Clinical Toxicology, 39(5), 473–568 (2001)

Platform Session 1 Saturday, October 6 10:30 am–11:30 am
Abstracts #1–#4

1 POTENTIAL HEPATOTOXICITY OF REPEATED ACETAMINOPHEN ADMINISTRATION IN CHILDREN.

Kozer E, Barr J, Evans S, Greenberg R, Soriano I, Bulkowstein M, Chen-Levi Z, Barzilay B, Berkovitch M. *Clinical Pharmacology Unit, Division of Pediatrics, Assaf Harofeh Medical Center, Sackler School of Medicine, Tel-Aviv University, Israel*

Background: Acetaminophen is the most frequent pharmaceutical product administered to children worldwide. Repeated doses of acetaminophen given for therapeutic reasons have been reported to cause hepatotoxicity in adults and children. We aimed to investigate glutathione and anti-oxidant status changes in the erythrocytes of febrile children receiving repeated supratherapeutic acetaminophen doses. Methods: 51 children aged 2 months to 10 years participated in the study. Three groups of children were studied: Group 1 (n = 24) included afebrile children who did not receive acetaminophen. Groups 2 (n = 13) and 3 (n = 14) included children who had fever above 38.5°c for more than 72 hours. Patients in group 2 received acetaminophen 50 ± 15 (30–75) mg/kg/day and those in group 3 received acetaminophen above the recommended therapeutic dose – 107 ± 28 (80–180) mg/kg/day. A blood sample was taken for reduced glutathione (GSH), glutathione reductase (GR), glutathione peroxidase (GPX), and anti oxidant status. Results: GSH, GPX and anti oxidant status were lower in group 3 as compared with groups 1 and 2 ($p < 0.001$, $p = 0.045$ and $p < 0.002$, respectively). GR activity was lower in groups 3 and 2 in comparison with group 1 ($p < 0.001$ and $p = 0.013$). Conclusion: Our data show that in febrile children, treatment with repeated supratherapeutic doses of acetaminophen is associated with reduced anti-oxidant status and erythrocyte glutathione levels. These significant changes may indicate an increased risk for hepatotoxicity and liver damage.

2 A META-ANALYSIS OF PROGNOSTIC CRITERIA DETERMINING NEED FOR LIVER TRANSPLANTATION IN PATIENTS WITH FULMINANT HEPATIC FAILURE SECONDARY TO ACETAMINOPHEN POISONING.

Bailey B, Amre DK, Gaudreault P. *Hôpital Ste-Justine, Université de Montréal, Montréal, Quebec, Canada*

Objectives: To summarize and compare different prognostic criteria used to determine need for liver transplantation in patients with fulminant hepatic failure secondary to acetaminophen poisoning. Methods: We performed a MEDLINE search (1966–2000) to retrieve relevant articles using a pre-established search strategy. Of the 15 relevant articles, 5 were excluded (2 × 2 tables could not be reconstructed or they assumed that subjects undergoing transplant would otherwise have died). Data were summarized using linear regression methods after accounting for possible threshold differences between studies and SROC (Summary Receiver Operating Characteristic) curves were generated. Results: King's criteria (pH <7.30 or PT >100 sec + creatinine >300 μmol/L + encephalopathy grade ≥3) were evaluated in 9 studies, pH <7.30 in 4, PT >100 sec in 3, PT >100 sec + creatinine >300 μmol/L + encephalopathy grade ≥3 in 3, creatinine >300 μmol/L in 2, and one each for increase in PT day 4, factor V <10%, APACHE II score >15 and Gc-globulin <100 mg/L. The King's criteria were more sensitive than pH <7.30: 69% [95% CI 63–75] vs 57% [95% CI 44–68]. Their specificities were however comparable: 92 % [95% CI 81-97] vs 89% [95% CI 62-97]. Accuracy for these 2 criteria using SROC methods was identical: Q values of 0.61. A APACHE II score >15 had the highest

positive likelihood ratio (16.4) and the lowest negative likelihood ratio (0.19) but was evaluated in only 1 study. The accuracy measures of all other criteria were lower than that of King's criteria or pH <7.30. Conclusions: Presently available criteria are not very sensitive and may miss patients requiring transplantation. Future studies should further evaluate the efficacy of the APACHE II criteria.

3 EFFECT OF ANTICHOLINERGIC DRUGS ON THE EFFICACY OF ACTIVATED CHARCOAL.

Tenenbein M, Green R, Sitar DS. *University of Manitoba, Winnipeg, Manitoba, Canada*

Background: Although it is a commonly held tenet that drugs with anticholinergic action would prolong the duration of time for effective gastric decontamination after drug ingestion, there are no published data in support of this belief. The purpose of this study was to determine whether activated charcoal is more effective in the presence of anticholinergic activity. Methods: This was an IRB approved, human volunteer, randomized crossover study. Ten volunteers ingested 4.0 g of acetaminophen on three occasions at least one week apart. One ingestion served as a control. One hour after the other two ingestions, 50 g of activated charcoal in water was administered to each volunteer. These intervention limbs differed in that each volunteer received 0.01 mg/kg of atropine intramuscularly 15 minutes prior to acetaminophen dosing in one of them. Eight blood samples were taken over the initial 8 hours for HPLC analyses of serum for acetaminophen, which were used for calculation of pharmacokinetic parameters. Repeated measures ANOVA and Tukey's HSD test were used for statistical analyses. Results: Control pharmacokinetic parameters for acetaminophen were consistent with literature values. The mean AUC $\pm$ SD for the control, charcoal and atropine/charcoal groups were 258 $\pm$ 122, 206 $\pm$ 120, and 138 $\pm$ 88 mg/L.hr, respectively. The decreases of bioavailability were 20% for the charcoal and 47% for the atropine/charcoal groups. Charcoal was more effective when the subjects were pre-treated with atropine compared to charcoal without atropinization ($p < 0.05$). Conclusion: Our data support the belief that activated charcoal is more effective in the presence of anticholinergic activity.

4 A RETROSPECTIVE REVIEW OF PHYSOSTIGMINE IN OLANZAPINE OVERDOSE.

Ferraro KK, Burkhart KK, Donovan JW, O'Donnell SJ. *Pennsylvania State College of Medicine, Hershey, PA*

Background: Olanzapine is a commonly prescribed atypical antipsychotic. The anti-cholinergic properties of this medication contribute to the severe agitation and altered level of consciousness that can be seen in acute olanzapine overdose. Physostigmine is a short acting cholinesterase inhibitor known for its efficacy in treating anticholinergic symptoms. The use of physostigmine in acute olanzapine toxicity has not been previously described. Methods: A retrospective review of all patients with acute olanzapine intoxication admitted to our regional poison treatment center between January 1999 and April 2001 was performed. Twenty-four patients between the ages of 7 and 76 years with moderate to severe outcomes involving olanzapine ingestion were reviewed. Of these patients, 9 were given physostigmine in doses ranging from 0.5 mg to a total of 14 mg. Results: Six of the nine patients became arousable with a rapid improvement of mental status and in 4 patients intubation was prevented. Two patients were self-extubated following physostigmine, with one requiring re-intubation. Two patients had inadequate documentation of response to physostigmine, and 1 patient's primary toxicity was from other coingestants. Conclusions: Large ingestions of olanzapine can result in severe toxicity with delirium and depressed mental status, often requiring invasive procedures such as intubation. Physostigmine successfully improved mental status in 6 of the 9 cases and prevented intubation in 4 of the 9 cases we report. The use of physostigmine was associated with agitation, however no significant adverse outcomes were seen. Physostigmine may be a safe and effective adjunct in the care of patients with acute olanzapine toxicity.

Poster Session I — Saturday, October 6 — 10:00 am–4:00 pm
Abstracts #5–#54 — Authors with Posters — 2:30 pm–4:00 pm

5 ELIMINATION OF BRODIFACOUM.

Lewis-Younger C, Horowitz Z. *Oregon Poison Center; OHSU: Portland, OR*

Background: Intentional ingestion of brodifacoum (BDF), the active ingredient in some rodenticides, can lead to many months of anticoagulant effect, which require vitamin K supplementation. The half-life of BDF in overdose is not well

established. Case Report: A 41-year-old female ate 15 boxes of BDF-containing rat poison, which contained 0.005% brodifacoum. Two months later the poison center was contacted because of continuing elevations of anticoagulation despite vitamin K therapy (50 mg PO tid). Her PT at that time was 101.8 and INR was 68.3. Other than a single guiac positive stool, there was no evidence of bleeding. An initial BDF level was 390 ng/mL. Four days later, a BDF level of 460 ng/mL was identified, consistent with a reingestion. Serial BDF levels were instituted (table).

SERUM BRODIFACOUM LEVELS (NG/ML)						
Day	0	4	21	28	54	65
Level	390	460	260	230	130	120

Curve estimation resulted in statistically significant results for both linear and exponential models, consistent either with 0 order or 1st order elimination, assuming a single compartment. However, a semilogarithmic plot of levels versus time was consistent with a multiple compartment model. Multiple curve models were statistically significant, including linear. A terminal half-life of 39.4 days was calculated; assuming a 1st order, multiple compartment model. Conclusion: The pharmacokinetics of BDF are not well delineated. BDF may be distributed into multiple compartments, and may exhibit a terminal half-life of 39.4 days.

6 USE OF BEBULIN® IN THE TREATMENT OF BRODIFACOUM POISONING.

Eng J, Ramstack T. *Michigan State University Emergency Medicine Residency, Ingham Regional Medical Center, Lansing, MI*

Background: Brodifacoum poisoning is a health concern. Scenarios in which it occurs include: accidental ingestion in children, suicide attempts, and Munchausen syndrome. This case reports a patient who attempted suicide with a brodifacoum product who was treated with Bebulin® VH Immuno (Factor IX Complex). Case Report: A 52-year-old man presented to the emergency department by ambulance in acute respiratory distress. He complained of throat swelling. The patient had an expanding hematoma to the anterior neck region superior to the supraclavicular notch. He also had blood present around his lips. Further exam revealed blood on the oral mucosa. The patient's respiratory status declined rapidly. Oral intubation was unsuccessful due to the large amount of blood in the oral pharynx and the loss of anatomical landmarks. Cricothyrotomy was not attempted in the emergency department due to the expansion of the neck hematoma. The patient was emergently taken to the operating room for placement of a tracheostomy. Initial laboratory values included a PT $>$ 100 seconds, INR $>$ 98.5, aPTT 111.3 seconds, Hb 10.7, Hct 31.6, Factor VII $<$ 4, and Factor VI $<$ 4. The patient was treated with Bebulin® 2000 units IV and vitamin K. He was subsequently discharged for extensive psychiatric evaluation and treatment with a PT $<$ 12 seconds. He was maintained on oral vitamin K. Conclusion: There have been many case reports of brodifacoum poisonings in the literature. These cases have been treated routinely with vitamin K therapy. We report the first case of brodifacoum poisoning treated with Bebulin® VH Immuno (Factor IX Complex) with an excellent patient outcome.

7 SURREPTITIOUS BRODIFACOUM POISONING.

Dahl B, Caravati M, Dunson W. *Utah Poison Control Center, Salt Lake City, UT*

Background: Brodifacoum is a long-acting anticoagulant found in many rodenticides. We report a surreptitious case of human poisoning that resulted in prolonged anticoagulation and unusual brodifacoum concentrations. Case Report: A 19-year-old man reported eating at a restaurant when he noticed "crunchy" pellets in his food. Thinking that the pellets resembled rodenticide, he immediately went to an ED where his exam was normal and PT-INR was 1.2. Six days post-exposure, he went to a clinic complaining of lightheadedness, diarrhea with bright red blood, chills, bloody nose, eye pain, chest pressure and wheezing. At this time his INR was 8.4. He was admitted to the hospital and treated with both oral and subcutaneous vitamin K. Lab studies revealed a brodifacoum serum concentration of 390 ng/mL and a warfarin serum concentration of 0.2 mcg/mL. After 83 days of vitamin K therapy, his brodifacoum was 12 ng/mL and INR was 1.0. Vitamin K therapy was continued 4 additional weeks. He returned 8 days after stopping treatment with rectal bleeding. His INR was 2.0 and his brodifacoum was 110 ng/mL. The patient denied any re-exposure. He required vitamin K therapy of 25 mg SQ BID and 50 mg PO BID for an additional 10 weeks until brodifacoum was

no longer detected in his serum, his INR was consistently normal and he was without further symptoms. Discussion: Human unintentional exposures with significant clinical effects are rare. It is unusual that this patient unintentionally ingested enough of a 0.005% brodifacoum product to cause the extremely elevated serum concentrations. Subsequent serial brodifacoum levels revealed re-exposure that was denied by the patient. He required high dose vitamin K therapy for over 6 months. Conclusion: Brodifacoum serum concentrations were useful in documenting a significant surreptitious exposure and predicting the duration of treatment required.

8 NO ADVERSE SEQUELAE FROM AN INTENTIONAL OVERDOSE OF SUPERWARFARIN AND GLASS DESPITE AN INR OF 38.

Tsutaoka B, Miller M, Patel M, Greethong J, Olson K. *California Poison Control System, San Francisco Division, San Francisco, CA*

Background: Deaths in patients with intentional overdoses with superwarfarins have been reported in the literature. We report a case of an overdose with mouse bait containing brodifacoum along with the consumption of broken pieces of glass. Case Report: A 23-year-old male presented to the Emergency Department (ED) after eating 4 boxes of mouse poison over 4 days and approximately 2 bottles of glass over the previous 2 weeks. He presented with a history of passing glass in his stools, but was asymptomatic with an INR = 38. A KUB showed diffuse radiopaque foreign bodies (presumably glass) in the large and distal small bowel. Treatment for the glass retained in the bowels consisted of stool softeners and bulk-forming laxatives. The patient developed gingival bleeding and received FFP and Vitamin K_1 orally. Vitamin K_1 was titrated up to 300 mg/day with correction of the INR to <2.0. The patient was discharged with follow up. He returned to the ED 26 days later complaining of hematuria and flank pain. An abdominal CT scan was negative. INR was 92. He was again administered FFP and Vitamin K_1 orally up to 800 mg/day with return of INR to baseline. Brodifacoum levels drawn 43 and 85 days after presentation were 350 mg/mL and 36 ng/mL respectively. At 5 month follow up the patient was doing well. Conclusion: Glass ingestions have rarely been reported to cause bleeding complications. Our patient, despite an extremely elevated INR in the setting of glass and superwarfarin ingestion suffered no serious sequelae. Appropriate treatment with large oral doses of Vitamin K_1 and very close follow up are necessary for a favorable outcome.

9 PYRETHROID PESTICIDE ILLNESSES IN OCCUPATIONAL SETTINGS.

Das R[1], Beckman J[2], Vergara X[2], Sutton P[2], Harrison R[1]. [1]*California Department of Health Services, Oakland, CA;* [2]*Public Health Institute, Berkeley, CA*

Background: The use of pyrethroid insecticides is encouraged because they are considered nontoxic to humans. In 1999, over 500,000 pounds of pyrethroids were used commercially in California. Because of prevalent use, large numbers of workers are exposed to these insecticides. However, the role of pyrethroids in causing disease in exposed workers has not been profiled. Methods: To characterize occupational pyrethroid-induced illness, a review was conducted of all cases reported by physicians, poison control centers and others to a state occupational pesticide illness surveillance system from January 1, 1998 to March 30, 2000. Results: Out of 844 occupational exposures to non-disinfectant pesticides, 134 cases (15.9%) representing 45 exposure incidents were related to pyrethroids. Selected variables are summarized below:

Variable	Indoor Exposure	Outdoor Exposure
No. illnesses related to pyrethroid pesticides (%)	32 (23.9)	102 (76.1)
No. males (% of illnesses in category)	7 (21.8)	81 (79.4)
Age: mean (range)	36.6 (20–55)	34.4 (13–68)
No. workers per exposure incident: mean (range)	2.9 (1–10)	3 (1–49)
Symptomatic (≥ 1 symptom): n (%)	31 (96.9)	87 (85.3)
Type II pyrethroid involved: n (%)	19 (59.4)	83 (81.4)

Conclusion: Pyrethroids were responsible for a large number of occupational pesticide illnesses. Illnesses occurred in a variety of workers and in a wide range of exposure scenarios, with the majority being reported in the outdoor setting. The longer acting Type II pyrethroids were responsible for more illnesses than Type I compounds.

10 ACUTE OCCUPATIONAL PESTICIDE-RELATED ILLNESS AMONG CHILDREN, 1988–1999.

Geoffrey M. *Calvert, Division of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Cincinnati, OH*

Background: Working children face many safety and health risks. Despite widespread concern about their vulnerability to pesticides, information on acute occupational pesticide-related illness among children is not available. The objective of this paper is to describe acute occupational pesticide-related illness among children, and to provide recommendations to prevent these illnesses. Methods: Survey data collected between 1988–1999 on acute occupational pesticide-related illness among individuals less than 18 years were obtained from agencies (generally state departments of health) in eight states. In addition, data from 1993–1998 were obtained from the Toxic Exposure Surveillance System, which collects poisoning reports submitted by approximately 85% of the US poison control centers. Results: A total of 531 children were identified with acute occupational pesticide-related illness. Insecticides were responsible for a majority (68%), and most illnesses were of mild severity (79%). There were no fatalities. The average annual incidence rate was 2.04/100 million hours worked, but was much higher among those employed in agriculture (19.7/100 million hours worked). Between 1993–1998, the overall risk among children compared to adults was 1.71 (95% CI = 1.53, 1.91). Conclusions: The findings suggest the need for greater efforts to prevent childhood acute occupational pesticide-related illness. This may require strengthening regulations and enforcement.

11 MOVEMENT DISORDER IN AN ADULT FOLLOWING EXPOSURE TO DEET.

Ratner M[1], Tahler D[2], Cabella D[1], Feldman R[1]. [1]*Boston University School of Medicine, Boston, MA;* [2]*New England Medical Center, Boston, MA*

Background: *N,N*-Diethyl-m-toluamide (DEET) is a widely used topical insect repellent known to produce central nervous system effects by an unknown mechanism. Consumer products contain 10–30% DEET in an alcohol base. DEET is absorbed through the intact skin. Elimination of a single dose of DEET is complete within 2–3 days in animals. Excretion of DEET by humans is rapid but not as complete as in animals with only about half of an absorbed dose of DEET excreted in 5 days permitting accumulation with repeated dosing. The most frequently reported neurotoxic effect of DEET in humans is seizures. Near lethal doses of DEET produce spongiform myelinopathy in the cerebellar roof nuclei of rats. Case Report: A previously healthy 30-year-old man developed an asymmetric bilateral action tremor that progressed over 2–3 weeks and then stabilized. His symptoms began after 3.5 months of exposure to DEET which he had been applying every day to his sheets, blankets, and pillows and bedclothes. He also wore clothing treated with DEET when in his home during this period. He used four 6 oz cans of spray containing 28.5% DEET per month. Each can contained 47 g of DEET. He was potentially exposed to 672 g of DEET in 3.5 months or 47 g per week. This can be compared with 442 g in 6 months among workers using repellents containing DEET. Neurotoxic effects are have been reported in workers exposed to $>$ 4 g of DEET per week. There was no history of exposure to other chemicals and an extensive work-up failed to reveal a non-neurotoxic etiology for the movement disorder. Conclusions: These findings suggest that subchronic exposures to high concentrations of DEET may produce movement disorders in susceptible persons.

12 INGESTION OF RATBANE 1080 SODIUM MONOFLUORACETATE.

Robinson R, Griffith J, Nahata M, Wolowich W. *Children's Research Institute, Central Ohio Poison Control Center, Ohio State University College of Pharmacy, Columbus, OH*

Background: The highly toxic agent (LDLo man 5 mg/kg) sodium monofluoracetate (SMFA) was banned as a rodenticide in the US in 1972. We report the first case of intentional ingestion in the US in over 15 years. Case Report: A 47-year-old male, was brought to the emergency room status post tonic clonic seizure. The patient was agitated, obtunded, acidotic (pH = 7.3, pCO_2 = 29 mmHg, bicarbonate 14 mEq/L, anion gap 24 mEq/L) his pO2 was 92 mmHg and serum creatinine 2 mg/dL. The urine screen was positive for benzodiazepines given for seizure, with no evidence of fluorescence or calcium oxalate crystals. An ethanol drip was started and folate, pyridoxine, and thiamine were given for possible ethylene glycol or methanol ingestion. At 34 hours post ingestion he responded only to noxious stimuli, pupils were equal and reactive, and EEG showed mild diffuse slowing. At 48 hours the patient became unresponsive to painful stimuli, diaphoretic, tachypneic (45 bpm), tachycardic (110 bpm), febrile, was intubated and placed on a ventilator. Chest radiograph showed pulmonary edema. Over the following 3 days, he was minimally responsive to external stimuli with bouts of agitation, and hypertension (144/83 mmHg), his renal function declined (BUN = 53 mg/dL, serum creatinine 4.3 mg/dL, urine output 100 mL/hr). Two days later, he was discharged from the hospital with

no evidence of neurologic sequelae. Conclusions: We report this case to increase awareness of SMFA and its ability to cause anion gap metabolic acidosis. Patients with access (farmers, exterminators) should receive aggressive decontamination and attempts should be made to identify the agent to avoid unnecessary antidotes and adverse outcomes.

13 LATE ADMINISTRATION OF OBIDOXIME IN MALATHION POISONING.

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Background: Organophosphate (OP) intoxication is due to accumulation of acetylcholine in synaptic clefts and myoneural junctions following phosphorylation of acetylcholinesterase. Early treatment with oximes results in reactivation of the enzyme and patient recovery. Data on the efficacy of late administration of oximes, particularly obidoxime, is limited. Case Report: A 42-year-old woman swallowed 60 mL of 50% malathion in a suicide attempt. She was immediately brought to the emergency department and her stomach was lavaged. Characteristic muscarinic, nicotinic and central manifestations of OP poisoning were observed. She received atropine and a total of 3 doses of 250 mg obidoxime I.V. with marked improvement. The next day severe bronchial secretions and muscular weakness recurred and were treated with atropine and mechanical ventilation. Ten days after admission the patient was transferred to another hospital as she could not be weaned from the ventilator. She manifested fasciculations as well as proximal and respiratory muscle weakness. Plasma and red blood cell cholinesterases were 0.8 U/mL (normal >2) and 1.6 U/mL (normal >6), respectively, and liver enzymes were elevated (ALT 136 U/L). Obidoxime was reinstituted after more than 9 days at a dose of 250 mg I.V. every 6 hours. Within 24 hours the patient could be weaned from the ventilator and plasma cholinesterase normalized (3.1 U/mL). Obidoxime was continued for another 4 days during which muscle strength recovered almost completely and liver enzymes returned to normal. Conclusion: The successful management of this patient shows that obidoxime treatment should be considered late in the course of untreated or partially treated OP intoxications, particularly when the culprit is a lipid-soluble compound such as malathion. This case does not support a cause-and-effect relationship between obidoxime and liver function abnormality.

14 OUTCOME OF METHANOL POISONING IN CHILDREN TREATED WITH ETHANOL: A 21-YEAR PERSPECTIVE.

Roy M, Bailey B, Chalut D, Senécal PE, Gaudreault P. *Hôpital Ste-Justine, Université de Montréal, and Montreal Children's Hospital, McGill University Health Centre, Montréal, Quebec, Canada*

Background: Although ethanol (EtOH) has been widely used for decades, there is little data documenting its efficacy in preventing pediatric methanol (M) poisoning complications. Methods: Charts review of children that were admitted to two pediatric hospitals and had received EtOH for M poisoning. Results are presented as median (range) or as mean ± SD (range). Results: From 1980 to 2000, there were 22 children that received EtOH for M levels ≥ 20 mg/dL. The initial M level was 38 mg/dL (20–280). 7 patients received only PO EtOH for 5 doses (1–19): bolus and maintenance doses were 1.0 g/kg (0.24–1.66) and 0.18 g/kg/h (0.05–0.23). 13 patients received only IV EtOH for 20.5 hours (5–65): bolus and maintenance doses were 1.0 g/kg (0.66–1.86) and 0.18 g/kg/h (0.07–0.44). The lowest EtOH level observed was 20 mg/dL (0–114). The variation with the highest EtOH level was 76 mg/dL (10–211). On admission, 7 had acidemia (bicarbonates (HCO_3^-) ≤20 mEq/L) with no further decrease in HCO_3^- during EtOH. 4 patients with normal HCO_3^- on arrival developed acidemia during EtOH. In those 4 patients, the mean HCO_3^- on arrival was 23.1 ± 1.6 mEq/L (20.8–24.2); the mean lowest HCO_3^- observed was 17.9 ± 0.6 mEq/L (17.2–18.7). 9 patients were hemodialysed: 8/8 with M levels >50 mg/dL; 1 with M level of 38 mg/dL and HCO_3^- of 18.5 mEq/L. No patient had visual impairment either on admission or on discharge. 9 patients were admitted to PICU for a stay of 24 hours (12–48) and transferred on a ward for a stay of 31 hours (0–96). 13 patients were admitted directly to a ward for a stay of 48 hours (13–216). Conclusion: Children treated with EtOH for M poisoning usually had good prognosis despite the wide variation in EtOH levels.

15 METHANOL CONTAMINATION OF ROMANIAN HOME-DISTILLED ALCOHOL.

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Background: Ţuica is a home-distilled alcohol consumed in great quantities throughout Romania. It is made from locally available fruits, primarily plums and apples. In a retrospective case review, methanol poisoning was found to be a

common and frequently lethal complication of Ţuica consumption. The objective of this prospective observational study was to characterize the incidence of potentially dangerous methanol contamination in home-distilled alcohol produced in Mures County, Romania. Methods: During a one-month period local Ţuica distilleries were visited. After obtaining permission from the distillery operator, 5-mL samples of Ţuica were collected and stored individually in sterile, additive-free glass vacutainers. The character of the distillery and the fruits used in the process were recorded. All samples were transported to a reference laboratory for determination of their ethanol and methanol content by gas chromatography. Laboratory staff was blinded to the sources of all samples. Results: 31 distilleries were visited, from which 35 samples were obtained. 26 samples (74%) contained detectable methanol levels (mean 0.48 ± 1.43 gm/dL, range 0.06–8.6 gm/dL). 9 samples (34%) contained methanol levels above 0.35 gm/dL, the threshold of safety allowed by the Bureau of Alcohol, Tobacco, and Firearms in the United States. Ethanol levels were divergent (mean 26.0 ± 12.1 mg/dL, range 0.10–49.07 mg/dL). Conclusions: Methanol was a contaminant in the majority of home-distilled alcohol analyzed in this study. A significant proportion of these samples contained toxic levels. Ethanol content varied greatly. Romanian public health policy should address the population health risks associated with methanol consumption and develop strategies to reduce the methanol content in the available home-distilled alcohol.

16 WHAT ARE THE ADVERSE EFFECTS OF ETHANOL USED AS AN ANTIDOTE IN THE TREATMENT OF METHANOL POISONING IN CHILDREN?

Roy M, Bailey B, Chalut D, Senécal PE, Gaudreault P. *Hôpital Ste-Justine, Université de Montréal, and Montreal Children's Hospital, McGill University Health Centre, Montréal, Quebec, Canada*

Background: Ethanol (EtOH) used as an antidote is said to have various adverse effects, particularly in children. However, the rate of these adverse effects is not known. Methods: The last 21 years (1980–2000) charts from ethanol-treated methanol (M) poisoning cases were reviewed in 2 pediatric hospitals. Cases were divided in 2 groups: 1) Received EtOH bolus and maintenance doses (BM group). 2) Received only EtOH bolus (B group). Results: A total of 60 children (median age of 24 months) received EtOH (39 PO and 21 IV): 49 BM group and 11 B group. No symptomatic hypoglycemia was reported [95% CI 0–5%]. Of the patients that had a glycemia measured, 0/50 [95% CI 0–6%] had a glycemia <50 mg/dL. However, 8/50 or 16% [95% CI 8–30%] had at least one glycemia between 50–65 mg/dL: 7 BM group and 1 B group. 42/50 or 84% [95% CI 70–92%] had all glycemia >65 mg/dL: 34 BM group and 8 B group. There was no difference in glucose intake between the 2 latter glycemia groups ($p = 0.95$): median 0.22 vs 0.18 g/kg/h, respectively. A total of 6/60 or 10% [95% CI 4–21%] of patients were described as more drowsy after EtOH; but none were comatose or needed intubation. 1/30 or 3% [95% CI 0.2-19%] had hypotension (BP < 2 SD for age): a patient that arrived already hypotensive 72 hours post ingestion with a pH of 6.76. 0/40 patients [95% CI 0–8%] was hypothermic (rectal T < 95 °F), 0/12 had hepatotoxicity (AST or ALT > 100 U/L) and 0/60 [95% CI 0–5%] had thrombophlebitis. Amylase or lipase levels were not measured. One patient had an erosive gastritis (IV EtOH). Conclusion: When ethanol was used as an antidote in the treatment of methanol poisoning in children, the rate of clinically important adverse effects was low.

17 OSMOLAR GAP AS TOXIC ALCOHOL SURROGATE: TESTING THE TEST.

Marcus S, Ruck B, Jennis T, Swenson R. *New Jersey Poison Information and Education System, Newark Beth Israel Medical Center, Newark, NJ*

Background: Few hospital laboratories are equipped to do stat assays for the toxic alcohols. Toxicologists often depend on calculating an osmolar gap as a surrogate marker. This study was conducted to ascertain the usefulness of this test. Methods: a retrospective review of data collected prospectively on all cases of suspected toxic alcohol exposure cases reported to a poison center over an 18 month period was performed. Seventy eight cases were identified, 20 met the inclusion criteria: an ingestion of a toxic alcohol suspected either on the basis of history or clinical presentation and a set of electrolytes and a freezing-point depression serum osmolarity were obtained on the same sample as an analysis in a reference laboratory for the toxic alcohol. Results: If one accepts as ''normal'' an osmolar gap of <8 mosm/L then 12 cases met this criteron with one false negative and 5 false positives for a sensitivity (sn) of 92 and a specificity (sp) of 38, a positive predictive value (ppv) of 69 with a negative predictive value (npv) of 75. If one raises the threshold to a gap of >12, then the values become sn = 85, sp = 71, ppv = 85 and npv = 71. Raising the threshold further does not result in a significant effect on the specificity but greatly reduces the sensitivity with little change in either the positive or negative predictive values, at a gap of >16 values become sn = 75, sp = 63, ppv = 75 and npv 63

and at a gap of >20 sn = 67, sp = 78, ppv = 80 and npv = 64. Conclusion: These data suggest that the osmolar gap calculation is only a fair surrogate marker for the presence of a toxic alcohol. Further it suggests that the previously accepted "normal" of <8 mosm may be too low and that by raising the bar to 12 the specificity increases dramatically without a drastic drop in sensitivity.

18 IS THE OSMOLAR GAP (OG) A RELIABLE SURROGATE FOR ETHYLENE GLYCOL (EG) MEASUREMENT?

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Background: EG levels are not readily available in most hospitals. The presence of an OG has therefore been proposed as a surrogate measure of serum EG. Some authors even suggest that absolute serum EG levels in mg/dL may be estimated by multiplying the OG by a conversion factor of 6.2. We report a patient with severe EG poisoning in whom serial measurements of OG were used to estimate EG levels and guide treatment decisions. Case Report: A 40-year-old alcoholic man drank 0.5 gallons of antifreeze in a suicide attempt. He was treated at a facility where 12–24 hrs were required to obtain serum EG results. The patient's initial OG was markedly elevated and this was used to justify the need for dialysis. There was no ethanol detectable. The OG was then followed serially to guide the timing and duration of dialysis treatments. The patient was dialyzed a total of 3 times until his OG was normal. Serial OG and EG measurements were:

Hours after ingestion	OG	EG (mg/dL)	Calculated EG (mg/dL)
8	161.3	676.0	1000.1
44	15.4	80.7	95.5
68	2.4	47.2	14.9

No further dialysis was performed after the OG reached 2.4, and the creatinine subsequently rose from 2.6 to 3.6 mg/dL. A month later it was 1.2 mg/dL. Conclusion: Although an elevated OG may be helpful in deciding to initiate therapy for EG ingestions, the OG may not accurately predict actual EG levels. Moreover, a normal OG does not exclude a potentially toxic EG level.

19 OUTCOME OF ETHYLENE GLYCOL TOXICITY AFTER ETHANOL TREATMENT.

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Introduction: There have not been prospective studies done with ethanol therapy (ETOHT) for ethylene glycol toxicity (EGT), or comparisons with fomepizole. The goal of this study is to summarize the outcomes after ETOHT. Methods: Using aspects of both meta-analysis and retrospective medical record review methodology all English-language reports of EGT receiving ETOHT were abstracted. Publications between 1942 and 2000 were identified using electronic and manual search strategies. A case abstract form and definitions were developed, pre-tested, and modified prior to case review. The primary outcome was renal impairment after initiation of ETOHT and associated adverse events. Information about EGT, ETOHT, and other treatment were documented. Results: 52 English language publications describe 133 cases of EGT that received ETOHT. EG concentrations were measured in 126 cases; 77 had arterial acidosis at presentation and 109 cases underwent hemodialysis. At presentation 26 cases had abnormal, 39 normal, and the remainder undocumented renal function. 48% (95% CI 32% to 63%) of cases that presented with normal renal function had abnormal renal function after ETOHT. Overall, 57% (95% CI 48% to 66%) of cases had documented abnormal renal function after EGT. 18 cases underwent hemodialysis treatment for EG renal toxicity. There were 8 cases with adverse events associated with ETOHT, including CNS depression and apnea. Conclusion: The English language literature (133 cases) suggests that approximately half of all EG exposures that present for treatment with normal renal function will experience renal toxicity even with ethanol therapy, and 5% will have adverse events from ethanol therapy.

20 AN APPROACH TO DIALYSIS IN ETHYLENE GLYCOL INTOXICATION.

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Methods: A cluster of 11 cases of ethylene glycol (ETG) intoxication is retrospectively analyzed. Patients were grouped according to the time it took them to receive medical attention. Results: Six patients presented within 12 hrs of ingestion

(group 1), while 5 presented later (group 2). Comparisons between groups were made for age, ETG level, anion gap, osmolar gap, pH, admission serum creatinine (Cr), time from ingestion to presentation, total time spent on hemodialysis (HD), number of dialysis treatments, recovery time, recovery Cr, hospital length of stay, and status. Significant differences between groups were observed for ETG level ($p = 0.01$), osmolar gap ($p = 0.003$), and presentation time ($p = 0.006$). Ten patients received single pass HD and 5 of these had one continuous cycle of HD (CHD). None of the patients who were treated with CHD developed a complication (defined as oliguric acute renal failure or death), whereas all patients treated with intermittent hemodialysis (IHD) had a complicated course ($p = 0.004$). An ETG index (ETG level times weight) was calculated for patients on CHD and plotted against time on dialysis necessary to close the osmolal gap. Regression analysis resulted in $R^2 = 0.923$. Patients presenting within 10 hrs of exposure had fewer complications than those who presented >12 hrs post exposure. Discussion: Timely delivery of CHD is superior to IHD in treating ETG intoxication in ETG poisoning. Furthermore, an adequate time course of HD can be approximated by using the ETG index.

21 TREATMENT OF TWO ADOLESCENT GIRLS WITH SEVERE ETHYLENE GLYCOL POISONING WITHOUT HEMODIALYSIS.

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Background: Hemodialysis is routinely recommended for treatment of ethylene glycol (EG) poisoning when initial serum concentrations are greater than 50 mg/dL. Experience in managing these poisonings with alcohol dehydrogenase (ADH) inhibitors alone is limited. We report 2 cases of EG intoxication with extremely elevated initial serum EG concentrations treated without hemodialysis. *Case Reports:* Two 13-year-old girls drank EG antifreeze solution and presented to the ED with lethargy, nausea and slurred speech. Initial labs were:

	EG (mg/dL)	CO2 (mmol/L)	Creatinine (mg/dL)	Anion gap	Osmol gap
Case 1	270	16	0.7	21	78
Case 2	113	17	1.0	20	30

Both girls were initially treated with loading doses of ethanol IV, then converted to fomepizole (15 mg/kg, then 10 mg/kg q 12 h). They were treated until the EG concentrations were <10 mg/dL which required 6–7 total doses of fomepizole each. Both developed transient 1+ oxalate crystaluria but no alteration in renal function. Total hospital duration was 72–82 hours. Conclusion: Adolescent patients with extremely high EG concentrations and normal renal function were successfully treated with ADH inhibitors alone and avoided hemodialysis.

22 FLUORESCENCE AS AN ADJUNCTIVE TEST IN SUSPECTED ETHYLENE GLYCOL INGESTION.

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Background: Fluorescein, an additive dye to commercial antifreeze, has been utilized as an adjunctive test in cases of ethylene glycol (EG) ingestion. Our past controlled, prospective study in volunteers given fluorescein in amounts to approximate a toxic exposure to EG showed fluorescence (F) was detectable. We now report a study to test the sensitivity of detection of F in patients that have ingested EG. Methods: IRB approval was obtained at 7 poison centers for collection of data for this study. Data was submitted over a 2 year period. Inclusion criteria include a history suspect for EG poisoning. Healthcare providers were asked to examine the mouth and urine for F. Exclusion criteria were a history of inhalation, dermal, or multiple exposures, or no evaluation for oral or urine F. Results: Two centers and one statewide network reported data for analysis for a total of 611 cases. 378 cases were inhalation or dermal exposures. In addition 217 cases were excluded due to lack of documentation of recommendation to check for F leaving 394 cases of which 378 were inhalation or dermal exposure only. Of the remaining 16 cases 5 (31%) had urine F detected. Three of these patients had EG concentrations measured (246 mg/dL ± 330 s.d.). One patient each had oral cavity and GI contents F detected. Conclusion: Examination of the mouth and urine for fluorescence while awaiting serum EG concentration

had a sensitivity of 31% in this study. Examination of the mouth and urine for fluorescence while awaiting serum EG concentrations may have utility in the early evaluation of a poisoned patient.

23 CONFIRMATION OF DEATH DUE TO ETHYLENE GLYCOL INGESTION BY MEASUREMENT OF BLOOD AND URINE GLYCOLIC ACID LEVELS.

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Objective: Due to its affinity for alcohol dehydrogenase, ethylene glycol is rapidly metabolized to a number of toxic organic acids. Death from ethylene glycol poisoning is usually secondary to the effects of profound anion gap metabolic acidosis with resulting cardiac, respiratory and renal failure. *Case Report:* A 42-year-old female was found slumped over in her car. An empty antifreeze container (ethylene glycol) was also found. Based on the accumulated evidence, the victim appeared to have ingested the antifreeze about 4 days prior to the discovery of the body. Autopsy findings showed death to be the result of pulmonary edema. Toxicological analysis using gas chromatography failed to show any evidence of ethylene glycol in either postmortem blood or urine samples. Results: Gas chromatography showed high levels of glycolic acid in both the serum and urine of the deceased confirming death secondary to ethylene glycol ingestion. Conclusion: The confirmation of death secondary to ethylene glycol poisoning may require additional testing for the presence of ethylene glycol metabolites. The finding of glycolic acid in biological specimens indicates recent exposure to ethylene glycol and may help substantiate the cause of death. The finding of calcium oxalate crystals should alert the pathologist to request testing for the presence of ethylene glycol and glycolic acid in the deceased.

24 ACUTE PROPYLENE GLYCOL INGESTION.

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Background: We describe a case of acute propylene glycol (PG) toxicity following ingestion of EtOH and PG-containing antifreeze in which serum PG, EtOH, lactate, and CO_2 were serially measured. Case Report: A 61-year-old man was transferred to our service after acute ingestion of EtOH and automotive antifreeze. Initial lab tests revealed serum ethanol level, 167 mg/dL, normal serum electrolytes, and osmol gap, 84 mOsm. IV 10% EtOH infusion was begun for suspected ethylene glycol toxicity and discontinued at approximately 17 hours post-ingestion. Toxicological analysis of urine was positive for EtOH and PG, and negative for ethylene glycol, methanol, and isopropanol. Serial assays of serum PG, lactate, EtOH, and CO_2 yielded the following results:

Time (hrs)	*PG (mg/dL)*	*Lactate (mmol/L)*	*EtOH (mg/dL)*	*CO_2 (mmol/L)*
1	—	—	167	23
7	470	—	145	23
11	419	2.8 (13 hrs)	138	—
18	336	3.1	33	24
25	188	3.1	<10	—
28	90	—	—	—
31	—	1.4	—	26
57	0	1.3	—	—

Conclusion: The elimination of PG followed zero-order kinetics. Our patient's clinical course was benign, resulting in his discharge in stable condition on hospital day 2.

25 SALICYLATE UNDETECTED FOR 8 HOURS AFTER ENTERIC-COATED ASPIRIN OVERDOSE.

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Background: Pharmacokinetic studies of enteric-coated aspirin tablets have shown that gastric emptying is random and often variable, delaying intestinal absorption for up to 12 hours. In contrast, overdose should increase the chance of at least one tablet leaving the stomach, and should decrease the chance of delayed absorption. Among the longest delays reported was an overdose of 88 enteric-coated aspirin 325 milligram (mg) tablets that resulted in a 7 hour level of 13.3

mg per deciliter (mg/dL) following undetectable levels at 1 and 3 hours. Such cases suggested that if serum salicylate levels remained at or below the limit of detection for 8 hours then a significant ingestion of enteric-coated aspirin was unlikely. Case Report: An Emergency Department contacted the Poison Center about a 13-year-old female who ingested an unknown amount of enteric-coated aspirin (325 mg each) about 2 hours prior to presentation. Her treatment included activated charcoal and intravenous fluids without sodium bicarbonate. Her aspirin levels were reported as follows:

Time postexposure:	2 hours	8 hours	14 hours	20 hours	26 hours	32 hours
Salicylate level:	<2.8mg/dL	4 mg/dL	36 mg/dL	34 mg/dL	30 mg/dL	25 mg/dL

She remained asymptomatic and was discharged after 2 days. Conclusion: This case reports that a significant salicylate level was not detected until 14 hours, and suggests that most absorption began 8 to 14 hours after exposure. The reasons for this lag-time are unknown. Possibilities include delayed gastric emptying, failed disintegration of the enteric coating, or desorbed aspirin from the activated charcoal. A delay of this magnitude could result in a patient's premature release from medical care.

26 HEMOFILTRATION–A POTENTIAL NEW TREATMENT FOR SALICYLATE POISONING.

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Objective: Hemodialysis is currently the recommended treatment for patients with severe salicylate poisoning. Hemofiltration, unlike hemodialysis, is available in a larger number of hospitals and is better tolerated in hemodynamically unstable patients. The aim of this study was to determine whether hemofiltration is a possible alternative to hemodialysis in severe salicylate poisoning. Methods: An *in vitro* hemofiltration model was used, with a Hospal AN69 filter and 500mL of 4% bovine serum albumin (BSA) as the carrier solution. Three different salicylate concentrations were studied (300, 600 & 900mg/L), with six runs for each starting concentration. Hemofiltration was performed for 180mins at a BSA flow rate of 150mL/min; ultrafiltrate was replaced with deionized water. Pre-filter, post-filter and ultrafiltrate samples were taken every 5mins for the first 60mins and then every 15mins. Results: The mean total amount of salicylate removed by hemofiltration (% amount initially present (±95% CI)) was 53% (±8%) for the 300mg/L runs, 69% (±12%) for the 600mg/L runs and 70% (±2%) for the 900mg/L runs. The sieving coefficient was constant over the 180mins studied; 0.4 (±0.05) for the 300mg/L runs, 0.65 (±0.08) for the 600mg/L runs and 0.75 (±0.04) for the 900mg/L runs. Conclusions: This *in vitro* model shows for the first time that hemofiltration can provide effective clearance of salicylate at toxicologically significant concentrations. The removal of 53–70% salicylate would be expected to be clinically significant in a severely poisoned patient. Although other factors will influence overall elimination and require further evaluation, this study shows that hemofiltration has important potential in treating severe salicylate poisoning.

27 ASPIRIN OVERDOSE IN MOTHER AND FETUS.

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Background: Although it is known that aspirin can cross the placenta, there are no studies describing the effects of aspirin on the live fetus and no correlation has been made between mother-newborn serum salicylate levels. Case Report: A 19-year-old woman in her 38th week of pregnancy ingested a total of 16.25 g of aspirin in a suicide attempt. On arrival to the emergency department, the maternal salicylate level was 31.7 mg/dL. Physical examination revealed stable vital signs with mild tachypnea. The patient, however, denied tinnitus, gastrointestinal, or neurological symptoms. Fetal monitoring applied in labor and delivery revealed fetal distress with bradycardia (HR- 60) and late decelerations. For this reason an emergency Cesarean section was performed and APGAR scores were noted to be 5 and 7 at 1 and 5 minutes respectively. Bag-valve-mask ventilations were required for a brief period after delivery. Maternal salicylate level drawn just prior to Cesarean section was 14 mg/dL. The baby's salicylate level, drawn immediately after delivery, was 35.2 mg/dL. Newborn vital signs soon after delivery were BP 65/46, HR 142, RR 58, pulse oximetry 100% on room air. Laboratories showed a pH of 7.49 with a pCO2 of 27 mm Hg; electrolytes were normal except for a bicarbonate of 18 mEq/L. Serial aspirin levels showed a value of 26.4 mg/dL at 28 hours, and 8.1 mg/dL at 101 hours post delivery. The baby was discharged without any obvious problems. Conclusions: We describe a case of perinatal aspirin poisoning, where the baby had higher levels than those of the mother. The baby had significant fetal distress with only minor

symptoms reported in the mother. We do not know the reason for higher salicylate levels in the fetus, although it may be due to excretion mechanisms that are not well developed. It is unclear as to which is the safest method for treatment in these cases.

28 BRINGING DONE'S NOMOGRAM INTO THE 21ST CENTURY.

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Background: In November 1960, Done published his sentinel "nomogram" observation based upon data from 29 children and 9 adults with acute salicylate (36) or oil of wintergreen (4) ingestions. It demonstrated that serum salicylate levels were predictive of clinical outcomes only when time since ingestion was factored into the predictive equation. In 1963, we confirmed his inferences in a sample of 102 children. In both instances, unfortunately, the assumption was made that absorption of the salicylate moiety took 6 hours. Even today, after 40 years, that assumption continues to be employed when one seeks to use Done's nomogram. It results in a delay in drawing initial salicylate levels till 6 hours has passed since ingestion. We decided to reexamine our 1963 data plus 50 additional cases occurring in that same year to determine the effect of using 4, rather than 6 hours, as the time delay required to achieve "absorption" of the salicylate moiety–based upon current pharmacokinetic understanding of aspirin. Method: Quite simply, the slopes resultant from Done's regression equation to achieve his S_0 levels remain unchanged because they reflected only excretion rather than absorption. Consequently, the resultant regression lines were simply "shifted" 2 hours to the left. Results: Not surprisingly, since the clinical groupings of outcome remained constant, the respective S_0 levels fell approximately 10% at the lower ranges to 7% at the higher ranges–in truth, both being clinically inconsequential. Conclusions: While these minor adjustments may help explain the small differences that occurred between the two prior studies, the consequences are, in fact, trivial. We see the resultant adjustment emphasizing no need to wait 6 hours, but rather relying on 4 hours after ingestion, to draw a representative blood sample just as is employed for the majority of other clinical poisonings.

29 HYPERTRIGLYCERIDEMIA CAUSING A FALSELY POSITIVE TOXIC SERUM SALICYLATE LEVEL.

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Background: Both acute and chronic salicylate toxicity often go undiagnosed and can result in great harm to the patient. We report a case of severe hypertriglyceridemia causing a falsely positive quantitative toxic serum salicylate level. Case Report: A 45-year-old male, type II diabetic presented to the ED for weakness. He occasionally took aspirin for headaches, but denied any significant acute or chronic salicylate ingestion. His neurological exam was normal. Initial chemistries showed a serum glucose of 534 mg/dL, sodium of 129 mmol/L, chloride of 94 mmol/L, CO2 of 13 mmol/L, an anion gap of 22, potassium of 5.1 mmol/L, a BUN of 22 mg/dL, a creatinine of 1.3 mg/dL, a serum osmolality of 295 mosm/K, an arterial pH of 7.42, pCO2 of 27, pO2 of 76, a HCO3 of 17.5, a lactic acid of 4.9 mmol/L, and a serum salicylate of 116.1 mg/dL. Serum ketones were negative. A UDS was negative for salicylate. Serum cholesterol was 618 mg/dL and triglycerides were 11,004 mg/dL. His blood was visibly lipemic. KUB x-rays were negative for concretions. Repeat serum salicylate level was 116.0 mg/dL. His urine was alkalinized to a pH = 8; multi-dose charcoal 1g/kg was given orally. Post dialysis serum salicylate level was 115.9 mg/dL. A Trinder test for rapid determination of salicylate in biological fluids was performed on his serum samples and was negative for salicylates. Conclusion: In light of the negative urine salicylate screen and the negative Trinder test, we suggest that this case demonstrates falsely elevated serum salicylate levels most likely caused by interference from severe hypertriglyceridemia.

30 ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS) AS A RESULT OF INADVERTENT INTRAVENOUS ACETAMINOPHEN SUSPENSION.

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Background: There are no previous reports of toxicity relating to intravenous administration of acetaminophen suspension. Case Report: A 16-year-old girl with Down's syndrome underwent a successful operative closure of a partial atrioventricular septal defect. Postoperatively she would only accept medication from her mother. At 48 hours post-operation her mother inadvertently gave 1 gram of acetaminophen oral suspension intravenously. 12 hours later respira-

tory decompensation occurred and oxygen and a heparin infusion (17 iu/kg/hr) were commenced. Her WBC increased to 30 × 10^9/L and platelets dropped to 45 × 10^9/L. 10 hours later, she developed worsening respiratory function and hypotension requiring intubation and ventilation, fluid resuscitation and inotropic support and transfer to PICU. In addition to lung protection strategies including high frequency oscillation for ARDS and presumed microemboli in the pulmonary vasculature, heparin was continued and she received methylprednisolone 2mg/kg/day and antibiotics (blood cultures later grew staphylococcus epidermidis). Over the next 24 hrs she continued to deteriorate, requiring increased ventilation to maintain oxygenation but by day 3 she began to improve and she was extubated on day 5. She was discharged from the hospital 10 days later self-ventilating on air and with a normal CXR. Conclusion: We describe a case of severe ARDS resulting from presumed microemboli in the pulmonary vasculature as a result of inadvertent intravenous administration of acetaminophen suspension. Therapy included heparin infusion and steroids, in addition to standard respiratory and cardiovascular support.

31 DOES INGESTED DOSE RELIABLY PREDICT TOXICITY IN ACETAMINOPHEN POISONING?

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Background: The toxic dose of acetaminophen is generally accepted as being 150mg per kg body weight. Our aim was to determine whether stated ingested dose correlates with subsequent plasma acetaminophen concentration and whether this 'toxic dose' is reliable in clinical practice. Methods: We conducted a prospective survey of all acetaminophen overdoses reported to the poisons unit over a fourteen week period between May and August 2000; overdoses presenting more than 24 h after ingestion were excluded because plasma acetaminophen concentrations are not interpretable in these cases. At the time of the initial call to NPIS data on the ingested dose, time of ingestion and the patient's weight was collected. A follow up was made the next day to determine the plasma acetaminophen concentration result (which was extrapolated back to the 4 hour level), treatment of the patient and outcome. Results: Data was collected on 261 cases. There was poor correlation ($r^2 = 0.11$) between stated ingested dose and plasma acetaminophen concentration at four hours. Of the 176 cases with a non-toxic acetaminophen concentration (according to the Prescott nomogram) of less than 200mg/L at 4 hours, 120 (68%) had a stated ingested dose more than 150mg/kg. Of the 85 cases with a toxic acetaminophen concentration of greater than 200mg/L, 17 (20%) had a stated ingested dose less than 150mg/kg. Conclusion: Stated ingested dose is a poor predictor of subsequent plasma acetaminophen concentration and hence risk of hepatotoxicity. On the basis of this study, all patients presenting after a non-staggered acetaminophen overdose should have a plasma acetaminophen concentration determined regardless of the stated dose ingested, if the time of ingestion is known.

32 COMPLIANCE WITH PCC RECOMMENDATIONS: DISCONTINUATION OF NAC.

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Background: In 1999, acetaminophen (APAP) ingestions accounted for 108,102 exposures nationally as reported in the AAPCC TESS 1999 Annual Report. The standard of care for this toxic agent has been the adminstration of *N*-acetylcysteine (NAC) for a course of 3 days, or 17 doses. Recent studies suggest that NAC can be discontinued prior to the full course without risk of hepatotoxicity, thus allowing early mobilization of patients with significant savings in healthcare costs. This study was performed to determine if health care facilities (HCF) were willing to accept early termination of NAC therapy as recommended by the PCC. Methods: All APAP toxic presentations that were handled by the PCC over a period of 5 months meeting the following criteria were included: acute acetaminophen overdose; a serum APAP level considered toxic by the Rumack-Matthew nomogram or positive acetaminophen level with unknown time of ingestion. All patients were treated with NAC for a minimum of 24 hours. After that time, if the patient's APAP level was undetected and there were no abnormalities in the liver profile, the poison specialist then recommend the discontinuation of NAC. Results: In 78.7% of the cases (37/47), the physicians followed the recommendations of the PCC, discontinuing NAC prior to 17 doses. Within this group, 48.6% of the patients received 6 doses of NAC; 48.6% received 7 to 12 doses of NAC; 2.7% of the patients received 13 to 16 doses of NAC. In 10/47 cases or 21.2%, the full course of NAC was given. Conclusion: With close collaboration between the HCF and the PCC, the majority of acetaminophen overdose patients will receive a short course of NAC therapy. In 78.7% of the cases examined, treating physicians reduced NAC therapy safely below the traditional 17 doses. No case demonstrated any untoward effect.

33 ACETAMINOPHEN-INDUCED FULMINANT HEPATIC FAILURE (FHF) TREATED WITH EXTRACORPOREAL HEMODIABSORPTION (EH) POSTPARTUM.

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Background: EH is an FDA approved treatment for acute hepatic encephalopathy and drug OD, but its use in postpartum patients with APAP (FHF) is unreported. We present a pregnant woman with poor prognostic criteria for APAP-induced FHF who was successfully treated by NAC and EH with a Liver Dialysis Unit®. Case Report: A 23-year-old woman, 27 weeks pregnant, presented with nausea and vomiting after heavy acetaminophen use for the 2 preceding days. She denied abdominal pain, fever, alcohol use or hepatitis. Vital signs were: BP, 144/98 mmHg; HR, 100/min; RR, 18/min; temperature, 97.8°F. She was alert, and her physical examination was unremarkable except for a gravid abdomen. Laboratory data were: APAP, 90 μg/mL, 6 hours after her last dose; AST, 1208 U/L; HCO_3^-, 6 mEq/L; Cr, 1.4 mg/dL; and INR, 1.7. She was started on standard oral NAC but required an emergent C-section for fetal distress. Despite maximal support, the baby expired soon after delivery. Cord blood revealed: APAP, 19.1 μg/mL and AST, <6 U/L. An autopsy on the baby was not performed. Following surgery, her clinical condition and laboratory values worsened to: AST, 9190 U/L; Cr, 3.1 mg/dL; INR, 3.8; and pH of 7.12. She was changed to intravenous NAC at 70mg/kg every 4 hours and underwent 2 courses of EH resulting in both numerical and clinical improvement. Recovery was complicated by pancreatitis and wound dehiscence. Conclusion: This patient met King's College criteria for poor outcome in APAP-induced FHF. Despite NAC therapy, she had worsening liver toxicity. Extracorporeal hemodiabsorption is designed as a bridge to liver transplant and may benefit those patients who fail NAC therapy.

34 DECREASED SERUM INTERLEUKIN-6 (IL-6) FOLLOWING ACUTE ACETAMINOPHEN (APAP) OVERDOSE IS ASSOCIATED WITH HEPATIC INJURY.

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Introduction: Acute phase reaction is a coordinated protective response to tissue injury and inflammation including the synthesis of acute phase proteins that are IL-6 dependent. C-reactive protein (CRP) belongs to this group and is considered a surrogate for IL-6. Animal studies have shown that inflammation plays a role in APAP induced liver damage. Objective: To explore serum IL-6 levels in patients following acute APAP overdose. Methods: Between May and December 2000, we conducted a prospective cohort study of patients who presented to an emergency department after an acute single overdose of APAP. We collected information on patient demographics, current ingestion including co-ingestants, past medical history, and current medication use. Serum AST, ALT, basic chemistry panel, INR, and IL-6 levels were performed. Hepatic injury was defined as AST or ALT > 50 IU/L and positive serum IL-6 was defined as greater than 40 pg/mL. Groups were compared with Fisher's exact test. Results: Thirteen patients were studied (male: 2, mean age: 26.8 yrs, range: 16–56). No one presented later than 24 hrs from reported ingestion. Serum IL-6 was statistically associated with hepatic injury ($p = 0.014$):

	Hepatic Injury	No Hepatic Injury	Total
Positive serum IL-6	0 (0%)	9 (69%)	9 (69%)
Negative serum IL-6	3 (23%)	1 (8%)	4 (31%)

Conclusion: Hepatic injury is associated with decreased serum IL-6 levels. Measuring IL-6 levels or the more clinically feasible CRP, may serve as a prognostic factor in APAP overdose.

35 PROSPECTIVE STUDY OF MULTIPLE SUPRATHERAPEUTIC ACETAMINOPHEN DOSES IN FEBRILE CHILDREN.

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Background: Repeated doses of acetaminophen given for therapeutic reasons have been reported to cause hepatotoxicity in adults and children. We studied the effects of repeated Acetaminophen (APAP) overdoses administered for therapeutic purposes. Methods: Forty four children, aged 2 months to 10 years, who were referred to the Emergency Department with a fever of >38.5° for more than 48 hours, and received APAP in a dosage >60 mg/kg/day. AST, ALT and APAP blood levels were measured. Results: The mean total daily dose of APAP was 92 ± 26 (63–171) mg/kg. There were no cases of severe liver injury. In 4 children (9.1%) an elevation of AST and ALT was found. One patient had a slight

elevation of AST only. Among children with AST and ALT levels of ≥ 45 IU/L, those who received APAP in a dosage of > 100 mg/kg/day had marginally significantly higher levels of AST (p = 0.05) and ALT (p = 0.07) as compared to those who received 60–100 mg/kg/day. APAP blood levels ranged from 0 to 23 mcg/mL. No correlation was found between the time since the last dose of APAP and the drug level in the serum. Conclusions: This study shows that in ill children receiving repeated supratherapeutic doses of acetaminophen, abnormalities in liver function may occur. Both the medical and the pharmaceutical industries should be responsible for providing adequate, accurate and easily understood information to parents and patients on the judicious use of this common medication.

36 ACETAMINOPHEN (APAP) OVERDOSE AND HEMOLYSIS IN A G6PD DEFICIENT PATIENT.

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Background: Glucose-6-phosphate dehydrogenase (G6DP) deficiency affects an estimated 400 million people worldwide rendering them more susceptible to hemolysis from oxidative stresses. While, therapeutic doses of APAP are considered safe for use by those with G6PD, in vitro and animal studies suggest that APAP can cause oxidative stress effects on erythrocytes. There have been at least two reports of hemolysis in patients with G6PD deficiency following APAP overdose. We describe another case. Case Report: A 22-year-old Iranian male with previously diagnosed G6PD deficiency presented to the emergency department with jaundice 4 days after ingesting an unknown amount of APAP in a suicide attempt. Initial lab data were: APAP 1.65 mg/L, total bili 25 mg/dL, AST 9650 U/L, ALT 11860 U/L, LD 7650 U/L, Alk Phos 138 U/L, Hgb 15.4 g/dL, INR 3.6, Hct 44%, WBC 12,600/uL (Neut 10,900/uL). He was started on IV *N*-acetylcysteine (NAC). The next day (day 5 post ingestion), his AST, ALT, LD, INR had improved but the following had worsened:total bili 26 mg/dL, Hgb 11.4 g/dL, Hct 31.7%, and WBC 16,400/uL (Neut 13,700/uL). Day 6 post ingestion, Hgb and Hct reached a nadir of 5.6 g/dL and 16.6%; WBC 21,700/uL (Neut 17,000/uL). He began to improve and was transferred to inpatient psychiatry on day 10 post ingestion with Hgb 10.8 g/dL, Hct 31.7%, and WBC 10,100/uL (Neut 4,600/uL). Conclusion: Acetaminophen overdose in a G6PD deficient patient may be associated with hemolysis. This may be due to oxidative effects of APAP or to the reactive neutrophilia caused by the liver damage.

37 DELAYED SEIZURES SECONDARY TO TRAMADOL OVERDOSE.

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Background: Tramadol is a centrally acting analgesic possessing both opioid and monoamine uptake inhibition effects. In overdose, seizures have been reported in approximately 8% of patients and are described as brief, self-limiting, usually single events, and occurring within 4 hours of ingestion. We report an overdose with tramadol with repeated seizures, the last occurring 10 hours post ingestion. Case Report: A 61-year-old F took #30 tabs of Ultram^{R} 50 mg (1.5 gm) at 0300. She was found unresponsive at ~0800. Paramedics observed clonic seizure-like activity of less than 1-minute duration with associated brief apnea. They administered naloxone IM and noted no response. On ED arrival at 0835, the patient had 2 clonic seizures with facial cyanosis and emesis. No pill fragments were seen. Pt was given 1 mg lorazepam IV and 750mg of phenytoin IV. PE: HR 74 and sinus, BP 142/86, RR 15, sats 99% on 2 liters, T 98.4. Pt was lethargic, pupils were 3 mm and equal, and bowel sounds positive. Drug screen + for THC, PCP, and benzos. CK 176, MB 1.5, Na 144, K 5.1, Cl 105, HCO3 27.1, BUN 15, Cr 0.7, gluc 133, Ca 8.8, Mg 1.5, acetaminophen <1 mcg/mL, salicylate<5 mg/L, EtOH 81.8 mg/dL, TCA 9.7 ng/mL. ABG on room air: pH 7.38, pCO2 40, pO2 69, sats 93%. At 1250, ~10 hours post ingestion, she had another seizure. The patient was alert and oriented by the next morning and confessed to the suicide attempt. Conclusion: Peak serum levels of tramadol occur within 2 hours with therapeutic dosing and the half-life is reported as 5–6 hours. This patient's low TCA level and lack of anticholinergic symptoms do not suggest a delay in absorption. Seizures from tramadol overdoses may occur longer than the 4 hours reported in the medical literature and patients should be monitored for this symptom for at least 12 hours.

38 POISONING FROM THE APPLICATION OF A SCROTAL TRANSDERMAL FENTANYL PATCH.

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Background: Toxicity has occurred from various administration routes of transdermal fentanyl patches. Topical application of multiple patches may result in significant toxicity. The degree of transdermal absorption of drugs is known to

inversely correlate with stratum corneum thickness. The scrotum is known to be a location with particularly effective absorption. *Case Report:* 55-year-old male was found with altered mental status, pinpoint pupils, and hypoventilation. Because little response occurred after 2 mg of IM naloxone, paramedics performed intubation. ED exam revealed a comatose patient with normal sized pupils who had a Duragesic®-50 patch (fentanyl transdermal system 50 mcg/hr) on his thigh that was immediately removed. Initial ABG revealed a pH of 7.24, and pCO2 of 59 mmHg. Thorough workup failed to identify an etiology of altered mental status. 25 hours after admission, a nurse discovered a second Duragesic®-50 patch adherent to the posterior aspect of the patient's scrotum. Patch was removed and escalating doses of naloxone were given that reversed his altered mental status, and allowed extubation. Patient admitted to intentionally placing the patches for abuse purposes and GC-MS confirmed the presence of fentanyl and excluded the presence of other opioids. Conclusion: This patient suffered severe fentanyl poisoning from the application of two fentanyl transdermal patches. The scrotal site of application is unique as an area of concealment and high absorption.

39 COMPARISON OF CONTINUOUS INFUSION AND SERIAL DOSES OF SODIUM BICARBONATE FOR SERUM ALKALINIZATION.

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Background: Nearly 3 decades after TCK Brown demonstrated the efficacy of sodium bicarbonate ($NaHCO_3$) in cyclic antidepressant poisoning, there is still no consensus about the best quantity or rate of $NaHCO_3$ administration. Objective: To compare the effects of two different $NaHCO_3$ regimens on serum pH, Na^+, K^+, and ionized Ca^{++} in a cross-over control pilot study. Methods: The subject underwent 2 study protocols 1 week apart. On each day, the subject underwent placement of two IV saline locks in separate arms (one IV for venous blood sampling, the other IV for drug administration). After baseline measurements of Na, K, venous pH, and ionized Ca, using a I-Stat portable analyzer with EC6+ cartridges (Abbott Laboratories), the subject received a bolus of 100 mEq of $NaHCO_3$ IV over 2 minutes. All measurements were repeated every 20 min during both 6-hr study periods. In the continuous-infusion (CI) arm, subject received an infusion of 150 mEq of $NaHCO_3$ in 1 L of D5W infused with a controllable pump over 6 hrs. In the serial-dose (SD) arm, the subject received 50 mEq of $NaHCO_3$ IV hourly for 3 hrs after the bolus. The subject had continuous cardiac monitoring during both study periods. Results: The CI method yielded a mean pH change of 0.044 (range 0.014–0.081) above the baseline pH of 7.379 with a calculated area under the curve (AUC) of 15.53 pH-min. The SD method yielded a mean pH change of 0.051 (range 0.004–0.178) above the baseline pH of 7.353 with a calculated AUC of 18.44 pH-min. No adverse decreases in serum K^+ or ionized Ca^{++} occurred. Conclusion: In this pilot study, Serial Dose $NaHCO_3$ appeared to produce a slightly higher mean pH change and a slightly greater AUC than Constant Infusion.

40 THE ECG INTERPRETATION FOR TRICYCLIC ANTIDEPRESSANT OVERDOSE.

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Background: Studies have shown variable interrater agreement in the interpretation of ECGs from patients with TCA OD. We hypothesized that there would be a toxicology (tox) training effect in ECG interpretation. Methods: Volunteers blinded to ECG source and patient data, measured maximum limb lead QRS, presence of terminal R wave in aVR and noted their impression of TCA toxicity manifest by ECGs selected from patients with known TCA toxicity and QRS ≥ 100msec and from an archive of normal ECGs. Results: 47 TCA and 37 normal ECGs were read by 13 readers. Complications were defined as seizures, dysrhythmias, and respiratory failure requiring intubation. Agreement was measured by intraclass correlation coefficient (ICC) (similar to kappa).

		QRS ≥ 120 & TCA Toxicity (95%CI)	QRS ≥ 120 & Complications (95%CI)	R-wave& Complications (95%CI)	ECG Signs& Complications (95%CI)	ICC (95%CI)
Tox Faculty (N = 4)	Sens	39(34–47)	44(34–54)	68(58 -77)	69(59–78)*	ICC0.94
	Spec	100(97–100)	87(82–91)	57(50–63)	74(68–80)*	(0.91–0.96)
Tox Fellows(2)	Sens	52(42–62)	58(43–72)	88(75–95)	94(82–98)	ICC0.88
	Spec	100(94–100)	83(75–89)	39(30–48)	42(33–51)	(0.82–0.92)
EM Faculty(4)	Sens	60(53–67)	66(56–75)	79(69–86)	88(80–93)	ICC0.93
	Spec	99(96–100)	78(74–84)	43(36–49)	53(46–59)	(0.91–0.95)
EM Residents(3)	Sens	51(43–60)	57(47–70)	70(59–80)	68(56–78)	ICC0.84
	Spec	99(94–100)	84(77–89)	46(39–54)	58(51–65)	(0.77–0.89)

Conclusion: In this sample of ECGs and readers, there was no significant toxicology training effect. EM faculty and residents demonstrated similar levels of sensitivity and specificity to Tox faculty and fellows in determining QRS ≥120 msec and recognizing ECG signs of toxicity in patients with complications. Tox faculty were less sensitive than fellows or EM faculty but were more specific than other readers in determining TCA toxicity by ECG. Overall interrater agreement was high.

41 USE OF DOSAGE AS A TRIAGE GUIDELINE FOR UNINTENTIONAL CYCLIC ANTIDEPRESSANT (UCA) INGESTIONS IN CHILDREN.

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Background: Triage guideline for UCA ingestions vary widely, with limited supportive evidence. Method: All records of UCA ingestion reported to 3 certified regional poison centers were evaluated for the years 1998–2000. Inclusion criteria included age < 6 years, patient with a known outcome and known ingested dose by history. Exclusion criteria was polydrug ingestion. Results: 233 cases were evaluated. Age ranged from 7 months to 6 years with a mean and median of 2.4 (±1.2) and 2, respectively. 128 patients (55%) were managed in a HCF. 105 patient were managed at home with observation and telephone follow-up. Mean and median follow-up time on all patients was 9.6 hours (±8.4) and 6 hours, respectively. Symptoms reported in all patients were drowsy (n = 58), tachycardia (n = 4), agitation (n = 2) coma (n = 1), respiratory depression (n = 1), ataxia (n = 1). Medical outcome was reported as; no effect (n = 174, 75%), minor effect (n = 55, 23%), moderate effect (n = 3, 1%) and major effect (n = 1, 1%). Drugs ingested were amitriptyline (n = 124), imipramine (n = 64), nortriptyline (n = 23), doxepin (n = 17) and other (n = 5).

Table 1

Dosage Vs Management Site and Symptoms

	Range in mg/kg	Mean dosage (Standard deviation)	Median dosage
Patients managed in HCF	0.3 to 44 mg/kg	5.2 mg/kg (±7.1)	3.3 mg/kg
Patients managed at home	0.3 to 6.6 mg/kg	1.9 mg/kg (± 1.2)	1.8 mg/kg
Patients *with* symptoms	0.5 to 44 mg/kg	6.3 mg/kg (± 9.3)	3.1 mg/kg
Patients *without* symptoms	0.5 to 26.3 mg/kg	2.9 mg/kg (± 3.1)	2.0 mg/kg
Patients with "Minor effects"	0.5 to 35 mg/kg	5.2 mg/kg (+7.9)	2.0 mg/kg

43 of 55 patients (78%) with minor symptoms reported a dosage of <5mg/kg. All patients with a moderate or major outcome reported a dosage of >5 mg/kg, with a range of 6.6 to 44 mg/kg and a mean of 19.8mg/kg. Conclusion: The majority of UCA ingestions produced limited or no symptamatology. In this series all children with ingestions of < 5 mg/kg developed no or minor effects. Home monitoring may be appropriate in such cases.

42 THE FATAL TOXICITY INDEX (FTI) OF ANTIDEPRESSANT DRUGS IN THE UK BETWEEN 1993 & 1998.

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Background: Large variations between antidepressant drugs in their FTI have been observed, with correlations with clinical toxicity in overdose.(Buckley 1998) Our aim is to examine the fatal toxicity in overdose of newer antidepressants compared to tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). Methods: We obtained data from 1993 to 1998 on the number of fatal poisonings due to a single antidepressant drug and the number of prescriptions in the UK (England, Wales & Scotland) for these drugs to derive a FTI of deaths per million prescriptions with 95% confidence intervals calculated as previously described (Buckley 1998). Results: Overall as a group, newer serotoninergic drugs had much lower FTIs than TCAs and MAOIs (1.5 vs 36 & 22 respectively). However there were large differences within the newer drugs. Venlafaxine (FTI 14 (95%CI 9 to 22)) had much greater fatal toxicity than all other SSRIs and had a higher FTI than that of some of the less toxic TCAs such as clomipramine (11) and nortriptyline (6).

The very low potential for toxicity in overdose (FTI < 1.5) was established for fluoxetine, paroxetine, sertraline, and nefazodone. Within the older antidepressants, our results confirmed previous analyses of the relatively greater toxicity of desipramine (FTI: 201), amoxapine (81), dothiepin (54), amitriptyline (40) and tranylcypromine (45) compared to other drugs in their class. Conclusions: The higher FTI of venlafaxine compared with other newer drugs is confirmation of concerns based on clinical reports that it has greater clinical toxicity. References: Buckley NA, McManus P. Drug Safety 1998;18(5):369–381.

43 PHEOCHROMOCYTOMA UNMASKED BY IMIPRAMINE.

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Background: Pheochromocytoma (PCC) is a rare catecholamine-secreting tumor that presents with paroxysms of severe tachycardia, hypertension and diaphoresis. It can also cause subtle symptoms or remain silent for long periods of time. We present a case in which the classic symptoms of a PCC developed after ingestion of Imipramine. Case Report: A previously well 8-year-old girl was prescribed Imipramine 25 mg/day for nocturnal enuresis. Five hours after taking her first dose, she developed abdominal discomfort, diaphoresis, and tachycardia. She presented 18 hours after ingestion with pallor, marked diaphoresis, pulse rate of 150 beats/minute, blood pressure of 150/120 mm Hg, a grade 2/6 systolic murmur, diminished peripheral pulses and cool clammy skin. An EKG showed sinus tachycardia with a normal QRS. A urinary toxicology screen was negative except for desipramine. Blood pressure was initially controlled with phentolamine and labetalol. Further evaluation included negative cultures, and imaging for renal artery stenosis, normal basic chemistries and thyroid studies. Serum levels of norepinephrine (24,000 pg/mL), Epinephrine (108 pg/mL) and dopamine (268 pg/mL) were markedly elevated as were 24 hour urine levels of norepinephrine (2,549 µg/gm Cr) metanephrine (12,973 µg/gm Cr) and VMA (30 mg/gm Cr). MRI revealed a left-sided periaortic mass consistent with a PCC. All symptoms resolved after a surgery. Catecholamine levels returned to normal post operatively. In retrospect the patient had a history of feeling hot and cold and episodes of paroxysmal diaphoresis for the past 6 months. Conclusion: Patients who develop symptoms consistent with a PCC when treated with a medication that potentiates catecholamines should be suspected of having a latent PCC.

44 FALSE POSITIVE URINE IMMUNOASSAY FOR TRICYCLIC ANTIDEPRESSANTS FOLLOWING QUETIAPINE (SEROQUEL®) OVERDOSE.

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Background: Quetiapine is a dibenzothiazepine antipsychotic whose chemical structure is similar to the tricyclic antidepressants (TCAs). We report the first incidence of a false positive immunoassay for TCAs associated with elevated quetiapine levels. Case Report: A 36-year-old woman ingested 15 grams of quetiapine and developed obtundation, hypotension, tachycardia, and prolonged QT interval. On admission, a urine enzyme immunoassay (Beckman Coulter SYNCHRON LX20) was positive for tricyclic antidepressants. The patient had no history of depression and was not taking antidepressants or cyclobenzaprine. A thorough search of her home revealed no medications other than quetiapine. A comprehensive serum drug screen for 250 analytes revealed no detectable levels of any analytes including TCAs. Gas chromatography/Mass spectrometry of a serum sample revealed no detectable levels of the tricyclic antidepressants, amitriptyline, clomipramine, doxepin, imipramine, trimipramine, desipramine, nortriptyline or protripyline and no detectable cyclobenzaprine. Conclusion: We report a false positive immunoassay for TCAs in the setting of a quetiapine overdose. This false positive may be due to the fact that substantial structural similarities exist between quetiapine and the tricyclic antidepressants. Physicians should be aware of quetiapine as a possible cause for false positive TCA assays.

45 PROPRANOLOL-INDUCED SYMPTOMATIC BRADYCARDIA AFTER INITIATION OF FLUVOXAMINE THERAPY.

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Background: Fluvoxamine is a popular selective serotonin reuptake inhibitor (SSRI) approved for treatment of obsessive compulsive disorder (OCD). Unlike other SSRIs, fluvoxamine is a potent inhibitor of the cytochrome P450 isoenzyme CYP1A2. It has been shown to increase plasma levels of several drugs metabolized by this isoenzyme including propran-

olol. We present a case of a patient on a stable propranolol dose who developed symptomatic bradycardia after the initiation of fluvoxamine therapy. Case Report: A 79-year-old male presented to the Emergency Department with bradycardia and syncope. The patient had been on a stable dose of propranolol for prophylaxis of migraine headaches for several years. He started fluvoxamine therapy and over the next several days developed fatigue and lightheadedness. After a syncopal event, the patient was admitted to the hospital. His heart rate was 38 beats-per-minute. Electrocardiogram showed no ischemic changes, while the cardiac enzymes, electrolytes and thyroid function tests were within normal limits. The fluvoxamine level was 168 ng/mL and propranolol level was 120 ng/mL. Both drugs were discontinued and the patient recovered uneventfully with resolution of bradycardia and symptoms. Conclusion: Fluvoxamine may cause potentially life-threatening interactions with medications that are metabolized by the CYP1A2 isoenzyme.

46 RIGHT BUNDLE BRANCH BLOCK AND DELAYED SEIZURE ASSOCIATED WITH CITALOPRAM AND FLUOXETINE INGESTION.

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Background: Selective serotonin re-uptake inhibitor (SSRI) agents are safer in overdose compared to older antidepressant agents. SSRI toxicity is usually limited to central nervous system depression and gastrointestinal effects. Cardiovascular effects and seizures have been reported but are rare. Serum concentrations are rarely obtained. We report an exposure to citalopram and fluoxetine that resulted in right bundle branch block (RBBB) and delayed seizure activity. Case Report: A 21-year-old male with a history of depression ingested twenty-five fluoxetine 40mg (1000mg), forty-five citalopram 40mg (1800mg) and five acetaminophen 500mg (2500mg). Patient was witnessed having a seizure and was taken to the emergency department (ED) 14 hours after ingestion. He was alert and had some lacerations and bruises. His BP was 107/55 mmHg and HR 137 bpm. Toxicologic analysis was negative for ethanol, aspirin, tricyclic antidepressants, benzodiazepines, cocaine, opiates and acetaminophen. Total creatine kinase was 8500 U/L. An electrocardiogram (EKG) revealed QRS prolongation (136 msec) with RBBB pattern. Drug concentrations were as follows: fluoxetine 181 ng/mL (therapeutic 50–480 ng/mL), norfluoxetine 205 (therapeutic 50–450 ng/mL) and citalopram 840 ng/mL, which is ten times the reported therapeutic concentration of 80 ng/mL. The patient was admitted to telemetry and the EKG abnormalities resolved spontaneously (QRS 100 msec, normal axis) within 36 hours of ingestion. Conclusion: High serum concentrations of citalopram were associated with new onset RBBB and delayed seizure activity that are rarely seen in SSRI overdoses.

47 FATAL OVERDOSE OF CITALOPRAM.

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Background: Citalopram is a selective serotonin reuptake inhibitor (SSRI) and, as such, is considered less toxic than cyclic antidepressants. Limited data exists in the medical literature regarding fatalities from this agent and blood levels associated with death. Case Report: A 38-year-old man with a history of depression and previous suicide attempts was found face down in his bedroom without vital signs. CPR was initiated and he was transported by EMS to the local hospital. The resuscitation attempt was unsuccessful and he was pronounced dead in the emergency department. A complete autopsy was performed which was unremarkable except for an alprazolam level of 190 ng/mL and a citalopram level of 3,402 ng/mL. Conclusion: The concentration of alprazolam in this case excludes it as a significant contributor to death. The therapeutic concentration of citalopram has been reported to be 0.3 ng/mL. A review of the literature revealed little information on citalopram blood levels in fatalities. Our case with a citalopram level of 3,402 ng/mL and a forensic absence of any other cause of death, suggests this agent as the sole basis for this man's death.

48 PAROXETINE AS A SOLE CAUSE OF SEROTONIN SYNDROME: A MASSIVE OVERDOSE WITH DOCUMENTED SERUM PAROXETINE LEVELS.

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Introduction: The serotonin syndrome is only rarely reported in patients taking only a single serotonergic agent. No case reports exist describing serotonin syndrome resulting from paroxetine alone. Case Report: A 57-year-old white male was brought to the emergency department 1 day after ingesting 3,600 mg of paroxetine. He denied the use of

any other drug or herbal products and was not a frequent consumer of alcohol. Upon arrival to the emergency vital signs were: BP188/103 mmHg, HR 114, RR 28, T 36.8°C, O2 sat 96% RA. The pupils were dilated and his face flushed. Prominent findings on physical examination included profuse diaphoresis, shivering. myoclonic jerks and tremors, as well as hyperreflexia and hypertonicity. A tentative diagnosis of serotonin syndrome was made. Symptoms abated slowly over 6 days during which a thorough medical evaluation failed to reveal any other potential causes for the patient's condition, including alcohol withdrawal. Laboratory analysis of serum paroxetine levels performed 27.5 hours and 40 hours post ingestion returned at 1800 ng/mL and 1600 ng/mL respectively (therapeutic range 20–200 ng/mL). Conclusions: Although serum paroxetine levels have not been shown to correlate with efficacy or toxicity of the drug, our patient's serum paroxetine level was nine times the upper end of this range. We have not seen any reports correlating paroxetine serum levels with serotonin syndrome. This is also the first case report of serotonin syndrome resulting from paroxetine alone.

49 VENLAFAXINE (VFX) AND A FALSE POSITIVE ABBOTT AXSYM IMMUNOASSAY FOR PHENCYCLIDINE.

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Case Report: A 13-year-old girl overdosed on 48 X 150 mg VFX (Effexor XR®). She was taking VFX regularly for depression. Her only other medications included topical Benzamycin and pyridoxine 50 mg daily for acne. A brief self-limited seizure occurred at home. When a second seizure occurred in the ED, 2 hours after ingestion, she was given Ativan and intubated. Simultaneously a Foley catheter was placed and urine obtained for a drug of abuse screen (Abbot AxSYM fluorescent polarized immunoassay® for phencyclidine, amphetamines, cannabinoids, cocaine, barbiturates, opiates, benzodiazepines) and comprehensive thin layer chromatographic drug screen (Toxilab®). The Abbot AxSYM assay was positive only for phencyclidine, but GCMS did NOT confirm the presence of phencyclidine. Toxilab® identified only one substance, confirmed by GCMS as VFX. The VFX was present in high concentration in the urine sample, which had to be diluted for the GCMS confirmation. Serum quantitative assay for VFX and metabolites is pending. Dextromethorphan (known to cross react on the Abbott Axsym PCP assay) was not detected on the Toxilab assay. Abbott has not received any reports of VFX causing a false positive assay for phencyclidine. Wyeth-Ayerst, manufacturer of Effexor®, has received unsolicited, unconfirmed, post marketing adverse event reports of VFX causing false positive drug screens for phencyclidine, amphetamine, benzodiazepines, cocaine, marijuana, opiates, propoxyphen, and tricyclic antidepressants. Details of other drugs and underlying illness are not described. The specific assays used are not described. No reports are published. Conclusion: VFX may cause a false positive Abbot AxSYM phencyclidine assay.

50 BUPROPRION EXPOSURES: CLINICAL MANIFESTATIONS AND OUTCOME.

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Background: Buproprion (Wellbutrin and Zyban) is commonly prescribed as an antidepressant and smoking cessation aid. Limited information describing the toxicity of intentional and unintentional buproprion-only exposures exists. Methods: A retrospective review of all buproprion-only exposures reported to the American Association of Poison Control Centers Toxic Exposure Surveillance System (TESS) from 1998–1999 was conducted. Data for all 3 buproprion products, Wellbutrin, Wellbutrin SR, and Zyban were obtained. Data analysis included demographics, reason for exposure, clinical effects, therapy, and medical outcome. Results: From 1998–1999, 7348 buproprion-only exposures were reported to the TESS: 56% were female, 54% were adults, 70% were acute, and 61% were unintentional. The majority of exposures involved Wellbutrin SR (51%); however, Wellbutrin exposures involved a higher percentage of intentional overdoses and serious clinical effects, including 4 deaths. Zyban involved the majority of allergic reactions. Clinical effects related to buproprion were noted in 2,247 (31%) exposures. Only 8% of children < 6 years of age developed clinical effects; 10% were categorized as moderate or major. In contrast, 46% of teens (13–19 years) developed symptoms; 26% were moderate or major. For all ages, neurological effects predominated. Seizures developed in 6% of all buproprion exposures; 87% resulted from intentional exposure. Seizures were extremely rare in children < 6 (0.2%) compared to teens (15%). Conclusions: The majority of buproprion-only exposures result in minimal or no clinical toxicity. Cardiovascular disturbances are extremely uncommon following buproprion overdose; however, a significant number of intentional overdoses result in seizures. Prospective studies are needed to establish the dosage necessary to produce toxic effects as well as time to onset of symptoms.

51 ANOXIC ENCEPHALOPATHY FROM CARBON DIOXIDE THERAPY (CDT) FOR THE TREATMENT OF DEPRESSION.

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Background: In the past, carbon dioxide therapy (CDT) was advocated as a treatment for various psychiatric disorders. The beneficial effects of carbon dioxide (CO_2) were presumed to be mediated through CNS alterations although a more precise mechanism of action was never elucidated. Although four deaths from CDT were reported prior to 1966, significant toxicity has not been reported since. We report a case of a woman having respiratory and cardiac arrest following CDT. Case Report: A 38-year-old female with a history of depression was being treated with CDT by an alternative medicine physician. She had 12 uneventful prior sessions. Therapy typically began with pretreatment of 100 mg of atenolol and two tablets of Fiorecet®. Approximately two hours later, the patient received via facemask varying mixtures of CO_2, nitrous oxide (N_2O), and oxygen (O_2). During the therapy, the physician adjusted the concentrations of the gases initially to attain a 1:1 ratio of CO_2 and O_2 and ultimately a 3:2 ratio of CO_2 and O_2. The CO_2/O_2 was then discontinued and the patient was given 100% O_2. Shortly thereafter, the patient was noted to be in respiratory and subsequently cardiac arrest. EMS was called and they found the patient receiving CPR by her physician. The patient was orotracheally intubated and initial cardiac rhythm determined to be asystole. She was resuscitated with a total of 8 mg of epinephrine and 3 mg of atropine, defibrillated 6 times, and given amiodarone 300 mg and 88 mEq of sodium bicarbonate with return of spontaneous circulation. She was subsequently admitted to the MICU with severe anoxic encephalopathy. Conclusion: Although the extent of CDT as an alternative form of psychotherapy is unknown and likely uncommon, this case highlights severe morbidity from its modern day use.

52 BIOPSY-PROVED ACUTE INTERSTITIAL NEPHRITIS IN CHRONIC LITHIUM NEPHROPATHY IN A LITHIUM OVERDOSE PATIENT.

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Background: Lithium (Li) is a drug of widespread use in psychiatric disorder. Long term use of Li causing interstitial nephritis with unique pathological picture can result in nephrogenic diabetes insipidus (NDI) clinically. However, biopsy-proved acute interstitial nephritis after acute overdose had rarely been described before. We report a biopsy-proved acute interstitial nephritis in chronic interstitial nephropathy associated with Li. Case Report: A 32-year-old female patient with a psychiatric disorder on chronic Li presented with vomiting and gradually impaired consciousness for 2 days after taking an unknown amount of Li carbonate. Physical examination revealed hyperthermia, comatose status and hyperreflexia. Initial laboratory studies included BUN 79 mg/dL, creatinine 8.1 mg/dL, Na^+ 135 mEq/L, K^+ 2.7 mEq/L, and glucose 190mg/dL. Blood Li level was 2.06 mEq/L. Acute hemodialysis (HD) was performed for 6 hours. After HD, blood Li level decreased to 0.5mEq/L; however, her drowsiness persisted for several days, and renal function did not recover completely. Renal biopsy showed acute interstitial nephritis and chronic interstitial nephropathy (tubular dilatation with vacuolar change). Polyuria and hypernatremia, however, worsened after improvement of renal function and consciousness. Hypertonic saline infusion test with ADH injection was done, which was compatible with NDI. Further follow up of the patient disclosed improvement of neurological sequelae and hypernatremia, but with moderate renal function impairment. Conclusion: Acute Li overdose can cause acute interstitial nephritis, which will aggravate previous chronic renal insufficiency in a chronic Li nephropathy patient. Careful monitoring of renal function and avoidance of dehydration is mandatory in chronic Li users.

53 DELAYED ABSORPTION AND PEAK CARDIOTOXICITY FOLLOWING MASSIVE THIORIDAZINE OVERDOSE.

Murray LM, Hackett LP, Ilett KF. *Departments of Surgery and Pharmacology, University of Western Australia, Perth, Western Australia*

Objective: Correlate clinical and EKG data with thioridazine concentration after overdose. Case Reports: 1. A 28-year-old female ingested 20 g of thioridazine. She developed coma, seizures, hypotension, QRS and QT prolongation, VT and torsades de pointes. She was managed with intubation, ventilation, fluids, serum alkalinization, inotropes, and isoproteronol. 2. A 21-year-old female ingested 9.6 g of thioridazine. She developed coma, tachycardia, ileus, QRS and QT prolongation, and VT. She was managed with intubation, ventilation, fluids, and serum alkalinization. Both made complete recoveries following ICU stays of 12 and 8 days respectively. Methods: Thioridazine concentrations were measured by HPLC on all plasma specimens. The primary metabolites, mesoridazine and sulforidazine, were also

measured in case 2. Heart rate, QRS, QT and QTc were determined from all EKGs. Results: In case 1, peak serum thioridazine concentration (11.4 mg/L) occurred at 120 hours post-admission. Maximal QRS occurred at 20 hours and QTc at 133 hours post-admission. In case 2, peak concentrations of thioridazine (4.4 mg/L), mesoridazine (5.9 mg/L) and sulforidazine (2.3 mg/L) were measured at 114, 138 and 143 hours post-ingestion respectively. Maximal QRS occurred at 27 hours and QTc at 121 hours post-ingestion. Conclusion: Absorption of thioridazine and peak clinical toxicity may be substantially delayed following massive overdose. The QTc appears to closely correlate with plasma thioridazine concentration and may provide a useful monitoring tool. The utility of more aggressive early gastrointestinal decontamination in these patients needs to be examined.

54 ANALYSIS OF GUANFACINE EXPOSURES IN CHILDREN < 19 YEARS OLD REPORTED TO POISON CENTERS.

McGrath J, Klein-Schwartz W. *Maryland Poison Center, University of Maryland School of Pharmacy, Baltimore, MD*

Objective: Guanfacine has been increasingly used in children with the diagnosis of Attention Deficit Hyperactivity Disorder (ADHD). The purpose of this study is to examine the clinical effects of guanfacine ingestions in children and adolescents. Methods: Guanfacine exposures reported to the Toxic Exposure Surveillance System from 1993 to 1999 in children and adolescents less than 19 years of age were analyzed. Inclusion criteria were single substance ingestions followed to known outcome. Results: There were 1,747 guanfacine exposures of which 870 cases met the inclusion criteria. There were 478 children <6 years; 304 children 6–12 years and 88 adolescents 13–18 years old. The number of cases increased 4 fold over the 7-year period with the largest increase in children less than 13 years of age. Analysis showed 258 exposures were managed on site (non-health care facility) and 594 were managed in a health care facility. There were no symptoms in 546 children. In symptomatic children, the most common symptoms were drowsiness/lethargy (76.8%), hypotension (25.8%) and bradycardia (30.0%). While the majority of cases were acute (674) there were 182 acute on chronic and 14 chronic. The 6 to 12-year-olds represented the majority of the acute on chronic and chronic exposures with 118 cases. Overall, there were 195 exposures coded as minor effect, 121 moderate and 8 severe. Conclusions: These data demonstrated a trend of increasing numbers of guanfacine exposures annually. Although the majority of children had minimal or no toxicity, serious toxicity can occur.

Platform Session 2 — Saturday, October 6 — 4:00 pm–5:30 pm
Abstracts #55–#60

55 EFFECTS OF MONENSIN ON CONTRACTILE RESPONSE IN TCA CARDIOTOXICITY.

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

Background: TCA toxicity inhibits inward sodium movement during cardiac depolarization, which may contribute to decreased contractile response. Monensin is a sodium ionophore which increases intracellular sodium content. Objective: To evaluate if increasing intracellular sodium content ameliorates TCA-induced cardiac dysfunction. Methods: Isolated rat hearts were perfused with a physiologic buffer (KHB) at a coronary flow of 10mL/min and paced at 300bpm. Experiments included 15 min intervals of Basal, Treatment, and Recovery. Left ventricular (LV) pressures were measured with a balloon-tipped catheter placed in the LV via the mitral valve. LV generated pressure (LVGP) was used as an index of cardiac function and was calculated by subtracting LV end diastolic pressure (LVEDP) from LV peak systolic pressure. Treatments included monension (MON, at 1uM, [n = 4]), imipramine (IMIP, at 1000ng/mL [n = 4] and 2000ng/mL [n = 4]) or IMIP + MON (IMIP at 1000ng/mL [n = 4] and 2000ng/mL [n = 4]). Values represent mean ±SE. Data were analyzed by ANOVA and $p < 0.05$ determined significance. Results: 1) IMIP progressively decreased LVGP over time and in a dose dependent fashion. At 1000ng/mL and 2000ng/mL, these values were 86 ± 1.04% and 79 ± 5.12% of basal, respectively. 2) MON transiently increased LVGP to a peak value of 127 ± 4.2% of basal. 3) IMIP + MON transiently increased LVGP to peak values of 111 ± 5.7% and 99 ± .4% of basal for IMIP at 1000ng/mL and 2000ng/mL, respectively. At the end of treatment, LVGP were similar to IMIP treatments alone.

Conclusions: 1) Increased intracellular sodium by monensin causes an unsustained increase in cardiac contractile performance during TCA toxic exposure. 2) Further investigations are necessary to define the significance of intracellular sodium content in TCA-induced cardiac dysfunction.

56 EFFECTS OF TCA TOXICITY ON FORCE-FREQUENCY RELATIONSHIP IN THE ISOLATED RAT HEART.

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

Background: A postextrasystolic potentiation study in the isolated perfused rat heart suggested inadequate sarcoplasmic reticulum (SR) re-uptake of calcium may contribute to TCA-induced cardiac dysfunction. Objective: To evaluate if cytosolic calcium content during diastole increases during TCA-induced cardiac dysfunction. Methods: A force-frequency model was used to evaluate the cytosolic calcium content during diastole. Isolated rat hearts were perfused with physiologic buffer (KHB) at a coronary flow of 10mL/min for 40mins and paced at 5Hz (300bpm). The protocol included basal, treatment, and force-frequency sequence. The force-frequency sequence was conducted by increasing the pacing rate in a stepwise manner in 0.5Hz increments from 5Hz to a maximal frequency of 8Hz. Measurements included left ventricular (LV) peak systolic pressure (PSP), LV end diastolic pressure (EDP), LV generated pressure (GP), and heart rate. GP was used as an index of cardiac function. EDP was used as an index of diastolic cytosolic calcium content. Treatments lasted 10mins and included KHB (control, N = 4), or imipramine (IMIP, 3000ng/mL, N = 4). Drug exposure was continued during the force-frequency sequence. Values represent mean ±SE. Data were analyzed by ANOVA and $p < 0.05$ determined significance. Results: 1) IMIP decreased GP to 67 ± 1% of basal. 2. During IMIP treatment, force-frequency increased EDP to 229 ± 18% (vs 258 ± 39% for Control, $p > 0.05$) and decreased PSP to 62 ± 1% (vs. 71 ± 2% for Control) of basal. Conclusion: 1) Imipramine-induced cardiac dysfunction is not associated with increasing EDP in a force-frequency model. This suggests the SR re-uptake mechanism for calcium to be intact. 2) Further studies are needed to define the mechanism responsible for decreased systolic performance in TCA-induced cardiac dysfunction.

57 A 31P NMR STUDY OF TCA-INDUCED CARDIOTOXICITY.

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

Background: Phosphorus 31 nuclear magnetic resonance (31P NMR) can be used to monitor the direct effect of drugs on intracellular pH (pHi) and phosphorus-containing metabolites of the heart. Objective: To evaluate if an intracellular acidosis or a decrease in high energy phosphate metabolites are associated with TCA-induced cardiac dysfunction. Methods: Isolated rat hearts were perfused with physiologic buffer (KHB) at a coronary flow of 10mL/min for ~60mins and paced at 300bpm. Temperature was maintained at 37C. The protocol included basal, treatment, and recovery. Left ventricular (LV) pressures were measured with a balloon-tipped catheter placed in the LV via the mitral valve. LV generated pressure (LVGP) was used as an index of cardiac function and was calculated by subtracting LV end diastolic pressure (LVEDP) from LV peak systolic pressure. 31P NMR was used to continuously monitor intracellular concentrations of inorganic phosphate ([Pi]i), phosphocreatine ([PCr]i), ATP ([ATP]i), and pHi. Treatments were for 30mins and included imipramine (IMIP, at 600ng/mL, n = 4) or KHB (Control, n = 4). Values represent mean ±SE. Data were analyzed by ANOVA and $p < 0.05$ determined significance. Results: 1) IMIP decreased LVGP to 45 ± 5.7% of basal (vs 87 ± 8.5% for Control). 2) pHi at basal for IMIP treatment was 7.08 ± 0.03 (vs 7.08 ± 0.02 for Control) and at the end of treatment was 7.09 ± 0.02 (vs 7.00 ± 0.06 for Control). 3) [ATP]i remained essentially unchanged from basal during IMIP and Control treatments. 4) [Pi]i decreased to 77% of basal with IMIP treatment (vs 98% for Control). [PCr]i increased to 114% of basal with IMIP treatment (vs 93% for Control). Conclusion: 1) TCA-induced cardiac dysfunction is not associated with an intracellular acidosis or a decrease in high energy phosphate metabolites.

58 INTERLEUKIN-18 (IL-18) IS INVOLVED IN ACETAMINOPHEN (APAP) INDUCED HEPATIC INJURY IN A FAS/FAS LIGAND INDEPENDENT MECHANISM.

Waksman JC[1,3], Fantuzzi G[2], Bogdan GB[1], Dart RC[1,3], Dinarello C[2]. [1]*Rocky Mountain Poison and Drug Center–Denver Health,* [2]*Division of Infectious Diseases,* [3]*University of Colorado Health Sciences Center, Denver, CO*

Introduction: IL-18 is a novel pro-inflammatory cytokine that participates in hepatocyte apoptosis in mice after treatment with a variety of hepatotoxins. The normal apoptotic pathway commences with Fas/Fas Ligand interactions. The role of IL-18 in APAP-induced hepatic injury has not been previously reported. Objective: To explore the role of IL-18 in

APAP-induced hepatic injury and determine if this mechanism is Fas/Fas Ligand mediated. Methods: C57BL/6 female mice (N = 10, 20–25g) were injected i.p. with 700 mg/kg APAP. Control mice (N = 10) received an equal volume of saline by the same route. Blood and liver were collected for ALT and IL-18 determinations at 1, 2, 4, 8, 24 hours following APAP injection. A second group (N = 25) was injected i.p. with neutralizing antibodies to IL-18 one hour prior to APAP injection. ALT and IL-18 levels were measured at 8 hours following APAP injection. Control mice (N = 25) received control protein followed one hour by APAP. Ten Fas deficient mice (Lpr/Lpr) were also injected i.p. with APAP to explore Fas/Fas Ligand participation. Results: IL-18 was detected in serum and liver one hour following APAP injection and peaked at 8 hours. Anti-IL18 antibodies significantly reduced hepatic injury: mean serum ALT (± SD) was 523 ± 428 IU/L compared to 1653 ± 1043 IU/L for controls ($p < 0.0001$). Mean serum ALT for Lpr/Lpr mice was not different from wild type mice. Conclusion: In this model liver IL-18 is involved in APAP-induced hepatotoxicity, but IL-18 antibodies do not completely prevent hepatic injury. The mechanism by which IL-18 is involved in APAP hepatotoxicity seems to be Fas independent.

59 NEUTRALIZATION OF UNITED STATES *LATRODECTUS MACTANS* AND *L. HESPERUS* VENOMS WITH A MEXICAN *L. MACTANS* ANTIVENOM.

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

Background: It has been suggested that an antivenom used for one species of *Latrodectus* (widow spiders) would be effective in treating envenomation by other *Latrodectus* species. Objective: To determine the efficacy of a *Latrodectus mactans* (black widow) antivenom, manufactured in Mexico, in neutralizing the lethality of *L. hesperus* (Western black widow) and *L. mactans* (Southern black widow) venom collected in the United States. Methods: A blinded, randomized, comparative, placebo-controlled trial in the mouse. Power analysis indicated that 7 animals per group conferred a 95% power to detect an 80% difference between the experimental groups with a two-tailed alpha of 0.05. The following were pre-mixed and incubated at 25°C for 1 hour prior to i.p. injection: 1) *L. hesperus* venom (at 5x LD_{50}) + antivenom (calculated to neutralize 120 x LD_{50}); 2) *L. hesperus* venom + protein control (non-specific equine IgG); 3) *L. mactans* venom (at 5x LD_{50}) + antivenom; 4) *L. mactans* venom + protein control; 5) 0.9% saline + protein control; and 6) saline + antivenom. Survival curves were compared with a Log-rank test. Results: All mice in the venom + antivenom and non-venom control groups survived (mean survival time 1440 ± 0 minutes) and were different from the *L. hesperus* + protein control group (7/7 died; mean survival time 689.3 ± 212.7 minutes) and *L. mactans* venom + protein control group (6/7 died; mean survival time 265.4 ± 518.4 minutes) ($p < 0.001$). Conclusion: The imported antivenom effectively neutralized the lethality of the *Latrodectus* venoms in a murine model. While our study is limited by the premixing of antigen and antibody, it concurs with the hypothesis that one antivenom could be effective worldwide.

60 ACRYLONITRILE INHIBITS CALCINEURIN PHOSPHATASE.

Stemmer PM, Sommer D, Swanson SA, Angle CR. *University of Nebraska Medical Center, Department of Pediatrics, Omaha, NE*

Background: Acrylonitrile is proposed to act as a non-genotoxic carcinogen by oxidative mechanisms and has been shown to have neurotoxic effects by unknown mechanisms. Calcineurin is a Serine/Threonine phosphatase prominent in brain. It is the target of the immunosuppressant drugs cyclosporin-A and tacrolimus and is inhibited by oxidants including superoxide, NO and H2O2. In order to test the hypothesis that acrylonitrile neurotoxicity is correlated with calcineurin inhibition the potency and efficacy of acrylonitrile as an inhibitor of calcineurin was examined in the SKN-SH neuroblastoma cell line. Methods: SKN-SH cells were grown to approximately 75% of confluence in 12 well plates. The cells were treated with acrylonitrile at concentrations between 10 μM and 1 mM for either 4 or 24 hours and were harvested in the presence of superoxide dismutase and catalase to prevent further oxidative inactivation or in the presence of those enzymes plus ascorbate to reverse oxidative inactivation. Phosphatase activity in cell lysates was measured using a 32-P labeled phosphopeptide substrate. Results: Acrylonitrile caused a dose-dependent inhibition of calcineurin under all conditions studied which decreased activity by approximately 65%. The inhibition curve was shifted to the right with increasing time of exposure and by ascorbate at the 24 hr time point but was shifted to the left by ascorbate at the 4 hr time point. Conclusions: Acrylonitrile is a potent inhibitor of calcineurin phosphatase activity. Increased potency at the 4 hr time is likely to be due to ascorbate free radical generation in the presence of acrylonitrile. Inhibition is reversed with time and by ascorbate at 24 hr suggesting the acrylonitrile is acting through an oxidative mechanism.

Poster Session II **Sunday, October 7** **10:00 am–4:00 pm**
Abstracts #61–#110 **Authors with Posters** **10:00 am–11:30 am**

61 DELAYED ABSORPTION OF VALPROIC ACID RESULTING IN COMA.

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Introduction: The length of observation period following acute asymptomatic valproate ingestions is unknown because of it's potential to induce coma and delayed peak levels. Case Report: A 24-year-old man presented to an emergency department (ED) 3 hours after ingesting unknown amounts of valproic acid, paroxetine, clonazepam and ethanol. Upon presentation, the patient was awake and alert. He stated this was an acute overdose and these were his medications although he was noncompliant. His vital signs (blood pressure 138/70 mm/Hg) and physical examination were normal. A valproic acid level obtained four hours post-ingestion was 70 mg/L [Therapeutic range: 50–100 mg/L]. Ethanol level was 133 mg/dL. Urine drugs of abuse (Enzyme multiplied immunoassay technique) screen was negative for benziodiazepines, cocaine, opioids, tricyclic antidepressants, marijuana and phencyclidine. An electrocardiogram was normal. Salicylate and acetaminophen levels were zero. Following six hours of ED observation he remained clinically well and awaited psychiatric consultation. Two hours later he was lethargic and was arousable only to painful stimuli. A repeat valproic acid level was 574 mg/L, serum ammonia level 176 mEq/dL [normal range 0–33] and liver function tests were normal. He was admitted to the intensive care unit (ICU) and received supportive care and L-Carnitine 25mg/kg IV q 6 hours. At 24 hours post ingestion, the patients' systolic blood pressure (SBP) was 70mm/Hg and he required fluid boluses and dopamine (6mcg/kg/min) to maintain SBP $\geq$ 90–100mmhg for two hours. At 32 hours post-ingestion a valproic acid level was 62mg/mL and ammonia level was 44 mEq/mL. He was transferred to inpatient psychiatry 46 hours post-ingestion asymptomatic and normal vital signs. Conclusions: Valproate ingestion may result in delayed toxicity despite therapeutic levels and a normal physical examination six hours post-ingestion.

62 VALPROATE-INDUCED HYPERAMMONEMIA: PRELIMINARY EVALUATION OF AMMONIA ELIMINATION WITH CARNITINE ADMINISTRATION.

Sztajnkrycer MD, Scaglione JM, Bond GR. *DPIC, Cincinnati, OH*

Objective: To determine the effects of L-Carnitine administration on valproate (VPA) and ammonia (NH_3) elimination. Methods: A retrospective poison center chart review of VPA ingestions. Results: A total of 6 patients met the criteria of elevated VPA levels, and more than a single NH_3 level. 4 patients (1,2,3,6) received L-Carnitine supplementation. Patient 1 also received hemodialysis and multi-dose activated charcoal. The mean peak VPA level was 858.0 (range 166 – 1688 mg/mL) and the mean peak NH_3 level was 253.3 (range 48-714 µg/dL). Patients with elevated NH_3 concentrations demonstrated a biphasic (1,2) or triphasic (6) NH_3 elimination. For pt 1, phase 1 β elimination demonstrated $t^1/_2$ 2.9 h, and phase 2 β elimination $t^1/_2$ 15.5 h. For pt 6, phase 1 β elimination demonstrated $t^1/_2$ 10 h, and phase 2 β elimination $t^1/_2$ 7.0 h. Pt 2 demonstrated a phase 1 β elimination $t^1/_2$ of 5.5 h; phase 2 β elimination $t^1/_2$ could not be determined secondary to lack of terminal data points but was biphasic in appearance. Analysis of published controls, neither of whom received L-Carnitine, had monophasic elimination half-lives ranging from 11 – 90 h. Conclusions: In patients with significantly elevated NH_3 levels, L-Carnitine appears to shorten NH_3 elimination. A multi-phasic process appears to occur. This suggests that dosage strategies for L-Carnitine may need to be reconsidered, with higher subsequent doses than our current recommendation of 100 mg/kg load followed by 250 mg Q 8 h.

63 HIGH-EFFICIENCY HEMODIALYSIS IS EFFECTIVE IN REMOVING VALPROIC ACID.

Sharma AN, Ilamathi E, Nelson LS, Hoffman RS, Howland MA. *New York City Poison Center, St. Catherine of Siena Medical Center and St. John's School of Pharmacy, New York, NY*

Background: Until recently, hemodialysis (HD) was not considered an effective means of extracorporeal removal of valproic acid (VPA). The protein saturation kinetics of VPA enable extracorporeal removal using current HD filters. Although two previous case reports describe successful treatment of VPA toxicity with high-flux HD, this is the first reported case of VPA toxicity effectively treated with high-efficiency HD. Valporate kinetics are also discussed. Case Report: A 19-year-old male taking VPA for an affective disorder presented to the hospital after ingesting approximately

84 Depakote tablets (total of ~ 21 grams). He was lethargic with an initial serum VPA concentration of 82.2 mg/L. The remainder of his initial evaluation was WNL. The patient received MDAC and WBI, but despite these measures his VPA concentration continued to rise and peaked at approximately 850 mg/L. Because of worsening mental status high-efficiency HD was performed at the bedside for 7 hours. Serial arterial, venous and dialysate VPA concentrations were obtained. Within six hours of HD therapy the patient awoke and his VPA concentration dropped from 849 to 240 mg/L. The extraction ratio ranged from 21.2 to 56.3%, HD clearance was 43.6 mL/min and 9.3 grams of VPA were removed over six hours of HD. VPA $T_{1/2}$ was 3.3h during HD and 7.44h after HD. Conclusion: High-efficiency HD of VPA achieved 69% of the extraction ratio and approximately 36% of the plasma clearance rate of high-flux HD and removed approximately 50% of VPA ingested. High-efficiency HD, which can be performed at the bedside and requires lower blood flow rates, is effective in removing VPA and should be considered when high-flux HD is unobtainable.

64 SERUM VALPROATE IMMUNOASSAY VARIABILITY FOLLOWING OVERDOSE.

Benton DC, McKay CA, Wu AHB. *Hartford Hospital, University of Connecticut School of Medicine, Hartford, CT*

Background: Valproic acid (VPA) overdoses are characterized by CNS and metabolic abnormalities, as well as frequently-reported delays to peak VPA concentration of many hours, as measured by immunoassay. This delay to peak has been attributed to delayed absorption, as well as possible assay cross-reactivity with VPA metabolites. Manufacturers report low cross-reactivity; however, these reports assume VPA measurements in the therapeutic range. We compare the performance of various assays after VPA overdose. Methods: Three samples collected over twelve hours from each of two patients with VPA overdose were assayed in duplicate by the Abbot, CEDIA, Roche Diagnostic Systems Inc., and Syva Co. immunoassays. Peak values (by Roche) were 561 and 332 mg/L. Results: Compared to CEDIA, Abbot read 29.8 mg/L lower, Roche read 25.8 mg/L lower, and Syva read 21.2 mg/L lower (standard of deviation 3.5 mg/L). Conclusion: The CEDIA assay produced consistently higher readings for VPA compared with the other three assays. It is unclear whether this represents problems with metabolites, although the results do not follow the relative cross-reactivity reported by the manufacturers. The assays' relation to the "true" VPA concentration is also uncertain. These questions are being addressed by a GLC procedure. Unfortunately, inadequate sample volume and infrequency of significant VPA overdoses have delayed this evaluation. Inter- and intra-assay measurement differences may become clinically significant at very high VPA concentrations following overdose.

65 DEATH ASSOCIATED WITH MASSIVE VALPROIC ACID INGESTION.

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Introduction: Most deaths reported from valproic acid (VPA) are associated with chronic use and hepatic failure. We report two deaths associated with acute VPA overdoses. Case Reports: Patient A, a 24-year-old woman, arrived at the ED 3 hours after ingesting 350 gm of Depakote® in a suicide attempt. She received gastric lavage and activated charcoal and was drowsy for 6 hours. Her initial VPA level was 407 mg/L (therapeutic: 50-150 mg/L). Eleven hours post-ingestion she suddenly became unresponsive requiring mechanical ventilation for respiratory support and dopamine for hypotension. Hypotension required maximal doses of dopamine, norepinephrine and epinephrine over the next 24 hours. She developed persistent metabolic acidosis and renal insufficiency, but no elevation in hepatic transaminases. Her VPA concentration peaked 14 hours post-ingestion at 2,204 mg/L. Hemodialysis was recommended but not done. Her pupils became fixed at 5 cm and she was pronounced dead 48 hours post-ingestion. Patient B, a 45-year-old man, presented to the ED 1 hour after ingesting 207.5 gm of Depakote® in a suicide attempt. He received gastric lavage and activated charcoal. His initial VPA concentration was 108 mg/L. He remained asymptomatic until approximately 6 hours post-ingestion at which time he suddenly became unresponsive. He had suffered respiratory arrest and developed hypotension and supraventricular tachycardia. Treatment included mechanical ventilation, dopamine, norepinephrine, diltiazem and amiodarone. His VPA concentration peaked 13 hours post-ingestion at 1,609 mg/L. He developed persistent metabolic acidosis and renal failure, without evidence of hepatic injury. Hemodialysis was held due to cardiac instability. He expired at 79 hours post-ingestion with multiorgan failure. Conclusion: We present two deaths associated with massive valproic acid ingestion. Clinical course exhibited a delayed onset of rapid decrease in level of consciousness, refractory hypotension, metabolic acidosis and renal insufficiency without hepatotoxicity.

66 CHOREATIC DYSKINESIA FOLLOWING LAMOTRIGINE OVERDOSE.

Miller M, Patel M, Tai W, Olson K. *California Poison Control-San Francisco Division, University of California at San Francisco, San Francisco, CA.*

Background: Lamotrigine is a relatively new antiepileptic drug that is finding increased use in other areas to include adjunctive therapy for depression and bipolar disorder. While adverse drug reactions such as rash and toxicity in overdose manifested by ataxia and diplopia have been described, we present a case of severe choreatic dyskinesia following a lamotrigine overdose. Case Report: A 23-year-old male with a history of bipolar disorder walked into the emergency department (ED) stating he had just taken all of his lamotrigine pills 30–60 minutes prior. He was noted to be alert and oriented with BP of 160/93, HR of 115, RR of 18, T of 98.4 F. He received a single dose of 50 grams of activated charcoal. During the subsequent hour, the patient became agitated and developed rapid, intermittent, choreatic movements of his trunk and extremities without any seizure activity. These movements continued every 30 to 45 seconds during the following 2 hours, but were reduced in frequency as well as severity by the administration of 2 mg of lorazepam IV. The movements subsided during the first 12 hours of admission. A lamotrigine level drawn 24 hours after admission to the ED was 62.4 μg/mL. While levels are not routinely used for monitoring this agent, it has been suggested 1–5μg/mL would be the probable therapeutic range. Conclusion: This is a case of a severe dyskinesia associated with an overdose of lamotrigine. With expanding indications for this drug, novel findings in overdose or therapeutic use are likely to continue to be reported. To our knowledge this is the first report of choreatic dyskinesia with lamotrigine overdose.

67 CONTROLLED-RELEASE CARBAMAZEPINE (CBZ) OVERDOSE RESULTING IN DELAYED PEAK SERUM CONCENTRATIONS AND TREATMENT WITH CHARCOAL HEMOPERFUSION.

Graudins A, Dowsett RP, Peden G. *Departments of Emergency Medicine and Clinical Toxicology, Westmead Hospital, Sydney, Australia*

Background: Peak serum levels following overdose with immediate-release (IR) formulations of CBZ have been reported to occur up to 2 days post-ingestion. We report a case of initially unrecognized poisoning with CBZ controlled-release (CR) resulting in peak levels 96 hours post-ingestion. Case Report: A 31-year-old female presented 12 hours following a suspected polypharmacy overdose. She was hemodynamically stable with GCS 3 and endotracheally intubated and ventilated. Two doses of activated charcoal (AC) were administered over the next 12 hours. The patient's neurologic status improved but she remained drowsy and intubated. Results of qualitative urine drug screen became available 24 hours post-admission revealing benzodiazepines and CBZ. Serum [CBZ] at this time was 66 μmol/L (therapeutic 17–42). A history of epilepsy and CBZ-CR therapy was also elicited from the patient's mother. Whole-bowel irrigation was attempted but poorly tolerated due to development of an ileus. Serum [CBZ] continued to rise and by day 4 post-ingestion peaked at 196 μmol/L. Coma, generalized seizure activity and hypotension were present. Charcoal hemoperfusion (HP) was instituted and serum [CBZ] fell from 176 μmol/L to 106 μmol/L after 1 hour of HP. The patient was rousable to voice and could obey commands. Serum [CBZ] continued to fall over the next 48 hours. Ingestion of 200 x 300 mg TEGRETOL-CR® was confirmed by the patient. Conclusion: CBZ-CR poisoning may result in significantly delayed peak serum [CBZ] and toxicity, particularly when GIT decontamination is delayed or inadequate.

68 INGESTION OF THE CALCIUM CHANNEL ANTAGONIST FLUNARIZINE WITHOUT HEMODYNAMIC CONSEQUENCE.

Sztajnkrycer MD[1], Bond GR[1], Uges DRA[2]. *[1]DPIC, Cincinnati, OH; [2]University Hospital Groningen, The Netherlands*

Background: Flunarizine (FNZ), a T-type calcium channel antagonist, is used in Europe and India for prophylaxis and management of migraine headaches. Case Report: A 25-year-old Indian woman on FNZ 10 mg daily for migraine prophylaxis, presented two hours after supra-therapeutic ingestion of FNZ 140 mg in an attempt to relieve a severe headache. Other than headache, she had no complaints. Upon arrival, BP 106/54, HR 80, RR 14. Physical and laboratory examination were unremarkable. Initial ECG demonstrated normal sinus rhythm and normal intervals. The patient was admitted to the ICU for hemodynamic monitoring. With the exception of asymptomatic sinus bradycardia (HR 58) while asleep which resolved upon wakening, the patient remained hemodynamically stable. The patient was discharged to home without incident. Telephone follow-up revealed no sequelae. A serum FNZ level on blood obtained 24 hours post ingestion was 191 ng/mL (usual therapeutic range 50 – 100 ng/mL). Conclusion: Unlike the majority of calcium

channel antagonists approved for use in the United States, FNZ affects T-type calcium channels. No previous overdose information is available in the English language literature. A serum level obtained 24 hours after acute ingestion was two to four times the expected therapeutic level. While management strategies should not be based on isolated case reports, it appears that FNZ is relatively safe in mild to moderate overdose.

69 VERAPAMIL-INDUCED HYPOTENSION REVERSED WITH DEXTROSE-INSULIN.

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Objective: Severe hypotension due to verapamil intoxication may become refractory to intravenous calcium, fluid, and vasopressor infusions. Dextrose/Insulin infusion may enhance ionotrope in the setting of Ca Channel Blocker toxicity. We report a case of toxicity following massive sustained-release verapamil ingestion in an adolescent. Hypotension and acidosis improved following initiation of a dextrose-insulin infusion. Case Report: A 13-year-old female with post-traumatic stress disorder ingested 25–30 120mg sustained-release verapamil tablets (3–3.6 gm). At 1-hour post ingestion she was alert, HR 120, BP 110/65. Gastric lavage and activated charcoal with sorbitol were completed. Within 30 minutes she became obtunded. Cardiac status deteriorated to A-V dissociation, bradycardia (HR 54), and hypotension (BP 68/32). Following intubation, IV resuscitation included 1 liter of NS, 54 mEq of calcium chloride, 15mgs of glucagon and the addition of norepi- and epinephrine infusions. BP was then 103/30 and HR 105. During air transport BP dropped to 75/50. On ICU admission pH was 7.25, blood glucose was 362, and she had marked deficit in clinical perfusion. BP increased to 100/70 along with an improvement in clinical perfusion following IV bolus of regular insulin as 0.1 U/kg and dextrose 0.5 g/kg.. An hourly infusion was maintained. Within 1.5 hours of the onset of the hourly infusion, norepi was discontinued as BP was held at 110/70, and pH improved to 7.37. Insulin/dextrose infusion was continued for 26 hours. An echocardiogram at 3 days revealed normal function. She had full neurologic recovery. Conclusion: Insulin/dextrose infusion improved hypotension and restored clinical perfusion following partial resuscitation with calcium and vasopressors in a severely intoxicated, verapamil overdosed adolescent. Early use of insulin/dextrose may be indicated following hypotension and acidosis due to verapamil.

70 MASSIVE PROPAFENONE OVERDOSE.

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Background: Propafenone, a Class 1C antiarrhythmic, antagonizes Na channels and β-adrenoceptors. Acute propafenone overdose has been rarely reported. We describe acute poisoning in a previously healthy 17-year-old girl. Case Report: A 17-year-old girl ingested 6.75 g propafenone. Paramedics found her 2.2 hrs later to be seizing, a pulse of 66, and an unobtainable BP. Glucose was 92 mg/dL. Seizures stopped spontaneously. In the ED she was in shock and underwent intubation. Charcoal was given per NG. About 3 hr post ingestion hypotension, a wide QRS complex, and bradycardia led to asystole. She was resuscitated with isoproterenol, dopamine, atropine, $NaHCO_3$, glucagon, norepinephrine and then captured with transient external pacing with a resultant BP in 70s and HR in 60s with wide QRS – no P waves were visible. After ICU admission repeated seizures treated with lorazepam and phenobarbital. Bradycardia and hypotension controlled with isoproterenol, epinephrine, norepinephrine, dopamine, and $NaHCO_3$ infusions; QRS was >.2 sec in ventricular or junctional rhythm. $NaHCO_3$ boluses were given for acute additional QRS widening and HR slowing on several occasions. 8 hrs post ingestion she reverted to sinus rhythm with 1st degree heart block and a QRS of .15 s. Echocardiography at this time while on pressors and $NaHCO_3$ showed good contractility and an estimated cardiac output of 6.1 L/min. Vital signs and the ECG normalized over the next 48 hr. Aspiration pneumonia and rhabdomyolysis resolved with supportive care, and she was discharged 5 days after admission. Conclusion: This patient presented with findings typical of massive Na channel blockade–seizures, hypotension, wide QRS, and bradyarrhythmias. The β-blocking action of propafenone may have compounded negative inotropic and chronotropic effects.

71 QUINAPRIL OVERDOSE-INDUCED RENAL FAILURE.

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Background: Angiotensin converting enzyme (ACE) inhibitor toxicity is infrequently reported and seldom produces significant clinical effects. To date, renal insufficiency associated with ACE inhibitor overdose has only been reported concurrent with systemic hypotension. Both effects are felt to be due to vasodilation from low angiotensin II levels. We report a case of renal failure without hypotension presenting two days after an intentional quinapril overdose. Case Report: A 24-year-old man presented to the Emergency Department complaining of bilateral flank pain and decreased

urine output. The patient had ingested between 30 to 40 of his mother's 5 mg quinapril tablets two days earlier in an attempt to "get high". He vomited twice the following day, but denied any orthostatic symptoms and otherwise remained asymptomatic for at least 24 hours. The patient presented complaining of intermittent, non-radiating, bilateral flank pain and decreased urine output described as a single small void in the prior 24-hour period. Vital signs were stable, including a blood pressure of 158/73 mmHg. Initial serum BUN and creatinine levels were 59 and 7.8 mg/dL respectively; potassium was 4.1 mEq/L. Renal ultrasonography revealed bilateral diffuse increased echotexture, without evidence of obstruction or stones. Work-up revealed no other evident cause of acute renal failure. The BUN and creatinine peaked at 74 and 8.3 mg/dL the next day, and decreased to 30 and 1.9 mg/dL at discharge on hospital day five. The patient recovered without hemodialysis. Conclusion: Overdoses with quinapril or other ACE inhibitors may result in renal failure. This effect is presumably due to reduced glomerular filtration pressure from efferent arteriolar dilation, and may occur in the absence of clinically evident systemic hypotension.

72 SEVERE HYPOTHERMIA CAUSED BY CLONIDINE.

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Background: Clonidine is a centrally acting alpha-adrenergic agonist that effectively lowers blood pressure and pulse. In overdose its toxic effects include central nervous system (CNS) depression, hypotension, bradycardia, hypoventilation and apnea. Victims of severe clonidine overdose may have, in addition to profound CNS depression, marked hypothermia. Cases of severe hypothermia, however, have not been published. We report of an unusual case of clonidine overdose resulting in severe bradycardia, respiratory depression and severe hypothermia, with the resulting misdiagnosis of death. Case Report: A 39-year-old FM arrived to a local emergency department unresponsive and apneic from a local funeral home after being declared a 'sudden death' from a failed suicidal attempt, by pre-hospital providers. When she was initially found, available history was that she had a mental health disorder for which she took multiple medications. Her medications, which had been refilled ten days prior, included: Amitriptyline (25mg), Serzone (100mg), Haloperidol (1mg), Quinne Sulfate (260mg), Seroquel (100mg) and Clonidine (0.02mg). The patient was located approximately 6 hours post ingestion when it was determined that she had expired at home, by qualified personnel. Twenty of the thirty of amitriptyline and clonidine were unaccounted for. She was removed to a local funeral home after the crime scene investigation was completed. However, she regained spontaneous movements and vocalizations, leading to her discovery by funeral home personnel. Initial vital signs were BP-110/70 PR-62 NSR, RR-intubated, GCS-3, skin cold and pallor. In the emergency department she was noted to be severely hypothermic with a core temperature of 84R. After warming and resuscitative measures she had a normal hospital stay and was medically cleared on day three, without any neurological defects. Conclusion: We report of an unusual case of clonidine overdose resulting in the mistaken diagnosis of death.

73 UNINTENTIONAL INGESTION OF BRIMONIDINE OPHTHALMIC DROPS NECESSITATING INTUBATION.

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Introduction: Brimonidine is a relatively new, highly potent alpha-adrenergic agonist, belonging to the same class of drugs as clonidine. Its systemic toxicity is mediated by both central and peripheral adrenergic agonism. Significant systemic absorption has been previously reported with ocular instillation. We report a case of accidental paediatric *ingestion* of this drug. Case Report: A previously healthy 28-month-old boy presented to the emergency department with "sleepiness". A 5 mL bottle of brimonidine tartrate 0.2% was found on the floor. The ingestion was estimated to have occurred 30 minutes prior to arrival. The child had ingested a maximum of 2 cc of the solution (0.26 mg/kg). Vital signs at triage were RR 36, HR 76, BP 98/68, and T 36.1 by axilla. The child had a fluctuating level of consciousness, but was responsive to noxious stimuli. Naloxone was given twice, with no response. An N/G tube was inserted, and activated charcoal (1 g/kg) was given. Due to a progressive decrease in level of consciousness, along with periods of apnea and desaturation, the patient was intubated and ventilated. All investigations, including CBC, electrolytes, BUN, creatinine, glucose, liver functions, serum osmolality, anion gap, blood gas, and EKG were normal. The patient was ventilated for 18 hours, and was extubated uneventfully. He remained in the ICU for 36 hours. He suffered no further complications related to the ingestion, after the first 18 hours. Conclusion: As little as 2 mL of the solution caused respiratory and CNS depression, necessitating intubation in a paediatric patient. To our knowledge, this is the first reported case of

toxicity due to accidental *ingestion.* Ingestion of a seemingly innocuous eye drop containing brimonidine led to a severe complication in a child. Childproofing this kind of product should be seriously considered.

74 A NEW CHEWABLE-SINGULAIR®.

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Background: Montelukast sodium, Singulair® is a leukotriene antagonist used to treat asthma. Children's daily dosage is 5 mg for ages 6 yr and older and 4 mg for those 2–5 years of age. Limited overdose information is available. *Methods*: A prospective study of calls placed to the poison center involving ingestions of Singulair® in children was performed for the 15 month period Jan 2000–March 2001. Patient demographics, dose and formulation ingested, clinical effects and outcome were reviewed. Results: 25 cases of Singulair® ingestion were documented in children ages 13 mo to 7 yrs; 21 cases (84%) were followed. 6 (24%) cases were evaluated in the ED; 4 of these 6 received charcoal. The largest estimated ingestion treated at home, without decontamination, was 60 mg in a 6 yr old; the largest estimated ingestion treated with decontamination (charcoal) was 96 mg in a 3 yr old. The average amount ingested for the 15 cases not sent to the ED was 18.2 mg. None of the patients developed any symptoms. Conclusion: More data must be collected to accurately predict a toxic dose in children. Preliminary information, based on our case series, indicates that up to 60 mg ingested by children produced no symptoms and required no decontamination.

75 THIRTY-MONTH RETROSPECTIVE ASSESSMENT OF THE TREATMENT OF SINGULAIR® EXPOSURES.

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Background: Montelukast, (Singulair®) is a leukotriene receptor antagonist used for prophylaxis and chronic treatment of asthma. Though prescription volumes have increased, limited information exists regarding toxic exposures. We present a case series of Singulair exposures. Case Series: During a thirty-month duration, (10/1/98–3/31/01), 74 Singulair human exposures were identified. 69 of these cases involved single exposures. Patients' ages ranged from 12 months to 67 years. 83% of reported case involved children under the age of 6, 7% of exposures were 6–12 years of age, and 10% were thirteen years or older. A dose variation ranged between 0.12–4.78mg/kg. 52 cases were reported as accidental exposures, while 14 cases were charted as therapeutic error, and 3 cases were reported as intentional suicide attempt. One patient received ipecac syrup previous to contacting the Poison Center. 48 (70%) patients remained at home for observation, while 20 (29%) patients presented to the emergency room. All patients presenting to an E.D. received a single dose of activated charcoal. Among the 69 Singulair cases presented, regardless of treatment instituted, no patients reported adverse or toxic symptoms. Conclusion: No adverse events related to ingestion of Singulair were identified in this case series. Give the similarity in outcomes between the two groups, activated charcoal may offer limited benefit.

76 REVIEW OF PEDIATRIC ACUTE UNINTENTIONAL ALBUTEROL AND RELATED SUBSTANCES EXPOSURES REPORTED TO TESS 1993 TO 1999.

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

Background: Bronchodilator exposures represent a common medication exposure reported to poison centers. The purpose of this study was to evaluate data regarding exposures to albuterol and related substances reported to the American Association of Poison Control Centers (AAPCC) Toxic Exposure Surveillance System (TESS) over a 7-year period. Methods: Exposures to albuterol and related substances were extracted from the AAPCC TESS database for 1993 to 1999. Inclusion criteria were children under 6 years of age with single acute ingestion resulting from an unintentional general exposure followed to known outcome. Summary statistics were compiled. Results: There were 24,977 exposures to albuterol and related substances in children under 6 years of age. Males accounted for 58.42% of cases. In 67.15% of children, no symptoms occurred. In symptomatic children the most common symptoms were tachycardia (19.09%), agitation/irritability (11.21%), vomiting (7.13%) and electrolyte abnormality (3.04%). Of 11,845 patients (47.42%) managed in a health care facility, 10,361 (87.47%) were discharged from the emergency department while 1,484 (12.52%) were admitted. The majority of exposures resulted in no effect (67.15%) or minor effects (23.36%). Moderate and major effects occurred in 9.36% and 0.13%, respectively. Symptoms in the 33 children with major effects included tachycardia (84.85%), electrolyte abnormality (39.39%) and hyperglycemia (33.33%). Conclusions: The majority of pediatric exposures to albuterol and related agents resulted in no or minimal toxicity.

77 COMA IN A TODDLER FROM LOW-DOSE CARISOPRODOL.

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Background: Carisoprodol is a centrally acting muscle relaxant. The number of exposures to children less than 6 years old from 1999 TESS report is 285. The minimum toxic dose in children is not well established. Previous case report established that ingestion of 3.5 grams by a 5-year-old child resulted in coma and death 40 hours postingestion. We report a case of a 2-year-old child ingesting 700 mg of carisoprodol that progressed to coma requiring respirator support before eventual recovery. Case Report: Grandparent reports setting out two 350mg tablets of carisoprodol on dresser, discovered tablets missing. A two-year-old (13kg) boy became increasingly drowsy over next hour and was brought to the emergency department. The patient was lethargic and hypoxic. Activated charcoal was administered. Over the next hour, his level of consciousness decreased requiring intubation to control airway and ventilation. Symptoms persisted for 12 hours. Child made full recovery. Conclusion: Ingestion of two tablets (700 mg, 54 mg/kg) of carisoprodol by a two-year-old boy resulted in severe central nervous system and respiratory depression with hypoxia.

78 SEVERE TOXICITY FOLLOWING 4-AMINOPYRIDINE OVERDOSE: CASE REPORT AND PHARMACOKINETIC DATA.

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Background: 4-Aminopyridine (4-AP) is a potassium channel blocker which increases the release of neurotransmitters, particularly acetylcholine and norepinephrine. It is currently under investigation for the treatment of multiple sclerosis; typical doses are 25–40 mg twice daily. Elimination is predominantly renal, and the half-life in therapeutic dosing is reported as 170–220 minutes. Case reports of 4-AP toxicity are rare, and kinetic data in human overdose is lacking. *Case Report:* A 14-year-old male intentionally ingested between 100 and 150 mg of 4-AP and soon thereafter complained of feeling ''twitchy''. Initial vital signs were: pulse, 114/minute; blood pressure, 146/89 mm Hg; respiratory rate, 20/minute; temperature, 96°F. The patient was diaphoretic. He had marked neuromuscular excitability, including fasciculations and increased muscle tone. Bowel sounds were hyperactive. The electrocardiogram showed sinus tachycardia with normal QRS and QTc intervals. The patient was intubated, underwent orogastric lavage, and received activated charcoal. He suffered one generalized seizure, which terminated after treatment with intravenous benzodiazepines and propofol. Sedation was weaned over the next few days, and the patient was extubated without sequelae. Analysis of serial 4-AP levels suggested first-order elimination with a half-life of 170 minutes. Conclusion: We report the first case of human 4-aminopyridine overdose with pharmacokinetic data. In this patient, who developed severe toxicity and whose peak serum level of 256 ng/mL is the highest yet reported, elimination appeared to be first-order with a half-life of 170 minutes. This value is similar to the half-life previously established for therapeutic dosing.

79 METAXALONE INDUCED MUSCULAR RIGIDITY.

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Background: Metaxalone (Skelaxin®) is a centrally acting muscle relaxant. We are unaware of any published cases of metaxalone overdose. Case Report: A 44-year-old female with a history of multiple sclerosis and ventriculoperitoneal shunt ingested 90 g of metaxalone. She developed intense muscular rigidity, encephalopathy with GCS 4, tachycardia, low-grade fever (99.1), diaphoresis, extensor posturing and tremulousness for 96 hours. Her legs were rigidly extended and internally rotated with feet plantar flexed. Tremulousness of her lower extremities was accentuated after light touch. These symptoms were accompanied by fixed and dilated pupils for 48 hours. EEG showed diffuse slowing and no seizure activity. Maximal CPK value was 950 ng/mL. CSF cultures were negative. CT of the brain demonstrated no evidence of shunt malfunction or other intracranial abnormality. The only serotonin agonist available to the patient was paroxetine and the level was non-detectable on admission. Gas chromatography-mass spectrometry of the patient's serum demonstrated an elevated peak consistent with metaxalone. Treatment included intubation, sedation with a propofol infusion and intermittent paralysis to control muscular rigidity. Her mental status returned to baseline and secondary to weakness she was released to an extended care facility for physical therapy and rehabilitation. Conclusion: Metaxalone, a muscle relaxant, can present in overdose with paradoxical rigidity accompanied by encephalopathy and tremulousness for a prolonged period of time.

80 SUCCESSFUL TREATMENT OF BACLOFEN (LIORESAL) OVERDOSE IN A DOG.

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Background: Baclofen overdose in pets is not uncommon. Case Report: A one-year-old 25 kg male Shetland Sheepdog presented after ingesting 280 mg (13 mg/kg) of Baclofen, a CNS GABA-B receptor agonist. Despite induction of vomiting by the owner within forty minutes, the dog was comatose, was in full respiratory arrest, and had a grand mal seizure within one hour of the ingestion. He was tachycardic and hypertensive with a respiratory acidosis. Both pupils were miotic with no direct or consensual light response. The patient was orotracheally intubated with a cuffed tube and manually ventilated with oxygen supplementation until independent respirations resumed twenty minutes later. An activated charcoal suspension containing a mild cathartic was instilled into the stomach via an orogastric tube, and intravenous crystalloid fluid diuresis was initiated. Intensive care therapy consisted of antiemetics, a beta-blocker to control tachycardia and hypertension, oxygen supplementation, gastric protectants and partial parenteral nutrition. Monitoring consisted of arterial blood gases, pulse oximetry, end-tidal carbon dioxide measurement, continuous electrocardiogram, pulse rate and quality, respiratory rate and effort, central venous pressure, urine output, coagulation status, hematocrit and standard chemistry values including electrolytes. Hematochezia, thrombocytopenia and a prolonged partial thromboplastin time prompted fresh frozen plasma transfusion and subcutaneous heparin therapy. Within 48 hours of presentation, the patient was alert, ambulatory and eating. He was discharged from the hospital within five days with no detectable abnormalities. Conclusion: When early aggressive detoxification and critical care measures are initiated, the mortality rate in dogs following acute Baclofen overdose is low despite the severity of initial clinical signs.

81 RHABDOMYOLYSIS IN A CHILD FOLLOWING UNINTENTIONAL DIPHENHYDRAMINE OVERDOSE.

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Background: Intentional overdose of antihistamines in adults has been associated with rhabdomyolysis and myoglobinuric renal failure. Case Report: A 23-month-old 14.5 kg male was brought to the emergency department 4 hours after unintentionally ingesting an unknown number of Equate® tablets containing 50mg of diphenhydramine. His parents reported no coingestants, fevers, or seizures since the time of ingestion. Vital signs included a heart rate of 192 beats/minute and temperature of 98.9°F. Examination was remarkable for dilated pupils to 6mm, dry, warm, and erythematous skin, diminished bowel sounds, and agitation. Since the child was easily consolable in the mother's arms, at no point was restraint necessary. Laboratory results included a CO_2 of 17 mEq/L, an anion gap of 11, potassium 4.9 mEq/L, BUN 8 mg/dL, creatinine 0.4 mg/dL, a normal urinalysis, and creatine kinase (CK) 1619 U/L (normal: 0–250 U/L). Intravenous fluid hydration was initiated, lorazepam was given 0.1mg/kg for sedation, and the child was admitted for observation to the intensive care unit. Repeat CK 6 hours later was 4505 U/L. Sodium bicarbonate was added to the intravenous fluids, and urine output was maintained at 2.0mL/hour. Subsequent CK levels of 2893 U/L, 2232 U/L, and 1205 U/L were noted on hospital days 2, 3, and 4. Serum diphenhydramine level was 136ng/mL 34 hours from time of ingestion. The central anticholinergic signs resolved 25 hours from the time of ingestion, and the child remained afebrile. Conclusion: Previous cases of rhabdomyolysis associated with diphenhydramine have been reported in intentional adult overdoses complicated by fever, seizure activity, or muscle damage by restraint. Despite mild central anticholinergic features and no other predisposing risks, the peak CK of 4505U/L in this patient is significant for his body weight. Antihistamine overdose warrants vigilance for rhabdomyolysis.

82 RETROSPECTIVE EVALUATION OF CETIRIZINE (ZYRTEC®) INGESTION.

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Background: There is limited published information on overdose of cetirizine, a new peripherally selective antihistamine. A single case reports suggests minor agitation. In response to this lack of information a multi-center retrospective case series was performed to evaluate the toxicity from cetirizine overdose. Method: All cetirizine exposures reported to 5 regional poison centers during the year 2000 were reviewed. Inclusion criteria included patient follow-up to a known outcome. Exclusion criteria were exposures involving unintentional double dosing and poly-drug ingestions. Results: 146 cases were evaluated. Patient age ranged from 4 months to 50 years with a mean and median of 5.4 years (±8.4) and 2 years, respectively. There were 83 (57%) males. Doses ingested ranged from 1 mg to 500mg with a mean and median of 43.4 (± 66.7) and 20 mg, respectively. Dosages ingested (evaluated only in children) ranged from 0.1 mg/

kg to 7.8 mg/kg with a mean and median of 1.8 (±1.7) and 1.1 mg/kg, respectively. Fifty-nine patients (40%) received direct medical evaluation in a HCF. Forty-two patients received activated charcoal, 7 received ipecac and 4 were lavaged. No other therapies were reported. Reason for ingestion was unintentional in 110, suicide in 17, therapeutic error in 15, and misuse in 4. Symptoms reported were drowsy (n = 9), restless/hyperactive (n = 7), tachycardia (n = 5), elevated blood pressure (n = 2) and mydriasis (n = 1). Neither coma nor seizures were reported. Medical outcomes reported were no effect (n = 124), minor effect (n = 17) and moderate effect (n = 5). The five patients with sinus tachycardia and labeled as moderate outcome were suicide attempts in adolescents and adults. The doses ingested in this subset ranged from 80 to 500 mg. Conclusion: In this case series cetirizine did not produce clinically significant CNS or cardiovascular toxicity, despite a mean dose ingested > 4 times the maximum recommended 24 hour dose in adults. Home monitoring with telephone follow-up may be appropriate for unintentional ingestions in children.

83 PROLONGED QT SEGMENT AND SYNCOPE WITH LORATIDINE USE.

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Objective: Loratidine is considered less cardiotoxic than terfenidine and astemizole, and clinically relevant loratidine-associated cardiotoxicity is rare. Though conflicting data exist, most suggest that loratidine does not affect cardiac conduction or prolong QTc. We report a case of loratidine-associated QTc prolongation and syncope. Case Report: A 46-year-old woman without cardiac disease presented to medical care after syncope. On the 5 previous days she had taken a loratidine 5 mg/ pseudoephedrine 120 mg tablet. In response to focused questioning regarding other agents that cause QTc prolongation or syncope, she denied use of any other product. Her vital signs and physical examination were normal and an otherwise normal 12-lead ECG revealed a prolonged QTc segment of 478 msec. Extensive laboratory investigations were all normal, including: potassium 4.4 mEq/L, magnesium 1.7 mg/dL, and calcium 8.7 mg/dL. The patient was monitored, received no medication and abstained from loratidine. The following day, her 12-lead ECG was normal with a QTc of 398 msec. Serum analysis by liquid and gas chromatography revealed therapeutic, non-toxic serum levels of: pseudoephedrine, 680 ng/mL; loratidine, undetectable in ng/mL; and descarboethoxyloratidine (loratidine metabolite), 3.6 ng/mL. Conclusions: Dysrhythmia from QTc prolongation was concluded to cause this patient's syncope. The clear history of loratidine use with non-toxic drug levels, syncope, QTc prolongation without other causality, and prompt normalization of QTc interval upon loratidine abstinence is apparently cogent evidence that this patient experienced QTc prolongation from loratidine use.

84 INADVERENT IV ADMINISTRATION OF ACYCLOVIR 800MG TO A CHILD.

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Background: Acyclovir is a commonly used antiviral medication. In large intravenous (IV) dosages, it may produce acute renal failure, crystalluria, agitation, irritability and hallucinations. We describe a case where IV acyclovir 800mg was inadvertently given to a child. No similar cases have been reported in this age group. Case Report: A 2-year-old, 12kg, male child was seen in the ED for cervical lymphadenitis. Oxacillin 800mg IV was ordered, but he was accidentally given IV acyclovir 800mg. Initially, no symptoms were noted; however, over the next several hours the child became increasingly agitated and confused. He was started on IV fluids, and frequent neurological exams were performed. Serial serum creatinine levels were obtained and are summarized in the following table:

Time since exposure	Serum creatinine
12 hours	1.1 mg/dL
16 hours	1.2 mg/dL
24 hours	1.3 mg/dL
36 hours	1.1 mg/dL
48 hours	0.8 mg/dL

All electrolyte and liver function tests remained normal following this event. Uri analysis performed 18 hours post exposure was completely normal. This child was managed with IV fluids and supportive care. Acetaminophen and

oxacillin were instituted for the lymphadenitis. Conclusion: Adverse effects seen in overdoses of acyclovir are renal and neurologic. These include renal insufficiency and failure, acyclovir crystalluria, agitation, tremor, and headache. This child experienced a transient increase in serum creatinine, lasting about 48 hours with no signs of renal failure. Agitation and irritability persisted for 12 hours. This case suggests that IV overdoses up to 67mg/kg in children may be managed by administering of IV fluids, monitoring renal function, and providing supportive care.

85 EMPIRIC TREATMENT WITH INTRAVENOUS (IV) ACYCLOVIR: IS IT SAFE?

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Objective: IV acyclovir is an effective treatment for herpes simplex encephalitis. Recently it has been increasingly used as an empiric therapy for patients with unexplained altered mentation. We reviewed all the patients who received IV acyclovir at an inner city, safety net hospital over a 6 month period to determine adverse sequelae associated with its use. Methods: Index case: a 35-year-old alcoholic with normal renal function empirically received 3 doses of IV acyclovir over 24 hr for unexplained confusion, despite a lack of focal neurologic deficits or abnormal LP results. Over the next 2 days the patient's serum creatinine rose from 0.6 to 6.4 mg/dL and he developed symptoms of psychosis, both attributed to the acyclovir. A chart review was then performed on all patients at our institution who had received IV acyclovir during the previous 6 months. Results: 36 patients were identified who had received IV acyclovir: 21 (58%) patients had no change in serum creatinine. Eleven (31%) had no serum creatinine measured during or soon after acyclovir therapy. Deterioration of renal function was discovered in 4 (11%) patients (the highest creatinine was 6.4 mg/dL), and 2 of these patients received acyclovir empirically despite a lack of focal neurologic findings or abnormal LP results. Both patients had extensive work-ups and prolonged hospital stays as a result of their renal injury, but eventually recovered within a week of stopping acyclovir. Conclusion: Despite serious potential adverse sequelae, empiric treatment with IV acyclovir is being used for patients with unexplained changes in mental status, even in the absence of focal neurologic deficits or an abnormal LP. Furthermore, many patients do not receive appropriate monitoring of renal function during therapy. Given the reported adverse effects of IV acyclovir, a careful risk/benefit analysis should be performed prior to empiric use.

86 ACYCLOVIR OVERDOSE IN A NEWBORN WITH CONSECUTIVE RENAL IMPAIRMENT.

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Background: Acyclovir has been used widely for treatment of documented or suspected herpes simplex infection in newborn. It is generally well tolerated and acute renal failure in the newborn has not been reported so far. Case Report: A 1.5-day-old, 3170-g girl had vesiculo-bullous skin lesions supporting a herpes simplex infection. An intravenous treatment with acyclovir was started. An unusual increase in weight to 3660g was noted on day 4 and a serum creatinine of 211 µmol/L on day 5. At this stage a dosing error of acyclovir was discovered. The patient had been given 100mg/kg instead of 10mg/kg 3 times daily intravenous over 4 days. Acyclovir then was discontinued. The creatinine decreased to normal within 2.5 days. On day 5 a renal ultrasound showed a marked enlargement of the kidneys. The follow up revealed no pathologic signs on day 12. The acyclovir blood levels were then measured. The highest level was 277 µg/mL (20-fold elevated over maximal therapeutic level).

Days after birth	1.50	3.50	5.25	5.50	6.00	6.25	6.50	6.75	7.00	7.25	8.00
Acyclovir treatment [0.3g/kg/d]	←			→							
Acyclovir blood level [µg/mL]		262	242	277	240	228	154	99	75	68	
Serum Creatinine [µmol/L]			211		174			156	138		89

Conclusions: To our knowledge this is the first case of a newborn with transient renal failure caused by acyclovir poisoning. The child recovered fully with conservative treatment only. In comparison with two cases previously reported, that experienced no renal toxicity (1), our case showed much higher serum levels of acyclovir. Hence, the case report

underlines the wide safety margin of acyclovir. Renal failure must be expected only after erroneous administration of very high doses and appears fully reversible after termination of acyclovir overdose. 1) McDonald LK et al. Lack of toxicity in two cases of neonatal acyclovir overdose. Pediatr Infect Dis J.1989: 529-32

87 SEVERE METHOTREXATE TOXICITY FOLLOWING ELECTIVE TERMINATION OF PREGNANCY.

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Background: Methotrexate (MTX), usual dose 50 mg/m^2 intramuscularly, is a common therapy for elective termination of pregnancies (ETOP) and non-ruptured ectopic pregnancies. Adverse effects from this regimen are rarely reported, and monitoring of patients for toxicity is believed unnecessary. However, complications may result from inappropriate dosing, idiosyncratic reactions, hypersensitivity or impaired renal function. We report the case of a woman developing significant toxicity from MTX therapy for an ETOP. Case Report: A healthy 30-year-old female, 6 weeks pregnant, received an intramuscular injection of 125 mg (80 mg/m^2) of MTX for an ETOP. Four days later, she presented to the ED with nausea, diarrhea, a rash on her hands, swelling to the face and a serum creatinine of 3.3 mg/dL. Her serum MTX level on admission was elevated at 1.1×10^{-7} M which when plotted on the MTX nomogram, necessitated leucovorin rescue therapy. During her hospital stay, she developed mucositis and severe bone marrow suppression with an absolute neutrophil count nadir of 358 on hospital day 9. The patient was treated with leucovorin (100 mg/m^2) every six hours continuously beginning on her second hospital day and was given a single dose of granulocyte colony stimulating factor for neutropenia on hospital day 9. She was discharged home on hospital day 11 with normal renal function and normal white blood cell and absolute neutrophil counts. Conclusion: MTX as therapy for ETOPs may cause significant toxicity particularly if the administered dose is higher than recommended based on body surface area. The narrow therapeutic index and potentially serious adverse effects of MTX, may thus require closer patient monitoring.

88 LEUCOVORIN RESCUE FOR MALICIOUS ABORTION ATTEMPT WITH METHOTREXATE.

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Background: Methotrexate, a dihydrofolate reductase and thymidine synthetase inhibitor, is used in cancer chemotherapy, and in the treatment of rheumatoid arthritis and trophoblastic disease. Because of its ability to inhibit purine synthesis, methotrexate is also used to abort ectopic and intrauterine pregnancies. Although leucovorin is routinely used to limit the toxic effects of methotrexate during chemotherapy, it has never been used to inhibit fetal demise. Case Report: A 31-year-old female who was 2 months pregnant presented to the ED immediately after maliciously receiving multiple intramuscular methotrexate injections into her thigh in an attempt "to induce an abortion." Although the exact dose was unknown, it was reported to be "a couple of milliliters" of the standard methotrexate solution (25 mg/mL). Thus her estimated dose was likely to be between 25–150 mg/m^2, which is consistent with the 50 mg/m^2 dose routinely used for abortion. Her initial vital signs, physical examination, and laboratory results were normal. An ultrasound showed a viable intrauterine pregnancy dated 7 weeks and 3 days. Leucovorin, 25 mg/m^2 (42.5 mg), was administered intravenously every 6 hours for 6 doses. A 3 hour post injection methotrexate level was 0.1 μmol/L, and repeat 24 hr methotrexate level was 0.0 μmol/L. The patient never developed signs or symptoms of methotrexate poisoning. Repeat ultrasound 10 days after the incident still revealed a normal viable pregnancy. A healthy baby boy was ultimately delivered. Conclusion: Although methotrexate is used as an abortifacient, there is no documentation of leucovorin use in preventing fetal demise. This case report suggests that leucovorin may be used to protect a fetus following an errant exposure of methotrexate.

89 DEATH BY QUININE.

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Background: Quinine has been used for centuries for various medicinal purposes. The major toxicities of quinine involve the vision, central nervous and cardiovascular systems. We present a case of a severe quinine toxicity that resulted in the death of the patient. Case Report: Our patient is a 32-year-old man who ingested thirty 325 mg tablets of quinine

(9.75 gm) and 6 beers 12 hours prior to presentation. He arrived to the Emergency Department in a lethargic state complaining of blurred vision and deafness. Vital signs on arrival were 117/68, 116, 20, 36.5. On exam, the patient had dilated (8mm) and sluggish pupils, lungs were clear to auscultation. During the initial evaluation, the monitor rhythm changed to a wide complex tachycardia (WCT) associated with hypotension. Cardioversion returned a normal rhythm and blood pressure. The patient was intubated due to decreased mental status, activated charcoal was given (100 gm.) and whole bowel irrigation was initiated. Further cardiac examination showed tachycardia with frequent extrasystoles. Sodium bicarbonate, amiodarone and dopamine were administered and he was admitted to the ICU. The bicarbonate was not continued, but the WCT and hypotension persisted despite pacing, cardioversion, 5L of fluids and maximal doses of vasopressors The patient was declared dead 52 hours after arrival to the hospital, following several episodes of PEA. Postmortem quinine levels were reported at 15 mg/L (Toxicity has been reported at > 9 mg/L). Conclusion: We describe a case of severe quinine toxicity with WCT that did not respond to conventional treatment and a syndrome of extreme peripheral vasodilation. Cases of quinine toxicity should be treated with aggressive decontamination, bicarbonate, overdrive pacing for refractory arrhythmias and enhancing elimination with multiple doses of activated charcoal.

90 ARRHYTHMIA AND DEATH IN AN ADULT ASSOCIATED WITH BENZONATATE.

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Background: The active ingredient of Tessalon Perles, benzonatate, is a topical anesthetic. Reports of toxicity are limited. Three deaths have reported, two in infants and the third in an 18 year old. Benzonatate has been associated with dysrhythmias and cardiac arrest in a 39 year old who was successfully resuscitated. Case Report: A 35-year-old female was found at home after being down an unknown number of hours, following a self-harm event. She was in ventricular fibrillation at the scene. She was intubated and resuscitated with lidocaine, and defibrillation. A bottle of Tessalon Perles was found empty. It is unknown how many capsules were in the bottle. Acetaminophen, aspirin and urine drug screens were all negative. She was initially acidotic, which was corrected with resuscitation. The patient's BP was unstable, requiring dopamine and phenylephrine. An ECHO showed an ejection fraction of 20% with global hypokinesia. The patient remained comatose. She developed a disconjugate gaze, and a positive Babinski reflex. She had no gag or cough reflex, and expired 14 days after the overdose. Conclusion: The patient suffered ventricular fibrillation arrest after an apparent benzonatate overdose from which she was resuscitated. She developed major cardiac and central nervous system dysfunctions and ultimately died.

91 STRYCHNINE POISONING: A CASE REPORT WITH TOXICOKINETIC DATA.

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Background: Strychnine poisoning is uncommon and most patients with severe strychnine poisoning die before reaching hospital. We describe a patient with strychnine poisoning who survived, together with some data on the toxicokinetics of strychnine. Case Report: A 42-year-old man, with no previous medical history, was brought to the ER one hour after ingestion of a bottle of wine together with some 'white powder' from his garden shed (this was later confirmed to be strychnine). Initially he was agitated and ataxic in keeping with his ethanol intake. Soon after arrival in the ER he developed a marked tremor and muscle spasms and shortly after this he had a respiratory arrest. He was intubated, paralysed and ventilated (and 50g nasogastric activated charcoal given), his temperature was 38.2°C, HR 95bpm and BP 85/40 mmHg. Blood gases showed a severe metabolic acidosis and he was given 3mL/kg 8.4% sodium bicarbonate. He was transferred to the ICU, over the first 24 hours he required norepinephrine to maintain his blood pressure (maximum dose 900mcg/hr). His temperature settled with simple cooling measures and rehydration and his CK peaked at 8218 IU/L. He was extubated on day 3, initially he had some muscle spasms, but by day 5 he was asymptomatic. A series of eight serum samples were taken over the first five days and subsequently analysed for strychnine concentrations. His initial strychnine level was 4.73mg/L at 1.5 hours after ingestion, this fell to 0.38mg/L and 74 hours and zero at 100 hours postingestion. This data conformed to a monoexponential elimination curve and the calculated elimination half-life was 12 hours. Conclusions: We describe a case of severe strychnine poisoning with a successful outcome with supportive care. Strychnine elimination half-life was 12 hours.

92 SURVIVAL AFTER INTENTIONAL HYDROFLUORIC (HF) ACID INGESTION.

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Background: Patients with intentional HF ingestions have a high mortality. Death results both from local injury and systemic complications of hypocalcemia and hyperkalemia. Calcium is a frequently used topical and systemic antidote, but inherent toxicity limits large intravenous doses. Comparatively, large doses of magnesium are better tolerated, however treatment with magnesium is not well described. We present a patient who survived a large HF ingestion after receiving only oral and intravenous magnesium therapy. Case Report: A 38-year-old man intentionally ingested about 50 mL of an HF containing rust remover (12% HF acid and 16% ammonium bifluoride). Initial vital signs were: BP, 139/83 mmHg; HR, 94/min; RR, 20/min; temperature, 96.8°F. Physical examination revealed a normal oropharynx and epigastric tenderness without guarding. On arrival, he received 60 mL of 50% magnesium sulfate via a nasogastric tube and 4 g magnesium sulfate intravenously. Calcium therapy was recommended but not given because there were no cardiac dysrhythmias. Pertinent initial laboratories were: Ca^{++}, 9.2 mg/L; Mg^{++}, 1.7 mEq/L; and K^{+}, 3.8 mEq/L. A 12-lead ECG showed sinus tachycardia at 100 beats/minute with a QTc interval of 450 msec. Repeat laboratories 10 hours later revealed: Ca^{++}, 8.3 mg/L; Mg^{++}, 3.4 mEq/L and K^{+}, 5.1 mEq/L. Esophageal endoscopy revealed severe erosive esophagitis and gastritis, a CT scan of the chest and abdomen was negative. The patient had an uneventful recovery. Conclusions: In HF ingestions, oral magnesium may provide a source of cations to complex with the toxic fluoride ions. This patient survived a potentially fatal ingestion of HF with magnesium sulfate therapy. Limited animal data suggests magnesium treatment as an alternative or addition to calcium therapy in HF burns.

93 NEBULIZED CALCIUM GLUCONATE FOR THE TREATMENT OF RESPIRATORY EXPOSURE TO HYDROFLUORIC ACID.

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Background: Hydrofluoric Acid (HF) is commonly utilized in the industrial setting. While skin exposure is common, there is relatively little information on HF inhalation injury. The use of 2.5% nebulized calcium gluconate has been suggested in the occupational literature and its use has been reported in one case study. We report the use of 2.5% nebulized calcium gluconate for the treatment HF gas inhalation. Case Series: A total of 5 patients were exposed to HF fumes while working at an industrial plant. They presented to the ED 4 hours after exposure. One patient was asymptomatic while another complained of nausea and abdominal cramps. The three others experienced the same symptoms of a terrible taste in their mouths, and a burning sensation to the tongue. There were no other respiratory complaints. Upon arrival to the ED all five patients received a 2.5% calcium gluconate nebulized treatment. Within moments of treatment the three patients with burning sensation to the tongue reported some resolution in their symptoms, and by the end of the treatment all symptoms had resolved. The patients were observed for a total of 6 hours in the ED and tolerated an oral challenge without difficulty. Upon discharge all patients were asymptomatic. All patients tolerated the treatment without adverse effects. Conclusion: We report a series of 5 patients from a single release of HF gas treated with nebulized calcium. This report suggests that 2.5% calcium gluconate nebulization is easily administered and is effective treatment for mild symptoms of HF inhalation with no adverse effects.

94 ELIMINATION OF PHENYLPROPANOLAMINE IN PERITONEAL DIALYSIS.

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Background: Phenylpropanolamine (PPA) is eliminated almost exclusively by the kidneys, with a half-life of 4 to 7 hours in patients with normal renal function. It is not recommended for persons with renal dysfunction. Although PPA has been removed from OTC medications, persons with renal failure may still utilize existing supplies in error. Case Report: A 10-year-old boy with a history of renal aplasia and failed renal transplant presented following a hypertensive crisis with a CVA. He had developed a URI about 3 days prior to admission, for which he was treated with an OTC cold preparation that contained PPA. Three days prior to admission his BP was 150/100. On the morning of admission, he woke up with headache and dizziness. He began vomiting and subsequently lost his vision. On a head CT, bilateral occipital infarcts were noted. After admission, he developed seizures. An MRI revealed 5 cortical lesions in the occipital

and parietal lobes, 2 frontal lesions and a left temporal lesion. An extensive infectious disease workup was negative. The patient underwent peritoneal dialysis nightly per his usual home routine. Serial serum PPA levels were obtained. At 300 ng/mL in the serum, 98 ng/mL were detected in the peritoneal dialysis fluid and none in the CSF. At 190 ng/mL in the serum, PPA was detected at 120 ng/mL in the dialysate fluid. The half-life of PPA in serum in this anephric patient was computed as 46 hours. Conclusion: PPA does diffuse into peritoneal fluid over time. Dialysate levels approached 75% of the serum level. Peritoneal dialysis may be an option for enhancing removal in patients without renal function. In this patient with no renal function who receives routine peritoneal dialysis, PPA was eliminated from the serum with a half-life of 46 hours.

95 USE OF WALD'S SEQUENTIAL TEST FOR GRADING THE SEVERITY OF ACETIC ACID POISONING (AAP) AND FOR PREDICTING ITS OUTCOME.

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Background: In clinical toxicology it is crucial to work out criteria of evaluation of the severity of a poisoning and prediction of its outcome by means of considering its characteristics and the results of observation of the patient in cases of AAP. Methods: In a retrospective study we included 197 patients with AAP 70%, admitted to the Toxicological center during the period of 1994–2000. As diagnostic characteristics, we chose data about the dose of the ingested substance, exposition time, the age of the patients, clinical data and data of certain well known and easy-to-use methods of study. Groups were formed based on the characteristic of the outcome of the poisoning (deceased–35, survived–162) and on signs: those who did not need resuscitation (n = 88) and those who did (n = 79), out of which 35 had an unfavorable outcome. Using a modification of the sequential analysis of Wald's[1], a diagnostic coefficient was estimated and a measure of informativeness of Kullback[2] in grades for each characteristic was determined. Results: A matrix for predicting the outcome and a matrix for recognition of the necessity of resuscitation were built. In the matrices criteria of severity of poisoning estimation developed by Persson et al. (1998) were used. Conclusions: The criteria of estimation of the severity of the case developed by Persson et al. (1998) are convenient and easy-to-use for practicing toxicologists. A grade-based method to estimate the patient's state is proposed. Auxiliary matrices will be helpful for the clinician in his work and help in a short period to determine an optimal technique of managing the poisoned patients with AAP. References: 1. Gubler EV. On using sequential statistical analysis in disease diagnostic. In: 5th International Congress on Cybernetics. Nature 1968, 881–883. 2. Kullback S. Information theory and statistics. New York: John Wiley and Sons, 1959.

96 SEVERE RHABDOMYOLYSIS FROM CERIVASTATIN (BAYCOL®).

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Background: Severe rhabdomyolysis is a rare complication of 3-hydroxy-3-methylglutaryl-coenzyme A (HMG CoA) reductase inhibitor, ''statin,'' therapy. Risk factors for developing statin-induced myopathy include older age, renal failure, high-dose therapy, concurrent use of hepatic cytochrome P450 (CYP) 3A4 inhibitors, and gemfibrozil. We report the first case of cerivastatin-induced rhabdomyolysis in the setting of CYP 2C8 inhibition along with the highest creatine kinase level reported for any statin-induced myopathy. Case Report: A 58-year-old female presented with two days of increasing muscle pain and weakness. She denied having any respiratory difficulty or bulbar symptoms, but noted having dark urine. Two weeks prior 0.8 mg of cerivastatin was added to her daily regimen of lisinopril and clopidogrel bisulfate (Plavix®). On examination, her vital signs were unremarkable with room air pulse oximetry of 97%. The neurological exam revealed intact cranial nerves, 3/5 proximal muscle and 4/5 grip strength. Initial labs included a serum creatinine of 0.6 mg/dL, CK 29,980 IU/L, ALT 868 IU/L, and urine myoglobin 62,303 ng/mL. Her urine was alkalinized and she received supportive care. By the fourth hospital day she had 2/5 generalized weakness, minimal respiratory compromise, normal renal function, and a peak CK of 135,500 IU/L. The patient remained hospitalized 7 days; her lower extremity weakness continued despite three months of rehabilitative services. Discussion: Cerivastatin, unlike other statins, is metabolized by two different CYP enzymes, 3A4 and 2C8 making it less susceptible to drug-drug interactions. Potent CYP 3A4 inhibitors increase the AUC and C_{max} of cerivastatin and its active metabolites while the clinical effect of CYP 2C8 inhibitors on cerivastatin metabolism remain unreported. Conclusion: In spite of dual metabolic pathways, CYP 2C8 inhibition may be a risk factor for cerivastatin-induced myopathy.

97 METFORMIN-ASSOCIATED LACTIC ACIDOSIS VS MESENTERIC ISCHEMIA.

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Background: Despite being widely reported, life-threatening metformin-associated lactic acidosis (MALA) may not be initially considered in patients with distracting chief complaints. The therapeutic range for metformin is 1–2 μg/mL, but MALA is reported with metformin levels ranging from 0.03–84.9 μg/mL. We describe severe MALA initially mistaken for mesenteric ischemia, and its successful treatment with hemodialysis (HD). Case Report: A 46-year-old man presented with 18 hours of severe, generalized abdominal pain without nausea, vomiting, or diarrhea. His PMH was significant for NIDDM, alcohol abuse, and an appendectomy. Medications included metformin, glyburide and quinapril. Initial vital signs were: BP, 80/50 mmHg; HR, 93/min; RR, 30/min; temperature, 92°F. Physical examination revealed an awake but uncomfortable man with dry mucosa, epigastric tenderness without rebound, and guaiac positive stools. Laboratory abnormalities included: WBC, 14.8 $\times$ 10^3/μL; HCO_3^-, 3 mEq/L; BUN, 40 mg/dL; Cr, 4.2 mg/dL; pH, 6.65; lactate, 21 mmol/L; and ethanol, 162 mg/dL. An abdominal x-ray showed mild ileus. He received IV crystalloids and sodium bicarbonate but continued to deteriorate. An exploratory laparatomy was negative for presumed mesenteric ischemia. In the ICU, he received 4 hours of HD for a presumed MALA during which time serial metformin levels were sent. The peak lactate was 40 mmol/L; pre-dialysis metformin was 8 μg/mL; and post-dialysis metformin was 3.7 μg/mL. The mean plasma clearance of metformin was 67.8 mL/minute. His hospital course was complicated by acute renal failure, which eventually recovered. Conclusion: Patients with MALA can present with nonspecific symptoms masquerading as mesenteric ischemia. After excluding ischemia, MALA is responsive to treatment with fluids, bicarbonate and HD.

98 METHEMOGLOBINEMIA INDUCED BY ROUTINE URATE OXIDASE ADMINISTRATION.

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Background: Urate oxidase, an enzyme derived from *Aspergillus flavus*, converts uric acid to the highly water soluble allantoin in patients with hyperuricemia. It has been used with few complications for over a decade in Europe against malignancy induced hyperuricemia in patients with acute lymphoblastic leukemia (ALL). It was recently introduced in the US for the same indication in place of allopurinol. Case Report: A 16-year-old African American male was admitted for chemotherapeutic treatment of B-cell ALL. On admission vital signs were normal, physical exam was unremarkable, and labs were notable for a hemoglobin of 5 and mild renal insufficieny (creatinine 4.0 mg/dL). Prior to urate oxidase infusion, one unit packed red blood cells was given without complication, and no other medicines were given. Within 2 minutes of intravenous urate oxidase infusion, the patient became dyspneic and his skin and lips appeared cyanotic. Pulse oximeter registered 85% despite administration of 100% oxygen. The patient's other vital signs and mental status remained normal. Arterial blood gas demonstrated pH 7.5, pCO2 38mmHg, pO2 110mmHg, Hb 7.0g/dL, and methemoglobin 14%. Other causes for cyanosis were excluded. Since the patient's G6PD status was unknown at this time, the oncology team withheld antidotal administration of methylene blue in favor of supportive therapy. Methemoglobin level 3 hours later was 10%. 12 hours later the dyspnea and cyanosis resolved and methemoglobin level was 2.3%. Subsequent testing confirmed the patient was not G6PD deficient. Conclusion: In the only previously reported case of methemoglobinemia associated with urate oxidase, the patient was G6PD deficient. Although the mechanism for methemoglobinemia in our patient is unclear, awareness of this potential reaction is warranted.

99 COLCHICINE RELATED DEATH PRESENTING AS AN UNKNOWN CASE OF MULTIPLE ORGAN FAILURE.

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Background: The clinical course following colchicine overdose has been well characterized. We describe the detection of an elevated colchicine blood level from approximately 72 hours post-admission in a case of multiple organ failure (MOF) and bone marrow suppression (BMS) found in a patient denying drug overdose. Case Report: A 45-year-old male clinical pharmacologist presented with acute renal and hepatic failure as well as hypotension and significant metabolic acidosis. Despite aggressive ICU care including CVVH, he had continued hypotension, leukocytosis, fever, renal and hepatic failure, lactic acidosis and hypocalcemia. On hospital day 3, his WBC fell to 200/mm^3 and platelet

count was 23,000/mm^3. Bone marrow biopsy showed marked aplasia without a specific etiology being elucidated. He received G-CSF and antibiotics, but died on hospital day 12. His past history was significant for gout and his medications included colchicine. Intentional drug ingestion was denied by the doctor and patient. The poison center recommended obtaining a colchicine level. Plasma colchicine level, 72 hours after admission, was 6.1μg/L. This level exceeds acute levels reported in prior fatalities. Conclusion: Colchicine overdose should be in the differential diagnosis in cases of MOF and BMS. Colchicine toxicity usually presents with GI symptoms, subsequent leukocytosis followed by severe pancytopenia. To our knowledge, this is the first case report of the use of a colchicine level to implicate this drug as the cause of MOF, BMS and death in a case of denied ingestion.

100 INGESTION OF A LARGE QUANTITY OF NEVIRAPINE IN A NON-HIV+ PATIENT.

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Background: There is very little published information regarding nevirapine overdose. The largest ingestion to date is 800mg with no reported adverse effects. This report is of an acute ingestion of an estimated 6 grams of nevirapine with no adverse events occurring. Case Report: A 26-year-old female took 30 Viramune® 200mg tablets in a suicide gesture. The patient was not HIV-positive and was otherwise healthy. She reported to the ED three hours after the ingestion, her vital signs were: 147/64, 102, 20, and 98.3°F. Lavage was not performed, nor was the patient given activated charcoal or cathartics. Salicylate and acetaminophen ingestion were ruled-out, her urine was positive for cannabis only. Approximately four hours after the ingestion, a basic metabolic panel and complete blood count were drawn and the results were completely within the normal ranges. The patient's liver function tests were: total protein = 7.4, albumin = 4.4, cholesterol = 199, total bilirubin = 0.6, direct bilirubin = 0.1, cpk = 134, ast = 14, alt = 16, alkaline phosphatase = 78, LDH = 136, ggtp = 24, again within the normal ranges. The patient was observed on a telemetry floor until 12 hours after the exposure and no adverse signs or symptoms were ever noted. At this time her vital signs were: 120/87, 82, 22, 97.5°F. Conclusion: The scientific literature contains no reports of acute overdoses of nevirapine; the manufacturer reports that 800mg was tolerated with no adverse effects in a single dose. There have been many adverse events reported with the chronic use of nevirapine, they include: rash, nausea, headache, fatigue, hyperlipidemia, hepatitis, liver enzyme abnormalities, Stevens-Johnson syndrome, and arthralgia. This patient was not HIV-positive and was otherwise healthy; she experienced none of these effects acutely. This case demonstrates that nevirapine overdoses is generally mild; even in acute ingestions up to 6 grams would cause only minor adverse effects at most.

101 CRYSTALLURIA AND RENAL DYSFUNCTION AFTER INDINAVIR OVERDOSE.

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Background: Indinavir (Crixivan®) is a protease inhibitor that effectively combats HIV infection but can cause crystalluria and nephrotoxicity during chronic therapy. The only published case of overdose describes a patient ingesting his own indinavir (6 grams) and presenting with nausea, dizziness and paresthesia. He received and vomited 50 grams of activated charcoal and was given prophylactic intravenous (IV) normal saline for 18 hours without developing crystalluria or any renal complications. This case suggested that indinavir overdose may not cause much renal toxicity. Case Report: A mother contacted the Poison Center (PC) about her 30-year-old son having ingested 16 to 24 grams of his HIV-positive brother's indinavir. At an emergency department (ED) the patient was lavaged, cleared medically and sent to a psychiatric facility, all within 3 hours of initially contacting the PC. Seven hours later the patient complained of severe left lower back pain and gave a urine sample that appeared bloody. After reluctance by an on-call physician for the psychiatric facility and after repeat urging by the PC, the patient was again transported to an ED for evaluation. His initial urinalysis was positive for blood, protein and crystals but his blood urea nitrogen (BUN), serum creatinine and urine output were normal. Intravenous (IV) normal saline at 200 milliliters per hour, IV morphine and IV anti-emetics were administered over the next 48 hours. His urine became increasing clear, his symptoms of flank pain decreased and his serum creatinine peaked at 2.1 with a BUN of 4. Within 72 hour of exposure, his symptoms resolved, his serum creatinine normalized to 1.1, and he was discharged from the hospital. Conclusion: Our case demonstrates that a single acute overdose of indinavir is capable of causing crystalluria and laboratory evidence of renal dysfunction. These renal complications developed within 12 hours, were managed with IV hydration, and resolved within 72 hours.

102 ACUTE TACROLIMUS OVERDOSE WITHOUT SIGNIFICANT TOXICITY.

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Background: Tacrolimus is a potent immunosuppressive agent that prevents organ rejection after transplantation. Chronic therapy is associated with nephrotoxicity, neurotoxicity, and increased risk of infections, malignancies and diabetes. Data on acute overdose in humans are rare and suggest no significant adverse effects. Although a single recent report associated myocardial ischemia with overdose, causality was lacking. We report a case of acute tacrolimus overdose confirmed by serum levels without the development of significant toxicity. Case Report: A 20-year-old female with a history of dysplastic kidneys and subsequent renal transplanation presented to the emergency department 14 hours after an acute intentional ingestion of approximately 41 mg (41 (1 mg) tablets) of tacrolimus. She also admitted to ingesting two tablets of prednisone and several tablets of mycophenolate mofetil. The patient complained of nausea, vomiting and headache but had normal vital signs. She was treated only with intravenous fluids and was admitted to the ICU for observation. A tacrolimus level of 42.6 ng/mL (therapeutic trough levels range from 9.8 to 19.4 ng/mL) was recorded. The next day she had a mild increase in serum creatinine from 0.8 mg/dL to 1.1 mg/dL but remained asymptomatic. No other signs or symptoms of toxicity occurred and she was discharged home after 48 hours. Conclusion: Although chronic tacrolimus use is associated with multiple adverse effects, we report one of the highest serum tacrolimus levels obtained following an acute ingestion with no associated significant toxicity. This single case report cannot be used to exclude potential toxicity following acute tacrolimus overdose and a prospective study is therefore necessary to properly evaluate this problem.

103 HYPOTENSION INDUCED BY MASSIVE CAFFEINE OVERDOSE RESPONSIVE ONLY TO VASOPRESSIN INFUSION.

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Background: We report a case of hypotension induced by massive caffeine overdose that was responsive only to vasopressin infusion. Case Report: A 41-year-old presented 3 hours after ingesting 50 gm of caffeine. Her pulse was 206 with a systolic blood pressure (SBP) of 140. She was agitated and had a tremor. A wide complex tachycardia was noted on EKG. Metoprolol (5mg) was infused and her rhythm degenerated into multiple dysrhythmias including ventricular fibrillation, wide complex tachydysrhythmias, bradycardia, asystole and pulseless electrical activity. Multiple defibrillations, 9 mg epinephrine, 2 mg atropine, 200 mg lidocaine, 2 gm $CaCl_2$, 2 gm $MgSO_4$, 350 meq $NaHCO_3$, and eight liters of normal saline were administered over the ensuing 4 hours. Despite dopamine (20mcg/kg/min) and norepinephrine (20 mcg/min) infusions, her SBP by doppler remained in the 50's. A vasopressin infusion was begun with good effect and titrated to sustain a SBP in the 80's. Hemodialysis could then be performed. The patient demonstrated dramatic improvement during dialysis resulting in discontinuation of all vasopressor support. A caffeine level was 405 mcg/mL. Twenty-four days after arrival, she was discharged to home and has made a full recovery. Conclusion: This case suggests that hypotension induced by caffeine toxicity and refractory to standard therapy may respond to vasopressin infusion.

104 HOW FEASIBLE IS IT TO CONFORM TO GUIDELINES ON ADMINISTRATION OF ACTIVATED CHARCOAL WITHIN ONE HOUR OF AN OVERDOSE?

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Objective: The role of activated charcoal in the treatment of poisoning has recently been reviewed in a position statement from the EAPCCT and AACT. This recommends its administration within an hour of ingestion of a potentially serious amount of a toxin. This study aimed to examine the treatment of acutely poisoned patients with activated charcoal in an ER of one hospital. Methods: 63 consecutive patients who had taken potentially serious overdoses and required hospital admission were identified over a six-month period. The patients' case notes were analyzed for age, sex, substances taken, and the timing of their management within the ER. Results: The median time of arrival after the overdose was 136 minutes, and only 15 of 63 patients presented within 1 hour of ingestion. 10 of these 15 patients were given activated charcoal, and only 4 of the 10 received it within the one-hour limit. 16 patients received charcoal outside the time limit. Sub-analysis of the individual cases given charcoal reveals that triaging is fast (median 5 minutes), but a significant time delay occurs after this before charcoal is administered (median 21 minutes). Conclusion: The majority of seriously poisoned patients presented after 1 hour, making them ineligible for therapy. For the significant number

who did present within one hour of taking their overdose, delays within the ER led to the majority receiving their charcoal late. It is important that those who have ingested a potentially serious overdose and have presented within the one-hour interval are rapidly identified and fast-tracked for activated charcoal therapy.

105 INTUBATION OF POISONED PATIENTS IN AN URBAN EMERGENCY DEPARTMENT.

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Background: There are limited data regarding emergent airway management of poisoned patients. Objective: We sought to characterize the experience with intubation of poisoned patients in an urban teaching hospital ED. Methods: Since 1992, the department of Emergency Medicine has maintained a database for all intubations performed in the ED. We queried the database for toxicology cases and examined these cases via structured chart review. Results: 100 cases were identified (3.7% of all intubations). Nine records were lost, leaving 91 for review. The source of poisoning was: intentional overdose (63), recreational overdose (19), snakebite (2), hydrocarbon aspiration (2), CO (2), and 1 each for caustic, smoke inhalation, and NMS. 47% were male. The median age was 34 yr (range: 9 mo – 82 yr). The primary indications for intubation were airway protection (69) and apnea (22). Eighty-six patients were intubated orally, 3 by blind nasal intubation, and 1 each via fiberoptic nasal intubation and laryngeal mask airway. No surgical airways were required. Most patients were intubated on the first (73%) or second (18%) attempt. Five patients required 3–6 attempts. The training of the operator was: PGYI 11%, PGYII 38%, PGYIII 45%, ED faculty 4%, and Anesthesia 1%. Induction agents were used in 74% and neuromuscular blockers in 91%. Etomidate (70%) and midazolam (26%) were the most common induction agents. Succinylcholine (96%) was the most common paralytic. Eight patients (9%) had immediate complications: emesis (4), right mainstem intubation (3), hypotension (1), and soft tissue bleeding (1). Of patients with emesis, 3 developed clinical pneumonitis. All but 2 patients survived, including 6 discharged directly from the ED. Neither death was due to intubation complication. Conclusion: ED intubation of poisoned patients is generally safe. The most common complication is emesis with subsequent aspiration pneumonitis.

106 URINE COLLECTION CONTAINERS: UNSUSPECTED DANGER?

Lucanie R, Chiang WK. *Hudson Valley Poison Center, Phelps Memorial Hospital, North Tarrytown, NY*

Background: Urine collection containers are often given to patients so that urine can be collected at home for later testing. These containers are large and the chemicals added are often in dangerous quantities. No information was found in Poisindex on the types and quantities of chemicals that may be used in these containers. Case Report: A previously healthy 18-month-old boy was discovered drinking from a urine collection container sent home for his father. The container was meant to be used to test for kidney stones and contained 30 mL of 10% hydrochloric acid; 15 mL was missing with some spillage. When the child vomited at home, he was brought to the local hospital. He arrived at the emergency department 15 minutes after the ingestion. The boy was given fluids and observed. Within 45 minutes of ingestion the patient vomited again. While no oral burns were observed at this time, an ENT consult was recommended. Within 2 hours, swelling of the epiglottis was noted and the patient was intubated. The child was then transferred to a tertiary center. 19 hours after the exposure, the patient underwent an endoscopy, and multiple 2nd degree esophageal burns were noted in addition to oral burns. The patient was started on steroids and antibiotics, and remained in the hospital for 8 days before he was discharged home. This case caused us to research the dangerous substances that may be found in urine collection containers. Our search found that urine containers did not have safety caps despite the addition of chemicals such as: glacial acetic acid 33–97%, nitric acid 70%, perchloric acid 60%, formaldehyde 37%, saccomanno fluid, and potassium fluoride 300mg, as well as the 10–50% hydrochloric acid seen in this case. Conclusion: Since dangerous chemicals may be added to urine collection containers without safety caps, medical professionals must be aware that exposure to these substances may lead to severe injury in children.

107 ACCURACY OF CAREGIVERS IN ASSESSING LIQUID MEDICATION SPILLS.

Branton T, Ciancaglini P, Benitez J. *Finger Lakes Regional Poison and Drug Information Center, University of Rochester, Rochester, NY*

Background: Poison Specialists manage pediatric liquid medication ingestions based on the caller's memory of how much product was used prior to the exposure and estimation of how much is spilled. We propose that caregivers (CG) are accurate in estimates of spill volume. Previous studies have not addressed this issue. Methods: A convenience sample of 62 CG of children between the ages of 14 months and 3 years attending a pediatric clinic with a home telephone for

follow up, were enrolled. A questionnaire asked for an estimate of the amount of liquid medication left in a bottle at home, the amount was confirmed by a telephone call that evening. An aliquot of 15 mL of Guaifenesin Syrup, USP was poured on a t-shirt and a floor tile. CG were asked to estimate the amount spilled on both. Results: Eleven subjects were excluded because of ambiguous or incomplete data. Of the 51/62 subjects included in the t-shirt spill segment, 28/51(55%) were able to assess the spill within 15 mL (difference range −13.25 mL to 225 mL, mean 33.6 mL). 21/51(41%) overestimated the spill by 30 mL or more. 55/62 CG were included in the tile spill segment. 30/55(55%) were accurate within 15 mL, (difference range −13 mL to 225 mL, mean 33.8 mL). 24/55(44%) overestimated the spill by 30 mL or more. 38/62(61%) CG were available for home follow up and 35/38(92%) were accurate within 30 mL of product in home medication. There was no statistical difference between the estimation and the actual amount of medication at home ($p = 0.08$). Conclusion: Within the limitations of this pilot study, caregivers recalled the bottle contents accurately within 30 mL. Spill amounts are overestimated more than 40% of the time. Though further study is needed, this suggests that ingestions may be greater than estimated and that children may be at increased risk of toxicity.

108 THE SUICIDAL PATIENT'S PERCEPTION OF TOXICITY AND RISK OF FATALITY FROM THEIR OVERDOSE–HOW MUCH IS ENOUGH?

Herrington L, Gorman S, Geller R, Kaslow N. *Georgia Poison Center, Atlanta GA*

Background: While suicide accounted for only 7% of the 2.2 million poisonings reported to the AAPCC Toxic Exposures Surveillance Survey in 1999, it accounted for 54% of all the reported fatalities. The purpose of this study is to evaluate the difference between the perception of risk held by the suicidal patient and actual toxicity. Methods: 200 women were enlisted in a suicide study conducted at a large metropolitan hospital. Demographics information, psychological and stress survey results, questions about the circumstances of the event, patient intent, a self assessment of the potential risk of fatality from their suicidal action, and medical outcome were obtained. Results: Ages ranged from 18–62 years; 92% were women of color; 65% had at least a high school education. Poisoning accounted for 88% of the attempts. Of this group, 59% wanted to "remove themselves" from their environment; 26% wanted to "change or manipulate" their environment. While 42% believed that their poisonings "exceeded or equaled what (they) thought was lethal", and that "death was probable or certain", only 10% reported significant sequelae. Comparative assessment by 2 Board Certified Toxicologists differed significantly from the patient's perception of risk. Conclusion: The perceptions of the risk of lethality of the suicidal women in this study group differed significantly from the toxicologist's assessment and the known outcome. The impact of emergency medical treatment on these outcomes could not be assessed from the study group. The suicidal patient's intention (attempt vs gesture) combined with the inaccurate perception of toxicity of their overdose could lead to unexpected outcomes–fatality or survival.

109 AVAILABILITY AND PRESERVATIVE TOXICITY OF IV PYRIDOXINE HCL.

Burda AM, Sigg T, Wahl M. *Illinois Poison Center, Chicago, IL*

Objective: Large doses of IV pyridoxine HCl (B6) are required for acute isoniazid (INH) overdose (e.g. adults: 5–10 grams, children: 70mg/kg). Standard references state that B6 is available in a concentration of 100mg/mL, in sizes of 1mL, 10mL, and 30mL. We attempted to determine which drug companies were currently marketing these products and to evaluate them for potential preservative toxicity. Methods: Companies were contacted from a compiled list of manufacturers of IV B6. These include all companies listed in: Drug Topics Red Book, PDR, Facts & Comparisons, Poisoning & Toxicology Compendium, AHFS-DI, Poisindex®, the FDA Drug Listing Branch, as well as various Internet sites. Package inserts were obtained to examine excipient composition. Results: Only two manufacturers were found to market B6 nationally. American Pharmaceutical Partners (APP) sells B6 in 1 mL vials (100mg/mL). The AWP is $2.83/vial and has a shelf life of 2 years. This product is preserved with 0.5% chlorbutanol. Legere Pharmaceutical Co. markets a compounded 30mL (3gm) vial; the cost is $17.95/vial and it has a shelf life of 1.5–2 years. This product is preserved with 1.5% benzyl alcohol. The 10mL vials are no longer sold. Discussion: Most pharmaceutical manufacturers of B6 no longer market IV B6 nationally. Some smaller companies compound this product and sell it only within their state borders. A 5–10 gram dose (50–100 1mL vials) of B6 using the APP product will deliver a 250–500 mg dose of chlorbutanol, which is related to chloral hydrate. This dose may produce mild CNS depression. Thus, in the setting of INH overdose, correction of metabolic acidosis and cessation of seizures may have to be used as therapeutic endpoints and not complete reversal of CNS depression. A 5-10 gram dose of B6 using 30mL vials from Legere Co. will deliver 750-1500 mg of benzyl alcohol. This preservative dose is not expected to pose significant toxicity.

110 HEPATOTOXICITY IN ACUTE SUSTAINED-RELEASE NIACIN OVERDOSE.

Paopairochanakorn C, White S, Baltarowich L. *Children's Hospital of Michigan Regional Poison Control Center, MI*

Background: Niacin has been used in the treatment of hyperlipidemia. There are several reports of niacin-induced hepatotoxicity in patients chronically taking sustained-release preparations. Recently, niacin has been promoted among drug users to enhance the elimination of marijuana and metabolites from the urine. We report 2 cases of hepatotoxicity associated with acute sustained-release niacin ingestion. Case Series: Both patients developed abdominal pain, nausea, and vomiting after the ingestion of 5 and 6 grams of sustained-release niacin during the 48 hour period preceding urine drug testing. Other than a history of marijuana abuse, their past medical histories were negative. There was no history of acetaminophen ingestion or chronic alcohol abuse. Both had elevated transaminases, coagulopathy and increased anion-gap metabolic acidosis, without evidence of encephalopathy or bleeding. Laboratory results from patients A & B respectively were: SGOT, 462 and 333 IU/L; SGPT, 285 and 312 IU/L; INR, 1.97 and 3.4; anion gap, 18 and 24. Serum acetaminophen and salicylate levels were negative in both patients. Urine drug screens did not reveal cannabinoids. A hepatitis panel was negative in one patient. Laboratory abnormalities improved within 72 hrs. of admission. Single dose NAC and daily parenteral vitamin K1 therapy were given to one patient. Conclusion: Acute sustained-release niacin ingestion should be considered in the differential for hepatotoxicity in drug-using patients.

Platform Session 3 — Sunday, October 7 — 11:30 am–12:30 pm
Abstracts #111–#114

111 THE ABRUPT CESSATION OF THERAPEUTICALLY ADMINISTERED SODIUM OXYBATE (GHB) DOES NOT PRODUCE WITHDRAWAL SYMPTOMS.

Hornfeldt CS, Pertile TL. *Orphan Medical, Inc., Minnetonka, MN*

Objective: A severe abstinence syndrome has been recently described following the abrupt cessation of illicit, chronic high dose gamma-hydroxybutyrate (GHB). Sodium oxybate, a pharmaceutical form of GHB being studied under FDA- and TPP-approved INDs, has demonstrated efficacy for the treatment of narcolepsy. The following long-term efficacy study provided a model to test the hypothesis that daily dosing of sodium oxybate in narcoleptics does not cause withdrawal following abrupt cessation. Methods: Fifty-five narcoleptic patients taking sodium oxybate (dose range 3–9 gm/night) for 7 to 44 months (mean = 21 months) were randomized into a 2-week single-blind period when they continued to receive sodium oxybate and a 2-week double-blind period when they received placebo or sodium oxybate. Narcolepsy symptoms and adverse events were recorded in daily diaries. Results: During the double-blind period, 29 patients received placebo and 26 received sodium oxybate. The sodium oxybate group had no median increase in weekly cataplexy attacks compared to an increase of 21.0 for the placebo group ($p < 0.001$). There were minimal signs of withdrawal such as insomnia, anxiety or tremor. Conclusion: The return of cataplexy symptoms following the cessation of long-term therapy demonstrates the efficacy of sodium oxybate for the long-term treatment of narcolepsy. Narcolepsy patients treated with sodium oxybate at doses of 3–9 gm nightly for up to 44 months demonstrated minimal evidence of withdrawal upon abrupt cessation of therapy. In contrast, reports of withdrawal describe the self-administration of illicit GHB with escalating doses and increasing frequency. Based on the results of this study, we conclude there is minimal evidence of withdrawal symptoms following abrupt cessation of chronic sodium oxybate dosing in the therapeutic range.

112 4-METHYLPYRAZOLE (4-MP, ANTIZOL®) DECREASES *IN VIVO* BLOOD GHB CONCENTRATIONS AND TOXICITY FROM 1,4-BUTANDIOL (1,4-BD).

Quang L[1], Desai M[2], Kraner J[3], Shannon M[1], Woolf A[1], Maher T[2]. [1]*Children's Hospital, Massachusetts/Rhode Island Poison Control Center, Harvard Medical School,* [2]*Massachusetts College of Pharmacy and Health Sciences, Boston, MA,* [3]*AIT Laboratories, Indianapolis, IN*

Background/Objective: Published studies have observed 4-MP, an alcohol dehydrogenase (ADH) antagonist, to block toxic manifestations of 1,4-BD in male CD-1 mice. We investigated if these observations corroborate with 1,4-BD and

GHB blood concentrations. Methods: Male CD-1 mice were *pretreated* with 4-MP 25 mg/kg i.p., or deionized, distilled water (controls), followed 5 min. later with 1,4-BD 600mg/kg i.p. (Toxic $Dose_{50}$). Another group of mice was administered 1,4-BD 600 mg/kg i.p. first, followed 5 min. later with 4-MP 25 mg/kg i.p. as an *antidote* or deionized, distilled water (controls). After 60 min., all mice were tested for the righting reflex and rotarod test (ability to log roll for 10 sec. on a 1-inch diameter rod revolving at 6 RPM) and sacrificed for blood 1,4-BD and GHB concentrations by GC/MS. Results:

	Avg. BD Level (μg/mL)	Avg. GHB Level (μg/mL)	Righting Reflex	Rotarod Test
4-MP Pretreat	1505	9	Present	Passed
Controls	<5	2532	Absent	Failed
4-MP Antidote	1069	95	Present	Passed
Controls	<5	1836	Absent	Failed

Conclusions: 4-MP, given as a pretreatment and as an antidote, was effective in blocking *in vivo* biotransformation of 1,4-BD to GHB. The presence of high 1,4-BD blood concentrations did not correlate with failure of the righting reflex or rotarod test. Conversely, the presence of blood GHB concentrations correlated with failure of the righting reflex and rotarod test.

113 *IN VIVO* TOXICITY OF GAMMA-VALEROLACTONE (GVL), A CONGENER OF GAMMA-BUTYROLACTONE (GBL).

Quang L[1], Desai M[2], Boyer E[1], Shannon M[1], Woolf A[1], Maher T[2]. [1]*Children's Hospital, Massachusetts/Rhode Island Poison Control Center, Harvard Medical School,* [2]*Massachusetts College of Pharmacy and Health Sciences, Boston, MA*

Background/Objectives: GVL, a methylated congener of GBL, now appears on internet "recipes" as a key ingredient for synthesis of gamma-hydoxyvalerate (GHV), a methylated congener of GHB. GHV has been reported by users to produce GHB-like effects, but GVL effects have not been described. Because GVL is the lactone ring structure of GHV, we hypothesize that it may undergo *in vivo* biotransformation to GHV by tissue lactonases or nonenzymatic hydrolysis. We investigated the potential of GVL to produce GHB-like toxicity. Methods: Male CD-1 mice received GVL 600mg/kg i.p. (n = 7), 1200mg/kg i.p. (n = 7), 1800mg/kg i.p. (n = 7), or control injections of deionized, distilled water i.p. Toxicity was assessed every 15 min. by observing for seizures, the righting reflex, and rotarod test (ability to log roll for 10 sec. on a 1-in. diameter rod revolving at 6 RPM). Results: Seizures, loss of righting reflex, and failure of the rotarod test were not observed in mice receiving control injections, GVL 600mg/kg, and GVL 1200mg/kg. Mice receiving GVL 1800mg/kg did not lose their righting reflex, but 6/7 of these mice failed the rotarod test 20 minutes after administration for a total duration of 210 minutes. Conclusions: Previous studies from our lab have observed seizures as well as failure of the righting reflex and rotarod test from the GHB prodrug, 1,4-butanediol (1,4-BD), at doses of 560mg/kg and 170mg/kg, respectively. GVL appears to have less toxicity than 1,4-BD because seizures or loss of the righting reflex were not observed at any dose, and impairment of the rotarod test occurred only at 1800mg/kg. The methyl group appears to decrease GVL toxicity as a GHB-like prodrug.

114 NCS-382 AND CGP-35348 DECREASE 1,4-BUTANEDIOL (1,4-BD) TOXICITY.

Quang L[1], Desai M[2], Boyer E[1], Shannon M[1], Woolf A[1], Maher T[2]. [1]*Children's Hospital, Massachusetts/Rhode Island Poison Control Center, Harvard Medical School,* [2]*Massachusetts College of Pharmacy and Health Sciences, Boston, MA*

Background/Objective: 1,4-BD is a GHB prodrug that produces toxicity after overdose by two GABAergic mechanisms: 1.) GHB interaction with GHB-specific receptors that modulate release of GABA in pathways expressing GABA-B receptors, and 2.) *in vivo* GHB metabolism by GHB dehydrogenase to GABA. We investigated if the toxicity of 1,4-BD and GHB by GABAergic mechanisms can be decreased with NCS-382 and CGP-35348, novel high affinity antagonists of GHB receptors and GABA-B receptors, respectively. Methods: Male CD-1 mice in group I were pretreated

with CGP-35348 200mg/kg i.p. (n = 5) or with control injections of deionized, distilled water (n = 5), followed 10 min. later by 1,4-BD 300mg/kg i.p. (TD_{50} = 170mg/kg for the rotarod test). Mice in group II were pretreated with NCS-382 200mg/kg i.p. (n = 5) or control injections (n = 5), followed 10 min. later by 1,4-BD. Mice in group III received NCS-382 *and* CGP-35348 (n = 5) or control injections (n = 5), followed 10 min. later by 1,4-BD. Toxicity was then assessed every 10 minutes by the rotarod test (ability of the mouse to log roll on a one in. diameter rod revolving at 6 RPM). Results: CGP-35348 decreased the duration of rotarod failure from 170 minutes in control animals to 80 minutes in pretreated animals. NCS-382 pretreatment did not improve rotarod performance. NCS-382 *and* CGP-35348 decreased the percentage of animals that failed the rotarod test from 100% in control animals to 40% in pretreated animals. Conclusions: Pretreatment with CGP-35348 and its combination with NCS-382 attenuate 1,4-BD toxicity in CD-1 mice. The potential therapeutic utility of these receptor antagonists in treating patients with 1,4-BD toxicity warrants further examination.

Platform Session 4 — Sunday, October 7 — 4:00 pm–5:00 pm
Abstracts #115–#118

115 1,4-BUTANEDIOL (1,4-BD) INTERACTIONS WITH ETHANOL AND "CLUB DRUGS."

Quang L[1], Desai M[2], Boyer E[1], Shannon M[1], Woolf A[1], Maher T[2]. [1]*Children's Hospital, Massachusetts/Rhode Island Poison Control Center, Harvard Medical School,* [2]*Massachusetts College of Pharmacy and Health Sciences, Boston, MA*

Background/Objectives: 1,4-BD is abused at dance raves and nightclubs, where ethanol (ETOH) and other drug use is prevalent. We examined interactions between 1,4-BD and ETOH as well as the "club drugs," flunitrazepam and ketamine. Methods: Drug doses were derived from our lab (1,4-BD, ETOH) or literature review (flunitrazepam, ketamine) and administered by i.p. route. Male CD-1 mice received 1,4-BD 600mg/kg alone (n = 10), ETOH 2g/kg + 1,4-BD 600mg/kg (n = 10), ETOH 2g/kg + 1,4-BD 1g/kg (n = 10), ketamine 100mg/kg + 1,4-BD 600mg/kg (n = 10), or flunitrazepam 5mg/kg + 1,4-BD 600mg/kg (n = 10). Toxicity was evaluated every 10 min. by the righting reflex and rotarod test. Results: Mice receiving 1,4-BD (600mg/kg) alone, and in combination with flunitrazepam, ketamine, and ETOH, lost their righting reflex for 110, 180, 110, and 0 min., respectively. Mice receiving *high dose* 1,4-BD (1g/kg) and ETOH lost their righting reflex for 300 min. Mice receiving 1,4-BD (600mg/kg) alone, and in combination with flunitrazepam and ketamine, failed the rotarod test for 210, 240, and 190 min., respectively. Mice receiving 1,4-BD (600mg/kg) and ETOH failed the rotarod test for the initial 50 min., followed by a 50-min. period when mice recovered their ability to perform the rotarod test. However, toxicity then recrudesced with all mice again failing the rotarod test for 120 min. Mice receiving *high-dose* 1,4-BD (1g/kg) and ETOH failed the rotarod test for a sustained 500 min. Conclusions: For the righting reflex, rank order of toxicity was: 1,4-BD (1g/kg) + ETOH > 1,4-BD + flunitrazepam > 1,4-BD alone and 1,4-BD + ketamine > 1,4-BD (600mg/kg) + ETOH. For the rotarod test, it was: 1,4-BD (1g/kg) + ETOH > 1,4-BD + flunitrazepam > 1,4-BD alone >1,4-BD + ketamine. 1,4-BD (600mg/kg) and ETOH recrudescing toxicity may represent dose-dependent, delayed 1,4-BD toxicity from greater alcohol dehydrogenase affinity for ETOH.

116 GHB-RELATED FATALITIES.

Dyer JE, Haller CA. *California Poison Control System-San Francisco Division, University of California at San Francisco, San Francisco, CA*

Objective: This descriptive surveillance study was undertaken to characterize common clinicopathologic features of fatalities associated with gamma hydroxybutyrate (GHB), and its chemical precursors gamma butyrolactone (GBL) and 1,4-butanediol (BD). Methods: Autopsy and investigative reports for GHB related fatalities were obtained by written request from state medical examiners and coroners. Demographic, toxicologic and pathologic findings were reviewed for each case. Results: Twenty GHB/GBL/BD death reports were reviewed. 15 of 20 cases were male, and 85% were between 20 and 34 years of age. The youngest victim was 15 years old. Post-mortem blood levels ranged from 140 to

2900 mg/L. Urine levels were 305 to 27,000 mg/L Coingestants were detected in 80% of death cases. Ethanol was the most common coingestant (60% of deaths) and plasma levels ranged from 10 to 170 mg/dL. Stimulants, including MDMA, cocaine, and amphetamine were detected in 4 cases. Other drugs were found in 5 cases including caffeine, butalbital, venlafaxine and acetaminophen. Autopsy showed four victims had aspirated gastric contents. Pathologic findings included pulmonary edema and congestion in 80% of cases, and cardiomegaly or left ventricular hypertrophy in 35% of cases. The cause of death was listed as drug intoxication in 15 cases, with other causes listed as cardiorespiratory arrest (3), drowning, idiopathic cardiac arrhythmia, and hypovolemic shock. Conclusions: 80% of GHB related fatalities involved a coingested drug, most commonly ethanol. Many deaths were a result of respiratory compromise due to aspiration of gastric contents, positional asphyxia, and pulmonary edema, supporting the need for prompt airway management in cases of GHB overdose. Some deaths were a result of traumatic injury or accident, possibly due to abrupt loss of consciousness seen with GHB. The finding of cardiac enlargement in 1/3 of death cases warrants further investigation.

117 LANGUAGE AND BARRIERS TO POISON CENTER UTILIZATION.

Griffin M, Barrera-Garcia V, Thompson G, Watson W. *South Texas Poison Center, University of Texas Health Science Center at San Antonio, San Antonio, TX*

Introduction: Poison center utilization varies when compared by age-adjusted population geo-political area. Variation may be secondary to need, awareness of and how to access centers, or perceived barriers that prevent use. The goal of this study is to determine the importance of potential barriers to poison center utilization in a population that includes a significant Spanish speaking population. Methods: A convenience sample of 835 adults completed a 2 page, IRB approved survey. Participants were identified through healthcare facilities, community service organizations, WIC programs, a state immigration council, and community health fairs. Surveys were provided in English or Spanish, and included questions about knowledge of and barriers to using poison center services. Results: 554 adults with children < 5 years of age at home comprise the survey group; 8% reported a past poisoning experience. 50% completed Spanish surveys, 44% of participants spoke Spanish at home and 29% lived in bilingual households. English speakers were more likely (74% vs. 50%) to know how to contact a poison center; Spanish speakers were more concerned that language would be a barrier (72% vs. 27%), or that there would be a charge for services (48% vs. 33%). Bilingual speakers were more concerned that calls would not be confidential and would be reported to the authorities (76% vs. 32% vs. 24%). Spanish speakers were less likely to call about common poisoning events including a child's exposure to plants, medications/drugs, cleaning supplies, and unknown substances. Conclusion: Poison center awareness campaigns should focus on minimizing perceived barriers to public use with messages directed to minimizing barriers. In the survey area English language and Spanish language messages need to be different.

118 HOME CALLS FROM PREDOMINENTLY LATINO COMMUNITIES TO A REGIONAL POISON CENTER.

Clark RF, Phillips M, Manoguerra AS, Chan TC. *San Diego Division, California Poison Control System. University of California, San Diego, CA*

Background: Penetrance values estimate the utilization of poison centers services. Nationally, poison centers seek to maintain penetrance values of greater than 7 (7 calls per 1000 population). For a variety of reasons, penetrance values may vary greatly among geographic areas of population. We examined the relationship between ethnicity and penetrance in our population. Methods: We conducted a retrospective review of data from 1/1/2000 to 12/31/2000 from our poison center's database. Home calls to the center were evaluated by zip code, age, gender, substance, route of exposure, and outcome. This data was compared with US Census 2000 geographic and community demographic data by zip code. Comparative statistics were employed to evaluate poison center penetrance into 5 communities with predominantly Latino residents, and 5 communities with predominantly Caucasian residents, matched for population. This data was compared with aggregate county data. Results: Aggregate penetrance values for the county were 43 and 6.5 calls per 1000 population for children less than age 4 and all ages respectively. Penetrance values for the communities with predominantly Latino populations were significantly lower, ranging from 16 to 23 for children, and 3.0 to 4.9 for all ages. All values were statistically significant when compared to county data and that from the predominantly Caucasian zip codes. Conclusion: Penetrance values were significantly lower in predominantly Latino communities. Language barriers only partly explain the difference since translators are on duty at the poison center around the clock. Other reasons for this variation are being investigated.

Platform Session 5 — Monday, October 8 — 10:30 am–11:30 am
Abstracts #119–#122

119 OPEN AIR CARBON MONOXIDE POISONINGS ON HOUSEBOATS: 74 CASES FROM 1990–2000.

Baron R, McCammon JB, Radtke T, Lovecchio F, Curry SC, Ruha M. *Good Samaritan Regional Medical Center, Departments of Emergency Medicine and Medical Toxicology, Phoenix, AZ; National Institute for Occupational Safety and Health Denver Field Office; Department of the Interior, Denver, CO*

Introduction: Carbon monoxide (CO) toxicity may result in deaths following recreational use of houseboats. CO sources include gasoline-powered propulsion and generator engines. Methods: A retrospective chart reveiw was conducted to review potential CO poisoning deaths from 1990-2000 in the Glen Canyon National Recreation Area (GCNRA). Medical records were reviewed of all emergency medical park service run sheets from GCNRA, all entries into GCNRA death registry and all patients who had carboxyhemoglobin (COHB) level obtained at a local hospital. A case was defined as a person with signs and symptoms of CO poisoning with elevated COHG (>2% in non-smokers, >9% in smokers) or exhaust exposure and signs and symptoms of CO poisoning. Data were collected regarding mode of exposure and patient presentations. Results: During this period 111 cases of CO poisonings occurred, 74 (67%) of the cases involved a houseboat with 64 (58%) of the cases involving generator exhaust alone. Three patients were poisoned by propulsion engines, two patients by both engine types, and five patients from unspecified engines. Of those involving a houseboat, 37 of these 64 patients (58%) became ill during open-air exposure commonly near the stern deck. Of those with open-air exposure, seven died, 17 suffered loss of consciousness and 12 patients required water rescue. Thirty-six patients (56%) were poisoned inside the cabin with no deaths and eight losing consciousness. Conclusions: CO exposure in open air on or behind a houseboat can produce death and loss of consciousness.

120 LEAD CONTAMINATED MOONSHINE: A REPORT OF ATF ANALYZED SAMPLES.

Parramore CS, Morgan BW, Ethridge MW. *Center for Injury Control; Emory Department of Emergency Medicine, Atlanta, GA; Buruea of Alcohol, Tobacco and Firearms National Laboratory, Rockville, MD*

Background: A recent study of Emergency Department patients in Atlanta, GA, revealed a significant association between reported moonshine consumption and elevated blood lead level (BLL)($\geq$10μg/dL). However, beyond anecdotal reports and isolated case histories, lab analyses confirming the presence and extent of lead contamination among moonshine samples are absent from modern scientific literature. Methods: 115 suspected moonshine samples seized by local law enforcement between 1995 and 2001 were voluntarily submitted to the Bureau of Alcohol, Tobacco and Firearms' National Laboratory for analysis. Determination of lead was achieved using Flameless Atomic Absorption Spectrophometry (FAAS). Descriptive statistics were calculated using EPIINFO 6.0 (CDC, Atlanta, Georgia). Results: Samples originated from 9 states, including 5 southeastern states, Missouri, Ohio, Wisconsin and West Virginia. Lead levels ranged between 0.0 μg/dL and 53,200 μg/dL (median = 44.0 μg/dL). Median percent alcohol by volume was 44.75% (range: 3.85%–65.80%). 33 samples (28.7%) contained lead levels greater than 300 μg/dL. Percent alcohol by volume did not predict lead content. Conclusion: OSHA guidelines have established a BLL of concern in adults at 40 μg/dL. The EPA quantitatively estimates that dietary lead exposure of 1520 μg/day will result in a BLL of 40 μg/dL. Consuming 500cc per day of moonshine contaminated with 300μg/dL of lead could result in a BLL of approximately 40 μg/dL. 28.7% of samples could produce lead toxicity at a low to moderate level of consumption. Moonshine production and consumption is an under-appreciated toxicologic and public health concern not restricted to the Southeast US.

121 *IN-VITRO* ADSORPTION OF COPPER AND LEAD TO ACTIVATED CHARCOAL.

Traub SJ, Nelson LS, Hoffman RS. *New York City Poison Control Center, New York, NY*

Background: Although it is commonly stated that metal salts are not adsorbed to activated charcoal (AC), when studied, mercury, arsenic and thallium are adsorbed in small, but clinically significant, quantities. We began to systematically study adsorptive properties of other metals. Methods: Stock aqueous solutions of copper sulfate and lead acetate were prepared in distilled, deionized water. Norit A® AC was desiccated for 24 hours at 100°C. Known quantities of AC were added to the stock solutions in AC:metal ratios ranging from approximately 10:1 to 250:1. The mixtures were

agitated at 25°C for 15 minutes, then gravity filtered. Supernatant metal concentrations were analyzed in triplicate using atomic absorption spectrophotometry. Each experiment was run 3 times. Langmuir isotherms were plotted and best straight lines were fitted by the least squares method. Maximum adsorptive capacity (MAC) for each metal was calculated from the Langmuir equations. Results: Lines representing the Langmuir isotherms had p values <0.05 and R^2 (Pearson's correlation coefficient) values >0.98 for both metals. Copper had a MAC of 9.1 mg/g AC, and lead had a MAC of 10.3 mg/g AC. Conclusions: Many drugs and toxins adsorb to AC with MACs on the order of 100–300 mg/g AC. By comparison, these metals demonstrate relatively poor adsorption. However, the significance of MAC must be considered in the context of dose-lethality. Lethal doses of copper sulfate are reported on the order of 1-2 grams. With a MAC of 9.1 mg Cu/gm AC, 100 grams of AC would be expected to bind up to 3.6 grams of copper sulfate pentahydrate, which could be consequential. Similarly, a 20 g dose of AC could theoretically adsorb all 200 mg of lead present in a typical paint chip. Further studies in-vitro (to determine the effects of varied pH on MAC) and *in vivo* are necessary to help define the clinical implications of these results.

122 THE EFFECTS OF DMSA ON URINARY LEAD AND MERCURY EXCRETION IN HEALTHY VOLUNTEERS.

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Background: Our toxicology clinic is frequently referred asymptomatic patients with normal baseline lead/mercury concentrations with a diagnosis of possible heavy metal poisoning based on a rise in their urinary lead/mercury excretion in response to a challenge with oral DMSA. Such challenge results are uninterpretable and our aim was to establish the urine lead/mercury response to an oral DMSA challenge in healthy volunteers without heavy metal poisoning. Methods: 10 volunteers (all healthy males) had a baseline blood lead and mercury estimation and were excluded if blood lead $>$ 10μg/dL or mercury $>$ 1.5μg/dL. A 24hr urine collection was taken prior to the administration of DMSA (30mg/kg). Urine was then collected for the periods 0–8hrs, 8–24hrs and 24–48hrs after DMSA and analyzed for lead & mercury concentrations. Results: Data was collected on all 10 volunteers. Blood lead 2.55 ± 1.21 μg/dL, blood mercury 0.68 ± 0.24 μg/dL. The table below summarizes the urine lead & mercury concentrations:

	Pre DMSA	0–8hr Post DMSA	8–24hr Post DMSA	24–48hr Post DMSA
Urine Lead (μg/dL)	0.18 ± 0.33	0.87 ± 0.73	1.03 ± 0.96	0.49 ± 1.17
Urine mercury (μg/dL)	0.51 ± 0.12	0.53 ± 0.23	0.58 ± 0.21	0.50 ± 0.44

Conclusions: There is a wide inter-individual variation in the urinary lead/mercury response to a challenge with oral DMSA in healthy volunteers without heavy metal poisoning making interpretation of this test difficult in clinical practice. In healthy volunteers without lead poisoning urine lead concentration increased by a factor of up to 5.7 after a DMSA challenge and so a positive response should not be used to indicate lead poisoning.

Poster Session III — Monday, October 8 — 10:00 am–4:30 pm
Abstracts #123–#178 — Authors with Posters — 3:00 pm–4:30 pm

123 TROUBLES WITH BUBBLES: AIR GAS EMBOLUS FROM CONCENTRATED HYDROGEN PEROXIDE INGESTION.

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Background: Ingestion of one milliliter of concentrated hydrogen peroxide can liberate 116 milliliters of oxygen. This can result in the formation of cerebral air gas emboli (CAGE). Hyperbaric oxygen therapy (HBOT) is the standard of

care in the treatment of CAGE. A search of MEDLINE from 1966 to present revealed one case in which HBOT was used to treat CAGE associated with ingestion of concentrated hydrogen peroxide. We report a case of CAGE after accidental ingestion of 33% hydrogen peroxide successfully treated with HBOT. Case Report: A 48-year-old male took two sips of 33% hydrogen peroxide, mistaking it for water. Within a few minutes he vomited and was taken to a local emergency department. Shortly after arrival, he developed hematemesis, left sided hemiplegia, confusion, and right hemianopsia. Initial laboratory studies, chest x-ray, and brain CT were normal. Brain MRI revealed multiple infarcts suggestive of an embolic source. After consultation with the poison center, a recommendation was made for HBOT. Twelve hours after arrival the patient underwent HBOT at 3 atmospheres absolute (ATA) for 30-minutes and 2.5 ATA for 60-minutes with rapid clinical improvement. He was discharged 4 days later. MRI 6 months later showed only a small right occipital infarct, with resolution of the earlier noted areas of ischemia. He has resumed his job as a truck driver and farmer and has no neurological impairment. Conclusion: Ingestion of even a small amount of concentrated hydrogen peroxide can result in air gas embolism. Hyperbaric oxygen therapy can be of benefit in reversing the symptoms and preventing permanent neurological impairment.

124 SCINTIGRAPHIC DETECTION OF CARDIAC INJURY OF ACUTELY CARBON MONOXIDE POISONED PATIENTS.

Pach J, Hubalewska A, Pach D, Staszczak A, Targosz D. *Department of Clinical Toxicology, Department of Endocrinology Jagiellonian University Medical College Kraków, Poland*

Objective: Transitory ischemia or necrosis due to metabolic abnormalities caused by carbon monoxide (CO) toxicity can be seen as changes in scintigraphy scans of myocardium in acutely poisoned patients. The mechanism of cardiac injury is different than that caused coronary occlusion, and usually takes a longer time. The changes in Tc99m-MIBI scans related to necrosis, hibernating and/or stunned myocardium in all the acutely CO intoxicated patients were seen in our previous study. Tc99m-glucarate the new radiopharmaceutical agent, by binding to the nucleoprotein subfractions and to a lesser extent to the DNA fractions, localizes in necrotic tissues and is the best agent for detection of acute necrosis and differentiates it from reversible myocardial changes. The aim of the study was to evaluate the cardiac injury of acutely carbon monoxide poisoned patients using both radiopharmaceutical agents. Material and Methods: In 18 acutely CO poisoned patients (7 male, 11 female) aged from 16–39, with no cardiac history or cardiac risk factors, Tc99mGlucarate and Tc99mMIBI scintigraphy were performed at 2–5 days post admission to the Kraków Department of Clinical Toxicology. Measurement of COHb, blood lactate level, duration of exposure and ECG and echocardiography examinations were carried out on admission. The enzymes activity (ALT, AST, CPK) were evaluated after 24 hours. The scintigraphy scans were performed using Siemens two-head gamma camera equipped with parallel high-resolution collimators. The Tc99m-Glucarate (700MBq) was injected intravenously and immediately afterward planar sequential 1-minute images were acquired for the first 60 minutes and then the static image was performed. Delay static images were carried out 3 and 24 hours after tracer injection. Tc99m-MIBI scan was performed 1 day later. The changes in Tc99m MIBI scan were graded: I^0- diminished and non-homogenic uptake, II^0- diminished and small foci of tracer absence, III^0- visible diminished uptake of tracer +one bigger "cold spot", IV^0- large and numerous "cold spots". Results: No elevation in enzyme activity was noted. The echocardiography did not revealed substantial changes in any patient. The electrocardiography examination revealed tachycardia in 13 of 18 examined patients. Of the 9 patients with negative Tc99mGlucarate scans 4 had I^0 but 5 II^0 of changes in MIBI scans. The dispersed accumulation of Tc99mGlucarate were found in 5 persons (1 patient–I^0; 2pts- II^0; 2pts- III^0 in MIBI uptake scale). The typical focal accumulation was noted in 4 patients, all of them had III^0 in MIBI uptake scale. In that group of patients the mean value of COHb was 34.8%, the blood lactate level was 6.43 mm/L. Conclusions: 1. The changes in Tc99mMIBI scintigraphy scans, confirming cardiac hypoxia followed by myocardial metabolic disturbances, were found in all the CO poisoned patients 2. Tc99mGlucarate scintigraphy scans confirmed the myocardial necrosis in 4 of the patients; dispersed accumulation of Tc99mGlucarate without typical focal necrosis was found in 5, and in 9 of the patients the myocardial necrosis was excluded by Tc99mGlucarate scintigraphy examination. 3. Myocardial scintigraphy is a more sensitive method than electrocardiography, echocardiography and measurement of enzyme activity for the evaluation of CO cardiotoxicity in acute poisoning.

125 ENCEPHALOPATHY FOLLOWING CHRONIC CARBON MONOXIDE POISONING.

Meggs WJ. *East Carolina University, Greenville, NC*

Objective: Chronic neurological deficits are well known to follow acute carbon monoxide poisoning. Neurological dysfunction following chronic carbon monoxide poisoning is seldom reported. Case Report: A 53-year-old Caucasian woman moved into an apartment in January that was heated with a natural gas space heater. Within 3 weeks she developed daily headaches, fatigue, difficulty getting out of bed, lightheadedness, nausea, anorexia, diarrhea, severe forgetfulness, mental confusion, and difficulty concentrating. She could read but had difficulty remembering what she read. Difficulty with gait developed. On May 15th, carbon monoxide poisoning was suspected, and the gas turned was turned off. On May 17th, two days after the gas was turned off, a technician using a portable meter measured carbon monoxide levels of 35 to 86 ppm in her apartment and ordered her to vacate the premises. On July 20th, the patient presented to the Emergency Department with continuing symptoms of headache, difficulty with memory and concentration, and disordered gait. She reported difficulty with driving and reading. She had difficulty with job performance. Neurological examination was normal except for mild cerebella signs and unsteady gait. CT scan and MRI studies of the brain were normal. Neuropsychiatric testing two months after the exposure ended revealed mild bilateral cerebral dysfunction, with deficits in attention, sustained concentration, problem solving, categorization, visual perception and visual motor integration. Her condition gradually improved. Driving difficulties resolved, and she passed a driving evaluation four months after termination of exposure. At that time, she returned to work but had difficulty with concentration and memory. Conclusion: Neurological deficits developed in association with chronic carbon monoxide poisoning and persisted for several months after the exposure was identified and terminated.

126 EFFECT OF KETAMINE ON PHENCYCLIDINE IMMUNOASSAYS (PCPIa).

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Objective: Reported cases of positive point-of-care urine PCPIa results associated with ketamine administration motivated us to systematically evaluate the effect of ketamine on these tests. Methods: A blinded, anonymous study of patients receiving parenteral ketamine was conducted. Patients (n = 50) submitted urine prior to, and 1–2 hours after receiving parenteral ketamine. Gas chromatography/mass spectrometry was performed on all urine samples to detect PCP, ketamine and ketamine metabolites. Urine was also tested with 5 point-of-care urine PCPIa to determine if ketamine administration caused a change in test result from negative pre-treatment to positive post-treatment. These PCPIa were devices by Forefront Diagnostics (Laguna Hills, CA), Princeton BioMeditech (Princeton, NJ), Roche Diagnostic Systems (Sommerville, NJ), American BioMedica (Kinderhook, NY), and Technical Chemicals and Products (Pompano Beach, FL). Results: No urine contained PCP, no pre-treatment sample contained ketamine or metabolites and all post-treatment samples contained ketamine and metabolites. No urine was PCPIa positive. Several assays appeared to have a weak partial reaction with post-treatment urine, and determination of a negative result was possible only after careful inspection of these test cassettes. Conclusions: Ketamine and metabolites are present in urine 1–2 hours after parenteral ketamine administration. In a previous report we note that these urine PCPIa performed after ketamine administration are negative. However, several assays were difficult to interpret: Reaction of ketamine or ketamine metabolites with these point-of-care urine PCPIa may cause ambiguous results leading to a false-positive interpretation.

127 ACUTE PARAPHENYLENEDIAMINE POISONING: MOROCCAN CASE.

Filali ZA, Soulaymani BR. *Morocco Poison Center, Institut National d'Hygiène, Agdal, Rabat, Morocco*

Introduction: Though uncommon in the west, ingestion of Paraphenylenediamine (PPD) is frequently reported from Africa and Asia, where it is commonly mixed with Henna (leaves of *Lawsonia alba*) and applied to colour hands and feet and to dye hair a dark red shade. PPD accelerates the dyeing process. Acute poisoning by accidental or intentional ingestion of PPD causes severe oedema of the face and neck frequently requiring emergency tracheotomy. This is followed by rhabdomyolysis and acute renal failure, culminating in death if not treated aggressively. Method: We report here a retrospective review of all reports received in the Moroccan Poison Center from 1992 to 1999, concerning PPD poisoning. Data analysis included reason of exposure, sex, age and evolution. Results: There were 175 PPD poisoning, 132 females (75.4%) and 43 males (24.6%). The ingestion was intentional in 139 cases (79.4%) and accidental in 36

(20.6%). The intoxication culminated to death in 48 cases (27.4%) and the evolution was favourable in 127 (72.6%). 25 cases (14.3%) concerned children less than 15 years old, 110 cases (62.9%) was between 15 and 30 years old, and 40 cases was more than 30 years old. Conclusion: Unhappy Moroccans, young females especially, have come to recognise the potential toxicity of PPD (The PPD poisoning was fatal in our study in 27.4% of cases) and have been ingesting it with suicidal intent. Stricter controls over the sale and distribution of PPD in Morocco should go some way towards alleviating this problem. Extra awareness of the clinical features is required in the western countries where, though not a common problem as yet, more cases may appear as a result of increasing migration of relevant ethnic groups to western cultures.

128 PEDIATRIC NICOTINE EXPOSURES FROM INGESTION OF SPITTOON CONTENTS.

Bryden PA McKnight RH, Spiller HA. *College of Medicine, University of Kentucky, Lexington, KY and Kentucky Regional Poison Center, Louisville, KY*

Background: This study describes the epidemiology of spittoon-related pediatric nicotine ingestions reported to a poison center for a 10-year span. Methods: Exposure reports received by a statewide regional poison center provided data for this retrospective study. First, exposures to children ≤ 48 months occurring from January 1, 1990 through December 31, 1999 were searched to identify reports in which a broad list of nicotine and tobacco-related substances had been implicated as the toxic agent. Next, each nicotine record was visually inspected to identify only those exposures where the child had reportedly ingested the expectorant of smokeless tobacco. Results: 104 ingestions of smokeless tobacco expectorant were identified. Spittoons included various containers, such as cups, soft drink cans and bottles, glasses, jars, and other receptacles. Ages ranged from 6 to 48 months (mean = 18.4 months). Almost two-thirds (64%, n = 67) occurred in males (mean age = 18.3 months), while 36% (n = 37) occurred in females (mean age = 18.7 months). For both genders combined, 63.3% were reported symptomatic, with vomiting the most frequent (n = 47), followed by lethargy (n = 12). 98% of exposures occurred in a residence or home. 66.3% (n = 69) of exposures were successfully managed outside of a health care facility, while 29.8% (n = 31) were seen in a hospital emergency department. Three cases required hospitalization. Conclusions: Although pediatric nicotine poisonings from spittoons are rare, prevention of these household exposures will require both education and engineering strategies. Pediatricians and parents should become aware of the hazards of smokeless tobacco expectorant ingestion. Cessation of smokeless tobacco use should be encouraged. Receptacles used as spittoons must be kept out of the reach of children during use, storage or disposal and should not be containers associated with food or drink which are very tempting to an inquisitive child. Spittoons should be engineered to eliminate access to their potentially toxic contents.

129 AN INTENTIONAL TOBACCO EXTRACT INGESTION: A CASE AND THE INFLUENCE OF pH.

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Introductions: The symptoms are severer and the onset is much more rapid after the ingestion of liquid nicotine such as insecticides or tobacco extracts compared with fresh cigarettes and butts. However we sometimes experience the tobacco extracts ingestion cases without any symptoms. To evaluate the toxicities of tobacco extracts, we analyzed the nicotine concentrations in patient's samples and also investigated the acute effects between tobacco extracts and other nicotine solutions. Methods: Animals; Male Sprague-Dawley rats (6 weeks old). Agents; tobacco extracts, nicotine sulfate and nicotine solutions. Dose; 90,135 and 180 mg/kg. The nicotine concentrations for samples were determined using HPLC. Case Report: A 47-year-old man ingested about 500 mL of tobacco extracts (estimated amount of nicotine; 500mg). On the arrival at the emergency room, his level of consciousness was almost clear. Heart rate, 88 beats per minute; blood pressure, 132/70 mmHg; pupil size, about 3 mm. Physical examination was normal except emesis and sialorrhea. He was treated supportively and discharged next day. Nicotine concentration of initial gastric contents and the patient's serum were 1.25mg/mL and 79.54ng/mL respectively. Results: The pH of tobacco extracts, nicotine sulfate and nicotine solutions were 4.5, 4.0, and 9.5, respectively. Time to death values and the onset of toxic effects in rats were significantly longer for the tobacco extracts and nicotine sulfates than for nicotine groups. Conclusion: The

results suggest that onset of toxic effects are slower for cigarette ingestion cases, because of the influence of pH for absorption.

130 THE EXTRACTION RATES OF NICOTINE FROM CIGARETTES IN SEVERAL BEVERAGES.

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Introduction: Accidental cigarette poisoning occurs often in children who ingest cigarettes, butts and tobacco extracts in an ashtray or a can used for an ashtray. To evaluate the toxicity in the cases of accidental tobacco extracts ingestion, we analyzed the extraction rates of nicotine from cigarettes in several beverages. Methods: A whole cigarette was placed in the test solutions and an aliquot was sampled at regular time intervals. After the pH measurement of the samples, they were filtrated through cellulose acetate filters (pore size: 0.45 μm). Then the filtrate was diluted with milliQ water and 20μl was injected into the ion-pair reverse phase HPLC with UV detection (262 nm). [Test solutions] milliQ water, milk, beer and 9 soft drinks. Results: The following table lists the values we obtained. Conclusion: Nicotine was extracted from cigarettes completely at 60 min in most test solutions except milk. Although the range of original pH

Test solutions	Samples	Original pH	Final pH	Extraction rates at 60 min (%)
milliQ water	control	6.50	5.63	100.1
Soft drinks	A	2.44	4.55	108.0
	B	3.03	4.41	97.5
	C	3.46	4.26	99.4
	D	3.53	4.07	105.0
	E	6.19	5.54	97.7
	F	6.38	5.48	90.2
	G	6.88	6.09	103.5
	H	6.54	6.1	110.0
	I	6.88	5.74	101.5
Milk	milk	6.69	6.49	67.7
Beer	beer	4.39	4.93	110.0

for soft drinks (2.44 ~ 6.88) was wide, all final pH values were gathered around 4.5 ~6.0. The results suggest that the original pH of beverages have almost no influence on absorption of nicotine in the tobacco extracts ingestion.

131 CHRONIC OCCUPATIONAL EXPOSURE TO AROMATIC HYDROCARBON GASES ASSOCIATED WITH DYSMENORHEA, DYSFUNCTIONAL UTERINE BLEEDING AND CNS EFFECTS.

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

Background: Limited information is available on effects to menstrual cycles and sex hormones from chronic exposure to aromatic hydrocarbon gases. Published studies involving toluene, benzene and xylene in the manufacturing setting, with higher concentrations, have reported an increase in dysmenorhea and dysfunctional uterine bleeding. Case Report: Five females worked in a small convenience store (600 Sq ft total) for 1 to 5 years. After complaints of a "chemical" odor that had persisted for more than one year, investigation showed air samples within the store of benzene 1.8 ppm, toluene 8340 mcg/m^3 (2.2 ppm), total xylene 4590 mcg/m^3 (1.1 ppm), and ethylbenzene 835 mcg/m^3. The site investigation began after one of the five patients was diagnosed with leukemia. All five women complained of new onset or significantly increased dysmenorhea and associated low back pain. Additional symptoms included increased number of cycles per month in two patients and increased duration of flow in five patients. One 23-year-old patient experienced prominent facial and neck flushing and "hot flashes" associated with her shifts in the store. All five complained of headaches, dizziness and reduced level of concentration when at the site. Preliminary investigation suggests venting from contamination of the sewer from an upstream site. Site investigation continues. Conclusion: The case report suggests that chronic exposure to low to moderate levels of aromatic hydrocarbons may effect menstrual cycles. Further research may be warranted.

132 EFFECTS OF RESPIRATORY ACIDOSIS AND ALKALOSIS ON THE DISTRIBUTION OF CYANIDE INTO THE RAT BRAIN.

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The question to be addressed is whether respiratory acidosis favors the cerebral distribution of cyanide, and conversely, if respiratory alkalosis limits its distribution. The pharmacokinetics of a non-toxic dose of cyanide were first studied in a group of 7 rats in order to determine the distribution phase. The pharmacokinetics were found to best fit a three-compartment model with very rapid distribution ($T_{1/2\,\alpha} = 21.6 \pm 3.3$ s). Then, the effects of the modulation of arterial pH on the distribution of a non-toxic dose of intravenously-administered cyanide into the brain of rats were studied by means of the determination of the permeability-area product (PA). The modulation of arterial blood pH was performed by variation of $PaCO_2$ in 3 groups of 8 anaesthetized mechanically-ventilated rats. The mean arterial pH measured 20 min after the start of mechanical ventilation in the acidotic, physiologic, and alkalotic groups were 7.07 ± 0.03, 7.41 ± 0.01, and 7.58 ± 0.01, respectively. The mean PAs in the acidotic, physiologic, and alkalotic groups, determined 30 s after the intravenous administration of cyanide, were 0.015 ± 0.002, 0.011 ± 0.001, and 0.008 ± 0.001 s^{-1}, respectively (one-way ANOVA $p < 0.0087$). At alkalotic pH the mean permeability-area product was 43% of that measured at acidotic pH. This effect of pH on the rapidity of cyanide distribution does not appear to be limited to specific areas of the brain. We conclude that modulation of arterial pH by altering $PaCO_2$ may induce significant effects on the brain uptake of cyanide.

133 A LIGHT-WEIGHT, LEVEL B, PROTECTIVE GARMENT WITH AIR PURIFYING RESPIRATOR HOOD FOR USE BY EMERGENCY PHYSICIANS.

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Background: In the event of a terrorist attack or a hazardous materials incident involving chemical agents, emergency physicians and staff personnel may need personal protective equipment (PPE), including respiratory protective equipment to safely attend to contaminated patients or to work in a contaminated environment. To date, no comfortable, practical, and economical solution for PPE has been developed for use by emergency physicians. Methods: Six (6) healthy, adult, volunteer emergency medicine residents were instructed in the use of a disposable Tyvek F, level B, protective suit and a butyl rubber air purifying respirator hood. They donned and wore this equipment under conditions which simulated customary working conditions in an emergency department for five (5) hours. This included a one (1) hour period of light exercise. At the end of five (5) hours the volunteers completed a questionnaire regarding the comfort and wearability of this PPE ensemble. Results: All volunteers completed the entire study period. No adverse effects of wearing this PPE were noted. All volunteers indicted that they could comfortably work in the Tyvek suit for at least five (5) hours and the respiratory hood for at least two (2) hours under working conditions in an emergency department. Conclusions: The use of a Tyvek F protective suit and butyl rubber air purifying respirator hood is a feasible solution when emergency physicians need to use PPE while working.

134 ANTHRAX: A BIOLOGIC OR PSYCHOLOGIC THREAT?

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Background: Last year in Florida, there were over 35 anthrax threats reported to the F.B.I. The purpose of this paper was to evaluate the psychological impact on the victims in 2 separate cases. Methods: Case A (family run business n = 13) and Case B (collection agency employees n = 4) were both decontaminated prehospital by HAZMAT; interviewed by the F.B.I/Health Dept., transferred to the E.D., and discharged after medical clearance. Once the cases were deemed hoaxes, all the victims completed a 92 'Yes/No' survey. It included questions screening for symptoms of post-traumatic stress. Pt's demographic information and proximity to the letter/powder were also recorded. Comparison of proportions used Chi squared with continuity correction or Fisher Exact tests ($\alpha = 0.05$). Results: Pt's initial responses to the threat were: "fearful for loved ones" (94.1%), "in disbelief" (88.2%), "felt helpless" (88.2%), and had "recurrent thoughts of the event" (82.4%). Once the hoax was declared, "recurrent thoughts" and "having difficulty making decisions" dropped (82.4% to 41.2% and 29.4 to 0%), respectively. Comparing sexes, females responded 'Yes' to 19 more symp-

toms. Comparing cases, Case B responded 'Yes' to 25 more symptoms than Case A. These included "difficulty going back to the building (75%), and felt "this experience has changed me in some way" (100%). Victims who were in close proximity of the letter/powder were more likely to "feel depressed/sad" (66.7%). Victims who met with a 'victim-witness counselor "felt the meeting helped" (87.5%). Conclusions: To date, in the U.S., all anthrax threats have been hoaxes. For the victims, however, the event is real and can lead to post-traumatic stress symptoms. Screening for, and counseling of these symptoms should become part of a hospital's biological disaster plan.

135 BLOOD LEAD LEVELS AMONG CHILDREN IN YAP STATE, FSM.

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Background: Following a January 2000 nutritional survey on Yap identifying elevated blood lead levels [BLLs] among some children, we performed a study to better characterize pediatric BLLs and identify associated risk factors. Methods: We collected blood samples from children at randomly selected schools and municipality sites. We obtained demographic and lead exposure information from parents by self-administered questionnaires. In a concurrent nested case-control study, parents of cases (BLL $\geq$ 15 micrograms/deciliter [mcg/dL]) and age-matched controls (BLL $<$ 5 mcg/dL) completed interviewer-administered questionnaires; environmental sampling was done at participants' homes. Results: The geometric mean [GM] of the BLLs for the 424 participants was 4.6 mcg/dL. While the GM BLLs of Native Yapese children and resident non-Micronesians were comparable (4.3 and 4.2 mcg/dL), Outer Island children had significantly higher levels (8.1 mcg/dL, p $\leq$0.05). Children living in neighborhoods where lead was recycled had significantly higher GM BLLs than those who did not (7.2 vs. 4.6, p 0.05). Children whose families made lead fishing sinkers at home also had higher GM BLLs (5.7 vs. 4.5, p = 0.06). Case children's homes were more likely than control homes to have detectable levels of lead in soil (odds ratio [OR] = 7.4, 95% Confidence Interval [CI] = 2.0–27.8) and household dust (OR = 11, CI = 2.1–56.7). Conclusions: The GM BLL of children surveyed on Yap exceeds that of same-aged US children as measured in National Health and Nutrition Examination Survey III, and Outer Island ethnicity was significantly associated with having an increased BLL. Local environmental contamination due to handling lead is a likely source of elevated BLLs.

136 EPIDEMIOLOGY OF ACUTE POISONINGS IN VIET NAM.

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Introduction: In Viet Nam a wide variety of chemical agents are used in agriculture, industry, and other fields. Over the last 4 years we have collected epidemiologic data on poisonings. Methods: Data from 1996 to 2000 was collected from 48 of the 61 provincial hospitals. The number of reported poisonings is stable, averaging 9.052 ($\pm$ 411)/year. The mortality from poisoning has been stable, at 3.3%/year. The following table describes the data from our 3 most common poisonings:

	Patients (%)	Death (%)
Agricultural Chemicals	5188 (24.8)	343 (6.6)
Sedatives	3714 (17.8)	32 (0.9)
Foods	3695 (17.7)	9 (0.2)

Agricultural poisonings were primarily from organophosphates, although paraquat contributed substantially in the South. Most of the sedative poisonings were from suicide attempts. Although food poisoning was primarily from bacterial agents, approximately 10% of these came from contamination with chemical agents and 17% from ingestion of poisonous plants, mushroom, snake venom, and fish gall (fresh water fish gallbladder or bile used as a traditional remedy). There has been a recent increase in the incidence of poisonings and death due to a new rodenticide from China. Our national laboratory has established its identity as trifluoracetamide. Recently there has been increased number of poisonings by CS in areas where CS containers were left behind by the U.S. Military. 92% of snake poisonings were from cobras, 8% were from viperidae. Conclusion: There are a significant number of serious poisonings in Viet Nam and the mortality

is high. The recent establishment of a central poison control institute is intended to collect epidemiologic data and to impact on the care of these poisonings.

137 HUMAN PESTICIDE EXPOSURES: TRENDS AND INFORMATIONAL NEEDS.

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Background: Pesticide exposures represent a consistent source of calls to poison control centers. The purpose of this investigation is to understand public information needs and health care utilization trends through an analysis of data from a national telecommunications-based medical monitoring program providing clinical pesticide-related information to all callers. Method: A retrospective analysis of data collected between 1996–2000 was conducted. Cases were referred from many sources (including physicians and state agency programs). Exposure information was collected from all cases, and reviewed by a medical toxicologist. Results: 2,834 cases were referred between 1996–2000. 60% of incident cases were acute exposures, 40% were chronic. The most common active ingredients implicated in cases of definite or probable pesticide-related illness were pyrethrins and pyrethroids (39%), organophosphates (28%), and antimicrobial products (14%). In the majority of exposure cases (54%), including those categorized as definitely or probably related to pesticides (n = 351), the site of exposure was the home. 17% of these incidents involved children. In 21% of exposure incidents, the relationship between the illness and the reported pesticide exposure was determined to be unlikely. Individuals with chronic exposures were more likely to see a physician (79%) or alternative health care provider (11%) than those with acute exposures (69% and 2%, X^2 36.3 and 97.9 respectively, $p < .001$). Conclusions: Unintentional household exposures to general use pesticides accounted for the majority of incident cases, including those involving children. Although many cases of illness were unlikely to be pesticide-related, there was a consistent need for health information and health care utilization among individuals with concerns about both acute and chronic exposures. Further research is needed to determine the proximate cause of household pesticide exposures.

138 RELATIVE MEDICAL OUTCOME FROM CHILD INGESTION OF ORGANOPHOSPHATE AND CARBAMATE INSECTICIDES IN U.S. POISON CENTER DATA, 1993–98 ACCORDING TO TOXICITY CATEGORIES BASED ON ANIMAL STUDIES.

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Objective: Determine the relative likelihood of symptomatic, moderate, and major/fatal outcome among children <6 orally exposed to cholinesterase-inhibiting insecticides according to toxicity categories based on animal studies. Methods: Oral LD_{50} values were obtained for formulated insecticides based on 15 organophosphate and carbamate active ingredients from a peer-reviewed database of pesticide toxicity studies. Pesticide data on childhood exposures (under 6 years) was obtained from the American Association of Poison Control Centers for the years 1993 through 1998. Proportion of symptomatic cases was based on those reporting known medical outcome, categorized as minor, moderate, major, or fatal. Proportions were also calculated for moderate or more serious outcome and for major (life-threatening) or fatal outcome. Results: Relative likelihood of symptoms, moderate or more severe outcome, and major or fatal outcome increased, as expected, with increasing toxicity as shown in the table below:

LD_{50} (mg/kg)	*N*	*Symptomatic*	*Moderate or greater*	*Major/Fatal*
0–500*	630	17.9%	3.17%	0.635%
501–1500*	410	18.0%	4.63%	0.732%
1501–5000	1061	12.0%	1.88%	0.283%
> 5000	2336	10.2%	1.16%	0.214%

* categories requiring child-resistant packaging

Conclusions: Children would receive additional protection if pesticides with an acute oral toxicity of 1501–5000 mg/kg were placed in child-resistant packaging.

139 FREQUENCY OF EARLY COMPLICATIONS OF ACUTE POISONINGS BY ACETIC ACID.

Sarmanaev SKh, Yamanaeva IE. *Toxicological Center, Hospital #21, Ufa, Russia*

Background: Acute peroral poisonings by corrosive agents and by acetic acid (AA), in particular, are severe poisonings. The structure of complications in cases of AA poisonings is insufficiently studied. Methods: We retrospectively analyzed 279 medical cards of adult patients with acute AA poisonings admitted in the Toxicology Center (Ufa) during 6 years. The mean dose is 136.5 ± 13.1 (vinegar), and 53.6 ± 3.2 mL of AA (70%). Mortality for vinegar is 1.1%, and for AA–13.4%. The statistical comparison was calculated by means of Fisher's angular transformation. Results: Complications developed in 62.1% of cases of poisonings by vinegar, in 91.1% of cases of 70% AA poisonings.

Complications	Vinegar, frequency (%)	70% acetic acid, frequency (%)
1. Chemical burn of GI	53.5	88.8
2. GI bleeding	1.2	11.7
3. Perforation of GI	0	0.6
4. Shock	1.2	7.8
5. Coma	0	0.6
6. Nephropathy	2.3	11.1
7. Acute kidney failure	0	7.3
8. Hemolysis	13.8	39.4
9. Coagulopathy	21.9	39.1
10. Others	6.4	19.3

Conclusions: In cases of AA poisoning during the first three days the following types of complication develop most frequently: chemical burn of GI, hemolysis, coagulopathy. The frequency of complication development depends on the concentration of the AA (statistically significant; $p < 0.01$).

140 STRUCTURE OF ACUTE POISONING BY CORROSIVE AGENTS (APCA).

Sarmanaev SKh, Yamanaeva IE, Aydarova LF, Vafina GM. *Hospital #21, Ufa, Russia*

Background: APCA take up one of major places in the structure of poisonings. Nonetheless, the structure of APCA in terms of individual nosologies are still poorly studied. Methods: A retrospective chart review of adult patients admitted to our referral center with APCA during the past 5 years was performed. We identified 701 patients. We studied the following epidemiological parameters: number of hospitalizations, mortality, length of hospitalization. Results:

Corrosives	Cases, %	Fatality, %	Length of hospital stay (days ± SD)
Acetic acid (AA)	39.09	12.7	8.86 ± 0.48
Hydrochloric acid	4.7	15.0	9.25 ± 0.48
Sulfur acid	7.13	0	10.20 ± 1.09
Bleach	20.99	1.3	4.36 ± 0.48
Others acids	0.99	0	8.14 ± 1.06
Sol. ammonia	11.13	1.5	7.97 ± 0.55
Other alkalis	2.71	0	14.80 ± 2.98
$KMnO_4$	2.43	0	4.67 ± 1.67
Unknown	9.7	18.9	7.05 ± 0.61
All APCA	100	8.13	7.70 ± 0.53

Conclusions: 1. Acute AA poisonings require special attention, since they take the first place in APCA structure of morbidity and have high mortality (the 3rd place). 2. Poisoning by bleaches are less dangerous, since the hospitalization length in these cases is the least among the APCA poisonings and has a statistically significant difference ($t = 4.9$; $p < 0.001$).

141 AN OVERVIEW OF MALICIOUS POISONINGS IN ANIMALS (1999–2000).

Hansen S, Murphy L, Khan S, Allen C. *ASPCA Animal Poison Control Center, Urbana, IL*

Objective: Alleged malicious animal poisonings are reported to poison control centers. The purpose of this retrospective case review was to characterize these incidents. Methods: Trend analysis was conducted on animal case data collected from Jan. 1999 through Dec. 2000 where the exposure intent was considered to be malicious. Results: 192 cases (0.20% of total cases) involving 249 animals were identified in which the poison center veterinarian or caller suspected malicious intent. Initial callers were the animal owner (55%) or attending veterinarian (29%). Species included 76% dogs and 16% cats. Of the dogs, common breeds (incl. predominate mix) were German Shepherd (15%), Labrador Retriever (12%), and Rottweiler (6%). The agent source was reported as unknown (30%), neighbor (27%), and owner (23%). Route of exposure was reported as oral or oral/dermal in 68% of the cases. Exposure locations were yard/garden (41%), home (31%), unknown (16%) and other (13%). Agent categories included pesticides (37%), car/home products (21%) and pharmaceuticals (17%). Final case outcomes were unknown (60%), died/euthanized (13%), and recovered (19%). Conclusion: Alleged malicious animal poisonings represent 0.2% of cases managed at this center. Dogs, especially German Shepherds, Laborador Retreivers and Rottweilers are frequently involved. The most common source of the agent is either unknown, the animal owner or the neighbor. Most agents are ingested in the yard or garden. Agents suspected to be used in malicious poisonings include pesticides, pharmaceuticals and car/home care products. Of the known outcomes, 13% of animals die or are euthanized.

142 PHARMACEUTICAL TERRORISM; A NATURAL EXPERIMENT INFLUENCING ANALGESIC POISONING.

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Background: Acetaminophen is commonly used in deliberate and accidental poisonings. Few studies have evaluated the impact of acetaminophen availability on the incidence of acetaminophen and non-acetaminophen poisoning. Methods: Following reports of extortion in Australia during 2000, there were two recall periods of acetaminophen-containing products. We therefore conducted a retrospective observational audit of calls to a national Poison Information Centre (PIC) and presentations to a regional toxicology service. We obtained the numbers of deliberate and accidental poisonings with tablet formulations of acetaminophen, ibuprofen and aspirin products, during the recall periods, and compared them with the number of cases during the same periods of the previous three years. Results: There was a trend towards a reduction in deliberate self poisoning with acetaminophen in recall 1 ($P = 0.057$) but no decrease overall, in calls to the PIC. There was no significant change in aspirin overdoses but a significant increase in ibuprofen overdoses ($P = 0.001$). There was a significant increase in aspirin overdoses for the toxicology service ($P = 0.043$). For accidental ingestions there was a significant increase in ibuprofen ingestion ($P = 0.001$), but no significant change in acetaminophen or aspirin ingestions. Conclusions: Reduced acetaminophen availability increased the use of alternate medications for deliberate and accidental poisonings. This may be deleterious because of the increased toxicity of these agents.

143 CHRONIC ILLNESS AND THE RISK OF SUICIDE IN THE ELDERLY.

Juurlink D, Herrmann N, Szalai J, Redelmeier D. *Sunnybrook and Women's College Health Sciences Centre, University of Toronto, Toronto, Canada*

Background: With age comes an increasing burden of physical illness and an increased risk of completed suicide. We evaluated whether selected chronic medical and psychiatric illnesses increase the risk for suicide in elderly residents of Ontario, Canada. Methods: We used provincial coroner's data to identify consecutive suicides occurring between January 1,1992 and December 31, 1998 in Ontario residents 65 years and older. Age- and gender-matched controls (1:1) were randomly selected from the general population. We analyzed prescription records for each individual to determine the presence or absence of nineteen illnesses suspected to increase the risk of suicide, as well as three control illnesses with no anticipation association. Results: During the 7 year study interval, 1197 elderly suicides were identified. The most common mechanism of suicide was firearm (38%), followed by hanging (23%) and self-poisoning (22%). Twelve illnesses were found to be associated with an increased risk for suicide in the univariate analysis ($p < 0.05$ for each). Adjustment for multiple illnesses and socioeconomic status confirmed a significantly increased risk for suicide with congestive heart failure (OR 1.82; 95% CI 1.17–2.82), chronic lung disease (OR 2.12; 95% CI 1.46–3.07), seizure

disorder (OR 3.78; 95% CI 1.22 to 11.8), depression (OR 3.55; 95% CI 2.40 to 5.26), anxiety (OR 5.58; 95% CI 4.11 to 7.59), bipolar disorder (OR 9.59; 95% CI 1.20 to 77.0), and malignant pain (OR 28.1; 95% CI 3.58 to 221.2). Within the cohort, individuals with multiple illnesses were 3.3 times as likely to commit suicide than those with a single illness, who themselves were 4.0 times as likely to commit suicide than those with no illness ($p < 0.0001$ for each). Conclusion: Several chronic medical and psychiatric illnesses are associated with a large increased risk of suicide in the elderly.

144 CAUSE OF DEATH: DO CORONERS AGREE WITH TOXICOLOGISTS?

Townsend J, Ekleberry S, Wolowich B, Casavant M. *Central Ohio Poison Center, Children's Hospital, Columbus, OH*

Objective: To evaluate the consistency of the coroner's report and the toxicologist's review when determining the cause of death in relation to a known exposure. Methods: All regional poison center cases were reviewed from 1998, 1999 and 2000 where death was the outcome. We included both accidental and intentional exposures. We excluded all cases for which we did not have a completed coroner report. Results: 42 cases met the inclusion criteria. Subject age ranged from 2 months to 80 years. Of the 42 cases, only 13 coroner's reports listed the specific toxicological agent as the primary cause of death. An additional 14 coroner's reports listed the specific toxicological agent as a secondary cause of death. The remaining 15 coroner's reports completely omitted the toxicological agent as a cause of death. A toxicologist reviewed the same 42 cases. In the 27 cases that listed the specific agent as primary or secondary on the coroner's report, the toxicologist agreed with the coroner that the specific agent was likely to be the primary cause of death. Of the 15 cases that omitted the specific agent from the coroner's report, the toxicologist found that in 4 of those cases, the specific agent was likely to be the primary cause of death. In the remaining 11 cases, both the toxicologist and coroner agreed the deaths were unrelated to the toxicological exposure. Conclusion: Our study suggests that while the coroner is very specific in listing the organ system that he believes responsible for the death, he will, at times, not link that organ system failure to the substance to which the patient was exposed. In our review, potentially 4 (9.5%) cases were misreported.

145 POISON CENTER DATA: PEER REVIEW.

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Background: The editorial board of the *Journal of Toxicology–Clinical Toxicology* receives multiple comments from authors concerning a perceived resistance to the publication of reports from Poison Control Centers (PCC). Given the potential importance of such data, analysis of the rejected and published reports seems indicated. Method: Retrospective review of all manuscripts originating from US PCC, 1997–2000 concerning source, category (case series, therapeutic protocols; policies; quality control; finance); year submitted; peer reviewer affiliation (ER or PCC); reasons for rejection. Results: Of the 52 PCC manuscripts submitted 1997–2000, 23 or 44% were published, compared with the publication of 53% of the 768 total manuscripts. Publication of PCC reports did increase from 37% in 1997–1998 to 61% in 1999–2000 associated with a decrease in the number of PCC reports submitted. The publication rate did not vary with data source-regional center; multi-center; or national. No judgmental bias could be related to the reviewers' medical specialty or primary affiliation (ER or PCC). The primary reasons for rejection were (1) the "Pollyanna Phenomenon" (Hamiltion RJ, Goldfrank LR. JTCT 1997;37:21–23), meaning that the low dose, unverified exposures seen by PCC provide only limited information concerning the risk of high doses (2) study design, concept, methodology, (3) duplicate or inadequate studies. A list of reviewers' red flags is intended to assist authors in manuscript preparation. Conclusions: Careful attention and critical analysis of the limitations of PCC data can result in reports judged meaningful by peer review.

146 APPRAISING INFORMATION DIFFUSION.

McMullin N, Robertson WO. *Washington Poison Center, Seattle, WA*

Background: Clearly the information explosion is only getting more intense. At mid-century, the American Chemical Society's registry documented some 1.2 million chemical entities since the origins of mankind. Over the next 50 years, that number saw an exponential increase to greater than 19 million at the Millennium. How well are Poison Center staffs keeping up? We've initiated inquiries to find out. Method: Every Monday, the Washington Poison Center holds a teleconference for its distantly located Associate Medical Directors; it's also recorded for staff review. The prior week's unusual cases are discussed; new local happenings are identified via newspaper clippings and some 12–15 national news items and journal articles are highlighted–and then attached to the dictated minutes of the session for personal review. After discussion with the entire staff, we prepared a 20-item multiple choice memory-based examination

for all staff to complete–including the administrative secretary—based on a pseudo-random item selection from some 60 topical reprints distributed over a 3-month period; we pre-tested the questions for clarity only. Results: Of the 35 total full- and part-time staff members, all except four completed the questionnaire. Interestingly, the mean percent of correct answers was 67%; and there was no evidence of the usually expected normal distribution curve. Overall, the staff seemed very enthusiastic about the exercise and were invigorated to hear that the administrative secretary actually had the third highest score! Conclusion: This ''exercise'', though intended to be provocative, seemed to stimulate group collegiality. Moreover, it documented that ''memory exams'' are most certainly relics of the past—because of information overload. No one would dream of tolerating a 30–35% error rate in medicine today; the aviation industry aspires to less than 0.1%. Our philosophic paradigm remains constant; access to answers rather than memory retention of information is the wave of the future.

147 INFLUENCE OF STAFF EXPERIENCE AND WRITTEN GUIDELINES ON POISON CENTER SEND-IN RATES.

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Background: In a previous survey, it was determined that the main factors influencing the percent of patients sent to health care facilities for treatment or observation were AAPCC certification, adequate staffing, and utilization of licensed professionals including pharmacists in the staffing mix. Two additional factors thought to affect send-in rates are the amount of experience working in the poison center and the extent of send-in guidelines. Methods: To verify this premise, all certified and non-certified poison centers were surveyed to compare human exposure call volume, send-in rates, years of SPI experience and number of substances included in send-in guidelines. Results: 20 poison centers responded to the survey. The average send-in rate was 11.5% (range 4.2% to 39.4%). Sites with larger numbers of human exposures tended to have lower send-in rates. The center with 5006 human exposures had a send-in rate of 39.4%, while the system with 220,965 human exposures had a send-in rate of 6.4%. Total months of experience per center ranged from 220.2 to 4298.5. Neither total months of work experience nor average months of experience per FTE showed an obvious correlation to the send-in rate. As the number of substances included in send-in guidelines increased, there was a trend towards decreasing send-in rates. The center with the highest number of substances (125) in their send-in guidelines had the lowest send-in rate (4.2%). Conclusion: Although months of staff experience did not affect send-in rates, larger numbers of exposures managed per FTE and higher numbers of substances included in the send-in guidelines did result in lower poison center send-in rates.

148 AN IMPORTANT BUT OVERLOOKED POISON CENTER RESPONSIBILITY: SURVEILLANCE OF DRUG INDENTIFICATION CALLS.

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

Background: Drug identification calls from the lay public are a source of annoyance to many poison centers. Although these calls may be an exaggerated extension of the poison center primary mission and tax personnel resources, they also provide valuable information about abused and diverted pharmaceuticals. Through daily toxicosurveillance our poison center identified a significant problem in advance of it being recognized by local law enforcement and the drug diversion task force. Methods: Daily quality audits of poison center exposures do not include routinely the review of drug identification requests. At our center a single person reviews every chart, every day which allows the recognition of drug identification trends that may otherwise go unrecognized. Results: On February 7, 2001 the poison center received its first request for the identification of a tablet with the imprint of M360 which was identified as a generic product that contained hydrocodone 7.5 mg/acetaminophen 750 mg (Mallinckrodt). In the first 21 days after the trend was identified, there were 78 identification requests and over two months the poison center received a total of 222 requests for M360 identification. This represented 5.9% of the 3,787 drug identification requests over the same period. Law enforcement officials were notified when the trend was identified and briefed weekly thereafter. This allowed drug intelligence to determine how this legal pharmaceutical was being diverted for illicit use. Conclusions: The poison center provides a valuable service by responding to requests for drug identification. Realtime surveillance of these calls can provide important information about drug abuse patterns within a poison center service region.

149 POISON CENTER–HOSPITAL CONSULTATION RATES FOR ACUTE POISONINGS.

Martin TG, Naef R, Robertson WO. *Washington Poison Center, Seattle, WA*

Background: Since most patients with moderate, major or fatal outcomes are hospitalized, PC hospital consultation rates (HCR) indicate the degree of a PC's utilization in serious poisonings (PO). The purpose of this study was to estimate PC HCR from easily obtainable data and the impact of hospital bed size and distance. Methods: After IRB approval, each hospital's number of poisoned patients was estimated by a computerized search of a statewide hospital discharge database for discharge code(s) (ICD9-CM) indicating acute PO. HCR was estimated by dividing the PC consultations (tabulated from the PC's annual report) by hospitalized PO for each hospital for each yr. from 1994–6 and within distance and bed size strata. Overall HCR (total hospital PC consults/total hospitalized PO) was calculated for each yr. and 3 yr. combined. HCR > 100% were set at 100%. Differences between strata means were analyzed by ANOVA with $p < 0.05$ (*). Results: Mean ± SD HCR for the 87 hospitals for the 3 consecutive yr. were 58 ± 0.34%*, 55 ± 0.32%* and 52 ± 0.32%, respectively, and 57 ± 0.31% for 3 yr. combined. The overall HCR for 3 yr. were 35%, 35% and 34%, respectively, and 35% for the 3 yr. combined. Mean HCR for distance strata (≤ 50, > 50 but ≤ 100, > 100 mi. from PC) were 42%*, 58%* and 70%*, respectively. Mean HCR for the 4 bed size strata (> 200, > 100 but ≤ 200, > 50 but ≤ 100, ≤ 50 beds) were 32%*, 44%*, 68%* and 79%*, respectively. Conclusion: For 3 yr. combined, mean HCR was 57% and overall HCR was 35%. HCR was found to be directly proportional to distance from PC and inversely proportional to hospital bed size. PC HCR can be easily estimated in some cases from readily available data and is an important estimate of PC utilization in serious poisonings.

150 THE ECONOMIC IMPACT OF A PROPOSED POISON CONTROL CENTER IN ECUADOR.

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Background: The economic impact of poison control centers in the United States have been estimated by the following methods: 1) The value of lost earnings because of poisoning deaths, 2) The increased cost to the health system when there is no poison center, and 3) the willingness of consumers to pay for poison centers. Methods: A survey is made of the published literature for each of the methods. Method 1 is used to estimate the impact of a proposed poison center in Ecuador. The value of lost lifetime earnings is estimated for the hypothetical poisoning death of a 20-year-old high school educated Ecuadorian male. Assumptions are made for probabilities of survival and employment, inflation, present value, purchasing power and growth in gross domestic product. Results: The estimated loss to the Ecuadorian economy due to this hypothetical death is $158,000 in 1999 dollars. This has a value of $480,000 when local purchasing power is considered. Conclusions: Poison centers make positive contributions to economic development. Economic losses result from each poisoning death. When multiplied by the total number of deaths they result in significant losses. This method is a valuable tool in planning and policy making.

151 KEYBOARDING SKILLS AND PERFORMANCE IN ON-LINE CHARTING.

Herrington LF, Geller RJ. *Georgia Poison Center, Atlanta GA*

Background: On-line data entry is the standard for record keeping in poison centers today. A significant increase in documentation time over prior methods of data collection has been identified in a previous study, though the reason is unclear. Narrative medical record requirements for the TESS data base are left to individual centers. Specialists in Poison Information (SPI) must have health professional training, for which keyboarding skills are generally not pre-requisites. The purpose of this study is to determine the role that typing ability plays in the length of the narrative in an electronic record. Methods: 24 SPIs at a certified regional poison center were given a standardized speed typing test. The most recent calls for each SPI regarding lay silica gel calls (n = 6), lay gasoline calls (n = 6), and medical calls involving acetaminophen in combination with diphenhydramine or a narcotic (n = 4–6) were collected and the average length of each category calculated. Results: Of participating staff, 29% were RNs, 33% pharmacists, 21% MDs and 17% other. Speeds ranged from 13–62 adjusted words per minute (AWPM). Trained typists averaged 39 AWPM, non-trained 23. Total calls handled per SPI (adjusted for a 40 hour week) from 1/1/01–3/31/01 ranged from 707–1924. No correlations between typing speed and number of calls handled or narrative length were identified (r^2 all < .06). Narrative lengths were similar for nurses, pharmacists and physicians (33-37 words); word length averaged 4.4 letters. Average narrative length increased with severity–silica gel = 20 words; gasoline = 36; acetaminophen with

diphenhydramine/narcotic = 50. Conclusion: Call severity, not keyboarding skill, has the greatest impact on narrative length. Keyboarding skills may become more of an issue if narrative requirements for TESS are expanded.

152 DEFINING POISONING HOSPITALIZATIONS FOR EPIDEMIOLOGICAL STUDIES.

Martin TG, Robertson WO. *University of Washington Medical Toxicology Consult Service, University of Washington & Washington Poison Center, Seattle, WA*

Background: A wealth of poisoning (PO)-related epidemiologic data is contained in hospital discharge datasets. Our dataset contains 1 E (intent) and 9 N (diagnosis) fields (ranked in order of importance) which may contain more than one PO ICD9-CM code/case. Our objective was to compare the numbers of cases identified by different definitions of hospitalized PO in the data from Washington (WA) State for 1987–96. Methods: After IRB approval, WA hospitalized PO were identified by a computerized search of a statewide hospital discharge database (CHARS) for cases with variable numbers of code fields indicating acute PO, then descriptive analyses was performed. Results: Use of the Ecode and the 1st Ncode (''primary'' diagnostic), the first 2 Ncode or the first 3 Ncode fields identified 90.8%, 94.2% and 96.8%, respectively, of the total cases (44,375) identified using all 10 code fields. Broadening the definition to include the 4th to 9th Ncode fields only added an additional 3.2% of the total cases. Because of mixed drug PO, a total of 0, 1, 2 or 3 PO Ecodes were identified in 7,760, 35,833, 778, and 8 cases, respectively. Likewise, a total of 0, 1, 2, 3, 4, 5, 6, 7 or 8 PO Ncodes were identified in 2,514, 31,430, 7,610, 2,198, 493, 108, 15, 5 and 2 cases, respectively. Conclusion: When searching for cases most likely hospitalized because of PO, limiting the inclusion criteria to the Ecode and first 1–2 Ncode fields is recommended. When searching for all PO related hospitalizations, inclusions of all E & Ncode fields is recommended. However, the differences are not very great with either approach.

153 EPIDEMIOLOGY OF WASHINGTON STATE POISONING HOSPITALIZATIONS.

Martin TG, Kenny RJ, Robertson WO. *University of Washington Medical Toxicology Consult Service, University of Washington & Washington Poison Center, Seattle, WA*

Background: Poisoning (PO) epidemiology is essential in prioritizing PO research, treatment and prevention efforts. Our objective was to describe the epidemiology of hospitalizations for acute PO in Washington (WA) from 1987–96. Methods: After IRB approval, WA hospitalized PO were identified by a computerized search of a statewide hospital discharge database (CHARS) for cases with a ICD9-CM discharge code(s) indicating acute PO and descriptive analyses performed. Results: Mean yearly # and rate/100,000 (range) of WA hospitalized PO during the 10 yr. interval was 4,437 (4,278–4,596) and 88.2 (77.8–100.4), respectively. PO related E (intent) codes were identified in 81% of cases (most after 1988) with 61% intentional, 32% unintentional and 7% other. Males accounted for 40% and females for 60% of PO cases. Those aged 15–54 accounted for 72% overall with the 25–34 yo group having the greatest overall proportion (22%) and 15–19 yo group having the greatest overall rate (145). Mean length of stay (LOS) was 3.5 d with 40% of LOS $\leq$ 1 d and 80% $\leq$ 4 d. Discharge status indicated: home ($\pm$ assistance) 76%, transferred 19%, AMA 3% and died 2%. Mean hospital charge per PO case was $4,694 with mean charges trending upward and more than doubling over 10 yr. Primary payer was Medicare/Medicaid in 46.5%, third party 35.7%, self-pay/charity in 14.1% and other government program 3.7%. Of 145 hospitals, 20 accounted for 52.5% of PO cases. Conclusion: A wealth of PO-related epidemiologic knowledge may be derived through analysis of hospital discharge data.

154 COMPARSION OF POISON EXPOSURE DATA: NHIS SURVEY AND TESS.

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Objective: The purposes of this secondary analysis were to identify age-adjusted poisoning rates, poison control center (PCC) contacts, and hospitalizations due to poisonings in young children based on 1997 National Health Interview Survey (NHIS) data; and where possible, compare findings with 1997 Toxic Exposure Surveillance System (TESS) data. Methods: Data were from 1997 NHIS poisoning episode data for children 5 years and younger. Data collection originally occurred via in-person interviews with 103,477 persons from 39,832 randomly selected households. Respondents were asked about poison exposures in the household during the previous 3 months. Results: Based on NHIS population weighted data, annual poisoning rates were highest in children 1–2 years old, and more poisonings resulted from non-pharmaceutical substances (Table 1). A PCC was called in most exposures to children $\leq$3 years, but only in about half of the 4–5 year old exposures. The PCC was called in 54% of the poisoning hospitalizations. The odds

of a child being hospitalized for poisoning were 3.75 times greater when the PCC was not called, compared to 0.27 when the PCC was called.

Table 1

Poisonings by Age & Substance, 1997 NHIS (Age-specific Rates/1000 pop.)

	<1 yr	1 yr	2 yrs	3 yrs	4 yrs	5 yrs
Pharmaceuticals	2.7	9.7	20.0	7.8	7.8	2.9
Non-pharmaceuticals	7.8	39.5	21.3	8.9	9.6	2.7

Comparison of NHIS data with data in the 1997 Annual Report of the TESS (Litovitz, et al, 1998) revealed the number of NHIS reported exposures are approximately ½ those in the TESS data (Table 2). Of all the exposures to children <6 years, children in the 1–2 year age group had the highest percent of poisoning exposures. Review of TESS data revealed boys and girls had approximately equal exposures; girls had a higher percentage of poisonings in the NHIS data at 1,2 and 4 years of age.

Table 2

Percent Poisonings in Children < 6 Years, by Age, Gender, & Data Source

	<1 year	1 year	2 years	3 years	4 years	5 years
NHIS data						
Males	5.2%	10.9%	8.0%	8.5%	3.9%	2.1%
Females	2.2%	23.5%	20.6%	4.0%	8.9%	2.2%
TESS data						
Males	6.5%	16.6%	16.8%	7.8%	3.8%	2.3%
Females	5.9%	14.6%	14.6%	6.4%	2.9%	1.7%

Note: NHIS weighted data: n = 550,568; TESS data: n = 1,145,564

Conclusions: Based on NHIS data, hospitalizations are diverted when the PCC is called. Differences in number of pediatric exposures and the predominance of exposures to girls may reflect recall bias in the NHIS data. Differences in data elements collected by NHIS and by TESS (e.g., NHIS does not provide clear definitions for in non-pharmaceutical categories) make comparisons difficult. Efforts should be made to standardize types of data collected so a more complete picture of poisonings to young children can be formulated.

155 DIURNAL AND SEASONAL VARIATIONS OF CALLS TO A POISON CENTRE.

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Background: Data collected by Poison Centers might give clues to temporal increases of particular types of poisoning thus enabling proper timing of preventive measures and campaigns. We hypothesized that such data will yield information about seasonal and diurnal accumulation of certain types of poisoning. Methods: Calls to a poison center concerning human poisoning were recorded during a period of 2 years (1998–99) with regard to patient age (adults >16 years old), circumstances of poisoning (intentional vs. accidental) and class of poison (therapeutic drugs, technical and industrial products, household products, agrochemicals, drugs of abuse, plants, mushrooms, envenomations). 24 one-hour intervals of all 730 days were created to analize diurnal variations. Both years were divided into 2-week intervals for analysis of seasonal variations. Results: 47554 calls were included (46% adults, 54% children; 76% accidental, 21% intentional). Call volume peaked during summer at the beginning of September for adult and pediatric poisoning. The

seasonal variations were entirely due to accidental poisoning (increasing from 300 to 475 per week) whereas intentional poisoning remained stable (100 per week) throughout the year. Poisoning with therapeutic drugs decreased slightly during summer, whereas poisoning with plants and agrochemicals increased markedly (5-fold and 4-fold resp.). Diurnal variations of poisoning is almost entirely the result of accidental poisoning in children with a marked peak from 11 to 12 a.m. and from 5 to 8 p.m. Conclusion: Seasonal and diurnal variations are almost exclusively due to accidental poisoning. This stresses the need for enhanced preventive activities, particularly in pediatric poisoning. In our geographical region campaigns for prevention of agrochemical poisoning are best carried out in March, for plant poisoning in June, whereas campaigns for prevention of therapeutic drug poisoning can take place all over the year.

156 TRENDS OF ACUTE POISONINGS IN INDIA.

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Background: The toxins involved in acute poisoning vary the world over and change over the years. This study was conducted to ascertain the pattern of acute poisoning in this part of the world over the last four decades. Methods: Series of acute poisoning cases reported from this country during the period 1960–1999 were searched using the Medline. Hand search was employed for pre-1966 series. Cases of food poisoning were excluded. Adult and pediatric series were reviewed separately. Analysis was done decade-wise. Results: In children, non-medicinal agents accounted for 94.8%, 73%, 72.9% and 68.4% of all cases of acute poisoning in the decades 1960–69, 1970–79, 1980–89 and 1990–99 respectively. Kerosene oil (a hydrocarbon) was the most common agent and was seen in 45.5%, 38%, 44.2% and 33.9% of all cases during the above-mentioned periods. The incidence of insecticides/pesticides poisoning rose from 4.4% in 1960–69 to 13.3% in 1990–99. Opioids and phenothiazines were the commonest medicinal agents. Information about acute poisoning in adults was scanty. In 1960–69, the commonest agent was copper sulphate (31.1%) but in 1990–99, aluminium phosphide (a fumigant) and other insecticides/pesticides accounted for 49.6% and 24.8% of all cases of poisoning. Barbiturate poisoning that was common in 1960s and 70s became uncommon in the last 2 decades. Conclusions: In the last two decades, dangerous chemicals like insecticides and fumigants have become common agents in acute poisoning cases in adults. Ingestion of kerosene oil continues to be common in young children. This calls for urgent need of public education regarding handling and storing of various chemicals in this part of the world.

157 INTERNET SURVEILLANCE OF PRO-DRUG WEBSITES. I. INCIDENCE OF CLUB DRUG REPORTING OVER A ONE-YEAR PERIOD.

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Background: Websites condoning drug use may post experiences of illicit drug use (''trip reports'') by date. Because the Internet is a population-wide phenomenon, data from pro-drug websites may represent real-time population-based information on illicit drug use. We explored temporal trends in drug experience reporting. Methods: We used a PERL CGI script program interfaced to monitor trip reports posted to Erowid (www.erowid.org) over a one-year period. We monitored reports of MDMA, GHB, and LSD (''club drug'') experiences. We also monitored the total number of drug reports. Results: We identified four bursts in reporting activity for all drugs, including MDMA, LSD and GHB; each ranged between 100–400% of baseline activity. These bursts occurred around June 22, 2000, Sept–Oct, 2000, and March 1, 2001. Each of these dates correlates with events in the academic calendar. Conclusion: Online postings are thought to reflect the offline activities of Internet-using populations. We identified bursts in reports of drug/club drug use coinciding with the beginning and end of the academic year, as well as with spring break. Although demographics of the reporting population and the temporal relationship between drug use and submission of reports is unknown, the observed activity may represent drug use in adolescents and young adults; high prevalence of Internet and club drug use has been observed in both populations. Moreover, these results suggest increased drug use at the beginning and end of the school year, and during spring breaks. We believe this methodology represents a mechanism for monitoring drug use in these populations. Furthermore, these results suggest that efforts to prevent club drug use may be effectively focused at specific age groups during specific periods in the academic year.

158 INTERNET SURVEILLANCE OF PRO-DRUG WEBSITES. II. IDENTIFICATION OF EMERGING DRUG USE TRENDS.

Boyer EW, Shih K, Karger D, Quang L, Case P. *Children's Hospital Massachusetts Institute of Technology, and Harvard Medical School, Boston, MA*

Background: Websites condoning drug use often carry postings from websites visitors describing experiences from illicit drug use. We observed these postings to determine if trends in drug use could be identified. Methods: Using a PERL CGI script interfaced with Erowid (www.erowid.com), we reviewed descriptions of drug experiences (''trip reports'') contributed over a 4-month period. Our searches, which involved a menu of 17 drugs of abuse, were repeated at two-week intervals. We searched for multiple reports describing use of a single drug. To determine if reports originated from random sources or from clusters of related drug users, we analyzed the content of each report. Results: On April 21, 2001 we identified a cluster of reports (N = 15) for 2-CT-7 (2,5-dimethoxy-4-propylthiophenethylamine, a hallucinogenic amphetamine), submitted April 2–16, 2001. This burst was 225% greater than baseline reporting activity for 2-CT-7. Content analysis revealed that 4/15 reports (26%) originated from two clusters of individuals using the drug. Conclusion: The global nature of the Internet may accelerate the dissemination of isolated or local drug practices to produce new drug trends. Existing drug prevention measures may not respond to emerging drug trends with sufficient rapidity to deter potential users. We identified, within 5 days of report submission to a prodrug website, a burst in reports of experience with a novel drug. Use of this drug was distributed between at least two groups of users. These results demonstrate that surveillance of pro-drug websites may be useful in identifying emerging drug trends. This methodology can also be used to develop Internet-based drug prevention measures that can be initiated while a trend is developing, thereby making drug prevention more proactive and timely.

159 INTERNET SURVEILLANCE OF PRO-DRUG WEBSITES. III. IDENTIFICATION OF EMERGING DRUGS OF ABUSE.

Boyer EW, Shih K, Karger D, Quang L, Case P. *Children's Hospital, Massachusetts Institute of Technology, and Harvard Medical School, Boston, MA*

Background: Websites condoning drug use often carry postings from visitors describing their experiences of illicit drug use. If visitors to the same website comprise a ''virtual peer group'', online descriptions of a new recreational substance may prompt others might try the same drug, thereby creating a new trend. Methods: Using text recognition and PERL CGI script program interfaced with Erowid, we reviewed descriptions of drug experiences (''trip reports'') contributed over an 8-month period. We performed repeat assessments at two-week intervals. We intended to identify an initial trip report of a drug not previously reported in Erowid. Results: We identified a single trip report describing tiletamine abuse; this report was posted on April 14, 2001. Using two separate programs, we confirmed that no other tiletamine reports were submitted to Erowid since inception of the website. Conclusion: Federally funded drug surveillance efforts monitor relatively few drugs of abuse; moreover, significant delay exists between obtaining and releasing survey information. In contrast, websites such as Erowid describe more than 150 illicit drugs, with new material added daily. Our methodology identified an initial report describing tiletamine abuse, a veterinary anesthetic similar to ketamine. Although continued surveillance is needed to establish a trend in tiletamine abuse, this methodology represents a novel surveillance tool for detecting emerging drugs of abuse. Ultimately, this methodology can assist in the creation of timely, preventative efforts focused upon specific drugs and drug trends. Furthermore, this methodology may permit, depending upon the information in the posting, region-specific warnings to clinicians regarding newly abused drugs.

160 A PHARMACOECONOMIC MODEL OF ANNUAL COSTS REQUIRED TO MAINTAIN AN ANTIDOTE STOCK.

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Background: Surveys in various jurisdictions have repeatedly shown severe understocking of essential antidotes. Perceived expense is an important barrier to adequate antidote stocking. These surveys, and recent expert consensus guidelines, cite only purchase price as a measure of cost. In most hospitals, many antidotes remain unused at year-end, and may expire prior to use. An accurate model of annual maintenance cost, beyond initial purchase, is needed to assist in rational formulary selection. Methods: Inputs for the model were: frequency of use (f), shelf-life (S), unit cost (C), and population served (N). Antidotes were selected from the WHO IPCS and expert consensus guidelines. Estimates of f

were derived from TESS data for 1996 to 1998. C and S reflect hospital pharmacy costs at our hospital based on current supplier contracts, as well as supplier policy of replacement for expired product. Results: The annual cost for a given antidote is $C/Sx\sum_{\iota}Pr(\iota)(\iota + 1\text{-}(\iota/\iota + 1))$, where $Pr(\iota)$ is the conditional probability of the antidote being administered $\iota = 0, 1, 2, \ldots$ times during S, namely $\exp(-p)p^{\iota}/\iota!$ with $p = fSN$. When expired antidote is replaced for free (S infinite), annual cost is fCN (ignoring inflation and using straight-line amortization). For small to medium hospitals (N = 20,000–100,000) stocking for the initial hour, total annual cost ranged from $Cdn 2300–4730; for the initial 4 hours, $3050–7580. A change in supplier policy to free replacement for fomepizole (as in the US) and for the nitrite/thiosulfate cyanide kit would reduce annual cost to as little as $645. Conversely, anti-digoxin F_{ab} fragments are relatively inexpensive due to credit on expiry. Conclusions: Beyond unit cost, shelf-life and supplier policy of replacement on expiry are major determinants of antidote cost. Efficacy and safety of available alternative therapy must also be considered in assessing formulary adequacy.

161 COMMUNICATIONS DURING CHAOS: A POISON CENTER'S ROLE IN MASS CHEMICAL EXPOSURE.

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Background: Flow of accurate, timely information is the most common problem in mass chemical exposures. An early notification system has been developed to improve communications between the incident scene and area hospitals. Methods: The notification system is activated whenever a HazMat incident has potential for injured patients, mass exposure or media interest. The poison center receives initial information from the county health department emergency responders. The SPI completes a structured worksheet for pertinent information such as location, type of incident, number of patients and suspected chemicals. The goal is to quickly alert EDs of an incident so they can prepare for contaminated patients or mass casualties. A computer generated fax is sent via network fax machine to area hospital emergency departments with an attached one-page fact sheet about the suspected substances involved. The quick reference fact sheet includes information about the expected symptoms, self-protection, initial treatment and additional resources needed. Because of the importance of early notification, information may not be completely accurate or reliable. The notification form addresses the reliability of the information and its accuracy with a 10 point rating scale. Periodic testing ensures accuracy of fax numbers and familiarity with the system. Conclusion: Poison centers can play a key role as a central information resource in mass chemical exposures. Timely information may be broadcast to local emergency departments in order to provide the best possible response.

162 REGIONAL PHARMACEUTICALS FOR BIOLOGICAL AND CHEMICAL TERRORISM.

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Background: The National Office of Domestic Preparedness has determined that the threat of a biological or chemical attack is very real. As an active participant of a 13 county regional task force, one of the roles of the poison center was to determine the pharmaceutical needs of the community in the event of a terrorist action and to develop a financially responsible method of acquisition and storage. Methods: Working with local health officials, an extensive literature review was conducted to identify possible biological and chemical poisons. Treatment recommendations were identified and an estimated amount to treat 5,000 people for 24 hours was determined. Instead of purchasing the medications, a unique solution utilizing a regional pharmacy wholesaler was used. Discussion: An important element in a biological or chemical terrorist event is the availability of the pharmaceuticals and the capability of delivering them rapidly. The poison center is the ideal agency to coordinate this endeavor since it is familiar with contemporary therapy and will be aware of the number, location and status of casualties. Based on the expense involved in the purchase and storage of a large quantity of medications, utilizing a local pharmaceutical distribution company is fiscally responsible. Rotation through normal stock and being readily accessible is another benefit. Conclusion: The poison center serves a number of roles in the surveillance, recognition and treatment of biological and chemical terrorism. Assisting in the development, implementation and procurement of a pharmaceutical cache is yet another role.

163 DEVELOPMENT OF A CENTRALIZED ANTIDOTE AVAILABILTY DATABASE.

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Objective: To develop guidelines and establish a method of rapid retrieval for locating antidotes in emergency situations that may be stocked in insufficient quantities. Methods: A retrospective review of cases reported to a certified poison center requiring usage of at least one antidote and a literature review of emergency antidote stocking were performed. The corresponding information was utilized to develop a survey assessing the availability, location and quantity of antidotes in an institution. Surveys were mailed to 98 acute care hospitals in the region serviced and a corresponding telephone follow-up was performed. A Microsoft Access® based database, with capability to generate reports, was created to analyze the survey data. Results: 45% responded initially to 57 different medications with toxicological indications / antidote use and the results were entered directly into a centralized database. The database provides facility information (name, address, telephone and fax numbers, contact person and email address) and antidote availability information (name, strength, dosage form, normally stocked, quantity and location). Brand name, generic name or facility name queries can be performed and formatted into reports, which are then printed and faxed immediately to facilities requesting information on specific antidotes during poison control center consultation. Conclusion: The creation of a centralized antidote availability database is a valuable asset in emergency situations. Fax on demand reports generated for health care practitioners may help decrease delays in obtaining and administering antidotes that may/or may not be present in a facility, or in an insufficient quantity. Providing the name(s), location(s) and telephone number(s) of the nearest health care facilities stocking the particular antidote accomplishes this.

164 SHARING OF ANTIDOTES: IS IT AN EXCUSE TO STOCK LOWER AMOUNT OF THOSE THAT NEED TO BE GIVEN IN LESS THAN 30 MINUTES?

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Introduction: Sharing of antidotes with a neighboring hospital for those that need to be given within 30 minutes may be a problem considering the potential delays in obtaining them. Methods: Written surveys were sent to 118 hospital pharmacies. The number of antidote doses kept in stock was compared amongst 1st, 2nd and 3rd line hospitals with or without agreement to share antidotes. Antidotes surveyed included those required within 30 minutes. The preparedness of each hospital was also assessed by evaluating if they had a 30 minutes dose for 1 average adult: atropine: 10 mg; cyanide kit: 1 kit; digoxin immune FAB: 4 vials; ethanol 100%: 70 mL or fomepizole: 1 vial; glucagon: 10 mg; methylene blue: 70 mg, naloxone: 4 mg; pyridoxine: 5 g. Results: Preliminary data was obtained from 69% of the pharmacies surveyed. There were 37 hospitals with agreement to share antidotes (22 1st line, 12 2nd line, 3 3rd line) compared to 44 hospitals with no agreement (28 1st line, 5 2nd line and 11 3rd line). Distribution of hospitals with agreement differed from those without ($p = 0.02$): 3rd line hospitals were less likely to have an agreement. Hospitals with agreement were not more likely to have an hospital within 30 minutes distance: 25/39 (64%) vs 24/43 (56%), $p = 0.44$. In 1st line hospitals, glucagon was kept in higher amount in hospitals with agreement (median 20 vs 10 mg, $p = 0.03$) and there were more hospitals with at least one 30 minutes dose [20/22 (91%) vs 17/28 (61%), $p = 0.02$] than in those without agreement. There was no difference for the other antidotes. Conclusion: First and second line hospitals are more likely to have agreements to share antidotes. Not all hospitals with such agreement are in close proximity. Hospitals with agreement to share antidote do not necessary stocked lower amount of antidotes than needs to be given within 30 minutes.

165 WASHINGTON'S ILLEGIBILITY SCANDAL.

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Background: In 1980, Tso and Robertson determined that many physicians' handwritten prescriptions at our Children's Hospital were deemed illegible by separate judging panels of physicians, pharmacists, nurses and medical students. They called for remedial action to be imposed–but none followed! In 2000, our state legislature modified a proposal to make "other than printed, typed or computer-generated prescriptions . . . unprofessional conduct . . ." subjecting the perpetrator to possible license revocation, to a law that simply mandated all prescriptions be legible–with a not-so-hidden message that no one could challenge their wisdom. We–and others–decided to do so. Method: As an obvious "quality assurance process" requiring no Institutional Review Board approval, several pseudo-random samples

of prescriptions already filled during the year 2000 were obtained with the cooperation of the Hospital's Pharmacy Department. Thirty prescriptions were arbitrarily classified "customary"; 15 as "difficult". Volunteer judges of legibility were sought and obtained. All 10 members of each of the 4 panels proceeded to interpret 45 separate prescriptions for their "legibility, possible legibility or illegibility." All were timed with a stopwatch. Results: Among the sample of 30 customary prescriptions, the mean rates of illegibility for each of the ten-member panels were A) pharmacists = 4%, B) physicians = 2%, C) nurses = 5%, and D) medical students = 8%. Among the sample of "difficult prescriptions" the comparable percentages were 23%, 12%, 20%, and 43%. For the "difficult prescriptions" each of the 600 interpretations consumed a mean of 16.4 seconds, compared to 8 seconds for the others. Conclusions: Clearly, illegibility remains a problem. Remember: each of these prescriptions had, in fact, been filled! Would you bring your child to our hospital?

166 COCAINE ASSOCIATED ACS: DO THE 2000 INTERNATIONAL TOX-ACLS GUIDELINES WORK?

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Objective: International consensus guidelines (ECC, 2000) recommend different treatment strategies for patients (pts) with ACS related to or unrelated to cocaine. The impact of these differences on clinical outcome has never been evaluated. We compared the treatment of chest pain pts with and without cocaine use to determine whether the different strategies resulted in differences in patient outcome. Methods: Consecutive low-intermediate risk chest pain pts (Braunwald criteria) presenting to an urban ED and admitted to the CDU and all cocaine/chest pain patients were enrolled. All ED pts <51 yrs without cardiac risk factors and all CDU patients received urine testing. Pts received serial markers and ECGs. Final diagnosis was per WHO criteria. Results: Of 1089 patients enrolled, 283 (25%) tested positive for cocaine. Cocaine users were younger (38.4 v 45.6 yrs; $p < 0.001$), more likely male (65 v 41%; $p < 0.001$), less likely to have htn (22 v 39%); increased cholesterol (5 v 20%) or diabetes (5 v 10%) but more likely to smoke tobacco (84 v 55%) and have a family history of CAD (31 v 16%): $p < 0.001$ for all. Cocaine users less often had a normal ECG (34 v 71%; $p < 0.001$). Both groups received aspirin (94%) and nitrates (92 v 97%). Cocaine users were more likely to receive benzodiazepines (31 v 2%; $p > 0.0001$) and less likely to receive beta-blockers (2 v 4%; $p = 0.05$). There was no difference in hospital admission from CDU (6% v 8%; $p = 0.4$) or rate of AMI (3 v 2%). Conclusion: Treatment of pts with cocaine associated ACS with aspirin, nitrates, increased use of benzodiazepines and decreased use of beta-adrenergic blockade resulted in clinical outcomes similar to low-intermediate risk CDU pts with ACS unrelated to cocaine. These findings support the consensus guideline recommendations for this patient cohort.

167 METHYLPHENIDATE ABUSE IN PRE-TEENS AND ADOLESCENTS.

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Background: A recent increase in methylphenidate trafficking and abuse has been reported in adolescents. This study evaluates pre-teen and adolescent methylphenidate abuse reported to the American Association of Poison Control Centers (AAPCC) Toxic Exposure Surveillance System (TESS). Methods: The AAPCC TESS database was queried for methylphenidate cases in children 10–19 years of age in 1993–1999. Cases were included if the reason was abuse and outcome known. Methylphenidate was assumed to be the child's medication in acute-on-chronic and chronic exposures. Results: There were 759 cases, of which 42.7% involved 10–14 year olds. Males accounted for 58.9% of cases. For the 530 (70.0%) cases involving methylphenidate as a single substance, the numbers of cases increased 7 fold from 1993 to 1999. Methylphenidate was the child's medication in only 117 (15.4%) cases. Of 570 patients (75.1%) managed in a health care facility, 398 were discharged from the emergency department while 172 were admitted. Symptoms occurred more commonly in exposures involving co-ingestants (84.3%) than in methyphenidate only exposures (71.1%). The most common symptoms in adolescents with methylphenidate only were tachycardia (31.7%), agitation/irritability (25.7%) and hypertension (11.5%). Outcomes were no effect in 189 cases (24.9%) and mild, moderate and severe in 318 (41.9%), 245 (32.3%), and 7 (0.9%) patients, respectively. Conclusions: Poison center data show a trend of increasing methylphenidate abuse. While the majority of adolescents experienced minor or no toxicity, 33% developed moderate-severe effects and the majority were treated in a health care facility.

168 A CASE OF ECSTASY (MDMA) BODY-PACKING.

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Background: The recreational use of Ecstasy (3,4-methylenedioxymetamphetamine-MDMA) is an increasing problem. We describe an unusual case of MDMA body packing in a 30-year-old male. Case Report: The patient presented to an emergency department 24 hours after an admitted ingestion of 750 tablets of MDMA. The patient was hemodynamically stable and the physical exam unremarkable except for complaints of mild abdominal discomfort. There were no neurological or psychiatric changes. Abdominal x-rays revealed several opacities in the stomach and mid-bowel. The patient initially received activated charcoal. Whole bowel irrigation was recommended according to standard protocol, however, it was administered over a 24 hour period p.o. ad libitum. Passage of intact packets, approximately 6 × 4 cm in size, began several hours post-admission and was completed in 10 days, with no apparent adverse effects. Discussion: A total of 25 packets of tablets were recovered. The patient described wrapping 10 tablets, arranged in 3 rows, with plastic food wrap. These were congealed with heat from a hair dryer and then placed into condoms which were tied-off with fishing line. The patient's fastidious method of packaging was apparently effective in preventing toxicity. Large doses of MDMA may lead to severe symptoms including arrhythmias, seizures, coma and hyperthermia. Conclusion: Body packing for the purposes of illegal drug transporting most often involves cocaine and heroin. To our knowledge this is the first reported case of Ecstasy (MDMA) body packing.

169 GAMMA-HYDROXYBUTYRATE (GHB) AND ITS ANALOGS: CURRENT LEGISLATIVE STATUS.

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Objective: In recent years, the federal government has taken increasingly stronger measures to regulate gamma-hydroxybutyrate (GHB), culminating in the passage of the Hilary J. Farias and Samantha Reid Date-Rape Drug Prohibition Act of 2000 (*Fed. Reg.* 65; 13235–13238, 2000). This legislation amended the Controlled Substances Act and made GHB a Schedule I agent while permitting it to be placed into Schedule III if used under an FDA-approved New Drug Application. This bill included language that addressed the manufacturing, distribution and possession of GHB analogs, such as gamma-butyrolactone (GBL) and 1,4-butanediol (1,4-BD) but left the responsibility of regulation to the individual states. Because each state has the option of imposing stricter controls over medications than the federal government, a lack of consistency exists at the present time regarding the regulation of legitimate and illicit GHB and GHB analogs. The objective of this presentation is to describe the current legal status of these compounds nation-wide. Results: To date, a "split" schedule (Schedule I/III) for GHB, consistent with Federal actions, has been adopted by 23 states. In contrast, only California, Nebraska, South Carolina and Pennsylvania have adopted analogue statutes that reflect Federal law and which can control the illicit use of GHB analogues. Conclusion: Because it has no widespread industrial use, GHB is easily regulated on a Federal level. In contrast, GBL and 1,4-BD are important industrial chemicals. Last year saw the manufacture of 725 million pounds of 1,4-BD in the United States alone. Thus, there is a very real need to introduce legislation addressing illicit manufacture, possession and distribution of GHB analogues to enable law enforcement to prosecute cases of distribution for consumption and sexual assault.

170 THE RELATIONSHIP BETWEEN FEDERAL PUBLIC LAW 106-172 AND MASSACHUSETTS GHB EXPOSURES.

Franck K, Hibberd P, Boyer EW. *Regional Poison Control Center and Children's Hospital, Boston, MA*

Background: On February 18, 2000, US Public Law 106-172 registered GHB as a Schedule I agent. We examined the timing of Poison Control Center (PCC) calls related to GHB exposure with the passage of Pub L 106-172. Methods: We reviewed all inquiries pertaining to GHB over a two-year period. We excluded non-exposure inquiries and non-Massachusetts exposures. We used a Fischer's exact test to compare the proportion of exposures which occurred from November, 1999–January 2000 (before passage of 106-172) with those occurring from February 2000–April 2000 (the peri-passage period). We also compared the proportion of exposures in the same peri-passage period with those occurring from May 2000–July 2000. Results: We identified a statistically significant increase in GHB exposures during Feb–March–April 2000 compared against baseline GHB exposures of the preceding three months ($P < 0.013$). Moreover, the proportion of GHB exposures decreased by a statistically significant amount from Feb–March–April 2000 to May–June–July 2000 ($P < 0.0053$). The burst of activity observed during Feb–March–April 2000 coincided with passage

of Pub L 106-172. Conclusion: Although causation cannot be established, poison center data suggest that Federal regulation of GHB led to a transient increase in use of GHB or its prodrugs. The pattern of GHB exposures potentially can be explained by accelerated purchasing of GHB prior to rescheduling, accompanied by its subsequent use. Consequently, clinicians might expect increased emergency department visits of patients abusing a drug about to be rescheduled. Furthermore, these data suggest that future drug prevention efforts should be coordinated with legislative efforts seeking to reschedule other illicit substances.

171 1,4-BUTANEDIOL WITHDRAWAL COMPLICATED BY URINARY RETENTION.

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Background: Gamma hydroxybutyrate (GHB) and gamma butyrolactone (GBL) withdrawal are both reported. Although entirely expected, a withdrawal syndrome associated with 1,4-butanediol (BD), which is metabolized to GHB *in vivo*, is not reported. Furthermore, GHB and GBL withdrawal are not reported to be associated with urinary retention. We present two brothers who took BD regularly for approximately one year and after voluntarily decreasing its intake, presented with a sedative-hypnotic withdrawal syndrome complicated by urinary retention. Case Report #1: A 22-year-old male presented to the ED with insomnia, hallucinations, constipation and inability to urinate after decreasing his daily BD intake. Sedation using approximately 120 mg of diazepam and 20 mg of haloperidol IV was required in the ED and he was intubated for airway protection. In the ICU, he received continuous infusions of midazolam and propofol. A Foley was placed and urine volume was estimated to be 400 mL. Case Report #2: The 19-year-old brother, presented several hours later with a complaint of palpitations, tremor and inability to urinate. He was confused, agitated and given a total of 45 mg of lorazepam IV and placed on continuous infusions of midazolam and propofol ultimately requiring intubation for airway protection. A Foley was placed and urine volume was approximately 500 mL. A product obtained from the patients was confirmed by gas chromatography mass spectroscopy to contain approximately 95% BD. Conclusion: The use of BD may be associated with a withdrawal syndrome similar to GHB and GBL withdrawal. Urinary retention may also be a prominent clinical feature of BD withdrawal by an unknown mechanism.

172 DETECTION OF 1,4-BUTANEDIOL (1,4-BD) IN URINE BY GAS CHROMATOGRAPHY-MASS SPECTROMETRY (GC/MS) AND RAPID *IN VIVO* CONVERSION TO GHB.

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Background: The gamma-hydroxybutyrate (GHB) precursors gamma-butyrolactone (GBL) and 1,4-BD are available as non-regulated substitutes for GHB. We demonstrate reliable GC/MS detection of 1,4-BD in human urine and rapid *in vivo* conversion to GHB in 2 human ingestions. Case 1: A 41-year-old male body-builder ingested 50 B 60 drops of liquid Rejov-at-nite™ (1,4-BD) associated with nausea and lost consciousness. Upon arrival at hospital, he was comatose with seizure activity, and a core temperature of 35°C. He remained comatose and hypothermic for 8 hours. Urine samples were obtained 1 and 9 hours post contact for GC/MS analysis. Case 2: A 25-year-old male body-builder ingested 32 ounces of Rejoov™ leading to respiratory depression, non-reactive pupils, and coma. Urine was collected 13 hours post admission for GC/MS analysis. Both cases recovered spontaneously within 24 hours and were discharged. Method: GC/MS methodology for detection, separation and semi-quantification of 1,4-BD and GHB in urine followed established methods (Pediatr Res (2000) **47**, 830–833). Representative chromatograms and mass spectra will be presented. Conclusion: Our data indicate that 1,4-BD is rapidly converted to GHB *in vivo*. Reliable identification in physiologic fluids can be made for diagnostic and legal purposes.

173 SEIZURE ASSOCIATED WITH 2C-T-7 (BLUE MYSTIC) INGESTION.

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Background: 2C-T-7 (2,5-dimethoxy-4-(n)-propylthiophenethylamine), also known as "blue mystic," is a hallucinogen developed around 1980 that purportedly share some of the entheogenic properties of LSD. No information could be found about this drug in the standard medical literature (i.e. MEDLINE, POISINDEX, and toxicology texts). Several Internet "recreational drug" web sites, however, provide extensive information on 2C-T-7. Case Report: The patient is an 18-year-old college student brought to the ED because of new onset seizure activity. He had been ingesting 2C-T-7 on a daily basis for about a month. He was familiar with web sites that discussed this drug and had purchased his

supply of 2C-T-7 over the Internet. He had no past medical or psychiatric history and denied alcohol or other drug use. The patient stated that prior to his most recent use of 2C-T-7 he had experienced less vivid hallucinations during his "drug trips." In order to intensify the hallucinations he increased the dose. Within a few minutes of ingesting the larger dose, he sustained a witnessed generalized tonic clonic seizure lasting less than a minute. On arrival in the ED he had some visual hallucinations and no recollection of the seizure activity. Vital signs were T: 36.6, RR: 18, P: 125, BP: 132/82. His pupils were about 7 mm bilateral and reactive. The rest of his examination was unremarkable. Over a three-hour observation, he remained stable. His hallucinations resolved, his heart rate slowed gradually to 90, and he was discharged from the ED. Conclusion: We report a case of 2C-T-7 use that resulted in seizure activity. Since the standard medical literature at present has little information about this drug, the health care provider is forced to rely on information garnered from Internet drug web sites. While such information may not have undergone medical peer review, the public, and adolescents in particular, may be quite influenced by their content.

174 SUPRATHERAPEUTIC USE OF OVER-THE-COUNTER (OTC) ANALGESICS BY PATIENTS REPORTING TO AN URBAN DENTAL CLINIC.

Havey JM[1], Hill RE[1,3], Robins CW[3], Bogdan GM[1], Daly FFS[1], Zallen R[2], Dart RC[1,3]. *[1]Rocky Mountain Poison and Drug Center–Denver Health, [2]Denver Health Dental Clinic–Denver Health, [3]University of Colorado Health Sciences Center, Denver, CO*

Objective: To examine the use of OTC analgesics among dental patients. Methods: Patients reporting to an urban dental clinic were interviewed by trained research assistants using a standardized questionnaire. We recorded patient demographics, the types and amounts of OTC analgesics taken over the past 3 days, and perceptions of OTC analgesic safety. The study was completed over 8 non-consecutive days. We defined supratherapeutic as any dose greater than the total recommended daily dose stated on package labeling. We defined primary OTC analgesic as the medication taken in the greatest amount by the patient if a combination of analgesics was used. Results: We approached 194 patients, of which 127 participated. Analgesic use within the last 3 days was reported by 100 of 127 patients, most (82%) used an OTC analgesic. The mean age of participants was 28 years: 52% were male. OTC analgesics reported in the 82 patients included ibuprofen (39), acetaminophen (28), acetaminophen/aspirin (8), naproxen (4), and aspirin (3). Seventeen patients reported supratherapeutic use of their primary OTC analgesic: ibuprofen (12/39), acetaminophen (2/28), and naproxen (3/4). Of these 17 patients, 82% agreed that they "could get sick [by taking] too much" OTC analgesic. Conclusion: OTC analgesic use was common in patients presenting to a safety net dental clinic (65%), as was OTC analgesic misuse (21%). Nonsteroidal anti-inflammatory drugs were the most frequently misused products. Although the study was limited by small sample size, it noted that analgesics requiring fewer total tablets per day for therapeutic dosing were more likely to be misused. Patient awareness of potential OTC analgesic toxicity did not prevent supratherapeutic use.

175 DIPHENDHYDRAMINE ABUSE AND WITHDRAWAL IN A PATIENT WITH A HISTORY OF A 2.5 GRAM/DAY DEPENDENCY.

Shiuh T, Wax P. *Department of Emergency Medicine, University of Rochester Medical Center, Rochester, NY*

Background: While excessive use of anticholingeric agents has been widely documented among individuals taking neuroleptic agents, cases of diphenhydramine (DPH) abuse and withdrawal are seldom reported. Case Report: A 38-year-old female with a history of depression presents to the ED with confusion and refusal to take her medications–olanzapine, gabapentin, mirtazapine, clonazepam. In addition, the patient has a history of escalating DPH use over the past 18 months, secondary to its "relaxing" effect, reaching a maximum of 100 25 mg tabs qd (2.5 gm) for a 10 month period of time and gradually tapering to 1.25 gm qd over the past 4 wks. She had not taken any of her medications including DPH over the 2 days prior to presentation. During the past 18 months she had not complained of dry mouth, urinary retention, constipation, confusion, sedation, or blurry vision. ED vital signs were T 36.1, P 109, BP 121/75, and R 16. She was calm with a normal exam except for disorientation and confusion. Her CBC and serum chemistries were normal. She was admitted to the hospital for observation of worsening withdrawal symptoms. By day 2 her mental status was clear but she complained of anorexia, nausea, and increased anxiety followed on day 3 by diarrhea, abdominal cramping and episodes of flushing. She was treated only with clonazepam, maintaining normal vitals. All somatic complaints gradually resolved, but she continued to complain of anxiety prior to discharge to outpatient drug rehabilitation on day 6. A DPH level drawn upon initial presentation (48 h after last DPH dose) was 550 ng/mL (ref. avg peak

serum DPH level 2h after a single 50 mg oral dose is 66 ng/mL.) Conclusion: This is a case of presumed DPH withdrawal presenting with mild confusion, anxiety and somatic symptoms of mild cholinergic excess without alteration in vital signs and demonstrating a self-limiting course. A remarkable tolerance to DPH characterized this case.

176 EIGHTEEN-MONTH MORTALITY IN A COHORT OF PATIENTS PRESENTING TO AN EMERGENCY DEPARTMENT WITH NON-FATAL OPIOID OVERDOSE.

Daly FFS[1,2], Morgan D[1], Fatovich DM[1,2], Bartu A[3,4], Quigley A[3]. *[1]Department of Emergency Medicine, Royal Perth Hospital; [2]University of Western Australia, [3]Next Step Specialist Drug and Alcohol Services, [4]Curtin University, Perth Western Australia*

Objectives: To study 18-month mortality in a cohort of patients following presentation to a metropolitan emergency department with non-fatal opioid overdose. Methods: A retrospective chart review was performed eighteen months after the conclusion of a prospective observational study, which enrolled all patients with non-fatal opioid overdose attending a tertiary referral emergency department over a 12-month period. The emergency department was the major metropolitan facility serving a geographically isolated city of 1.2 million. The study was approved by the local Institutional Review Board. All medical charts were reviewed by a single non-blinded investigator using a pre-formatted abstraction tool. Intra-rater reliability was measured using Cohen's un-weighted Kappa score. Coronial and state death certificate data were also searched using all known aliases. Results: Two hundred and twenty eight patients (60% male, mean age 25 years, range 15–49), presenting on 253 occasions, were enrolled in the original prospective study. Fifteen patients (6.6%) presented with opioid overdose on more than one occasion during the original study. Eighteen months following the conclusion of the year-long prospective study, 223 of 228 patient charts were reviewed (3 unknown males, 2 charts lost); 12 had died (5.4%). Three of 15 (20%) recidivists in the original cohort died compared to 9 (4.3%) of 208 patients with single presentations (Odds ratio 5.5, 95% CI 1.3–23.1, p = 0.04). Conclusions: This study is limited by its retrospective nature and relatively small numbers. However, short-term mortality among patients presenting to a metropolitan emergency department with non-fatal opioid overdose was high, especially among those with multiple presentations.

177 NEBULIZED NALOXONE EFFECTIVELY AND GENTLY REVERSES METHADONE INTOXICATION.

Mycyk M, Szyszko A, Aks S. *Toxikon Consortium, University of Illinois Hospital, Chicago, IL*

Background: Naloxone administered by intravenous (IV), intramuscular, subcutaneous, sublingual, and endotracheal routes has reversed opioid intoxication. We describe the successful use of nebulized naloxone in methadone overdose. Case Report: A 46-year-old woman in a methadone maintenance program developed respiratory distress and lethargy shortly after ingesting her daily methadone dose late in the afternoon. Medical history included HIV and COPD requiring supplemental home oxygen. Heart rate was 76 beats/minute, blood pressure 122/66 mmHg, respiratory rate 12 breaths/minute, and pulse oximetry on 3L nasal cannula was 61%. Physical exam was remarkable for a pale, lethargic woman with shallow respirations. Oxygen with 100% nonrebreather face mask failed to improve the patient's oxygenation or clinical status. An arterial blood gas was obtained: pH 7.28, pCO_2 96 mmHg, pO_2 46 mmHg, $pHCO_3$ 47 mmHg. The patient's baseline pCO_2 is 60 mmHg. Since IV access was initially unobtainable, 2mg naloxone mixed with 3cc normal saline was administered via nebulizer. Within five minutes the patient's respirations deepened and rate increased to 28/minute, pulse oximetry improved to 100%, and mental status improved to normal. Repeat arterial blood gas demonstrated pH 7.36, pCO_2 83 mmHg, pO_2 67mmHg, $pHCO_3$ 47mmHg. 25 minutes after nebulized naloxone, the patient was noted to have yawning and lacrimation but no other signs of opioid withdrawal. Other toxicologic, infectious, and structural causes for hypoxia and lethargy were ruled out and the patient was admitted to the intensive care unit despite her request for discharge. Discussion: Naloxone administration via nebulizer effectively reversed methadone overdose in an opioid dependent patient. Nebulizer administration of naloxone may avoid the violent symptoms of withdrawal seen after IV administration.

178 THIAMINE DEFICIENCY IN ALCOHOLIC PATIENTS.

Hexdall A, Pardo S, Hoffman RS, Goldfrank LR. *Bellevue Hospital Center, New York, NY*

Background: Thiamine deficiency (TD), a recognized complication of alcoholism, can result in substantial morbidity and mortality if untreated. Until recently, insensitive laboratory techniques hampered both an understanding of the

incidence of TD and the role of T supplementation. The following study was designed to prospectively evaluate TD in the ED. Methods: Adult ED alcoholics by DSM criteria were enrolled if they were able to give informed consent, and had no other conditions associated with or clinical signs of TD. Trained observers recorded standardized demographic data that included vitamin use and previous T supplementation. Blinded T levels were determined by a highly sensitive HPLC technique. A healthy control group was included to confirm normal laboratory values. TD was defined as a T level <0.7pg/mg of total protein (P) to correlate with predicted pyruvate dehydrogenase dysfunction. Mean T levels were compared using the student's t-test. Numbers of TD subjects in each group were compared by Fisher's exact test. A $p < 0.05$ was considered statistically significant. Results: 105 alcoholics were enrolled; 20 used vitamins or had recently received T. There were 18 controls. The study groups were therefore defined as follows: C = 18, A = 85, A + V = 20. The A + V group was not demographically different from the A group. Mean T levels (in pg/mg P) were: C = 1.67; A = 0.856; A + V = 1.95. (p values: C vs A, <0.001; A vs A + V, <0.001; C vs A + V, NS). Numerical TD was identified in 1/18 C patients, compared with 35/85 A and 0/20 A + V patients ($p = 0.003$, RR 1.3, 95% CI = 1.12–1.51 for C vs A). Conclusions: Although severely ill and clinically TD patients were excluded from study by design, numerical TD remains prevalent in alcoholic ED patients with obvious metabolic, neurologic and cardiovascular implications. Supplementation appears to protect against numerical TD in alcoholic patients and should be continued.

Platform Session 6 Monday, October 8 4:30 pm–5:30 pm
Abstracts #179–#182

179 OUTBREAK OF METHANOL INTOXICATION, EL SALVADOR, OCTOBER 2000.

Armero JA[1], Suarez GI[1], Kilbourne EM[2], Hernandez R[1], Mixco M[1]. [1]*Ministry of Public Health and Social Assistance and Institute of Legal Medicine, El Salvador;* [2]*Agency for Toxic Substances & Disease Registry, Atlanta, GA*

Background: We investigated one of the largest outbreaks of methanol toxicity on record. Our aims were to describe the outbreak, assess the adequacy of therapy rendered, and identify factors indicating a poor prognosis. Methods: We developed statistics from questionnaires, medical records, and autopsy and laboratory reports. Results: During the month of October 2000, a total of 211 cases occurred, predominantly in the East-Central part of El Salvador, when methanol was introduced into the alcohol supply. The outbreak principally involved males (98%), largely of middle age (median age 42; 10^{th} & 90^{th} percentiles 24, 64), who were primarily agricultural day laborers (84%). The death-to-case ratio was high (61%). Patients had been regularly intoxicated with ethanol for from 1 to 90 days (median 4 days) prior to illness. The following numbers and percentages of the 159 patients reaching a healthcare facility alive received: hemodialysis (2, 1.2%), ethanol (58, 37%), fomepizole (0, 0%), sodium bicarbonate (11, 7%), folinic or folic acid (33, 21%), and intravenous fluids (126, 79%). Only 4 (2.5%) had a blood pH or serum bicarbonate measurement, and only 1 (0.6%) had blood ethanol and/or methanol measurements made during life. Having visual symptoms or altered mental status at the time of presentation to the health care facility was associated with a fatal outcome ($P = 0.005$ and 0.004, respectively). Conclusions: A large proportion (over 40%) of the 128 deaths occurred before the victim could receive health care. Standard diagnostic and therapeutic maneuvers were infrequently used. Acute visual symptoms and altered mental status heralded a fatal outcome. Improved secondary prevention will require improved access to necessary diagnostic tests, drugs, and equipment and enhanced knowledge among Salvadoran physicians of how to treat methanol poisoning.

180 HYPERTONIC SODIUM BICARBONATE FOR *TAXUS MEDIA*–INDUCED CARDIAC TOXICITY IN SWINE.

Ruha AM, Tanen DA, Graeme KA, Curry SC, Miller MB, Gerkin R, Reagan CG, Brandon T. *Good Samaritan Regional Medical Center, Phoenix, AZ*

Objective: To determine if intravenous (IV) hypertonic sodium bicarbonate is effective in the reversal of QRS widening associated with severe *Taxus* intoxication. Methods: 17 anesthetized swine were poisoned with an IV extract of *Taxus*

media until doubling of the QRS interval on EKG was achieved. After poisoning (time 0), animals received either 4 mL/kg IV 8.4% $NaHCO_3$ (Experimental Group; 6 animals), a similar volume of 0.7% NaCl in 10% mannitol (Mannitol Group; 6 animals), or nothing (Control Group; 5 animals). Main outcome parameter was QRS duration. Secondary outcome parameters were mean arterial pressure (MAP), heart rate (HR), and cardiac index (CI = cardiac output/kg). Additionally, arterial pH, PCO_2, and plasma ionized calcium, sodium and potassium were monitored. Results: *Taxus* toxicity, defined as a 100% increase in QRS duration, was produced in all animals. Animals were similar in regard to baseline and time 0 physiologic parameters as well as amount of *Taxus* extract administered. Swine treated with $NaHCO_3$ had a statistically significant increase in pH and base excess compared with other groups. From time 5 through 30 minutes, following assigned treatment, no significant difference was detected between groups in QRS duration, MAP, HR, or CI. With a 2-tailed á of .05, we had 98% power to detect a 50% narrowing in QRS duration between groups if it would have occurred, but were unable to do so. Conclusion: Hypertonic sodium bicarbonate was ineffective in reversing the widening of QRS interval associated with *Taxus* poisoning in this swine model.

181 SPIDERBITE IN AUSTRALIA : PROSPECTIVE STUDY OF 371 BITES FROM FORMALLY IDENTIFIED SPIDERS.

Isbister GK[1], Gray MR[2]. [1]*Department of Clinical Toxicology, Mater Hospital, Newcastle, Australia;* [2]*Australian Museum, Sydney, Australia*

Introduction: There is little information on confirmed spider-bites in Australia with considerable mythology surrounding spider-bite. Methods: Subjects were recruited prospectively from February 1999 to March 2000. Subjects either presented to participating hospitals or contacted 3 of the 4 Poison's Information Centres in Australia. Subjects were included if they had a confirmed bite and the spider. The spider was identified by an arachnologist. All subjects were followed for at least 1 week. Results: Of 717 enrolled subjects, 371 were included with confirmed bites. Spider families involved included : Araneidae (''Orb-weavers'') (100), Sparassidae (''Huntsman spiders'') (89), Lamponidae (''White-tail spiders'') (52), Theridiidae (including ''Widow spiders'') (29), Lycosidae (''Wolf spiders'') (22), Salticidae (''Jumping spiders'') (16), Desidae (13), Clubionidae (''Sac spiders'') (9), Zodariidae (7), Actinopodidae (''Mouse spiders'') (6) and Hexathelidae (''Funnel web spiders'') (5). Clinical effects were local pain in 98% (severe 24%), erythema in 74% and swelling in 22% of cases. The rate of skin necrosis in the study was 0% (95% CI; 0–1%). Systemic effects occurred in 15% of cases. Analysis of clinical effects and circumstances of bites showed that there were unique characteristics of bites by particular spider groups, which, in the future, would allow determination of spider type in many cases, without spider collection. Conclusion: The study demonstrated minor effects in most spider-bites in Australia. Unique circumstances and clinical effects occurred with different groups of spiders.

182 CROFAB® FOR TREATMENT OF RATTLESNAKE ENVENOMATION.

Ruha AM, Beuhler M, Brooks D, Wallace K, Graeme KA, Curry SC, Gerkin R, Lovecchio F. *Good Samaritan Regional Medical Center, Phoenix AZ*

Background: CroFab antivenin was recently FDA-approved. Methods: Prospectively collected data from 7 CroFab-treated rattlesnake bites seen in March/April 2001. Our treatment guideline, based on package insert, comprised attempts at initial control followed by 2 vials every 6 hrs × 3 doses with repeat outpatient labs 48–72 hrs later. Control was defined as a halt in proximal swelling and normal fibrinogen (fib), PT, and platelet count. Results: 7 pts received 1st doses of 6 or 8 vials in attempts to achieve control, but additional initial vials were given if control was not achieved. 2 pts with fib levels < 35 mg/dL remained hypofibrinogenemic despite 22 and 28 initial vials CroFab. Fib levels then became normal with scheduled dosing of 2 vials q 6 hrs, but both had recurrence of afibrinogenemia within 72 hrs. Both were readmitted and retreated, but neither achieved normal fib despite 12 and 19 additional vials of CroFab. 2 pts had thrombocytopenia which corrected with 6 vials without recurrence. 7 pts had swelling, and we achieved initial control of swelling in all pts with 6 to 16 vials. 2 pts then redeveloped progressive swelling during the next 24 hrs despite receiving 2 vials q 6 hrs. No immediate hypersensitivity reactions or serum sickness has been seen to date. Antivenin expenses and length of hospitalization were much greater than those with past use of Wyeth Antivenin. Conclusion: Initial ''control'' of coagulopathy is frequently difficult to achieve with CroFab, and recurrence of severe coagulopathy developed within a few days of discontinuing CroFab. Swelling can progress after what appears to be initial control, despite regular dosing of antivenin.

Poster Session IV **Monday, October 8** **8:00 am–3:00 pm**
Abstracts #183–#237 **Authors with Posters** **1:30 pm–3:00 pm**

183 IBOGAINE FATALITIES.

Cienki J, Mash D, Hearn W. *University of Miami School of Medicine/Jackson Memorial Hospital, Miami, FL*

Background: Ibogaine (IBO) is an indole alkaloid derived from the root of an African shrub: *tabernathe iboga.* Indigenous peoples use IBO to combat fatigue and high doses to induce spiritual experiences. Research is underway to prove the efficacy of IBO and its metabolite in abstinence from addictive substances. The promises of IBO and its psychoactive effect have lead to a proliferation of illicit sources. Web sites offer iboga and IBO. Addiction treatment centers improperly administer the drug. We offer data from 2 IBO deaths as well as experience in therapeutic dosing. Case one: A 24-year-old female was treated for narcotic withdrawal with 29mg/kg IBO. At 16 hrs, the patient experienced back pain. In an hr, she vomited and 1 hr later died. No attempts at resuscitation were made. IBO and norIBO blood levels were 0.74mg/L and 11.28mg/L. Case two: A 40-year-old male took approximately 85mg/kg IBO prepared by an herbalist, for opiate dependence. Initially, the patient experienced GI symptoms. 24 hrs later he collapsed. Attempts at resuscitation failed. IBO levels were 0.36mg/L. Discussion: In trials patients received 8–12mg/kg IBO and were monitored for 24 hrs. No significant adverse events were seen. The most frequent effects were nausea and tremor. Hypotension noted in cocaine dependent individuals, responded to fluids. Conclusion: 8–12mg/kg IBO has not shown serious adverse events. Larger dosing is associated with delayed toxicity. Our findings suggest large or unknown IBO ingestions should be admitted for monitoring.

184 AN INVESTIGATION OF ACKEE FRUIT POISONING IN HAITI.

Belson M, Joskow R, Kaiser R, Vesper H, Backer L, Rubin C. *CDC/NCEH Atlanta, GA*

Background: In March 2001, investigators in Haiti reported unexplained illness and death in over 100 locals of the Cap Haitian region from November 2000 – March 2001. Symptoms included vomiting, hypoglycemia, and altered consciousness. Similar reports in 1988 and 1991 were possibly caused by the consumption of unripe ackee fruit. Unripe ackee contains a high concentration of the toxin hypoglycin, which can produce the symptoms noted in the recent outbreak. Methods: We reviewed hospital records of patients suspected by Haitian physicians of suffering from the recent illness, established a case definition, and administered a questionnaire for additional case finding to determine exposures, risk factors, and dietary history. Serum and urine samples were collected for analysis of hypoglycin, organic acids, and micronutrients. Results: Medical records were available for 16/23 hospitalized patients. Hypoglycemia ($<$ 35 mg/dL) and vomiting or CNS effects were noted in 11 patients; 2 had only vomiting. Urine was collected from one patient on initial presentation and subsequent analysis revealed marked elevations of organic acids and carnitines (suggestive of hypoglycin toxicity). Based on medical records, our case definition was illness with documented hypoglycemia or vomiting $\geq$ 2 times per hour (without fever or diarrhea). Three of the 16 hospitalized patients were excluded because of fever or diarrhea. A field investigation of the non-hospitalized case patients identified by the local investigators found an additional 48 case-patients. Of the total of 61 patients who met our case definition, the median age was 7 years (6 months–88 years), and 57% died. Ackee was reportedly eaten within 24 hours of symptom onset in 36 cases (59%), and 65% of these had consumed unripe fruit. Conclusions: The consumption of unripe ackee fruit may be responsible for the recent illness in Haiti. Our recommendations are to establish ackee educational programs and begin active surveillance to identify new cases and confirm ackee toxin exposure.

185 PHYSICIAN KNOWLEDGE OF HERBAL TOXICITIES.

Steinfeldt J, Suchard M, Khu J, Suchard J. *Division of Emergency Medicine, UC Irvine Medical Center, Orange CA; UCLA Department of Biomathematics, Los Angeles, CA*

Background: Herbal ''dietary supplements'' are commonly used without an understanding of their potential toxicities and drug interactions. Physicians' knowledge of these topics has not before been formally assessed. Methods: We distributed an anonymous survey and 16-question, multiple-choice quiz about herbal toxicities and herb-drug interactions to attendees at educational meetings of emergency medicine (EM) and internal medicine (IM) physicians. We analyzed the data to determine if significant associations existed between quiz score and amount of clinical experience or self-

assessed familiarity with the subject matter. Results: 142 surveys were completed by 59 attendings, 57 residents, and 26 medical students. Calculated mean quiz score (± SD) was 4.63 ± 2.03, versus an expected mean of 4 from random guessing ($p = 0.0003$). There was a non-significant trend toward higher scores with greater clinical experience; student mean 4.26 ± 2.20, resident mean 4.42 ± 2.05, attending mean 4.98 ± 1.91. No significant difference in scores was found between EM and IM groups. No participants rated their familiarity with the topic as "Good" or "Excellent" on a 5-point Likert-like scale; 73.9% (105) gave themselves a "Poor" rating, 22.5% (32) a "Fair" rating, and only 3.5% (5) a "Moderate" rating. No significant difference in quiz scores was found between those with the median "Poor" rating versus those above the median. Only 9% of participants (14) reported any prior formal training regarding herbal therapies; although this group reported a higher familiarity with the subject matter (median = "Fair"; $p < 0.0001$), they did not score significantly better on the quiz (means of 5.0 vs. 4.59). Conclusions: The EM and IM physicians surveyed have little training or knowledge of herbal toxicities and herb-drug interactions.

186 A CASE OF SEVERE CONSEQUENCES AFTER MISLABELING HERBAL PREPARATIONS.

Martinez-Arrieta R, Ramón MF, Ballesteros S. *Poison Control Center, Instituto Nacional de Toxicología, Madrid, Spain*

Objective: *Illicium anisatum* belongs to Illiciaceae family. This plant is very toxic due to its contents of shikimine and may be confused or mixed with the much less dangerous plant *Illicium verum.* The latter is rich in anethole and is used in traditional medicine as a carminative. We describe the clinical manifestations of a herbal preparation intoxication caused by an error in labeling. Case Report: In March 2001, a thirty-day-old female received 6–7 flowers of "star anise" (6-7 grams in 100 mL of water). The bag containing the flowers was sold in a pharmacy as a carminative product; it was labeled with the words "*Illicium anisatum*" and had no use, dose or administration advises. The baby was admitted into a hospital and our Poison Control Center received the call from the pediatrician. Two hours post ingestion she presented with vomiting, 3–4 seizure episodes, nystagmus and irritability with somnolence phases during 12 hours. We did not know the exact composition of the flowers so we merely recommended supportive and symptomatic treatment with neurological, respiratory, cardiovascular, fluids and electrolytes monitoring, and chemical analysis of the plant. Diazepam (2 mg) was administered. The clinical manifestations resolved in 2 days. Chemical analytic studies showed that the bag contained flowers of *Illicium verum.* We contacted the drug agency authorities of our country in order to take the appropriate measures. Nevertheless high doses of *Illicium verum* have caused pediatric convulsions according to our own experience. Conclusions: Labels of herbal preparations can be confusing not only in the proper name but also in the lack of use and administration recommendations. We want to highlight the significant role of toxicological surveillance of the Poison Control Centers in herbal potential risks.

187 STRATEGIES OF MUSHROOM POISONING MANAGEMENT IN A PCC.

Ballesteros S, Ramón MF, Martínez-Arrieta R, Iturralde MJ. *Spanish Poison Control Centre, Instituto Nacional de Toxicología, Madrid, Spain*

Introduction: The principal question when assessing a patient following an exposure to a mushroom should be whether it is a toxic mushroom Identification remains the best method to answer this question although not always properly accomplished so PCC has to manage it in a different way. The purpose of this project was to describe our experience. Methods: A retrospective review of documented cases of poisoning from 1991 to 2000 was performed. Data including patient age, mushroom type, clinical presentation, prognosis, and seasonal distribution of the exposures were recorded. Results: A total of 355 mushroom exposures were reported, 84.5% of them in adults and 50.7% in groups. Seasonal distribution of cases was clearly demonstrated since 75.2% of them occurred between September and December. A percentage of 32% of the specimen were properly identified with the Latin name; in 6.4% of cases the common name was given to our centre. On the other hand the substrate of growth of the specimen was given in 6% of consults but this mostly did not help to identification. 24.8% of the patients were asymptomatic at the moment of the consult. According to the toxins or symptoms, the global incidence was: Gastrointestinal (31%), delayed gastrointestinal symptoms and/or hepatotoxicity (24.5%), muscarine (14.4%), muscimol (4.5%), hallucinogenic (4.5%) and coprine (1.4%). At the end only 13.4% of all mushrooms exposures were not classified. Most patients had a favorable outcome with no long-term morbidity except one death. Conclusion: Our data show a general picture of what is occurring in a PCC. Clinical features of toxicity remain our best weapon to discriminate between low-risk and high-risk mushrooms. Nevertheless when one cannot exclude the possibility of a toxic species of mushroom in the exposure, the patient should be referred for evaluation.

188 BEWARE OF SALICYLISM AND HERBAL MEDICINES.

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

Background: A deluge of herbal medicines have become readily available in the United States. These can be purchased in ethnic groceries, by mail order and on the Internet and also brought in to the country by visitors and immigrants. Due to ambiguous labeling, sometimes in a foreign language, it is difficult, if not impossible to determine the ingredients in some of these products. This raises concerns about their potential danger and appropriate treatment from both acute and chronic exposures. We report an unusual overdose in a demented patient from a product called Red Oil Chinese containing a high concentration of methyl salicylate. Case Report: A 72-year-old Vietnamese male with a history of dementia arrived in the emergency department 5 hours after ingesting 2 ounces of a product called Red Oil Chinese. The label was in Chinese and initially there was no one available to translate the ingredients. He had diarrhea and hyperventilation and based on these symptoms, salicylism was considered. His initial salicylate level was 56 mg/dL with a peak at 10 hours post exposure of 64.5 mg/dL. The label was later translated to reveal a methyl salicylate concentration of 70%. It also contained turpentine oil, palm olean, and cinnamon oil. The patient recovered over the next 24 hours with supportive therapy including IV fluids, sodium bicarbonate and potassium. Conclusion: With the increasing prevalence of alternative medicines and the frequent inability to evaluate ingredients from the label, the presence of salicylates should be considered if the symptoms are consistent with salicylate toxicity.

189 ACUTE HEPATITIS INDUCED BY KAVA KAVA, AN HERBAL PRODUCT DERIVED FROM *PIPER METHYSTICUM.*

Humbertston CL, Akhtar J, Krenzelok EP. *Pittsburgh Poison Center, Children's Hospital of Pittsburgh, University of Pittsburgh Schools of Pharmacy and Medicine, Pittsburgh, PA*

Background: Herbal preparations are widely available and generally regarded by the public as harmless remedies for a variety of medical ailments. We report a case of acute hepatitis associated with the use of Kava Kava, derived from the root of the pepper plant, *Piper methysticum.* It is used in the United States as an anti-anxiety and sedative agent. Case Report: A previously healthy 14-year-old female was admitted to the hospital with fulminant hepatic failure. Her admission liver function tests were markedly abnormal. Initial therapy was unsuccessful and she continued to deteriorate. Plasmapheresis was of no benefit. She ultimately required a liver transplant and now remains well. The liver biopsy showed hepatocellular necrosis consistent with chemical hepatitis. A work up of alternative causes of liver failure was negative. The patient gave a history of taking a Kava Kava-containing product for 6 months. She stopped taking it for a month and then resumed its use. The use of Kava Kava and liver failure is supported by its use, a negative work up of alternative causes of liver failure, and histological changes in the liver. Conclusion: Health care professionals need to be aware of the possibility of Kava Kava-induced hepatotoxicity. The toxicity of these allegedly 'natural' remedies underscores the importance of surveillance programs and quality control of the manufacture of these products. Clinicians must remain aware of the toxic potential of herbal products and always inquire about their intake in cases of unexplained liver injury.

190 A FATALITY FROM *PHYTOLACCA AMERICANA* (POKEWEED) ROOT INGESTION.

Brooker J, Obar C, Courtemanche L. *New Hampshire Poison Information Center, Dartmouth Hitchcock Medical Center, Lebanon, NH*

Background: *Phytolacca americana* is a well known toxic plant. The root of *Phytolacca americana* contains the highest concentration of toxins including triterpene saponins and glycoproteins. Ingestion of these toxins generally cause gastroenteritis. Pokeweed also contains mitogens that normally induce a plasmacytosis. The white root of pokeweed can be readily mistaken for parsnip, or horseradish. We present a fatality due to ingestion of pokeweed root. Case Report: An 18-year-old, previously healthy male, while landscaping, dug up and ate a 4–5 inch piece of white root. He mistook this for parsnip. Within 45–90 minutes he complained of epigastric pain and vomited. Approximately 2 hours post ingestion he collapsed at home and an ambulance was summoned. He was found to be in ventricular fibrillation. Despite aggressive ACLS treatment, resuscitation efforts were unsuccessful. A botanist identified the plant, including the root ingested, as Phytolacca americana. Discussion: To date, there have been no deaths reported to the AAPCC following ingestion of pokeweed. No cardiac toxin has been identified despite the development of bradycardia, Mobitz type 1 heart block, tachycardia, hypotension and v-fib in some patients. Cardiac effects may be related to a parasympathetic

response to severe GI effects, but this remains unproven. Conclusion: There is now a documented fatality related to the ingestion of pokeweed root. More aggressive public education is needed to heighten awareness about the serious consequences of ingesting unknown plant roots. Health care providers should not only be aware of the well known effects of pokeweed ingestion, but also the potential for life-threatening toxicity when the root is ingested.

191 CONTRARY TO THE LITERATURE, VOMITING IS NOT A COMMON MANIFESTATION ASSOCIATED WITH PLANT EXPOSURES.

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Background: A review of clinical toxicology references and the contemporary literature illustrates that vomiting is reported as a nearly universal symptom associated with exposure to cultivated and wild plants. However, the literature fails to put the symptom of vomiting into a clinical perspective with regard to its frequency of occurrence. The ambiguity results in the dissemination of this admonition to those who call poison centers about plant ingestions. To clarify this issue a retrospective review of plant exposures was conducted. Methods: AAPCC TESS 1997–1999 was queried electronically to identify and extract all plant ingestion exposures where vomiting was documented as being related to the exposure. All exposures with documented ipecac syrup use were excluded. Data were stratified through the use through the use of a relational database. Results: Plant ingestion exposures accounted for 229,538 reports and vomiting was reported in 5,917 (2.6%) of the exposures. Vomiting was attributed as a related symptom in 753 different plants. It was reported one time in 323 plants, 1–3 times in 495 plants (65.7%) and $\leq$ 10 times in 657 (87.4%) of all plants in the database. Ten plants accounted for 32.3% of the reports of vomiting (1. Philodendron; 2. Spathiphyllum; 3. Narcissus; 4. Dieffenbachia; 5. Phytolacca; 6. Epipremnum; 7. Euphorbia; 8. Eucalyptus; 9. Ficus; 10. Hedera) and represented 1.3% of the plants associated with vomiting. Conclusion: Even with the most common plant exposures, vomiting is not a frequent adverse event.

192 A HUMAN FATALITY FROM "GRAZING" ON A YEW PLANT.

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Background: Despite the reputation of the Yew plant (*Taxus* spp.) for being inordinately toxic when ingested, a recent 10-year retrospective poison center study suggested significant morbidity is rare. This case illustrates that mortality can occur. Case Report: A 45-year-old male was seen "grazing" on a yew plant in the yard of his residential center. Past medical history was significant for developmental delay and pica throughout adulthood. There was no indication that the yew ingestion was suicidal in nature. One hour later the patient was found on a couch in cardiac arrest; resuscitation efforts by EMS and ED staff were unsuccessful. Gross postmortem examination demonstrated no evidence of trauma and was significant for mild hydrocephalus and berries, leaves, twigs, and sticks matching the description of a yew in the stomach and intestines. Thorough postmortem toxicologic analysis of the patient's blood and bile was significant only for 3,5-dimethoxyphenol in the blood (21 ng/mL) and bile (104 ng/mL). 3,5-dimethoxyphenol is the aglycone of taxine, the toxic alkaloid of the yew plant. Discussion: Although this patient was witnessed to be "grazing" on a yew plant shortly before his death, intoxication by taxine was not considered until undigested fragments of the yew were found on postmortem examination. Unlike previously reported fatalities thought to be linked to yew ingestion, systemic absorption was confirmed by toxicologic analysis and other causes of death were ruled out. Although significant morbidity is rare, this case confirms that a significant ingestion of the yew can lead to death.

193 RETAINED GASTRIC LEAD FOREIGN BODY RESULTING IN MARKEDLY ELEVATED BLOOD LEAD LEVELS IN TWO CHILDREN.

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Background: Previous human case reports of acutely ingested lead foreign bodies have documented only mild elevations in blood lead levels. We report 2 patients who ingested single lead foreign objects that resulted in markedly elevated blood lead levels. Case 1: A 3-year-old swallowed a lead musket ball. He was brought to the emergency department

(ED) 2 days later where he was asymptomatic and a x-ray revealed the ball retained in the stomach. The lead ball was removed by endoscopy without complication. A 48-hour post ingestion venous lead level (VLL) was at 89 mcg/dL. Case 2: A 3-year-old swallowed a lead-fishing sinker. He was evaluated in the ED 24 hours later, was asymptomatic, and a x-ray revealed it retained in the stomach. The sinker was removed by endoscopy without complication. A 36-hour post ingestion VLL was at 48 mcg/dL. Both children remained asymptomatic and chelation with succimer was performed. A repeat VLL drawn prior to initiation of chelation for child 1 was 44 mcg/dL. A VLL 33 days post ingestion (8 days following completion of chelation) was 22 mcg/dL. Child 2 had a postchelation VLL of 18 mcg/dL. Our toxicology service and the health department performed a complete environmental evaluation with no other lead sources identified. Conclusion: These cases demonstrate that acute marked elevation of blood lead levels may occur following ingestion of a single lead foreign body, especially if they remain retained in the stomach. Endoscopic removal should be considered in these cases.

194 LEAD ENCEPHALOPATHY IN A CHILD TREATED WITH WHOLE BOWEL IRRIGATION (WBI) AND COMBINATION Ca_2Na_2EDTA AND DMSA.

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Objective: This case report describes the successful use of whole bowel irrigation in a child with lead encephalopathy, allowing early and successful combination EDTA and DMSA therapy. Case Report: A 13-month-old girl presented in November 1999 with a one-month history of vomiting and lethargy. Soon after arrival in the ER she had a convulsion. Initial blood results showed Hb 7.7g/dL, MCV 67fL, blood lead of 244μg/dL, renal function was normal. She had a history of pica of paint (paint lead 16.6% on subsequent analysis) and an abdominal XR (AXR) showed multiple flakes of paint throughout the GI tract. She was intubated and ventilated and transferred to the PICU. Initial chelation was with intravenous EDTA at a dose of 40mg/kg/d for ten days. She received WBI with polyethylene glycol at a dose of 500mL/hr for 36 hours; a repeat AXR was clear of paint flakes and she was commenced on oral DMSA at a dose of 10mg/kg twice daily for five days followed by 10mg/kg twice daily for fourteen days. Her urinary lead on combination therapy with EDTA and DMSA was 166000μg lead/g creatinine. She was extubated on day 2 and discharged from PICU on day 5. Initially she had a tremor and hyper-reflexia, but by discharge from hospital on day 24 she was walking and her blood lead had fallen to 41μg/dL. Over the last sixteen months she has required a further six courses of chelation with DMSA and her current blood lead is 30μg/dL; she has continued to have normal physical and neurological development. Conclusions: Whole bowel irrigation resulted in clearance of paint flakes from the GI tract over a thirty six hour period and allowed early combination EDTA and DMSA chelation therapy.

195 ELEVATED BLOOD LEAD LEVELS ASSOCIATED WITH THE CONSUMPTION OF ILLICITLY DISTILLED MOONSHINE.

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Background: In February and March of 2000, 4 moonshine consuming patients were diagnosed with lead toxicity. This suggested that lead exposure from moonshine was under-recognized. Methods: We conducted a retrospective cohort study over a 14-day period. A total of 581 patients were screened for moonshine consumption. A full interview and blood lead analysis were conducted on all moonshine consumers (N = 35) and a randomly selected comparison group of non-moonshine consumers (N = 68). Results: 8.6% of our patients reported that they had consumed moonshine in the past five years. Moonshine drinkers were predominately male, 40–49 years of age and reported heavy alcohol use. The median blood lead levels in moonshine drinkers and nonconsumers were 11.0 μg/dL and 2.5 μg/dL respectively. Moonshine consumers were significantly more likely to have elevated blood lead levels ≥ 10 μg/dL (Odds Ratio = 10.94 95%, CI 3.76–31.85). Patients who consumed moonshine in the previous week were significantly more likely to have a blood lead level ≥ 10 μg/dL than individuals who reported more remote consumption. Patients who consumed moonshine more than once per month were significantly more likely to have a blood lead level ≥ 10μg/dL than those reporting less frequent exposure. Conclusions: Moonshine consumption was strongly associated with elevated blood lead levels particularly among recent consumers.

196 ENDOGENOUS LEAD STORES IN RETIRED LEAD WORKERS: AN ARGUMENT FOR MONITORING.

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Background: The toxicokinetic behavior of lead results in lead accumulation in bone with a half-life substantially longer than that in blood. Lead in bone may be a source for endogenous exposure during periods of bone demineralization, yet many believe that in the bony matrix, lead is inert and stable unless specific conditions (surgery, pregnancy, bony trauma) intervene. We report a retired lead battery worker with career-long blood lead elevations who was not exposed to lead for over five years and developed severe elevations in blood lead levels (BLL). Case Report: A 65-year-old male was employed in the lead battery industry for 35 years. Work records documented monthly BLL monitoring with consistently elevated BLLs (> 40 mg/dL). Five years after retirement, he began to experience arthralgias. Serial determinations demonstrated BLLs as high as 100 mg/dL requiring oral chelation therapy. A painstaking search (by history) failed to uncover ongoing exposures to lead. Testing of cohabitants was negative for lead. Conclusion: The release of lead from bony stores due to age related bone remodeling can cause elevations in BLLs. In the aging lead worker, large bone burdens may exist, thus large amounts of lead may be released into the blood in these individuals. An epidemic of endogenously derived lead exposures may occur as this segment of the workforce ages. We recommend monitoring of retired lead workers for elevations in BLLs as a new and essential public health initiative.

197 LEAD CONTAMINATED DRINKING WATER IN PHILADELPHIA PUBLIC SCHOOLS.

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Background: Lead exposure is an important health concern for children and lead contaminated drinking water is a potential source of lead exposure. We report the results of lead level determinations in Philadelphia public school drinking water as well as remediation efforts aimed at dealing with this public health concern. Methods: Water samples were collected from drinking sources in 290 school buildings in Philadelphia from May, 2000 through January, 2001. These samples were sent to a reference laboratory for determination of lead levels. Results: The mean levels of lead in drinking water (in parts per billion) found are summarized below:

School buildings	Mean Water Lead Level (ppb)
102 (35.2%)	3.0–14.9
112 (38.6%)	15.0–49.9
29 (10.0%)	50.0–99.9
47 (16.2%)	> 100

Conclusions: 64.8% of Philadelphia public school buildings had water containing mean lead levels exceeding current Environmental Protection Agency (EPA) action levels (15 ppb). 26.2% of school buildings had water with mean lead levels in excess of 50 ppb. Depending on the volume of water consumed by a given individual, drinking water from Philadelphia school buildings may be an important source of lead exposure for school children. School buildings in other urban areas may have similar water lead levels and testing programs in other locales is desirable.

198 SYSTEMIC ILLNESS FROM SUBCUTANEOUS ELEMENTAL MERCURY INJECTION.

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Background: Many cases of subcutaneous (sc) elemental mercury (Hg^0) injection resulting in skin granulomata are reported, but there is little associated systemic illness. We report a case of debilitating illness in a patient with repeated sc Hg^0 injections. Case Report: A 31-year-old Belizian woman complained of lumps on her fingers. Other symptoms included weight loss, nausea, weakness, and oral pain. She reported multiple injections of Hg^0 sc in the mouth and small joints of the hands and feet over 3 years, done for medicinal purposes. On exam, she had cachexia, granulomatous

nodules on her face, digits and extremities, severe stomatitis in areas of oral injection, and a normal neurologic exam. Radiographs and CTs revealed Hg deposits in the soft tissues of the hands, face, palate, tonsils, ears, eyelids, and occiput. Punctate deposits of Hg occurred in the liver, spleen and lung periphery. The patient had a hypoproliferative anemia, low CD4 count, and impaired gas exchange (DLCO 52 % predicted). Work-up for autoimmune and HIV disease was negative. Biopsy of a nasal bridge lesion showed suppurative and granulomatous inflammation surrounding dense droplets. One month after her last injection, blood Hg was 3.38 μg/gm blood, and urine Hg 11,000 μg/L. The blood Hg declined over several months, but eventually returned to a similar level. Five months after last injection, she developed proteinuria. The skin lesions responded to topical steroids, but new lesions continue to appear. Months after the treatment of the stomatitis, she remains cachectic. The safety of chelation in this patient is questioned, and she has not been chelated. Conclusion: Hg^0 injection has resulted in a wasting illness, with recurring skin granulomata, anemia, impaired cell mediated immunity, impaired gas exchange, and renal pathology, but no evident CNS disease. The patient's blood Hg levels are among the highest reported.

199 ACUTE ZINC TOXICITY IN A CHILD RECEIVING TOTAL PERIPHERAL NUTRITION (TPN).

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Background: Zinc is a naturally occurring trace element that must be maintained to ensure proper cell growth and repair. Zinc toxicity rarely occurs and most cases are related to accidental ingestion of zinc salts. Case Report: A three-month-old child with a history of congenital short-gut syndrome was admitted and received TPN formulated by the hospital pharmacy. After two days, she become hypotensive, obtunded and apneic. She was also noted to have varying degrees of heart block and a PR interval prolongation greater than 0.5 msec. Laboratory findings were compatible with sepsis and showed renal insufficiency, elevated liver enzymes, decreased hemoglobin, hemolysis and thrombocytopenia. Antibiotics, fluids and pressor agents were instituted. After further review it was determined that she had received TPN containing 200 mg of zinc chloride (normal 200 mcg) for two days. Blood zinc was measured at 41.4 mcg/mL (normal 0.66–1.05 mcg/mL). The patient received four daily doses of intravenous calcium EDTA and showed improvement in renal and liver function. Results: Blood zinc levels were monitored and remain elevated above normal. Chelation therapy reduced the blood level to 5.4 mcg/mL after four days of administration. Conclusion: Although rare, zinc toxicity can cause serious life-threatening problems. Calcium EDTA can be used to quickly and safely lower zinc blood levels in seriously intoxicated patients. Zinc toxicity should be considered when patients receiving TPN develop abrupt life-threatening symptoms involving multiple organ systems.

200 ZINC TOXICITY ASSOCIATED WITH MASSIVE COIN INGESTION.

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Background: Since 1982, pennies consist of 99.2% zinc and 0.8% copper. Massive human coin ingestion resulting in clinical zinc toxicity has only been reported once. Case Report: A 47-year-old male history of schizophrenia and AIDS, developed 2 episodes of syncope during a 24 hour period. He reported progressive generalized fatigue and vomiting over a 3-week period. He had a previous history of coin ingestion. Work-up revealed severe sideroblastic anemia (hematocrit = 10%), renal insufficiency (creatinine = 1.9 mg/dL), and mild transaminase elevation. Severe copper deficiency was diagnosed [serum copper = 7 mcg/dL (normal range 70–155)]. Secondarily, zinc toxicity was discovered [(serum zinc = 2891 mcg/dL (normal range 60–130)]. The abdominal X-ray revealed numerous coins, distributed throughout the GI tract. Endoscopy revealed inflammation of gastric mucosa, and numerous coins that were not amenable to removal. Treatment included chelation with calcium disodium ethylenediaminetetraacetate ($CaNa_2EDTA$), copper supplementation, and gastrotomy for coin removal. Two hundred and ten pennies, one nickel and one dime were removed. About 50 coins, inaccessible to removal at gastrotomy, were advanced into the colon during surgery. Post-treatment, zinc levels declined and renal function and anemia improved. Conclusion: Zinc toxicity should be considered in patients with history of massive coin ingestion. Zinc and copper have complex interactions at absorptive sites. Furthermore, elevated zinc levels appear to reduce effective copper utilization. Patients may present with sideroblastic anemia or other clinical manifestations of copper deficiency due to zinc toxicity.

201 ASYMPTOMATIC COLONIC IRON BEZOAR DESPITE 3 DAYS OF WHOLE BOWEL IRRIGATION.

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Background: Concretion formation is a documented complication of large iron ingestions. The accepted preventive and therapeutic treatment is whole bowel irrigation (WBI) to a clear effluent, and deferoxamine in cases of systemic toxicity. While experiments suggest that iron is poorly absorbed in the colon, no case reports of iron overdose have shown a lack of systemic toxicity despite retained colonic bezoar. Case Report: A 16- month-old male presented to an Emergency Department 19 hours after an iron ingestion. Vital signs on arrival were stable and initial laboratories revealed an anion gap of 20mEq/L, and a 20 hour serum iron level of 429 mcg/dL (*nl 65–175mcg/dL*). A KUB showed multiple pills throughout the stomach and small bowel and WBI was initiated. Deferoxamine was administered at a rate of 10mg/kg/hour and then stopped when the serum iron level reached 27mcg/dL 36 hours later. At this time, the KUB showed an iron bezoar remaining in the ascending colon despite a clear rectal effluent from WBI. Over the next 36 hours, the serum iron level never became elevated and the iron bezoar actually migrated proximally in the colon despite continued WBI. On the third day WBI was stopped and a normal diet was instituted with prompt passage of the bezoar. Conclusion: We present a case in which a large iron ingestion resulted in a retained colonic bezoar that did not lead to significant iron absorption. WBI did not incite passage of the iron bezoar but a normal diet did. This case report calls into question the efficacy and necessity of WBI for the clearance of *colonic* iron bezoars.

202 FINDING THE ELUSIVE PRUSSIAN BLUE FOR THALLIUM TOXICITY.

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Background: Prussian Blue (PB) has been shown to decrease the elimination half-life of thallium by more than 50% and to decrease mortality in animal models. Partly due to PB's lack of FDA approval and its lack of availability in pharmaceutical grade in the US, it is likely underutilized in thallium poisoning. We present a case of thallium poisoning and describe our means used to rapidly obtain PB. Case Report: A 27-year-old male presented to the emergency department (ED) 5 days after ingesting an estimated 2000–3000 mg of thallium. He complained only of diffuse abdominal pain and nausea. Admission vital signs were within normal limits. The patient received activated charcoal. PB was unobtainable from local pharmacies and pharmaceutical vendors. During the ensuing days, the patient developed severe painful dysethesias in both lower extremities that precluded ambulation. We were able to obtain PB (Radiogardase®) from Radiation Emergency Assistance Center (REAC) in Oakridge Tennessee within 72 hours of our request and by day 5 of hospitalization. The patient received doses in the range of 1.5 grams of PB orally three times a day for approximately 2 weeks. His blood thallium levels peaked at 1084 μg/L. PB was stopped when his blood level dropped below100 μg/L, and the patient was discharged. The patient had some resolution of dysethesia but eventually developed diffuse alopecia. Conclusion: Prussian Blue can be obtained rapidly within the US. It is a useful antidote for thallium poisoning and has few risks. Obtaining the substance early may enhance elimination and decrease toxicity. REAC (www.orau.gov/reacts/prussian.htm) rapidly assisted in obtaining Prussian Blue.

203 SURVIVNG A BARIUM SULFIDE INGESTION.

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Background or Objective: Barium sulfide, an ingredient of depilatories, is a water-soluble barium salt that is highly toxic ($LD_{50} \sim 1$ g). After oral absorption, barium is rapidly sequestered in the skeletal muscle cells and then blocks the exit channel for potassium ions. This produces a profound hypokalemia characteristic of barium intoxication. Barium also stimulates striated, smooth and cardiac muscle resulting in peristalsis, arterial hypertension and cardiac arrhythmia. Case Report: An adult female prisoner ingested an unknown amount of depilatory ($CaOH_2$ 25%, $BaSO_2$ 25%, pH = 12) mixed with water 12 hours prior to PC (poison center) notification. The patient was complaining of difficulty swallowing and throat pain. She had an extensive psychiatric history, her concurrent medicines were valproic acid and sertraline. PC encouraged squad transport to ED for evaluation of symptoms. Six hours after the initial call patient had severely deteriorated. Potassium (K+) was 1.6 mEq/L; she was having cardiac arrhythmias and required intubation and ventilation. A K+ infusion was started at 10–15 mEq/hour with K+ levels ordered every 4 hours. At follow up 5 hours later, the K+ was only 1.7 mEq/L. Patient was now in NSR but still requiring respiratory support. Twenty-

nine hours post ingestion, patient's K+ stabilized to 4.3 mEq/L. Endoscopy was performed 2 days later and mild to moderate esophagitis was visualized. Patient was extubated 3 days post ingestion and was tolerating PO on day 6. She was discharged back to prison 7 days post ingestion with mild esophagitis and mild gastritis. Conclusion: Only a handful of case reports document patients that survive soluble barium salt ingestion. We report a prisoner that survived ingesting an unknown amount of corrosive barium sulfide containing depilatory.

204 A CASE OF SERIOUS BISMUTH POISONING TREATED WITH 2,3-DIMERCAPTOPROPANE-1-SULPHONATE (DMPS).

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Background: Previous published case reports describe acute renal failure with severe acute bismuth poisoning, but this case report demonstrates that, with early use of DMPS, renal impairment can be avoided. Case Report: A thirty-year-old man presented two hours after ingestion of 40 De-Nol tablets (4.8 g tripotassium dicitratobismuthate). On admission he was vomiting, examination was unremarkable. His renal function was normal, his initial blood bismuth was 42.4 μg/dL and urine bismuth was 1000 μg/dL. Chelation with oral DMPS was started on day 2 at a dose of 200 mg four times daily for ten days followed by 200 mg twice daily for a further ten days; the DMPS was well tolerated with no adverse events. His renal function remained normal throughout and he developed no further clinical features of bismuth poisoning. At the end of the course of chelation his blood bismuth had fallen to 1.7 μg/dL and his urine bismuth was 3.7 μg/dL. A month later his blood bismuth was 1.2 μg/dL, urine bismuth was 2.3 μg/dL and he remained well with normal renal function. Conclusions: We describe a case of severe bismuth poisoning is which acute renal failure was avoided with the early use of chlelation with DMPS. DMPS was well tolerated and should be considered in all cases of significant bismuth overdose.

205 MAGNETIC RESONANCE IMAGES IN THE FOLLOW-UP OF OCCUPATIONAL MANGANESE-EXPOSED WORKERS.

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Objective: High signal intensity of several regions of the brain is observed on T1-weighted Magnetic Resonance Imaging sequences (MRI) in Parkinson-like syndrome due to manganese (Mn) intoxication. This signal is also described in asymptomatic workers with high blood Mn concentration (MnB) exposed to low levels of Mn. It could be a predictive factor for neurological disorders. As the neurological damages induced with Mn are irreversible even if the exposure has ceased, it would be interesting to know if MRI could be used in the medical follow-up of occupational Mn-exposed workers and if the signal intensity is correlated with other biological or environmental parameters. Methods: 9 active and former workers at a braking Mn and iron factory had to answer to a standardised questionnaire to evaluate exposure and search for subjective neurological symptoms. Environmental and biological parameters (MnB), physical examination, cerebral MRI and neuropsychological tests when possible were conducted. Results: 2 retired subjects with 22 years exposure were asymptomatic and had normal results; one of them had had a high signal on globus pallidus on MRI two years before when he was active. 7 active exposed workers had a 4-years mean length of exposure at an average airborne Mn concentration of 28 mg/m^3 and a mean MnB of 27 μg/L ($N < 10$ μg/L); All had an increase in signal intensity on T1-weighted sequences on the globus pallidus that seemed correlated with MnB. The subject with the highest MnB had physical and neuropsychological disorders. Conclusion: High signal intensity on T1-weighted MRI reflects recent Mn exposure as well as high MnB, and these two parameters seem to be correlated. High signal intensity disappears when the exposure stops. These results are consistent with the literature data.

206 ACRODYNIA AND ASTHMA IN ADOLESCENCE.

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Background: Overt mercury poisoning is uncommon in modern western society. Still less prevalent is the toxic-allergic syndrome of acrodynia (Swift's disease). Characteristic manifestations include swelling, erythema, pain, and desquamation of the hands and feet. Rash, gingivostomatitis, and hypertension may be noted. While specific immunologic mecha-

nisms are not clear, the syndrome appears unique to children. Case Report: A 16-year-old male with previously mild asthma presented to the emergency department with 2 days of rash, dyspnea, cough and fever. Symptoms were unrelieved by his usual asthma therapy. He also noted headache, dizziness, insomnia, vomiting, and cold intolerance in his feet. Examination was remarkable for wheezing and diffuse milial rash, with desquamation of the digits. A thorough environmental history disclosed significant inhalational exposure to elemental mercury. Initial blood mercury was 20 mcg/L, while a 24-hour urine mercury was 651 mcg/L. He was treated with succimer, but continued to excrete significant amounts of mercury for months. Headaches, insomnia, refractory asthma, and lower extremity dysesthesia persisted at 9-month follow-up. Conclusion: Mercury toxicity, while uncommon, may manifest as an unusual immunologic syndrome in children. Delayed elimination may prolong toxic manifestations. Diagnosis of this now rare toxic entity requires that the clinician perform a vigilant environmental history to identify exposure and determine precipitating causes.

207 GOLD-INDUCED PERIPHERAL NEUROPATHY WITH RECHALLENGE.

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Background: Gold salts are frequently used in the treatment of rheumatoid arthritis. Common side effects include proteinuria, rash and thrombocytopenia. Peripheral neuropathy is a rare complication of gold administration. Typical features include weakness, distal sensory loss, loss of deep tendon reflexes, elevated CSF protein, axonal loss and sometimes demyelination on nerve biopsy. It is generally reversible, in contrast with peripheral neuropathies associated with rheumatoid arthritis. Case Report: A 50-year-old male with rheumatoid arthritis was placed on gold salts and at a total dose of 545 mg noticed weakness of his lower extremities. On examination, he was areflexic, had some muscle weakness in both upper and lower extremities, but had no decrease in sensation or in vibratory or position sense. There was no proteinuria or thrombocytopenia. His lumbar puncture revealed elevated protein. Nerve conduction velocities were decreased as were amplitudes; no myopathic features were seen. A muscle biopsy revealed focal atrophy, myxomatous degeneration of nerve fibers and no arteritis. Gold was stopped, and he was treated with tapering doses of prednisone. After 6 weeks, he had no neurologic sequelae. Nine years later, he was taking prednisone, 10 mg. a day, and was again started on gold. At a total dose of 500 mg, he noted weakness in his upper extremities and paresthesias in his hands and feet. On neurologic examination, he was areflexic, had upper and lower extremity weakness, with decreased vibratory sense and light touch. There was no proteinuria or thrombocytopenia. No LP was done. Nerve conduction showed decreased slowing and amplitudes, and again no myopathic features. No biopsy was performed. Gold therapy was discontinued, and after 8 weeks, he had no neurologic deficits. Conclusion: This is the only known report of gold peripheral neuropathy with inadvertent rechallenge. Neurotoxicity recurred following administration of the same total dose of gold.

208 PROLONGED QT AND POLYMORPHIC VT WITH CHRONIC CESIUM USE.

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Background: Cesium is gaining popularity as a homeopathic treatment for various cancers and other conditions. There are no reports of chronic cesium therapy causing toxicity in humans in the literature, although it has been used experimentally to duplicate Long QT Syndrome in animals. We report a case of a patient with cardiac toxicity and electrolyte imbalance while on cesium therapy. Case Report: A 39-year-old female was given cesium powder, 1/4 tsp. in water BID, by a homeopathic practitioner to "shrink away" her uterine fibroids. She had been doing this for 3 weeks when she presented to the ED. Prior to presentation she experienced 3 syncopal episodes. The third episode was witnessed. No seizure activity was noted. Cardiopulmonary resuscitation was performed until a pulse returned. Upon presentation to the ED she was alert and oriented, BP 114/48, HR 58, RR 20. Labs upon presentation included: Na 139, K 3.1, Mg 1.4, BUN 11, SCr 1, and glucose of 168. The EKG revealed a markedly prolonged QT interval at 656 msec and QTc at 622 msec. She was admitted to telemetry and K and Mg were aggressively replaced. During this time she had several episodes of nonsustained polymorphic ventricular tachycardia. Her QTc remained prolonged for 9 days and did not reach baseline for 3 weeks, during which time she required continued replacement of Mg and K. Twenty-four hour urine collections were obtained and cesium levels were 768 mg/spec and 673 mg/spec on days 2 and 8 of admission, respectively. On follow-up at one month, the patient continued to require K and Mg replacement. Her QTc was 424 msec. Conclusion: This is the first reported human case of chronic cesium therapy causing Mg and K wasting, QT prolongation and ventricular tachycardia.

209 AIRWAY ABNORMALITIES ASSOCIATED WITH SICK BUILDING SYNDROME.

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Objective: To objectively assess airway abnormalities in the Sick building. Methods: Standardized histories and physical examinations, record reviews, PFTs, rhinomanometry, nasal smears, and fiberoptic rhinolaryngoscopy were performed. Industrial hygiene reports were reviewed. Results: 33 patients with illness arising while employed in a poorly ventilated office building were examined. Mean age was 38 (range 18 to 60), with 7 men and 26 women. Complaints associated with the workplace included asthma (100%), rhinosinusitis (100%), fatigue (97%), headache (94%), difficulties with memory (79%), mental confusion (48%), and difficult menses (64% of women less than 45 years-of-age). 61% reported weight gain during the period of employment. Obesity was common, with 45% weighing more than 100 kilograms. Prior or current treatment with beta agonist inhalers, steroid inhalers, and montelukast was universal. PFTs were abnormal in 60% without discontinuation of medications. Nasal resistance was abnormal in 93% (mean right 1.39 ± 0.18 Pa/cm3/sec, range 0.45 to 4.41 Pa/cm3/sec, left 1.12 ± 0.14 Pa/cm3/sec, range 0.32 to 4.41 Pa/cm3/sec). Rhinolaryngoscopy was abnormal in the 29 of 33 who underwent the evaluation, with findings characteristic of chemical irritant rhinitis. Discoloration of mucosa with injection, cobblestoning, edema, and injection of the uvula and soft pallet was common. Nasal smears showed lymphocytes. 3 were still employed in the building, 4 were employed elsewhere, 10 were unemployed, and 15 were on medical leave. Industrial hygiene evaluations documented poor to no fresh air exchange. Volatile organic chemicals were not measured. Conclusion: Asthma, rhinosinusitis, fatigue, headaches, difficulty with memory, difficult menses, weight gain, and objective measures of airway inflammation were found in association with employment in a sick building.

210 MULTIPLE THROMBOTIC OCCLUSIONS OF VESSELS AFTER RUSSELL'S VIPER ENVENOMATION.

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Background: Systemic bleeding due to consumption coagulopathy and thrombocytopenia resulted from activation of procoagulants from Russell's viper venom (RVV) is the leading manifestation and cause of death in Russell's viper systemic envenoming. Thrombotic occlusion of vessels is rare in cases of snakebite. Here, we report two cases of RVV systemic envenoming presenting with multiple cerebral infarctions and digital gangrenes. Case Report: A 67-year-old male presented with delirium, hemolysis and oliguria six hours after being bitten by a Russell's viper over his right hand. Multiple cerebral infarction and digital gangrene were noted in addition to acute renal failure and systemic bleeding. Specific antivenin was administrated 4 days after systemic envenoming. He recovered with neurological sequelae and loss of multiple toes. Another 52-year-old female had altered consciousness and bleeding from several organs 2 hours after snakebite. Twenty-four hours later, Russell's viper was identified and specific antivenin was used. But it was too late to restore normal vital functions due to multiple cerebral infarctions and suspected diffuse ischemia in multiple organs. The patient passed away due to multiple organ failure and recurrent septic conditions after 49 days' intensive treatment. Conclusion: Thrombotic occlusion of small and midsize vessels might occur in systemic envenoming by Formosan Russell's viper. It develops early and tends to occur in patients with atherosclerotic vessels. Venom-induced vasocontriction in atherosclerotic or damaged vessels might play a key role in the mechanism of RVV systemic toxicity.

211 CLINICAL SURVEY OF *TRIMERESURUS MUCROSQUAMATUS* SNAKEBITES.

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Introduction: Envenomation by *Trimeresurus mucrosquamatus* (TM) is the most encountered poisonous snakebite in Taiwan. Symptoms following its bite vary widely. Here, we review all cases of TM bites admitted to our hospital, and summarize their clinical significance. Case Series: From 1986 to 1998, there were 37 cases totally, male/female = 24/13, and age ranged from 15 to 72 years old (mean 42). Sites of envenomation included feet (n = 17), hands (n = 15), lower legs (n = 4) and forearms. Extents of local swelling were classified as grade I (<1 joint, n = 1), II (<2 joints, n = 11), III (<3 joints, n = 15), IV (<4 joints, n = 4) and V (≥ 4 joints, n = 6). Most of the swelling (n = 31) subsided in 5 days. Creatine kinase (CK) increased in 17 cases, and one had severe swelling with CK >10,000 U/L. Platelets (PLT) ranged from 124,000/μL to 382,000/μL, and changes of PLT (ΔPLT/PLT) varied from −47% to +16%.

PT, aPTT and fibrin degradation products were normal in patients checked. There was no compartment syndrome, systemic complication or fatality found. There is no correlation between the degree of swelling and lab data. All received ≥ 1 dose of antivenin, and 28 cases received it within 3 hours. Subsequent doses were given in 10 cases. Dosages were 1 dose in 20 cases, 2 doses in 7 cases, and maximally 13 doses in 1 case, and none with an adverse reaction. Routes of administration were IV (n = 35), IM (n = 2), and local (n = 3). Conclusion: Unlike cases previously reported, no severe systemic complication following TM bites was found. TM venom is known to be a strong platelet inhibitor in vitro, but reduced platelet counts were not significant in our series. There are no clinical data to predict the degree of swelling caused by TM bite; therefore, the dosage of antivenin is still equivocal. However, 1–2 doses of the antivenin seemed to be adequate.

212 LIFE THREATENING ANAPHYLAXIS AFTER FIRST TIME CROTALIDAE ENVENOMATION.

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Background: Anaphylaxis with life-threatening complications rarely occurs following rattlesnake envenomation. To our knowledge this is the first report of venom-induced anaphylaxis with airway swelling that required emergent intubation. Furthermore, this is the first case to suggest possible sensitization to rattlesnake venom from dermal and oral exposure to snake proteins following the decapitation and consumption of rattlesnakes. Case Report: A 26-year-old man with a history of prior rattlesnake consumption rapidly developed airway compromise and hypotension, requiring intubation and an epinephrine drip, following a first-time rattlesnake envenomation. He had a complicated acute medical course; including venom-induced thrombocytopenia and defibrination requiring antivenin administration (Wyeth-Ayerst's Crotalidae Polyvalent Antivenin), rhabdomyolysis, renal dialysis for acute renal failure, and continued airway edema with eventual tracheostomy. He subsequently developed, and was treated for, serum sickness during his course at our facility. Six-month follow-up found him hospitalized following repeat abdominal surgery for enterocutaneous fistula, sepsis, and over 35% weight loss. Conclusion: Rattlesnake envenomation may cause anaphylaxis, with airway and hemodynamic compromise, in patients without history of previous invasive rattlesnake exposure. Dermal and oral exposure to snake proteins may predispose to these life-threatening allergic reactions.

213 PROBABLE INTRAVENOUS CROTALIDAE SNAKE ENVENOMATION WITH RAPID ONSET SHOCK AND CROFAB THERAPY: SURVIVAL WITH GOOD OUTCOME AND TIME COURSE OF COAGULATION RECOVERY.

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Introduction: Crotalid polyvalent immune Fab (ovine) (cpif; CroFab) is a new crotalid antivenom with markedly improved safety compared to the previously available horse serum IgG crotalid antivenom. We report use of cpif (CroFab) in a patient with rapid onset shock, probably due to intravenous envenomation. Case Report: A 41-year-old man presented to the Emergency Department, hypotensive and bradycardic, about 45 minutes after envenomation to a leg by a rattlesnake. The envenomation site was in an area of multiple varicose veins. There was ecchymosis, swelling, and profuse bleeding at the envenomation site. 6 vials of cpif were obtained and given as a 1 hour infusion starting 45 minutes after arrival to the hospital. An additional 3 cpif infusions, each of 2 vials, was given every 6 hours. During the first 12 hours post injury, the patient experienced episodes of intermittent severe abdominal pain, each lasting 15–30 seconds. There was no further increased swelling at the envenomation site and muscle compartmental pressures remained low. He was discharged from the hospital five days after envenomation. Results: No adverse effects of cpif were noted clinically. He had a residual 2 cm area of skin sloughing on the leg. Clinical and laboratory time-course data will be presented. Conclusion: We conclude: 1. This patient likely experienced an intravenous envenomation of crotalid venom; 2. The patient recovered with minimal sequelae due to both rapid supportive care and early cpif infusion; and 3. Relatively rapid clinically effective correction of coagulation abnormalities occurred in this particular case with complete laboratory normalization at 7 days.

214 LOCAL MANIFESTATIONS OF *AGKISTRODON CONTORTRIX* (COPPERHEAD) ENVENOMATION SUCCESSFULLY TREATED WITH CROTALIDAE POLYVALENT IMMUNE FAB (OVINE) CROFAB®.

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Background: Although the CroFab® package insert lists its indication for treatment of minimal or moderate North American crotalid envenomation, the clinical trials included no patients that were bitten by *Agkistrodon contortrix.* To date the only evidence of this product's effectiveness against Copperhead venom is in an animal model. We present a case of a moderate human envenomation by the North American Copperhead successfully treated with CroFab. Case Report: A 45-year-old white male was bitten by a copperhead snake on the dorsal surface of his right hand while working in his yard. On presentation to the ED, edema was confined to the hand only. No coagulopathy or neurologic complaints were noted and the patient was admitted for observation. Within three hours the edema progressed to the elbow and was continuing. Four vials of CroFab were administered at five hours post-envenomation. Within one hour of this infusion, the edema progression ceased. The patient subsequently received six additional vials of CroFab per package insert dosing recommendations. No acute allergic reactions or other complications were noted during his two-day hospital stay and no symptoms of serum sickness have been reported on follow-up. Conclusion: We report a case of a moderate human envenomation by the North American Copperhead successfully treated with CroFab. Further study is needed to determine the most efficient dosing schedule for treatment of mild to moderate Copperhead envenomations.

215 COPPERHEAD ENVENOMATION IN THE CAROLINAS.

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Introduction: Although the Copperhead (*Akistrodon contortrix*) is responsible for most Crotalid envenomations in the Carolinas, manifestations and treatment are poorly characterized. Objective: We sought to better describe the clinical course of Copperhead envenomation. Methods: Copperhead exposures reported to a regional poison center from 1997–2000 were identified. Structured review of cases was performed, including hospital records if available. Phone follow-up was attempted. Results: 182 cases were identified. 75% were men. The median age was 31 yr (range 2–93). The bite site was recorded in 168 cases: hand (84), foot (62), leg (14), and arm (8). Bites were classified as: dry (8%), mild (48%), moderate (39%), and severe (5%). The most common symptom was pain (93%). In cases with ≥ mild envenomation, local findings included: fang marks (93%), edema (92%), ecchymosis (48%), erythema (35%), bullae (11%), and tissue necrosis (7%). In 37 patients with coagulation studies, 11 developed abnormal PT and PTT. Two patients had significant bleeding. Treatment was rendered at a healthcare facility in 166 cases, with 80 patients admitted. Opioid analgesics were the most common therapy (81%). Fourteen patients received equine-derived crotalid antivenin (range 2–30 vials), 2 of whom developed urticaria. Two patients required blood products. Surgical treatment included: debridement (6), grafting (2), digit amputation (2), and digit dermotomy (2). No patients died. Phone follow-up was successful in 18 cases. Patients reported limb dysfunction ranging from 5–365 days (mean 103 days). Conclusion: Copperhead bites typically result in mild to moderate envenomation due to local tissue effects. Significant systemic manifestations are rare. Limb dysfunction can be prolonged.

216 FIVE-YEAR EXPERIENCE OF SERUM SICKNESS FOLLOWING THE ADMINISTRATION OF WYETH® ANTIVENIN IN RATTLESNAKE ENVENOMATIONS.

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Background: The incidence and characteristics of serum sickness following the administration of Wyeth® rattlesnake antivenin (WRSA) is rarely described. Methods: A retrospective systemic poison center chart review was conducted involving rattlesnakes' victims referred to our poison center. Serum sickness (SS) was defined as unexplained rash between 3–21 days following the administration of WRSA and resolution defined as the resolution of all associated symptoms (rash, subjective fever, arthralgia and pruritus). Data collected included extremity involved, total vials administered, associated signs, symptoms, duration and medications used to treat SS. A second reviewer independently re-

viewed 10% of randomly selected charts. Results: A total of 414 patients were referred for presumed rattlesnake envenomation between January 1, 1996–December 31, 2000. Of these, 32 refused care and 202 received WRSA. Of the 202 patients receiving WRSA, 181 (90%) completed the required the minimum 21-day follow-up period. SS occurred in 102 (56%) patients. The frequency of SS in patients receiving < 20, < 30, ≥ 30 or ≥ 40 vials was 34%, 36%, 88%, 100% respectively. The risk ratio for developing SS was .54 for < 20 vials, 1.86 for ≥ 20 vials and 2.4 for patients receiving ≥ 30 vials respectively. Duration of SS was 6.1[range 1–21] days. Associated symptoms included subjective fever (49%), arthralgia (20%), and pruritus (40%). Reported medications use included prednisone (98%), antihistamines (92%), and H-2 blockers (2.7%). Kappa for inter reviewer reliability was .69 with confidence intervals of .45–.95. Limitations: Retrospective nature, 10% drop out rate, lack of ability to confirm physical examination findings and medication use. Conclusions: SS following Wyeth rattlesnake antivenin is common.

217 RESPIRATORY COMPROMISE IN PATIENTS WITH RATTLESNAKE ENVENOMATION.

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Introduction: Respiratory compromise (defined as airway obstruction or bronchospasm) following rattlesnake envenomation is an uncommon yet potentially lethal complication. We were interested in determining the frequency of respiratory compromise in patients (pts) treated for rattlesnake envenomation. The medical indications for intubation were also determined. Methods: A retrospective chart review of all pts treated by medical toxicologists at a tertiary referral hospital between 7/1994 and 11/2000. Findings: Out of 294 total pts, 289 charts were reviewed (3 pts were excluded because of enrollment in an experimental protocol, 2 charts were not located). Two hundred fourteen pts (74%) received Crotalidae Polyvalent Antivenin (Wyeth-Ayerst). Of all pts, 21 (7%) had clinical evidence of respiratory compromise: 3/21 pts (1%) developed venom-induced anaphylaxis and 18/21 pts (6%) developed antivenin-induced respiratory compromise (2 with facial swelling, 1 with nasal congestion, 15 with wheeze or bronchospasm which resolved with medications). Thirteen out of 289 pts (4.4%) were intubated following rattlesnake envenomation (1 for venom-induced anaphylaxis, 1 for tongue envenomation, 1 for alcohol withdrawal requiring propofol infusion, and 10 for surgical fasciotomy, dermotomy, or wound care). No one was intubated for antivenin-induced complications. There were no deaths. Conclusion: Only 2 of 289 pts were intubated as a direct consequence of rattlesnake envenomation, no one required intubation for antivenin-induced hypersensitivity reactions. The majority of intubations were done electively for surgical procedures requiring general anesthesia.

218 ANTIVENOM: IT'S NOT JUST FOR RATTLESNAKES ANYMORE.

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Background: Coagulopathy, neurotoxicity and compartment syndrome are life and limb threatening effects of rattlesnake envenomation often warranting polyvalent crotalidae antivenin administration. These effects are rare with copperhead bites. However, tissue destruction (pain, edema and necrosis) is common and may cause significant loss of function and impaired activities of daily living. We believe antivenin use in copperhead envenomation is limited due to a perceived disproportionate risk/benefit ratio for anaphylaxis and serum sickness. Methods: We examined outcome severity (Grade I–IV Scoring Method) and use of conventional antivenin for venomous snakebites reported from Mar 1993 to Dec 1999. Results: Of 596 venomous snakebites reported, 87.2% were copperhead, 8.5% were rattlesnake and 4.3% were cottonmouth. Tissue effects overwhelmingly accounted for the majority of symptoms; systemic effects were uncommon. For rattlesnake envenomations, known outcomes were 26% minor, 42% moderate and 8% major. 50% (25/50) had symptoms which might have benefited from antivenin therapy; 72% (18/25) of these were treated. For copperhead envenomations, known outcomes were 35.7% minor, 46.5% moderate and 2.7% major. 49.2% (255/518) had symptoms that might have benefited from antivenin therapy yet only 22.7% (58/255) were treated. Conclusions: The outcome profile for both groups is similar, yet copperhead envenomation is undertreated compared to rattlesnake envenomation. This disparity in antivenin use represents a therapeutic gap. While not yet FDA approved for copperhead bites, the superior risk/benefit profile for Fab crotaline antivenom makes it a viable treatment option in the management of tissue destruction, even in the absence of systemic toxicity, for all crotaline envenomations.

219 DENDROTOXIN POISONING IN A NEUROBIOCHEMIST.

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Background: Dendrotoxins are highly potent blockers of KV1.1, KV1.2 and KV1.6 potassium channels that are derived from the venom of the green mamba (*Dendroaspis angusticeps*). It is commonly used to inhibit the function of whole nerve preparations *in vitro*. Despite its widespread use and potency, neurotoxicity in humans has not been described from refined toxin. We report a case of dendrotoxin toxicity from dermal exposure. Case Report: A healthy 40-year-old female neurobiochemist presented with complaints of progressive numbness of the left malar region and lateral orbit that progressed to include the medial orbit and tongue. One hour prior to presentation she used her bare hands to remove residual petroleum jelly from a dish that had previously contained 500 nanoliters of 500 nanomolar dendrotoxin. She recalled rubbing her left eye prior to the onset of symptoms. Before touching the dish, she had washed it with running water and then 70% ethanol while using latex gloves. Physical examination was remarkable only for weakness to superior gaze and some mild tongue fasciculations. Within 12 hours of exposure, she was completely asymptomatic. Conclusion: Dendrotoxin is a highly potent neurotoxin that can cause localized impairment of nerve function after mucous membrane exposure. The spreading of the sensory complaints and the tongue fasciculations likely represent spread of the toxin through the nasolacrimal duct.

220 A ONE-YEAR REVIEW OF 241 BLACK WIDOW SPIDER BITE REPORTS.

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Background: Controversy exists regarding the severity and treatment of a black widow spider bite (BWSB). Treatment of BWSB includes antivenin, calcium, analgesics and/or muscle relaxants. In this retrospective study, calls regarding bites from BWSB were reviewed. Method: The poison center database was searched for all cases of human BWSB reported to the poison center in the year 2000. Results: A total of 241 cases were reported. A bite was reported in 79.7% of cases. In the other 20.3% of cases, a BWS was seen but no bite mark was found. A BWS was positively identified in 65.6% of cases. Average patient age was 31.5 years (range 10 months to 79 years) and 58.1% of patients were males. After the bite, 55.2% developed no further symptoms. Only 37 (15.4%) of total cases were observed/treated in a HCF. Of the HCF-managed cases, PCC staff referred only 6 patients (2.5% of total) in for treatment. Of patients seen in a HCF, only 8 (3.3% of total) were admitted. Systemic symptoms developing after the bite included cramping 13.7%, severe pain 8.3%, nausea 8.5%, vomiting 2.9%, dizziness 2.9% and hypertension 0.8%. A few patients (1.2%) reported fever, chills, HA, anxiety, sweating, or SOB. The average length of admission was 37.5 hours. Patients treated in the ED or MD's office were treated/observed for an average of 3.6 hours. Treatment included analgesics 8.7%, muscle relaxants 6.6%, calcium 4.1%, and antihistamines 3.3%. BWS antivenin was used in only one case and that patient was admitted for 28 hours. Patient outcomes: 24.1% minor outcome; 51.9% minimal possible effects; 6.2% moderate effect; 6.6% unrelated effects. Conclusions: Admission to a HCF due to a BWSB occurred in 3.3% of cases. Antivenin administration is rare. More than half of the patients do not develop symptoms beyond the local bite. It is reasonable to monitor BWSB cases with only local symptoms at home with follow-up calls at 1 and 2 hours.

221 DEATH FROM *CENTRUROIDES* SCORPION STING ALLERGY.

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Background: Envenomation by scorpions of genus *Centruroides* can lead to death from ventilatory or respiratory compromise, but mortality in the United States has not been reported since 1964. We describe the first fatality reported in Arizona in 36 years, in an adult with an apparent anaphylactic reaction to the sting. Case Report: A 62-year-old woman in rural Arizona was stung on the toe by a scorpion. Within minutes she experienced dyspnea, then vomited and fell to the floor. Paramedics found her pulseless and apneic. She was intubated with difficulty, because of laryngeal edema; resuscitation involved CPR, defibrillation, and aggressive pharmacologic support. She stabilized hemodynamically, with subsequent physical examination notable for mottled skin, sluggish pupils, tongue edema, diffuse wheezes, toe erythema, and Glasgow coma scale of 3. At no time did she develop peripheral or cranial motor hyperactivity characteristic of scorpion envenomation syndrome. Despite intensive care, she remained neurologically unresponsive for 6 days and was removed from life support after confirmation of global anoxic injury with head CT and nonreactive EEG. Further history revealed that she had been stung by similar scorpions on several prior occasions, without significant reactions.

The scorpion was identified as *Centruroides exilicauda* (aka *C. sculpturatus*). Conclusions: This patient died of consequences of anoxia from anaphylactic reaction to *Centruroides* sting, rather than from neurotoxicity. This phenomenon, although rare in Arizona, is consistent with published reports of hypersensitivity to venom of other scorpions in the Buthidae family.

222 PHARYNGEAL IRRITATION AFTER EATING COOKED TARANTULA.

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Background: Certain species of tarantulas have "urticating hairs" on their abdomen that produce dermal and ocular irritation. When served as a delicacy, a hot fire or blow torch is recommended to destroy these hairs prior to consumption. We report several cases of pharyngeal irritation which probably resulted from the ingestion of poorly prepared tarantulas. Case Series: Several members of a club, whose purpose it is to undertake novel experiences, shared an exotic dinner which included batter-dipped, deep-fried Chilean Rose-haired tarantula (*Grammostola spatulata*). Although a blow torch was used prior to batter-dipping, the ventral surfaces of the spiders were not properly treated. Symptoms consisting of tingling in the mouth and throat occurred in several people who ate the tarantula dish. There was no suggestion of systemic allergic reaction in any patient, and no patient was ill enough to seek medical care. Epidemiological investigation revealed that symptoms did not develop in dinner guests who did not ingest tarantula. Evaluation of a remaining cooked specimen by an entomologist confirmed the presence of urticating hairs on the spider's abdomen. Conclusion: Tarantula hairs, which have previously been reported to cause both dermal and ocular irritation, appeared to cause pharyngeal irritation in a group of persons eating them as part of a prepared dinner. Although other causes of symptoms could not be rigorously excluded, we conclude that cooked tarantula is a potentially irritating food, and those who consume it should be aware of this potential toxicity.

223 THE VIETNAMESE CENTIPEDE–WHERE EASTERN MEDICINE MEETS WESTERN MEDICINE: A CASE REPORT.

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Background: There are approximately 3,000 species of centipedes. The Vietnamese Centipede (Scolopendra subspinipes) is normally found in the high humidity of Southeast Asia and is among the largest of the centipedes. Case Report: A healthy 16-year-old male pet store worker brought a water dish to a Vietnamese centipede which subsequently bit him on his index finger. The patient's finger reddened, swelled to twice normal size, and became very painful. The patient was taken to an emergency department, when our poison control center was contacted. Initial management included tetanus prophylaxis, an antihistamine, and analgesia. The pet distributor and reptile museum were unavailable. We contacted the Embassy of Vietnam to consult with their reptile expert. The following faxed to us from Bach Mai Hospital in Hanoi: "Transfer IV fluids, inject pain relief directly into the wound area (lidocaine) and use allergy medication. The other way is Vietnamese Traditional treatment: To take slaver (spittle) of the cock after it did its usual morning crow and put directly to the wounded area. (If can not wait until the morning, you may try the simple slaver of the cock put in the wound)." Based on availability, lidocaine and diphenhydramine were chosen over cock spittle. The patient responded well, and was discharged without further sequella. Conclusion: The global neighborhood has contributed to, among other things, the commercialization of international pets. A consultation network is necessary to maintain readiness to treat the unexpected. Openness to alternative therapies, as is asking for "western equivalents" whenever possible, is recommended.

224 CENTRUROIDES STING COMPLICATED BY ANAPHYLAXIS TO ANTIVENOM IN AN ASTHMATIC ADULT.

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Background: Bark scorpion envenomation is very painful and can be associated with airway complications in young children, however adult patients rarely suffer life-threatening complications. Caution should be used in administering scorpion antivenom to adults with a history of risk factors such as asthma, without life threatening airway compromise. Case Report: A 33-year-old woman with a history of asthma treated with albuterol inhalers was stung by a single scorpion twice. She presented to the emergency department 1.5 hours later, agitated with generalized motor twitching and mild extraocular muscle involvement. Initial treatment was 4 mg of IV lorazepam and 4 mg IV morphine. Her

condition worsened, with increasing twitching, but without airway compromise. The decision was made by the ED physician to treat with scorpion antivenom after a negative skin test. After 5 minutes of infusion (≈1/5 vial) she developed a rash, tongue swelling, wheezing, hypotension and coma requiring intubation, SVNs, methylprednisolone, diphenhydramine, cimetidine and epinephrine infusion. Epinephrine was discontinued 11 hours later and she was extubated the following day with concomitant visualization of the cords demonstrating no further swelling. Patient was discharged two days later but developed serum sickness 12 days after discharge. Conclusion: Scorpion envenomation can cause severe symptoms. Treatment with antivenom should be considered after assessment of risk/benefit. We recommend scorpion antivenom not be given to patients with a history of asthma if the envenomation itself is not life threatening.

225 SEVERE TOAD VENOM POISONING BY DERMAL ABSORPTION.

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Background: In Viet Nam it is common practice to eat toad livers. Although these toads secrete digitalis-like glycosides, poisoning does not occur when eating the livers. We report a case of severe cardiac glycoside-like poisoning in a patient with extensive dermal exposure to toads. Case Report: A 16-year-old male presented with emesis and fatigue approximately 2 hours after removing and eating 15 toad livers (roasted). He had eaten these livers in the past, but no more than 5 at a time. On presentation his vital signs were: HR 120, BP 130/90. He was treated by potassium permanganate gastric lavage, fluids, diazepam, and furosemide, and was then transferred to our poison control centre. He arrived 1-1/2 hours later with stable vital signs. Within an hour and a half of his arrival his heart rate dropped to 60 with a third degree AV block and a narrow QRS (100 msec). His blood gases showed pH 7.44, PCO_2 45.7, PO_2 119. Electrolytes were: sodium 129, potassium 4.7, and bicarbonate 30.4. Hemoglobin was 42 g/L and hematocrit was 40%. He was treated with supplemental oxygen and activated charcoal. Over the course of the next 8-hours his heart rate dropped to below 50 and his systolic blood pressure dropped to < 80. He was treated with transcutaneous pacing to a heart rate of approximately 60/minute using 30 milliamps of current. He progressively improved and by the next morning showed only first degree AV block with a resolution of his bradydysrhythmia. Discussion: This patient appears to have developed a digoxin-like syndrome after handling toads for a prolonged period of time while he removed the livers. Although anti-digoxin Fab fragments may have been useful, they were not available to us. He was treated with supportive care and transcutaneous pacing until resolution of his toxic syndrome. This toad venom poisoning appeared to occur strictly from dermal exposure.

226 DIFFICULTY IN DIAGNOSING A CASE OF HUMAN RABIES.

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Background: Since 1990, there have been 32 reported cases of human rabies reported in the US. Exposure to bats was implicated in 74% of the cases, even though only 2 cases had an established history of a bat bite. Case Report: A 49-year-old male presented to his PMD with right arm pain, weakness and increasing paresthesias. He returned the next day with increasing symptoms and was diagnosed with cervical radiculopathy. The patient presented to the ED that night with pain unrelieved by opiates and anxiety that was relieved with lorazepam. The next AM he presented again to the ED with increased pain, agitation, right-sided diaphoresis, inability to swallow, tremulousness, jerking movements in the trunk, confusion and hyperventilation. He was admitted and diagnosed with either encephalitis, toxin induced alterations or a psychiatric condition. He was transferred to a tertiary care HCF with a diagnosis of possible viral encephalitis, including the need to rule-out rabies. The patient quickly became unresponsive and was placed on a ventilator. A head CT and LP were negative. He was treated with vecuronium, morphine and midazolam drips, K+ replacement, rabies immunoglobulin, and IV acyclovir. Corneal impressions, saliva and CSF sent to the State Health Dept Lab confirmed a diagnosis of rabies by immunofluorescence. Although the family denied any bites, the patient had removed a bat from the house a month prior. His hospital course was complicated by laryngeal spasm, rhabdomyolysis, renal failure, elevated LFTs, hypokalemia, hyponatremia, respiratory alkalosis, anion gap acidosis, and an MI. Autopsy revealed the cause of death as rabies polioencephalitis contracted from a Mexican free-tailed bat, determined by DNA probe studies. Conclusion: Rabies is rare but should be considered in cases of rapidly progressing encephalitis. History of wild animal exposure should be excluded and rabies should be ruled out as a potential diagnosis.

227 REPEATED DOSES OF INTRAVENOUS MANNITOL FOR CIGUATERA POISONING.

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Introduction: Mannitol is a recognised treatment for ciguatera poisoning. In previously reported cases only a single dose has been administered. The use of repeated doses of mannitol have been recommended to treat recurrent symptoms. We report a case of ciguatera poisoning in which a second dose of mannitol was effective in controlling recurrent symptoms. Case Report: A 50-year-old Fijian man presented 2 days after ingesting "bati" or red bass (*Lutjanus bohar*) imported from Fiji. 24 hours following ingestion he developed myalgias, lethargy, paraesthesia of tongue, hands and feet, and a burning sensation when he washed his hands. On examination he was afebrile and had no focal neurological signs. Ciguatera poisoning was diagnosed and treated with a 200mL intravenous infusion of 20% mannitol. This relieved all of his symptoms within 2 hours and he was discharged. He returned the following day with similar symptoms plus arthralgia in the elbows and knees. His symptoms had returned 6 hours after completing mannitol. His pain responded to oral ibuprofen and he was discharged on regular ibuprofen. Three days later he represented with headache, lethargy, pruritus in his lower back and paresthesiae of tongue, hands and feet. An additional 250mL of 20% mannitol was given intravenously. Within 1 hour, his symptoms had completely resolved and the patient was discharged. On review the following day, some minor symptoms had returned but were controlled with ibuprofen. By 8 weeks the patient had fully recovered, was back to full-time employment and was tolerating seafood and alcohol. Conclusion: Mannitol is an effective agent for ciguatera poisoning but symptoms may return after initial treatment. A second dose of mannitol 5 days after initial exposure appeared to be at least partially effective in controlling recurrent symptoms in a patient with ciguatera poisoning.

228 EOSINOPHILIC PNEUMONIA ASSOCIATED WITH FOOT INJURY FROM BLACK SEA URCHIN.

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Background: Hypersensitivity reactions after sea urchin "spine injuries" have been described. Most often these are local granulomatous nodules or rashes. We describe a case of eosinophilic pneumonitis (EP) without a clear cause in a patient who stepped upon a black sea urchin a day prior to onset of symptoms. Case Report: A 21-year-old male arrived at the emergency department (ED) with fever, headache and 3 days of dyspnea and cough. He had no significant medical history, was on no medications, denied alcohol use, and smoked tobacco occasionally. He denied recent unpurified freshwater exposure. He noted stepping upon a black sea urchin while in the ocean 3 days prior to presentation. In the ED, he had a T: 100.7, BP: 122/59, P: 98, R: 36, Pulse OX 84%. His exam was notable for diffuse rhonchi, respiratory distress, and areas of black tattooing on the plantar surface of his foot consistent with his recent sea urchin injury. No cellulitis was noted. The patient underwent CT of his head and an LP, which were unremarkable. Blood cultures, urine cultures and cultures for TB, leptospirosis, mycoplasma, Hantavirus and fungal pathogens were negative. HIV test was negative. Bronchoscopy confirmed an eosinophilic process. The patient developed increased respiratory distress and was intubated. He received broad-spectrum antibiotics and methylprednisolone. His condition improved over 10 days and he was discharged on hospital day 13. Conclusion: We describe a case of severe EP possibly related to black sea urchin exposure. In marine environments, black sea urchin exposure should be considered as a possible cause of EP.

229 STINGRAY ENVENOMATIONS AND ANTIBIOTIC USE: A PILOT STUDY.

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Objective: There are approximately 1800 stingray envenomations annually in the US. Although many textbooks recommend prophylactic antibiotics (ABX), few studies have evaluated this. The goal of this pilot study is to determine the characteristics of patients with stingray envenomations and the use of ABX. Our null hypothesis is that patients prescribed prophylactic ABX will have better outcomes as compared to those that did not. Methods: We performed a retrospective chart review on all reported southern stingray (Dasyatis Americana) envenomations that presented to a Gulf coast community based ED during 1997–1999. Data collection included patient demographics, local and systemic effects, ABX use, and outcomes. We compared patients who received prophylactic ABX with those that didn't. The treating physician decided on the use of ABX. Outcome measures were: need for subsequent hospitalization or need

for further medications. At this institution, it is departmental policy to obtain telephone or direct follow up for all discharged patients. Results: 66 patients identified. All were treated as outpatients. 53% (35/66) were males. 89.4% (59/66) were stung in the lower extremity. 36.3% (24/66) were lost to follow up. Of the 42 patients who had follow-up: 4 patients were prescribed ABX for cellulitis diagnosed on initial presentation. 66.7% (28/42) were prescribed prophylactic ABX. One patient prescribed prophylactic ABX was later treated for *Clostridium difficile.* No other patient required further medications or hospitalization. There were no statistical differences between the measured outcome of the two groups (Fisher's exact test, $p = 0.736$). Conclusion: Patients with envenomations from southern stingrays may be treated as outpatients. We did not detect a significant difference in outcomes between patients who received prophylactic ABX versus those that did not.

230 ORGANIC ANION TRANSPORTING POLYPEPTIDES (OATPS) MEDIATE UPTAKE OF MICROCYSTIN INTO BRAIN AND LIVER.

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Background: Microcystins (MC) are toxins produced by freshwater cyanobacteria. They are cyclic heptapeptides that exhibit neuro- and hepatotoxicity. In rat hepatocytes, basolateral uptake of MC was inhibited by substrates of the organic anion transporting polypeptide (rodent: Oatps; human: OATPs) family of membrane transporters. However, the transport systems that mediate uptake of MC across the blood-brain barrier and into hepatocytes have not yet been identified. Methods: Using the *Xenopus laevis* oocyte expression system we tested whether members of the Oatp/OATPs would be involved in transport of the most common MC variant, MC-LR. Results: OATP-A expressing *X. laevis* oocytes accumulated MC-LR, which led to a severe damage of the oocytes. Accumulation and damage could be inhibited by co-incubation with taurocholate or bromosulfophthalein (BSP). Similarly, expression of the liver specific rat Oatp4 as well as the human OATP-C and OATP8 resulted in MC-LR uptake into oocytes that could be inhibited by taurocholate and BSP. However, no MC-LR transport was observed with oocytes expressing the rat Oatp1, rat Oatp2 and the human OATP-B. Conclusions: Given the tissue specific expression of the involved transport systems, these results can explain the observed organ specific toxicity of MC-LR. OATP-A mediates MC-LR transport across the blood-brain barrier, while the liver specific Oatp4, OATP-C and OATP8 are responsible for transport into hepatocytes.

231 SYSTEMATIC REVIEWS IN CLINICAL TOXICOLOGY.

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Background: The quality and extent of clinical trial evidence on the effectiveness and safety of treatments of poisoning is often not described in conventional review articles and texts. Systematic reviews focus solely on these aspects of the literature. Methods: We performed systematic reviews of trials of treatment(s) of paracetamol, paraquat and organophosphate poisoning. Methods used were those formulated by the Cochrane collaboration and/or the journal Clinical Evidence. Results: Similar problems were encountered in all reviews. There were few trials, which were mostly small, from a single centre with concerns about the effectiveness of randomization and blinding processes. None used the CONSORT guidelines for reporting clinical trials (some pre-dated these guidelines). No trials had addressed dose-response and/or compared different protocols. Important prognostic factors such as dose, agent, time to treatment, and severity of poisoning were not stratified in any trial and were commonly unequally distributed. Strong conclusions by the authors were often based on over interpretation of post-hoc analyses. Conclusions: For acetylcysteine, further studies are required to attempt to improve the current cumbersome and long treatment protocols. For other treatments (such as activated charcoal, oximes, and cyclophosphamide/prednisone) much larger trials are required before firm conclusions about the effectiveness (or lack thereof) of the treatments should be made. These should also be designed to explore aspects of the dose and time response, stratify using predefined prognostic variables and be reported using CONSORT guidelines.

232 IMPROVING QUALITY OF PEER REVIEW: RANKING OF GUIDANCE CRITERIA.

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Background: A 21-item non-hierarchical Reviewers' Guideline has been used by the editorial office of *Journal of Toxicology–Clinical Toxicology* to aid peer reviewers in judging the suitability of manuscripts for publication. The

concordance of these items with the personal criteria for quality held by the reviewers and the leadership of the supporting toxicology organizations has not been examined. Methods: E-mail questionnaire containing the 21 items of the guidance criteria was sent to the 41 members of the Journal editorial board and to the 26 officers and board members of AACT, EAPCCT, AAPCC, CAPCC, and ACMT. Participants were asked to rank the 5 most important items in peer review. Optimal participation was achieved by multiple requests followed, if necessary, by a telephone interview. Results: The respondents expressed considerable dissatisfaction with the non-hierarchical categories of the checklist. A clear emphasis emerged on the primary ranking of 3 criteria: Relevance of the Objectives; Study Design; Analysis and Accuracy. Of lower ranking were Clarity in Abstract and Introduction; Adequate Description of Methodology; and Focus on Discussion and Literature Reviews. Several reviewers voiced particular attention to the exclusion of duplicate publication, repetition within the document, inadequate or inaccurate background material. There was a wide divergence of opinion on manuscripts with major difficulties in organization and language but the majority of reviewers saw these as remediable. Conclusions: Relevant objectives, good study design, and scientific accuracy were the primary criteria preferred for peer review of manuscripts by editors and leaders in clinical toxicology. Attention to these ranked criteria should improve the overall quality of peer review.

233 CURRENT IPECAC RECOMMENDATIONS, WHAT DOES IT MEAN FOR FUTURE NATIONAL MATERIALS?

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Background: Development of national guidelines for poison management and public education materials is currently being discussed in the US. Consensus on these issues will take a cooperative effort. Syrup of ipecac is one topic for which a consensus decision will need to be made. Objective: The hypothesis of this study is that poison center (PC) medical directors are now recommending both home ipecac availability and ipecac administration less often. Methods: An 8-question survey pertaining to syrup of ipecac was distributed by e-mail to all medical directors of AAPCC certified regional poison centers. A separate e-mail was sent to all AAPCC member PC's requesting their educational materials. These materials were surveyed for ipecac recommendations. Results: Of 25 medical directors who responded (32% response rate), 21 (84%) currently recommend that parents keep ipecac at home. Twenty-two (88%) of medical directors reported recommending ipecac administration less frequently compared to 5 years ago. When asked if national educational materials should include information concerning ipecac, 21 (84%) of the medical directors responded "yes." Of the 34 centers that submitted educational materials (43% response rate), 100% recommend keeping ipecac in the home. Conclusion: Although the majority of medical directors are recommending ipecac administration less frequently than 5 years ago, the majority stated that future national materials should contain information regarding syrup of ipecac and that syrup of ipecac should continue be available in the home.

234 A SURVEY OF KNOWLEDGE AND ATTITUDES CONCERNING ACCIDENTAL POISONING IN MINNESOTA.

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Background: The goal of this survey was to document and benchmark the level of knowledge and attitudes concerning accidental poisoning in Minnesota. The survey was funded by the Minnesota Department of Health. Methods: A total of 301 heads of households, 18 years and older, were interviewed by telephone. Interviewing was based on a computer assisted random digit dialed sample of households. A standard questionnaire was developed to assess knowledge and attitudes. Results: Only 21% of those surveyed rank accidental poisoning as a significant concern when considering major issues across the State. Household cleaning products were considered the leading cause of accidental poisoning (29%). 8 in 10 respondents were at least somewhat confident that they would know what to do if someone were accidentally poisoned. 94% store medicines and household products in their original containers. 75% said they never lock up medicines. When faced with a poison emergency, 47% said they would call 911, and 28% a poison center. Conclusion: Accidental poisoning was considered to be a much less significant concern than other major issues. Despite the fact that substantial prevention and education efforts have been ongoing in Minnesota for over 20 years, respondents were less likely to provide appropriate responses regarding prevention strategies and emergency action than would be

expected, though positive behaviors were noted. Prevention, education, and awareness efforts are in need of further evaluation.

235 COMMUNITY HOSPITAL'S RESPONSE TO ANTHRAX THREATS: A CASE SERIES

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Background: Because of its seriousness, many individuals have used *B. anthracis* contamination as a threat. In Florida, last year, there were over 35 anthrax threats reported to the F.B.I. All were proven to be hoaxes. We report 2 such cases involving a community hospital. Case 1: A letter, in a meat processing plant, was opened that dispersed brown powder in the room. The letter said they've been exposed to anthrax and to call "911." EMS/HAZMAT/Police/F.B.I./ and Dept. of Health arrived on the scene. The hospital was alerted and a "Code Orange" (Mass Casualty/Natural Disaster) was called for the hospital, which required the whole hospital staff to stay on notice. 14 victims, were decontaminated, placed in tyvek suits and transported to the E.D. After being medically evaluated, all were discharged. Only those who came in to direct contact with the letter/powder received prophylaxis. After 72 hr, testing revealed the substance to be *B. pharagirus*, an organic pesticide. Case 2: Two months later, a letter was opened at a collection agency, which contained a white powder and the words "Anthrax- Die." 4 pts were again decontaminated by HAZMAT, and medically cleared after an E.D. evaluation. Anthrax testing was negative 24 hr later. Discussion: After the 1st hoax, multiple meetings took place to improve performance against biological threats. These were implemented in Case 2. The Command Center agreed to notify the hospital on any environmental precautions needed, based on the substance. Arrangements were made to secure personnel "contaminated" belongings. The hospital created a "Code Grey", specifically for biologic threats. It limited the number of staff involvement and educated them regarding their personnel risk. Antibiotic prophylaxis would be based on F.B.I.'s threat assessment. Conclusion: By creating a separate plan for Biologic (vs. Chemical/Nuclear/Mass Casualty) Disasters, hospital resources can be better utilized.

236 DETERMINING TARGET POPULATION CHARACTERISTICS THROUGH A FOCUS GROUP BASED PROCESS.

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Objective: To determine baseline information about a regional poison center's target population using a focus group process. Methods: A series of ten, 90 minute, focus groups were held throughout our service area. Participants were both English and Spanish speaking parents of children <4 years, and English speaking adults > 30 years. A self reported demographic data form was collected on each participant prior to the start of each group. Results: There were 85 participants, 51 were parents of children <4 with 23 of these Spanish speaking and 34 participants were adults >30. Doctors, the Internet, friends and health fairs were the source of child health information for 95% of the participants. Even though 70% had heard of something called poison control in a poisoning emergency 80% said they would call 911 or go to the emergency room. The local poison center name was not recognized by 85% of the participants. Their emergency phone numbers were kept on the refrigerator or near the phone 90% of the time. Participants, 85%, indicated that any poison prevention message should be attention getting and appeal to the intellect with some scare tactics. Strong magnets are the most often kept and most readily available method of keeping the poison control number on hand was agreed upon by 99%. Conclusion: Based on the 10 focus groups a regional poison center is generally unknown by name and is not a preferred source of care in a poisoning emergency. Therefore significant efforts should be made to market the poison center and its services.

237 HOW WELL DO DOCTORS AND NURSES ESTIMATE THE WEIGHT OF PATIENTS?

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Background: The weight of patients is important in clinical toxicology because the toxic dose of many compounds is expressed in mg/kg body weight and also the dose of some drugs used to treat poisoned patients (e.g. N-Acetylcysteine) is weight dependent. The authors have observed that, commonly, patients are not weighed and that doctors and nurses often estimate the weight of patients. The aim of this study was to investigate how reliable such estimates of body weight are. Methods: 6 patients on a poisons ward were chosen with body weight ranging from 53-122kg. 6 experienced nurses and 12 doctors (2 consultants, 4 middle grade and 6 junior doctors) were asked to estimate the weights of these patients. Results: The following table summarises the estimates of the patient's weight.

Actual weight (kg)	122	56	76	53	74	53
Range of all estimations	72–118	48–85	67–89	50–90	58–90	52–75
Mean of all estimations (mean ± SD)	100 ± 10.1	65 ± 9.2	79 ± 5.9	68 ± 7.9	77 ± 7.9	61 ± 8.5
Mean of doctors' estimations (mean ± SD)	102 ± 8.4	65 ± 8.5	80 ± 4.6	67 ± 11.8	78 ± 7.5	61 ± 7.8
Mean of nurses' estimations (mean ± SD)	96 ± 12.9	66 ± 11.3	78 ± 8.3	69 ± 12.2	74 ± 8.9	60 ± 10.3

Conclusions: The doctors and nurses in this study were not good at estimating the weight of patients, sometimes overestimating and sometimes under-estimating true weight. If the weight of the patient is going to be used in a calculation of the toxic dose of a substance taken in overdose or of the dose of a drug to be used in treatment the patient must be formally weighed.