hepatotoxicity had a sensitivity of 75%, a specificity of 61.1%, a positive predictive value of 30%, a negative predictive value of 91.7%, and an accuracy of 68.2%. The half-life determination had a sensitivity of 100%, a specificity of 83.3%, a positive predictive value of 57.1%, a negative predictive value of 100%, and an accuracy of 86.4%. The half-life determination was superior to time to NAC therapy in selecting patients to receive shorter oral NAC therapy without hepatotoxicity. <u>Conclusion</u>: This study suggests that a shorter course of oral NAC therapy is safe and effective in the patients whose APAP half-life calculated within 36 hours is less than 4 hours.



202. CLINICAL FEATURES OF A REPEAT-DOSE MULTIPLE-DAY PHARMACOKINETICS TRIAL OF ACETAMINOPHEN AT 4, 6, AND 8 g/DAY

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<u>Background</u>: Acetaminophen (APAP) pharmacokinetics (PK) has been studied at recommended single doses (1 g), at recommended total daily doses (4 g/day) and in single, large, simulated overdoses (up to 9.1 g). Repeat-dose PK studies at 4 g/day in healthy and hepatic-impaired adults do not demonstrate APAP accumulation to higher concentrations with continued dosing. <u>Methods</u>: This double-blinded, placebo-controlled, 3-regimen study in healthy subjects assessed clinical and PK parameters after 1 g, 1.5 g, and 2 g APAP were given every 6 hours for 13 consecutive doses. Subjects were divided into 2 groups: (1) 12 adults on APAP at 4 g/day and 6 g/day and 6 on placebo and (2) 12 adults on APAP at 4 g/day and 8 g/day and 6 on placebo. Liver function and other safety parameters were regularly monitored during the baseline, treatment, and washout phases between treatment periods and for a 3-day washout phase following the final treatment. <u>Results</u>: Pharmacokinetics determinations from the first and last doses of each 4 g, 6 g, and 8 g APAP per day regimen demonstrated that plasma APAP concentrations did not accumulate with repeated dosing. No subject discontinued due to an adverse event. All reported adverse events were non-serious in severity. There were no adverse hepatic or renal events reported. Results of all liver function tests for subjects taking 4 g/day, 6 g/day, and 8 g/day APAP and placebo were within normal limits for the duration of the study. Ranges of individual interday variations for both AST and ALT during the drug administration and washout phases reflected the expected day-to-day and time-of-day biological variations in adults. <u>Conclusion</u>: Repeat doses of APAP, up to 8 g/day over 3 days in healthy subjects, were well tolerated.

203. PLASMA REDUCED GLUTATHIONE (GSH) IN ALCOHOLICS RECEIVING MAXIMAL THERAPEUTIC DOSES OF ACETAMINOPHEN

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<u>Background</u>: Reduced glutathione (GSH) is important in the detoxification of NAPQI, the toxic metabolite of acetaminophen (APAP). Alcoholics are thought to have diminished GSH levels causing vulnerability to APAP toxicity. The maximum time of vulnerability is reported to be 8 hours after drinking cessation. We examined plasma GSH in 53 alcoholics receiving either placebo or maximum therapeutic APAP doses. <u>Methods</u>: A randomized double-blinded study was conducted using inpatients at a closed campus alcohol detoxification center. Patients received APAP (4 g/d) or placebo and were dosed for 3 days. All patients received the same diet. Reduced glutathione levels were drawn in the mornings at time 0 (Day 1) and after 2 days of dosing (Day 3). <u>Results</u>: Paired (Days 1 & 3) GSH samples were available for 53 patients to date. Average plasma GSH levels were 1.82 μ M (placebo) and 2.07 μ M (APAP) on Day 1 and 1.81 μ M (placebo) and 2.21 μ M (APAP) on Day 3. No significant difference in means was found at the 95% confidence level, p = 0.07. Nonparametric testing indicates no significant difference between Day 1 and Day 3 GSH levels within either group (Wilcoxin Signed Ranks test p = 0.687 and p = 0.292, respectively). When all 81 patients are completed, this study will have 70% power to detect a difference of 0.42 μ M at the 95% confidence level. There was no change in AST throughout the study. <u>Conclusion</u>: Plasma GSH concentrations in alcoholics are not significantly affected by 2 days of maximal therapeutic dosing with APAP.

204. EFFICACY OF DIGIBIND VERSUS DIGIFAB IN BINDING CINOBUFOTALIN

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<u>Background</u>: Cane toad venom is used to prepare various Chinese medicines like Chan Su, Lu-Shen-Wan, and Kyushin and has resulted in lethal exposures. Cinobufotalin (CINB), a bufadienolide, is one component of this venom. Digibind[®] (DB) has been shown to bind various bufadienolides but has never been compared to the new Fab preparation, DigiFab[®] (DF). This study compares the binding of DB vs. DF to CINB. <u>Methods</u>: HPLC grade methanol was utilized to create a stock

solution CINB then diluted with fresh frozen plasma to 50 µg/mL. Stock solutions of DB and DF were made with normal saline to a concentration of 200 µg/mL. Three hundred micro litre aliquots of the DB and DF solutions were separated and then mixed with 2.7 mL of CINB solution so that the final DB and DF concentration was $20 \mu g/mL$. DB + CINB, DF + CINB, and an aliquot of just CINB solution were incubated at 37° C for 1 hour. The protein-free ultrafiltrate of the three aliquots was prepared by centrifuging a specimen with the Centrifree Micropartition system for 40 min at 1500g. Digoxin-equivalents were measured utilizing the turbidometric Tina Quant assay (Roche[®]) in triplicate for all specimens after centrifuging. <u>Results</u>: The protein-free ultrafiltrate of CINB alone had a mean of 2.59 µg/mL of digoxin-equivalents. The DF + CINB solution yielded a mean digoxin-equivalent of 2.13 µg/mL compared to $1.52 \mu g/mL$ of the DB + CINB solution. Independent two tailed *t*-test showed a *p* < 0.0001 between the DB + CINB vs. DF + CINB; *p* < 0.0001 for DB + CINB vs. CINB and DF + CINB vs. CINB. <u>Conclusions</u>: Fab decreases the amount of free digoxin-equivalents. Digibind appears to be superior to DigiFab in this in vitro model at these particular concentrations. In vivo investigation is required to determine if this difference offers a therapeutic advantage.

205. SEVERE TOXIC REACTIONS TO EPHEDRA: NATIONAL TRENDS FROM 1993–2002

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<u>Objective</u>: *Ephedra* is a herbal remedy widely used for enhancing alertness, as a decongestant, and as a weight loss aid. We compared botanical products containing *ephedra* with non-*ephedra* products regarding the severity, outcomes, and trends of poison control center (PCC) toxicity reports. <u>Methods</u>: TESS was used to access the frequency and outcomes of cases of poisonings involving botanicals (excluding botanicals + drug exposures) reported from 1993–2002. Only cases with definitive outcomes were included in the analysis. Products were stratified to those containing *ephedra* only vs. combination ingredients. Hazard rates: mod + major severity + deaths per 1000 exposures were used to compare outcomes. <u>Results</u>: There were 43,133 toxic exposures to botanicals only reported to PCC over 10 years, of which 21,638 had definitive outcomes and were analyzed. Of these, 4307 (19.9%) had moderate or severe outcomes and 2 deaths, for an overall hazard score of 199 per 1000 exposures. There were 2604 exposures to *ephedra/Ma Huang* only and 10,690 to combination-botanicals. Comparisons revealed:

	Ephedra only	Ephedra combo	Other botanicals
Exposures	2,604	10,690	8,344
Mod + severe	649	2,855	803
Deaths	2	0	0
Hazard Rate (per 1000)	250	267	96

The frequency of *ephedra* only exposure reports to US PCC increased 20-fold over the 10 years of study. <u>Conclusion</u>: *Ephedra* products accounted for a significant number of exposures with severe outcomes. Hazard rate analysis suggests events involving *ephedra*-containing botanicals were over 2.5 times as toxic as other non-*ephedra* containing products. This supports concerns by policy makers that *ephedra* should be more closely regulated.

206. SAGE TEA RELATED CONVULSIONS IN A PEDIATRIC PATIENT

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<u>Background</u>: In the United States and Europe, there is increasing popularity for complementary medicines, including various herbal preparations. Contrary to their purported safety, these agents may produce several serious side effects, notably on the central nervous system. Dalmatian sage (*Salvia officinalis*), as an essential oil or extract, is a popular home remedy for various ailments, but may pose health risks because they contain epileptogenic monoterpene ketones (which





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include camphor and thujone). We report a case of convulsions in a pediatric patient following ingestion of tea made from dalmation sage. <u>Case Report</u>: A previously healthy 18 month-old female with 3 days of intermittent vomiting and diarrhea without fevers, was given a tea made from water and a home-grown herb. Two hours after drinking the tea, the child developed tonic–clonic contractions of the upper extremities, left eye deviation, and unresponsiveness that lasted less than 1 min. There was no prior history of convulsions. The child was evaluated in the ED, where she was afebrile with a normal physical exam, head CT, CBC, and serum chemistries. She was discharged home, but 18 hours after her initial ingestion, she developed three subsequent seizures requiring treatment with lorazepam. An EEG the following morning showed some parietal lobe slowing, interpreted as a possible seizure focus. A sample of the herb was identified by a botanist as *S. officinalis* or sage. <u>Conclusion</u>: Tea made from sage may have kindled convulsions in a child with a previously unmasked seizure focus.

207. MISDIAGNOSED FATAL MEADOW SAFFRON POISONING IN A TODDLER

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<u>Background</u>: Meadow saffron (*Colchicum autumnale*) is a plant widely found throughout Switzerland. In springtime its leaves can be confused with bear's garlic (*Allium ursinum*), which is collected for alimentary purposes. The mistake can lead to severe and even fatal colchicine poisoning. Colchicine is a tropolonic alkaloid which interferes with microtubular-dependent cell functions by blocking microtubular protein polymerisation. <u>Case Report</u>: A 3-year-old boy was referred to the hospital in a somnolent, confused, hypovolemic state, and cardiovascular shock. He had a 2 days history of vomiting, severe abdominal cramps, and profound diarrhoea. Laboratory testing: Plasma/serum osmolality 305 mosmol/kg (after 500 mL Ringer infusion), creatinine 172 µmol/L, BUN 25.5 mmol/L, ASAT 846 U/L, lactate dehydrogenase 8157 U/L, ammonia 800 µmol/L, and metabolic acidosis (pH 7.13, base excess -18.4). Despite aggressive medical management, the child died 5 hours after admission. Based on the symptoms and a plasma salicylate level of 0.41 mmol/L a diagnosis of Reye syndrome was made. However, 1 year later, a relative of the child read in a newspaper about two fatal cases of *C. autumnale* ingestion and remembered that the boy had eaten leaves on a meadow the day before the symptoms started. Meadow saffron was found at the place where the boy had been playing. Analysis of a serum sample still available revealed a colchicine level of 7 µg/L. <u>Conclusion</u>: Colchicine poisoning, either from ingestion of *Colchicum* leaves or colchicine tablets, should be considered as a differential diagnosis in cases of unexplained multiple organ failure.

208. AN OUTBREAK OF FOOD-BORNE ILLNESS ASSOCIATED WITH PLANT MATERIAL CONTAIN-ING RAPHIDES—CHICAGO, 2003

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<u>Background</u>: Many botanicals, particularly ornamental houseplants, contain raphides that have known toxic effects when chewed, including painful edema, vesicle formation, and dysphagia. We report a food-borne illness outbreak associated with ingestion of raphides. <u>Methods</u>: On 2/25/03, the Chicago Department of Public Health was notified of multiple cases of oral burning and facial edema associated with lunch in an office cafeteria on 2/21/03. The investigation included a case-control study, interviews with kitchen staff, an environmental inspection, and laboratory analysis of leftover foods. <u>Results</u>: Ten cases were identified, including one admitted to the ICU for potential airway obstruction secondary to severe edema, and two seen by ER staff for oral edema and pain. Ten of ten case-patients reported oral stinging and burning, and 8 of 10 reported dysphagia. Four of ten case-patients continued to have symptoms 2 weeks later. Food from the cafeteria's international buffet was consumed by 10 of 10 case-patients, and by 1 of 22 control subjects (odds ratio = undefined); each of the 10 case-patients reported consumption of a Chinese vegetable entrée from the international buffet, and had no other foods in common. Plant material from the Chinese vegetable entrée, which was brought to the attention of the cafeteria staff by a complainant, contained raphides. <u>Conclusion</u>: This outbreak was associated with consumption of raphides resembling those from common botanicals. Clinicians and public health practitioners should be aware of raphide-containing plants as a potential cause of food-borne illness.

209. POISONING BY SUSUMBER BERRIES

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<u>Objective</u>: To describe an unusual poisoning and the potential health effects of a common native dish. <u>Background</u>: Akee and salt cod is a common native dish in Jamaica. It is well known that akee may only be eaten when ripe because of the toxicity of hypoglycine in the unripe fruit. Not commonly known, is that, when the akee fruit is not ripe for picking that the "turkey berry" or susumber bean is substituted. Jamaican folk lore has it that this bean is also "poisonous" in the unripe state, but no descriptions of the toxicity are available. <u>Case Studies</u>: Six members of a Jamaican community ate a meal of salt fish and unripe turkey berries prepared in the traditional way at 1100, three of the members eating minute amounts. At 14 hours following the meal, the three who had eaten the greatest quantities, presented to the same emergency department. Two had gastrointestinal symptoms of diarrhea. All felt profoundly weak with facial paralysis, slurred speech, ataxia, and trunkel greater than peripheral weakness. Two of these patients required intubation and ventilation for respiratory failure and had a prolonged fluctuating course. Biological samples and extracts from remaining meal were tested for botulism, domoic acid, and other neurologic toxins. All tests were negative. Extracts from remaining susumber fruit revealed significant levels of solanine, the toxin found in the Solanaceae family, of which the susumber is a member. No measurable chaconine was found. <u>Conclusion</u>: These cases illustrate significant toxicity, neurologic and gastrointestinal, from the unripe susumber berry.

210. NEUROTOXICITY ASSOCIATED WITH 1-BROMOPROPANE EXPOSURE

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Background: 1-Bromopropane (1-BP) is an alternative to ozone depleting solvents. There is limited human toxicity data, however, animal data suggests that it may cause neurotoxicity. We report a series of patients chronically exposed to a glue containing 1-BP while working in a foam cushion factory. Case Series: Workers were exposed to glue containing 70% 1-BP, 0.3% 1, 2-epoxy butane, 10% styrene-butadiene rubber, and 20% rosin ester. Three individuals worked directly with the glue applying it with a spray gun and 3 other individuals worked in close proximity to the gluing station. No workers wore latex gloves and it is unclear if any workers wore approved respiratory devices. All the gluing was done in an poorly ventilated work area. The duration of exposure was 30-40 hours per week for approximately 3 months. All six workers experienced gradual onset of lower leg weakness, pain, difficulty in standing and walking, and numbness of the legs and feet. In addition, four workers also demonstrated hyperreflexia and hypertonicity of the lower extremities. Three workers complained of occipital headaches and two workers complained of dizziness and shortness of breath. All workers had elevated serum bromide levels ranging from 44 to 170 mg/dL and concurrent serum chloride levels ranging from 105 to 139 mmol/L. The mean air concentration of 1-BP obtained from the factory was 130 ppm (range, 91–176 ppm). Manufacturers of 1-BP recommend that workplace air concentrations remain below 25 ppm. An evaluation of the workplace did not reveal any substantial exposures to other known neurotoxins. Patient plasma bromide levels decreased after cessation of exposure but signs and symptoms of neurotoxicity persisted for more than 2 months. Conclusion: Chronic 1-BP vapor and dermal exposure was associated with chronic lower extremity neurotoxicity and elevated serum bromide levels.

211. LEAD DUST TRANSPORT FROM FIRING RANGES ON THE FOOTWEAR OF RECREATIONAL SHOOTERS—A PILOT STUDY

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<u>Background</u>: The purpose of this study is to determine if individuals who participate in target shooting transport lead dust from firing ranges on their footwear. <u>Methods</u>: Swipe samples were taken from the soles of footwear worn during one hour sessions of handgun firing on indoor ranges as follows: Immediately prior to entry onto the firing range (site A),



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after shooting for one hour (site B), after leaving the range and immediately prior to entering shooter's automobile (site C) and prior to entering shooter's home (site D). <u>Results</u>: Average lead levels obtained by surface swipe sampling of the footwear are listed below:

	Range 1 (mcg/ft ²)	Range 2 (mcg/ft^2)	Range 3 (mcg/ft^2)
Site A	<10	<10	<10
Site B	260	95.5	590
Site C	100	34.5	405
Site D	60	29	54.5

<u>Conclusion</u>: Lead dust is indeed transported on footwear from firing ranges to the home. This mode of transport for environmental lead is not widely known but may pose a cumulative health hazard for recreational shooters and their families.

212. LEVELS OF POLYCHLORINATED DIBENZO-P-DIOXINS (PCDDs), POLYCHLORINATED DIBEN-ZOFURANS (PCDFS), AND POLYCHLORINATED BIPHENYLS (PCBs) IN HUMAN MILK AT TWO US LOCATIONS

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Background: Polychlorinated biphenyls (PCBs), Polychlorinated dibenzo-p-dioxins (PCDDs), and Polychlorinated dibenzofurans (PCDFs) are globally distributed in the environment and in people. Human milk is an advantageous sample to monitor for exposure, and allows for the estimation of infant intake. Objective: To estimate the overall exposure of a local population to PCBs, PCDDs, and PCDFs from human milk samples. Method: The protocol from the Third WHO-coordinated exposure study was used. Ten primipara women from each lactation center (California and North Carolina) were conveniently sampled from December 2002 to January 2003. The milk at each location was pooled to form two samples. The milk was analyzed by HRGC/HRMS for 29 PCBs and 15 PCDDs/PCDFs congeners. All levels and toxic equivalent quotients (TEQ) were expressed as pg/g fat. The project was not eligible for human subjects review because the milk samples were pooled and no personal identifying information collected. Results: (1) The ages of the women participants at both locations ranged from 23 to 41 years. (2) The patterns of the PCB and PCDD/PCDF congeners at the two locations were similar. The PCDDs/PCDFs with the higher levels included OCDD, 1234678HpCDD, and 123678HxCDD. The TCDD and PeCDD levels were 0.80 and 2.26 for CA, and 0.92 and 2.63 for NC. The levels of the indicator PCBs (28, 52, 101, 138, 153, 180) were as follows: for CA 1755, 186, 368, 22,642, 24,218, 14,584; and for NC 1307, 322, 509, 14,609, 15, 868, 10,825. (3) The sum WHO-TEQ (PCB + PCDD/F) was 11.74 for CA and 11.84 for NC. Polychlorinated dibenzo-p-dioxins/polychlorinated dibenzofurans contributed to about 60% of the sum WHO-TEQ (PCB + PCDD/F). This was the same for CA and NC. Conclusion: These PCBs, PCDDs, and PCDFs levels suggest a decrease in the US population's exposure to these chemicals.

213. METHEMOGLOBINEMIA FROM NITRITE-CONTAMINATED PUNCH

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<u>Background</u>: A myriad of substances can induce methemoglobinemia (MetHb). A public health emergency occurred when sodium nitrite was accidentally added to a dry-powder punch mix. <u>Case Report</u>: Approximately 63 guests from a wedding reception presented to local ERs after consuming a peculiar-tasting punch. Within 15 min of ingesting the punch, they experienced headaches, dyspnea, dizziness, and nausea. Thirteen had cyanosis and nine collapsed. Pulse oximetry remained in the mid 80s despite oxygen therapy. Chocolate-brown colored blood was noted in all of the cyanotic patients. MetHb levels for all patients ranged from 1.0 to 60.2%. The mean MetHb level of the 20 hospitalized

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patients (13 children, 9 adults) was 14.4%. Thirty-five patients (19 hospitalized, 16 treated and released) were treated with methylene blue. All admitted patients were discharged the next day. Our poison center took calls on 98 people from the reception who were affected by the punch. All food and water was tested and the sodium nitrite concentration in the punch was 500–820 mg/L. An investigation revealed that sodium nitrite was accidentally substituted for citric acid during the processing of the punch mix. Two additional batches of the punch mix were recalled before distribution. A fourth batch was distributed and consumed by 13 people at a baby shower. Eight developed symptoms but none sought medical attention. <u>Conclusion</u>: This is one of the largest reported outbreaks of MetHb from food contamination. It emphasized the poison center's role in providing medical advice to treating physicians and for triaging the "worried-well" who called the poison center.

214. VISUAL SIDE EFFECTS FROM BOTULINUM TOXIN TYPE B INJECTIONS

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Background: Botulinum toxin B (BotB) has been used to treat hyperhidrosis and cervical dystonia. The most common side effects reported by the manufacturer in clinical trials are xerostomia, dysphagia, dyspepsia, and injection site pain. We describe three cases exhibiting visual symptoms after BotB injection. Case Report: Case 1. A 26 year-old male with a 6 year history of hyperhidrosis was injected with 20,000 units of BotB in both axillae and palms, on two different occasions. He developed blurry near vision in 4–7 days which lasted 2.5–3 months. While symptomatic examination by a neuro-ophthamologist revealed a parasympathetic abnormality of the ciliary muscles and papillary sphincters bilaterally. Case 2. A 20 year-old female with a 7 year history of hyperhidrosis, was injected with 10,000 units of BotB in both axillae. Two weeks later she developed blurry near vision and ptosis of her left eyelid. Her visual changes returned to baseline in 6 weeks. Case 3. A 32 year-old female with 13 year history of torticollis was injected with 30,000 units of BotB into her cervical musculature. She developed blurred vision when attempting to read 10 days post injection, which resolved in 8 weeks. Because of the side effects she was injected in the same site with 20,000 units, and no visual symptoms were noted. All of these patients elected to continue with a dosing regimen that alleviated the symptoms of their disease, despite the visual effects. Conclusion: Injection of BotB may result in prolonged but self-limiting ophthalmologic adverse effects. All of the patients chose to have blurry near vision over the symptoms of their disease. These patients did not report problems with daily living while suffering from visual changes. Whether this visual impairment will threaten the safety or livelihood of other patients is not known.

215. COLLABORATION OF MULTIPLE AGENCIES IN THE MANAGEMENT OF A HOME MERCURY SPILL

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<u>Background</u>: Prolonged inhalation of elemental mercury may result in significant organ damage (e.g., CNS symptoms, renal dysfunction, pneumonitis, necrotizing bronchiolitis, and death). Improper cleanup of a home mercury spill resulted in clinical symptoms, hospital admission, toxic urine mercury levels (>100 μ g/L), home levels 23 times acceptable measures, and environmental contamination resulting from furniture disposal. <u>Case Report</u>: A concerned neighbor contacted the regional poison control center (PCC). The PCC contacted both the OH EPA and the mother. Despite the advice by the PCC to vacate the premises, the family returned. To prevent further exposure, OH EPA involved the PCHD who has the authority to declare a residence unsafe for habitation. Subsequently, the OH EPA, PCHD, US EPA, ATSDR (Agency for Toxic Substances and Disease Registry), OSPERRA (Ohio Spill Planning, Prevention and Emergency Response Association), private physician and PCC collaborated on the care of this family, including chelation therapy, and decontamination of the environment. <u>Conclusions</u>: Successful collaboration of multiple agencies prevented prolonged symptoms and further environmental contamination. A protocol for interagency collaboration is described.

216. STROKE AND ENVIRONMENTAL LEAD EXPOSURE IN ADULTS: IS THERE AN ASSOCIATION?

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<u>Background</u>: An association between lead exposure and stroke has been theorized but never proven. The purpose of this study was to determine if there is an association between exposure to environmental lead and stroke in adults. <u>Methods</u>: Blood lead concentrations (BLCs) were measured in patients with recent hemorrhagic or ischemic, CT confirmed, stroke. Blood lead concentrations were also measured in non-stroke, hospitalized, control patients matched for age, sex, race, and hypertension. <u>Results</u>: Blood lead concentrations obtained from ischemic, hemorrhagic, and control patients are shown below. This data was analyzed using the Mann–Whitney U test (p = 0.714) as well as unpaired *t*-test (p = 0.705). Power analysis showed 80% power to detect a difference of 2 µg/mL between control and study patients.

	Mean BLC (µg/mL)	Median BLC (µg/mL)
Hemorrhagic stroke	2.82	3.00
Ischemic stroke	2.43	1.00
Controls	3.05	2.00

Conclusion: Stroke in adults does not appear to be associated with environmental lead exposure in our study population.

217. RELATIONSHIP BETWEEN ATMOSPHERIC LOW LEVEL OZONE CONCENTRATIONS AND ED VISITS

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<u>Background</u>: Ozone is the principal oxidant in photochemical air pollution which can cause increased airway reactivity. Previous studies have correlated high ozone levels with bronchospasm. We studied the effect of low level ambient ozone concentrations on emergency department (ED) visits and asthma. <u>Methods</u>: A computerized database of urban ED visits (annual volume 71,000) was retrospectively compared with daily ambient ozone concentrations averaged over 24 hours. Linear regression analysis was performed on data from every third day from April 1, 2002 to June 30, 2002. <u>Results</u>: ED visits ranged 168–243 patients daily. Hospital admissions from the ED ranged 39–75 patients daily. Ozone levels measured hourly ranged 0.0063–0.0468 ppm daily. There was no correlation between daily ED visits and ozone levels. A significant inverse correlation $(r^2 = 0.18)$ was found between all ED generated hospital admissions and daily ozone concentrations (P = 0.02). There was no correlation between levels. <u>Conclusion</u>: Ozone exposures well below the EPA national ambient air quality standard (0.12 ppm) have no relationship to total ED visits or the subset of ED asthma visits. With regard to total ED generated hospital admissions, ozone's exposure–effect relationship may not be one of an expected nature (i.e., linear), but may actually be paradoxical (i.e., biphasic or "U" shaped). An effect of hormesis may actually occur with low level ozone concentrations and total ED generated hospital admissions.

218. PLANNING HAZARDOUS MATERIALS EMERGENCIES RESPONSE: THE ROLE OF THE POISON CONTROL CENTER

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<u>Introduction</u>: After the chemical disaster in Seveso (Italy), where, in 1976, a release of TCDD contaminated 7 square miles and 706 citizens had to be evacuated, the European Community developed Council Directives (Seveso Directives) on the major-accident hazard which aim at the prevention of hazmat accidents and at the limitation of the consequences of such accidents for man and for the environment. The Directives also contain an obligation to prepare the internal and

external emergency plans for response measures and to regularly test them in practice. <u>Results</u>: In the province of Bergamo (1.070 square miles, 968.000 citizens, 58 high-risk chemical plants), our Poison Control Center has been planning hazardous materials incidents response since its institution in 1999, focusing on the following activities: chemical plants identification and collection of information of substances kept on site, collaboration, and coordination with emergency responders (EMS, Fire Department, Civil Protection, Regional Environment Protection Agency) in particular during alert activation and sharing information, elaboration and dissemination of toxicological treatment guidelines, antidotes stocking and distribution, training and education for first responders of industries and EMS personnel. The result is a computer-aid procedure, which use metafile documents, with a database of the most important chemical substances (over 600) used and stocked in the chemical plants linked to the simplified (one page) medical treatment procedures. <u>Conclusion</u>: The optimal response to hazardous materials emergencies need a foregoing plan with validated operating procedures, where the poison center's role is strictly integrated with all the different agencies involved for resolving the accident.

219. DETECTION OF A TOBACCO-SPECIFIC CARCINOGEN IN THE URINE OF CHILDREN EXPOSED TO ENVIRONMENTAL TOBACCO SMOKE

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<u>Background</u>: The US Environmental Protection Agency classifies environmental tobacco smoke (ETS) as a known human carcinogen. Studies of adult volunteer nonsmokers exposed to ETS have revealed the presence in urine of a tobacco-specific nitrosamine metabolite (NNAL), a potent rat-lung carcinogen, and its glucuronide (NNAL-Gluc). Although ETS plays a role in numerous childhood diseases, NNAL and NNAL-Gluc have not been investigated in children under age 3 years (except in newborns exposed via the placenta from mothers who smoked during pregnancy). Because ethical considerations preclude studies of children exposed intentionally to ETS, we conducted a pilot study of pediatric emergency department (PED) patients exposed incidentally to ETS at home. <u>Methods</u>: We enrolled, via convenience sampling, patients 0-3 years of age presenting to two PEDs for medical evaluation. If parents consented and surplus urine became available during evaluation, we analyzed urine for NNAL and NNAL-Gluc by gas chromatography/nitrosamine selective detection. <u>Results</u>: We detected NNAL (total NNAL range, 0.13-4.11 pmol/mg Cr) and NNAL-Gluc in the urine of 10 of 11 children (mean \pm SD, 1.04 ± 0.92 years) exposed to ETS. <u>Conclusion</u>: Urine samples of children under age 3 years exposed to ETS reveal the presence of a potent rat-lung carcinogen derived specifically from tobacco.

220. EFFECTS OF MUSTARD GAS EXPOSURE IN PEDIATRIC PATIENTS (LONG TERM HEALTH STATUS OF MUSTARD-EXPOSED CHILDREN, 14 YEARS AFTER CHEMICAL BOMBARDMENT OF SARDASHT)

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Mustard agents are chemical weapons which act through alkylation of cellular components, causing acute symptoms which include severe blistering of tissue exposed to vapor or liquid agent and a diverse range of chronic illnesses. During the 8 year war between Iran and Iraq, extensive employment of chemical munitions by Iraqi forces was documented, including attacks on both military and civilian targets. One of these incidents, an aerial bombardment of the Iranian border town of Sardasht in June 1987, is the focus of the present investigation. Here we report on distribution of mustard-induced lesions among 20 female and 30 male victims less than 10 years of age at the time of exposure. Physical examinations revealed that lesions of the lungs were most common (100%), followed by skin (98%), and eye (86%) lesions, with 0–8% classed as severe; 4–16% as moderate; and 82–84% as mild lesional coverage. It was also noted that individuals exposed to the agent as children exhibited more severe chronic effects than adults exhibiting comparable acute symptoms. Results of this investigation yield insight into the extend which mustard exposure affects a particular symptom (lesions for this study) in adults when exposure occurs in childhood.





221. OSMOTIC ACTIVITY OF ETHANOL IN SALINE AND SERUM

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<u>Background</u>: Calculations to determine the serum osmolal gap require a term to account for ethanol's contribution to total osmolality. To convert measured ethanol (EtOH) concentrations in mg/dL into relevant units, one divides by 4.6 to yield mOsm/L, or by ~4.2 to yield mOsm/kg which corrects for serum water content of roughly 0.93 L/kg. Several studies, however, have empirically shown that EtOH contributes more osmotic activity than predicted by its serum concentration in mg/dL, and the proper conversion factor is somewhat <4.2. Thus, EtOH has an in vivo "osmotic activity coefficient" >1, probably in the 1.12–1.25 range. We sought to determine whether this non-ideality of EtOH in solution was due to EtOH itself or to an interaction of EtOH with serum. Methods: Saline (0.9%) and pooled human serum were spiked with EtOH in concentrations ranging from 0 to 500 mg/dL. A basic metabolic panel, EtOH level, and osmolality by freezing-point depression method were concurrently determined for each sample. The slopes of the EtOH concentration vs. total osmolality lines were determined by linear regression and compared. Results: The slope of the EtOH concentration vs. total osmolality line was 0.209 ± 0.0019 mOsm dL/mg kg in saline and 0.222 ± 0.0084 mOsm dL/mg kg in serum. These slopes were not significantly different (p = 0.17). Conclusion: The osmotic activity of EtOH is similar in 0.9% saline and serum, and is close to that predicted by its measured concentration. The greater osmotic activity coefficient of EtOH seen in vivo is therefore likely due to an interaction of EtOH with blood's cellular components.

222. ARTERIAL AND VENOUS DIFFERENCES IN POSTMORTEM MORPHINE CONCENTRATIONS IN HEROIN OVERDOSE DEATHS

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<u>Background</u>: Postmortem morphine concentrations are often used to assist in determining cause of death. Limited animal and human data suggest central sampling sites yield higher drug concentrations than peripheral sites. However, it is not known whether there are differences in morphine levels between artery and vein at a given site or between left and right ventricle. <u>Methods</u>: Suspected heroin overdoses (n = 29) had femoral artery and vein, left and right ventricle and pooled heart blood samples obtained at autopsy. Specimens were stored in refrigerated sodium fluoride tubes. Morphine (free and total) concentrations were determined by GC/MS. Site differences are reported as ratios and compared by signed rank test. <u>Results</u>: The femoral artery to vein ratio for total morphine was 1.2 (range 0–4.5) $p \le 0.282$. The ratio was greater than 1 in 18 cases and less than 1 in 13 cases. The ratio for left heart to right heart total morphine was 1.1 (range 0.4-3.2) $p \le 0.85$. Left ventricular to femoral vein total morphine ratio was 2.0 (range 0.6–6.9) $p \le 0.001$. <u>Conclusion</u>: In heroin OD deaths, femoral artery and vein morphine levels are usually similar, although up to 4.5 fold differences were noted in some cases. Left and right ventricular morphine concentrations were usually similar although up to 3.2 fold differences were noted (L side higher). Centrally obtained morphine levels are on average twice as high compared to peripheral morphine concentrations.

223. MONITORING OF OCCUPATIONAL EXPOSURE TO METHYLENE CHLORIDE: SAMPLING PROTOCOL AND STABILITY OF URINE SAMPLES

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<u>Background</u>: Methylene chloride (MeCl₂) is a volatile halogenated hydrocarbon with various industrial applications. It has irritant, defattening, and especially neurotoxic, cardiotoxic and carcinogenic properties. Biomonitoring is needed in occupationally-exposed workers and the ACGIH has noticed an intended BEI. <u>Objective</u>: To establish a sampling

protocol for biomonitoring occupational exposure to MeCl₂ by analysis of urine. Adequate sampling of MeCl₂ is essential as it is a highly volatile solvent. <u>Methods</u>: An analytical procedure for gas chromatograph based on solid phase microextraction technique (SPME) was developed. MeCl₂-spiked urine samples were tested for stability at room temperature and refrigeration. The effect of the time elapsed until clamping the vial was also evaluated. <u>Results</u>: MeCl₂-spiked urine samples stored in headspace vial (HSV) showed a 31% decrease in the first 4 days at room temperature. The concentration remained stable up to 3 weeks thereafter. Refrigeration did not improve recovery. The time elapsed from introducing the spiked urine into the HSV until clamping had a major effect on MeCl₂ concentration, 72.5% and 61% of control after 5 and 15 min, respectively. <u>Conclusions</u>: Because of the volatility of MeCl₂, time to clamping of the HSV should be no longer than 5 min. Clamped HSV can be stored at room temperature until analysis, but no longer than 3 weeks. Standard samples should be prepared on the same day of test sample collection and handled under the same conditions. This will prevent dissimilar loss of MeCl₂ due to its volatility.

224. IN-VITRO EFFECTS OF QUETIAPINE ON TRICYCLIC ANTIDEPRESSANT IMMUNOASSAYS

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<u>Background</u>: Quetiapine is a dibenzothiazepine antipsychotic agent with structural similarity to the tricyclic antidepressants (TCA). The potential for quetiapine to cross react with tricyclic antidepressant immunoassays was investigated in vitro. <u>Methods</u>: Quetiapine was added to nine samples of pooled drug free plasma at concentrations from 1 to 640 ng/mL that were verified by gas chromatography. No quetiapine metabolites were present. The samples were tested using the Abbott Tricyclic Antidepressant TDx assay on the TDxFLx (Abbott) in two separate laboratories, the Syva[®] Emit[®] toxTM Serum Tricyclic Antidepressant Assay on the AU400 (Olympus), and the S TAD Serum Tricyclic Antidepressant Screen on the ACA[®]-Star 300 (Dupont) autoanalyzers. The TDxFLx assay is quantitative, while the Emit and S TAD assays are qualitative screening assays with a threshold of 300 ng/mL for TCA positivity. <u>Results</u>: The quantitative assay showed a concentration-related TCA cross-reactivity beginning at quetiapine concentrations ≥ 5 ng/mL. The 640 ng/mL spiked sample produce TCA results of 379 and 385 ng/mL in lab 1 and lab 2 respectively. The qualitative assays were screened as positive at 160 and 320 ng/mL for the S TAD and Emit assays, respectively. <u>Conclusion</u>: Quetiapine cross reacts with quantitative and qualitative TCA immunoassays in a concentration dependent fashion. Therapeutic use or overdose of quetiapine may result in a false-positive TCA immunoassay result.

225. ERYTHROCYTE ACETYLCHOLINESTERASE ACTIVITIES ACCORDING TO AGE IN KOREANS

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<u>Objectives</u>: Acetylcholinesterase (AchE) activities in patients intoxicated with organophosphates or carbamates are important to evaluate the severity and progress of the intoxication. This study was conducted to obtain basal AchE activities according to age in Koreans. <u>Methods</u>: 210 healthy subjects, who were randomly recruited and grouped according to age, participated in this study. Erythrocyte AchE levels were measured colorimetrically. <u>Results</u>: The mean (\pm SD) AchE activities in age of <1, 1–5, 6–10, 11–20, 21–30, 31–40, 41–50, 51–60, and >60 groups were 24.1 \pm 9.2, 36.9 \pm 5.5, 41.0 \pm 5.4, 41.0 \pm 7.8, 37.8 \pm 5.5, 35.1 \pm 5.6, 37.9 \pm 12.6, 33.4 \pm 4.2, and 37.7 \pm 2.1 U/gHb, respectively. There were statistically significant differences in AchE activities between different age groups (One-way ANOVA, p < 0.0001). The AchE activity of infant group was significantly lower than those of all other age groups (Bonferroni test, p < 0.001). A significant difference in AchE activity was found between male and female in age of 1–5 group (unpaired *t*-test, p = 0.009), while there were no significant differences between both sexes in all other age groups. <u>Conclusion</u>: Infant group had lower AchE activity than those of other age groups, while the AchE activities in older children and adults showed no difference between age groups. These data are similar to results previously reported in other ethnic groups.

226. RAPID CYANIDE DETECTION USING THE CYANTESMO[®] KIT

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<u>Background</u>: Sources of cyanide exposure are many, including combustion of plastics and vinyls, laboratory or industrial exposures including exposure in the electroplating industry both of printed circuit boards and in jewelry work. Rapid and definitive diagnosis of cyanide poisoning is, currently, unavailable in the emergency department setting. We investigated a technique utilized by water treatment facilities to detect cyanide to see if it could be applied to rapidly detect concentrations in the clinically important range. <u>Methods</u>: Dilutions of KCN ranging from $0.25 \,\mu\text{g/mL}$ to $30 \,\mu\text{g/mL}$ were acidified with a drop of sulfuric acid in a closed system under a ventilation hood. Cyantesmo[®] test strips were placed into the test tubes above the fluid level where liberated HCN gas interacted with the test strip to effect a color change. Color changes were compared to negative controls and to each other. <u>Results</u>: The test strips demonstrated an incrementally increasing deep blue color change over a progressively longer portion of the test strip in less than 5 min for each concentration of KCN including 1, 3, 10, and $30 \,\mu\text{g/mL}$. The concentrations of 0.25, 0.5, and 0.75 required more than 2 hours to begin to demonstrate any color change. <u>Conclusion</u>: The Cyantesmo[®] test strips accurately and rapidly detected in a semi-quantifiable manner, concentrations of CN greater than $1 \,\mu\text{g/mL}$ contained in each test sample. Future work to validate this test in blood and in clinical specimens, is planned.

227. FALSE POSITIVE ETHYLENE GLYCOL LEVEL

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Background: Enzymatic assays may be unable to differentiate ethylene glycol (EG) from propylene glycol (PG). Therefore, gas chromatography (GC) is the gold standard for measuring serum EG. We present a case of an acidotic patient who received fomepizole after an enzymatic assay provided a false positive EG level due to PG. <u>Case Report</u>: A 55 yr female presented with encephalopathy and abdominal pain after a chronic acetaminophen (APAP) overdose, but without history of EG exposure. *Admit labs*: AST 6012 U/L, ALT 2310 U/L, calcium 8.3 mg/dL, BUN 40 mg/dL, creatinine 2.5 mg/dL, acetaminophen 1 mcg/mL, pH 7.25, HCO₃ 11 mEq/L, lactate 4.0 mmol/L, and urinalysis with 2+ protein, and trace blood. *N*-Acetyl cysteine was administered, and i.v. lorazepam was given for agitation. The acidosis and renal failure prompted measurement of serum EG which was 12 mg/dL. The patient was treated with one dose of fomepizole. The liver failure resolved, and the acidosis improved with hydration and without hemodialysis. It was believed that the liver failure failure failure for the acidosis and renal failure. Because of this, the initial serum sample was analyzed again using the enzymatic method producing an EG level of 10 mg/dL. However, confirmatory testing of this sample using GC with flame ionization detection was negative for EG, but PG was present at 8 mg/dL, presumably from the lorazepam. <u>Conclusion</u>: Physicians should be aware of which analytical method is used to determine serum EG levels. Enzymatic assays may result in false-positive results leading to incorrect diagnoses and unnecessary treatments.

228. COMPARISON OF URINARY PARANITROPHENOL AND PLASMA/RBC CHOLINESTERASE MEASUREMENTS IN THE EVALUATION OF DOMESTIC METHYLPARATHION EXPOSURE

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<u>Background</u>: Methylparathion, an organophosphate pesticide was illegally used to spray homes for cockroach control. An investigation was performed to determine if this was causing any adverse health effects. Plasma and RBC cholinesterase levels provide a measure of the degree of enzyme inhibition and paranitrophenol (PNP) is a metabolite of methylparathion. <u>Methods</u>: Homes were sampled in several rooms using 10 cm^2 adsorbent sheets. These were analyzed for methylparathion by GC/MS. Residents of homes with high levels of methylparathion (>150 µg/100 cm²) were considered the high-exposure group and compared with those from homes with zero or extremely low levels

 $(<15 \,\mu\text{g}/100 \,\text{cm}^2)$. RBC, plasma and total cholinesterase measurements and urinary PNP levels were obtained and compared to the likelihood of subacute toxicity based upon evaluation by a medical toxicologist. <u>Results</u>: There were no differences in any of the cholinesterase measurements between the low-exposure and high-exposure groups or between those felt to have subacute toxicity and those who did not. The total cholinesterase had the least variability, CV—15%, compared to CVs of 23% for plasma and 21% for RBC. The urinary PNP levels were highest in the asymptomatic subjects with an average of 183 ppb compared to an average of 71 ppb for the group felt to have subacute toxicity (p > 0.05). <u>Conclusion</u>: Neither cholinesterase nor urinary PNP measurements were useful in identifying those individuals who may have subacute symptoms resulting from chronic, domestic methylparathion exposure.

229. CARBARYL INHIBITION OF PLASMA CHOLINESTERASE ACTIVITY

Long H, Kirrane B, Nelson LS, Hoffman RS. New York City Poison Center, New York City, New York, USA.

<u>Objective</u>: Carbamate-inhibited cholinesterases undergo spontaneous regeneration in-vivo. Confirmatory testing may be misleading if regeneration occurs in-vitro. Our objective was to determine under what conditions plasma obtained for laboratory analysis following suspected carbamate exposure could be used as a reliable indicator of exposure. <u>Methods</u>: Carbaryl (Sevin[®]) was added to pooled fresh frozen plasma (FFP) to produce plasma cholinesterase (PChE) inhibition. Using the Michel method, PChE activity was measured at baseline and at successive time intervals until it returned to baseline. To simulate specimen collection practices, enzymatic activity was measured in specimens maintained at ambient temperature (77°F) and refrigerated at 38°F. Plasma cholinesterase activity was then compared to that of FFP to which an organophosphate malathion had been added (positive control) and to FFP to which no carbaryl had been added (control [C]). Specimens were run in triplicate, and results are expressed as means. <u>Results</u>: Plasma cholinesterase activity returned to baseline by 48 hours following addition of carbaryl to FFP in specimens maintained at ambient temperature but did not differ significantly from control in specimens maintained at 38°F. (See Table).

Agent	C at 1 hour (%)	C at 24 hours (%)	C at 48 hours (%)	C at 53 hours (%)
Carbaryl 77°F	16	41	83	88
Carbaryl 38°F	_	15	18	17
Malathion 77°F	_	14	14	
Malathion 38°F	—	17	26	—

<u>Conclusion</u>: Refrigeration of specimens may improve reliability of results. These findings may vary with different carbamates.

230. EXPOSURE ASSESSMENT OF CHILDREN EXPOSED TO ARSENIC IN AN URBAN PLAYLOT

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<u>Background</u>: In June 2001 local public health authorities closed an urban playlot due to a 10×10 foot square hot spot of arsenic (As) contamination (>800 ppm). <u>Methods</u>: Health authorities conducted an 8-week exposure assessment that began 2 months after abatement that included exposure questionnaires, health assessments, spot (creatinine adjusted) urine As, and 24-hour urine As. Bioassays were analyzed using ICP-MS. <u>Results</u>: 216 children were evaluated (51 spot and 24-hour urine; 88 spot urine only; 87 24-hour urine only). Geometric mean age of the children was 6.89 years (range 2 months to 18 years); 53% male. None of the children reported any symptoms or demonstrated any signs of acute or chronic arsenicosis. Geometric mean 12.7 mcg/gCr of the spot urine samples had a range 0.35–87.17; geometric mean 8.5 mcg/L of the 24-urine samples had a range 7.5–101.0 (lowest detection limit = 15, 1/2 detection limit = 7.5). <u>Conclusion</u>: Children's exposure to As from CCA treated wood is currently an area of active public health investigation. Since urine assays were collected 2 months after abatement and no child had evidence of arsenicosis, these data most likely represent background levels of As burden in children living in urban housing developments. Normative data on As distributions in pediatric populations are sparse, and these data provide useful comparative data for other exposure assessment studies.



231. CLINICAL OUTCOMES IN INORGANIC MERCURY SALTS INGESTIONS

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<u>Background</u>: Ingestion of elemental mercury is generally not accompanied by major adverse effects. However, ingestion of inorganic mercury salts may cause severe gastrointestinal corrosion, renal failure and death, with acute lethal doses ranging from 1 to 4 g. <u>Methods</u>: We analyzed and present clinical material from nine patients, hospitally treated, after ingestion of various amounts of mercury chloride. <u>Results</u>: All treated patients presented with upper gastrointestinal complaints and underwent endoscopies that showed caustic lesions in the upper gastrointestinum. Four of them (44%) had life-threatening upper gastrointestinal bleeding, along with numerous esophagus and stomach caustic lesions, and spread fibrinonecrotic plagues, deepest in the prepyloric region. Five (55%) of them developed acute renal failure. Four patients (44%) underwent dialysis. Antidotal therapy was given to two patients only, because of unreadily availability or lack of opportune timing. The overall lethal outcome of the group was 3 (33%) even after dialysis, antidote administration, and conservative treatment for the upper gastrointestinal injuries. <u>Conclusion</u>: Inorganic mercury salts ingestions present with a broad spectrum of fast-developing clinical impairments, have various and often poor clinical outcomes. Our clinical experience emphasizes preparedness to administer on-time, multifaceted/combined treatment, including antidotal, dialysis, and other approaches in all suspected cases.

232. A CASE OF SEVERE IATROGENIC BISMUTH POISONING

Dargan PI,¹ Bailey CA,² Greene SL,¹ Murray SA,² Jones AL.¹ ¹National Poisons Information Service (London), Guy's and St. Thomas' NHS Trust, London, UK; ²The North of England Bone and Soft Tissue Tumour Service, Freeman Hospital, Newcastle, UK.

Background: Bismuth Iodoform Paraffin Paste (BIPP) is widely used for packing small wounds and cavities in maxillofacial surgery. Case Report: A 66 year old man presented with a sacral mass due to a chondroid chordoma. He required extensive surgery, the tumour was excised and the sacrum reconstructed with an allograft internally fixed to the lumbar vertebrae. Two weeks later his sacral wound broke down exposing the metalwork. He was taken to theatre and the wound was irrigated and packed with BIPP-soaked gauze. 5 days later he developed acute confusion and gram-negative septicaemia. His confusion failed to settle with treatment of the sepsis and he developed a tremor. Blood and urine bismuth concentrations were checked and were raised (340 and $2800 \,\mu g/L$ respectively). The BIPP packing was therefore removed and he was started on 2,3-dimercaptopropane-1-sulfonate (DMPS) which he received for 61 days (initially IV 5 mg/kg qds for 5 days, then 5 mg/kg tds for 5 days and 5 mg/kg bd for 17 days; then oral 200 mg tds for 10 days; and 200 mg bd for 14 days). His confusion and tremor improved gradually over the next month and his bismuth concentrations were normal after 55 days of chelation; his renal function remained normal throughout. He was discharged after 12 months of rehabilitation; 7 months later his sacral wound has reduced in size and continues to heal and he has no chronic neurological sequelae. Conclusions: We describe a case in which BIPP was used to pack a large wound after pelvic surgery resulting in significant bismuth toxicity requiring DMPS chelation. If BIPP is used in large quantities rather than for its standard maxillo-facial indications, careful patient monitoring, with urine bismuth concentrations, is probably warranted.

233. BENIGN OUTCOME WITH TOXIC SERUM IRON LEVELS FOLLOWING IV IRON DEXTRAN OVERDOSE IN THREE PATIENTS.

Beuhler MC, Wallace KL. Good Samaritan Regional Medical Center, Phoenix, Arizona, USA.

<u>Background</u>: Iron dextran (FeDex) is an IV formulation used in patients with contraindications to oral iron supplementation. We describe three patients accidentally overdosed with FeDex. <u>Case Series</u>: Three TPN-dependent patients, ages 9 y, 7 y, and 22 m were admitted for coagulase-negative line sepsis. All were scheduled to receive one weekly dose of 0.7 mg/kg FeDex supplementation in their TPN. On a single occasion, as a result of a pharmacy error,

	Iron (µ	g/dL)	Serum CO	2 (mmol/L)	AST/AI	.T (U/L)	
	24 h	48 h	24 h	48 hr	48 h	72 h	Follow-up
9 y	902	677		25	21/36	26/41	15 days
7 y	952	686		30	28/28		37 days
22 m	1,000	775	24	23	88/27	58/40	60 days

each instead received 7 mg/kg. Serum iron levels, electrolytes and hepatic enzyme levels were obtained over 48 hours following parenteral FeDex overdose:

During the first 48 hours following overdose, none developed nausea, vomiting, diarrhea, fever, or evidence of systemic toxicity. All were re-admitted at the follow-up times shown above for recurrent line infections. Each had been well until the date of re-admission. None of the patients had evidence of hepatotoxicity at re-admission. <u>Conclusion</u>: Although significantly elevated iron levels occurred in our three patients after parenteral overdose of FeDex, there was no evidence for acute or subacute toxicity.

234. DARKNESS ON THE EDGE OF TOWN: A RURAL FAMILY MALICIOUSLY POISOINED BY THALLIUM

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<u>Background</u>: Thallium was banned as a rodenticide in 1975. Now acute thallium poisoning is rarely seen outside of the industrial setting. <u>Case Report</u>: A 41 year-old female was admitted to the hospital for increasing lower extremity weakness and back pain. Within 2 days, rapidly ascending paralysis and respiratory failure necessitated intubation. A 24 hour urine 13 days after admission revealed a thallium level of 5000 mcg/mL, and the serum thallium was 471 ng/mL 14 days after admission. Prussian blue (250 mg/kg/day) was administered orally for 17 days, until the serum thallium level was 41 ng/dL. Her hospital course progressed from alopecia, quadriparesis, hyporeflexia, and disconjugate gaze, to recovery of some facial muscles and diaphragm strength, but still requiring pressure support ventilation and a tracheostomy. Her 10 year-old son with reported elevated levels and peripheral neuropathy was treated with Prussian blue. Other possible victims included an 18 year-old son, a visitor with gastrointestinal symptoms and hair loss, and a visiting dog that died after a two day course of vomiting and diarrhea and the inability to ambulate. <u>Conclusion</u>: Thallium poisoning should be considered in a case of rapidly ascending paralysis. If the diagnosis is made, surveillance of associates of the patient must also be made because of the high possibility of homicide.

235. ARSENIC AND LEAD SOIL CONTAMINATION NEAR A HEAVY METAL REFINERY IN THE ANDES MOUNTAINS

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<u>Background</u>: In a remote town in the Andes Mountains in Peru lies one of the world's largest lead smelting plants. Because the smelting process releases arsenic and other heavy metal particulates into the air, we hypothesized that arsenic and other heavy metals may be present in the soil in higher than normal quantities. <u>Methods</u>: We conducted random soil sampling at seven sites within an approximately 10 kilometer radius of the plant, and one site approximately 220 kilometers from the plant. Using appropriate chain of custody and USDA soil transfer permits, soil samples were sent to the United States for analysis of eight heavy metals: arsenic, barium, cadmium, chromium, lead, selenium, silver, and mercury. <u>Results</u>: Mean arsenic and lead concentrations were 627.2 and 1200.1 ppm, respectively. At the control site 220 kilometers away in Lima, Peru arsenic, and lead concentrations were 25.6 and 379 ppm, respectively. Superfund cleanup goals at toxic sites in the United States range from 5–65 ppm to 200–500 ppm for arsenic and lead respectively.



<u>Conclusion</u>: Arsenic and lead were found in the soils of this remote town in Peru at many times the Superfund cleanup goals. Because over 30,000 people live in close proximity to the lead smelting plant, human exposure to these heavy metals is possible and human health assessments should be conducted.

236. BLOOD LEAD LEVELS IN PEOPLE LIVING NEAR A HEAVY METAL REFINERY: A CAUSE FOR CONCERN AND FURTHER STUDY

Krause, E,¹ Nussle P,¹ Santana D,² Wilson, B.¹ ¹Joining Hands Against Hunger, St. Louis, Missouri, USA; ²Filomenas Clinica, La Oroya, Peru.

<u>Background</u>: In a remote town in the Andes Mountains in Peru lies one of the world's largest lead smelting plants. Based on anecdotal stories from the local midwifery clinic and reported lead dust emissions from the plant, we supplied materials to the clinic to start a lead screening program so people could find out what their blood lead levels are. <u>Methods</u>: The first 77 people who showed up at the midwifery clinic were screened, including three United States visitors. The portable LeadCare Analyzer with accompanying lead-free supplies and training video from ESA, Inc., D-Wipe cleaning cloths from Esca Tech, Inc., and sterile alcohol swabs were used to conduct the fingerstick screening. <u>Results</u>: All the blood lead levels of the local people were above the World Health Organization's and Centers for Disease Control's safe threshold of 10 mcg/dL.

Blood lead level (mcg/dL)	Number of people (Peru)	Number of people (US)
>60	21	0
45-60	9	0
20-45	39	0
10–20	5	0
0–10	0	3

<u>Conclusion</u>: All blood lead levels of the people living near the lead smelter were above 10 mcg/dL and further human health assessments should be conducted.

237. LEAD-TAINTED HERBAL REMEDY USED FOR DEVELOPMENTAL DELAY

Schier JG, Hoffman RS, Nelson LS. NYC Poison Center, New York City, New York, USA.

Introduction: Herbal medicines sometimes contain contaminants that have the propensity to cause significant toxicity. We report a case of a child who was being treated with an herbal medicine for developmental delay that contained an unusually high concentration of lead. Case Report: A 14-year-old female developed a multitude of nonspecific gastrointestinal and neurologic symptoms for which she was hospitalized on two separate occasions. She had a history of mild developmental delay for which she was recently started on a Hindu herbal remedy by her parents. Her major complaints during this time period included: nausea, vomiting, abdominal pain, generalized weakness, and headache. Physical and neurologic examinations were within normal limits and no etiology for her symptoms was found. A serum lead level sent during the second hospitalization was 112 (μ g/dL and the patient was called back to the hospital for chelation therapy. Routine laboratories (chemistry, urinalysis, and abdominal radiograph) were within normal limits with the exception of a complete blood count, which revealed a mild microcytic anemia (hemoglobin, 9.6 g/dL; hematocrit, 29%; mean corpuscular volume, 82 fL). Her treatment regimen included oral succimer and intravenous edetate calcium disodium (EDTA). Analysis of the herbal product revealed a lead concentration of 8000 parts per million. She was discharged to home in good condition after completion of intravenous EDTA to finish her oral succimer regimen. Conclusion: Herbal medicines may contain contaminants such as lead, which have the propensity to cause toxicity.

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238. CANINE ZINC TOXICOSIS FROM INGESTION OF A DECORATIVE BATHROOM FIXTURE

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<u>Background</u>: Most zinc toxicoses in animals are associated with the ingestion of pennies minted after 1982. Other sources of zinc reported in the literature include cage wires and cage nuts, zinc oxide ointment, and brass buttons. We report the development of zinc intoxication in a dog following ingestion of an ornamental brass knob from a toilet paper holder. <u>Case Report</u>: A 3 year old, male, neutered, 15.4 kg Welsh Corgi presented to a local veterinary clinic with a 2-week history of intermittent vomiting, inappetance, and lethargy. The dog did not respond to symptomatic treatment, developed diarrhea, and 2 days later presented to a veterinary emergency clinic with fever (40.3°C), icterus, and abdominal pain. Urine and blood collected at that time had a brown-black color. Serum chemistry analysis indicated severe hemolysis, anemia, and acute renal failure. A radiograph revealed a large, radio-opaque object in the stomach. The dog was immediately transferred to a veterinary teaching hospital where supportive care, administration of calcium EDTA, and blood transfusions were initiated in anticipation of surgery to remove the foreign body. The dog was euthanized 24 hours post-surgery due to the development of anuric renal failure. Blood drawn at the time of surgery had a serum zinc concentration of 89.8 ppm (N = 0.7-2.0 ppm). The retrieved object had a shiny brass-like coating that was pitted, and weighed 79.1 g. <u>Conclusion</u>: The serum zinc concentration in this dog is the highest reported concentration found in the literature. Ingestion of any zinc-containing metal object can potentially result in a severe zinc toxicosis, and zinc intoxication should be suspected when hemolysis is accompanied by the finding of a metallic object in the gut.

239. COMPARISON OF "NORMAL" REFERENCE RANGES FOR SELECTED HEAVY METALS WITH BIOMONITORING EXPOSURE DATA OF US POPULATION

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<u>Background</u>: The 2nd National Report on Human Exposure to Environmental Chemicals by the National Center for Environmental Health (NCEH) released in 2003 reports biomonitoring exposure data on a US sampling of participants in the National Health and Nutrition Examination Survey (NHANES). We compared the 95th percentile of selected heavy metal urine concentrations in the NCEH report with the "normal" reference ranges provided by a sampling of commercial toxicology laboratories. <u>Methods</u>: We contacted 5 commercial laboratories across the country that routinely perform a large variety of heavy metal analyses. Each lab director was asked to provide their analytical technique, the reported normal reference range, and the origin of that reference range for a variety of metals included in the NCEHs report. The normal reference ranges for creatinine corrected urinary concentrations of antimony, cadmium, thallium, and uranium were compared to the 95th percentile in the NCEH report. <u>Results</u>: For thallium, the NCEH 95% was 0.305 mcg/g creat with the commercial labs normals ranging from <0.4 to <1.0 mcg/g creat. For cadmium the NCEH 95% was 1.13 mcg/g creat and the commercial labs normals ranged from 1.2 to 3.0 mcg/g creat. For uranium and antimony only one lab could be identified that reported creatinine corrected urine normal ranges: uranium NCEH 95%was 0.034 mcg/g creat, commercial lab normal <0.013 mcg/g creat and antimony NCEH 95% was 0.352 mcg/g creat and commercial lab normal <0.013 mcg/g creat and antimony NCEH 95% was 0.352 mcg/g creat and commercial lab normal <0.013 mcg/g creat and antimony NCEH 95% was 0.352 mcg/g creat and commercial lab normal <0.013 mcg/g creat and antimony NCEH 95% was 0.352 mcg/g creat and commercial lab normal <0.013 mcg/g creat and antimony NCEH 95% was 0.352 mcg/g creat and commercial lab normal <0.014 mcg/g creat. <u>Conclusion</u>: Relatively small variations exist betwee

240. MERCURY AND CADMIUM TOXICITY IN A HAITIAN VOODOO MINISTER THAT RESULTED IN ACUTE RENAL FAILURE

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Background: Mercury (Hg) is widely used during religious ceremonies in some Latin American and Caribbean cultures. Multiple cases of Hg poisoning related to these ceremonies are reported but few are reported with cadmium (Cd).

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We report the case of acute renal failure (ARF) developing after a suicide attempt in a Haitian voodoo minister with elevated Hg and Cd levels measured. <u>Case Report</u>: A 53 year-old woman with a past medical history of hypertension presented to the ED after she was observed to have a 10 min episode of tonic-clonic seizure-like activity followed by fecal and urinary incontinence. Early that monring, she complained of nausea, abdominal pain, and had multiple episodes of vomiting. On hospital day two, she was noted to have a serum creatinine (SC) of 6.3 mg/dL (0.9 mg/dL on presentation) and she developed oliguric ARF. At this time, the patient revealed that she ingested a white powder obtained in Haiti that she was "saving for a suicide attempt." The patient required multiple episodes of hemodialysis (HD) prior to her SC decreasing to 1.7 mg/dL 2 weeks later. A kidney biopsy demonstrated acute tubular necrosis. On hospital day 20, she was discharged home and no further HD was necessary. Her initial blood mercury concentration was 5000 µg/L and her measured 24-hour urine Cd concentration was 7.4 mg/dL. <u>Conclusion</u>: This is a rare case of both Hg and Cd toxicity resulting from the ingestion of religious "remedies/poisons." Clinicians should be aware that people from Caribbean cultures have access to agents containing significant quantites of Hg and Cd. The development of unexplained ARF in such patients, especially in the circumstances of a suicide, attempt should prompt testing for heavy metals such as these.

241. SMOKING: A NOVEL ROUTE OF OLANZAPINE ABUSE

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<u>Background</u>: Although antipsychotics are generally not considered to be drugs of abuse, an Internet drug advocacy site has devoted an entire "vault" to the discussion of olanzapine and promotes euphoria as one of its positive effects. While there have been numerous reports of olanzapine toxicity, there are no reported cases of olanzapine abuse. Additionally, although the MSDS warns of "toxic fumes" from burning olanzapine, no clinical data refer to smoking as a route of exposure. We report a case of toxicity following inhalational olanzapine abuse. <u>Case Report</u>: A 24 year-old man with a history of depression and heroin and cannabis use was found obtunded at home. Initial vital signs were: blood pressure, 120/80 mmHg; pulse, 90 min⁻¹; respirations, 14 min⁻¹; afebrile. Physical examination was remarkable only for deep obtundation and an absent gag reflex. Finger stick glucose was normal, and an initial ECG showed sinus tachycardia at 110 min⁻¹ with normal intervals. He was intubated for airway protection, lavaged (no pills or fragments were retrieved), and given activated charcoal by nasogastric tube. The patient remained stable in the ICU and was extubated the following day. He later admitted to lacing a cannabis cigarette with approximately 20 tablets of crushed olanzapine and smoking it. A serum olanzapine level drawn approximately 36–48 hours after presentation was 150 ng/mL, twice the upper limit of the therapeutic range. <u>Conclusion</u>: Olanzapine may be abused and although unstudied, appears to be bioavailable through pyrolysis and inhalation. The pharmacokinetics and stability of olanzapine by this route are unknown. Further investigation of its abuse potential is warranted.

242. TACHYCARDIA AND RHABDOMYOLYSIS AFTER INTENTIONAL INGESTION OF N,N-DIPRO-PYLTRYPTAMINE

Dailey RM, Nelson LD, Scaglione JM. Drug and Poison Information Center, Cincinnati, Ohio, USA.

<u>Background</u>: *N*,*N*-Dipropyltryptamine (DPT) is a synthetic tryptamine with hallucinogenic properties that is available through the Internet as a research chemical. Although it may be ingested, abuse through insufflation, smoking freebase, or IM injection is more common. We report a case of DPT ingestion resulting in tachycardia and rhabdomyolysis. <u>Case Report</u>: An 18-yearold female arrived to a community hospital ED 90 min after ingestion of an unknown amount of DPT. The patient had visual hallucinations, was extremely agitated, screaming, and scratching her skin to the point of bleeding. Clinical findings included tachycardia (200 bpm), mydriasis, and diaphoresis. A commercial vial labeled DPT, with the cautionary statements of "human health hazard" and "for research purposes only" was brought in with the patient. Lorazepam (3 mg) was administered in the ED prior to admission to the ICU. Within 4 hours of presentation the patient was no longer agitated, but was confused, with a heart rate of 99 bpm. Laboratory values obtained after admission included serum creatine kinase (greater than 8000 U/L), CK-MB (25.1 ng/mL), troponin (1.1 ng/mL), BUN (7 mg/dL), creatinine (0.9 mg/dL), AST (566 U/L), and ALT (105 U/L). Rhabdomyolysis was treated with aggressive IV crystalloid. Sixty hours after admission, the patient was discharged with no complications. The patient admitted to purchase of 1 g of DPT from the Internet, emptying a vitamin C

capsule out, then filling it with an unmeasured amount of powder for experimental purposes. <u>Conclusion</u>: Abuse of DPT has not been previously reported in the literature. *N*,*N*-Dipropyltryptamine and other hallucinogenic tryptamines should not be overlooked when patients present to the ED with hallucinations, agitation, and tachycardia.

243. "MELLOW YELLOW"—INTENTIONAL ABUSE OF CLOZAPINE

Hadley C, Griffith J, Casavant M. Central Ohio Poison Center, Children's Hospital, Columbus, Ohio, USA.

Background: Intentional abuse of clozapine has not been reported in the literature. TESS data for 2000, 2001, and 2002 shows 1327 cases of atypical antipsychotics used for Intentional Abuse. Abuse of this agent may be an alarming trend. <u>Case Reports</u>: Two sixteen year old boys developed altered mental status at school and confessed to taking a pill called "mellow yellow." Naloxone was given by EMS with no response. Charcoal with sorbitol was given in the ED. Urine tox screen was negative in one case and positive for THC in the other. Mental status improved over a few hours. "Mellow yellow" was identified as Clozaril[®] 100 mg. Serum screens for clozapine were 196 and 92 ng/mL with norclozapine levels of 48 and 89 ng/mL, respectively. The Poison Center advised family doctors for the boys to check WBC within 2 weeks of discharge. Results were normal. <u>Conclusion</u>: Consider clozapine in patients with altered mental status, history of recreational drug use, and negative urine tox screens. The risk of agranulocytosis makes identification and long term follow-up advisable.

244. ROLE OF CONTINUOUS ARTERIOVENOUS HEMODIALYSIS (CAVHD) IN METHANOL POISONING

Schier JG,¹ Shapiro WB,² Howland MA,^{1,3} Hoffman RS,¹ Nelson LS.¹ ¹NYC Poison, Center and ²Brookdale Medical Center and ³St. John's University, New York City, New York, USA.

Introduction: The use of CAVHD in methanol poisoning is not well studied. We report a case of methanol ingestion in which serum methanol levels were measured during this process. <u>Case Report</u>: A 31-year-old female was found unresponsive at home and brought to the ED. She was intubated for airway protection and admitted to the ICU. Over the next 24 hours she developed a severe anion gap metabolic acidosis with a serum pH of 7.06. She was started on intravenous fomepizole and placed on CAVHD until hemodialysis (HD) could be started. She received approximately 8 hours of CAVHD followed by 4 hours of HD and was then placed back on CAVHD for another 8 hours. During the second episode of CAVHD, two separate and simultaneous blood samples were taken from both the arterial and venous dialysis lines on two occasions 4 hours apart. Mean arterial and venous methanol concentrations for the first set were 16.5 and 16 mg/dL respectively. Mean arterial and venous methanol concentrations for the second set were 11 and 10.25 mg/dL respectively. After extubation, the patient admitted to ingesting a de-icing agent containing 100% methanol. She suffered no visual impairment. Calculated methanol extraction ratios by CAVHD were 3.1% and 6.8% respectively. Methanol extraction ratios by HD vary depending on the level but may be >80%. <u>Conclusion</u>: In this patient, methanol extraction ratios and longer exposure times to formate are more likely to cause retinal toxicity, CAVHD would be a less optimal method of extracorporeal elimination when compared to HD.

245. THE EFFECT OF ALCOHOL CO-INGESTION ON PATIENT BEHAVIOUR IN ACUTE DRUG OVERDOSE

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<u>Background</u>: Alcohol is commonly co-ingested in acute drug overdose (OD). We examined the relationship between alcohol co-ingestion and patient behaviour during drug OD. <u>Methods</u>: A prospective observational study of adult patients presenting to the Emergency Department following drug OD. In addition to baseline patient data, alcohol co-ingestion, time of alcohol ingestion (<1 hour or >1 hour before OD), number of drugs ingested, and time of decision to take overdose (immediate, 1–4 hours, or >4 hours before OD) were recorded. <u>Results</u>: There were 291 patients with ODs, and

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47% of these had co-ingested alcohol. Males comprised 48% of the population who had taken an OD, but represented 56% of the population who had co-ingested alcohol. Patients who ingested alcohol ingested a significantly greater number of different drugs in overdose (p = 0.02). Amongst those with alcohol co-ingestion, males were more likely to ingest a greater number of different drugs than females (p = 0.03). The time of alcohol ingestion before OD, and the time of decision to take an overdose did not have a significant effect on the number of different drugs ingested. However those patients who had co-ingested alcohol took significantly fewer tablets in total (34.2 vs. 50.8, p < 0.01). There was no significant correlation between patient age and the likelihood of alcohol co-ingestion in drug OD. Conclusion: Alcohol co-ingested, but decreasing the total number of tablets ingested.

246. FATAL COMPLICATIONS OF ECSTASY AND AMPHETAMINE ABUSE

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Background: Amphetamine (A) abuse, including the hallucinogenic amphetamine MDMA "Ecstasy" and methamphetamine (M), can result in fatal outcome. We studied demographics, clinical features, and unique complications of A fatalities. Method: The 2000 and 2001 AAPCC TESS database was searched for human intentional fatal exposures to unspecified A (UA), E, and M. Individual fatality abstracts were reviewed for coingestants, age, gender, and presence of 11 complications: hyperthermia, rigidity, cardiac dysrhythmias, shock, coma, seizures, intracranial hemorrhage, rhabdomyolysis, DIC, acidosis, and hyponatremia. Complications were analyzed using binary logistic regression. Results: Of the 112 fatalities listed as A deaths, 68 were directly related to an amphetamine compound: 33 E, 19 M, 16 UA. Age for all A was 12–59 yrs (mean 36.2 ± 16.1), for E 12–40 yrs (mean 21.6 ± 5.7), for M 19–39 yrs (mean 30.9 ± 10.1). The predominant gender for all A fatalities was male: 44 males vs. 24 females. The four most frequent complications associated with a fatal outcome, found in >40% of all amphetamine deaths, were: coma, cardiac dysrhythmia, shock, and hyperthermia. In the UA group, coma was the best single predictor of mortality odds ratio (OR) = 0.38; p = 0.23]. In the E group, hyperthermia/fever was the best single predictor of mortality (OR = 4.44; p = 0.08). In the M group 73% of deaths were associated with acidosis. Conclusion: Amphetamine fatalities showed a predominance of the male gender with a mean age of 29.7 + 14.0 yrs. The best predictors of fatality were coma for unidentified amphetamines and hyperthemia for hallucinogenic amphetamines. Hyponatremia was unique to E documented in 12% of cases. Hyperthermia and rigidity were documented together in only 5.8% of all A fatalities.

247. MOONFLOWER ABUSE AND INTOXICATION IN NORTHERN OHIO

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<u>Background and objective</u>: In the fall of 2002, the Cincinnati Drug and Poison Information Center (DPIC) received a number of calls from a four county area in northern Ohio regarding intoxication after abuse of "moonflower seeds." Several parents and ED physicians sought help in identifying and understanding the toxicity this plant. <u>Case Series</u>: Between October 11th, 2002 and November 20th, 2002, DPIC was consulted on 13 patients between the ages of 12–19 years old after abuse of "Moonflower" seeds. The patients either ate the seeds or soaked them to make a tea. The patients presented with an anticholinergic toxidrome including mydriasis, dry mucus membranes, tachycardia, hallucinations, and urinary retention. The signs and symptoms typically lasted about 24–48 hours and all patients did well with supportive care and benzodiazepines. <u>Discussion</u>: The patient's symptoms were characteristic of Datura species, but neither Jimson weed nor Jimson seeds came up in histories from the patients or their friends. Poisindex indicates that Moonflower is a common name for Ipomoea (*I. Alba* and *I. Muricata*), adding to confusion. Conversations with the parents of some of the victims indicated that *Datura Inoxia* is a well-known ornamental plant in suburban gardens in Northern Ohio. Moonflower is a common name for *D. inoxia*, but not *Datura stramonium* (Jimson Weed). When the original seven cases were identified, the Cincinnati DPIC emailed a health alert throughout Ohio via the state's early warning system alerting physicians to the outbreak of abuse, likely cause and appropriate management. <u>Conclusion</u>: Common names for plants may be misleading. Poison Centers serve an important role in identifying abuse trends and alerting health care providers of these trends.

248. MONOAMINE OXIDASE INHIBITOR POISONING RESULTING FROM INTERNET INFORMATION ON ILLICIT SUBSTANCES

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Background: The Internet is a medium that may increase the dispersal of illicit drug information among vulnerable populations such as adolescents and young adults. Adverse outcomes related to internet based drug information are rarely identified. We report a case of an adolescent whose Internet use led to experimentation with illicit substances, an increase in drug use, and poisoning from a naturally occurring monoamine oxidase inhibitor (MAOI), harmaline. Case Report: A 17 year old male was brought to the ED by ambulance after he ingested Syrian Rue seeds purchased from the internet, and then smoked 20 mg of 5-methoxydimethyltryptamine (5-MeO-DMT). He presented with an axillary temperature of 105.2°F, HR 190 bpm, BP 136/66 mmHg, confusion, and extremely combative behavior. Benzodiaze-pines effectively controlled agitation, however, his mental status remained altered throughout his stay in the ED. His ICU course was notable for bradycardia, hypotension, rhabdomyolysis (peak CK 26,219 U/L), mild renal insufficiency (Cr. 1.7 mg/dL), and mild elevation of hepatic transaminases (ALT 167 U/L, AST 415 U/L). GC/MS analysis of urine revealed harmaline, a naturally occurring MAOI found in Syrian Rue. Conclusion: A disturbing trend of adverse events stemming from internet based drug information has emerged. Many of the drugs discussed in these forums are unfamiliar to physicians. Therefore, clinicians must be aware of alternate drug information sources used by patients in order to accurately predict and diagnose toxicity.

249. SERUM HOMOCYSTEINE LEVELS DO NOT CORRELATE WITH SEVERITY OF ALCOHOL WITHDRAWAL

Chan GM, Su M, Donnelly JG, Hoffman RS, Nelson LS. NYC Poison Center, New York, USA.

<u>Background</u>: Homocysteine is implicated in the genesis of the alcohol withdrawal syndrome (AWS) because it may be metabolized to an excitatory neurotransmitter. Folate and vitamin B12 are important cofactors in the safe elimination of homocysteine and may limit accumulation of the excitatory metabolite. This study was designed to investigate a previous hypothesis suggesting that the serum homocysteine levels can be used to predict the severity of AWS. <u>Methods</u>: During a 1 year period in a large urban ED, we enrolled a prospective convenience sample of patients determined to have AWS following written consent. Each enrolled patient had determinations of concomitant serum levels of homocysteine, folate, vitamin B12, and blood alcohol. Assessment of severity of AWS was determined by The Clinical Institute of Withdrawal Alcohol Scale, Revised (CIWA-Ar) at presentation and by surrogate markers, including the need for benzodiazepines and other agents. <u>Results</u>: Preliminary results of 31 patients showed no correlation ($R^2 = 0.01$) between homocysteine levels and CIWA-Ar scores. There was also no correlation between homocysteine levels and the need for benzodiazepines in the 23 patients in whom sufficient data is available. However, a significant negative correlation was noted between folate levels and CIWA-Ar ($R^2 = 0.17$; p = 0.01). <u>Conclusions</u>: Serum homocysteine levels do not appear to correlate with severity of alcohol withdrawal based on initial CIWA-Ar scores or medication requirements. The suggestion that lower folate levels are noted in patients with more severe AWS at ED presentation merits further investigation into the potential beneficial role for nutritional supplemention.

250. SURVIVAL AFTER RECREATIONAL IV ADMINISTRATION OF BENZONATATE

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<u>Background</u>: Benzonatate, a non-narcotic antitussive, is also a local anesthetic structurally related to tetracaine. It is a mixture of related polyglycol analogs. Its action is due to an effect on lung stretch receptors and a central mechanism. Literature search reveals only one other case (death) from intentional IV administration. We report a case of recreational IV administration who suffered cardiac arrest and survived. <u>Case Report</u>: A 27 y. o. woman and her male cousin were



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given "some" 100 mg Tessalon Perles[®] to "get high." The Perles were heated in a spoon and injected intravenously by the patient. Her cousin did not achieve IV access. He contacted 911 within 10 min of the injection after the patient collapsed and seized. She was found unresponsive (full cardiopulmonary arrest) by first responders. Upon arrival, EMS noted a faint auscultated heart sound and initiated CPR, epinephrine and atropine. She was noted to be in VFib and was defibrillated. In the ED she was intubated, administered lorazepam, vecuronium and placed on a ventilator. She exhibited some decorticate posturing and was admitted to the Medical Neurology Unit. The patient did not require inotropic support to maintain her systolic BP in the 90s and NSR at 100 bpm. On day 2 she was extubated and remained stable. Within 2 days she was self-ambulating, following commands but occasionally agitated with hallucinations. On day 4 she was transferred to a psychiatric facility. <u>Conclusion</u>: IV administration of benzonatate may result in seizures and cardiac arrest with Vfib, but survival is possible with supportive care.

251. PROLONGED HALLUCINATIONS FOLLOWING INGESTION OF ALPHA-METHYL-TRYPTAMINE

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<u>Background</u>: Alpha-methyl tryptamine (AMT) is a hallucinogenic agent that has recently been made a schedule I drug. Human AMT intoxication has never been reported in the medical literature even though it is prominently advocated on internet sites such as the Vaults of Erowid. We describe the first reported case of AMT intoxication. <u>Case Report</u>: A 21-year-old male college student presented to the ED after ingesting 270 mg of AMT he had purchased on the internet. He used the same batch of AMT numerous times previously for its "psychedelic properties," but this time miscalculated and took ten times what he had previously used. He presented 1 hour after ingestion with vital signs of: BP 183/93, P 52, RR 20, and T 36.4. He was awake, hyper-vigilant, orientated to only person, with mydriasis (10 mm diameter), mild tremor, delayed response time, restlessness, exaggerated startle reaction, and visual hallucinations. Ten hours later, his symptoms began to resolve. Diagnostic tests (CBC, Chemistry, PT, PTT, EKG) were unremarkable except for potassium of 3.2. He was discharged home without complication. <u>Conclusion</u>: Alpha-methyl tryptamine is a potent hallucinogen with effects that can last greater than 10 hours following ingestion of large doses.

252. COMPARATIVE RATES OF OXYCONTIN ABUSE: ANECDOTAL HIGHS

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<u>Background</u>: Several prescription opioids are commonly abused in the United States. Anecdotal information suggests that OxyContin, a continuous release formulation of oxycodone, has a higher rate of abuse than other opioids. We used intentional exposure calls reported to poison centers as abuse indicators to support the hypothesis that call rates for OxyContin abuse are higher than other opioids. <u>Methods</u>: We reviewed 3 months of exposure calls, excluding information calls, from eight regional poison centers, serving a broad geographic area of over 65 million people, involving five commonly abused opioids (oxycodone (excluding OxyContin), OxyContin alone, methadone, morphine, and hydrocodone. <u>Results</u>: The total intentional exposure call rates for these opioids per 100,000 population served for 3 months for all eight poison centers are:

Opioid	Hydrocodone	Methadone	Morphine	Oxycodone	OxyContin
Rate	0.89	0.14	0.10	0.24	0.12

Intentional exposure call rates for hydrocodone are 371% greater than oxycodone and 636% more than methadone. OxyContin had the fourth highest rate of intentional exposure calls, only greater than morphine. The 2001 Annual Report of the AAPCC Toxic Exposure Surveillance System indicates that the rate for oxycodone, with OxyContin included, is 0.44 per 100,000 people for 3 months nationwide while the 8 centers combined rate was only 0.36.

<u>Conclusion</u>: OxyContin does not have a higher intentional exposure call rate than hydrocodone, oxycodone, and methadone as reported to 8 regional poison centers in the United States.

253. CYCLOSPORIN PHARMACOKINETICS WITH MULTIDOSE CHARCOAL AFTER A TEN-FOLD DOSING ERROR

Qureshi ST, Smolinske S. Children's Hospital of Michigan, Regional Poison Control Center, Detroit, Michigan, USA.

<u>Background</u>: Although cyclosporine (CSA) is used widely as an immunosuppressant in transplant recipients, experience with an overdose is limited. We report the kinetics data on a neonate with 2 ten-fold dosing errors, treated with multi-dose activated charcoal (MDAC). <u>Case Report</u>: A 21 day old infant with a heart transplant for hypoplastic left heart received two consecutive oral doses of CSA of 21 mg/kg each (10-fold dosing error), 8 hours apart. The patient developed fussiness shortly after the first overdose, but did not manifest any other signs of toxicity. Cyclosporine level was 2037 ng/mL just before the second dose, and 2430 ng/mL 8 hours later (desired level 50–300 ng/mL). Multi-dose activated charcoal was started 8 hours after the last dose and was given every 4 hours for three doses only (until 16 h after the last dose), secondary to lack of adequate data supporting its use. Cyclosporine level was 930 ng/mL 15.5 h after starting MDAC, corresponding to a half-life of 11 h. Subsequently the CSA level declined to the 295 ng/mL 86.5 h after starting MDAC, with the longest calculated half-life of 74 h. <u>Conclusion</u>: Current recommendations for CSA overdose include general gastric lavage and/or activated charcoal, as appropriate, and supportive care. One case reports a half-life of 2.7 h during MDAC and 9 h after MDAC cessation. In two other overdose cases the elimination half-lives were 6.87 and 70 hours. Our data suggests that MDAC enhances the elimination of CSA. More frequent CSA monitoring during MDAC administration would have improved the kinetics data.

254. URINARY SODIUM AND POTASSIUM EXCRETION AS MEASURES OF IBUPROFEN NEPHRO-TOXICITY

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<u>Background</u>: Overdose of ibuprofen can cause acute renal vasoconstriction and acute tubular necrosis (ATN). We have studied urinary sodium (Na) and potassium (K) excretion after ibuprofen overdose. Renal vasoconstriction Na retention occurs and fractional excretion of Na (FeNa) should decrease. In ATN FeNa should exceed 2–3% producing a biphasic dose response. Renal vasoconstriction increases K excretion. Fractional excretion of K (FeK) should increase as dose of ibuprofen ingested increases. Methods: We studied 11 patients admitted with an ibuprofen overdose. Creatinine, Na, and K were measured on paired plasma and urine samples, taken on or as near as possible to admission, and FeNa and FeK calculated. Results were correlated with ibuprofen dose. Results: FeK correlated with ibuprofen dose $(r^2 = 0.37, p < 0.05)$, consistent with increasing aldosterone action on the distal tubule as renal perfusion falls. There was no correlation between ibuprofen dose and FeNa $(r^2 = 0.22)$. When overdoses of >20 g were excluded, FeNa did fall as dose ingested increased $(r^2 = 0.49, p < 0.05)$, consistent with increasing renal vasoconstriction with increasing dose ingested. <u>Conclusion</u>: This study suggests that FeK, measured early after overdose, may provide a useful guide to patients requiring follow up of renal function. FeNa may also point to those patients with significant vasoconstriction, however, care would be needed in interpreting higher values as these may equally represent Na wastage from damaged tubules in severe overdose, or normal renal perfusion.

255. DOES FLUMAZENIL PROLONG GHB POISONING?

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Objective: Gamma hydroxybutyrate is a sedating drug of abuse that has affects on GABA receptors. Animal studies of the interaction between flumazenil, a GABA A receptor antagonist and GHB are inconclusive. Our prior study reported

a delay of GHB intoxication when pre-treated with flumazenil. We proposed that flumazenil might affect the length of the sedative affect of GHB. <u>Methods</u>: We performed an in-vivo study of the interaction between flumazenil and GHB in a murine model. Three groups of mice received 1.5 g/kg of GHB intraperitoneally. Group 1 (n = 8) received sham IP injections at time 0, then GHB at 5 min. Group 2 (n = 12) received 0.3 mg/kg of flumazenil at time 0 and then GHB in 5 min. Group 3 (n = 12) received sham injection at time 0, GHB at 5 min, then four escalating IP doses of flumazenil every 3 min (0.003–1 mg/kg). Righting reflexes, postural time, and recovery times were recorded. <u>Results</u>: One animal in the group 1 died and all others recovered within 4 hours. No animals in group 2 or 3 died, but 4 did not regain righting reflex and 8 did not regain postural tone within 4 hours. P = 0.05. <u>Conclusion</u>: In this murine model, the combination of flumazenil and GHB was associated with prolonged time to regain postural tone and righting reflex.

256. CYTOFLAVINE IN EXPERIMENTAL TRAUMATIC BRAIN INJURY COMPLICATED WITH ACUTE ALCOHOLIC INTOXICATION

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Background: Every second case of traumatic brain injury (TBI) is accompanied by acute alcoholic intoxication (AAI) which may contribute to hypoxia, reactive oxygen species production, and lipid peroxidation. Cytoflavine (C) is a novel medication, each vial of it contains a combination of succinate (500 mg), riboflavine (10 mg), nicotinamide (50 mg), and inosine (100 mg). The aim of this study has been to assess C activity in acute TBI complicated with AAI. Methods: 89 albino male rats were subjected to TBI (0.4 J) induced by a weight-drop device 1 h after intragastric administration of ethanol (6–8 g/kg). Cytoflavine (150 mg of succinate/kg i.p., b.i.d. for 8 days) was compared with mexidol (emoxipine succinate 50 mg/kg i.p. once a day), placebo (P) (normal saline), and intact control. Neurologic severity score (NSS) and passive avoidance (PA) conditioning were assessed. On the 9th day animals were decapitated. Their blood was collected for malonyl dialdehyde (MDA), reduced glutathione (RG), ascorbate (A), superoxide dismutase (SOD), and catalase (CAT) assessment. Brain pathology was evaluated. The data are presented as mean values and 95% Confidence Intervals. Results: By the 7th day NSS decreased to 4.2 (3.4–5.2) in C group vs. 6.1 (4.9–7.3) and 5.4 (4.7–6.1) in P and M groups respectively [intact values were 0.6 (0-1.2)]. Cytoflavine group showed 100% PA performance vs. 50% in P and 62% in M groups. Malonyl dialdehyde (nmol/mL) was 7.1 (5.8–8.5) in C vs. 10.4 (8.5–12.3) in P group (in intact rats 4.5(3.4– 5.6), M group differed non-significantly). Cytoflavine prevented the 50% depletion of RG, A, SOD, and CAT found in P group. Conclusion: Cytoflavine reduced neurologic and behavioral disorders due to TBI and AAI in rats and attenuated lipid peroxidation.

257. FUNCTIONAL MRI ASSESSMENT OF ORGANOPHOSPHATE POISONING

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<u>Background and Objectives</u>: Recent studies have emphasized the CNS component of hyperacute organophosphate (OP) toxicity. Advances in MRI technology now allow imaging of awake, restrained, animals. However, the profound fasciculations after OP toxicity have limited the use of functional MRI (fMRI) in OP studies due to motion artifact. We sought to develop a method of assessing the CNS effects of OP poisoning using functional MRI. <u>Methods</u>: Four Wistar rats were anesthetized with 2% isoflurane and an intraventricular catheter was placed. Rats were then immobilized in a customized rodent restrainer with surface MRI coil in a 4.7 T horizontal MRI. Five minutes of continuous blood oxygen level dependent (BOLD) imaging was performed, followed by intraventricular injection of 50 µg dichlorvos. This was followed by another 5 min of continuous BOLD imaging. Blood oxygen level dependent signal changes were analyzed using STIMULATE PC software. <u>Results</u>: All animals died within 5 min of poisoning. Functional MRI images after poison administration contained little appreciable motion artifact, with image deviation of roughly 1% from baseline. Significant BOLD signal changes were present in the cortex and medullary respiratory centers. Neither hypoxia nor hypotension occurred until cessation of respiration. <u>Conclusion</u>: Using customized restrainer and surface MRI coils, functional MRI can be used to determine the CNS effects of acute, lethal, organophosphate poisoning in the rat. Changes in BOLD signal did not appear to be due to hypoxia or hypotension. Further studies examining these CNS effects are warranted.
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258. THE EFFECT OF AMIODARONE ON SURVIVAL IN A MURINE MODEL OF FLUORIDE TOXICITY

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Objective: Systemic fluoride (F) toxicity results in hypocalcemia, hypomagnesemia, and hyperkalemia, which lead to cardiac dysrhythmias. A previous in vitro study showed attenuation of F-induced hyperkalemia with amiodarone (A). We hypothesize that A pre-treatment improves survival time in a murine model of escalating F toxicity. Methods: We performed a randomized, blinded, placebo-controlled trial using 77 mice. The mice were randomized to pre-treatment with normal saline (C) or 50 mg/kg A. After 30 min, the mice were randomized into four groups and received escalating doses of sodium fluoride (NaF) intraperitoneally: (1) 50 mg/kg, $C_1 n = 15$, $A_1 n = 16$; (2) 66 mg/kg, $C_2 n = 10$, n = 10A₂; (3) 95 mg/kg, C₃ n = 8, A₃ n = 8; (4) 151 mg/kg, C₄ n = 5, A₄ n = 5. After NaF administration, the mice were continuously observed for 3 hours and again at 24 hours for survival. Kaplan-Meier analysis was used to compare the primary endpoint of 3-hour survival of A to C within each group. A Cox Proportional Hazard analysis was performed to determine the effect of A on the risk of death across all doses of NaF compared to C. Results: Median survival times in minutes (95% CI) for each group are: (1) 50 mg/kg, $C_1 = 41.5$ (21.1–61.8), $A_1 = 52.2$ (0–297.1); (2) 66 mg/kg, $C_2 = 29.1$ (26.5–31.6), $A_2 = 28.9$ (23.4–40.2); (3) 95 mg/kg, $C_3 = 14.9$ (6.3–23.4), $A_3 = 15.5$ (14.7–16.2); (4) 151 mg/kg, C₄ = 10.1 (8.9–11.2), A₄ = 14.4 (7.2–21.6). Two C mice and 6 A mice in group 1 survived longer than 3 hours. A Cox Proportional Hazard analysis showed a risk ratio of 1.38 (0.851–2.25) indicating that C mice have a 1.38 times risk of death versus A treated mice in severe F toxicity. Conclusion: Pre-treatment with amiodarone shows a trend toward improved survival in severe fluoride toxicity.

259. HIGH-DOSE MAGNESIUM INDUCES APOPTOTIC NEURODEGENERATION IN THE DEVELOP-ING MOUSE BRAIN

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<u>Background</u>: Magnesium (Mg) is the most abundant divalent cation in the human body, and is essential for many vital physiological functions. NMDA receptors play an important role in development, learning and memory. Transient blockade of these receptors during synaptogenesis (first 3 weeks of life in rodents) causes widespread apoptotic neurodegeneration in the developing brain. Magnesium, by binding to a specific site in the NMDA receptor channel, can inhibit neuronal conductance. Many human fetuses are exposed to high doses of Mg late in pregnancy (corresponding to the vulnerable synaptogenic period) for preterm labor and preeclampsia/eclampsia. Therefore, we investigated whether acute exposure to Mg would produce apoptotic neurodegeneration in the developing mouse brain. Methods: Seven-day-old C57BL/6 mice were given MgSO₄ 250 mg/kg IP hourly for four or five doses, and after survival intervals from 10 to 24 hrs, the brains were examined for degenerative changes, using the DeOlmos cupric silver technique, or immunohis-tochemical staining for activated (cleaved) caspase-3, a sensitive marker for apoptosis. <u>Results</u>: In specific layers of the neocortex, subiculum, caudate nucleus, thalamus, hippocampus, and cerebellum/brain stem, the number of neurons displaying positive silver staining or caspase-3 activation was significantly increased, compared to saline controls. <u>Conclusions</u>: Exposure of the developing brain to Mg at a time of heightened sensitivity produces widespread apoptotic neurodegeneration with NMDA receptor interference as a candidate mechanism. This suggests the possibility that similar toxicity might occur in human fetuses exposed in utero to Mg.

260. GHB, GBL, AND 1,4-BD REDUCE INFARCT VOLUME FROM REPERFUSION INJURY FOLLOW-ING TRANSIENT MIDDLE CEREBRAL ARTERY OCCLUSION (MCAO)

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Background: GHB has been shown to have neuroprotective properties in cerebral ischemia, but its effect on reperfusion injury has not been studied. Objective: To evaluate the neuroprotective effect of GHB, GBL, and 1,4-BD in the rodent



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model of reperfusion injury by transient MCAO. Methods: 32 male SD rats were anesthetized with isoflurane. A small incision was made in the left external carotid artery and 3/0 monofilament passed 2 cm proximally into the left internal carotid artery until it lodged in the anterior cerebral artery, thereby occluding the origin of the MCA. The filament was sutured into place and the incision closed. After 90 min, the filament was removed and blood flow restored to the brain. Eight rats were sham controls and 24 rats were divided into groups treated with GHB, GBL, or 1,4-BD at 30 min before as well as 180 and 360 min after infarction (dose 300 mg/kg i.p.; N = 8 each group). Twenty four hours later, brains were removed, cut into 1mm coronal slices and stained with 2% TTC, a water soluble, colorless solution reduced to an insoluble red pigment by viable neuronal cells. The coronal sections were photographed with a 4.0 megapixel digital camera and analyzed with SigmaScan Pro[®] 5.0 image analysis software. Data for ischemic injury in each group were represented as the mean volume of infarction (cu.mm) \pm SEM and compared to sham rats by ANOVA with post-hoc Neuman-Keuls Test. Results: The mean volume of infarction for sham rats was 464.4 \pm 17.9 cu.mm vs. 273.6 \pm 53.1 (P < 0.05), 233.3 \pm 44.7 (P < 0.05), and 275.4 \pm 39.9 cu.mm for rats treated with 1,4-BD, GBL, and GHB, respectively. Conclusion: GHB, GBL, and 1,4-BD appear to offer neuroprotection from reperfusion injury following transient MCAO in the rat by significantly reducing infarct volumes.

261. NEUROPROTECTIVE EFFECT OF GHB, GBL, AND 1,4-BD ON RAT FOCAL CEREBRAL ISCHE-MIA BY PERMANENT MIDDLE CEREBRAL ARTERY OCCLUSION (MCAO)

Quang LS,¹ Sadasivan S,² Maher TJ,² Shannon MW.¹ ¹Children's Hospital Boston/Harvard Medical School, Massachusetts, USA; ²Massachusetts College of Pharmacy & Health Sciences, Boston, Massachusetts, USA.

Background: A recent study demonstrated a neuroprotective effect of GHB in rat global cerebral ischemia. Objective: To evaluate the neuroprotective effect of GHB, GBL, and 1,4-BD in the rodent model of *focal* cerebral ischemia by permanent MCAO. Methods: 48 male SD rats were anesthetized with isoflurane. A small incision was made in the left internal carotid artery and 4/0 monofilament passed 2 cm proximally, lodging it in the anterior cerebral artery and occluding the origin of the MCA. The filament was sutured into place and the incision closed. six rats were sham controls and 36 rats were divided into groups treated with GHB, GBL, or 1,4-BD at 30 min before as well as 180 and 360 min after infarction (dose 300 mg/kg i.p.; N = 12 each group). Twenty four hours later, brains were removed, cut into 1 mm coronal slices, and stained with 2% TTC, a water soluble, colorless solution reduced to an insoluble red pigment by viable neuronal cells. The coronal sections were photographed with a 4.0 megapixel digital camera and analyzed with SigmaScan Pro[®] 5.0 image analysis software. The infarct areas for each group were represented as the mean volume $(cu.mm) \pm SEM$ as well as the mean % volume $\pm SEM$ of ischemic injury and compared to sham rats by ANOVA with post-hoc Neuman-Keuls Test. Results: The mean volume of infarction for sham rats was 323 ± 29.5 cu.mm vs. 149.7 ± 45.2 (P < 0.05), 103.5 ± 35.1 (P < 0.05), and 229.4 ± 40.5 cu.mm for rats treated with 1,4-BD, GBL, and GHB, respectively. The mean percent volume of infarction for sham rats was $30.1 \pm 2.0\%$ vs. 13.1 ± 4.0 (P < 0.05), 8.2 ± 3.0 (P < 0.05), and $19.1 \pm 3.4\%$ (P < 0.05) for rats treated with 1,4-BD, GBL, and GHB, respectively. Conclusion: GHB, GBL, and 1,4-BD offer neuroprotection in rodent *focal* cerebral ischemia by permanent MCAO.

262. LABORATORY DETECTION OF ACUTE 1,4-BD AND GHB OVERDOSE BY ROUTINE URINE ORGANIC ACID ANALYSIS

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<u>Background</u>: Laboratory detection of GHB and 1,4-BD remains a significant challenge for suspected overdoses at most hospitals. However, the urine organic acid analysis (u.o.a.a.) may provide an alternative laboratory method for confirming a clinically suspected acute overdose of GHB or 1,4-BD. As an endogenous catabolite of GABA, 4-hydroxybutyric acid (GHB) is among about 250 endogenous organic acids the u.o.a.a. can detect. <u>Objective</u>: We conducted a controlled, longitudinal murine study to evaluate the diagnostic accuracy of routine u.o.a.a. for detecting GHB after acute 1,4-BD overdose. Methods: 25 CD-1 mice were divided into five groups (N=5 each group) and placed into metabolic cages for

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urine collection in 2 phases. In the control phase, baseline urines were collected from each group for 4 hours. In the overdose phase, all mice were given 1,4-BD 600 mg/kg i.p. and urine was collected for 4 hours. All samples underwent solid phase extraction and derivatization with bis(trimethyl-silyl) trifluoroacetamide followed by double organic extraction with ethyl acetate and ether. They were then analyzed on GC 5890/MS 5792 (Hewlett-Packard Co., Wilmington, DE), using MSD Productivity ChemStation Software. <u>Results</u>: There was a library match for GHB in 5/5 mouse overdose urine samples, with a retention time of 9.4 min, molecular weight (MW) 248, methylene unit (MU) 1225, and ion pattern inclusive of 233 m/z. Conversely, 0/5 mouse control urines had a library match for GHB. <u>Conclusion</u>: In this murine pilot study, the urine organic acid analysis is a sensitive and specific diagnostic tool for confirming acute overdoses with GHB and 1,4-BD.

263. ABSTRACT WITHDRAWN

264. EMERGENCY DEPARTMENT CLINICAL COURSE OF OPIATE OVERDOSES

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<u>Background</u>: A scarcity of outcome data has contributed to many controversies in the management of opiate OD. *Purpose*: Determine the disposition, complications and naloxone dose requirements for an ED opiate OD cohort. <u>Methods</u>: An inner city EDs charts over 2 yr were retrospectively reviewed. <u>Results</u>: 366 cases met inclusion criteria; mean age 38 yr (range 19–66 yr), 78% male. Mean length of ED stay: 3 hr 10 min (range 8 min–23 hr 50 min). Naloxone was given in 92% initially by EMS (290) or ED (48) staff. One hunderd and seven (32%) required additional ED naloxone for no gag 18, hypoventilation 15, persistent somnolence 26 or decreased LOC 14, SpO2 23, RR/volume 16. Mean total naloxone dose was 0.9 mg (range 0–3.4 mg) not including 13 IV infusion cases. The mean interval between ED arrival and the 1st ED naloxone was 1 hr 7 min (range 0 min–7 hr 20 min). Serious complications: Aspiration pneumonia 11, pulmonary edema 8, hypoxic encephalopathy 3, rhabdomyolysis 5, and seizure 2 cases. Coingestants were suspected in 73% of cases receiving additional ED naloxone and 83% of 70 cases requiring hospitalization. <u>Conclusions</u>: In this cohort, opiate OD frequently were confounded by coingestants and required additional ED naloxone and had surprising high rates of serious complications and hospital admission.

265. THE EFFECTS OF ADENOSINE RECEPTOR ANTAGONISTS ON AMITRIPTYLINE-INDUCED CARDIOVASCULAR TOXICITY IN RATS

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<u>Objective</u>: To determine the effects of adenosine receptor antagonists (A₁, A_{2a}, A₃) on amitriptyline-induced cardiovascular toxicity in an anesthetized rat model. <u>Methods</u>: In this randomized, controlled animal study, toxicity was induced by the infusion of amitriptyline 0.94 mg/kg/min until 40–45% reduction of mean arterial pressure (MAP). Intravenous cromolyn was injected before amitriptyline administration to inhibit the A₃ receptor-mediated activation of mast cells. After amitriptyline, while control animals (n = 8) were given dextrose solution, treatment groups received an A₁-antagonist, 8-cyclopentyl-1,3-dipropylxanthine (DPCPX, 20 µg/kg/min, n = 8) or an A_{2a}-antagonist, 8-(3-chlorostyl)caffeine (CSC, 24 µg/kg/min, n = 8) for 60 minutes. Outcome measures were MAP, heart rate (HR) and QRS duration. <u>Results</u>: Amitriptyline infusion significantly reduced MAP by 42.9 ± 0.9% and prolonged QRS by



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 $30.4 \pm 9.3\%$ within 15 minutes. HR was not changed significantly during the experiments. While dextrose did not improve MAP and QRS prolongation, DPCPX and CSC increased MAP significantly ($103.3 \pm 3.7\%$ and $95.5 \pm 4.3\%$ at 60 min, p < 0.0001, respectively). DPCPX and CSC administration resulted in significant improvement in MAP compared to the dextrose group at 10 min ($88.5 \pm 2.8\%$, $75.6 \pm 4.7\%$ and $50.1 \pm 14.7\%$, p < 0.05, respectively). Both DPCPX and CSC decreased QRS prolongation (p < 0.01) and increased median survival time significantly (log-rank test, p < 0.00001). Conclusion: As adenosine antagonists were found to be effective in improving hypotension, QRS prolongation and survival time in our amitriptyline toxicity model, these agents may be promising agents for reversing amitriptyline-induced cardiovascular toxicity.