Clinical Toxicology, 36(5), 427-532 (1998)

Platform Session 1 Saturday, September 12 8:30 am - 10:00 pm
AACT/AAPCC Research Awards and General Presentations
Abstracts #1-#6

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1 PHARMACOKINETICS OF ORAL VS INTRAVENOUS BOLUS VS INTRAVENOUS INFUSION OF PYRIDOXINE IN HEALTHY HUMAN SUBJECTS.

Caliva T, Anguish D, Kalapodis C, Powers R, Stork CM. *Central New York Regional Poison Control, Department of Emergency Medicine, SUNY HSC Syracuse, NY*

Background: Isoniazid overdose causes seizures through depletion of pyridoxine, a required co-factor for the formation of gamma amino butyric acid (GABA), a major inhibitory neurotransmitter. Treatment includes benzodiazepines and high doses of pyridoxine, at a dose of gram per gram equal to the amount of isoniazid ingested up to 5 grams, typically given by intravenous bolus. It is unknown whether an alterative dosing method of pyridoxine may provide more active substrate for the co-factor mediated generation of GABA. Methods: This was a prospective, randomized, cross-over trial in four healthy human subjects comparing the pharmacokinetics of pyridoxine after intravenous bolus, intravenous infusion over 2 hours and orally. One gram of pyridoxine was administered to each subject by each of route with a one week washout period. Serum pyridoxine concentration was measured at time 0, 10, 20, 30, 60, 120, 240, 480 and 720 minutes. Peak concentration, area under the concentration vs time curve (AUC), and elimination half-life of pyridoxine were statistically compared using anova. Significance was accepted at a p value $\leq$ 0.05. Results: The peak concentration of pyridoxine was 9.97 $\pm$ 7.30 ng/uL, 44.6 $\pm$ 21.07 ng/uL and 15.75 $\pm$ 12.47 ng/uL after intravenous infusion, intravenous bolus and oral administration, respectively. (p = 0.077) AUC was 2058 $\pm$ 1178 ng min/uL, 1403 $\pm$ 619 ng min/uL, and 1942 $\pm$ 1653 ng min/uL after intravenous infusion, intravenous bolus and oral administration, respectively. (p = 0.061) Finally, elimination half-life was 106.85 + 32.31 min, 18.25 + 0.777 min and 42.53 + 19.76 min after intravenous infusion, intravenous bolus and oral administration, respectively. (p = 0.796) Conclusion: No significant differences were found with respect to peak concentration, AUC, and elimination half-life of pyridoxine, although there was a trend towards increased AUC (p = 0.077) after pyridoxine infusion. More subjects are required to effectively determine that there is no difference between the groups.

2 PARTIAL LIQUID VENTILATION (PLV) ATTENUATES LUNG INJURY IN A HYDROCARBON ASPIRATION INJURY (HAI).

Kahai J, Rotta A, Quadrani D, Alashari M, Joshi P, Steinhorn D. *Pediatric Critical Care, Western New York Poison Control Center, Children's Hospital, Buffalo, NY*

Introduction: HAI causes significant morbidity, leading to pneumonitis and ARDS. PLV has been investigated as an adjunctive therapy in the treatment of ARDS. We postulated that PLV, if instituted early in an experimentally-induced HAI model would recruit alveoli, improve gas exchange and decrease lung injury. Methods: 12 New Zealand white rabbits were sedated, instrumented, and injected intratracheally with 0.15ml/kg kerosene. Rabbits breathed 100% oxygen via a nose cone for 2 hours or until the PaO2 fell below 100 torr. They were then randomized to either PLV with Perflubron (18 ml/kg)(n=6) or conventional ventilation (CMV/controls) (n=6), intubated and supported for three hours. Blood gases were determined every 30 minutes. The myeloperoxidase activity (MPO) was determined spectophotometrically and protein content via the BCA method. A pathologist blinded to treatment groups scored the lung injury score (LIS), using an established 4 point scale. Data were analyzed globally and regionally using the *t*-test and are presented as means$\pm$SEM. Results: Mean global LIS was 43 for controls versus 11 for the PLV group. The controls showed significant regional injury with a dependent LIS of 32 *vs.*7(p=.039) in the PLV group. The dependent MPO for the control group was 53 *vs.*36 (p=.2) for PLV. The PaO2 for the control *vs.* PLV animals at 210, 240, 270 and 300 minutes were 144$\pm$61 *vs.*300$\pm$80, 220$\pm$104 *vs.*264$\pm$88, 188$\pm$87 *vs.*413$\pm$7.5, and 203$\pm$158 *vs.*396$\pm$2 respectively. Conclusions: The LIS observed in the PLV and control rabbits suggest that PLV reduces the histologic and biochemical evidence of injury particularly in dependent regions where consolidation

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is prominent in ARDS. The higher PaO2 seen in the PLV-treated group during the final hour would suggest that PLV in HAI improves oxygenation. We speculate that the improvement in lung injury seen during treatment with PLV will lead to a reduction in long-term morbidity following HAI.

3 CARBARYL POISONING: 2-PAM OR NOT 2-PAM.

Mercurio-Zappala M, Hack J, Salvador A, Hoffman RS. *New York City Poison Center, New York, NY*

Background: Treatment for severe organophosphate and carbamate poisoning includes atropine for muscarinic effects, and 2-PAM to regenerate cholinesterases. Most literature, however, cautions against use of 2-PAM in carbaryl (1-naphthyl *N*-methyl carbamate) poisoning based on limited animal models which suggest that 2-PAM exacerbates carbaryl toxicity. The purpose of this study was to define the effects of 2-PAM on carbaryl toxicity in a controlled, clinically relevant, animal model. Methods: Female Swiss albino mice were acclimated to 12-hour alternating light and dark cycles and allowed food and water ad libitum. Following preliminary studies, 110 mice were randomized to one of 4 placebo controlled treatments: 1) carbaryl 5 g/kg SC, 2) carbaryl 5g/kg SC with atropine 4mg/kg IP, 3) carbaryl 5g/kg SC with 2-PAM IP, 4) carbaryl 5g/kg SC with atropine 4mg/kg IP and 2-PAM IP. Protocols 3 and 4 were subdivided 3 times using 2-PAM doses of 100, 50, and 25mg/kg (3A,3B,3C,4A,4B,4C). Atropine, or placebo were given 10 minutes after carbaryl injection which coincided with the onset of visible symptoms. Lethality was assessed at 24 hours. Results: Percent lethalities were as follows (p values are compared to carbaryl alone using Fisher's Exact test): 1) carbaryl, (60%), 2) carbaryl with atropine, (15%, $p<0.05$), 3A) carbaryl with 2-PAM (100mg/kg), (80%, $p>0.05$), 4A) carbaryl, atropine, 2-PAM (100mg/kg), (67%, $p>0.05$), 3B) carbaryl with 2-PAM (50mg/kg), (60%, $p>0.05$), 4B) carbaryl, atropine, 2-PAM (50mg/kg), (10%, $p<0.05$), 3C) carbaryl, 2-PAM (25mg/kg), (25%, $p<0.00$), 4C) carbaryl, atropine, 2-PAM (25mg/kg), (10%, $p<0.05$). Conclusion: In this animal model, atropine protected against carbaryl poisoning. High dose 2-PAM, with or without atropine offered no benefit over placebo. While intermediate dose 2-PAM was no different than placebo, the addition of atropine at this dose enhanced survival. Low dose 2-PAM, both alone and with atropine statistically improved survival. Although not statistically different, animals that received intermediate or low-dose 2-PAM with atropine tended towards better outcomes when compared to animals treated with atropine alone. This study casts doubt on the relevance of previous models of carbaryl poisoning.

4 PYRIDOXINE HYDROCHLORIDE INFUSION EFFECT ON ACID-BASE STATUS.

LoVecchio F, Curry SC, Wallace KL, Graeme KA, Suchard JR. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: Pyridoxine hydrochloride (B6), the antidote for INH-induced seizures, is available in solution at a concentration of 100 mg/ml at pH < 3. B6 is often infused rapidly in large doses for INH-induced seizures. We hypothesized that this infusion would result in transient worsening of acidosis. Thus, clinicians obtaining blood gases soon after pyridoxine infusion, might erroneously attribute a decrease in base deficit to an additional intoxication, or question the diagnosis of INH poisoning. Patients may then be subject to further unnecessary or potentially harmful diagnostic tests or interventions. Effects of B6 infusion on base deficit, in amounts given for INH poisoning, have not been studied in human beings. Methods: We conducted a randomized, controlled, crossover trial in human volunteers. Five healthy volunteers [mean age:35, range 29-43] were randomized to receive IV placebo (50 ml normal saline) or 5 grams of B6 (50 ml) over 5 minutes. A peripheral intravenous catheter was established in each arm and heparinized venous blood was obtained for base deficit at baseline, and 3, 6, 10, 20, and 30 minutes after infusion. Following at least a 4 hour washout period the volunteers were assigned to the alternate arm of the experiment, thus acting as their own control. Data were analyzed using the 2-tailed Student t-test with Yate's correction for multiple comparisons. Results: No difference was noted between groups at baseline ($p>.6$). A statistically significant increased base deficit was noted following the B6 infusion versus control at 3 ($p<.023$), 10 ($p<.01$), and 20 ($p<.0002$) minutes. Statistical significance was approached at 6 minutes ($p<.056$), but not at 30 minutes ($p=.1$). Maximal mean increase in base deficit (2.74 mEq/L) was seen at 3 minutes. Conclusions: A transient increase in base deficit occurs following the infusion of 5 grams B6. Physicians should be aware that larger B6 infusions, commonly given in INH overdose, may produce larger increases in base deficit.

5 ENHANCED MORTALITY FROM PARTIAL LIQUID VENTILATION IN A RAT MODEL OF KEROSENE ASPIRATION.

Burns M, Dickson E, Hocker M, Sivilotti M. *Beth Israel Deaconess Medical Center, Boston, MA; University of*

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Massachusetts Medical Center, Worcester, MA

Background: Low-viscosity hydrocarbon aspiration is associated with the development of chemical pneumonitis and acute respiratory failure. Animal and human studies suggest that perfluorocarbon liquid ventilation (PLV) improves gas exchange and pulmonary function in acute respiratory failure. We sought to determine the efficacy of PLV to enhance survival in a rat model of acute hydrocarbon aspiration. Methods: Twelve Wistar rats (450-500g) were anesthetized with halothane, intubated via direct laryngoscopy, and administered 0.3 ml/kg intratracheal kerosene. Positive-pressure ventilation using a Harvard rodent pump was instituted until resumption of spontaneous breathing, followed by extubation and recovery in an oxygen chamber. Five min after kerosene instillation, animals were randomized to PLV or sham treatment (standard gas ventilation). PLV group animals (n=6) received 20 ml/kg of pre-oxygenated FC-77 (3-M) by direct intratracheal instillation in a fashion analogous to kerosene delivery. Sham treatment animals (n=6) were also re-anesthetized, reintubated and received positive-pressure ventilation ($FiO_2=1.0$) in an identical fashion. All animals recovered in an oxygen chamber for two hours and were then returned to animal quarters with free access to food and water. Five-day mortality was compared using the Fisher Exact test. Results: Six rats died, all from acute respiratory failure. Mortality was significantly greater in the PLV group (83%) as compared to the gas ventilated group (17%; p=.039). Conclusion: Early PLV appears to increase mortality in this model of hydrocarbon aspiration.

6 THEOPHYLLINE INDUCED SEIZURES IN RATS; PROPHYLAXIS WITH LORAZEPAM AND PHENOBARBITAL.

Daniel B, Wurpel J, Hoffman RS, Howland MA. *St. John's University College of Pharmacy, New York City Poison Center, New York, NY*

Objective: To determine the role of lorazepam (0.5 and 1mg/kg) and phenobarbital (30 and 60mg/kg) for prophylaxis against theophylline induced seizures in rats. Methods: This was a prospective, randomized, blinded, and controlled animal study. Male Sprague Dawley rats (290-548g) were anesthetized with ketamine and xylazine, an indwelling jugular venous catheter was inserted, and they rested for 24 to 48 hours before the onset of the experiment. Twenty-nine rats were infused with aminophylline (300mg/kg theophylline total dose given as a 20mg/kg/min load over 5 minutes followed by a maintenance infusion of 10mg/kg/min over 20 minutes). At 10 minutes into the protocol the rats were randomized to receive: placebo, lorazepam 0.5mg/kg, lorazepam 1mg/kg, phenobarbital 30mg/kg, or phenobarbital 60mg/kg. The time to seizure (defined as forelimb extension, tonic hind limb extension, and loss of righting reflex), death (absence of respiration and pulse), or decapitation (determined by the appearance of imminent demise) was recorded. Theophylline serum concentrations were measured at onset of seizure, death or decapitation. The combined time to seizure, death, or decapitation was analyzed by Kruskal Wallis (ANOVA). ANOVA was used to compare the mean serum concentrations measured at the time of seizure, death, or decapitation. The alpha for significance was set at 0.05. Results: There was a statistically significant difference in the combined time to onset of seizure, death, or decapitation among the groups (p=0.011). The combined mean ± SEM time in minutes to onset of seizure, death, or decapitation was: control, 28.7 ± 14; lorazepam 0.5mg/kg, 77 ± 23; lorazepam 1mg/kg, 52 ± 9; phenobarbital 30mg/kg, 109 ± 18; and phenobarbital 60mg/kg, 111 ± 13. Theophylline serum concentrations were statistically higher than control for all treatment groups ($p<0.05$). Conclusion: In this rodent model, prophylaxis with either lorazepam or phenobarbital appear to be beneficial in delaying the time to significant toxicity. Further study is needed to elucidate differences among these agents.

Poster Session 1 — Saturday, September 12 — 12:15 pm – 5:15 pm
Abstracts #7–#57

7 MORTALITY RATES AMONG 1192 POISONED PATIENTS AND DIRECT CAUSES OF DEATH.

Vieira RJ, Madureira PR, Zambrone FAD, Bucaretchi F. *CCI, Hospital de Clínicas, Faculdade de Ciências Médicas, Universidade Estadual de Campinas, SP, Brasil*

Background: Preventing death in intoxicated patients requires knowledge beyond mortality rates and substances involved. It is necessary to know and understand the mechanisms of the immediate cause of the death. Patients and Methods: The Hospital de Clinicas of University of Campinas is a tertiary level service in regional system that accepts intoxicated patients with high mortality rates related to severity. Between January 1971 to December 1996, the Emergency Infirmary cared for 1192 poisoned patients of various etiologies, of which, 97 died. Among these fatalities

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we studied the immediate cause of death according to product or product group, age, and days of hospitalization. We tried to understand factors related to the process of patient care, aiming to prevent death. Results: The most common products involved were pesticides (502 cases, 67 deaths), and drugs (392 cases, 15 deaths). Other agents accounted for the remaining 298 cases and 15 fatalities. Regarding the immediate cause of death of 97 patients, 38 were from respiratory failure, 16 were from sepsis, 13 were due to cardio-repiratory arrest, 13 were from multisystem organ failure, 12 were from shock, 9 were from other causes, and in 9 the cause was not established. All patients in the not established group died in the first 3 days of hospitalization. Some patients had more than one cause of death. All the 16 patients who died from sepsis spent 10 or more days at the hospital. Conclusions: The most common cause of death of 88 cases was respiratory failure followed by cardiac respiratory arrest leading to organ failure, and cardio-respiratory arrest which occurred before arrival to the hospital or during transportation. Sixteen patients who died from infection or sepsis were hospitalized for 10 or more days.

8 THE EPIDEMIOLOGY OF DEATHS RELATED TO TOXIC EXPOSURES IN THE US WORKPLACE, 1992-1996.

Greenberg M, Hamilton R. *Division of Toxicology, Department of Emergency Medicine, Allegheny University of the Health Sciences, Philadelphia, PA*

Background: This study describes the epidemiology of accidental deaths in the workplace caused by exposures to toxic substances over a five year period. Methods: Data were derived from information reported to the United States Bureau of Labor Statistics and were verified by at least two independent sources before inclusion in the data files. Data regarding etiology, demographics, worker activities, and workplace characteristics of 31,567 work related deaths occurring between 1992 and 1996 were analyzed. Deaths from suicidal or homicidal use of toxins were excluded. In addition, workplace toxins which caused fires or explosions which contributed to worker deaths were excluded. Results: During the years 1992 through 1996 inclusive, there were a total of 401 accidental workplace fatalities specifically related to toxic exposures. This represents 1.27% of all reported work related deaths during that period. Toxin related deaths of male workers exceeded toxin related deaths of female workers by more than thirty times. The majority (28.4%) of reported fatalities occurred in the 35-44 year age group. However, 10 workers age 19 or less and 21 workers aged 65 and over died from toxic exposures. Seventeen percent of toxin related deaths involved self employed individuals and 44% of deaths involved individuals working in businesses employing 10 or fewer employees. The construction trades, followed by mechanics represented the occupational groups at greatest risk for toxin related death in the workplace. Carbon monoxide was responsible for 35.8% of all toxin related deaths, followed by hydrogen sulfide (7.7%). Conclusion: This investigation demonstrates that toxic exposures are important causes of death in the workplace. The data reported in this study indicates the need for comprehensive workplace surveillance as well as improved risk assessment and risk communication regarding the potential lethality of specific toxins in the workplace, especially for small business.

9 POISON CENTER-BASED SURVEILLANCE: ADOLESCENT TOXIC EXPOSURES IN THE WORKPLACE.

Flynn B, Woolf A. *Massachusetts Poison Control System, Harvard Medical School, Children's Hospital, Boston, MA*

Background: As many as 5 million American children are legally employed; an additional 1 to 2 million are probably employed in violation of current regulations. While there are previous reports describing injuries to adolescents in the workplace, scant attention has been given to toxic substance exposures. There is currently no tracking of such poisonings among younger adolescents by federal agencies such as the Occupational Safety & Health Administration (OSHA). Yet low skill, entry-level jobs are those most commonly secured by adolescents; and such jobs may pose a particular hazard of toxic exposure due to the frequent use of cleaning compounds, solvents, and/or paints. Objective: To analyze the types and severity of adolescent occupational toxic exposures. Methods: Secondary analysis of the 1991-96 database at a regional PCC concerning children 14-19 yrs old with occupational exposures. Follow-up was performed on a sample to confirm worksite type. Results: Of 7024 occupational toxic exposures recorded over the 6 years, 269 incidents(3.8%) involved adolescents aged 14-19 years (median age=18 yrs; 65% males). The most frequently involved toxins included cleaning products (27.8%), paints/glues/solvents (9.0%), hydrocarbons (8.7%), caustics (8.7%), bleach (7.3%) and automotive supplies. Exposure routes included: inhalations (27.5%), ocular (27.5%), dermal (21.1%), ingestions (18.2%). Worksites were identified in 88 (32.7%) cases; they most commonly included: food services (30.7%), automotive services (14.8%), retail-general merchandise (12.5%), health services (9.1%), recreation (8.0%), and retail-food (6.8%). 157 of these cases were triaged to a health care facility for evaluation; 7 adolescents were hospitalized. 43 of 269 incidents(16.0%) resulted in moderate or severe injuries. The

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majority of these involved cleaning agents (29.1%) and caustics (18.8%). There were no deaths. Conclusions: Adolescent occupational exposures to toxins are an under-recognized but important injury category. Further studies are needed to outline the scope of the problem, to analyze the impact of such injuries on health resource expenditures, and to explore the use and effectiveness of preventive strategies.

10 EMERGENCY DEPARTMENT PREPAREDNESS OF HAZ-MAT INCIDENTS.

Quail MT, Woolf A. *Massachusetts Poison Control System, Boston, MA*

Background: There are no JCAHO or other guidelines for Emergency Departments (ED) to follow when establishing hazardous material protocols (e.g., treatment of chemically-exposed patients arriving from an exposure site to the ED), or to provide guidance in the building of adequate decontamination facilities. Objective: To determine the preparedness of EDs in this state for adequate medical management of victims of Hazardous Materials disaster incidents. Methods: All hospitals with active EDs listed with the state's Hospital Association, were sent a written survey in October 1997 asking 6 questions dealing with the management and treatment of the patient exposed to toxin(s) in the course of a HAZ-MAT incident. Results: 51 of the 89 EDs returned the survey (57%). 41 facilities (80%) had a written policy in place guiding staff on how to respond to patient medical management demands resulting from a HAZ-MAT incident. 11 (21%) EDs have had to evacuate their department at least once within the last 5 years. 23 (45%) facilities had both an area with separate ventilation and water containment; 11 (21%) had separate ventilation only; 13 (25%) had a portable decontamination unit; and 4 (7%) had no decontamination facility. 10 (19%) EDs have had to utilize their decontamination facilities within the last year. Conclusions: EDs in this state may not be adequately prepared to handle contaminated patients due to Hazardous Materials events. More specific oversight and regulations are needed to provide ED personnel with recommendations concerning the adequate preparation to respond to in HAZ-MAT emergency and to subsequently ensure they are in compliance with each recommendations.

11 HAZARDOUS MATERIALS INCIDENTS REPORTED.

Quail MT, Woolf A. *Massachusetts Poison Control System, Boston, MA*

Background: The National Fire Protection Association defines hazardous materials as "any substance that causes or may cause adverse effects on the health or safety of employees, the general public, or the environment and/or any biological agent and other disease-causing agent and/or waste or combination of wastes." Hazardous materials are all around us. Every day inhabitants of our society become potential victims because of an inadvertent release of a hazardous substance. Poison Control Centers (PCC) are utilized as a resource to assist the public and health care providers about the health risks and treatment management of victims. Objective: To determine the locations, frequency and type of hazardous material exposures reported to a PCC. Method: Each AAPCC exposure record was reviewed from year 1997. All exposures from residence were discarded except for 2. Only those caller sites reported from a Health Care Facility (HCF) or Other were evaluated. Results: In 1997 our center received 35,834 exposures. 69 incidents involving 90 toxic substances were reported involving over 3,972 people; 247 required transport to an emergency facility for treatment; 2 fatalities occurred directly from the incident. Exposures sites occurred in 32 commercial buildings (0.46%); 15 schools (0.22%); 8 HCF (0.12%); 4 unknown public areas (0.06%); 3 private labs (0.04%); 2 residence (0.03%); 1 railroad yard (0.01%); 1 manhole; 1 fuel storage area; 1 prison; and 1 unknown. 3 explosions occurred (1 residence), 1 manhole (involving 1 death), 1 school lab, and 1 explosive level of natural gas at an HCF. The route of exposures were inhalation (41), dermal (14), inhalation & dermal (11), ocular and inhalation (2), and none (1). Conclusion: Hazardous material exposures involve a large assortment of toxic chemicals and other substances that are frequently reported to PCC. The sites of exposures are variable; however in this study commercial buildings, schools, and health care facilities comprised 80% on the exposures.

12 AN EVALUATION OF THE INCIDENCE OF EMESIS FOLLOWING ORAL BOLUS DOSING VS GRADUAL ADMINISTRATION REGIMENS OF ACTIVATED CHARCOAL.

Holmes C, Schauben J, Hasan MY. *Florida Poison Center-Jax, UFHSC, Jacksonville, FL*

Background: Activated charcoal is frequently used for gastrointestinal decontamination of the poisoned patient. To date, no studies have evaluated the incidence of emesis associated with bolus vs. gradual dosing of activated charcoal. This study was designed to investigate the incidence of emesis associated with these two regimens. Methods: All patients, 18-65 years old, presenting to the emergency department with suspected overdose were considered for enrollment. After verbal consent was obtained, patients were randomly assigned to one of two treatment groups. Group A was administered a 50 g oral "bolus" dose of activated charcoal diluted with 120 ml of water, and 108 g

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of sorbitol, over a 15 minute interval. Patients in group B received the same dose as group A, but in equal increments at 15 minute interval for one hour. Route of administration (orally vs. use of nasogastric tube) was determined by the treating physician. Patients were observed for 4 hours in the emergency department to document any episodes of emesis. Demographic data, previous incidence of emesis, the substance ingested and time to emesis were also recorded. The data were evaluated by chi-square analysis. Results: A total of 250 patients were enrolled; 136 females and 114 males. Group A and Group B had 135 and 115 patients respectively. The incidence of emesis was 17% (n=250). No difference existed between group A and group B with respect to route, dose, time to administration of charcoal, incidence of emesis, episodes of emesis, time to emesis or age. Females had a statistically significant higher incidence of emesis compared to males (77% and 23% respectively; $p<0.001$). Conclusion: The results of this study indicate that the incidence of emesis after charcoal administration is independent of rate or route of administration. Gender seemed to play a role in the incidence of emesis. The results of this study will need to be confirmed with larger study groups.

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13 EMESIS IN THE POTENTIALLY POISONED PATIENT—IS IT WORTH THE RISK?

Cantrell FL, Phillips M. *California Poison Control System-San Diego, UCSD Medical Center, San Diego, CA*

Background: Induction of emesis has long been considered a viable treatment modality. Emesis has been induced when contraindicated, ill advised, or unnecessary. Ipecac is traditionally used, although unorthodox and potentially dangerous methods are often used. This series evaluated various methods used in the community, the appropriateness of the situation, and any negative outcomes. Methods: Retrospective analysis of human exposure cases called into a poison control center (PCC) in which emesis was induced prior to contacting PCC were prospectively collected over a nine-month period. Results: 317 cases identified, 129 (41%) utilized ipecac, 159 (50%) used foreign objects (alone or in combination with ipecac) (eg. fingers, toothbrushes, cotton swabs), 22 (7%) used various methods (eg. ingesting salt water, cooking oil, or raw eggs, pressure on the abdomen, inverting and shaking children, and giving noxious medications, in 9 (3%) the method was unspecified. In 107 cases (33%), emesis would have been contraindicated or ill advised based on the substance ingested. In 53 (17%), the substance ingested was non or minimally toxic. Emergency departments induced emesis in 34 cases (11%), private physicians advised emesis at home in 11 (3%), other health care personnel (pharmacists, paramedics) recommended home emesis in 7 (2%). Induction of emesis was initiated within 30 minutes of ingestion in 226 cases (71%), 30-60 minutes in 75 (24%), and >60 minutes in 16 (5%). In 310 cases in which follow-up was obtained, significant adverse effects directly related to the induction of emesis occurred in 8, including protracted vomiting delaying activated charcoal and chemical pneumonitis secondary to aspiration. Conclusions: While the use of emesis for gut decontamination is declining in popularity, the public as well as health care professionals are still utilizing it. We demonstrated that the public is using potentially hazardous methods in the majority of cases. Health care professionals and the public both induced emesis in situations considered dangerous or unnecessary. Further education is needed.

14 AVAILABILITY OF ACTIVATED CHARCOAL AND IPECAC AT PHARMACIES IN CALIFORNIA.

Nordt SP, Manoguerra A, Williams S, Clark R. *CPCS-San Diego Division, Division of Medical Toxicology-Emergency Medicine, University of California, San Diego Medical Center, San Diego, CA*

Background: Gastrointestinal (GI) decontamination is often employed in an effort to remove or to adsorb potentially toxic ingested substances. There are several limiting factors to effective GI decontamination, specifically the rapidity of administration and the availability of the antidote. Traditionally, ipecac has been used for home GI decontamination, however, more recently attention has been focused on pre-hospital and home activated charcoal (AC) administration. In a effort to assess the availability of AC and ipecac and need for education, we conducted a telephone survey. Methods: One-hundred eighteen pharmacies were randomly identified from the Yellow pages from the 59 counties in California to assess availability of AC (liquid or powder) and/or ipecac. Results: Ninety-four (80%) pharmacies were available for analysis. There was no difference between community (44) and chain (50) pharmacies ($p=0.7046$). Seventy-nine of the pharmacies had ipecac compared to 8 which had AC. Three pharmacies had pre-mixed aqueous AC while 5 had AC in powder formulation. There was no difference between chains and community in AC (5 vs. 3, $p=0.8905$) or ipecac (42 vs. 37, $p=0.8812$) availability. One pharmacy provided ipecac free of charge but did not stock AC. One pharmacist said activated charcoal was not recommended for poisonings. Seven (0.08%) of the pharmacies that did not have AC could order AC if needed. Conclusions: The major limitations of effective GI decontamination are the rapidity in administration and the availability. Our results identify there would be a significant delay in the administration of AC if a parent was referred to a local pharmacy for home or pre-hospital decontamination. In addition, when AC was available most was the powdered formulation which would

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require the parent to constitute further delaying decontamination. As more toxicologists and poison centers move towards pre-hospital and home AC much education of pharmacists as well as parents needs to be done. Pharmacies should be encouraged to carry ready-made, aqueous AC.

15 DOES INSUFFICIENT STOCKING OF EMERGENCY ANTIDOTES IN HOSPITAL PHARMACIES PERSIST?

Bogdan G, Holt S, Mlakar P, Dart R. *Rocky Mountain Poison & Drug Center, Denver, CO*

Background: Insufficient stocking of poisoning antidotes in hospital pharmacies was reported in *JAMA* (1996;**276**:1508-1510) and garnered considerable media attention. Fifteen months after the report, hospital pharmacies were again surveyed to determine antidote stocks. Objective: Determine whether insufficient stocking of emergency antidotes for poisoning and overdose still exist in hospitals that provide emergency care compared to prior published data. Methods: A written survey of hospital pharmacy directors of all hospitals with emergency departments in Colorado, Montana and Nevada. Respondents reported the amount currently in stock of 8 different antidotes. Insufficient antidote stocking was defined as lack of the antidote or an amount inadequate to initiate treatment of 1 seriously poisoned 70-kg patient: crotalid antivenin (5 vials), cyanide kit (1 kit), deferoxamine (1-g), digoxin immune Fab (20 vials), ethanol (70-g), naloxone (2-mg), pralidoxime (2-g), and pyridoxine (5-g). The proportion of hospitals with insufficient antidote stocking from the current survey was compared to prior published data. Results: Questionnaires were mailed to 168 hospital pharmacy directors, 126 (77%) responded. Only 2 (1.6%) of the 126 hospitals stocked all 8 antidotes in adequate amounts. The rate of insufficient stocking for antidotes ranged from 3% (naloxone) to 98% (digoxin immune Fab), as in the prior survey. There was no significant change in the insufficient stocking of 5 antidotes as indicated by two-tailed Fisher's Exact testing ($p < 0.5$). There was significant improvement (negative % change) in the insufficient stocking for 3 antidotes: ethanol (-19.3%), pralidoxime (-16.3%), and pyridoxine (-18.6%). Conclusions: Although some improvements have been made, insufficient stocking of antidotes continues to be a wide spread problem in Colorado, Montana, and Nevada. Although these states are served by a certified regional poison center, potentially lifesaving antidotes are frequently not available to treat a single poisoned patient.

16 IMPACT OF *N*-ACETYLCYSTEINE AND NALOXONE CONSUMPTION ON ANTIDOTE AVAILABILITY.

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Background: Recent reports suggest insufficient antidote stocking by hospital pharmacies. Infrequent antidote use has been proposed as an explanation. Objective: To study the impact of *N*-acetylcysteine and naloxone consumption on the availability of 13 antidotes. Methods: Written surveys were sent to 147 hospital pharmacies. Amount of *N*-acetylcysteine and naloxone purchased over the 1996-97 fiscal year were recorded and used as a surrogate for yearly consumption of these antidotes. Adequate stocking was defined as minimum stocking level suggested by the San Francisco Bay area Regional PCC. The number of adequately stocked antidotes was correlated separately with the amount of *N*-acetylcysteine and the amount of naloxone consumed and the number of hospital beds. Results: Preliminary data was obtained from 66% of the pharmacies surveyed. Number of adequately stocked antidotes per hospital ranged from 0 to 10 out of 13. The proportion of hospitals carrying minimum stocking levels of antidotes was: *N*-acetylcysteine 87%, alcohol 55%, cyanide kit 17%, deferoxamine 27%, digoxin immune FAB 12%, dimercaprol 1%, flumazenil 43%, glucagon 0%, methylene blue 12%, naloxone 41%, physostigmine 12%, pralidoxime 8%, and pyridoxine 0%. There was a correlation between the number of hospital beds and the number of adequately stocked antidotes ($r = 0.47$ $p < 0.001$). Correlation between the number of adequately stocked antidotes and the amount of *N*-acetylcysteine and naloxone consumed were weak but significant ($r = 0.53$ $p < 0.001$ and $r = 0.56$ $p < 0.001$). A stepwise logistic regression model showed that the amount of *N*-acetylcysteine and naloxone consumed independently predicted the presence of adequately stocked antidotes but not the number of hospital beds ($r = 0.62$ $p = 0.002$). Conclusion: Larger hospitals are more likely to have adequate antidote stocking. Adequate antidote stocking is significantly correlated with the amount of *N*-acetylcysteine and naloxone purchased. This suggests that hospitals more likely to see serious acetaminophen and opiate poisonings are more likely to maintain adequate antidotes stocking.

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17 OCULAR EXPOSURES: A SUMMARY AND COMPARISON OF FIVE REGIONAL POISON CENTERS.

Thomas R, Stevens D. *Samaritan Regional Poison Center, Samaritan Health System, Phoenix, AZ*

Background: Ocular exposures account for 7% of human exposure cases reported annually to AAPCC-TESS. No multi-center review of this type of exposure has been reported previously. Methods: All human, ocular exposures recorded by five regional poison centers in 1997 were analyzed for the following: most frequent AAPCC generic substance categories, site of exposure, treatment location and referral frequency, signs and symptoms, and patient outcomes. Results: A total of 16,099 cases were reviewed. The most frequent substance category for overall calls and referrals to a health care facility (HCF) was household cleaning products (24%). The other categories in order of overall frequency were cosmetics and personal care items (15%), plants (7%), chemicals (6%), hydrocarbons (6%), topical medications (5%), insecticides (5%), adhesives and glues (4%), ENT preparations (3%) and foreign bodies, etc. (3%). Referrals to HCFs by both the patients and poison centers were the same as the overall top three categories. However, glues and adhesives which ranked only 8th overall, ranked 4th among the categories receiving treatment in a HCF. 89% of exposures occurred at home followed by the workplace (7%) and schools (2%). Patients complained primarily irritation and pain (74%) and lacrimation (8%). Only 3% had a corneal abrasion. Approximately 25% of ocular calls were for exposures for which no symptoms were listed. Approximately 75% (range=64-77%) of calls were managed at home. On average, 18% of callers self-referred to a health care facility (range=11%-25%) while only 9% of calls to a poison center resulted in referral (range=7-11). Outcomes were distributed as follows: No effect-12%, Minor effect-47%, Moderate effect-6%, Major-<1% (1 case), non-toxic, not followed-1%, minimal toxicity expected, not followed-28%, potentially toxic, unable to follow-5%. Treatment consisted almost exclusively of irrigation. Documentation was insufficient to accurately describe treatment in HCFs. Conclusions: A majority of ocular exposures can be safely treated at home. Self-referral rates are significantly higher than referrals by poison centers. Those requiring evaluation are routinely treated and released and rarely admitted.

18 SCHOOL NURSE UTILIZATION OF THE POISON CENTER: FOCUS ON OPHTHALMIC EXPOSURES.

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Background: In 1996 the American Association of Poison Control Centers reported 27,691 poisoning exposures in schools. A study was undertaken to specifically characterize ocular exposures reported to the Poison Center by school nurses in our state in order to more precisely focus on prevention. Methods: A retrospective study was done reviewing calls placed to the Poison Center by school nurses from September 1996 through June 1997. Data reviewed included the age and sex of the patient, town of origin, route of exposure, symptoms before treatment, and symptoms or outcome after treatment. Only calls initiated by school nurses were included. Results: The results of the study are as follows: 579 poisoning exposures were documented; 108 (19%) of these were ocular exposures. 26 (24%) were due to cleansers; 21 (20%) were science or shop related. The number of eye exposures from cleansers was higher in the spring months with 16 occurring in March, 11 in April and 13 in May. Many different soaps, Formula 409®, Fantastik®, white board cleaner, and ammonia were among the cleaning products involved. All of the children with ocular exposures required eye irrigation and 13 (12%) required further medical evaluation due to prolonged irritation. Other causes of ocular exposures included nitric, sulfuric and hydrochloric acids, copper sulfate, and formaldehyde used in science and shop classes. 2 cases of corneal burns were the result of sulfuric acid, and a mixture of hydrochloric acid and carbon disulfide, used in this setting. Conclusions: Poison education should be aimed at school nurses. They should be instructed in adequate eye irrigation, and made aware of the importance of calling their poison center for any suspected exposure. These ocular exposures may have been preventable with proper safety education, an area where school nurses can make a difference. They should also be encouraged to work with the school authorities to purchase and mandate the use of protective eye gear in science and shop classes. Cleaning agents should be kept out of the reach of children and used by housekeeping. School nurses and Poison Control Centers must join together to promote children's health.

19 ROUTINE URINE DRUG SCREENING OF PREGNANT PATIENTS IN THE EMERGENCY DEPARTMENT.

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Background: It has been estimated that 10-27 percent of pregnant women in the US have a pattern of illicit drug use, which is felt to be underreported. All studies up to now have been conducted in obstetric clinics or at time of

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delivery. This study was designed to determine the prevalence of recent illicit drug use and the reliability of self-reporting in pregnant patients presenting to the emergency department. Methods: Prospective convenience sample of 75 pregnant women presenting to an urban university hospital emergency department were asked about patterns of recent illicit drug use during routine their social history. Anonymously, urine samples were then qualitatively tested for drugs of abuse by EMIT (Cannabs, Benzos, Cocaine metabolites, Opiates, PCP, Amphet, Barbs). Results: Of the 75 women, 13% (10) tested positive for one or more substances, with cocaine being most prevalent at 56%. 80% of the patients with positive urinary assays denied illicit drug use, for a disclosure rate of only 20%. Of the 5 women (7%) who admitted to drug use within the past ten days, only 2 had a positive urine assay. There was no correlation between drug use and diagnosis of pregnancy complications. All but one patient with recent drug use (either positive urine assay or admitter) had previously known she was pregnant. Conclusion: This study stresses the high prevalence of illicit drug abuse in pregnant, inner city, women. Patient self-reporting of drug use is highly inaccurate and unreliable. Given such a small disclosure rate, routine urine drug screening, in this population, may be warranted.

20 IS NALOXONE CONTRAINDICATED IN TRAMADOL OVERDOSE?

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Background: Tramadol is an analgesic with opioid and monoaminergic activity. Tramadol is metabolized by CYP2D6 to (+) *o*-desmethyltramadol, a potent μ-opioid-receptor agonist. Monoaminergic effects result from inhibition of norepinephrine reuptake and stimulation of serotonin release, primarily by the parent compound. In tramadol-poisoned patients either opioid or monoaminergic manifestations may predominate, with the balance potentially being affected by prior therapeutic use of CYP2D6 inhibitors, acute coingestants, and naloxone administration. In a three-year period (1995-1998), 6 of 42 patients with tramadol toxicity reported to our center exhibited dominant monoaminergic features, with several being on chronic therapy with CYP2D6 inhibitors. Of these, we report two contrasting cases. The first had improvement in mental status after naloxone administration but the second developed seizure activity. In humans, seizures have been reported with therapeutic doses of tramadol as well as overdose. In animals, administration of naloxone in tramadol poisoning increases the risk of seizures. The relationship between naloxone administration and seizures in tramadol-poisoned patients has not been characterized. Case Reports: A 56-year-old female taking paroxetine, lorazepam, imipramine, and ranitidine, presented after tramadol overdose. She was unresponsive to verbal stimuli but had spontaneous respiration, systolic hypertension (160/80 mmHg) and increased muscle tone. After 2mg of naloxone she became communicative. A 37-year-old female taking fluoxetine and furosemide presented after tramadol overdose. She was responsive to verbal stimuli with good respiratory effort. She received 2mg of naloxone without initial CNS change. Shortly afterwards she suffered repeated short-lived seizures necessitating benzodiazepine administration. This patient's response may be explained by CYP2D6 inhibition due to fluoxetine and resultant accumulation of monoaminergic parent compound. Conclusion: Tramadol toxicity can present with dominant opioid or monoaminergic features. Naloxone reverses opioid effects in tramadol poisoning, but its use may be associated with seizures. Further investigation is needed to determine the role of naloxone in tramadol toxicity when chronic CYP2D6 inhibition, acute coingestants with monoaminergic activity, or monoaminergic clinical manifestations are present.

21 CONTINUOUS INTRAVENOUS FLUMAZENIL INFUSION FOR BENZODIAZEPINE POISONING.

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Background: This is the first United States (US) report of continuous intravenous (IV) flumazenil infusion for benzodiazepine poisoning. A MEDLINE search from 1966 to 1998 revealed no similar reports in the US literature. Case Report: A 24-year-old woman ingested fifty, 2 mg flunitrazepam tablets in a suicide attempt. She presented 30 minutes after ingestion with a temperature of 36.5°C, blood pressure of 89/36 mmHg, pulse of 83/min, and shallow respirations of 8/min. Her Glasgow coma scale (GCS) was 8. Her ECG showed sinus rhythm at 80/min, a QRS axis of 30° with no terminal 40 msec deviation, and a QRS interval of 84 msec. She received 0.2 mg of IV flumazenil and her GCS improved to 15. She was orogastrically lavaged and given 50 g of activated charcoal. Resedation to a GCS of 8 recurred twice, requiring additional 0.3 mg and 0.5 mg IV boluses of flumazenil, totaling 1.0 mg over 1 hour. Then, a continuous IV flumazenil infusion was started at 1.0 mg/h, maintaining her GCS at 15. Fourteen hours later, the flumazenil continuous IV infusion was terminated, resulting in resedation and hypoventilation. Therefore, the flumazenil infusion was restarted at 1.0 mg/h with resolution of sedation and hypoventilation. Thirty hours after overdose, her flumazenil infusion was terminated without resedation. Conclusion: This is the first US report

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describing the successful use of a continuous IV flumazenil infusion. This is not FDA approved and further study is necessary to determine its safety and efficacy in carefully selected patients.

22 A COMPARISON OF CHARCOAL AND CHOLESTYRAMINE DIALYSIS OF PHENYTOIN AND PARACETAMOL *IN VITRO*.

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Background: Activated charcoal is used for treating phenytoin and paracetamol toxicity, both to reduce drug absorption from the gut and, on repeated administration, to enhance elimination. Despite the understanding that cholestyramine reduces drug absorption from the gastrointestinal tract, its use in treating drug intoxication remains skeptical. Here, we use equilibrium dialysis to compare charcoal and choiestyramine bonding of phenytoin and paracetamol *in vitro*. Methods: Into the chamber of a Slid-A-Lyzer dialysis Cassette (Pierce, USA; membrane pore diameter of 3000 mol wt), was injected 3 mL of plasma spiked with phenytoin sodium 91.3 mcg/ml or paracetamol 7.29 mg/ml. This was then dialyzed against 250mL of plain phosphate buffer pH 7.40, or charcoal or cholestyramine in similar buffer. Drug concentrations in plasma and dialysis buffer were determined at intervals. Linear regression was used to derive half-life. Results: The rate of drug loss from plasma chamber was enhanced by both charcoal and cholestyramine as follows: In plain buffer, the half-lives for paracetamol and phenytoin where 0.96 ± 0.03 and 2.78 ± 0.18 hours, respectively. With activated charcoal, these values fell to 0.52 ± 0.08 and 1.2 ± 0.10 hours for paracetamol and phenytoin, respectively, as compared to cholestyramine which yielded half-lives of 0.5 ± 0.01 and 1.17 ± 0.16 hours again for paracetamol and phenytoin, respectively. After six hours of dialysis, the plasma concentration of paracetamol was 78.9 ± 8.3mcg/ml with plain buffer, 35.21 ± 7.71mcg/ml with cholestyramine and 1.48 ± 1.76mcg/ml with charcoal. Corresponding concentrations for phenytoin were $9.67 \pm$ 0.79mcg/ml, $2.89 \pm$ 0.99mcg/ml and $1.82 \pm$ 1.41mcg/ml respectively. Of note, doubling the concentration of cholestyramine decreased paracetamol concentration at six hours to 20.79 ± 5.82mcg/ml. Conclusions: Results indicate that activated charcoal is superior to cholestyramine for paracetamol, while both compounds were equipotent for phenytoin dialysis. However, since the concentrations of paracetamol at which charcoal and cholestyramine differ are non-toxic, clinical studies are required to determine the significance of this observation and to design appropriate regimens of cholestyramine as an alternative to charcoal in the treatment of paracetamol and phenytoin toxicity.

23 TOXIC BABY FORMULA: FLEETS® ENEMA CONFUSED FOR BABY WATER BOTTLE.

Hung OL, Nelson LS, Howland MA, Hoffman RS. *New York City Poison Control Center, New York, NY*

Introduction: Toxic exposures involving children are often the result of mistaken identification and inappropriate usage of a toxic product due to incorrect labelling or storage. Fortunately, most exposures are benign. We report a rare case of life-threatening toxicity in an infant resulting from an error in identification and storage of an OTC remedy. Case Report: An 8-week-old healthy female presented to the hospital after ingestion of 2-3oz of formula prepared with Fleets enema solution. The Fleets enema solution was placed in the refrigerator in its original container and the babysitter mistook it for "bottled water." Shortly after the baby was fed, the baby-sitter reported to the mother that the baby "did not look right" and soon became lethargic. When the parents arrived home, the baby was lethargic and unresponsive. Initial vital signs were: temperature 101°F, blood pressure 86/50 mm Hg, pulse 170/min, respirations 50/min. The baby was pale and unresponsive to deep pain. She had dry mucous membranes and a depressed anterior fontanelle. Her arms were flexed, her legs were straight with increased tone. Initial serum electrolytes revealed: sodium 164mmol/L, potassium 5.3mmol/L, chloride 107mmol/L bicarbonate 19mmol/L, blood urea nitrogen 10mg/dL, creatinine 0.8mg/dL, calcium 4.3mg/dL, and phosphate 49mg/dL. Initial resuscitation included multiple fluid boluses, intravenous calcium gluconate, oral aluminum hydroxide, and intravenous antibiotics. In the ICU, the patient had two seizures. She was intubated but became bradycardic (pulse=50/min) shortly after intubation. The patient's bradycardia was responsive to multiple doses of atropine and epinephrine. A dopamine infusion was started because of hypotension and oliguria, and she was transferred for emergency peritoneal dialysis. The patient's electrolytes normalized by hospital day #2. Dialysis was discontinued and the patient was extubated. The infant was discharged home with her parents after a child abuse investigation was completed. Conclusion: This case illustrates that potentially catastrophic results may occur when common household products are mistakenly identified. Treatment of life-threatening hyperphosphatemia includes: aggressive volume repletion, correction of hypocalcemia, and extracorporeal phosphate elimination.

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24 OCTREOTIDE FOR PEDIATRIC SULFONYLUREA POISONING.

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Background: Octreotide, a somatostatin analogue, has been reported to be effective in treating hyperinsulinemic hypoglycemia resulting from sulfonylurea poisoning in adults. Although it has been used in the treatment of other conditions in infants and children, its use in children sulfonylurea-induced hypoglycemia has not previously been reported. Case Report: A 5-year 9-month weighing 20 kg male was brought to the ED in status epilepticus. He had a history of Attention Deficit Hyperactivity Disorder but no history of seizures. The seizures stopped after lorazepam 1 mg IV. Initial blood sugar was 12mg/dl. Despite treatment with IV glucose (up to 18 g/h) he developed recurrent hypoglycemia. 5 hours after presentation, it was discovered that glipizide (5 mg) had been dispensed instead of Adderall® (amphetamine and dextroamphetamine), and that he had been given 7.5 mg of glipizide BID for the preceding 3 days, with the last dose 14 hours prior to arrival. IV octreotide (25μg) was administered. The glucose concentration rose promptly to 150-200mg/dl, allowing for the rapid weaning of supplementary glucose and cessation of such therapy 4 hours after octreotide administration. Subsequent laboratory analysis confirmed both the glipizide exposure and the efficacy of octreotide: the initial serum glipizide concentration was 0.4μg/ml with 0.5μg/ml expected as the peak concentration after 5 mg in adults (half-life - 3.5 hours); serum insulin [simultaneous glucose] concentrations were 53 μIU/ml [45mg/dl] before octreotide and 16μIU/ml [183mg/dl] 3 hours later (physiological fasting insulin concentration - < 20-30μIU/ml). Conclusions: As in adults octreotide is effective in blocking pancreatic insulin secretion in response to glucose administration and in treating recurrent hypoglycemia following sulfonylurea poisoning in children. Octreotide is recommended as adjunctive therapy for pediatric sulfonylurea poisoning.

25 OCTREOTIDE: AN ANTIDOTE FOR SULFONYLUREA-INDUCED HYPOGLYCEMIA.

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Background: Sulfonylurea agents produce hypoglycemia by facilitating insulin release from pancreatic B-cells. Dextrose is commonly used to treat sulfonylurea-induced hypoglycemia but is a potent stimulus for additional insulin secretion and frequently causes rebound hypoglycemia. Octreotide, a long-acting analogue of somatostatin, is a potent inhibitor of pancreatic insulin release and has been used as a treatment for sulfonylurea-induced hypoglycemia in one volunteer study and in isolated case reports. We assessed the impact of octreotide treatment on rebound hypoglycemia in nine patients with sulfonylurea-induced hypoglycemia from 1995-1998. Case Series: The age range of these nine cases was 20 to 65 years, and included six glyburide ingestions and three glipizide ingestions. Three cases involved therapeutic misadventures and six cases were from intentional overdose. Dosing of octreotide ranged widely from a single dose of 40mcg SQ to an infusion of 125mcg IV per hour. There were no side effects from octreotide administration. Prior to the first dose of octreotide, there was a combined total in all nine patients of 29 episodes of hypoglycemia (less than 60mg/dL) requiring 27 ampules of 50% dextrose. After the first dose of octreotide, there was a combined total of 2 episodes of hypoglycemia requiring 2 ampules of 50% dextrose. The mean number of hypoglycemic events recorded per patient before octreotide (3.2) was significantly greater than the mean number of hypoglycemic events recorded after octreotide (0.2), $p=.001$ (paired t-test). Similarly, the mean number of ampules of 50% dextrose required per patient before octreotide (2.9) was significantly greater than the mean number of ampules required after octreotide (0.2), $p=.001$ (paired t-test). Stabilization of blood glucose and cessation of rebound hypoglycemia occurred immediately after the administration of octreotide in all nine patients. Conclusion: Octreotide appears to be safe and highly effective at stabilizing blood glucose and preventing rebound hypoglycemia after sulfonylurea ingestion. Controlled studies are needed to confirm these results and to investigate optimal dosing and observation requirements.

26 PROLONGED HYPOGLYCEMIA FOLLOWING ACUTE GLIMIPERIDE OVERDOSE.

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Background: We describe a case of prolonged hypoglycemia following acute overdose of glimiperide, a new oral hypoglycemic drug of the sulfonylurea class (second generation). Case Report: A 22-year-old male presented to the ED within an hour after ingestion of #90 glimiperide, #15 furosemide, and unknown amounts of clarithromycin, acetaminophen, and ethanol. Upon arrival to the ED vital signs were as follows: pulse 105bpm, BP 130/70mmHg, RR 29/min, temp 97.0°F. Pupils were mid-range and reactive. Labs revealed a pH 7.4, white count 145,000/mm^3,

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hematocrit 40.9%, EtOH 114mg/dL, acetaminophen 0mg/dL, serum toxicology screen negative, urine tox screen positive for cocaine. Glucose was 118mg/dL. The patient was administered a one ampule D_{50} bolus, started on D_5W drip and transferred to a tertiary care facility. While in the second facility, his glucose dropped to 64mg/dL and he was again administered D_{50}. His glucose continued to decrease to 45mg/dL. He remained on a D_5W drip until transferred to the ICU where fluids were changed to $D_{10}NS$ at 100 ml/h. Over the ensuing 8 days, the patient's blood sugar ranged from 49mg/dL to 158mg/dL and was extremely variable. D_{50} bolus was ordered for blood sugar less than 100mg/dL, and the patient received 49 D_{50} boluses in the first three days. Conclusion: Glimiperide overdose can result in prolonged hypoglycemia that can cause wide swings in blood sugar and require large amounts of glucose supplementation.

27 PAMIDRONATE AS A TREATMENT FOR SEVERE HYPERCALCEMIA SECONDARY TO ACUTE VITAMIN D POISONING.

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Background: Pamidronate disodium, a bone resorption inhibitor through osteoclast mediation, has been used to treat chronic hypercalcemia secondary to malignancy and chronic renal failure. There is scant data on the use of pamidronate for acute hypercalcemia secondary to vitamin D poisoning. Case Report: A 77-year-old female with a history of chronic lymphocitic leukemia, peptic ulcer disease, multiple cerebral infarct, dementia, congestive heart failure, and osteoporosis was discharged from a hospital to a nursing home facility with a normal renal function and calcium status. The patient's medications included furosemide, cephalexin, cimetidine, vitamin D, and calcium supplements. The patient was accidentally given 50,000 units of oral vitamin D rather than the correct dose 5,000 units. The patient presented to the emergency department six days later with a four day history of lethargy, abdominal pain, and vomiting. The patient's initial laboratory values were: calcium, 5.25mmol/L (21mg/dL); ionized calcium, 1.87mg/dL; 25 OH vitamin D3, 280ng/mL (nl 10-53); 1,25 vitamin D3, 39pg/mL (nl 24-65); BUN, 17mg/dL; creatinine, 1.9mg/dL. The patient was initially treated with hydration and furosemide. However the patient developed congestive heart failure. A single dose of 90mg of intravenous pamidronate was administered over four hours. The patient had a normal serum calcium concentration within 24 hours after treatment. Calcium concentrations (in mmol/L) post-pamidronate treatment were as follows: presentation, 5.25; 6 hours, 3.75; 12 hours, 2.75; 18 hours, 2.50; 24 hours, 1.50; 30 hours, 2.45; 36 hours, 2.45; 42 hours, 2.45; 48 hours, 2.20. The patient's calcium concentration troughed on the 30th hour of post-treatment at 1.50mmol/L. The patient was asymptomatic at this time. The patient recovered successfully and no significant sequelae was observed. The patient was discharged from the hospital with the corrected dose of medication. Conclusion: We report a case of the use of pamidronate for significant hypercalcemia secondary to acute vitamin D toxicity. Although evidence of congestive heart failure and renal failure was evident, dialysis was avoided without significant sequelae. Pamidronate therapy should be considered in patients with hypercalcemia secondary to acute vitamin D poisoning.

28 GREEN-BLACK URINE IN THE SETTING OF METRONIDAZOLE OVERDOSE.

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Background: Metronidazole is a commonly prescribed drug with antibacterial and antiparasitic properties. Side effects have included headache and gastrointestinal symptoms. We report a case of dark urine in the setting of metronidazole overdose. Case Report: A 15-year-old female presented 12 hours after ingesting 15-20 metronidazole tablets in a suicide attempt with a chief complaint of nausea, vomiting and diarrhea. Her symptoms began 5 hours post ingestion. Vital signs were Temp 37.2 P 92 R 16 BP 108/94. Past medical history was notable for depression. She denied taking other medications and reported no known allergies. To our knowledge this medication did not belong to the patient. Physical examination was normal. Laboratory evaluation was remarkable only for green/black colored urine with specific gravity of 1.025, positive bilirubin, protein, nitrite, leukocyte esterase and blood on macroscopic urinalysis. Microscopic analysis was notable for WBC 2-3, RBC >200, and marked bacteria. Urine toxicology could not be performed secondary to the intense color of the urine. Serum bilirubin and liver transaminase studies were normal. Methemoglobin was 0.3% This discoloration began to clear at 23 hours post ingestion. The patient was medically cleared without further incident the following day. Conclusion: Metronidazole overdose may result in transient urine discoloration. A review of the literature was notable for one previous report of urine discoloration in the setting of treatment of amebic dysentery with metronidazole. In that case, the urine was reported to be dark brown with an otherwise normal urinalysis. To our knowledge our case is the only such color change described in the overdose setting. This color change appears to interfere with laboratory analysis of urine. The etiology is unclear although a metabolite of metronidazole appears to be the likely culprit.

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29 MISUSE OF ANALGESICS FOR ODONTALGIA AND OTHER DENTAL- AND ORAL-CARE PRODUCTS.

Suchard JR. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: Poison control centers receive many calls regarding exposures to various dental- and oral-care products. We determined the volume and nature of calls regarding these products, and found which exposures resulted in significant toxicity or a referral for medical evaluation. Methods: Retrospective review of all 65,849 calls to the local regional poison control center, as entered into a computerized database during the calendar year 1997. Cases were included if the caller reported exposure to, or had a question regarding the use or misuse of a dental- or oral-care product. Cases involving the use of analgesics or local anesthetics specifically for dental pain were also included. Results: 676 calls met inclusion criteria, representing 1.02% of all calls received. The most commonly involved products were toothpaste (160 cases), mouthwash (111), local anesthetics (68), dental rinses (49), analgesics (48), and fluoride supplements (36). Ten patients were admitted to the hospital; 4 for salicylate intoxication occurring as a therapeutic misadventure (one patient was hemodialyzed for coma and seizures), 4 for *N*-acetylcysteine treatment following acetaminophen overdose, 1 for seizures from prilocaine and lidocaine injected by the dentist, and 1 for unresponsiveness following use of methamphetamine and cinnamon oil for odontalgia. Fourteen additional cases were referred to the Emergency Department for medical evaluation but did not require admission; 6 for benzocaine ingestions, 3 for aspirin overdose, 2 for acetaminophen overdose, and 1 each for ingestion of eugenol, camphor/phenol local anesthetic, and toothpaste. The 24 cases admitted or evaluated in the Emergency Department represent 3.55% of all calls regarding oral- and dental-care products. Conclusion: Among calls to poison centers regarding oral- and dental-care products, over-the-counter analgesics and local anesthetics used for dental pain appear to pose the greatest risk for toxicity and the need for hospital admission.

30 I CAN'T BELIEVE IT'S NOT TOOTHPASTE: INADVERTENT TOOTHBRUSHING WITH NON-TOOTHPASTE PRODUCTS.

Suchard JR. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: Poison control centers receive many calls regarding exposures to household products not used for their intended purpose. A cluster of inadvertent toothbrushings with non-toothpaste products prompted a review of this type of incident. We sought to determine the number of such incidents, the nature of the non-toothpaste products involved, and whether such exposures resulted in any significant toxicity. Methods: Retrospective review of all 65,849 calls to the local regional poison control center, as entered into a computerized database during the calendar year 1997. Cases were included if the caller reported an incident where a non-toothpaste product was used to brush teeth. Results: 122 cases met inclusion criteria. 115 cases were truly inadvertent; while in 7 cases, the patient intentionally used a non-toothpaste product and then called the poison center regarding actual or potential side effects. The products most commonly mistaken for toothpaste were methyl salicylate muscle rubs (30 cases), corticosteroid creams (18), zinc oxide diaper rash ointments (16), and capsaicin creams (8). Two calls came from medical practitioners, when the patient sought medical evaluation prior to poison center notification; neither had significant symptoms or sequelae. One referral for emergency medical evaluation was made for a 15-kg toddler who may have ingested up to 436mg/kg of methyl salicylate; however, the parent chose instead to observe the child at home and reported no adverse effects. Conclusion: Toothbrushing with non-toothpaste products did not result in significant recognized toxicity. However, the potential for serious toxicity exists when non-trivial amounts of methyl salicylate-containing creams are ingested when mistaken for toothpaste.

31 CLINICAL EFFECTS OF ACUTE LORATADINE EXPOSURE: A REVIEW OF 71 CASES.

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Background: Antihistamines have been associated with significant side effects in overexposure including torsades de pointes. Data regarding acute loratadine toxicity is limited. This study sought to determine the clinical effects of loratadine exposure. Methods: We reviewed all acute single- and multiple-agent exposures involving loratadine that were reported to a regional poison control center between 1/1/93 and 6/30/97. Patient age, the amount of loratadine ingested, symptomatology, disposition and outcome were documented. Results: Of 54 single ingestions, 45 (83%) were asymptomatic (avg dose: 33.6mg; range: 10-180mg). Nine patients had manifestations including syncope, lethargy, drowsiness, urticaria, vomiting, nausea and headache (avg dose: 32.5mg; range 10-70mg). One patient had right bundle branch block and was discharged after six hours of observation. It is unknown if this was a new finding. Of these nine, five patients remained at home, three patients were treated with activated charcoal (AC) and released

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and one patient was admitted to a critical care unit. All patients had minor outcomes. Of 17 multiple drug exposures, 11 (65%) were asymptomatic (avg dose of loratadine: 51mg; range: 10-300mg). Six patients (35%) had manifestations including confusion, mydriasis, vomiting, lethargy, and headache (avg dose of loratadine: 220mg; range: 40-400mg). One patient had sinus tachycardia (rate 110) and was admitted to a critical care unit. This patient was released after 24 hours of observation. One patient remained at home, one patient was treated with AC and released, and three patients were admitted to an unmonitored floor. All patients had minor outcomes. Conclusion: In our study, loratadine did not exhibit serious clinical toxicity in overdose. We recommend that individuals who ingest less than 35mg of loratadine and are not exposed to any other substances may be observed at home.

32 NAIL PRIMER COSMETICS: CORRELATIONS BETWEEN CORROSIVITY AND ADEQUACY OF PRODUCT LABELLING.

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Background: We previously reported on the corrosive hazard to young children of methacrylic acid-containing nail primers. However some newer primers contain alternative ingredients, which may not pose the same risk. Objective: To investigate the relationship between corrosivity of different primer products, product contents, and appropriateness of product labeling and packaging. Methods: 23 commercially available primers were grouped by product contents: (methacrylic acid vs others). Product pH was measured using an Orion Model 520A electrode. Product labels were scored on 7 warning points: 'poison and/or corrosive', a 'caution to avoid contact and/or to use a barrier when handling the product', a 'skin first aid', an 'eye first aid', an 'ingestion first aid', a caution to 'keep away from children' and a 'in an emergency, contact poison center'. A summative 'global hazard notification score' (GHNS) was calculated for each product. Data were analyzed using correlations and the two-sample t-test. Results: None of 23 products tested were contained in a child-resistant container (CRC) and none included all 7 label items. Product pH ranged 1.90-8.55 (median pH 4.58; 20 products had $pH<7.0$). Only 1 product advised, in the event of a poisoning, that a poison center be contacted. Of 20 acidic products, only 7 alerted users that the contents were corrosive. The mean GHNS (MAX=7) was 3.6; GHNS did not correlate with pH. Methacrylic acid-containing products had a lower pH (mean 3.43) than those without methacrylic acid (mean 5.34), $p=0.008$. When the primer bottle was separated from the rest of the packaging, 50% of products lost all their warning information. Conclusions: Not all artificial nail primers used in the home are highly corrosive, although most are acidic. Labeling is generally inadequate. We recommend that cosmetics manufacturers repackage methacrylic acid-containing products in CRCs and alert consumers to their hazard with better labelling. Manufacturers should also investigate the feasibility of either substituting other chemicals or lowering the concentration of methacrylic acid.

33 LIMITED HEPATOTOXICITY FOLLOWING A MASSIVE CHLOROFORM INGESTION TREATED WITH ORAL *N*-ACETYLCYSTEINE.

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Background: Although chloroform has been used for decades as an anesthetic and solvent, there are few published cases of oral exposure. Ingestions exceeding 120 mL have been uniformly fatal with death occurring either immediately as a result of respiratory depression or cardiac dysrhythmias or 4 to 5 days later from fulminant hepatic failure. Animal models suggest that glutathione depletion plays a role in this hepatotoxicity and *N*-acetylcysteine has been proposed as a possible preventive therapy. We report a patient who ingested 230 mL of chloroform, was treated with oral *N*-acetylcysteine, and developed limited hepatotoxicity. Case Report: A 38-year-old depressed male ingested chloroform, rum, and tetrahydrozoline. Upon presentation, the patient developed stupor and hypoventilation necessitating intubation. Treatment included gastric aspiration and activated charcoal administration via nasogastric tube, atropine, lidocaine (prophylaxis against ventricular dysrhythmias), and oral *N*-acetylcysteine. Over the next 12 hours episodes of hypotension were successfully treated with saline boluses and Trendelenburg positioning. Catecholamine pressors were avoided due to chloroform's ability to sensitize the myocardium to catecholamines. The patient was extubated on day 2 without sequela. Gas chromatography-mass spectroscopy identified chloroform and tetrahydrozoline in his urine and his initial serum ethanol level was 21mg/dL. *N*-acetylcysteine dosing (70 mg/kg) was continued for 21 doses over 4 days. Hepatic transaminase levels were initially normal but began rising on day 2 and peaked with an AST 582U/L and an ALT 232U/L on day 4. By day 6 they had normalized and the patient was transferred to an inpatient psychiatric facility. Conclusions: Oral *N*-acetylcysteine appears to prevent significant hepatotoxicity after massive oral chloroform exposure. Further animal work should be done to confirm this but until more definitive data is available *N*-acetylcysteine should be recommended for any chloroform ingestion.

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34 NEPHROTOXICITY FOLLOWING DERMAL ABSORPTION OF BROMATE.

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Background: Bromate induced nephrotoxicity has followed ingestion. Such injury has not been reported following dermal absorption. Case Report: ProLine SuperCurly Neutralizer was applied twice to the hair of a healthy 9-year-old girl with normal intelligence. 30 minutes after application, which induced a severe burning sensation on the scalp, she vomited. 2 hours later, loose stools, along with the vomiting, led to hospitalization for acute gastroenteritis. BUN was 35mg/dL and creatinine (C_R) 1.9mg/dL on admission. The 4th day after exposure, BUN/C_R were 73mg/dL/7.0mg/dL. She was transferred to a tertiary center where the association with bromate was entertained. The girl denied any ingestion of the product. Her mother, present during the application, reported no incident where the products would have entered her mouth. The second application was triggered by severe stinging during the first, yet, it also caused stinging. The dermal route of absorption was the only route identified. A serum bromide was 11mg/dL. Her scalp showed diffuse thin brown desquamation; however, there was no active dermatitis at that time. The hair was brittle. On day 5, peritoneal dialysis was started and continued 6 days. BUN/C_R peaked at 95mg/dL/11.7mg/dL on day 7. On day 5, AST was 47U/L; CPK 375U/L; and LDH 832U/L. Total parenteral nutrition was needed because of emesis and anorexia which waned by day 12. She was discharged on day 14 eating normally and on iron for a drop in hemoglobin from 12.5 to 8.5 g/dL. Audiometry on discharge revealed normal hearing. Results: No oral exposure was identified. The scalp was injured more than expected from routine use. This would occur if the concentration of the product were too high. Bromide in the serum showed absorption. Injury to the kidney from bromate is supported. The manufacturers, notified about the case, are reviewing their marketing of this product. Conclusions: Dermal absorption of bromate in toxic amounts can occur. The authors think bromate should be removed from the cosmetics market.

35 ACUTE ALLERGIC REACTION TO HAIR WAVING AGENTS.

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Background: Historically, cosmetic products when used according to labeling information present a limited health threat. Commonly reported toxicity is of a contact dermatosis. Most Hair Waving Agents are classified as caustic agents with a potential for serious chemical burns when misused. Acute systemic symptoms are unusual. We report two cases of an "*Immunologic Contact Urticaria Syndrome*". Case Series: 1) A previously healthy, 31 year-old female was evaluated 30min after a mixture of African Pride® Relaxer and Activator was applied to her scalp hair. She developed pruritus that started with scalp burning. She stated that her lips were swollen. Her vital signs were BP 139/80; HR 125 (narrow ST); RR 24; T 97.4; POx 95%^{RA}. Her airway, breathing, and circulatory assessment were normal. Facial flushing and urticaria was noted. Treatment consisted of decontamination, antihistamines and corticosteroid. 2) A 26 year-old female with history of asthma fainted shortly after applying AAA Bond® to her scalp hair. First, she experienced scalp burning and itching. Subsequently, she became nauseous, light-headed, diaphoretic, short-of-breath, and complained of substernal and stomach burning. She became unconscious and awoke without confusion. Her vital signs were BP 86/60; HR 124 (narrow ST); RR 24; POx 97%^{RA}. Her airway, and breathing assessment were normal. She had hyperemic conjunctivas. Significant lacrimation was noted. Urine drug screen was negative. Treatment consisted of decontamination, a fluid challenge, antihistamines, and corticosteroid. Conclusions: Due to allergen induced sensitization, most contact cutaneous disorder that begin as an irritant dermatitis may progress to a localized Allergic or Atopic Dermatitis when reapplied. Because of release of vasoactive compounds from the immune cells, severe reactions can result in airway obstruction, hypotension, and cardiorespiratory arrest. The Food, Drug, and Cosmetics Act should be enforced. The Fair Packing and Labeling Act should be modified to state ALL ingredients of a product are required to be label specified. By doing so, the consumer could have avoided exposure to previously identified allergens, thus minimizing a potentially life threatening event.

36 THE UTILITY OF ACETAMINOPHEN SCREENING IN UNSUSPECTED SUICIDE INGESTIONS.

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Background: Acetaminophen (APAP) is one of the most common medications taken in overdoses. Because of the lack of specific symptoms associated with initial APAP toxicity and the availability of a specific antidote. Potential unsuspected APAP toxicity is a significant concern in all suicide ingestions. Objective: To identify the likelihood of unsuspected APAP toxicity in suicide ingestions. Methods: We performed a retrospective study of all suicide ingestions without a history of APAP exposure reported to a poison control center over a 6 month period. History, symptoms, and APAP levels were reviewed to assess the incidence of unsuspected APAP ingestions. All ingestions with a history of potential APAP involvement were excluded. Results: A total of 471 cases were identified, 151 cases

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had APAP levels requested but the results were not documented. Of the remaining 320 cases, 23 (7.1%) were found to have APAP levels > 10 ug/mL. Twelve patients were treated with *N*-acetylcysteine (NAC) in the hospital, 7 patients (2.2%) had potentially toxic levels based on the Rumack-Matthew nomogram. The other 5 patients had non-toxic or un-interpretable levels. Even if we assume the 151 cases of undocumented levels were all non-toxic, the overall incidence of APAP toxicity is 1.5%. Conclusion: The risk of unsuspected acetaminophen toxicity is small but definite. Continued universal APAP screening in all suicide ingestions may be warranted. The use of NAC therapy in the hospitals often deviated from standard recommendations.

37 OBSERVATIONAL STUDY OF ACETAMINOPHEN OVERDOSE.

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Objective: To evaluate whether elevated AST, early in the clinical course of patients following acute acetaminophen (APAP) overdose might predict which patients might not need prolonged therapy with oral *N*-acetylcysteine (NAC). Background: Prior to the availability of an antidote, as many as 60% of patients, following an acute overdose, developed liver damage defined historically as aspartate aminotransferase (AST) greater than 1000 IU/L. Studies have documented the efficacy of a 72 hour course of oral NAC following acute ingestion of APAP. Those that do not have evidence of injury by 36 hours may do as well with a shorter course of NAC. Methods: This was a prospective observational study evaluating patients with toxic APAP blood levels, according to the Rumack-Matthew nomogram, following acute overdose. Five regional poison centers participated. Therapy with NAC was recommended for patients with continued elevation of AST four times the upper limit of normal past 36 hours. Otherwise, duration of therapy was determined by the treating physician. Results: Data was collected on 338 patients. 60 cases were excluded due to unknown time of ingestion, non-toxic APAP level, or insufficient AST measurements. There were 193 cases with AST under 4 times the upper limit of normal at 36 hours. 62 of these received no further NAC; None of these developed AST > 1000. 131 received further treatment, 2 (1.5%) developed AST > 1000. None developed liver failure. Conclusions: We found that following acute ingestion of toxic amounts of APAP, patients treated with NAC, rarely develop liver injury, as defined historically by AST > 1000, if the AST remained less than 4 times the upper limit of normal up to 36 hours post ingestion. These patients did well regardless of continued therapy with NAC.

38 DISPOSITION OF ACETAMINOPHEN OVERDOSE PATIENTS BY EMERGENCY PHYSICIANS.

Harris C, Engebretsen K. *Emergency Medicine Department, Regions Hospital, St. Paul, MN; International Poison Center, Bloomington, MN*

Objective: There are wide variations in the Emergency Department (ED) disposition of patients with acetaminophen (APAP) overdose, from ICU admission to treating the patient at home with the antidote, *N*-acetylcysteine (NAC). It is widely known that the early treatment with the antidote is very effective, and mortality from APAP overdose is low. The objective of this study was to evaluate the most common level of care involved in the hospital management of significant APAP overdose (requiring NAC therapy). Methods: This was a descriptive study design using a self-reporting survey instrument. A random sample of 1000 Emergency physicians (EPs) was obtained from the active member list of the American College of Emergency Physicians. A pilot survey was mailed to 50 randomly selected EPs from this list. After these were returned survey questions were reformatted or revised for clarification before mailing to the remaining 950 EPs. The survey requested information regarding hospital size, practice experience, and the typical disposition of acute APAP overdoses. A follow-up post card was sent to non-responders. Data from completed surveys was entered into a computer database for descriptive statistical analysis. Results: Of the 1000 surveys mailed, 591 were returned for a response rate of 58%. The completion rate was 57% (565). Over 2/3 (67.6%) of the responders of the total responders admitted their patients to the ICU. Only 19.4% admitted patients to the Step-down unit, and 12.6% to a ward bed. Nearly ninety percent (88.8%) are able to administer NAC in the ED. Conclusions: Two-thirds of EPs admit patients with significant APAP overdose to critical care bed in spite of the availability of an effective antidote and low mortality. Are critical care beds needed for APAP overdoses requiring NAC therapy? Further study is required to determine a safe and cost effective admit level.

39 ACETAMINOPHEN HEPATOTOXICITY AFTER CHRONIC RECTAL DOSING.

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Background: Recent pharmacokinetic studies in children administered a single dose of acetaminophen per rectum have suggested that up to 40 mg/kg/dose is required to achieve adequate serum concentration (Anesthesiology 1997;87:244). Some health care workers have extrapolated from these single-dose studies to recommend higher doses with chronic rectal administration. We report a case of a child we believe developed acetaminophen hepatotoxicity after his parents were instructed to administer per rectum 15-20 mg/kg every 3-4 hours. Case Report: An 8-year-old boy with congenital muscular dystrophy underwent a bilateral orchidopexy under regional anaesthesia. Following surgery and continuing after discharge, he was administered acetaminophen suppositories (17 mg/kg) every 3 hours for 60 hours (115 mg/kg/d). He was well for 24 hours, then developed anorexia, lethargy and vomiting. His parents sought medical care when they found him responsive only to pain. He was afebrile and his serum glucose was 1.0 mM. With glucose and fluid resuscitation he became lucid, but remained lethargic. He was noted to have tender hepatomegaly and ALT of 7777 IU/L, AST 7450 IU/L, PT 26.8 sec (INR 2.71), albumin 28 g/L, with normal NH_4 and bilirubin. He was treated with intravenous *N*-acetylcysteine for 60 hours with rapid improvement in liver function, appetite and mental status. On follow-up, 4 weeks later, ALT was 80 IU/L and all other liver function tests were normal. The clinical course and very high AST are most consistent with acetaminophen hepatotoxicity. Investigations were negative for an alternative toxic, viral or metabolic etiology for his liver dysfunction. Conclusion: This case suggests that the therapeutic index for chronic rectal dosing of acetaminophen is potentially as low as with oral administration. Health care workers should not extrapolate from pharmacokinetic studies of single-dose rectal administration of acetaminophen to make recommendations regarding chronic dosing per rectum that exceed 15 mg/kg/dose every 4 hours.

40 INHERITED METABOLIC DISEASE MASQUERADING AS CHRONIC ACETAMINOPHEN TOXICITY.

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Background: There have been recent concerns about chronic acetaminophen toxicity in pedatrics. Acetaminophen-induced hepatotoxicity should be considered early in the differential diagnosis of fulminant hepatic failure (FHF) so that antidotal treatment can be initiated. A case of FHF is presented in which initial investigations supported a possible diagnosis of acetaminophen toxicity, but the eventual etiology was a rare inherited metabolic disease. Case Report: A previously well 11-month-old male presented unresponsive with profound hypoglycemia after a 3-day illness of fever, vomiting, stomatitis and decreased intake. Recent medications included acetaminophen as suspension, suppositories, and as part of a multi-symptom cold remedy. In the emergency department, he was treated with IV dextrose with an excellent response, evaluated for presumed sepsis and started on antibiotics. He was maintained on dextrose containing IV fluid and on the morning after admission, he became unresponsive, requiring intubation. Significant laboratory studies at that time revealed a normal blood glucose, AST, ALT > 10,000U/L, LDH-6180U/L, NH_3-190mcmol N/L/, PT > 50sec, PTT-55sec, bilirubin-9.2/6.1mg/dL. He fulfilled the criteria for stage IV hepatic encephalopathy and was treated with aggressive supportive therapy including a double volume exchange transfusion. He was placed at the top of the liver transplant list. Multiple studies looking for infectious, toxic, and metabolic causes were performed. An acetaminophen level was 10mg/L. The last known dose of acetaminophen was prior to admission, more than 24 hours previously. Based on the exposure history and this clinical presentation, *N*-acetylcysteine therapy was initiated and continued for a full 72-hour course. He progressed to a full recovery and was discharged 10 days after admission. After discharge, metabolic studies revealed the etiology to be Long-Chain 3-Hydroxyacyl-CoA Dehydrogenase (LCHAD) Deficiency. Conclusion: LCHAD deficiency is an extremely rare disorder of fatty acid oxidation that presents as episodes of acute illness triggered by prolonged fasting. In the future, the use of specific markers for acetaminophen-induced hepatotoxicity may help to separate acetaminophen-induced hepatic failure from other causes of FHF.

41 DO WE ADMINISTER *N*-ACETYLCYSTEINE (NAC) ON TIME? ADMISSION TO INPATIENT UNITS IS ASSOCIATED WITH DELAY IN NAC ADMINISTRATION.

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Background: The objective of this study was to determine whether admission to the hospital floor after receiving a 1st dose NAC in the ED was associated with delayed administration of subsequent doses. Methods: We reviewed hospital records for all APAP overdoses presenting to a university hospital over 2 years who had a toxic APAP level and were admitted for NAC therapy. Information obtained included: time of ingestion, time of first three NAC doses,

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location of NAC administration, and peak AST level during hospitalization. Results: Ninety-eight cases were reviewed. Sixteen were excluded because of incomplete data. Forty-three percent (35/82) of patients received their first NAC dose within 8 hours of APAP ingestion. Of the 82 cases that were analyzed, mean time between first and second NAC dose was 4.7 hours (SD 1.9) with median interval of 4 hours and range of 45 minutes to 12 hours. Time interval between doses was ≥ 6 h in 23% (19/82) of cases. 41% (34/82) received the 2nd NAC dose in the ED. Fifty-nine percent (48/82) received the 2nd NAC dose on the floor. Mean time between 1st and 2nd NAC was 5.1 h (SD 2.2) for patients transferred to the floor and 4.1 h (SD 1.0) for ED patients. In 2 patients who were transferred to a second hospital after the 1st NAC dose, time between 1st NAC dose and 2nd NAC dose was 8.5 and 12 hours. Of the 35 patients who received their first dose of NAC within 8 hours of ingestion, mean AST was 51 IU/L (SD 71) for those who received their 2nd NAC on the floor, and 29 IU/L (SD 12) for those who received their 2nd NAC in the ED. Conclusions: Timely administration of the second NAC dose may be delayed upon patient transfer from the ED to the floor. Delay is particularly problematic with inter-hospital transfers.

42 IMMEDIATE HYPERSENSITIVITY RELATED TO ORAL *N*-ACETYLCYSTEINE ADMINISTRATION FOR ACETAMINOPHEN TOXICITY.

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Background: The administration of *N*-acetylcysteine (NAC) is the treatment of choice for acetaminophen (APAP) poisoning. Allergic reactions have been associated with IV administration of NAC. However, immediate IgE mediated hypersensitivity reactions have not been well substantiated with orally administered NAC. We report such a case in the setting of acute APAP toxicity. Case Report: A 15-year-old female ingested 10 g APAP 6 hours prior to ED presentation. Her 6 hour post ingestion APAP level was 562 mcg/mL. Her first dose of NAC was administered 8 hours after ingestion followed by additional doses every 4 hours. Following the 6th NAC dose, she developed an urticarial rash over both forearms associated with pruritis. There were no associated respiratory or vital sign abnormalities at this time. She received diphenhydramine (DPH) with resolution of her rash. Following subsequent NAC doses she had recurrent urticaria and pruritis with the development of swelling, erythema and tenderness of the plantar aspect of her right foot. She was managed successfully with DPH, methylprednisolone and discontinuation of the NAC after the 11th dose. Serum IgE after the last NAC dose was elevated at 624 u/mL. A CBC at that time was normal with no eosinophilia. Conclusion: A case of IgE mediated immediate hypersensitivity is presented associated with the oral administration of *N*-acetylcysteine for acetaminophen toxicity.

43 DOES ORAL *N*-ACETYLCYSTEINE REDUCE THE ADSORPTION OF ACETAMINOPHEN BY ACTIVATED CHARCOAL?

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Background: Whether concomitant oral administration of *N*-acetylcysteine and activated charcoal reduces the bioavailability of *N*-acetylcysteine has been studied many times. This study tests the hypothesis that concomitant administration reduces the adsorptive capacity of charcoal for acetaminophen. Methods: This was an *in vitro* study. Reagents included were the pharmaceutical preparation of *N*-acetylcysteine, powdered acetaminophen and powdered activated charcoal. Acetaminophen analyses were by HPLC and all measurements were done in triplicate or greater. All experiments were performed at pH 1.8 and 7.4 and *N*-acetylcysteine was added after the incubation of acetaminophen and charcoal. Percent binding of acetaminophen was calculated after its incubation with charcoal both with and without the addition of *N*-acetylcysteine over a range of clinically relevant charcoal to acetaminophen ratios. To further test the hypothesis the latter experiment was repeated with increasing amounts of *N*-acetylcysteine to provide this antidote with a competitive advantage for charcoal binding sites. Data were compared using the unpaired two-sided student t-test. A difference was accepted when $p<0.05$. Results: Differences between control and experimental data were similar at both pH values. At pH 7.4 the addition of *N*-acetylcysteine altered the binding of acetaminophen by activated charcoal from 90.6% to 89.6% ($p=0.005$) and from 98.5% to 98.6% ($p=1.0$) at activated charcoal to acetaminophen ratios of 5:1 and 10:1 respectively. Although the first comparison is significant it is likely clinically unimportant as a 1% difference equals 100 mg of acetaminophen when 10.0 g are ingested. Increasing the amount of *N*-acetylcysteine decreased the amount of bound acetaminophen from 98.5% to 82%. Conclusions: The hypothesis is supported. Concomitant oral administration of activated charcoal and *N*-acetylcysteine reduces the adsorptive capacity of charcoal for acetaminophen. The extent is likely clinically unimportant for mild to moderate acetaminophen ingestions. However, it becomes important in situations of diminished charcoal to ingestant ratios (< 5.0:1) such as massive acetaminophen overdose or the presence of coingestants. Intravenous administration of *N*-acetylcysteine circumvents this interaction.

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44 SALICYLATE TOXICITY: CURRENT TRENDS IN MANAGEMENT.

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Objective: This study sought to evaluate current treatment practices and outcomes for salicylate ingestions. Methods: All exposures reported to a RPCC between 1/1/95 and 4/1/98 were evaluated. Products ingested, blood concentrations, management, and outcomes were analyzed on all hospital admissions. Elimination half-lives were calculated on patients with 2 or more levels drawn after 6 hours post ingestion. Results: 605 ingestions, 96 admitted to hospitals. 27 excluded because admitted for reasons unrelated to salicylates, leaving 69 admitted for salicylate toxicity. Aspirin alone =44, multiple ingestants =25; ICU admissions =48, floor =21. Accidental pediatric ingestions =1. Peak serum levels for admissions: <30mg/dl in 11, 30-49mg/dl in 28, 50-69mg/dl in 25, >69mg/dl in 5. There were 2 fatalities (peak serum levels 118mg/dl and 152mg/dl); all survivors appear to have recovered completely. Data from coroners' offices indicated there were 7 other pre-hospital deaths caused solely (3) or partly (4) by salicylates. Management provided for the 69 admissions: single dose activated charcoal =42, multiple dose (MDAC) =19, gastric lavage =28, alkalinization (NaHCO3) =50, whole bowel irrigation =1, hemodialysis =1. Pills recovered by lavage: 4/8 patients who presented within 1 hour post ingestion, 1/5 who presented within 1-2 hours, 1/5 within 2-3 hours (patient with massive ingestion who died), 0/6 >3 hours, 0/4 chronic/unknown. Only 21/50 patients alkalinized had 2 or more urine pH's checked, of these 10 were alkalinized successfully (pH 7.5-8). Mean elimination half-lives according to treatment received: NaHCO3 + MDAC (n=17) t1/2 =17.4 hrs, NaHCO3 without MDAC (n=29) t1/2 =17.6 hrs, MDAC without NaHCO3 (n=3) t1/2 =20.4 hrs, no NaHCO3 or MDAC (n=9) t1/2 =24.1 hrs. Conclusions: There was frequently a failure to achieve adequate urinary alkalinization in salicylate intoxications; despite this mortality and serious morbidity were low in patients who were hospitalized. Our data suggested limited value of lavage after 1 hour. We found a trend toward shorter elimination half-life in patients who received NaHCO3 and/or MDAC compared to those who received no treatment.

45 ALTERED MENTAL STATUS IN SALICYLATE POISONING: REVERSAL WITH IV GLUCOSE.

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Background: Studies in salicylate poisoned rodents found low brain glucose and glycogen, altered consciousness, seizures and death, despite normal blood glucose. These CNS abnormalities reverse with glucose treatment. Since salicylate uncouples oxidative phosphorylation, high energy phosphate in brain is maintained by inefficient glycolysis. Brain glucose therefore falls as utilization exceeds the rate of supply from blood. We report a case of reversal of altered consciousness with IV glucose (IVG). Case Report: A 48-year-old woman was brought to the ER with confusion and malaise. The patient exhibited disorganized speech, poor response to commands, no focal CNS signs, temperature 37.6°C, HR 112, RR 20, BP 100/65. Chart review showed past alcohol excess, SLE. She was given lorazepam 2 mg, $MgSO_4$, normal saline, thiamine with no response. 1.5 hours post admission, K^+ 3.8mmol/L, anion gap (AG) 22, glucose 5.4mmol/L, pH 7.44, pCO_2 22, pO_2 88, HCO_3 15. Salicylate level on original blood was 4.1mmol/L and ethanol undetectable. Renal function, liver enzymes, WBC, platelets, Ca^{++} and Mg^{++} were normal. Five hours post admission her CNS status had deteriorated with GCS of 11, and tremor. Vital signs and temperature remained unchanged. Nine hours post admission the poison centre medical consultant recommended repeat glucose, K^+, and salicylate levels, infuse bicarb and potassium, and give a bolus of 50 mL of D50W. During the glucose infusion, GCS quickly became 15 and she conversed normally. She provided a history of joint pains, and having taken 10 or more acetylsalicylic acid(325 mg) daily for one week. Salicylate level dropped to 2.7mmol/L, K^+ 3.7mmol/L, AG 12. D10W infusion was continued for 12 hours and mental status remained normal until discharge. Salicylate level 22 hours post admission was 1.2mmol/L, K^+ 3.4mmol/L and glucose 5mmol/L. Conclusion: Altered mental status with salicylism, and its response to IVG suggests that this is due to low brain glucose. A controlled trial is being initiated to evaluate this.

46 INTRA-ALVEOLAR HEMORRHAGE IN FATAL SALICYLATE TOXICITY.

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Background: Severe salicylate poisoning is complicated by multiple pathophysiologic changes, including inhibition of vitamin K epoxide reductase, hypoprothrombinemia and inhibition of platelet aggregation. Nevertheless, overt hemorrhage is rarely seen. We describe two cases of a previously unreported complication of severe salicylate poisoning. Case Reports: A 41-year-old man presented 4 hours after ingestion of 65 g (761 mg/kg) of aspirin with tinnitus, nausea, vomiting, compensated metabolic acidosis and a serum salicylate concentration of 52.1 mg/dL. Initial

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treatment included potassium supplementation, multiple-dose activated charcoal and urinary alkalinization. Attempts to administer charcoal were unsuccessful due to refractory emesis and inability to place a nasogastric tube. By 11 hours after admission, the salicylate concentration had risen to 96 mg/dL. Intubation was required for mental status changes and hypoxia. Dialysis was arranged. Despite attempts at high minute ventilation, his pCO_2 rose to 77 mmHg with markedly increased airway pressures and expectoration of blood. Shortly afterwards, he had a sudden asystolic arrest with unsuccessful resuscitation. At autopsy, an esophageal stricture immediately above the cardiac sphincter was found, totally occluded by a 5 cm inspissated mass of aspirin and charcoal, together with extensive bilateral intra-alveolar hemorrhage, confirmed by light microscopy. Another 41-year-old man presented 6 hours after ingestion of 65 g (784 mg/kg) of aspirin. He was obtunded, tachypneic and diaphoretic, with a partially compensated metabolic acidosis and a serum salicylate concentration of 122 mg/dL. During transfer for emergent dialysis, he became hyperthermic (108°F) with extreme tetany, then asystolic. Resuscitative measures including cricothyrotomy were unsuccessful (despite succinylcholine, tetany had prevented oral intubation). Autopsy findings were remarkable for diffuse intra-alveolar hemorrhage, also confirmed by light microscopy. In neither case was intra-alveolar hemorrhage attributable to cardiopulmonary resuscitation. Conclusion: These are the first reported cases of intra-alveolar hemorrhage in salicylate poisoning. Extensive intra-alveolar hemorrhage will impair respiratory compensation for salicylate-induced metabolic acidosis. Intra-alveolar hemorrhage in this setting may be an idiosyncratic reaction or a complication of known pathophysiologic changes.

47 URINARY ALKALINIZATION COMPARED TO MULTIDOSE ACTIVATED CHARCOAL FOR THE ENHANCEMENT OF SALICYLATE ELIMINATION.

Ruskosky D, Schauben J, Kunisaki T. *Florida Poison Information Center/Jax, UFHSC, Jacksonville, FL*

Background: To date, no human studies have compared the efficacy of the two common modalities to enhance the elimination of salicylate. The purpose of this study was to compare the efficacy of urinary alkalinization and multidose activated charcoal in enhancing the elimination of aspirin. Methods: After informed consent was obtained, 10 healthy volunteers were asked to participate in each of the three phases of the study. In phase 1 (control phase) 650mg of aspirin was administered every 4 hours for 2 days. Serum salicylate levels were drawn at 0, 1, 2, 4, 6, 8, 10, 12, 18, and 24 hours after the aspirin regimen. In phase 2, using the same protocol for aspirin administration and laboratory assessments, the volunteers ingested 50 g activated charcoal (AC) with 1g/kg of sorbitol after the aspirin administration was complete. This was followed by 25g of AC every 4 hours for 24 hours and 1g/kg of sorbitol every 12 hours for 24 hours. Phase 3 included the same aspirin regimen, followed by a loading dose (1mEq/kg) and infusion (100mEq/L) of sodium bicarbonate titrated to a urinary pH of 7.5-8. The results were analyzed using Wilcoxon ranked sum tests. Results: 9 patients completed all 3 phases. Urinary alkalinization shortened the half-life of the salicylate by 48.4% (4.741hrs) compared to control ($p=0.008$) and 42.7% (3.767hrs) compared to the AC phase ($p=0.011$). AC did not statistically decrease the half-life when compared to control ($p=0.441$). The area under the curve was statistically less for the urinary alkalinization group compared to the control ($p=0.008$) and the activated charcoal group ($p=0.028$). Conclusion: Based on these results, urinary alkalinization is an effective modality to enhance the elimination of salicylate. Multidose AC does not appear to enhance salicylate elimination beyond normal clearance values.

48 FALSELY ELEVATED SALICYLATE LEVELS IN AN OVERDOSE OF DIFLUNISAL.

Leff K, Delgado P, Marcus S, Shih RD, Szucs P. *Department of Emergency Medicine, Morristown Memorial Hospital and The New Jersey Poison Center, Newark Beth Israel Medical Center, Newark, NJ*

Background: Diflunisal (Dolobid®) is an atypical NSAID. It has structural similarities to salicylate. Standard tests for salicylate have been reported to be falsely elevated by diflunisal. We report a case of a "highly toxic" level of salicylate in an otherwise clinically stable patient that had ingested diflunisal and famotidine. Case Report: A 24-year-old male self-reported ingesting 7 tablets of 500mg diflunisal and 100 tablets of 20mg famotidine. He denied salicylate ingestion or ingestion of other toxins. He was alert and oriented and denied tinnutis or GI symptoms. His initial laboratory tests revealed normal electrolytes with no anion gap, normal arterial blood gas analysis, and a normal urinalysis without ketosis. His salicylate level was 133.9mg/dL at 6 hrs post-ingestion, 72mg/dL at 11 hrs, and 54mg/dL at 15 hrs. Serum tox screening was otherwise negative. The patient remained clinically nontoxic including repeat electrolytes throughout his hospital stay. Conclusion: We present a case of a patient with a very high salicylate level and lack of clinical salicylate toxicity. This salicylate level typically prompts consideration for aggressive management including hemodialysis. Consideration of this cross-reactivity needs to occur in the setting of diflunisal ingestions.

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49 INFLUENCE OF NON-STEROIDAL ANTI-INFLAMMATORY DRUGS ON THE MATERNAL LIVER DURING PREGNANCY.

Burdan F, Burski K, Siezieniewska Z, Maciejewski R, Urbanowicz Z. *Department of Human Anatomy, Department of Pathology; Medical University School of Lublin, Poland*

Background: Due to its increased susceptibility to toxic damages, the maternal liver is a good experimental model for monitoring side effects in various pharmaceutical studies. This research evaluates the combined hepatic effects of paracetamol (P), propyphenazone (R), and caffeine (C). Methods: Pregnant rats were administered intragastrically a mixture of P+R+C in Tween 80 solution once a day, during the whole second trimester (A, B, D groups) and on 10th day of pregnancy (groups A0, B0, D10). Drugs were administered in the constant proportions 5:3:1 in 3 different doses. Dose A, A0 - 3.5 mg/kg (P), 2.1 mg/kg (R), 0.7 mg/kg (C). Dose B, B0 was 10 times higher, and D, D0 100 times higher than dose A, A0. There were two Tween control groups. On 21st day of pregnancy female rats were sacrificed. Histological preparations of livers were made in using stains: Hematoxylin & Eosin, Periodic Acid-Schiff, Van Giesson, and Silver-Gomorii. Slides were verified under the light microscope. Maternal body and liver weight was also checked. Results: Drug administration resulted in reactive and minimal degenerative changes in D0, B and D groups. There was a statistically significant ($p < 0.05$) drop in the body weight in the second trimester of pregnancy in the D group, as well as in D0, B and D groups in the third semester. Conclusions: The simultaneous administration of paracetamol, propyphenazone and caffeine failed to produce significant histological evidence of hepatotoxicity. Reactive and minimally degenerative liver changes were only present at D0, B and D groups.

50 BROMFENAC-INDUCED CENTRI-LOBULAR NECROSIS.

Cortes-Belen E, Benitez JG, Verrico P. *Toxicology Treatment Program, Drug Information Center, University of Pittsburgh, Pittsburgh, PA*

Background: Bromfenac is a new non-steroidal anti-inflammatory agent which has been recently deemed as potentially hepatotoxic whenever it is used beyond the recommended course of ten days. We present two cases where hepatotoxicity in the form of centri-lobular necrosis was found in liver biopsy after inappropriate use of bromfenac. Case Reports: #1 44 year-old male (without history of liver disease), presented with jaundice, flu-like symptoms, nausea, vomiting, RUQ pain, dark yellow urine and steatorrhea after approximately 15 weeks of taking two bromfenac 25 mg capsules two to three times weekly for severe migraine headaches. Liver function tests (BILI 26.7mg/dl, ALT 1792 IU/L, AST 2127 IU/L, GGTP 206 IU/L, and alkaline phosphatase 235 IU/L) and sonogram suggested acute hepatitis but hepatitis screens failed to demonstrate an infectious etiology. A liver biopsy revealed acute hepatitis with evidence of centri-lobular hemorrhagic necrosis without evidence of cirrhosis. Signs of hepatic encephalopathy or coagulopathy did not develop. Transaminase levels improved after several days and the patient was released with gastroenterology follow-up. #2 55-year-old woman, was transferred to our institution after post-operative evidence of liver failure after back surgery. The patient had been given a prescription for 360 capsules of bromfenac 25-mg of which she had consumed about a hundred in the 2 weeks prior to surgery. During that period she also used acetaminophen and acetaminophen/oxycodone occasionally. Liver fuction tests from a month prior to surgery were normal. No liver profile was available just prior to surgery. Patient suffered hypotension (60/40) secondary to a retroperitoneal hematoma post-surgery and liver function tests were elevated (AST 10308 IU/L, ALT 877 IU/L), and PT was 17.3. Liver biopsy revealed a centri-lobular necrosis. The patient developed hepatic encephalopathy, multiple organ failure, and expired. Conclusion: Bromfenac is indicated for short-term analgesia only. As our cases testify, severe drug-induced hepatotoxicity can occur if the drug is used beyond the recommended schedule. Additional risk factors (hypotension and other hepatotoxins such as anesthetics or acetaminophen) may potentiate the hepatotoxic effect of bromfenac. Centri-lobular necrosis occurred in both these patients.

51 COMPLICATIONS FOLLOWING ACUTE ACETIC ACID POISONING.

Borisovsky V, Birtanov E. *Toxicology Treatment Department (TTD), Toxicology Information Center, City Emergency Medicine Hospital (CEMH), Almaty, Kazakstan*

Background: Acetic acid poisoning is one of the most common and severe types of acute human poisonings in Kazakstan. Injuries following an acute ingestion of the concentrated acetic acid include corrosive damage of the gastrointestinal and respiratory tract and hemolysis resulting in further severe complications. Methods: During 1997, 187 patients were hospitalized at the Toxicology Treatment Department of City Emergency Medicine Hospital and 28 of them died (15%). We studied the complications following acute acetic acid ingestion in this group including clinical syndromes and laboratory findings. Results: The following complications were most commonly found in this group of patients (n=187): chemical burns of gastrointestinal tract (89.3%), hemolysis (65.2%), chemical pneumonitis

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(19.2%), gastrointestinal bleeding (11.2%), toxic shock (7.5%) and acute renal failure (3.7%). Among fatalities (n=28) the most common complications were: chemical burns of gastrointestinal tract (100%), hemolysis (100%), chemical pneumonitis (57.1%), gastrointestinal bleeding (42.9%), toxic shock (96.4%), acute renal failure (78.6%) and DIC (10.7%). Conclusion: Acute acetic acid poisoning causes serious complications resulting in high mortality rate and delayed recovery. Thus, it is extremely important to develop a specialized poisoning prevention programs aimed at the reduction of this type of exposure. One of the important ways to reduce the risk of human exposures to a strong concentrated acetic acid should be a contribution of a regional poison control centers in promotion of safe packing, storage and use of corrosive products.

52 PROFOUND IONIZED HYPOCALCEMIA WITH ARRHYTHMIA AND COAGULOPATHY IN A FATAL HYDROFLUORIC ACID EXPOSURE.

Hung OL, Flomenbaum M, Flomenbaum NE, DiMartino RN, Goldfrank LR, Hoffman RS. *New York City Poison Center, Cornell Medical Center, OCME, New York, NY*

Background: Although hydrofluoric acid (HF) is recognized as a caustic agent capable of causing significant tissue destruction, its potentially lethal systemic effects are often unrecognized. While HF exposures involving 2.5% body surface area burns may be fatal, actual fatalities following cutaneous exposure, ingestions, and inhalation to HF are rarely reported. We report a fatal HF exposure with associated postmortem coagulopathy and provide detailed electrocardiographic, photographic and pathologic findings, including the first reported comparison of post-mortem blood and vitreous ion levels. This case also highlights the hazards of pulmonary toxicity following dermal exposure to concentrated HF. Case Report: A 51-year-old male sanitation worker presented to the ED after a garbage canister containing 80% HF ruptured, spraying him on his face and torso. He developed immediate dyspnea and chest pain. In the ED, he was noted to have respiratory distress and second degree burns to his face and neck (5% total body surface area). An initial ECG revealed normal sinus rhythm and a QTc interval of 491msec with peaked T waves. He received 2g IV calcium gluconate. Despite this therapy, his QRS complex continued to widen and shortly thereafter he became asystolic. Serial ionized calcium levels were: 0.57, 1.84, 0.63, 0.74mmol/L despite 4g IV calcium gluconate, 10g IV calcium chloride, and 3g IV magnesium sulfate. His serum potassium was 5.8mEq/L. He expired 4 hours after presentation. Autopsy revealed coagulopathic manifestations of hypocalcemia including diffuse multiorgan hemorrhages and severe laryngeal edema. The concentrations of ions detected in the blood and vitreous humor were: fluoride, 0.98mEq/L and 0.63mEq/L; magnesium, 2.3mmol/L and 9.46mmol/L; potassium, 43.3mEq/L and 10.3mEq/L; and calcium, 2.74mmol/L and 16.2mmol/L, respectively. Conclusion: Limited exposure to concentrated hydrofluoric acid may be potentially lethal. Hydrofluoric acid-induced severe hypocalcemia is refractory to large quantities of intravenous calcium. Aggressive decontamination and therapy is indicated for patients with exposure to concentrated HF. Appropriate IV calcium dosing should be based on measured ionized calcium response.

53 FACTORS INFLUENCING MORBIDITY AND MORTALITY ASSOCIATED WITH LOW CONCENTRATION HYDROFLUORIC ACID.

Kao WF, Dart R, Kuffner E, Bogdan G. *Dept of Emergency Medicine, Veterans General Hospital-Taipei, Taiwan, Medical School, Yang-Ming University, Taiwan, ROC; Rocky Mountain Poison and Drug Center, Denver, CO*

Background: Exposure to hydrofluoric acid (HF) may result in severe toxicity or death in a minority of cases. AAPCC data suggests that cases involving ingestions of HF are at a disproportionate risk of death when compared to other routes of exposure. There are no currently reviews in the literature that focus on HF ingestion. The purpose of this study was to assess the morbidity and mortality associated with ingestion of low concentration hydrofluoric acid. Methods: Retrospective analysis of data collected by trained personnel using a standardized data collection system. All charts involving HF exposures for a two-year period a large regional poison center were identified using a computerized search. Each chart was abstracted by trained and blinded personnel. Results: There were 1772 exposures to HF; 135 involved ingestion. Twenty eight animal exposures and eight cases of human HF ingestion which could not be located were excluded, leaving 99 records for analysis. All ingestions involved consumer products containing 6-8% HF. Symptoms were reported by 49 patients in the ingestion group; the most common effects were mild gastrointestinal effects. Two patients with minimal clinical effects suddenly decompensated and died; both involved ingestion of HF. All remaining patients recovered completely. Of 29 cases in which calcium levels were recorded, 4 cases of hypocalcemia occurred. All patients who developed major effects or died were adults who ingested more than 3 ounces of HF with suicidal intent. Death occurred precipitously in patients who had appeared well moments earlier. Conclusion: Death occurred in two patients; both of whom were adults who ingested more than 3 ounces with suicidal intent. Ingestion of a household product containing hydrofluoric acid is a potentially life-threatening condition that requires close monitoring and prompt therapy. There appears no convenient method for

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monitoring of toxicity. The abrupt deterioration and lack of warning signs indicates the need for better diagnostic methods.

54 TREATMENT OF OCULAR POTASSIUM PERMANGANATE EXPOSURE WITH 5% ASCORBIC ACID SOLUTION.

Sigg T, Leikin JB, Sigg K, Mullin T. *Illinois Poison Center/Toxicon, Chicago, IL*

Introduction: Potassium permanganate ($KMn0_4$) has many uses, including: bleaching various materials, dyeing wood, photography, purifying water, and as a reagent in analytical and synthetic organic chemistry. When splashed into the eye it can frequently result in ocular injury which is difficult to treat. We describe a novel approach to this problem. Case Report: A 45-year-old male splashed a concentrated solution of $KMn0_4$ into both eyes while cleaning a well water filter. In the Emergency Department (ED) each eye was irrigated with three liters of 0.9 NaCl which resulted in the conjunctival sacs achieving a pH of about 7.5. Both corneas were clear by fluorescence stain and slit lamp evaluation. However, the patient complained of foreign-body type irritation and had several hard eroded lesions which appeared as granular deposits on the conjunctival tissue. The ED physician had been moderately successful at physically removing the deposits but the process was arduous. The original recommendation was to irrigate the eyes for at least 15 minutes with 0.01-0.05 M disodium EDTA, which was unavailable, neither was a parenteral ascorbic acid solution. Then the recommendation was to compound a 5% solution of ascorbic acid by dissolving a 500 mg chewable tablet in 25mL of 0.9% NaCl and irrigate the eyes. The ascorbic acid solution was then dripped into the eyes off of a piece of gauze. This dissolved the granular deposits on contact. The solution caused no residual damage to the ocular tissues and the patient was released. Conclusion: The solution utilized in this patient was 5% or 0.114 Molar ascorbic acid and has a pH=2.56(pKa=4.17). This solution would not be expected to damage any ocular tissues on contact. In patients that present with significant ocular exposures to potassium permanganate with visible eroded lesions and/or granular deposits, the use of 5% ascorbic acid solution aids in the removal of the manganese oxide deposits. Parenteral ascorbic acid solutions are preferable (due to sterility concerns), and may be compounded extemporaneously.

55 ANEMIA SECONDARY TO GASTRIC ULCERATION FROM A SOLDERING FLUX INGESTION.

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

Background: Soldering fluxes often contain zinc chloride (10-30%), a corrosive. From 1/1/93-12/31/96, 1816 soldering flux exposures were reported in the AAPCC TESS database. One-third were treated at a HCF. Of 1089 cases with known outcomes, 15 reported major effects. One death was reported. Of the few cases in the medical literature, most involved pediatric patients who experienced spontaneous vomiting, progressive lethargy, and varying degrees of upper GI injury. Most sought immediate medical attention. We report a case of soldering flux ingestion in an adult male presenting 2 weeks post exposure. Case Report: A previously healthy 42y.o. male accidentally ingested soldering flux from a drink container at work. Initial oral irritation occurred, with vomiting lasting for several days. He sought hospital care for abdominal pain, hematemesis, and melena beginning 3 days prior. Vital signs were: BP101/55mmHg, P101beats/min, R18/min, T99.1°F. Labs included: WBC 14.0K/uL, Hct 18.1%, Hgb 6.7g/dL, platelets 196K/uL; amylase 98(37-110IU/L), calcium 7.7(8.4-10.2mg/dL). Nasogastric suction yielded coffee ground fluid. IV fluids, famotidine, omeprazole, and sucralfate were administered. Five units of packed RBCs were given. Endoscopy revealed a 20-25cm fundal gastric ulcer of the greater curvature surrounded by hypertrophic gastritis. The duodenum, antrum, esophagus, and pharynx were normal. No active bleeding was seen. Biopsies were negative for *helicobacter pylori* and no malignancy was seen. The patient was discharged on day 4, and did not return for medical follow-up. No further symptoms were reported on telephone follow-up with a family member. Conclusion: Ingestion of soldering flux may cause significant gastric burns with or without oral or esophageal injury, possibly delaying medical treatment. The majority of cases reported in the medical literature have sought immediate medical attention following initial symptoms, and recovered following treatment without significant sequelae. We report a case of soldering flux ingestion in an adult presenting 2 weeks post exposure with severe anemia secondary to persistent GI bleeding from gastric ulceration. Immediate medical evaluation of symptomatic soldering flux ingestions is recommended.

56 GASTRIC BURNS SECONDARY TO GRANULAR LAUNDRY DETERGENT.

Herrington LF, Lewis JD, Geller RJ. *Georgia Poison Center & Children's Gastroenterology Group, Atlanta, GA*

Background: Granular laundry detergents contain anionic/nonionic surfactants with alkaline builders (phosphates, carbonates, and silicates) often added to boost cleaning. From 1995-1997, 24,719 exposures to granular laundry

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detergents were reported to the AAPCC TESS data base; 21,418 (86.65%) involved children under 6 years. These products seldom produce significant symptoms; 14,070 (96.75%) of known outcomes reported minor or no effect. Case Report: On 2/12/98, this Regional Poison Center (RPC) was contacted about a 15 month old girl who had eaten some Surf® powder. She had vomited spontaneously. The mouth had been rinsed and p.o. fluids given. The child presented to her physician's office one hour post ingestion, listless and drooling. Exam revealed a previously healthy child with perioral edema, but no intraoral lesions and in no respiratory distress. The RPC recommended evaluation for esophageal injury. On admission, vital signs were normal. Stool was negative for occult blood; hemogram and chemistry profile were also normal. Gastroesophageal endoscopy revealed no hypopharyngeal or esophageal injury, but isolated superficial erosions with erythema and ulceration on the lesser curvature, posterio-lateral to the gastroesophageal junction. The child was discharged home the next day on cimetidine and sucralfate, and was asymptomatic on follow-up 2 months later. Discussion: From 1/1/95-12/31/97, 85 human exposures to Surf were reported to this RPC. Ages ranged from 9mos to 74yrs. Seventy of these cases involved ingestion or ingestion/eye/skin contact in children < 6 years. None of these were recognized to have clinically significant adverse effects. Surf granular laundry detergents are combinations of anionic/nonionic detergents with sodium silicate (1-20%) and/or sodium carbonate (1-50%) builders (product pH = 10.3-11 for 0.1-10% solutions). Sodium silicate and sodium carbonate can be corrosive at high concentrations. Conclusion: Alkali corrosives can cause oral burns, with or without esophageal injury. Gastric burns are uncommon. We report a case of granular laundry detergent ingestion with gastric burns in the absence of hypopharyngeal or esophageal injury.

57 LACK OF HYPERKALEMIA WITH WAX-MATRIX POTASSIUM CHLORIDE STOMACH CONCRETION CAUSING GASTRIC OUTLET OBSTRUCTION.

Tunget-Johnson CL. *San Diego Division, California Poison Control System. San Diego, CA*

Background: Three recent case reports involving sustained release nifedipine highlight the potential for chronic therapeutic administration of sustained-release medications to cause stomach concretions and gastric outlet obstruction. This is the first report of stomach concretion and gastric outlet obstruction associated with therapeutic administration of potassium chloride wax-matrix tablets. Case Report: A 59-year-old white female underwent a Whipple procedure for a pancreatic neoplasm. Oral potassium chloride 40 mEq bid was started 10 days later using 10 mEq wax-matrix tablets. The dose was increased to 60mEq am and 40 mEq pm 2 days later. Seventy-two tablet were given over 8 days. Other oral medications included ranitidine 50mg bid, magnesium oxide 400mg qd, methyldopa 250mg tid, atenolol 50mg qd and enalapril 5mg qid. All of these medications were compressed tablets. Stenosis of the jejunal segment distal to the hepaticojejunostomy but proximal to the gastrojejunostomy was suspected on post-op day 14. Possible tablets were interpreted as normal ruggae on a abdominal CT scan on post-op day 17. A bezoar of 50-60 or more tablets was discovered by direct visualization through the G-tube on post-op day 18. A chest film the same day showed the bezoar filling approximately one-third of stomach. Lavage through the G-tube was unsuccessful in breaking up the bezoar. Only 5-6 tabs were able to be crushed and retrieved with an endoscope and gastric lavage on post-op day 20. On post-op day 21, three more attempts were made to retrieve the bezoar by endoscopy and gastric lavage. The bezoar was finally evacuated on day 22 endoscopically with Trochar and repeated crushing of tablets with laparoscopic forceps and irrigation. No increase in serum potassium was noted either before or after crushing tabs. The patient was actually hypokalemic before discovery of the bezoar and during and after its removal. Conclusion: Potassium chloride wax-matrix concretions may be mechanically removed. Hyperkalemia may not have occurred due to appropriate release from matrix or due to adequate irrigation during removal.

Platform Session 2 — Sunday, September 13 — 10:00 am - 11:00 am

Alcohols

Abstracts #58–#61

58 PHARMACOKINETICS OF FOMEPIZOLE (4MP) IN PATIENTS.

McMartin KE, Brent J, and META Study Group. *Louisiana State University Medical Center, Shreveport, LA; University of Colorado Health Sciences Center and Toxicology Associates, Denver, CO*

Background: Fomepizole (4-methylpyrazole) has been approved for treatment of ethylene glycol (EG) poisoning in humans. We have determined 4MP pharmacokinetics in 17 patients from the META prospective multicenter clinical

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trial. Methods: Serial blood samples were obtained from 17 patients enrolled in the EG arm of the META trial, with 14 fitting the criteria for EG poisoning. Patients received loading doses of 4MP of 15 mg(183 μmol)/kg with supplemental doses by defined schedule. Plasma 4MP concentration-time data were analyzed for the saturation kinetic parameters during periods without dialysis and first order parameters during hemodialysis. Results: The peak 4MP plasma concentrations ranged from 200-400 μM (mean 342) and occurred generally after the first dose. Because of the repeated dosing, there were defined troughs, with mean Cmin of 105 μM and Tmin of 8 h. Zero order elimination rates, based on multiple data points, could be determined after the first dose and in 4 cases after multiple doses. 4MP elimination was very consistent within each patient. The mean (±SEM) rate of elimination in all patients was 16±3 μmol/L/h. The mean volume of distribution was 0.69±0.05 L/kg. Data from 9 patients were sufficient for analysis of kinetics during dialysis, when 4MP elimination was first order. The elimination half-life and plasma clearance during dialysis were 3.0±0.4 h and 230±25 ml/min, respectively. No significant adverse effects were attributable to the 4MP plasma concentrations obtained in patients in the META trial. Conclusion: The elimination kinetics of 4MP in EG-poisoned patients were very similar to those previously reported in healthy volunteers. The dosing protocol used in the META trial maintained 4MP plasma levels well above those that are considered therapeutic (10 μM), without producing any apparent adverse effects.

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59 PHARMACOKINETICS OF ETHYLENE GLYCOL AND METHANOL DURING FOMEPIZOLE THERAPY: RESULTS OF THE META TRIAL.

Sivilotti M, Burns M, McMartin K, Brent J, META Study Group. *University of Massachusetts Medical Center, Worcester, MA; Beth Israel Deaconess Medical Center, Boston, MA; Louisiana State University Medical Center, Shreveport, LA; University of Colorado Health Sciences Center, Denver, CO*

Objective: To characterize the pharmacokinetics of ethylene glycol (EG) and methanol in poisoned patients treated with fomepizole. Methods: Patients who ingested EG or methanol and received fomepizole as part of a prospective multicenter trial were evaluated. Serial blood samples from 18 EG and 10 methanol-poisoned patients were analyzed for EG, methanol, and ethanol (GC), and fomepizole (HPLC). Linear regression was performed on postabsorption concentration-time data to define the operative kinetics and elimination rates for EG and methanol during fomepizole monotherapy. Monotherapy was defined as the interval when ethanol < 10mg/dL and hemodialysis not in progress. Individual patient data were normalized and pooled to estimate overall elimination rates (expressed as means±s.e.). Elimination half-lives during fomepizole monotherapy were compared to those calculated when fomepizole plasma concentrations fell below 10μmol/L, and when ethanol was present with fomepizole. EG elimination half-life was correlated with measures of renal function (pretreatment serum creatinine and calculated creatinine clearance). Results: During fomepizole monotherapy, EG and methanol elimination appeared first-order with half-lives 19.7±1.3 (r^2=0.72) and 54.4±4.8hrs (r^2=0.68), respectively. When fomepizole concentrations fell below 10μmol/L, EG (7.5±1.0hrs) and methanol half-lives (10.5±0.5hrs) significantly decreased ($p<.001$). When ethanol was present with fomepizole, there were no significant differences in EG (19.6±1.1hrs; $p=.95$) and methanol half-lives (54.7±4.7hrs; $p=.96$) as compared to fomepizole monotherapy. During fomepizole + hemodialysis, EG and methanol half-lives were 3.5±0.4 and 2.8±0.4hrs, respectively. Patients with pretreatment elevated creatinine (>1.2mg/dL) had significantly longer mean EG half-lives than those with normal creatinine (48.6±6.4 vs 17.7±1.2hrs; $p<.001$). Creatinine clearance correlated with EG half-life ($r^2=.57$; $p<.001$) during fomepizole monotherapy. Conclusion: The slow rate of EG and methanol serum elimination during fomepizole monotherapy and significant increase when fomepizole concentrations are < 10μmol/L suggest strong inhibition of hepatic metabolism by fomepizole. Pretreatment creatinine > 1.2mg/dL and depressed creatinine clearance predict prolonged EG half-life during fomepizole monotherapy.

60 A COMPARISON OF FOMEPIZOLE WITH HEMODIALYSIS VS FOMEPIZOLE ALONE IN THERAPY OF SEVERE ETHYLENE GLYCOL TOXICITY.

Donovan JW, Burkhart K, McMartin K. *Central Pennsylvania Poison Center, Pennsylvania State University, Hershey, PA; Louisiana State University, Shreveport, LA*

Background: Fomepizole (4-MP) is an alcohol dehydrogenase inhibitor recently approved for therapy of ethylene glycol (EG) toxicity. It may be effective as sole therapy even in cases of severe EG toxicity with high plasma EG levels, but the resultant delay in elimination of EG necessitates multiple doses and may prolong hospitalizations. Hemodialysis may additionally hasten EG elimination and thus shorten the clinical course. This report compares the course, outcomes, and costs of fomepizole alone vs fomepizole with hemodialysis in a small group of patients. Methods: The clinical course and hospital charges of 2 patients who received fomepizole and hemodialysis were

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contrasted with 2 patients treated with fomepizole alone. All were treated at the same center within one month. Data analyzed included hours post-ingestion at presentation (HPI), peak plasma EG, lowest CO2, peak creatinine, hemodialysis duration, fomepizole doses, duration on ventilator, length of stay (LOS), and charges. Results: All patients had good outcomes without progression of acidosis or development of renal failure. Maximum acidosis and creatinine occurred at presentation in all cases. The two dialyzed patients each had a peak EG level of 100 mg/dL, presented at 13.5 and 14.5 HPI, received 2 and 3 doses of fomepizole, had dialysis for 2 and 3 hrs, and LOS of 2.5 days, and hospital charges of $15,616 and $24,315. The two non-dialyzed patients had EG levels of 92 and 574 mg/dL, presented 12.5 and 3 HPI, received 3 and 5 doses of fomepizole, had an LOS of 4.5 and 3.5 days, and hospital charges of $30,072 and $16,790. Conclusion: Therapy of EG toxicity with fomepizole and hemodialysis vs fomepizole alone in similar patients had the same good outcome but dissimilar courses. The LOS was longer in fomepizole only patients. There was no direct correlation between charges and use of HD vs fomepizole alone. Charges appeared to be related to factors other than use of hemodialysis or fomepizole doses.

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61 A COMPARISON OF THE BIOAVAILABILITIES OF ORAL AND INTRAVENOUS ETHANOL IN HEALTHY FEMALE SUBJECTS.

Goldberg J, Cobaugh DJ, Wax PM, Krenzelok EP, Kwong TC, Schneider SM. *Finger Lakes Regional Poison & Drug Information Center/University of Rochester, Rochester, NY; University of Pittsburgh, Pittsburgh, PA*

Background: Ethanol (ETOH) is administered orally (PO) or intravenously (IV) in treatment of methanol and ethylene glycol toxicity. Serum concentrations (SCs) of 100 mg/dl are recommended. Studies in males demonstrated a difference in ETOH bioavailability between routes of administration. In males, the mean peak serum concentration following PO ETOH was 71.3 mg/dl and 103.6 mg/dl following IV administration. Mean time to peak was observed at 103.5 minutes after oral dose initiation and 46.5 minutes after IV dose initiation. Male and female metabolism have been shown to differ. The objective of this study was to characterize the bioavailability of ETOH in females following a 700 mg/kg ETOH loading dose. Methods: 20 healthy female volunteers between 21 and 40 years old were enrolled. Subjects abstained from ETOH 48 hours before each phase. They were randomized to receive either PO or IV ETOH. After a 7 day washout period they crossed over to the other group. Before each phase, baseline ETOH SCs, pregnancy tests and drug screens were obtained. Two hours after a standard breakfast, they received 700 mg/kg of ETOH. PO ETOH was administered as a 20% solution in juice over 10 minutes. IV ETOH 10% in D_5W was administered over 30 minutes. Blood was drawn for ETOH SCs at timed intervals over 6 hours. Statistical analysis was done with a dependent T-test ($T=9.3$). Results: Mean peak SC was 146.9 mg/dl after IV administration and 98.1 mg/dl after PO administration ($P<.0001$). Mean time to peak SC was 30.4 minutes after initiation of IV administration and 83.8 minutes after initiation of PO ($P<.0001$). Conclusion: Significant differences exist between mean peak SC and mean time to peak SC following PO and IV ETOH loading doses in females. This is similar to previously reported male data. Unlike males, following a 700 mg/kg PO loading dose females achieved a therapeutic SC.

Poster Session 2 — Sunday, September 13 — 1:30 pm – 6:30 pm
Abstracts #62–#116

62 ACUTE INTOXICATIONS BY MUSHROOMS AND PLANTS IN SLOVAKIA.

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Background: Every year the Poison Information Center in Bratislava responds to about 1,600 inquires from all over Slovakia. Poisonings by plants comprise about 3.6% and mushrooms 3.7% of inquires. Mushroom intoxications can be the serious often resulting in death. This report describes our experience with plant and mushroom poisoning. Methods: Review of cases reported to the PCC during the last 8 years. Results: Of 638 mushroom cases, 18 were fatal (12 adults; 6 children). Death was caused by *Amanita phalloides* with death occurring about 7 days after ingestion. The primary symptoms appeared 22 (16-28) hours after ingestion. Treatment was started 35 (24-46) hours after ingestion. In one instance 19 children were intoxicated by *Amanita phalloides*. The age range was 20 months to 17 years; (57%) were boys. The diagnosis was based on history, gastrointestinal symptoms, and laboratory parameters (AST, ALT prothrombin, bilirubin). Primary symptoms appeared after 19 (10-28) hours. Treatment consisted of: single and multiple-dose activated charcoal, cathartics, forced diuresis, supportive treatment (plasma,

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Vitamin K, antihaemorrhagics, vitamins, hepatoprotectives), antidote therapy (Legalon Sil IV (Silibinin)). All survived. In another case, a mother breast fed her 1 month old son, 13 hours after eating amatoxin-containing *Lepiota*. The mother became ill and was considered for liver transplantation, although renal insufficiency was absent. Her status improved with supportive care and transplantation was unnecessary. The infant displayed no clinical symptoms, but did have moderately increased transaminases which resolved within 3 months. Most plant intoxications were caused by *Diffenbachia*, *Datura stramonium*, and *Atropa belladonna*. One death occurred. A man mistook the leaves of *Colchicum autumnale* for *Primula veris* and ingested them.

63 THE MOST PREVALENT SYMPTOMS ASSOCIATED WITH COMMON PLANT EXPOSURES.

Krenzelok EP, Jacobsen TD, Aronis J. *Pittsburgh Poison Center, Children's Hospital of Pittsburgh; Hunt Institute for Botanical Documentation, Carnegie Mellon University; Keck Center for Computational Biology, University of Pittsburgh, Pittsburgh, PA*

Objective: The symptoms associated with plant exposures are chronicled by a scant number of case reports, animal poisoning data, limited experience and myth. The objectives of this study were to examine the common plant exposures and to profile the most prevalent symptoms associated with those exposures. Methods: AAPCC TESS (1993-1996) was searched to identify all plant exposures. A subsequent search was conducted to identify a subset of exposures that were associated with symptoms. The ten most common exposures were identified and the symptoms associated with the exposures were categorized by frequency for single symptoms and symptom complexes. Results: There were 412,457 plant exposures reported during the first four years of AAPCC TESS symptom documentation. The ten most common exposures accounted for 26.5% of all reported plant exposures. Within this category 70.6% of the patients were symptom-free following the reported exposure. 73.0% of the symptoms occurred in patients exposed to various Toxicodendron and Capsicum species. The ten most common exposures accompanied by the most prevalent symptom and symptom complex are: 1. Capsicum--dermal irritation/pain (54.2%) and dermal erythema, flushed, pain; 2. Philodendron--oral irritation (5.9%) and oral/throat irritation; 3. Euphorbia pulcherrima--vomiting (1.5%) and nausea/vomiting; 4. Ilex--vomiting (1.4%) and diarrhea/vomiting; 5. Dieffenbachia--oral irritation (18.2%) and oral/throat irritation; 6. Toxicodendron--rash (50.8%) and rash/pruritis; 7. Ficus--vomiting (3.3%) and vomiting/cough-choke; 8. Phytolacca--vomiting (2.6%) and diarrhea/vomiting; 9. Crassula--vomiting (0.5%) and vomiting/cough-choke; 10. Solanum--vomiting (2.4%) and nausea/vomiting. An extensive list of symptoms and the frequency of those symptoms will be presented. Conclusions: This is the first attempt to profile the reported plant exposure symptoms. Knowledge of the most common symptoms can allow poison specialists and toxicologists to anticipate patient prognosis, assist in the confirmation of a suspected diagnosis and to guide referral decisions.

64 THE MEDICINAL PLANTS OF THE KIKUYU AND NDOROBO TRIBES AND MAASAI HEALING TRADITIONS.

Stipetic ME. *Texas Poison Center Network, Central Texas Poison Center, Scott & White Memorial Hospital, Temple, TX*

Background: Beneath the divine blue canopy sky of the Great Rift Valley of Kenya lingers the origin of our ancestors. Below the flat-topped acacia trees the majestic lion relaxes among the first healing roots, literally, known to man. The origin of a number of medicinal plant species can be attributed to Africa. Among the Kenyan savannah flourishes an astonishing plant life in both number and diversity. The various rural and remote communities of Kenya depend on the land for their livelihood and health. Knowledge of which plants can be consumed and which ones can be used as medicinals is crucial to their well-being. As Paracelsus recognized in the sixteenth century, "Dosage alone determines poisoning." It goes without saying that this knowledge has been imparted to the enduring occupants of the Kenyan terrain. Methods: Take a trip through the eyes of a CSPI as Paul Githinji Kabochi, a Ndorobo hunter-gatherer and resident naturalist, takes an ethnobotanical field trip revealing fascinating plant specimens and their traditional medicinal uses. Dominick Njugona reviews some of the forest medicinals in the Lewa Downs area based on the knowledge passed down by his father, a medicine man of the Kikuyu Tribe. A walk on the Maasai Medicine Trail gives further insight into the healing traditions and medicinal plant uses of this tribe. Results: The forest community of Kenya contains trees ranging from the camphor to the giant St. John's Wort. Locate euphorbias and aloes among the mighty acacias of the grassland and desert landscape. Discover how the asanti blood plant, a relative of the coffee plant, is used to treat intestinal disorders and how the calla lilly is used to treat sores. Identify the cardiac glycoside containing thelvetia plant and many more medicinal and toxic plants. Conclusion: Familiarity with the local florae is a way of life for the inhabitants of the African communities. This is a fascinating and practical topic to all involved in toxicology.

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65 MULTI-SYSTEM TOXICITY FROM INTENTIONAL HELLEBORE INGESTION.

Schuetz E, Adams J. *Maryland Poison Center, Baltimore, MD*

Background: Hellebore (common name for *Veratrum viride*) is a coarse, erect, herbaceous plant found in low, moist habitats throughout the United States, but most commonly in the Eastern US. The Veratrum genus of plants has been known to be poisonous since antiquity. The principle poisons are the veratrum alkaloids, most of which are glycoalkaloids. The principle signs of intoxication are severe nausea and vomiting, bradycardia, hypotension or hypertension, syncope, parasthesias, and ECG changes. Case Report: A call was placed to the Poison Center (PC) by a 52-year-old woman who reported eating a plant while walking in the woods. Her initial complaints included severe sweating, nausea, and numbness all over. The PC recommended immediate transport to the ER and the patient was flown by helicopter, with an additional plant specimen, to the hospital. While en route, the patient was given a dose of activated charcoal, and experienced nausea, vomiting, diarrhea, syncope and bradycardia (HR=42 beats/min). The initial EKG showed a sinus bradycardia. At the ER, the patient received a second dose of charcoal and vomited again. The nausea and vomiting were treated with metoclopramide 10mg I.V. and she was given another dose of charcoal, which she retained. Initial chemistries and CBC were WNL. The patient continued to complain of parasthesias. The plant was positively identified as hellebore by calling a local university botany professor and comparing it with an illustrated text in the ER. After approximately 45 minutes in the ER, the patient was still bradycardic (HR=40s) and had become hypotensive (BP=50s systolic). She was given 0.5mg of atropine and her pulse increased to 100 beats/min. Dopamine was started at 5mcg/kg/min but discontinued immediately after the rapid response to the atropine. The patient was admitted to the ICU for cardiovascular monitoring. In the ICU, there were no further complaints of parasthesias or further syncopal episodes, only minor nausea, which resolved without further treatment by the next day. The patient was discharged asymptomatic two days post ingestion. Conclusion: Significant toxicity is possible after intentional hellebore ingestion.

66 A GAP IN THE SAFETY NET: A MULTI-CENTER PROSPECTIVE STUDY OF HERBALS AND OTHER DIETARY SUPPLEMENTS.

Palmer M, Haller C, McKinney P, Everson G, Klein-Schwartz W, Grimm K, Woolf A, Foley M, Smolinske S, Bartlett D, Nelson L, Dahl B, Horowitz Z, Todd-Butera T. *New York City Poison Control Center, New York, NY*

Background: Poison Centers (PC) are currently unable to report detailed data concerning herbals and other dietary supplements (DS). While regulations, research and surveillance are minimal, consumption of DS is explosive. This study was designed to prospectively assess this gap using 1998 PC cases. Methods: A database (Microsoft Access) was developed to analyze standard data collected from 13 PCs. Symptomatic cases were evaluated for probable causality. Status of information about products in Poisindex (PI) was recorded. Results: Among 412 cases, 124 (30%) involved symptoms and the remainder were asymptomatic exposures (172, including 46 unintentional pediatric cases) or product information requests (116). Dieting and athletic/body-building performance were commonly stated as motivations for product use (62 cases). DS are believed to cause the majority of symptomatic cases (61%) with at least 50% probability. Search of PI yielded no information on over half of 241 products (59%). The most frequently reported non-homeopathic products were: St. John's Wort (15/58 symptomatic), Melatonin (4/24 symptomatic), Echinacea (6/21 symptomatic), Ginkgo (5/13 symptomatic), Creatine (6/11symptomatic). Ma Huang and/or caffeine-containing botanicals also stand out in the list of products that have probable/definite causality of mild-moderate symptoms (13 cases) in addition to several unknown remedies (3 cultural, 5 domestic not in PI), Yohimbe, Tea Tree Oil, Bee products, Melatonin, Spirulina (2 each) and a multi-ingredient laxative also containing Comfrey and Aloe, both potentially toxic orally. Severe cases included two hospitalizations for hepatitis (related to multi-ingredient products) and two deaths by myocardial infarction in patients with clean coronary arteries and no other clear etiology on autopsy (Pau d'Arco in a 57 year old female; Ma Huang/caffeine product in a teenage male). Conclusions: The PC community cannot assume safety of DS products or adequacy in information tools (PI) and database compared to standards maintained for other product types. Multi-ingredient formulations compound the difficulty of surveillance.

67 POISONING BY HERBAL INFUSIONS INGESTED AS ABORTIFACIENT AGENTS.

Laborde A, Ciganda C. *Department of Clinical Toxicology and Poison Control Center (CIAT) Montevideo, Uruguay*

Background: Limited scientific information is available about the toxicity of plants and herbal preparations popularly ingested to induce abortion. Since mixtures of plants could be ingested, and many patients deny intentional abortion, the real cause of disease and abortion is very difficult to establish. This investigation assesses the use of plants as abortifacients in our facility, and describe the clinical patterns and kinds of plants used, in order to facilitate diagnosis and treatment. Methods: A retrospective descriptive study of cases with a history of the ingestion of herbal infusions

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as abortifacient agent between 1990 and 1997 was performed. Results: A total of 57 cases were identified. All cases occurred during the first trimester. Plants most commonly involved were: *Lycopodium selago*, *Ruta chalepensis*, *Mentha pulegium*, *Margyricarpus punnatus* and *Petroselinum hortense*. All patients were symptomatic with genital bleeding and digestive symptoms the most frequent cause of ED consultation. Multisystemic alterations occurred in severe cases, all were at first presumed to be post-abortion septacemia. Hepatotoxicity, nephrotoxicity and hematological alterations occurred in 9 cases. Hysterectomy was done in 2 cases; with normal pathology. Early gastrointestinal symptoms, absence of respiratory distress and persistent hypokalemia distinguished intoxication from an infectious etiology. Abortion always occurred as part of multisystemic disease (3 cases) and never as an only symptom. *Ruta chalepensis* was involved in all of the severe cases. *Ruta chalepensis* was involved in 2 fatal cases. *Lycopodium selago* did not cause abortion or severe poisonings. Conclusion: Abortion was associated with *Ruta chalepensis*, *Mentha pulegium*, *Margyricarpus pinnatus* and *Petroselinum hortense*. *Ruta chalepensis* and *Mentha pulegium* induced abortion as part of systemic poisoning. Both reportedly contain principles with abortifacient properties and are considered toxic plants. Intentional ingestion of herbal infusions to induce abortion can lead to severe multisystemic disease. Although some clinical features indicate herbal poisoning as an etiology, in severe cases diagnostic difficulties still persist. Further studies are needed to develop clinical and analytical patterns that allows suitable diagnosis and treatment.

68 NICOTINIC TOXICITY FROM TINCTURE OF BLUE COHOSH (*CAULOPHYLLUM THALICTROIDES*) USED AS AN ABORTIFACIENT.

Rao RB, Hoffman RS, Desiderio R, Raysor D, Palmer ME, Hung OL. *New York City Poison Control Center, Glens Falls Medical Center, New York, NY*

Background: *Caulophyllum thalictroides*, (blue cohosh) is used as a uterine stimulant, abortifacient and emmenagogue. Caulosaponin and caulophyllosaponin found in blue cohosh have proven oxytocic effects in animals. Methylcytisine, a quinolizidine alkaloid with nicotinic effects is also found in *Caulophyllum thalictroides*. This is a unique case of nicotinic toxicity from blue cohosh used as abortifacient. Case Report: A 21-year-old female 6 weeks pregnant spent four days drinking 15 cups/day of slippery elm tea, along with 10-20 doses/day of tincture of blue cohosh. She also used a douche of parsley dipped in slippery elm tea. She presented with two days of abdominal pain and bilious vomiting. Her temperature was 39°C; blood pressure 149/62 mmHg; pulse 148 beats/minute; respiratory rate 24/minute. Physical examination was significant for diaphoresis, abdominal muscle fasciculations, soft abdomen, and diffuse muscular weakness. Her pupils were normal size, equal and reactive. The electrocardiogram demonstrated sinus tachycardia. The electrolytes were normal. Urine was strongly positive for ketones. Ultrasound of the uterus demonstrated a viable intrauterine pregnancy. She responded to 24 hours of intravenous hydration with resolution of symptoms. Conclusions: Tincture of *Caulophyllum thalictroides* used as an abortifacient (blue cohosh) can produce significant nicotinic toxicity in the absence of detectable oxytocic effects. Slippery elm is used an a demulcent with limited clinical effects. Clinicians need to be aware of toxicities of naturally occurring products, especially those used as abortifacients.

69 DIEFFENBACHIA SPECIES EXPOSURES... AN EVIDENCE-BASED ASSESSMENT OF SYMPTOM PRESENTATION.

Pedaci L, Krenzelok EP, Jacobsen TD, Aronis J. *Pittsburgh Poison Center, Children's Hospital of Pittsburgh; Hunt Institute for Botanical Documentation, Carnegie Mellon University; Keck Center for Computational Biology, University of Pittsburgh, Pittsburgh, PA*

Objective: Dieffenbachia species exposures are among the most common plant exposures reported to poison centers. The literature often ascribes significant morbidity to Dieffenbachia exposures. However, poison center experience suggests a discordant view from that presented in the literature. The purpose of this project was to determine if the symptoms described in the literature were consistent with clinical practice. Methods: To profile the symptoms reported in the literature, an extensive literature search using Medline and Webspirs (http://infoshare.library.pitt.edu/webspirs/webspirs.htm) was conducted to identify publications that described Dieffenbachia toxicity; standard toxicology references and medical botany texts found in a poison center were used to profile symptoms in the tertiary literature; and an Internet search using the Savvy Search program (http://www.guaraldi.cs.colostate.edu:2000) to search simultaneously all other search engines was conducted. The symptom information was compared to AAPCC TESS (1993-1996) actual symptom data on Dieffenbachia exposure reports. Results: Nineteen sources were utilized to profile the literature perspective on Dieffenbachia exposures. AAPCC TESS contained 10,796 Dieffenbachia exposures and 34.7% of the reports documented symptoms. Oral irritation was reported in 18.2% of the reports and

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100% of the queried literature listed this symptom (18.2%/100%). Dermal pain 8.7%/89%; vomiting 2.6%/26%; erythema 2.5%/5%; throat irritation 2.3%/32%; oral edema 2.2%/100%; pruritis 2.1%/5%; ocular irritation 1.7%/47%; rash 1.2%/37%; cough/choke 1%/5%; loss of speech 0%/42%; salivation 0%/37%; respiratory obstruction 0%/68%; death 0%/16%. When multiple symptoms occurred the most common toxidrome was oral and throat irritation that occurred in 2.9% of the reports. Conclusions: There is similarity between the literature and the clinical experience with regard to the irritant properties of the Dieffenbachia species, but there is also significant disparity because the literature fails to put the symptoms into perspective. In general, the literature portrays Dieffenbachia exposures to be associated with more morbidity and mortality than what is actually observed in clinical practice as exemplified by the AAPCC TESS data.

70 *AMANITA BISPORIGERA* - DOSE RELATED TOXICITY FROM INTENTIONAL ABUSE.

Scalzo A, Blume C, Weber J, Madhok M, Thompson M. *Cardinal Glennon Regional Poison Center, Saint Louis University Health Sciences Center, St. Louis, MO*

Background: AAPCC data (1991-1996) revealed 240 cyclopeptide mushroom exposures. We report three cases of *amanita bisporigera* ingestion, illustrating dose related toxicity. Medline search yields no reports of exposure to this mushroom species. Case Series: Three teenage males ingested varying amounts of mushrooms. The Regional Poison Center was contacted, potential victims identified and referrals made to the emergency department. Patient 1 presented with nausea, repeated emesis, abdominal cramps, hypotension and cyanosis at 10-12 hours post ingestion of eleven mushrooms. Initial laboratory revealed metabolic acidosis and mild ↑ PT. Within one week, the patient developed fulminant hepatic failure (Factor V <1%, peak AST 2459U/L and ALT 3649U/L, APTT 54.8sec, NH_4 67mmoles/L). The patient was designated status one for orthotopic liver transplantation. ERCP was performed with nasobiliary duct placement to interrupt the enterohepatic recirculation of the toxin. Charcoal hemoperfusion was instituted while receiving oral multidose activated charcoal (AC). Supportive therapy for hepatic failure included: NAC, penicillin G, cimetidine, thioctic acid, silybum marianum, cholestyramine and sandostatin. He recovered with aggressive, supportive care and was discharged on hospital day 8. Patient 2 presented with severe, watery diarrhea, nausea, vomiting and abdominal pain at 10-12 hours post ingestion of three mushrooms. Laboratory revealed mild elevation of LFTs. Oral AC and penicillin G were administered. A trial of charcoal HP was performed to remove circulating toxins. An attempt was made to place a nasobiliary tube via ERCP. Factor V was 89%. He was discharged asymptomatic on hospital day 6. Patient 3 presented asymptomatic after ingesting one-half mushroom. Laboratory studies were normal. Mushrooms harvested for mycological examination yielded a positive identification for *amanita bisporigera*. Conclusion: Thorough history and physical assessment coupled with accurate mycological identification are essential to proper management of mushroom ingestion. Aggressive supportive care, enhanced elimination and ERCP for naso-biliary drainage may improve outcome. This case series demonstrates dose related toxicity.

71 POISONING FOLLOWING OLEANDER SMOKE INHALATION.

Khasigian P, Everson G, Bellinghausen R, Geller R. *California Poison Control System, Fresno Division, Fresno, CA, Mercy Healthcare, Bakersfield, CA*

Background: Oleander (*Nerium oleander*) is a common ornamental plant found throughout many parts of the United States including the Central Valley of California. Despite its potential for toxicity, Oleander is utilized in landscaping by many homeowners. Poisoning from accidental ingestion of plant parts or intentional ingestion from teas brewed using Oleander leaves is well documented in the literature. However, clinical signs of oleander poisoning along with a positive digoxin assay have not, to our knowledge, been reported following inhalation of smoke from the burning of Oleander. We report such a case as follows. Case Report: A 42-year-old female presented to the emergency department with a history of nausea, vomiting, and dizziness for two days. She also experienced a brief syncopal episode. The patient had a history of fibromyalgia but was otherwise previously healthy and did not have access to digoxin or digitoxin. Upon admission to the emergency department, the patient continued to vomit and complain of severe dizziness which worsened upon movement. At this time the patient revealed that she had been burning Oleander, two days earlier, to dispose of the prunings. She had been exposed to the smoke outdoors periodically for two hours. An electrocardiogram revealed a sinus bradycardia of 51 with no ST changes or evidence of AV block. Her blood pressure was 149/85. A digoxin assay performed 48 hours post-exposure was measured at 0.3 ng/ml and her potassium level was 3.8 mEq/L. She was treated with one liter of 5% Dextrose in ½ Normal Saline, given 10mg of prochlorperazine intravenously, 100mg hydroxyzine intramuscularly and was discharged following a three hour observation period. Her heart rate and blood pressure were 59 and 108/47 respectively, prior to discharge. Conclusion: Inhalation of smoke from burning oleander may result in Oleander toxicity. Patients with symptoms

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consistent with a cardiac glycoside toxidrome after inhalation of oleander smoke should be evaluated for cardiovascular, electrocardiographic and laboratory evidence of cardiac glycoside toxicity.

72 CARDIOTOXICITY FROM INGESTION OF UNPROCESSED NERIUM OLEANDER LEAVES TREATED WITH FAB FRAGMENTS.

Graeme KA, LoVecchio FA, Selden BS, Kunkel DB, Higgins TJ, Perkins KM, Wong A. *Good Samaritan Regional Medical Center, Thunderbird Samaritan Medical Center, and St. Joesph's Hospital, Phoenix, AZ*

Background: *Nerium oleander* ingestions treated with digoxin-specific Fab antibody fragments have rarely been reported. Most previous cases involve ingestion of *Nerium oleander* teas. A literature search revealed one previous case of unprocessed *Nerium oleander* leaf ingestion treated with Fab fragments. We report severe cardiotoxicity following intentional ingestions of unprocessed *Nerium oleander* leaves treated successfully with digoxin-specific Fab antibody fragments (Digibind®). Case Reports: 1) A 66-year-old female, without previous cardiac disease or antidysrhythmic use, presented after ingesting *Nerium oleander* leaves. Upon arrival, she was lethargic and vomiting leaves, with a pulse of 75 bpm. She developed bradycardia at 20 bpm with asystolic periods of 4 seconds, then spontaneously converted to PAT at 140 bpm, followed by a normal sinus rhythm (with first degree AV block and nonspecific ST and T wave abnormalities) alternating with a junctional escape rhythm with frequent PVCs. Serum RIA for digoxin was 0.8ng/ml with a potassium of 4.8mmol/L. Five vials (200 mg) of Digibind were infused. Following Digibind, she had no further dysthymias, but continued to have nonspecific ST and T wave abnormalities. Repeat serum RIA for digoxin was 0.1ng/ml, corrected for Digibind. Her serial cardiac enzymes and discharge ECG were normal. 2) A 26-year-old male, without previous cardiac history or antidysrhythmic use, ingested 3 bowls of unprocessed *Nerium oleander* leaves. He presented 2 hours after ingestion, with vomiting and an initial pulse of 57 bpm. He developed bradycardia at 30 bpm, with first, second and third degree AV blocks, asystolic periods of greater than 5 seconds, junctional escape rhythms, and nonspecific ST and T wave abnormalities. Serum RIA for digoxin was 0.77ng/ml with a potassium of 5.8mmol/L. Ten vials (400mg) of Digibind were infused. His dysrhythmias resolved. His pulse and echocardiogram were normal at discharge. Conclusion: Cardiotoxicity induced by ingestion of unprocessed *Nerium oleander* leaves was treated successfully with Digibind.

73 DIGITALIS POISONING FOLLOWING THE INGESTION OF AN HERBAL DIETARY SUPPLEMENT.

LoVecchio F, Seby MV, Johnson D. *Department of Medical Toxicology, Good Samaritan Regional Poison Center, Phoenix, Arizona; Flagstaff Medical Center, Flagstaff, AZ*

Introduction: Herbal supplements may contain numerous combinations of plants and herbs that on occasion are contaminated with toxins. The FDA had warned that Plantain, a key ingredient in Chomper®, had been identified as *Digitalis lanata*, containing digitalis. We describe two family members who ingested Chomper and suffered cardiac glycoside toxicity. Case Reports: Case One: A 31-year-old woman ingested Chomper, a "cleansing herbal dietary supplement," in an attempt to "clean her intestines." She ingested two 1410 mg tablets twice daily for three days prior to presentation. She complained of emesis, malaise, lethargy and blurred vision. Heart rate, 73 beats per minute; blood pressure, 123/69 mm/Hg; respirations, 16 breaths per minute. She was afebrile. Physical examination was normal except for emesis. An electrocardiogram revealed normal sinus rhythm with "digitalis-effect" in the precordial leads. Serum potassium level was 3.9 mEq/L, and serum digitalis level was 4.2 ng/mL; other laboratory data were normal. She was treated supportively and discharged two days later with a digitalis level of .7 ng/mL and a normal ECG. Case Two: The patient's husband presented with similar signs and symptoms. He had ingested three 1410 mg tablets three times daily for three days prior to presentation. Physical examination was significant for a heart rate of 52 beats per minute and emesis. An ECG revealed sinus bradycardia. Serum potassium level was 4.2 mEq/L, and serum digitalis level was 5.2 ng/mL; the remainder of his laboratory data were normal. He was treated supportively and discharged with a digitalis level of 1.3 ng/mL two days later. A Chomper tablet provided by the patient was dissolved in water, and digoxin or a digoxin-like substance was identified using an enzyme immunoassay (EIA) technique. Presumeably this represented digitalis. Conclusion: Herbal supplements may become contaminated with toxic substances. In this case *Digitalis lanata* was mistaken for plantain and resulted in subacute digitalis poisoning in our patients.

74 PROGNOSTIC IMPLICATIONS OF HYPERKALEMIA IN TOAD TOXIN INTOXICATION.

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Taichung, Taiwan, Republic of China

Background: The toad possesses several toxic substances. Toad toxin poisoning manifests itself primarily with digitalis-like, cardioactive effects which results in bradycardia, varying degrees of atrio-ventricular block, ventricular tachycardia, ventricular fibrillation, and sudden death. Case Report: A cluster poisoning in a family who became intoxicated after ingestion of cooked toad soup for a skin problem. The youngest one (15 months old) died of refractory bradydyarrhythmias soon after arriving at our hospital. A second child (20 months old), who survived, arrived in shock with hyperkalemia (potassium 7.3 mEq/L) and varying degrees of atrio-ventricular block. She was successfully treated with atropine, lidocaine, and cardioversion, and had a transvenous temporary pacemaker implanted for one day. The third boy (16 years old) had hyperkalemia (potassium 6.3 mEq/L) and bradycardia. The remaining three adults had only mild symptoms of nausea, vomiting, watery diarrhea, and a sensation of numbness over their oral mucosa. Conclusion: The level of serum potassium had prognostic implications in toad intoxication. Determination of serum potassium level is readily available in almost every hospital and is therefore more convenient to measure than serum digoxin level. We conclude that if hyperkalemia develops, the treatment of toad intoxication must be more aggressive to prevent mortality.

75 CORRELATION OF THE SNAKEBITE SEVERITY SCORE WITH ANTIVENIN USE AND LENGTH OF HOSPITAL STAY.

Jordan J, McNally J, Boyer L. *University of Arizona College of Pharmacy, Tucson, AZ, Arizona Poison and Drug Information Center, Tucson, AZ*

Objective: A research tool for clinically evaluating snakebites, the snakebite severity score (SSS) was developed by Dart *et al* in 1994. The purpose of this study was to correlate this recently validated research tool with antivenin use and length of hospital stay in patients reported to a poison center. Methods: A retrospective review of 100 snakebite cases collected by the Arizona Poison and Drug Information Center from 1994-97 was done. Initial and worst-case SSS scores were derived from the free text portion of the AAPCC TESS form. Additionally the number of vials of antivenin used and the length of hospital stay were recorded from the case forms. The data were compared statistically using correlation and simple regression analyses. Results: Using a Pearson correlation, initial and worst-case SSS values had low correlation with antivenin use and length of hospital stay. Regression analysis showed initial SSS and antivenin use are significantly associated ($p = 0.019$). Initial SSS was associated with hospital time when all cases were used ($p = 0.029$), but not when an outlier was excluded ($p = 0.067$). Worst-case SSS was significantly associated with both antivenin use ($p = 0.0083$) and time in hospital ($p = 0.0001$). Conclusions: The regression analysis shows antivenin use has a significant association with SSS values. Time in hospital has an association with the worst-case SSS, but may not with the initial SSS. The worst-case SSS associations are primarily useful for research purposes, as it is determined at the conclusion of a snakebite case. The initial SSS association with antivenin use is important because in the future, the SSS may be used to guide antivenin use. Further research must be done to support this.

76 RESOLUTION OF ELEVATED COMPARTMENT PRESSURES AFTER RATTLESNAKE ENVENOMATION WITH ANTIVENIN.

Tunget-Johnson CL, Pearigen PD, McDermott MJ, Gillingham BL. *San Diego Division, California Poison Control System; Naval Medical Center at San Diego, San Diego, CA*

Background: Objective evidence for development of compartment syndrome in patients after rattlesnake envenomation is not well documented in the literature and the incidence of true compartment syndrome is considered rare. Evaluation and treatment of suspected compartment syndrome secondary to rattlesnake bites is controversial. Fasciotomy for suspected compartment syndrome has been advocated by some clinicians. However, fasciotomy may result in disfiguring scars and carries risks of infection, contractures, and nerve damage. Inadequate antivenin administration as a cause of compartment syndrome has been advanced as a theory in the literature but no evidence has been presented to support or refute this theory. Case Report: A 13-month-old black male was found crying in his room and an 8-10 inch western diamondback (by description) was also present. Examination at the Emergency Department revealed 2 puncture wounds approximately 1cm apart over the 3rd and 4th metacarpals of the dorsum of the left hand. Soft tissue swelling involved the entire hand distal to the wrist. Initial laboratories revealed no coagulopathy. Ten vials Wyeth Antivenin were administered 80 minutes after envenomation and swelling halted at the level of the deltoid muscle. Approximately 6 hours after envenomation, volar and dorsal compartments of the hand and forearm were noted to be tense and swollen. Compartment pressures were: volar forearm 45mmHg, dorsal forearm 61mmHg, volar hand 50mmHg, dorsal hand 51mmHg (Stryker hand-held digital monitor). Upper normal

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limit is 30mmHg. Seven more vials of antivenin were immediately infused over 90 minutes. Repeated measurements immediately prior to a planned fasciotomy demonstrated all forearm and hand compartment pressures were 15-17mmHg. The patient was discharged 3 days later with no residual soft tissue swelling and painless active range of motion of all muscle groups. No fasciotomy was performed and no adverse outcome was noted over the ensuing 6 months. Conclusion: Elevated compartment pressures following crotalid envenomation may resolve with administration of adequate amounts of antivenin avoiding the need for fasciotomy.

77 PROFOUND FIBRINOGENOLYSIS AFTER A GABOON VIPER ENVENOMATION.

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Background: Gaboon viper (*Bitis gabonica*) envenomations are rare but severe manifestations including hypotension, pulmonary edema, hematologic abnormalities including fibrinogenolysis and disseminated intravascular coagulation, and death have been described. We report a gaboon viper bite producing systemic toxicity including massive but clinically silent fibrinogenolysis. Case Report: A 25-year-old male was feeding his pet gaboon viper when he was bitten on the dorsum of his left hand. He immediately sucked on the wounds to remove venom, tied a towel and plastic bag around his wrist as a tourniquet, and came to the hospital. Upon arrival he complained of left hand pain and swelling. His physical examination was remarkable for a grossly swollen left hand that was warm with delayed capillary refill and that had 2 puncture wounds 2cm apart on its dorsum. The local zoologic herpetologist was contacted and within one hour an infusion of 5 vials of Central African viper specific antivenin (Schlangengift-Immunserum Behring Zentralafrika, Germany) was initiated. The "tourniquets" were then removed and over 6 hours the swelling progressed up to the shoulder. Eighteen hours after envenomation the patient complained of chest tightness, dizziness, and nausea and 5 more vials of antivenin were infused with resolution of all symptoms. Over the next 4 days, his fibrinogen fell precipitously to zero with a corresponding rise in fibrin split products and prothrombin time. His platelet count and hematocrit remained unchanged. No specific therapy was initiated for these hematologic findings and no bleeding developed. On day 7 he was discharged with a low but rising fibrinogen level. At follow up 2 weeks later, he had not developed serum sickness or bleeding and his prothrombin time and fibrinogen level were normal. Conclusion: Gaboon viper venom contains a thrombin-like enzyme, gabonase, which cleaves fibrinogen and activates factor XIII. Asymptomatic fibrinogenolysis and elevated prothrombin times after a gaboon viper envenomation may be expected and appears best managed with observation and monitoring alone rather than transfusion of blood products.

78 A CONTROLLED STUDY OF THE SYSTEMIC EFFECTS OF TOPICAL NITROGLYCERIN IN A RABBIT MODEL OF BROWN RECLUSE SPIDER ENVENOMATION.

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Background: Topical nitroglycerin applied to the envenomation site has been recommended as a treatment for brown recluse spider bites, on the assumption that vasodilation removes venom from the site of the bite. We have reported earlier that this treatment has no significant clinical efficacy in a controlled trial in rabbit. Objective: To determine if topical nitroglycerin increases systemic toxicity in rabbits experimentally envenomated with brown recluse spider venom. Methods: 24 New Zealand white rabbits were experimentally envenomated with 20 ug of brown recluse spider venom. Rabbits were randomized to a treatment group which received one inch of 2% topical NTG ointment, and a control group which received vehicle. Ointments were applied every 4 hrs for 3 days. Animals were phelobotimized on days 0, 2, and 4. Hgb, Hct, CPK, BUN, were measured by a blinded laboratory technician. Means $\pm$ standard deviations were calculated, and comparisons between means were made using the Student's t-test, with $p < 0.05$ considered statistically significant. Results: CPK rose from 781 $\pm$ 96 to 3505 $\pm$ 733 in the treatment group from day 0 to day 2 ($p=0.002$) and from 718 $\pm$ 77 to 1794 $\pm$ 319 in the control group ($p=0.004$). The day 2 CPK was significantly higher in the control group with $p=0.03$. Hct fell from 34.6 $\pm$ 0.8 to 26.8 $\pm$ 3.4 from day 0 to day 2 in the treatment group ($p=0.00005$). Hct fell from 32.6 $\pm$ 10.2 to 27.6 $\pm$ 0.6 in the control group ($p=0.058$). There was no significant difference in the day 2 Hct between the 2 groups ($p=0.28$). No abnormalities in BUN and creatinine were noted in either group. Conclusions: Topical NTG applied to the site of Brown Recluse spider envenomations increased CPK rise and anemia in rabbits relative to controls, but only the CPK rise was statistically significant. Recommendations that NTG be applied to the site of human envenomation are not supported by this model.

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79 HUMAN EXPOSURES TO THE *LOFOCAMPA CARYAE* CATERPILLAR: A REVIEW OF 365 EXPOSURES.

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Background: Caterpillars are perceived as gentle, harmless creatures to the lay person. They are a source of curiosity to children because of their size, accessibility and slow mobility. In reality, some harmless-appearing caterpillars are endowed with guard hairs, spinals and spines that secrete biogenic amines as a self-defensive mechanism. Contact with them can produce an irritant or histamine reaction in humans. The purpose of this project was to characterize the presentation and treatment associated with *Lofocampa caryae* caterpillar (tiger moth, white woolly) exposures. Methods: 365 exposures to the *Lofocampa caryae* caterpillar managed by a RPIC over a two year period were reviewed. Results: Pediatric exposures (80%), had a mean age of 4.6 years (SE $\pm$ 3.5) and adults (20%) had a mean age of 37.6 years (SE $\pm$ 16.2). Within the pediatric group 92.1% were dermal exposures, 7.5% oral and 0.4% ocular. The dermal exposures with minimal symptoms were treated at home with the supportive measures of hair and spine removal, irrigation, antihistamine and/or topical steroid administration. Resolution of symptoms occurred within 24 hours. Patients with oral exposures who presented with symptoms of refusal to drink, drooling, oral irritation and positive visualization of hairs or spines, were referred to an emergency department for medical evaluation and removal of the caterpillar hairs. Those children were treated with oral antihistamines and corticosteroid therapy. The routes of exposure in adults were 98.6% dermal and 1.4% oral. Treatment in 97.3% of the exposures included removal of the hairs, irrigation, antihistamine and topical steroid administration at home. The oral exposure was treated at home with dilution--the throat irritation resolved within 24 hours. Conclusion: Symptoms are encountered commonly following dermal and oral exposure to the *Lofocampa caryae* caterpillars. Removal of the defensive guard hairs or spines is the primary treatment. Supportive care with irrigation, antihistamines and/or corticosteroids can decrease the intensity of symptoms.

80 BEE SWARMINGS IN CHILDREN.

LoVecchio F, Graeme KA, Gerkin R, Kunkel DB, Curry SC. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: Massive bee envenomation in adults is associated with a greater than 15% mortality. Melittin, phospholipase A_2, and hyaluronidase in bee venom, may produce vomiting, diarrhea, shock, hemoglobinuria, rhabdomyolysis, multi-organ failure and death. Animal data suggests death is inevitable when $\geq$20 stings/kg are present. We describe our experience with multiple bee stings in five children. Methods: All bee stingers were counted. All signs and symptoms were recorded by the admitting medical toxicologist and a standard form was retrospectively completed. Electrolytes, CPK, CBC, PT/PTT, fibrinogen, and renal function were measured serially in all patients. Laboratory data were measured at baseline, 8, and 16 hours following presentation. Results: Five children had a median age of 4.8 years [range 2-9 years old]. The median total stingers were 36 per patient [range 25-50] or 2.24 stingers per kg [range 1-4.5]. No child had any signs of anaphylaxis. Four of five children had vomiting that resolved within 90 minutes of envenomation, with three patients receiving one dose of an antiemetic (droperidol or hydroxyzine). Three of five children had at least one episode of non-bloody diarrhea within two hours of envenomation. Mild elevations were noted in CK (median 171 IU/L; range 141-191 IU/L), while all other laboratory data remained normal. All patients were discharged the following day with complete resolution of the symptoms and normal laboratory data. Telephone follow-up at 4 days post-envenomation was unremarkable. Conclusions: Bee envenomation resulted in mild systemic illness in our series of children receiving 1 to 4.5 stings/kg body weight. Nausea, vomiting, and diarrhea do not always portend a poor prognosis or involvement of other organ systems.

81 CONTINUOUS INTRAVENOUS MIDAZOLAM INFUSIONS FOR *CENTRUROIDES EXCILICAUDA* ENVENOMATIONS.

Gibly R, Williams M, Walter F. *University of Arizona, Tucson, AZ*

Background: There is no FDA approved antivenin for *Centruroides exilicauda* scorpion envenomation. A MEDLINE search from 1966–1998 revealed no published cases of *Centruroides exilicauda* envenomation treated with continuous intravenous midazolam infusion. This is the first such case series. Methods: A retrospective chart review from July 1,1993 through January 1, 1998 identified all patients treated in our hospital with ICD-9 codes 989.5 (toxic effect of venom) and E905.2 (scorpion sting causing poisoning). *Centruroides exilicauda* envenomation severity was graded from I-IV using Curry's system. Results: 104 patients were identified in our search. Of these, 34 had grade III or IV envenomations, and were treated in the ICU with intravenous midazolam infusions. These 34 patients were

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analyzed for this study. The median patient age was 3.5 years (range 1–68 years). The midazolam dose was adjusted to control agitation and involuntary motor activity. The median amount of midazolam needed to produce the first observed decrease in agitation and involuntary motor activity was 0.26mg/kg (range 0.03-1.76mg/kg) and was given over a median time of 0.83h (range 0.00-3.75h). The median midazolam infusion rate that optimally controlled the agitation and involuntary motor activity was 0.29mg/kg/h (range 0.00-1.29mg/kg/h) and was reached at a median infusion time of 2.5h (range 0.00-8.50h). The median total amount of midazolam given during a hospitalization was 3.30mg/kg (range 0.29-7.37mg/kg). The median duration of midazolam infusion was 9.25h (range 0.00-20.50h). The median duration of ICU stay was 15.08h (range 6.0-28.0h), with 82% of patients being discharged directly from the ICU to their homes. Complications attributed to midazolam included 4 episodes of hypoxia ($\leq$ 90% SaO_2) that resolved with supplemental oxygen, with no permanent sequelae. No patients developed other complications attributable to midazolam. Conclusion: Continuous intravenous midazolam infusion is a safe, effective, and readily available option for treatment of patients with grade III or IV *Centruroides exilicauda* envenomations.

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82 A PROSPECTIVE ANALYSIS OF 558 COMMON STRIPED SCORPION *(CENTRUROIDES VITTATUS)* ENVENOMATIONS IN TEXAS DURING 1997.

Stipetic ME, Lugo A, Brown B, Beard D, Colbert J. *Texas Poison Center Network, Central Texas Poison Center, Scott & White Memorial Hospital, Temple, TX*

Background: The most frequently encountered scorpion in Texas is the *Centruroides vittatus*. This scorpion usually inflicts a painful sting. A retrospective study of scorpion stings in central Texas has reported effects to the nervous system. In the United States, only the *Centruroides exilicauda* has been documented to cause systemic symptoms. We present a prospective study of patients stung by a scorpion who contacted one of the six poison centers in Texas. Methods: All human exposures, between January 1 and December 31, 1997, documented on the Texas Poison Center Network Scorpion Envenomation Study Form were reviewed. Inclusion criteria were human envenomations to a scorpion described as tan in color, presence of a black intra-ocular triangle or black stripes on the thorax. Descriptive analysis of the symptoms was also performed. Results: There were 763 human exposures to scorpions reported and 558 (73%) met the inclusion criteria. Of these cases, 537 (96%) experienced local symptoms including bleeding, pain, burning sensation, erythema, edema, welts/hives, local paresthesia and/or pruritus. The data revealed 109 (20%) displayed one or more systemic symptoms; 43 had facial/perioral paresthesia, 42 had tongue tingling/numbness, 22 had dysgeusia, and various reports of chills, sweating, dysphagia, fasciculation, nausea and vomiting. Discussion: Systemic reactions and more specifically neurological responses following *Centruroides vittatus* stings had not been previously supported by prospective analysis. The Texas scorpion population is known to include the *Centruroides vittatus* and 16 other species. This study confirmed the manifestation of systemic symptoms in this species. Conclusion: Systemic reactions were noted in 20% of *Centruroides vittatus* stings described in this study, which supports the 19% reported in a retrospective study. Other Texas scorpions are tan and may have been included in this study, placing limitations on attributing these effects to the common striped scorpion. Although neurological symptoms can occur following these stings, the mild sequelae suggests that home treatment of these patients is acceptable.

83 ANAPHYLAXIS FROM A *CENTRUROIDES SCULPTURATUS* ENVENOMATION.

Welch S, Thomas R. *Samaritan Regional Poison Control Center, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: Anaphylactic reactions from *Centruroides sculpturatus* envenomations have never been reported. We present a case report of anaphylaxis after a *Centruroides sculpturatus* envenomation. Case Report: A 35-year old healthy female felt a stinging sensation to her lower lip in the early morning hours, while she was in bed. She looked down on her pillow and saw a scorpion. She experienced pain and paresthesias to the lips immediately after the sting (characteristic symptoms seen with *Centruroides sculpturatus* envenomations). Within 30 minutes she developed a diffuse urticarial rash. She experienced chest tightness with respiratory difficulties. She presented to the closest emergency room approximately one-hour post envenomation with swollen lips, mild periorbital edema and bilateral inspiratory wheezes. Vital signs were; BP 147/101, HR 86 and respiratory rate 26. 70 minutes post envenomation she was given 0.3ml epinephrine SQ, 50 mg diphenhydramine IV and 125mg methylprednisolone IV. 15 minutes after medications were administered the swelling decreased by 75% to her lips and the rash was gone. She still had c/o mild tightness to her chest. She was discharged home on diphenhydramine (50mg) every 4 hours and a tapering dose of prednisone starting at 40mg daily. The patient went to an urgent care clinic the next morning upon awakening with diffuse urticaria, swelling to her lips and periorbital edema. Patient still had the paresthesias to her lips. She was treated and discharged home. The patient had been previously envenomated by a scorpion one month earlier.

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Conclusion: We present the first case of an anaphylactic reaction to a *Centruroides sculpturatus* envenomation. Although rare, patients who experience a scorpion envenomation should be instructed to watch for anaphylaxis.

84 PROSPECTIVE STUDY OF IMMEDIATE AND DELAYED HYPERSENSITIVITY TO *CENTRUROIDES* ANTIVENIN.

LoVecchio F, Klemens J, Welch S, Curry SC, Thomas R. *Samaritan Regional Poison Center, Phoenix, AZ*

Background: *Centruroides* scorpion envenomation produces prolonged, excessive firing of neurons resulting in autonomic and voluntary muscle effects, including cranial nerve dysfunction, uncontrolled movements, and respiratory compromise. Goat serum-based antivenin to *Centruroides sculpturatus* is used by many physicians for severe envenomations (Grade III or IV). We performed a prospective study for 12 months evaluating the incidence of anaphylaxis/anaphylactoid reactions and serum sickness following *Centruroides* antivenin administration. Methods: Standard forms documenting delayed or immediate hypersensitivity reactions were completed prospectively by poison specialists on all patients who received *Centruroides* antivenin reported to our poison center or evaluated in our emergency department for 12 months. Telephone follow-up was obtained 7, 14, and 21 days and discontinued after resolution of serum sickness or after 21 days if no symptoms developed. Anaphylaxis/anaphylactoid reactions were defined as stridor, wheezing, urticaria/erythroderma, or shock within one hour of antivenin infusion. Serum sickness was defined as unexplained rash between 1 and 21 days after infusion. Results: 116 grade III or IV patients received *Centruroides* antivenin; 77 cases < 13 years-old. Three developed rash during infusion but completed the infusion at lower rates with adjunctive medications (hydroxyzine, methylprednisolone, and/or epinephrine infusion). One of three was admitted for possible aspiration pneumonia/croup that preceded the envenomation. An atopic asthmatic woman developed wheezing treated with epinephrine infusion and an inhaled β-agonist. Ten were admitted for social reasons, sedation for drugs used before antivenin, or to rule out aspiration and were discharged within 24 hours. The remaining 106 patients were discharged from the emergency department after resolution of symptoms. Seventeen did not complete the required 21 day follow-up period. Of the 99 remaining, 61% [n=60] developed serum sickness (n=47 < 13 years-old). Serum sickness quintessentially responded to oral steroids and/or antihistamines; mean duration of symptoms was 2.8 days with medication. Conclusions: Anaphylactic reactions are uncommon following *Centruroides* antivenin infusion. Self-limited serum sickness that is easily controlled with corticosteroids and antihistamines commonly follows use of antivenin.

85 PROLONGED COAGULOPATHY AFTER BRODIFACOUM INGESTION, AND THE CHALLENGES IN PROVISION OF CONTINUING CARE.

Boyer LV, Griffith L, Fosdick CC III, Fernández M. *Arizona Poison and Drug Information Center, Tucson, AZ; Center for Family Medicine, Casa Grande, AZ; South Texas Poison Center, Austin, TX*

Background: Acute ingestion of superwarfarins has been associated with coagulopathy lasting many months. Monitoring and treatment may be complicated by changes in venue and health care providers. Case Report: A 35-year-old alcoholic male parolee ate approximately 1.5 pounds of brodifacoum on Valentine's day in an effort "to die like the rat he was." He had no symptoms that day, but over 2 weeks he had 3 ER visits for gross hematuria, gingival bleeding and ecchymoses. Eventually, a prothrombin time (PT) greater than 50 seconds was noted; the history of ingestion was elicited; and he received intravenous vitamin K and FFP. He was released on 100mg of Vitamin K1 daily. His parole officer having opined that the state penitentiary was probably not the safest place to treat severe coagulopathy, a judge consented to ongoing parole on condition that he visit a clinic regularly. This system worked moderately well, except for brief interludes of medication noncompliance and bleeding, until Independence Day, when he vanished from medical and legal surveillance. 40 days later he returned to the notice of the Corrections department, following a scuffle with a police officer. A wrist injury resulted in ecchymosis to the axilla, and the man admitted to having stopped taking K1. The PT was 32, now 6.5 months postingestion. Oral K1 was resumed with supervision by prison staff, and PT normalized during a taper that ended 9 months postingestion. Early in his course, the patient was urged to contact his regional poison center for any coagulation-related problems. Numerous calls came to the Center over 9 months, from 3 hospitals, 2 clinics, and the state corrections system. Conclusion: Coagulopathy may persist for up to 9 months following acute brodifacoum ingestion. Optimal treatment requires close monitoring and adjustments in therapy. Regional poison centers can provide continuity of care in cases involving frequent changes of venue.

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86 POISON CENTER'S ROLE DURING COUNTY-WIDE AERIAL MALATHION SPRAYING.

Chambers-Emerson J, Normann SA, Speranza, VC. *Florida Poison Information Center - Tampa, Tampa, FL*

Background: In the summer of 1997, the US Department of Agriculture discovered Medflies (Mediterranean Fruit Flies) in orange trees outside Tampa, FL. In an effort to protect the area's citrus industry, a 20% malathion solution was sprayed from low-flying aircraft. Because spray zones covered residential areas, there was concern about potential toxicity from the spray ingredients and breakdown products. Methods: Anticipating a large number of malathion-related calls that might interfere with normal access and operation of the center, Florida Poison Information Center-Tampa (FPICT) established a special "Medfly/Malathion Line" to answer questions about the potential health effects from the spray. A survey of caller's self-reported symptoms was conducted and served as a basis for a more formal review done in conjunction with the University of South Florida College of Public Health. FPICT organized a meeting of state health, toxicology, environmental and agricultural experts to evaluate risks to human health and address communication/public information issues. Although the spraying stopped after four months, the controversy and planning for future Medfly eradication continues today. Results: FPICT received over 5,000 calls, and out of 584 reported exposures, no cases of malathion poisoning were confirmed. However, the general public attributed "malathion poisoning" to a variety of human and wildlife effects, generating negative publicity about "cover-ups" of potential toxicity by all agencies that provided information. Conclusions: Poison Centers play a vital role in providing public information in a community-wide crisis, but planning and partnerships are needed to do this successfully. Recommendations include setup of a dedicated telephone line and staff to provide standardized information based on Risk Assessments, Reports of Citizen Illness, and Frequently-Asked-Questions from agencies experienced in dealing with similar incidents. Poison center staff may require time to research toxicity, attend community meetings, create a public relations/risk communication plan, collect and record data. The FPICT has entered into a formal agreement with other local, state, and federal agencies to plan for and respond to future pesticide eradication programs.

87 ACCIDENTAL DEATH FROM METHYL BROMIDE EXPOSURE.

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Background: Methyl Bromide (CH_3Br) is an odorless and colorless gas used as an insecticidal fumigant. It is an extremely toxic compound which primarily affects the central nervous system, respiratory tract, and cardiovascular system. To prevent toxic exposures from Methyl Bromide during residential use, the structure is "tented" and guarded for 24 hours. It is also commonly used in industrial settings as a pesticide with similar safety precautions. Case Report: The Poison Center was contacted regarding the management of a 33-year-old female patient experiencing seizures after exposure to a reportedly "unknown insecticide." The boyfriend of the patient had illegally obtained two cylinders of gas that he thought contained propane from an industrial work site. He took these to his girlfriend's home where they attempted to connect them to her stove for use as a fuel source. During attempts to connect the fittings to her stove, one of the cylinders was opened briefly. Continued attempts to connect the tanks to the stove were abandoned after someone at a local hardware store informed them that the tanks did not contain propane gas. The previously opened cylinder remained in the home overnight and continued to slowly leak methyl bromide throughout the night. The next morning the female was found by her 8 year-old son unresponsive and seizing. The son was also symptomatic with lethargy, muscular incoordination, and fine motor tremors. Both patients were transported to the hospital where the female patient had to be intubated and paralyzed for seizure control. After three weeks of continued seizure activity, she developed a flat EEG and was declared brain dead. The 8 year-old continues to require physical rehabilitation for injuries attributed to the long-term central nervous system sequelae of methyl bromide exposure. Conclusion: This case demonstrates the severe consequences associated with the unauthorized use of methyl bromide. Poison Specialists and Physicians should be aware of the extreme toxicity of these products.

88 ORGAN PROCUREMENT AFTER BRODIFACOUM POISONING.

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Background: Ingestion of brodifacoum rodenticides is a common but rarely serious poisoning, with only three deaths reported. Both the increased volume of distribution and potency of brodifacoums has raised concern regarding organ procurement in these cases. The liver itself has been reported to contain 20 to 40 times the brodifacoum concentration of plasma. We present a fourth brodifacoum death with the first reported successful organ procurement of the heart, liver and kidneys. Case Report: A 21 year old male was evaluated after ingestion of an unknown quantity of

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brodifacoum. Charcoal was administered, coagulation studies were unremarkable, and the patient was discharged and lost to follow-up. Six months later the patient presented with hematemesis, abdominal pain, hematechezia, hematuria, and bleeding gums. He was hospitalized for 14 days. His initial hemoglobin of 12.3 g/dl dropped to 6.9 g/dl and he was given packed red blood cells, fresh frozen plasma, and vitamin K_1. His initial PT was 102 seconds, INR was 72, and PTT was 136 seconds. The day before discharge his symptoms had resolved and his PT was 14.9 seconds. However, on the day of discharge his PT had risen to 17.2 seconds. He failed to return for follow-up. Three weeks later the patient was found comatose and incontinent of grossly bloody stool. Initial laboratory tests were significant for a PT of 101.0 seconds, INR of 81.4, and PTT 97.9 seconds. Head CT revealed bilateral intraventricular hemorrhage with a right parietal parenchymal hemorrhage and bilateral perihippocampal herniation. Within 24 hours, he was declared brain dead. The family consented to organ procurement. Blood from the organ donor was positive for brodifacoums. After careful consideration, the patient's liver, heart, and kidneys were procured and transplanted. Six months post-transplantation, the recipients are doing well with no unusual bleeding complications reported. Conclusion: In this case, organs were safely transplanted from a patient pronounced brain dead after complications from a brodifacoum poisoning.

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89 ACUTE PEDIATRIC BRODIFACOUM INGESTIONS.

Shepherd G, Klein-Schwartz W, Anderson B. *Maryland Poison Center, Baltimore, MD*

Background: Brodifacoum is the major rodenticide used in the United States today. Although structurally similar to warfarin, brodifacoum is approximately 200 times more potent and has a more prolonged effect. Brodifacoum 0.005% is available in a variety of forms. Symptoms from large overdoses and chronic intoxications include coagulopathies lasting months and death. Management of acute unintentional ingestions is poorly defined and controversial. Methods: 1993-1996 TESS/AAPCC data were retrospectively searched for all brodifacoum exposures; data on acute exposures without co-ingestions followed to a known outcome in children under 7 years of age were analyzed by management site, symptoms, therapy and outcome. Significant symptoms were defined as evidence of bleeding, hemolysis and coagulopathies. Results: There were 23,481 reported brodifacoum exposures in children <7 years old. Of these, 10,733 cases involved single acute unintentional ingestions and were followed to a known outcome. In this cohort there were no deaths or major effects reported. Minor and moderate effects were reported in 310 (2.88%) and 57 (0.53%) children, respectively. Prothrombin time prolongation was noted in 39 (0.36%) children. Evidence of bleeding occurred in 16 (0.15%) cases. Management outside a healthcare facility occurred in 5,388 (50.2%) cases; 5,345 (49.8%) patients were evaluated at healthcare facilities. Approximately half of the children received some form of GI decontamination. Decontamination had no effect on the distribution of outcome. Adverse effects (GI symptoms) to decontamination therapy were reported in 44 cases. Only 90 children (< 1%) were treated with phytonadione, of which 71 had no effect from brodifacoum. Conclusions: Acute pediatric ingestions of brodifacoum rarely caused clinical evidence of morbidity and was not associated with any life threatening symptoms or deaths. Since GI decontamination did not alter outcome its role is unclear. Phytonadione use was low and appeared to be prophylactic. Considering these data it seems reasonable that acute unintentional ingestions of brodifacoum by children can be adequately managed with observation and parent education. However this approach should be studied in a controlled prospective manner before defining therapy.

90 METHYL BROMIDE POISONING TREATED WITH HEMODIALYSIS.

Palatnick W, Tenenbein M. *University of Manitoba, Winnipeg, Manitoba, Canada*

Background: Methyl bromide is widely used as a fumigant insecticide. However, there is limited experience with poisoning from this insecticide. We report a man severely poisoned with methyl bromide who was treated with multiple anti-convulsants and hemodialysis and was left with significant neurologic sequelae. Case Report: A 35 year old male was fumigating a greenhouse with methyl bromide. He entered it four hours later to clean up. About one hour after the exposure he complained of jaw pain, abdominal cramps and diarrhea. An hour later he was noted to be twitching, although still alert. Within another 30 minutes he developed status epilepticus. He was taken to hospital where high doses of diazepam and phenytoin were ineffective. Seizure activity was eventually terminated with thiopental and neuromuscular blockade. The next day he was transferred to our centre. He was hemodialyzed for 9 hours with a decrease in his bromide concentration from 2.8 mmol/L to 0.6 mmol/L which rose to 0.9 mmol/L after hemodialysis. However extracorporeal removal hemodialysis did not seem to alter his clinical course. He required three weeks of tracheal intubation, two weeks of pentobarbital infusion for recurrent myoclonic seizures, along with phenytoin, phenobarbital, valproate, clonazepam and lamotrigine to eventually control his myoclonus. A CT scan showed possible right frontal demyelination, and an EEG showed cortical and spinal myoclonus. An MR scan of his

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head was normal. He was transferred to a rehabilitation hospital where he spent seven months. He continues to have severe disability with dystonic posturing, action myoclonus, generalized hyperreflexia and difficulties with the activities of daily living despite treatment with L-5-hydroxytriptophan and carbidopa/levodopa. Conclusion: Methyl bromide intoxication may be associated with recalcitrant seizures and serious long-term sequelae. In our patient, hemodialysis decreased his serum bromide concentration, but had no effect upon his clinical course.

91 MOTHBALL BLUES: CONFUSION IN NAPHTHALENE TOXICITIES AND TREATMENT.

Chun T, Perrone J, Osterhoudt K, Ugur S, Henretig F. *The Poison Control Center, The Children's Hospital of Philadelphia, Philadelphia, PA*

Background: Unintentional ingestion of mothballs by young children is a common occurrence, resulting in infrequent but potentially serious hemolysis in G6PD deficient patients. A rare consequence of naphthalene toxicity cited by many toxicology references is methemoglobinemia. We report 2 cases of children with naphthalene induced hemolysis inappropriately treated with methylene blue in the mistaken context of presumed methemoglobinemia. Case Reports: Patient 1 is a 13 y.o. boy, who presented with a 2 day history of abdominal, back, and flank pain, with dysuria, urgency, and hematuria, and a known history of mothball ingestion 3 days earlier. His physical examination was remarkable only for scleral icterus and a soft flow murmur. Initial labs revealed a hemoglobin of 9.2 g/dl, a reticulocyte count of 8.2%, and large blood, but only 3-5 RBC/hpf on urinalysis. He was given 1 mg/kg of methylene blue IV for presumed methemoglobinemia. At 12 hours post-admission, his hemoglobin dropped to 7.1 g/dl, and he required 2 transfusions over the next 48 hours. He was discharged after 4 days, in stable condition. Patient 2 is a 20 m.o. boy who presented with a 1 day history of fever, lethargy, irritability, pallor, jaundice, vomiting, and a known mothball ingestion 3 days earlier. He was noted to be pale and tachycardic on examination. His hemoglobin level was 3.1 g/dl. A slow methylene blue infusion was started for suspected methemoglobinemia. On referring the patient to a tertiary care pediatric hospital, the accepting physician indicated that the methylene blue should be immediately discontinued, and a slow transfusion of PRBC's and urine alkalization initiated. He was transferred to the pediatric hospital where he had an uneventful 2 day stay. Conclusion: Toxicology references often cite methemoglobinemia as a potential consequence of naphthalene exposure, in addition to hemolysis in G6PD deficient patients. It may be insufficiently emphasized that for virtually all patients who present with mothball-induced hemolysis, empiric treatment with methylene blue is contraindicated.

92 FUMIGANT RELATED ILLNESSES: WASHINGTON STATE'S FIVE YEAR EXPERIENCE.

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Background: Acute exposure to fumigants may have severe acute and/or long-lasting health effects. To determine the circumstances surrounding fumigant exposures and their resultant health effects, a review was undertaken of pesticide-related illnesses reported in Washington state over a five year period (1992–1996) and investigated by the Department of Health's pesticide surveillance staff. Methods: Pesticide-related illnesses within the state database were searched, selecting for fumigants as a class. Additional searches were done for individual chemicals known to be used as fumigants in the state, regardless of whether they were classified as fumigant exposures. The combined cases (each exposed individual being one case) were analyzed by nature, route of exposure, and clinical outcome. Results: From 1992-1996, 2759 cases of possible pesticide-related illness were reported, of which 1186 (43%) were suspected or confirmed of being related to a pesticide exposure. Forty-two (3.5%) of these suspected or confirmed pesticide-related illnesses involved exposures to fumigants. The most common fumigant exposures during this period were to aluminum phosphide (12 incidents, 15 cases), methyl bromide (6 incidents, 12 cases), metam-sodium (9 incidents, 9 cases), and zinc phosphide (3 incidents, 3 cases). The nature of exposure for these four products (39 cases) included 19 cases of exposure to applicators, 9 cases of reentry into a fumigated structure, 3 cases of drift, 6 cases of improper storage or disposal, and 2 other types of exposures. Routes of exposure were as follows: inhalation 21, dermal/ocular 8, inhalation/dermal 7, and ingestion 1. Symptoms varied by fumigant, but included irritation (mucous membrane and/or skin) and respiratory symptoms for the majority of exposures. No fatalities occurred. Conclusion: Despite established regulations and training of fumigant applicators, exposures to fumigants continue to occur. Of particular interest are the relatively high number of aluminum phosphide and methyl bromide exposures, and the substantial number of exposures due to reentry.

93 CASE REPORT OF DONEPEZIL OVERDOSE.

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Background: Donepezil (Aricept®) is a centrally acting reversible acetylcholinesterase inhibitor that is used in the treatment of Alzheimer's disease. Donepezil is highly specific for neural acetylcholinesterases. It preferentially binds AChE by greater than 3 orders of magnitude over butyrylcholinesterases. This specificity minimizes peripheral side effects at therapeutic doses. Clinical trial data suggest that donepezil has a dose dependent half-life, at a dose of 5mg/day the t1/2 is 70hrs. Animal studies have demonstrated cholinergic symptoms following large doses. There are no cases of human donepezil overdose currently reported in the literature. Case Report: A 79-year-old nursing home patient with a history of Alzheimer's disease and hypertension was inadvertently given 50mg of donepezil instead of her usual 5mg dose. She presented to the ED with nausea, vomiting and a persistent bradycardia in the 40's. Serum chemistries and blood counts were all within normal limits. Basilar rales were noted 5 hours after arrival. She was treated with 0.2mg of atropine PRN bradycardia < 50/minute. A total of 3.0mg was administered over 18 hours to control bradycardia. Each atropine bolus kept her heart rate in the 60s for 0.5-2 hours. Atropine therapy caused dry mouth and urinary retention requiring foley catheterization. After atropine therapy was stopped she was incontinent of urine. No further vomiting or evidence of pulmonary edema occurred after her initial episodes. RBC cholinesterase and serum donepezil concentrations were not measured. She returned to baseline by day 2 with a heart rate in the 70's and was returned to the nursing home. Discussion: A large overdose of donepezil produced systemic symptoms of cholinergic excess in this patient. The most notable feature of this overdose was the persistent bradycardia.

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94 AGRICULTURAL AVERMECTINS: ARE THEY REALLY SAFE?

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Background: Avermectins are macrocyclic lactones used in the control of nematode and arthropod parasites. Among them, ivermectin has been widely employed as an anthelmintic agent in treating onchoceriasis, while abamectin is an agricultural miticide and insecticide. The toxicity of therapeutic ivermectin is generally not severe and may at best cause hypotension, somnolence and skin rashes. The clinical spectrum of agricultural avermectins poisoning, however, remains not clear. Methods: A retrospective study was conducted to evaluate the manifestations of agricultural abamectin and ivermectin exposures reported to the National Poison Center-Taiwan during the period between September 1994 and September 1997. Results: A total of 13 patients, including 12 patients with Agrimec® (2% abamectin) exposure and one with Ivomec® (1% ivermectin) exposure, were recorded. Males (8) outnumbered females (5) in these exposures. All cases aged between 23 and 81 years old with a mean of 49.8 years. The majority of patients were exposed secondary to a suicidal attempt (9), followed by occupational (2) and accidental exposures (2). Not surprisingly, oral ingestion (10) was the most common way of exposure. Inhalation was noted in 2 patients and dermal contact in another 1 patient. Among these 13 patients, 2 were asymptomatic and 6 presented with minor symptoms, such as vomiting, dizziness and transient bradycardia after ingesting 0.25-0.75g abamectin (mean = 0.5g). Five patients manifested severe symptoms, including coma (5), hypotension (3) and aspiration pneumonia with respiratory failure (2) following an ingestion of 1g ivermectin or 2.5-12.5g abamectin (mean = 6.9g). Although intensive care was needed in these 5 patients, all recovered well within 6 days. Conclusion: Due to its GABA-potentiating effect, agricultural avermectins may produce life-threatening central nervous system depression and hypotension following large amount of oral exposure. Prompt institution of intensive supportive measures, such as ventilatory support, fluid replacement therapy and inotropic agents are needed in reaching a favorable outcome.

95 A RETROSPECTIVE STUDY OF 148 CASES WITH ACUTE TRIPHENYLTIN ACETATE INTOXICATION.

Lin T, Wu M, Deng J. *National Poison Center, Taipei, Taiwan*

Background: Agricultural chemical intoxication is the leading poisoning morbidity and mortality in Taiwan. Excluding organophosphate, carbamate and other herbicides, triphenyltin acetate (TPTA) was also widely used. We recently found two mortality cases of TPTA ingestion, which were inconsistent with the description of the literature. This has triggered our intention to review the clinical significance of TPTA poisoning in our series. Methods: All cases registered in Taiwan National PCC with TPTA poisoning, during the period of March 1986 and June 1996, were retrospectively collected. Data including sex, age, route and dose of exposure, clinical symptoms and signs, and outcome were abstracted and analyzed by Chi-square test. Results: Excluding 59 cases with incomplete data, 111 male and 37 female cases of acute exposures with TPTA were identified. Fifty seven cases (38.5%) were suicide ingestion, 16 (10.9%) miswallowing, and 75 (50.6%) occupational exposure. The age ranged from 1-85 years with an average of 46.6 years (±19.6). The common symptoms including nausea & vomiting (30.4%); skin burn (10.1%); conscious disturbance (10.1%); dizziness (8.9%); urticaria (8.9%). Nineteen percent were asymptomatic. Among the 7 deaths (5.4%), all were suicide ingestion. Three patients died of aspiration pneumonia with ARDS who ingested 45%

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products of 100 g, 75 g and 50 g respectively and all of them combined with delayed CNS depression. Dosage of exposure and old age were found to be significantly associated with mortality ($p < 0.005$). Conclusion: In general, acute TPTA exposure is relatively mild and symptomatic treatment shall be the mainstay of management. However, in suicide ingestion, aspiration pneumonia and delayed CNS depression are poor prognostic indicators.

96 MULTI-SYSTEM FAILURE IN A SUICIDAL CHLORDANE INGESTION. A CASE REPORT.

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Background: Chlordane is a chlorinated hydrocarbon insecticide which has been previously thought to act primarily as a neurotoxin in acute intoxications. Although it has not been manufactured in many years, clinicians will occasionally have to treat patients exposed to these presumed extinct products. Most acute overdoses have produced gastrointestinal symptoms of nausea, vomiting, and diarrhea. Neurologic symptoms include confusion, agitation, ataxia, tremors, muscle weakness, and convulsions. We present a unique case report of an adult who died following an intentional chlordane ingestion. Case Report: The patient was a 46 year old female, brought to the hospital after she reportedly had a seizure. She admitted to taking 10-12 paroxetine tablets, and to drinking 6-8 ounces of chlordane. Her mental status returned to baseline while in the emergency department. ABG analysis revealed a severe metabolic acidosis with a pH of 7, pCO_2 of 31mmHg and HCO_3^- of 7.8mEq/L. She experienced a generalized tonic-clonic seizure which was controlled with lorazepam 1mg. Her liver enzymes, amylase, and bilirubin were elevated. The patient was transferred to the ICU. The following day she became confused and disoriented. The patient had a persistent metabolic acidosis (pH 6.97), DIC (platelets 10,000, pt 27.1, ptt 33.5, INR 4.94, fibrinogen 99mg/dL), and renal failure (BUN 16, Creatinine 3.6). She was started on hemodialysis. Her liver enzymes and bilirubin continued to increase. She was diagnosed with a severe global encephalopathy. On Day 3 she was still severely acidotic (pH 7.16). DIC persisted despite packed red blood cell and fresh frozen plasma transfusions. Hepatic failure and pancreatitis worsened. Her glucose was 1081mg/dL and her CK was 4644U/L. On Day 4 she developed asystole which could not be reversed and she was pronounced dead. Conclusion: This is an atypical case of chlordane overdose that resulted in multi-system failure and a fatal outcome. The paroxetine was not thought to have contributed significantly to the clinical presentation of this patient.

97 LINDANE: A CASE OF DANGEROUS PLAYTIME.

Ryan ML, Arnold TC, Dick RM. *Louisiana Drug and Poison Information Center, Northeast Louisiana University, Monroe, LA*

Background: Lindane is a chlorinated hydrocarbon insecticide. Its toxicity is a result of central nervous system stimulation. Lindane is absorbed by ingestion, inhalation, and dermal exposure. Symptoms of toxicity include nausea, vomiting, tremor, dizziness, weakness, and seizures. This is a case report of a child who exhibited symptoms of toxicity including seizures on two consecutive days following dermal exposure to lindane 20.0%. Case Report: A healthy 9 month old female was exposed to a 20.0% lindane insecticide which had been applied to a wooden deck at her home. The child played on the deck for 1 hour the day after application, and again the following morning on the dew moistened deck. Later that day the child had seizure activity witnessed by a Registered Nurse. She was transported to a clinic where the physician diagnosed strep throat and apparent febrile seizure activity, although her temperature was never noted over 100.2 degrees. The mother said she was extremely fussy that evening. The next morning she again played on the moist deck for 45 minutes. Within 2 hours the child was having seizures. She was again brought to the clinic where she was seizing upon arrival. Seizure activity ceased shortly after arrival. Discussions with family members at this time revealed the presence of the lindane, her play while wearing only a diaper on the treated deck area, and the likely correlation between the lindane and the symptoms. The child was released with instructions to avoid the deck area. She vomited several times that night. She also seemed wobbly and extremely irritable to the mother for the next two days. Follow up has revealed complete resolution of all symptoms and no further seizure activity. Conclusion: Lindane is a common insecticide with toxic potential by all routes of exposure. This case demonstrates the efficiency by which lindane may be dermally absorbed. Clinicians and Poison Center personnel should consider this toxin when cases of seizures of unknown etiology are present.

98 ACUTE ETHYLENE CHLOROHYDRIN INTOXICATION: REPORT OF 13 CASES.

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Background: Ethylene chlorohydrin (ECH), which can be used to hasten grape germination, may cause severe toxicity upon dermal or oral exposure. Although it is prohibited in Taiwan, ECH intoxication still occurs following its illegal usage. Case Series: A retrospective study was conducted to better understand the clinical spectrum of ECH intoxication in Taiwan. All cases reported to the PCC-Taiwan as ECH intoxication between July 1985 and June 1997 were carefully reviewed. Their demographic and clinical data were then analyzed. Thirteen patients, including 9 males and 4 females, were recruited. Their age ranged from 2 to 62 years with a mean of 44.3years. The intent for exposure was suicidal in 5, accidental in 6, and occupational in 2 patients. Oral ingestion was the most common way of exposure (84.6%). Five out of the 13 patients (38.5%) died within 24 hours after oral ECH exposure. A 55-year-old male even expired in 3 hours after a suicidal ingestion of 20-30 ml ECH. The ingested amount was 5-10 ml in 2-year-old boy, but unknown in the other 3 fatal patients. Respiratory failure was the leading cause of death in these 5 patients. No possibly useful antidotes, such as alcohol, had been tried in these 5 cases. The symptoms in the rest 8 patients who survived were much less severe. Three patients developed nausea, vomiting, dizziness, tachycardia and chest tightness, and the other 5 patients manifested only mild oral discomfort. All of these 8 patients recovered without any sequelae. Conclusions: ECH is highly toxic and can cause sudden respiratory failure and death following either intentional or unintentional exposure. Intensive supportive measures remain the cornerstone in the management of ECH intoxication, but antidotal agents may also be investigated to antagonize its major effects. Nevertheless, education of farmers and complete removal of this illegal chemical from agriculture use are of utmost importance in future prevention of ECH intoxication.

99 TOXICITY FROM VOLCANIC ERUPTIONS.

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Background: An estimated 1000 people die annually as a result of volcanic eruptions. While this number is relatively small, most of the risk is borne by those populations living near active volcanoes. Limited information is available concerning health effects of and disaster preparedness for volcanic disasters. This study was conducted to review available data from previous volcanic eruptions in one at risk location and abroad in order to identify observed and potential toxic hazards. Methods: The available literature pertaining to health effects from volcanic eruptions in this location and others was reviewed. The literature included descriptive reports by eyewitnesses, relevant geological investigations of the eruptive materials and reports of health effects and mortality resulting from volcanic eruptions. A literature review was also conducted to benefit from experience abroad. Results: The most common and abundant substances released in eruptions in this location for which data are available are: sulfur dioxide, hydrogen fluoride, hydrogen chloride, carbon dioxide, carbon monoxide, hydrogen sulfide, metallic mercury, and crystalline silica. Pulmonary sequelae from inhalation of volcanic gases have ranged from minor respiratory complaints in mild exposures to acute bronchospasm, pulmonary edema and death in severe cases. Those most quickly affected have been children, the elderly and people with asthma or other respiratory diseases. Fluoride poisoning has been observed after volcanic eruption. Deaths have been attributed to asphyxia from carbon dioxide accumulation in the vicinity of the crater both here and abroad. Other potential toxic hazards of volcanic eruption include chemical asphyxiants from carbon monoxide or hydrogen sulfide release, silicosis from fine particles of crystalline silica adsorbed to volcanic ash, heavy-metal poisoning from the release of metallic mercury or thallium chloride and radiation exposure from release of radioactive radon. Conclusion: A review of the experience with volcanic eruption both in this location and abroad identified a number of observed and potential toxic hazards. This information may be used to guide efforts at education, prevention and disaster preparedness by our poison center and others.

100 EPIDEMIOLOGY OF US WORKPLACE CARBON MONOXIDE FATALITIES.

Hamilton RJ, Greenberg MI. *MCP-Hahnemann School of Medicine, Allegheny University of the Health Sciences, Philadelphia, PA*

Background: Sources of CO are common in the workplace. The epidemiology of occupational CO fatalities has not been previously reported. Methods: Bureau of Labor Statistics data were searched for carbon monoxide fatalities for the years 1992 to 1996 inclusive. Data was analyzed using EPI Info 6.04b software. Fatalities which involved fires or explosions were excluded. Demographics of the worker and the workplace, nature of work performed, activity at the time of death, and the source of CO were included in the final analysis. Results: There were 203 CO deaths in this five year period, and it was the leading cause of toxin (non-drug) related deaths. There were 54 suicides, three homicides, and 146 accidental CO deaths. The number of CO deaths was not statistically significant year to year. The

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largest number of fatalities were in the Midwest, followed by the South, Northeast, and West. Managers, mechanics, construction workers, farmers, and janitors were most frequently affected, with managers leading the suicidal group and construction workers leading the accidental group. Thirty percent of fatalities were among the self employed, and 62% were in business with < 10 employees. Motor vehicles were the source of CO in 84% of the suicides and 41% of the accidental deaths. Half of all CO deaths occured from November to March whether by accident or suicide, or in warm or cold regions. Seventy three percent of deaths were in workers between the age of 25 and 54. All CO fatalities decreased as time on the job increased (Linear regression, $r = -0.66$, $P < .0.005$). Conclusion: CO fatalities are more often seen in the small business or the self employed. The winter months of November to March are particularly concerning. On the job experience may play a role in decreasing the incidence. The incidence of the occupational CO deaths does not appear to be decreasing. There may be a role for CO detectors in the workplaces.

101 DELAYED NEUROLOGIC SEQUELAE (DNS) FROM OCCUPATIONAL CARBON MONOXIDE (CO) EXPOSURES.

Hamilton RJ, Greenberg MI. *MCP-Hahnemann School of Medicine, Allegheny University of the Health Sciences, Philadelphia PA*

Background: CO exposures which do not cause syncope and produce COHb levels less than 25% are thought to be at low risk for developing DNS. Case Report: Two males (patient A 39 y/o and patient B 56 y/o) were removing ceramic tiles from a suburban shopping store with a propane operated forklift. The work area was sealed in a plastic tent and was inadequately ventilated. On day one, both patients spent approximately 6 hours in this work area and developed symptoms of a flu-like illness which resolved at home. Upon returning to work the next day they rapidly developed symptoms of headache, nausea, dizziness, and dyspnea. Hours later, confusion, lapses in judgement, swings in mood, weakness, and fatigue forced both workers to seek medical attention. At the hospital, their COHb was measured at 22.0% (patient A) and 19.7% (patient B). Patient B developed chest pain with a normal ECG and serial cardiac enzymes. Both patients were treated with supplemental oxygen and released when their symptoms improved and the COHb had dropped below 10%. The next day patient A complained of fatigue and headache and was treated with monoplace hyperbaric oxygen therapy. Ten days later, both patients were found to have labile and depressed moods, deficits in short and long term memory, and an unsteady Romberg with hesitancy on nose to finger pointing. Neuropsychiatric testing of patient A revealed difficulty completing trail-making tasks and digit span memory (5 digits). Neuropsychiatric testing of patient B demonstrated borderline difficulty with digit symbol recognition and a score below the lowest dysfunction range for trail making. Patient A has experienced mild improvement and patient B has continued to deteriorate over the subsequent three months. Conclusion: These cases serve as a reminder that debilitating CO exposures can be insidious and result in DNS without syncope or COHb greater than 25%. This may be especially true for exposures that occur over time ("soaking").

102 HYDROGEN SULFIDE-INDUCED CHANGES IN COIN APPEARANCE.

Graeme KA, Wallace KL, Curry SC, Tanen DA, Higgins TJ. *Department of Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: A sewer worker found unconscious at the work site presented to the ED combative and smelling of rotten eggs. Initially, faces of coins (quarters, dimes) found in his pockets appeared normal, however, the corrugated edges of coins were blackened (the usual copper-colored rim was absent). Twenty hours later, the patient's condition was markedly improved, but the coins were noted to have slightly blackened faces and increased blackening of the corrugated edges. Early blackening of the corrugated edges of coins and delayed blackening of coin faces contrasts with previous reports of black coins found on victims of hydrogen sulfide (H_2S) exposure at autopsy. We evaluated changes in coin appearance after exposure to H_2S. Methods: Untarnished, recently minted coins (penny, nickel, dime, quarter) were suspended in a cloth pocket and exposed to experimentally generated H_2S at various concentrations between 65 and 310 ppm for 15 minutes within a closed container. Coins were evaluated for color changes at 20 minutes, 1, 2, 3, 6, 12, and 24 hours. Experimental coins were compared to unexposed control coins and to coins that became completely black within 10 minutes of experimental exposure to much higher H_2S concentrations. Results: Purple and purple-blue discolorations of pennies were the earliest and most consistently observed change, noted in all trials at 20 minutes. Purple-blue to blue discolorations of the corrugated edges of dimes were consistently noted at higher concentrations, and usually occurred within 20 minutes. Discoloration of the faces of nickels, dimes and quarters generally occurred later and inconsistently. No delayed blackening of the faces of experimental nickels, dimes or quarters was noted after 12 hours (in contrast to our patient's coins). Conclusions: Purple and blue discolorations of pennies and the corrugated edges of dimes may be an earlier and more sensitive surrogate marker

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of clinically significant H_2S exposure than the classically described blackening of coins reported after fatal exposure. Survivable H_2S exposures may not reliably result in coin darkening.

103 CYANIDE EXPOSURES - A CASE SERIES.

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Background: Cyanide (CN) exposures are uncommon, but can result in significant toxicity. We reviewed CN exposures from 2 poison control centers and the authors' experiences over the past 8 years. Case Series: Five cases of CN exposures were identified. The routes of exposures included oral (3) dermal (1), and pulmonary (1). The actual history of CN exposure was available in only one of three suicidal cases and neither the occupational nor the unintentional case upon presentation to the emergency department. CN toxicity was considered in the other cases because of the significant symptoms: cardiac arrest; altered mental status, metabolic acidosis, and a dermal burn to the foot; coma and hypotension; or respiratory arrest and metabolic acidosis. Historical information that alerted clinicians to potential CN exposure included: employment in an electroplating factory; employment in a jewelry factory; home electroplating; and ingestion of an unknown metal polish. pH's ranged from 6.90 to 7.47, and CN levels (in mcg/mL) ranged from 0.45 to 13.90. Four of five patients were treated with the cyanide antidote kit, one clinically improved without therapy. Two cases had temporary improvement with prompt therapy, but subsequently expired from multiorgan failure. One patient did not have any clinical improvement despite treatment. The patients who died were more acidotic and more symptomatic on presentation. The two survivors had initial pH's of 7.41 and 7.47. Conclusion: CN exposures often have dramatic presentation and result in significant consequences, even with proper therapy. A brief history and certain clues may suggest CN toxicity, but often can not be conclusive on initial presentation. Only a heighten suspicion of CN exposure by clinicians can permit prompt therapy.

104 METHEMOGLOBINEMIA AND SYNCOPE INDUCED BY BUTYL NITRITE INHALATION.

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Background: Federal restriction of amyl nitrite has led to widespread commercial availability of volatile butyl and isobutyl nitrite. Although used ostensibly as room oderizers or video head cleaners, they are abused recreationally via inhalation for vasodilatory effects. Although several reports have identified inhalational amyl and isobutyl nitrite as causing clinically significant methemoglobinemia, this is the first reported case implicating inhalational butyl nitrite as a cause of symptomatic methemoglobinemia. Case Report: A 52 year old male presented to the emergency department after a syncopal episode while dancing at a nightclub. He appeared anxious and complained of shortness of breath. Physical exam revealed a blood pressure of 75/55mmHg, respiratory rate of 26/min and pulse of 112/min. A pallor complexion with cyanotic lips, ears, and extremities were noted. Heart, lung, abdomen and neurological exam were normal. He was incontinent of stool (guaiac negative). ECG showed a sinus tachycardia without evidence of ischemia. Chest radiography revealed clear lungs without effusions or mediastinal widening. Continuous pulse oximetry was 87%. Arterial blood gas values at 6L oxygen were; pH 7.44, PCO2 31 and PO2 123mmHg. Cooximetry revealed a methemoglobin of 23% and oxyhemoglobin of 74.5%. Intravenous methylene blue was administered over a five minute period and his cyanosis resolved after 45 minutes. Intravenous dopamine was administered and his hypotension resolved over the following four hours. The patient admitted to nitrite inhalation shortly before "passing out". He submitted an unmarked colored bottle containing 5ml of remaining liquid which was identified as butyl nitrite by gas chromatography. The patient's NADH methemoglobin reductase activity was determined to be 15 IU/gHb(nl=10-19 IU/gHb). Conclusion: This patient presented to an ED in a state where commercial availability of butyl and isobutyl nitrites are prohibited. Lack of similar restrictions by bordering states have undermined local attempts to control availability and widespread abuse. Despite increasing reports implicating inhalation of butyl and isobutyl nitrites causes of significant morbidity, they are not restricted at a federal level.

105 ACUTE DAPSONE INTOXICATION AND METHEMOGLOBINEMIA IN CHILDREN. A CASE SERIES STUDY.

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Background: Dapsone, a widely used anti-leprosy, anti-parasitic and anti-inflammatory drug, can cause methemo-

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globinemia and hemolytic anemia at both therapeutic and overdose levels. Although several cases of acute dapsone poisoning have been reported, there have been few cases series studies in children. We describe the outcome of 18 children under 14 years old admitted to our hospital from January, 1988 to December, 1996, with acute dapsone intoxication complicated by a methemoglobin concentration greater than 20%. Patients and Methods: Eighteen patients (1-13 years old, median= 3 yr), were admitted 1-72 hours after the ingestion of 100-1200 mg (median= 350 mg, 10 patients) or an unknown amount (8 patients) of dapsone. The methemoglobin concentration at hospital admission (T0) ranged from 23.5-49.7% (median=37.7%) and the main clinical features at T0 were cyanosis (18), tachycardia (18), vomiting (11), tachypnea (9) and restlessness (5). All of the children received activated charcoal orally or by nasogastric tube in multiple doses (1 g/kg, 3-6 times/day, 3-16 doses, median= 8 doses). Twelve of the 14 patients with methemoglobin levels greater than 30.0% were treated with a single dose of 1-2 mg/kg methylene blue 1-2% infused IV over 5 min. Results: A progressive decrease in the methemoglobin levels was observed after the start of treatment, mainly between 4-8 h after admission. No additional dose of methylene blue after the first one was necessary. Acute anemia was confirmed in eight children. Conclusions: Repetitive doses of activated charcoal plus a single IV dose of methylene blue given when methemoglobin levels were greater than 30% was found to be a safe and efficient treatment in this pediatric series.

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106 MULTIPLE LIFE-THREATENING METABOLIC COMPLICATIONS IN SEVERE VALPROIC ACID OVERDOSE.

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Background: Valproic acid (VPA) is a branched-chain fatty acid used for treatment of petit mal epilepsy and migraine prophylaxis. Manifestations of VPA toxicity are protean, though target organs in acute overdose (central nervous and cardiorespiratory systems) differ from those following chronic exposure (liver, bone marrow). Case Report: A 29-year-old female was admitted 30h after ingestion of an unknown amount of VPA. Her current therapy included venlafaxine but not VPA. On arrival, she was unresponsive, hypotensive (BP_{syst} 70 mmHg) and acidotic (pH 7.20) with an elevated anion gap (34 mmol/L). Endotracheal intubation and dopamine were required. EKG showed peaked T-waves with a prolonged QT_c interval (593). Laboratory findings 36 h post ingestion (p.i.): plasma VPA 660 mg/L, venlafaxine and metabolites within therapeutic range, ethanol 45 mg/dL, K^+ 7.0 mmol/L, AST 407 U/L (peaked 72 h p.i. at 609 U/L), plasma ammonia 179 μmol/L, creatine kinase 19,025 U/L, creatinine 2.9 mg/dL, total serum Ca^{++} dropped within 6h from 6.0 to 2.9 mg/dL; urinary calcium excretion was not elevated. Serum 25-OH-Vit.D and 1,25-OH-Vit.D were normal. At 72 h p.i. platelets were 37,000/μL without bleeding. Plasma ammonia declined rapidly. Thrombocytopenia and hypocalcemia persisted for 10 days despite aggressive replacement. QT_c normalized within 56 h, following VPA concentrations closely [r_s = 0.52, Spearman's rank coefficient, $p<0.05$]. The patient was extubated on day 11 and left hospital on day 18 with minor neurologic deficits. Conclusions: This patient suffered metabolic complications of VPA toxicity hitherto described only in separate patients (hyperammonemic encephalopathy, thrombocytopenia, hypocalcemia). The correlation between QT_c-prolongation and plasma-VPA-concentration is a new observation, possibly due to a direct effect of VPA on cardiac Na^+ channels. The mechanism of VPA-related hypocalcemia is unknown. Renal Ca^{++} loss was excluded in our patient because urinary Ca^{++} excretion was low. Possible VPA-induced disturbance of parathyroid hormone and vitamin D metabolism, and sequestration of Ca^{++} secondary to disrupted fatty acid metabolism, require further investigation.

107 VALPROIC ACID INTOXICATIONS IN 96 DOCUMENTED CASES 1995-1998: THE ROLE OF HEMOPERFUSION IN THERAPY.

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Background: Valproic acid intoxications with doses higher than 100mg/kg body weight in the adult require primary toxin elimination via gastric lavage and activated charcoal combined with a laxative. In the case of serious clinical symptoms and high blood levels hemoperfusion is useful as a secondary elimation technique. Case Report: A 21 year old epileptic woman committed suicide with 22,5g sodium valproate. Gastric lavage began after 2.2 hours and activated charcoal was given. The patient developed a deep coma with severe cardiovascular depression requiring assisted ventilation and catecholamines. Blood levels were 1066mg/l (2.2hrs) and 1562mg/l (5hrs). Hemoperfusion was initiated twice with resulting valproic acid levels of 835 and 413mg/l after 4/8 hours. Epinephrine doses could be reduced significantly during the second hemoperfusion and the patient experienced a complete recovery. Case Series: Among 53,534 intoxications registered from 1995 until April 1998 96 patients (0,2%) aged 1 to 83 years

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(mean 29 years, 63% females) took 0.15g to 90g of valproic acid as mono- or mixed intoxication. 77% were suicide attempts; 12% nonintended intakes. Using the poison severity score at initial evaluation 33% showed minor, 18% moderate and 9% severe symptoms. In 7 cases (8%) we recommended hemoperfusion and in 15 cases as therapeutic option. 9 cases showed a life threatening clinical picture and 3 patients died (one combined intoxication with carbamazepine/valproat). Conclusion: Different valproic acid-formulations can lead to a delay in the rise of blood levels even after sufficient primary elimination. The clinical picture of high dose valproic acid intoxications includes beside electrolyte disturbances a cardiovascular depression. Due to the high protein binding and low distribution volume hemoperfusion can lead to a significant shortening of the plasma half time by increasing valproic acid clearance.

108 FATAL TCA-LIKE CARDIOTOXICITY FOLLOWING CARBAMAZEPINE OVERDOSE.

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Background: Although carbamazepine is structurally similar to tricyclic antidepressants (TCAs), carbamazepine poisoning rarely results in significant cardiotoxicity. Sinus tachycardia, AV block, and QRS prolongation have been reported after carbamazepine overdose but life-threatening tachydysrhythmias have not. We desribe a case of severe carbamazepine intoxication in which atrial fibrillation, wide-complex tachycardia, and pulseless electrical activity (PEA) followed a brief seizure more than 13 hours after an acute overdose. Case Report: A 27-year-old epileptic male was brought to the ED because of an unusually prolonged postictal state following a generalized seizure. No further history was available at that time. He was obtunded and combative with P=96, BP=132/71, R=20, T=100.9°F (R), and O_2 Sat=94%. ECG, head CT, and routine serum chemistries were normal. Two hours after arrival, he had a one-minute seizure. After finding his medication levels to be carbamazepine=48.9ug/ml and phenytoin=27.2ug/ml, activated charcoal was given and whole bowel irrigation was initiated. No other substances were detected in the serum (by GC) or urine (by TLC). The carbamazepine 10,11-epoxide level was 12.5ug/ml (nl=10-20% carbamazepine). Subsequent treatment included endotracheal intubation for oxygen desaturation and dopamine for hypotension. Repeat drug levels 5 hours after arrival were carbamazepine=70ug/ml and carbamazepine-epoxide=19.2ug/ml. Vital signs were stable until 13 hours after arrival when he had another brief seizure. A minute later, his cardiac rhythm went from NSR@80 with normal QRS/QT intervals to rapid atrial fibrillation to an irregular wide-complex tachycardia to PEA. Resuscitation, which did not include $NaHCO_3$, was unsuccessful. Drug levels one hour before his demise were carbamazepine=71ug/ml, carbamazepine-epoxide =24.4ug/ml, and phenytoin=19.6ug/ml. Conclusion: TCA-like cardiotoxicity may occur in severe carbamazepine poisoning. As in TCA poisoning, seizures may precipitate dysrhythmias. In contrast to TCA poisoning, the QRS duration does not appear to correlate with life-threatening events and dysrhythmias may not occur until many hours after carbamazepine ingestion. The late onset of dysrhythmias in carbamazepine poisoning may be related to the activity of its epoxide metabolite. $NaHCO_3$ therapy for carbamazepine-induced dysrhythmias merits investigation.

109 STATUS EPILEPTICUS (SE) AFTER CARBAMAZEPINE OVERDOSE.

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

Background: SE following carbamazepine overdose has not been previously described. We report two cases of SE. Case One: An 18yo female with a history of epilepsy ingested 40g of carbamazepine, and unknown amounts of phenytoin and valproic acid (VA). In the ED she was alert, cooperative with HR 100bpm, BP 145/90. Laboratory results were carbamazepine 14.5mcg/ml, phenytoin 5.5mcg/ml and VA <4mcg/ml. Chemistries were normal and Toxicology screen was negative. She was lavaged and given 50g AC. Mental status deteriorated and 5hrs post-arrival the patient began having frequent grand mal seizures 30sec to 1min in length. 6hrs post-arrival she was intubated and placed on a ventilator. Repeat levels were carbamazepine 47.7 mcg/ml, phenytoin 4.6 mcg/ml and VA 76 mcg/ml. Amobarbital 100mg was given followed by 0.5mg/min infusion, without effect on seizures. HR was 119, BP 130/80. The patient experienced cardiac arrest, with successful resuscitation, including dopamine and norepinephrine infusion. SE continued and amobarbital infusion was increased to 1mg/min and 2mg/min without effect on seizures. 10hrs post-arrival she experienced an apparent MI and ventricular fibrillation to asystole. Resuscitation was unsuccessful. Case Two: A 19 yo male with a history of bipolar disorder was found unresponsive after ingestion of an unknown amount of carbamazepine. In the ED he experienced two brief seizures and was administered 5mg diazepam. HR was 90bpm, ECG Normal sinus and BP 160/90. CXR revealed infiltrates with suspicion of aspiration. The patient was lavaged, given 50g AC, intubated and ventilated. Initial carbamazepine level was 37.4 mcg/ml. Over the next 10 hours he experienced 4 brief discrete seizures, without intervention. 12 hours post-arrival he began seizing continuously.

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Laboratory revealed carbamazepine 44mcg/ml. Diazepam 45mg and phenytoin 1.25g produced no effect. BP dropped to 60/systolic. ECG showed an accelerated nodal rhythm and intermittent ventricular tachycardia. Temperature rose to 105.9°F. Dopamine and a cooling blanket were begun. Hypotension and seizures persisted. Phenobarbital 1.1g produced no apparent effect on seizures. SE halted after 5hrs, during a midazolam infusion. Conclusion: This is the first report of SE after carbamazepine overdose, which may be resistant to conventional therapy.

110 FOSPHENYTOIN-INDUCED BRADYASYSTOLE ARREST IN AN INFANT TREATED WITH CHARCOAL HEMOFILTRATION.

Rose R, Cisek J, Mickell J. *Virginia Poison Center, Department of Emergency Medicine, and Department of Pediatrics, Medical College of Virginia of VCU, Richmond, VA*

Background: Fosphenytoin is a water-soluble prodrug of phenytoin that lacks that propylene glycol diluent thought to be responsible for cardiovascular toxicity following IV phenytoin. There is no experience with fosphenytoin overdosage in humans. Case Report: An 8-month-old infant with no PMH was admitted to hospital with bronchiolitis. Upon arrival the child was noted to have intermittent generalized rhythmic activity with preserved mentation, and was treated with lorazepam, phenobarbital, phenytoin and fosphenytoin. Head CT, LP, metabolic and septic work-ups were negative. On hospital day 5, due to increased seizure activity, serum phenytoin level of 10.8 mcg/ml and serum phenobarbital level of 46 mcg/l, the child inadvertently received a 15-minute infusion of 750mg fosphenytoin which stopped all observable seizure-like activity. 12-15 minutes after the infusion the child developed progressive bradycardia and asystole. CPR was started immediately and the child was resuscitated. Serum phenytoin level was 77.2 mcg/ml 1 hour after the infusion, 73.8 mcg/ml (free = 13.0 mcg/ml) 8 hours later, and 82 mcg/ml 16 hours later. Serum phosphate measured 1 hour post infusion was 8.4 mmol/L. Serum calcium, magnesium and blood urea nitrogen were normal. Approximately 24 hours after the overdose the child underwent 4 hours of continuous venous-venous hemofiltration with a charcoal filter. Total and free serum phenytoin levels approximately one hour after completion of CVVH were 33.9 mcg/ml and 3.9 mcg/ml, respectively. There was no subsequent cardiac sequelae, however the child continued to exhibit tonic-clonic activity and was subsequently treated with carbamazepine, valproate, and was restarted on phenytoin during the one month hospital course. Conclusion: Infusion of 71 mg/kg fosphenytoin (as phenytoin equivalents) in an 8-month-old with new onset seizure activity resulted in bradyasystolic arrest. This dose represent a five-fold error in dose, but maximum safe rates of infusion are not established in children. This case suggests that fosphenytoin can produce severe cardiac toxicity in the absence of propylene glycol.

111 CARDIAC ARREST FOLLOWING LARGE INTRAVENOUS FOSPHENYTOIN OVERDOSE IN AN INFANT.

Lieber BL, Snodgrass WR. *Texas Poison Center Network--UTMB, Galveston, TX*

Background: Phenytoin blocks sodium channels in cardiac and nerve tissue (at concentrations of 0.0001 M, i.e., about 25 mg/liter). Phenytoin (old formulation with propylene glycol) given IV at less than 1 mg/kg/minute dose rate infrequently resulted in cardiac arrhythmias and rarely cardiac sinus arrest. These adverse cardiac rhythm effects occurring at therapeutic doses were attributable to the propylene glycol additive. Any fosphenytoin (new formulation without propylene glycol) adverse cardiac rhythm effects more likely would be due to the drug itself. We report a large overdose of IV fosphenytoin. Case Report: A 13 day old 2.72 kilogram infant with seizures received 300 mg (110 mg/kg) IV phenytoin equivalent as fosphenytoin in a 15 minute infusion. Immediately afterward bradycardia, hypotension (systolic blood pressure 30 torr), and then asystole occurred. After 25 minutes of cardiopulmonary resuscitation (CPR) including endotracheal intubation, sinus rhythm recurred before IV albumin or sodium bicarbonate were given. Initially nonresponsive with fixed dilated pupils and requiring dopamine and mechanical ventilation, the infant gradually regained consciousness with a return to baseline neurological status over the next week, with extubation at 9 days and discharge to home 5 weeks later. Phenytoin plasma levels of 77 and 14 mg/liter were measured at 5 hours and 8 days post-dose, respectively. Conclusions: 1. Large overdose IV fosphenytoin may produce cardiac sinus arrest most likely related to its known sodium channel blocking properties. 2. CPR should be continued for a sufficient time period to allow for possible return of spontaneous cardiac sinus rhythm. This time period perhaps may reflect partially the time for redistribution of phenytoin from heart tissue to other sites in the body.

112 ANAPHYLACTIC REACTION FOLLOWING TOPICAL EXPOSURE TO CEPHALEXIN.

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Background: The incidence of anaphylaxis between cephalosporins and penicillin has been estimated to be between 5-15%. This cross reactivity is generally seen following either parenteral or oral administration. We present a case of anaphylaxis in a penicillin allergic following topical exposure to the cephalosporin cephalexin. Case Report: A 32-year-old pencillin allergic female presented to the ED after spilling cephalexin 250mg/5ml suspension on her right arm. Rather then immediately washing of her arm, the patient refilled the measuring device and administered her son his dose of cephalexin. After administering the child's medication the patient then washed off her arm. Immediately after washing her arm she felt throat tightness and urticaria over her arm. The urticaria became more generalized spreading over both arms, back, chest, scalp, and right thigh. Vital signs in the ED were 90, 169/116, 22, 98.6F. The patient had a pulse oximeter of 100% on room air. An erythematous urticarial rash was noted over the scalp in the occipital region, neck, dorsal aspect of both forearms, right medial thigh, and anterior chest. The lungs were clear to auscultaion bilaterally. The patient was administered 0.3 mL 1:1000 epinephrine subcutaneously. The patient was given 50 mg diphenhydramine intravenously and 125 mg of methylprednisolone. The patient's rash began to abate, however, the patient still complained of symptoms. A second subcutaneous dose of epinephrine was given with 400 mg cimetidine with complete resolution of symptoms. The patient remained asymptomatic in the ED and was dicharged to home on oral diphenhydramine and prednisone. Conclusion: While the incidence of cross reactivity between cephalopsorins and pencillin has been estimated to be as low as 5% clinicians should be aware of the potential severe reaction following a topical exposure to one of the offending medications. Patients with a history of severe pencillin reaction should use extreme caution when handling potentially cross reactive medications such as cephalosporins.

113 CIPROFLOXACIN-INDUCED DYSKINESIA.

Burkhart K, Park A, Donovan JW. *Central Pennsylvania Poison Center, Pennsylvania State University, Hershey, PA*

Background: Movement disorders are common adverse drug effects. A tourette-like syndrome has followed ofloxacin administration. There are no similar reports, however, for ciprofloxacin. Case Report: A 90 yo male presented for evaluation of body tremors and facial spasms. The symptoms had begun the previous day, but were increasing in frequency. The patient's only current medication was ciprofloxacin 500mg BID. This treatment was begun three days earlier for suspected cellulitis. The patient's PMH was significant for Alzheimer's disease, but no other neurological diseases. In the ED, the patient required assistance with walking and undressing. On physical exam, the vital signs were temperature 37.1°C, pulse 84, respirations 24, and BP 150/80 mmHg. The skin exam was normal. On neurological exam he was alert and oriented to person, place and time, cranial nerves II - XII intact, sensorimotor intact, strength 5/5, DTRs 2+ symmetric, and no finger-to-nose ataxia. The patient was observed to have facial spasms and contortions that lasted about five seconds, but recurred every one to three minutes. Simultaneous extremity tremors were present. During these movements the patient could speak fluently. Intravenous diphenhydramine, 25mg was given without effect. The ciprofloxacin was discontinued and the symptoms resolved within 24 hours. With his permission, a video has been made of the patient's movement disorder and recovery. Conclusion: An elderly man quickly recovered from ciprofloxacin-induced dyskinesia that had begun after two days of therapy. The mechanisms for this drug-induced movement disorder have not been clearly elucidated.

114 CONTAMINATION OF ANTIBIOTICS RESULTING IN SEVERE PEDIATRIC METHADONE POISONING.

Lalkin A, Kapur B, Koren G. *Division of Clinical Pharmacology and Toxicology, The Hospital for Sick Children, Toronto, Ontario, Canada*

Background: The use of methadone hydrochloride substitution therapy for heroin addiction is widely accepted. Accidental ingestion of carelessly stored methadone by children has been reported, resulting in severe toxicity. With the advent of an increase in methadone treatment programs, more and more community pharmacies have started to dispense methadone to patients. Errors in dispensing can cause poisonings unless precautions are taken while dispensing this narcotic. We report here a case where an antibiotic preparation got mixed with methadone solution and resulted in an accidental infant overdose. Case Report: A 4½ year old healthy boy was prescribed amoxicillin syrup for cough and fever. Shortly after receiving the second dose of 5 ml he became unresponsive, with difficulty in breathing. On admission, he was arousable by deep pain and pinpoint pupils were noted. A urine sample sent for toxicology screen revealed methadone and its metabolite. Blood methadone levels were 0.23 mg/L and 0.14 mg/L, 5 and 9 hours after the second dose was given, respectively. The amoxicillin solution was tested for methadone and was found to have a concentration of 2.4 mg/L. The child gradually improved and was discharged on day 4 in good condition. The pharmacy in which the antibiotics was dispensed has been a dispensing center for local methadone

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maintenance program, and methadone was accidentally mixed with the antibiotics. Discussion: In this case, an almost fatal outcome occurred when an opiate, methadone, was inadvertently mixed with antibiotics in a pharmacy. We were able to locate 2 previous reports of opiate toxicity in children following ingestion of oral antibiotics preparations. In order to prevent this dangerous situation and minimize such accidents in the future, prompt action is needed. Conclusion: This case suggests that possible fatal errors can occur in community pharmacies. Vigorous quality control in pharmacies is strongly recommended. General practitioners, pediatricians and emergency room physicians should recognize and suspect this rare cause of opiates toxicity in a child.

115 ATRIAL FIBRILLATION FOLLOWING ORAL CYCLOSPORINE OVERDOSE.

LoVecchio F, Curry SC. *Good Samaritan Regional Medical Center, Phoenix, AZ*

Introduction: Cyclosporine toxicity has resulted in atrial fibrillation following intravenous administration in at least one case. We report the first case of oral cyclosporine-induced atrial fibrillation following accidental overdose. Case Report: A 61 year old anephric man was scheduled for a renal transplant and inadvertently received 100 mg Neoral®, an oral cyclosporine suspension, instead of mycophenolic acid. The patient has a history of end-stage renal disease secondary to glomerulonephritis three years prior. He had no prior history of cardiac disease. His medications were ketoconazole and phosphate binders. The patient developed agitation, and flushed within one hour of administration of the cyclosporine. His physical examination revealed a fine tremor of upper and lower extremities. Fasciculations were present in his shoulders, biceps, thighs and calves. The cardiovascular examination was significant for an irregular rate, but was otherwise normal. The remainder of the physical examination, including vital signs were normal. A serum cyclosporin level drawn 60 minutes post-ingestion was 2438 nanograms/ml (normal range 100-300 nanograms/ml; using TDX, Fluorescent Polarization Immunoassay Technology) and fell to 123 nanograms/ml 18 hours after ingestion. An electrocardiogram was significant for right bundle branch block and atrial fibrillation with ventricular rate of 75 beats per minute. The electrocardiogram returned to his baseline normal sinus rhythm with right bundle branch within one hour. Serial CK levels over 24 hours were normal. Thyroid testing prior to admission were normal. An echocardiogram revealed borderline left ventricular hypertrophy with trace tricuspid and mitral regurgitation. The patient was asymptomatic at discharge 24 hours later and received a kidney transplant one month later. Conclusions: Acute cyclosporine toxicity was associated with atrial fibrillation, anxiety, tremors, and fasciculations in our patient.

116 OPTIC NEURITIS IN ACUTE ISONIAZID OVERDOSE.

Lockman P, Shum S. *Texas Panhandle Poison Center, Northwest Texas Healthcare System, Amarillo, TX*

Background: Isoniazid (INH), considered primary treatment of tuberculosis, is remarkable for its toxicity in an overdose. Severe acute toxicity is known for the classic triad of symptoms: seizures, metabolic acidosis and coma. While symptoms of optic neuritis, peripheral neuropathies and hepatitis are typical only in chronic INH toxicity. Case Report: A 17 yo female arrived at the emergency department two hours after ingesting 9 g (146.6mg/kg) of INH. Patient denied INH use prior to the suicidal attempt. The chief complaint was poor vision. She stated she could not see fingers placed in front of her but was able to differentiate light areas from dark areas. Minor symptoms of headache and vomiting were also present. Initial vital signs were: T98.4, HR88, RR16, BP135/70. Blood gas revealed PH 7.41, PCO2 37.3, HCO3 23.3. Anion gap was 15mmol/L The patient received 50 grams activated charcoal with sorbitol, IV fluids and 5 g of pyridoxine in two doses over the following two hours. 6 hours post ingestion a fundal exam revealed hyperemic fundi bilaterally. The Poison Center was contacted and 5 more g of pyridoxine was recommended. Other than visual disturbances, the patient remained asymptomatic. The final 5 g of pyridoxine was administered 10 hours post-ingestion. 16 hours post-ingestion, examination by an ophthalmologist revealed normal fundi and the visual disturbances had resolved. Discussion: Acute INH overdose results in a decrease of GABA synthesis secondary to pyridoxal-5-phosphate (active form of pyridoxine) binding with INH. This decrease in GABA typically results in cerebral excitability which may lead to seizures. Peripheral neuropathies and optic neuritis in chronic INH dosing is secondary to deficient pyridoxine causing demyelination of nerves. Current literature does not describe a mechanism for optic neuritis in acute toxicity. Conclusion: In our patient no sign of acute toxicity developed, rather a sign of chronic toxicity developed. Clinicians may consider acute INH toxicity based on history, presence of the classic triad of symptoms or, while uncommon, chronic toxicity symptoms.

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Platform Session 3 — Sunday, September 13 — 4:00 pm - 5:00 pm

Treatment Protocols
Abstracts #117–#120

117 CHRONIC ETHANOL USE AND ACUTE ACETAMINOPHEN OVERDOSE TOXICITY.

Smilkstein MJ, Rumack BH. *Rocky Mountain Poison Center, Denver, CO; Oregon Poison Center, Portland, OR*

Objective: To compare acute acetaminophen overdose hepatotoxicity in patients with and without chronic ethanol use. Methods: Cases from the National Multicenter Study of Oral *N*-acetylcysteine meeting the following criteria were included: acute acetaminophen overdose, age >10 years, completed *N*-acetylcysteine treatment, valid plasma acetaminophen concentration ([APAP]), and adequate aminotransferase data. If chronic ethanol abuse or alcoholism was noted, cases were considered ethanol$_c$(+). If ethanol use was negative, acute only, or not documented, cases were considered ethanol$_c$(-). Maximum recorded AST, and the proportion of cases with AST or ALT >1000 IU/L were compared between ethanol$_c$(+) and ethanol$_c$(-) cases overall, as well as within subgroups defined by [APAP] risk stratification, time to first *N*-acetylcysteine dose or both. Statistics included t-test of independent samples (means), Kruskal-Wallis 1-way Anova (medians), and Chi-square (proportions). Results: 115 ethanol$_c$(+) and 2321 ethanol$_c$(-) cases were included. As a group, ethanol$_c$(+) cases were older and more often male, but no different in APAP dose by history, [APAP] risk stratification, or time to first *N*-acetylcysteine. Overall, ethanol$_c$(+) cases had higher mean AST(1647±322 vs 608±38 IU/L, p=.002), median AST(49 vs 38 IU/L, p=.013) and proportion with AST or ALT>1000 IU/L(26% vs 13%, p=.0001). The same was true within traditional at-risk subgroups (e.g., "possible", "probable", "high" risk) defined by [APAP]; and among all cases first receiving *N*-acetylcysteine after >8 hours (N=1730). Among cases traditionally considered at low risk ([APAP] below the "200/probable risk" line (N=1014), or *N*-acetylcysteine started within 8 hours (N=706)), there was no difference in any outcome variable between ethanol$_c$ groups. By multiple linear regression, the ethanol-AST association remained significant (p=.0001) after effects of age, gender, time to *N*-acetylcysteine, and [APAP] risk group were considered. Conclusions: Acute acetaminophen overdose with chronic ethanol use was associated with increased hepatotoxicity among at-risk cases. Chronic ethanol use did not increase hepatotoxicity among low risk cases, suggesting that changes in *N*-acetylcysteine initiation guidelines on the basis of chronic ethanol use are unwarranted.

118 VALIDATION OF A SHORTER TREATMENT PROTOCOL FOR ACUTE ACETAMINOPHEN POISONING BASED ON EVIDENCE OF LIVER INJURY.

Roth B, Woo O, Olson K, Albertson T, Geller R, Clark R. *California Poison Control System, University of California San Francisco, San Francisco, CA*

Background: The 72 hour treatment regimen for acetaminophen poisoning is based on an uncontrolled, open study performed for the purposes of showing efficacy and safety to gain FDA approval. The decision to utilize a 72 hour regimen in the original protocol was not based on experimental data (e.g., biokinetic monitoring). A prospective observational study was undertaken to test the hypothesis that patients with normal AST and ALT levels determined 36 hours after acetaminophen overdose do not subsequently develop hepatic injury with N-acetyl cysteine (NAC) is discontinued. Methods: If AST and ALT were normal 36 hours after the ingestion, poison control center (PCC) recommendations were that NAC be discontinued. If AST and ALT were abnormal it was recommended that therapy with NAC be continued until normalization of liver function studies or until the patient received 72 hours of therapy. Data was collected prospectively in the course of usual telephone consults provided by the Poison Control System. Results: 250 consecutive patients were evaluated. 60% of patients had normal AST and ALT 36 hours from the time of ingestion. All of these patients did well without subsequently developing biochemical or physical evidence of liver injury. Of the 149 patients with normal AST and ALT at 36 hours 102 were treated for an average of 16 hours longer than PCC recommendations; 31 were treated for an average of 5 hours less than PCC recommendations; 16 were treated exactly as recommended. Despite inexact adherence to PCC recommendations average duration of therapy with NAC for this group was 36 hours. Follow up was obtained in 90% of cases. Conclusions: Patients without biochemical evidence of liver injury 36 hours after acute acetaminophen overdose require no further therapy with NAC.

119 GASTRIC LAVAGE FOR LIQUID POISONS.

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

Background: Although gastric lavage is no longer considered as routine therapy for the overdose patient, there are

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limited data regarding its efficacy for liquid ingestants. The present study addresses this question. Methods: The experimental design is a randomized controlled crossover study in 8 males and 2 females 22-32 years old ranging in weight from 63.3 - 90.1 kg. A solution of 4.0 g of acetaminophen in 60 ml room temperature water followed by another 60 ml of room temperature water was ingested on two occasions two weeks apart. Blood for serum acetaminophen concentration was collected at 0, 0.5, 1.0, 2.0, 3.0, 4.0, 6.0 and 8.0 hours and analysis was by HPLC. One acetaminophen ingestion served as a control. Gastric lavage through a 34 F orogastric tube was performed 1.0 hour after the other ingestion. The endpoint was a clear effluent with a minimum requirement of five 200 ml lavage cycles. Standard pharmacokinetic calculations were performed. A one-tailed t-test was used for statistical analysis and $p<0.05$ was set as the level of significance. Data are presented as mean $\pm$ SD. Results: Mean Cmax for control and gastric lavage were 59 $\pm$ 12 and 46 $\pm$ 13 mg/L respectively ($p<0.04$). Mean AUC for control and gastric lavage were 195 $\pm$ 10 and 154 $\pm$ 17 mg/l.h respectively ($p<0.03$). The mean decrease of bioavailability was 20% $\pm$ 28 (95% C.I.; 0,40). Conclusions: In this model of liquid poisonings, gastric lavage at one hour resulted in a significant decrease of bioavailability. This treatment effect is of marginal clinical importance because of its modest size and the marked inter-subject variability.

120 EFFICIENCY OF CIVILIAN PARAMEDICS AT PERFORMING MEDICAL INTERVENTIONS WHILE IN CHEMICAL PROTECTIVE GEAR.

Dyer S, Brinsfield K, Woolf A, White D, Haley R. *Massachusetts Poison Control, Boston, MA; Boston Emergency Medical Service, Boston, MA*

Background: With increasing concerns about global chemical terrorism as well as HAZMAT disasters, many civilian EMS systems are evaluating the adequacy of the emergency response to the chemically contaminated patient. Critical ALS procedures need to be performed in the field to save patients suffering a life-threatening toxic exposure. Objective: To determine if civilian EMS paramedics, who do not routinely use chemical protective gear, can efficiently perform medical interventions. Methods: Using a crossover design, 20 EMS paramedics were timed and graded by two physician reviewers on accuracy of endotracheal intubation and intramuscular (IM) injection while in two types of gear: standard uniform and Chemical Protective Equipment (CPE). Chemical Protective Equipment consisted of self-contained breathing apparatus (SCBA), butyl suits and nitrile gloves. Duration of time required to successfully intubate a mannequin and to administer an IM injection were separately standardized and scored. Error rates (e.g. esophageal intubation) were also noted. Comparison between standard uniform and CPE were made using a paired t-test. Results: Intubation was accomplished in 26.58 sec $\pm$ 6.75 in CPE vs. 23.3 sec $\pm$ 4.91 standard uniform ($p=0.02$). IM injection was accomplished in 50.23 sec $\pm$ 12.13 in CPE vs. 42.3 sec $\pm$ 10.79 in standard uniform ($p=0.004$). In CPE 2 recognized esophageal intubations occurred, compared to one in the standard uniform group. Conclusions: Although small but statistically significant differences exist between CPE and standard uniform, these delays in treatment are probably not clinically significant. Decontamination prior to medical intervention will result in a treatment delay of approximately 12-25 minutes for the chemically exposed patient. Despite infrequent use of CPE civilian paramedics can perform life saving maneuvers in a toxic environment.

Poster Session 3 Monday, September 14 7:00 am - 12:00 pm
Abstracts #121–#174

121 EMERGENCY DEPARTMENT COMPLIANCE WITH POISON CENTER DECONTAMINATION ADVICE.

Weiner A, McKay C, McCormick M, Bayer M. *Connecticut Poison Control Center, Farmington, CT*

Background: Emergency Departments (EDs) frequently contact Poison Control Centers (PCCs) for advice on gastrointestinal decontamination of the poisoned patient. No data exists on the frequency of ED compliance with this advice. Methods: Patients being evaluated in EDs for suspected drug overdose were eligible for the study if decontamination had not been done prior to PCC consultation. Chi square analysis was done to compare the rates of compliance with decontamination advice given for pediatric patients vs. adults, for community hospitals vs. university hospitals and for recommendations to use activated charcoal vs. orogastric lavage. Statistical significance was set at $p<0.05$. When decontamination advice was not followed, the treating physician was contacted to determine the reason. A panel of three toxicologists reviewed these cases to determine if the recommendations were appropriate. Results: 400 patients were entered into the study over four months. Decontamination advice was not followed in 12%

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of cases. No difference was demonstrated in the rate of compliance for decontamination advice given for pediatric patients relative to adults ($p=0.9$). Decontamination advice given to community hospitals was more likely to be followed compared with university hospitals, but this difference did not reach statistical difference ($p=0.4$). Activated charcoal was much more likely to be given when recommended (compared with doing gastric lavage), although the difference did not quite reach statistical significance ($p=0.07$). The most common reason cited for not complying with decontamination advice was a sense that it was too late after the ingestion for decontamination to be of benefit. The three reviewing toxicologists felt that the recommendations made were appropriate in 92-98% of these cases, with inter-rater agreement greater than 90%. Conclusions: PCC decontamination advice is not followed by EDs in a significant minority of cases. When decontamination is felt to be critical to the patient's care, early follow up should be done. Future work will attempt to determine if adverse outcomes result from not following PCC decontamination advice.

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122 EFFICIENT DATA COLLECTION - BACK TO PAPER?

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Background: AAPCC certified regional poison control centers are required to submit case data to the TESS® database as a criterion for certification. Over the past several years, most certified centers have been moving away from paper "bubble-sheets" to on-line data collection systems. Questions about the efficiency of entry, poison specialist time, and smooth case follow-up have been repeatedly raised concerning current on-line systems. New technologies now allow acceptable handwriting recognition from scanned paper-based forms to serve as a means of rapid data collection. These technologies can be utilized to improve poison center efficiencies and case follow-up. Methods: A duplex scanner and handwriting recognition form software were obtained to begin this study. Forms were designed with the software to provide both "bubble" boxes and handwriting recognition areas required to meet TESS data collection requirements. The software was customized to export data read from the forms into the currently existing on-line database and to store an image of each form by case number on a CD-ROM. Evaluation of the new entry form and system was provided by current poison specialists and staff. Results: Evaluations of the new data entry/collection system by eight poison specialist over a one month period resulted in unanimous preference of the new paper-based system over the on-line data collection system that was previously used. Handwriting recognition accuracy increased during the one month evaluation period to the point where staff correction time decreased to an average of less than 30 seconds per case. Conclusions: An evaluation of any data collection system is largely subjective. Poison specialists in our center have constantly complained about the amount of typing they had to do and the time required, which decreased the number of calls they could handle. The new paper-based system eliminates SPI typing requirements and provides easy follow-up. In addition, the system automatically produces a case image stored on CD-ROM which allows the paper forms to be destroyed while keeping an exact copy of the written case for archival purposes.

123 AN ONLINE REFERENCE SYSTEM FOR THE POISON CENTER.

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Background: Over the past few years there has been tremendous interest in the collection of poison center data utilizing online and direct entry computerized systems. Although progress has been made in the ability of online systems to collect our data, there has been little concurrent movement towards computerizing the vital support systems (other than POISINDEX®) upon which the Center staff depend. We describe the development of an online reference system which places many of these needed materials on the SPI's workstation screen. Methods: The idea originated as a desire to place digitized primary literature at the fingertips of the SPI's for immediate fax transmission to clinicians or HCFs, and for maintenance of the health practitioner rotators' reading list in a form which would not disappear so readily. From this grew a system which places virtually all printed materials on the desktop screen. The system resources reside on a local area network, utilizing a standard web browser interface for access. Items not originally in electronic form are scanned utilizing standard software. The web-style motif of the system allows for easy access to resources. The browser interface will display graphics images directly or launch external viewers to accommodate many different file formats. Any image (e.g. scanned text, picture, slide format) or word processing file can be printed to laser or sent directly to the network fax system. The system enables rapid access to various materials such as; primary literature files, journal club articles, reference materials, program syllabi, health professional rotator readings, center statistics, quality improvement reports, meeting minutes, an electronic "FYI" section, a "photo gallery," the P&P/operations manual, and many other useful tools. Conclusion: The online reference system has taken the place of many traditional hard-copy methods of information transfer within the Poison Center. Among the advantages of the system are accessibility, reliability, and the ability to cross-reference materials. Linked

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with staff email, this system moves us significantly closer to the truly "paperless" Center. Critical evaluation of the system is ongoing.

124 TESS DOCUMENTATION: IMPROVEMENT THROUGH QUALITY MANAGEMENT.

Blume C, Thompson M, Scalzo A, Weber J. *Cardinal Glennon Regional Poison Center, St. Louis University Health Sciences Center, St. Louis, MO*

Objective: Documentation of poison calls is a responsibility of the Specialist in Poison Information (SPI). Our Quality Management Team examined the effectiveness of the TESS form in contributing to concise documentation. It became apparent in the course of audits that SPIs were experiencing difficulty in documenting information extracted during calls. The history, symptoms and calculations were scattered about the TESS form, and the assessment statement and management plans were often omitted or incomplete. Methods: It was noted that accurate documentation contributes significantly to the efficiency with which a case is managed. Weekly audits were randomly chosen according to medical outcome. We selected 100% of cases resulting in Major Effect or Death, 10% Moderate Effect, and 5% Minor or No Effect. Quality Control Auditors (AAPCC certified SPIs) evaluated 1327 records over five months. Audits considered ten specific criteria. Three categories were found to be deficient (<90% threshold) for the group. All but two SPIs had deficiencies in at least one criteria. Several SPIs had deficiencies in five of the ten criteria. In response, a new approach to documentation was developed which involved the addition of initials to designate key points: **H**=History, **S**=Symptoms, **C**=Calculations, **A**=Assessment and **P**=Plan of Management (**HSCAP**). We hypothesized that this addition would organize data collection and encourage more accurate documentation. Results: Results were reexamined for a five month period. This sample included 1594 audits. No areas were found deficient for the group and individual performance was drastically improved. Conclusion: With the institution of the **HSCAP**, staff quickly responded to the need for improved documentation. Staff reported liking the revised format and found that it helped them to organize their questions when extracting information initially and on follow up. Despite the organized structure of TESS, fine tuning of its format contributed to more concise and orderly documentation. We recommend the addition of the HSCAP initials to the TESS form.

125 TECHNOLOGY-FACILITATED RECOGNITION OF A POTENTIAL CHILD ENDANGERMENT CASE.

Morentz E, Keller KH. *California Poison Control System-San Francisco Division, University of California, San Francisco, School of Pharmacy, San Francisco, CA*

Background: In March, 1998, Poison Center staff became aware of a mother calling repeatedly regarding a variety of possible ingestions by her two children. Recent operational and data collection changes within the center permitted investigation into the frequency and nature of these calls in a way not practical a few years earlier. Methods: The center's computer case files were initially searched by mother's name and zip code, two identifiers most commonly obtained by all staff. Using the children's names, ages and city located additional cases. Inclusion of "Caller I.D." service on a newly installed phone system alerted staff to the existence of this repeat caller and increased case identification. Results: A total of 102 original cases were located from 1/1/97 through 3/30/98. Evaluation revealed a pattern of apparently escalating exposure risk, beginning with "My child touched a table cleaned with Windex," culminating in an incident where both children were evaluated by their pediatrician for possible acetaminophen ingestion from a baggie of tablets left in a closet. Raising concerns about the children's ultimate safety, poison center staff questioned their legal and moral obligation to report suspected child endangerment to local authorities. Group consultation (by phone) involving the caller, her husband, their pediatrician, poison center medical director and staff, determined the children were safe and successfully directed the caller into counseling. Eliminating her need for repeated calls benefited both the caller and the center. At an average of 8.35 minutes per case for management and documentation, based on a 1995 study, this sole caller represented a minimum of 14 hours of emergency services time prior to recognition of the underlying problem. Conclusion: Since repeat callers may contact the poison center at different times, speaking with different center personnel, they often go unnoticed. Technological innovations in data collection and communications systems at the poison center allowed the identification and successful resolution of a situation possibly involving child endangerment.

126 COST SAVINGS ASSOCIATED WITH A POISON CENTER / 911 TELEPHONE LINK.

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Objective: To characterize calls received by an AAPCC certified poison center (PC) via a dedicated 911 telephone line and the cost savings associated with this linkage. Methods: Records of 911 calls received by the PC from July 1996 through December 1997 were reviewed. Data collected included: substances, management site, therapies provided, hospital admissions, and outcomes. Cost savings was calculated based upon regional charges for pre-hospital transport ($205) and an emergency department visit ($269) multiplied by those cases managed on-site. Results: 656 exposure calls received via 911 were managed by the PC during the study period. Eighty percent (544/656) were managed on-site and 17% (112/656) were transported to a healthcare facility. Twenty-four cases resulted in an admission. Most commonly involved substances were: cleaning substances (97 calls), cosmetics and personal care products (57 calls), and analgesics (54 calls). Of interest, 7 calls involved silica gel and 12 involved mercury thermometers. Medical outcomes were: no effect 15.4% (101/656), minor effect 21.5% (141/656), moderate effect 3% (20/656), and not followed (judged as non-toxic or minimal clinical effects expected) 56.1% (368/656). Only 2 cases resulted in major effects and no deaths were reported. Twenty-four cases were lost to follow-up and in 22, the effect was classified as unrelated to the exposure. Calculated cost savings were $111,520.00 for prevention of pre-hospital transport and $146,455.00 for prevention of emergency department visits with a total savings of $257,975.00. Conclusions: Poisoning center impact on cost savings associated with on-site management is often discussed. Given that the calls involved in this study arrived directly via 911, a strong assumption can be made that in the absence of this link or in the absence of a PC, these patients would have been transported via ambulance to an emergency department. Therefore, a direct PC/911 telephone link can result in significant cost savings associated with emergency care of the poisoned patient.

127 PROSPECTIVE EVALUATION OF POISON CONTROL CENTER 911 INTERFACE FOR MANAGEMENT OF POISON EXPOSURES.

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Background: The CNYPCC and the Onondaga County 911 have developed a system of cooperative management of poison exposures called to 911. This system is expected to decrease the number of transports, and ED visits compared to a protocol of dispatching ambulances and transporting all poison exposure patients. Prospective analysis of this interaction will help define the ability of this system to manage poison exposures. Methods: Calls to the CNYPCC from 12/1/97-4/15/98 were included. Cases were prospectively entered into the study by the poison information specialist taking the call if the 911 dispatcher was identified as the source of the call. Data regarding the site of treatment, outcome of cases, incidence of ambulance dispatch, and incidence of patient transport to healthcare facilities, and disposition from emergency department including site of admission was collected. Results: 49 poison exposure cases called in by the 911 dispatcher were identified. On site management occurred in 35 (71.4%) of these cases, while 14 (28.6%) were managed at a healthcare facility. Of those cases managed at home 13 were non-toxic exposures, 18 were followed-up with no adverse events, and 4 were lost to follow up. Of those evaluated at a healthcare facility, 7 were treated and released, one was admitted to a critical care area, 1 was admitted to a non-critical care area, 2 were admitted to psychiatry, and 3 were lost to follow up. An ambulance was dispatched in 19 (38.7%) of the cases, of which fourteen were transported to a healthcare facility. No patient that was initially treated at home required transport to a healthcare facility at a later time. Conclusion: Cooperative management of poison exposure calls to 911 by EMS and poison control allowed for a large percentage of the calls to be managed without dispatch of an ambulance or transport to a medical facility without requiring later transport of any of the cases managed at home. This approach will lead to significantly decreased resource utilization.

128 911/EMS POISON CONTROL TOXIC EXPOSURE TRIAGE CALL PATTERNS AND THE COST-AVOIDANCE IMPACT OF POISON CONTROL CENTERS.

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Background: The benefit of poison center triage for 911/Emergency Medical Services (EMS) toxic exposure-related calls provides significant cost savings and/or avoidance for supporting government agencies, health care institutions and payers. Methods: We created an on-line 911/EMS patient flow encoding scheme integrated into our on-line patient management tool. The data system would automatically query the staff person for additional 911 patient flow information on all 911 related calls. Eleven categories were assigned and each 911 call was encoded to the appropriate category. 1,946 cases involving patients 6 years or older and 2,561 (4507 total) cases involving patients 5 years or younger were encoded (1997 calendar year). Each call was evaluated for EMS flow, reason, and outcome. Flow codes: 1) En route (necessary) (9.7%) 2) En route (unnecessary) (2.1%) 3) PCC no transport-transported (EMS Evaluation) (2.3%) 4) PCC no transport-transported (Patients desire) (1.4%) 5) PCC no transport-can go by private

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transportation (3.4%) 6) PCC no transport-NOT transported (60.5%) 7) PCC recommends immediate transport (15.7%) 8) PCC recommends calling 911 (3.8%) 9) Patient refuses transport (0.9%). Results: 71% of pediatric cases (<6yrs) and 46.8% of non-pediatric (>6yrs) were not sent to ED's based on Poison Center advice. Non-toxic outcomes were seen in over 97% of these cases. EMS referral against PCC advice was only 2.1% of all calls but was 1.5 times more likely in pediatric cases. When PCC recommendation lead to ED referral, the non-toxic outcomes were 70.9% in peds and 52.3% in non-peds with 15.7% and 33.4% unable to follow to known outcomes respectively. Average marginal cost savings are estimated to be up to $356,000 and estimated charges avoided are $2,124,000. Conclusions: Poison Center 911 call triage results in significant cost and charge avoidance with no increase in morbidity.

129 AUTOMATIVE ANSWERING SYSTEMS: GOOD OR BAD FOR POISON CENTERS?

Woodard-Jenkins JA, Woolf A. *Massachusetts Poison Control System, Boston, MA*

Background/Objectives: New credentialling criteria for AAPCC certification focus on improving the quality of care given to callers. How incoming calls are triaged and the use of automated answering service are ongoing sources of question and controversy. We wanted to conduct a survey to ascertain how many poison centers in the United States have these devices, how they're used in this setting and how they might affect accessibility of a trained specialist to the caller. Methods: All 77 US poison control centers (PCC) listed by the AAPCC were ascertained and sent a brief survey on automated answering systems (AAS). Eleven key questions covered the type and use of AAS. Results: All 77 poison centers completed the survey (100% compliance). Fifteen (19.5%) poison centers indicated that they utilized such a automative system; sixty-two (80.5%) answered they did not. Three of the fifteen centers who stated they only utilized automated call routing systems were not further analyzed. None of the 12 centers (15.6%) that had AAS had the same type. Total number of incoming telephone lines in these 12 PCC ranged from 4 to 24. All 12 had initial messages which varied in length: < 20 sec (6), < 60 sec (4), > 60 sec (2). 9 out of 12 had additional messages with lengths from < 20 sec (6), < 60 sec (1), >60 sec (2). Only 2 PCCs had a message in a language other than English; 3 out of 12 used music as a time filler. Only 3 PCCs had the option for the caller to elect to reach a SPI immediately for an "emergency" situation. Conclusion: Only 15.6% of US Poison centers utilize automated answering systems. For those that use an AAS it may pose barriers to: accessibility to the poison center's personnel, timely triage of emergency callers, or foreign-speaking callers. Future studies of AAS should include the sampling of customer satisfaction to ensure quality of service and accessibility to all callers.

130 HAZARDOUS MATERIALS INCIDENTS: AN INDUSTRIAL COMPARISON.

Burgess JL, Kovalchick D, Kyes K, Harter L, Thompson J. *University of Arizona Prevention Center, Tucson AZ; University of Washington, Seattle WA; Washington State Department of Health, Olympia WA*

Background: There is very little research available identifying industries at high risk for Hazardous Materials (Hazmat) incidents. This information could help fire departments and industries prepare for and potentially prevent such incidents. Methods: A retrospective review was conducted on Hazmat incidents from 1993 to 1995 collected by Washington State Department of Health in a program supported by the Agency for Toxic Substances and Disease Registry. The program excludes incidents with petroleum products exclusively. Some of the criteria for incident inclusion have changed over time, and reporting differences have occurred within industries. Using Standard Industrial Codes (SIC), the number of Hazmat events per industry were ranked. Annual US Census data were used to determine the number of facilities and employees per SIC in the state of Washington. SICs with the most incidents per 10,000 employees were ranked and characterized by type of releases. Fifteen industries were not ranked because they did not have SIC information. Industries with only 1 event during the 3 year period were considered "chance events" and were not ranked. Results: Over three years, 1373 incidents were recorded, with 317 involving human victims. Industries with the greatest number of reported Hazmat incidents were trucking service (115), pulp, paper, and paperboard mills (110), electric light and power (75), petroleum refining (64), and sanitary services (49). However, industries with the greatest number of incidents per 10,000 employees were agricultural chemicals (146/10,000), petroleum refining (124/10,000), electric light and power (64/10,000), industrial and miscellaneous chemicals (54/10,000), and pulp, paper, and paperboard mills (40/10,000). Between SIC the specific chemicals released in the Hazmat incidents varied. Within SIC, certain chemicals were consistently released each year while the presence of other chemicals varied. Conclusion: Controlling for number of employees permits more practical comparison of Hazmat incidents by industry. Industries with high incident rates should continue to develop methods of preventing accidental Hazmat releases.

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