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Poster Session 1 — Friday, October 1 — 10:00 am–4:00 pm
Abstracts #1–#56

1 HEALTH CARE PRACTITIONER SATISFACTION WITH POISON CENTER CONSULTATION SERVICE.

McCormick MA, Fish SS, Bayer MJ. *Connecticut Poison Control Center, Farmington, CT*

Objective: Our training program uses senior emergency medicine residents (SEMRs) as toxicology consultants, with the back-up of the physician toxicologist on-call. We assessed health care practitioner (HCP) satisfaction with this toxicology consulting service. Methods: For 5 months, HCPs who spoke to a toxicology consultant (SEMR or attending on-call toxicologist) were contacted by telephone and a 12 question survey was implemented. Results: 48 toxicology consultant cases were identified. 9 HCPs did not respond to initial or 3 follow-up calls. 3 surveys were eliminated because they were incomplete. Of the 36 completed surveys, 21 used the SEMR as consultant, and 15 used attending toxicologists. 92% (33/36) felt the response time of the consultant was very quick and 100% felt that advice given was relevant to the poisoning case. 94% (34/36) followed the advice given by the consultant. 83% (30/36) felt that his/her knowledge base was expanded by discussion with the consultant. 94% (34/36) felt that all of his/her questions were answered. 92% (33/36) said they could access the consultant easily if needed again. 80% (29/36) had used the service before, and 100% stated they would use it again. When asked to rate the service on a scale of 1–5 (5 = extremely valuable), the median score was 5 (range 4–5). When asked for comments, 13 had no comments, 3 had negative comments, 6 had suggestions for improvement, and the remaining 14 had praise for the service. When SEMR survey results were compared to those of the toxicologist, no differences were noted. Conclusion: The poison center consultation service meets with high satisfaction and is well received. Use of SEMRs as toxicology consultants in the poison center compared favorably to the use of attending toxicologists.

2 TARGETED GROUP EDUCATION AS A MEANS OF PUBLICIZING POISON CENTER SERVICES.

Herrington LF, Garrettson LK. *Georgia Poison Center, Atlanta, GA*

Objective: Numerous methods may be used to teach potential clients about poison center (PC) services. Measuring the impact of PC public relations (PR) and educational activities is difficult. This study was conducted to identify correlations between the lecture schedule of a single PC toxicologist discussing drug therapy during human lactation and breastfeeding information calls (BFIC) received by this regional PC. Methods: All (BFIC) were tagged with a special code. Coded calls were entered into a data base for analysis by date and location. A time line was created correlating lecture timing and location with monthly call rates by date and state locations. Results: Between 1/21/92 and 3/31/99, 24 lectures were given to both medical and lay audiences, 3 out of state. Over the same time period, 8,241 BFIC were received, 2/3 from non-medical callers. BFIC increased from 2–275/mo. (0.23–20.84% of all info calls). There was a > 4000% increase in BFIC between 1992–1998 (0.5–203% annually), compared to a 29% increase in non-BFIC. Increase call rates followed lectures temporally. Of 165 BFIC from out of state, 64% originated in 4 surrounding states. Calls from a neighboring state rose 178% following a single lecture to a large state-wide audience. Conclusion: While it is difficult to correlate PC educational and PR activities to increases in workload, this regional PC had no other program promoting

itself as a reference for medication use and breastfeeding. We report a significant increase in a specific type of PC call (BFIC) that followed presentations on this specific topic.

3 A PROGRAM TO PREVENT IRON POISONING UTILIZING PUBLIC HEALTH NURSES IN A COUNTY HEALTH DEPARTMENT.

Broderick M, Dodd-Butera T, Wahl P. *California Poison Control System (San Diego Division) and San Diego State University, San Diego, CA*

Background: Despite 50 years of educational and legislative efforts aimed at increasing public awareness of the toxicity of iron, it remains the leading cause of poisoning deaths in children under the age of 5. This study addressed the issue of preventing iron poisoning through an educational program developed for Public Health Nurses (PHNs) in the local county health department. PHNs were chosen as study subjects because of their contact with patients prescribed iron supplements during the perinatal period. Methods: Six educational programs were held at local public health departments throughout the county. A convenience sample of 70 attended. The programs consisted of a pre-test, lecture, post-test, and evaluation. In addition, a follow-up survey was conducted at 8 months post-training in order to determine the application of iron poison prevention principles. Education and evaluation of subjects were based on behavioral and cognitive approaches to iron poison prevention. Results: Prior to attending the program, 60% of the PHNs reported storage habits of medication that predispose children to poisoning. Further, 71% of the attendees were unable to identify iron as one of the three most dangerous products from a list of six hazardous household substances. The overall score in the pretest was 56% compared to the mean post-test score of 96%. Of the 45 respondents to the follow-up survey, 87% had utilized the program information to educate patients. In addition, 84% of the participants educated a family member on the dangers of iron poisoning. Conclusions: This Iron Poison Prevention Program provided a successful educational program for PHNs that was then applied to patient education. This program represented a unique interagency partnership for disseminating vital poison prevention to health professionals and can serve as a model for public health departments throughout the country.

4 USING PUBLIC LIBRARIES TO DISSEMINATE POISON PREVENTION MESSAGE.

Cropley J, Gibson J, Christoff D, Bobbink S, Robertson WO. *Washington Poison Center, Seattle, WA*

Objective: To determine a simple and cost-effective way for a regional poison center's public education department to reach a state population with information concerning National Poison Prevention Week and year-round poison center services. Method: We designed four bookmarks each with a different poison prevention message. At the beginning of February we distributed 200 bookmarks, along with one National Poison Prevention Week poster to each public library in the state. In early May we mailed each library a questionnaire to (1) determine how many distributed and posted our materials, and (2) obtain feedback on the librarians' perceptions of the project's effectiveness. Results: Fifty-one percent (167) of 326 libraries responded to our questionnaire. Of the respondents, 94% (157) reported the bookmarks had been distributed to patrons during National Poison Prevention Week. Ninety-one percent (153) felt the bookmarks were an effective way of letting people know about both the poison center and National Poison Prevention Week. Forty-three percent (72) thought too many bookmarks had been mailed. Forty-four percent (74) thought the number of bookmarks had been sufficient and 8% (14) thought the number had been too small. Sixty-three percent (106) had displayed the poster on one of their bulletin boards and 28% (47) had not. Ninety-seven percent (163) noted that they would like to receive similar materials in the future and 3% (4) indicated they would not. Based on our daily statistics, there had been an actual increase in the number of calls to the poison center during the month of March, including more than the expected number of requests to our public education department for materials and information. Conclusion: Distribution of bookmarks via the public library system appeared to be a successful way to both educate and inform the public about National Poison Prevention Week and the availability of our statewide poison center and its services.

5 DECREASING ACCIDENTAL POISONINGS THROUGH EFFECTIVE EDUCATION.

Brogan H, Lobell D. *The Poison Control Center, Philadelphia, PA*

Background: When reading the AAPCC certification criteria for public education, there is no requirement to measure the quality or success of educational efforts. One theoretically could relate "increased penetrance" within a center's region as an effectiveness measurement tool, although no verification of "penetrance" validity can be found. Theoretically, an organized effective education program will over the long run decrease penetrance and educational efforts can

be measured. The Philadelphia Poison Center's Health Educator visits schools and teaches a 30-minute program for children ages three to six that includes: stories, activities, and each child is given a packet to take home for their caregivers to reinforce the lesson in the home. A study was designed to analyze the children's comprehension and short-term retention of the information taught to them by the Health Educator at the end of the 30-minute classroom experience and after a seven-month period. Method: A pre-test consisting of 10 questions was presented to 493 children between the ages of three and six. At the end of each presentation, a post-test with the same questions as the pre-test was given. Also, a post-test consisting of the same questions was presented to 368 children who had been taught the Program seven months before. Results: Out of the 493 children who were given the pre-test, 36% answered all 10 questions correctly. At the end of the 30-minute presentation when the post-test was given, 96% of them answered the same questions correctly. Out of the 368 children who were taught the Program seven months earlier, 95% answered all the post-test questions correctly. Conclusion: According to the results of the tests, children clearly understand and are able to retain the information that was taught to them over a period of time. This study proves that educating children works and can theoretically reduce the number of accidental poisonings.

6 EDUCATION AND FUND RAISING COMBINED IN ARIZONA RATTLERS PROGRAM.

Krueger AM. *Samaritan Regional Poison Center, Phoenix, AZ*

Background: Poison centers are constantly challenged to provide education and prevention activities as well as raising funding to sustain program components. This joint program created a unique program that combined poison prevention education and fundraising with the community relations program of a professional sports team. Methods: The poison center began an affiliation with the Arena Football League local franchise in 1993 during their first season. It was felt that a team with the name ''Arizona Rattlers'' was a good match with the local poison center. The affiliation began first with the ''Extra Point for the Poison Center'' program and expanded over the years into a combination of educational presentations at local schools while at the same time fundraising at games and special events. The poison center was designated as the sole charity of the team and over the next five years a solid cooperative program was developed. Results: Over the length of the program more than 45,000 kids were exposed to education about venomous creatures of the Arizona desert. The educational programs were presented by the Rattler players and a representative of the poison center. The community outreach program earned the team national recognition by the Arena Football League. Over $300,000 has been raised to benefit the public education programs of the poison center. Conclusion: The success of this program can serve as a model for other poison centers to develop joint programs with other professional sports organizations.

7 SURVEY OF STATE LEGISLATION FOR POISON CONTROL CENTERS.

Kearney T, Heard S. *California Poison Control System, University of California, San Francisco, CA*

Background: State legislation directs many facets of PCC operations in the US. Trends in state legislation may forecast and influence future public policy and funding of PCCs. The purpose of this study was to perform a cross-sectional survey of US state legislative bills from the 1990's that mentioned PCCs. Methods: A key word search was conducted on the Lexis-Nexis database (Dayton, OH) from 1991 to 1998 for state legislative bills mentioning PCCs throughout the US. The search strategy included the word grouping of ''poison'' within 5 words of ''center'', ''control'', or ''information'' to locate defeated and enacted bills by year and by state. Results: State legislative bills mentioning PCCs were revealed for 37 states. Thirty-four states had enacted legislation. Enacted bills that appropriated funds for PCC operations were the most common type of legislation and included 26 states. Of these, 8 states had enacted bills with a dedicated fund source for PCC operations (e.g. telephone surcharge, hospital fee, and reimbursement). Ten states had defeated bills to establish a dedicated funding source for PCC operations. Twenty-two states had enacted other types of bills encompassing a range of PCC-related issues (e.g. statewide systems, pesticides, protocols, indemnification, product disclosure). The annual frequency of enacted bills increased in the late 1990s. Conclusions: Although the majority of states have enacted bills that mention the appropriation of state general funds to PCCs, the amounts were variable and inconsistent by year and state, and subject to line-item vetoes. Few states have enacted bills to provide a dedicated funding source for PCCs. There was an increasing trend of enacted state legislative bills mentioning PCCs for a variety of issues in the 1990s.

8 FINANCIAL REVIEW OF A YOUNG CLINICAL TOXICOLOGY SERVICE.

Eng J, Miller MB. *Ingham Regional Medical Center and Sparrow Hospital, Lansing, MI*

Background: The financial performance for the practice of clinical toxicology is modest. A clinical toxicologist may be affiliated with a Poison Control Center (PCC) or a fellowship training program and receive financial support. Many toxicologists also volunteer time to the clinical practice. We would like to present data regarding patient demographics and financial performance from our toxicology service established approximately two and a half years ago. Case Report: The toxicology service was established in August 1996 with practicing emergency physicians forming a private consultation and admitting toxicology service not affiliated with a PCC. Consulting privileges were obtained at two area hospitals with admitting privileges at one of these hospitals. Patients for occupational exposures are occasionally seen in an outpatient clinic. Results: For the period August 1996 through March 1999, 275 patients were seen. Of these, 209 were adults (>18 yo) and 66 were children. Two hundred sixty-two patients were seen in consultation and 13 patients were admitted to the clinical toxicology service. As of March 1999, the total amount billed was $117,009.00 with collections equaling $31,295.87. To date, the aging amount is $35,578.14 with an adjusted amount of $50,134.99. Our total expenses equal $6,629.58 with a remaining net income of $24,666.29. Conclusion: Our clinical toxicology service is small but already it shows evidence of growth. Although the net income our service generated thus far cannot support all of our personal finances, we hope to see continued growth and return. This report serves as an example that establishing a clinical toxicology practice independent of a PCC is possible; however, its development may take time.

9 THE UTILIZATION OF EMERGENCY DEPARTMENT OBSERVATION UNITS FOR THE POISONED PATIENT.

Dribben W, Welch J, Dunn D, Kirk M. *Indiana University School of Medicine, Indianapolis, IN*

Background: Only 5% of poison exposures have toxic effects serious enough to admit to the hospital. In addition, only 10–30% of those hospitalized patients required specific treatments or antidotes. Many variables exist for poisonings that cloud management issues and patients are frequently admitted, particularly to critical care units, for observation. Admission to the critical care unit for observation is an expense that has contributed to the escalation of health care costs. Emergency department observation units have been studied for a variety of conditions, but no studies describe their use for the observation of the poisoned patient. Methods: A retrospective study describing the volume and outcome of poisoned patients admitted to our observation unit by our toxicology admitting/consultation service during the previous 10 months. Results: From June 1998 to March 1999 there were a total of 1932 patients admitted to our observation unit. Poisoned patients ranked 5th on the list of diagnoses accounting for 135 (7.0%) of the total admissions. The majority of patients were asymptomatic or exhibited signs of mild to moderate toxicity due to a wide variety of exposures. Most patients were either discharged home or transferred to psychiatry. A total of 8 patients (0.41%) required admission for further medical care. There were no complications during this period. Conclusions: In our experience to date, the observation unit has been a valuable resource for safely managing poisoned patients and reducing unnecessary hospitalizations. Further prospective studies are needed to provide a valid foundation for developing predictive factors and clinical guidelines to identify toxic patients that can be appropriately managed in an observation unit. In addition, studies analyzing cost savings by utilizing observation units in lieu of ward or critical care beds should be examined.

10 UTILIZATION OF AN EMERGENCY DEPARTMENT OBSERVATION UNIT FOR ACUTE INTOXICATIONS.

Gummin DD, Butler JR, Roberts RR, Erickson TB. *Cook County Hospital, Toxikon Consortium, The University of Illinois at Chicago, Chicago, IL*

Objective: To review the effectiveness of short-term observation in an emergency department (ED) observation unit for patients presenting with acute intoxications. Methods: Retrospective chart review of consecutive adults presenting to an urban ED with known or suspected toxic presentations, who were subsequently observed for up to 24 hours in an observation unit. Admission to the observation unit was determined by specific eligibility criteria. This group was compared with a control group of patients who also met criteria for observation unit admission but who were admitted to the hospital because the unit was closed. Results: Eighty-nine acutely intoxicated patients admitted to the observation unit over a one-year period served as the primary study group; eight patients were identified as hospitalized controls. Seven patients (8%) failed observation and required admission to the hospital. Mean length of stay for patients discharged from the unit was 15.1 hours vs 38.8 hours for hospitalized controls. Eighty-eight percent of observation unit patients

were discharged within 24 hours, compared with only 3 of 8 hospitalized controls. Despite eligibility criteria constraining use of the unit for isolated ethanol ingestion, ethanol was the most common intoxicant, followed by anticonvulsants, multiple ingestions, and unknown ingestions. Four of the seven patients who failed observation and required admission did so for delayed metabolism of phenytoin. Follow-up was obtained in 66%. No mortality or major morbidity was identified. Conclusions: The observation unit successfully discharged patients within 24 hours in 88% of cases with low failure rate, low morbidity and no mortality. Length of stay was decreased. ED observation units appear to be an efficacious alternative means of monitoring acutely overdosed patients. More long-term prospective studies are required to explore cost-effectiveness.

11 THE IMPACT OF DEMOGRAPHICS AND PRACTICE PATTERNS ON MEDICAL TOXICOLOGY BILLING PRACTICES.

Wax P. *University of Rochester Medical Center, Rochester, NY*

Objective: To investigate demographics and practice patterns that may impact medical toxicology billing practices. Methods: Data from a recent written survey to ACMT members regarding workforce issues in medical toxicology was analyzed with respect to how the demographics and practice patterns of medical toxicologists may impact their billing practices as they pertained to 2 patient scenarios: inpatient evaluation of a hypotensive CCB overdose and outpatient evaluation of steelworker presenting with multisystem complaints. Billing practices were assessed with regard to geographic location, primary specialty, and % of professional time devoted to medical toxicology. Results: 150 of 236 ACMT members completed the survey. 133 of 150 are currently practicing medical toxicology. Mean inpatient bill was Western States $628 (collected $354), Midwest $309 ($190), Northeast $270 ($127), and South $188 ($138). Mean outpatient bill was West $768 ($697), Midwest $281 ($181), Northeast $243 ($179), and South $125 ($75). Regarding primary specialty: mean inpatient bill was EM $427 (collected $235), Peds $157 ($85), and OM $650 ($463). Mean outpatient bill was EM $421 ($355), Peds $149 ($96), and OM $908 ($875). Those with 100% Med Tox practice had mean inpatient bill of $1160 (collected $600), 50–99% Med Tox practice $354 ($190), and <50% Med Tox practice $272 ($180). Mean outpatient bill for 100% Med Tox practice was $847 ($847), 50–99% Med Tox practice was $276 ($206), and those with <50% Med Tox practice was $267 ($203). Conclusions: These billing scenarios suggest that medical toxicologists from the West bill more and collect more than in other parts of the U.S. Those from OM bill and collect the most revenue, and those from pediatrics bill and collect the least. Billings and collections appear to be significantly greater for full-time medical toxicologists than those who practice toxicology part-time. Collection to billing ratio is best for outpatient consultations, especially for full-time and OM medical toxicologists.

12 A DESCRIPTION OF CONTINUING EDUCATION (CE) FOR SPECIALISTS IN POISON INFORMATION IN US POISON CENTERS.

Fishman C, Cobaugh D, Ciancaglini P, Wax P, Lawrence R. *Finger Lakes Regional Poison & Drug Information Center, University of Rochester Medical Center, Rochester, NY*

Background: Specialists in Poison Information (SPIs) in U.S. poison centers obtain CE through a combination of internal and external programs. The purpose of this study was to describe existing educational programs and to characterize the CE needs of SPIs. Methods: A 13-item self administered survey was mailed to the managing directors of 72 US poison centers and a 17-item self administered survey was mailed to 948 SPIs. Only one mailing was completed for each survey. Results: Completed surveys were received from 65/72 (90%) centers. A mean 13.6 hours (range 0–150 hours) of CE were provided per year by the centers. 21/65 (32%) centers indicated that CE is not required for SPIs. 51/65 (79%) of the centers indicated that formal evaluation methods are not included in their CE programs and 49/64 (75%) indicated that CE credits are not available for poison center based CE. The return rate for the SPI needs assessment survey was 430/948 (45%). SPI demographics were: RN 252/430 (59%), RPh 170/430 (40%), MD 5/430 (1%), other 3/430. Mean length of poison center experience was 7 years and 336/430 (78%) of the respondents were CSPIs. Respondents reported a mean of 38.9 hours per year are spent on CE. 133/430 (30.9%) of respondents indicated that attendance at a poison center based conference is not required for their job. 68/430 (15.8%) of respondents indicated that poison center based conferences are not provided. 369/430 (86%) believed that CE was very important. Both the SPIs and the centers identified mushrooms, drugs of abuse, herbals and heavy metals as preferred CE topics. Conclusion: SPIs regard CE as very important to their professional practice. There is inconsistency across US poison centers in provision of CE.

13 EVALUATION OF AN ELECTRONIC CONTINUING EDUCATION (CE) PROGRAM FOR SPECIALISTS IN POISON INFORMATION.

Ciancaglini P, Cobaugh D, Wax P, Lawrence R. *Finger Lakes Regional Poison & Drug Information Center, University of Rochester Medical Center, Rochester, NY*

Background: A regional AAPCC certified poison center developed an electronic CE program for Specialists in Poison Information (SPIs). The program was distributed to participants via one of three electronic mediums including the www, E-mail, and fax. Participants chose the most appropriate electronic medium for delivery of their CE modules. Each two-week module provided information on a toxicology-related topic, in a brief question/answer format followed by a discussion of the answer. Following each module, participants were asked to respond to a test question and a satisfaction survey. Thirteen modules were completed at the time of analysis. The purpose of this analysis was to describe the participants' response to the program. Methods: 948 SPIs from 72 poison centers were included in the educational program. Responses to the satisfaction survey were to be indicated by marking strongly agree, agree, disagree, or strongly disagree, and were then to be submitted via fax or E-mail along with the completed test question. Results: As of the completion of the 13th module, 2,134 visitors logged onto the website. 553 responses to test questions and 456 satisfaction surveys have been received. Response to individual test questions ranged from 7.7% (73/948) for Module I (AACT/EAPCCCT Gastric Decontamination Guidelines) to 2.0% (19/948) for Module 13 (Herbal/Dietary Supplements Part II). Response rate steadily declined over the duration of the program. 97% agreed that the activity met its stated objectives. 95% agreed that the content was at an appropriate level for the audience. 97% said they would apply the information/skills learned from this activity to their clinical practice. 90% agreed that the overall quality of the activity was excellent. Conclusion: Although responders were generally satisfied with the activity, the response rate for both the test questions and satisfaction surveys was much lower than anticipated. This analysis was limited because we were unable to determine the number of participants who completed the module but elected not to respond.

14 ESTABLISHING CLINICAL TREATMENT GUIDELINES FOR A STATE-WIDE POISON SYSTEM USING CONSENSUS.

Alsop J, Heard S, Albertson T, Ekins B, Clark R. *California Poison Control System, Sacramento, CA*

Background: When multiple sites consolidate into a single system, differences in triaging and treatment recommendations can cause conflict among staff at other sites. This results in confusion to patients and health care professionals who are on the receiving end of conflicting and differing recommendations. Developing uniform and consistent guidelines for triage and treatment was seen as an essential need of the system. Methods: Medical Directors, Managers and Staff were queried for input on topics requiring attention. Problem issues generating the most controversy were addressed first. The Managers researched the topics to find toxicity and evidence-based outcome data from the published literature. Outcome information from statewide and national databases was also examined. Past clinical experience from the Manager and Medical Director group was pooled and included. Drafts of the guidelines were widely circulated across the system by E-mail for review and comment. Staff was also invited to participate in the process if they desired. Consensus decisions were decided in advance. When making a choice was difficult, it was agreed that adjustments and compromises would be made until a consensus agreement resulted. The last Manager draft was reviewed, discussed and agreed upon by the Medical Directors. The final documents were signed by the Executive Medical Director and Executive Administrative Director prior to implementation with the staff. Results: Cooperation by using consensus resulted in development of a Statewide Provider Triage List, Provider Treatment Management Guidelines, SPI Send-In Triage Guidelines, and CSPI Clinical Management Treatment Guidelines. Conclusion: Buy-in and cooperation by all members of all groups, including the staff, were seen as the essential elements in the successful development and implementation of guidelines.

15 PARENT/CAREGIVER COMPLIANCE WITH POISON CONTROL ADVICE.

Yamada J, DiRezze P, McGuigan M. *Ontario Regional Poison Centre, Toronto, Canada*

Background: Measuring the effectiveness of the services provided by poison control centres (PCC) can be achieved by evaluating the level of compliance of callers with the advice provided by poison control specialists. The purpose of this study was to assess compliance of parents/caregivers, whose children (ages 5 years and under) had accidentally ingested an antihistamine and/or decongestant. Methods: Using a retrospective chart review, a convenience sample of all eligible calls was retrieved during the months of January and February 1999. Calls were excluded if they included

other co-ingested products. The sample was divided into two groups: those which were considered toxic by a poison control specialist and required treatment in hospital and those which were considered non-toxic and did not require treatment. Compliance with PCC advice was measured by a follow-up telephone call made by a research assistant within seven days of the initial call. Results: A total of 147 calls were retrieved and followed. One hundred and seven (73%) callers were advised that no treatment was required and of these, 100 (93%) followed PCC advice. Forty (27%) callers were advised to take their child to the emergency department and of these, 39 (97%) complied with the advice. There was no statistically significant difference in compliance rates between the two groups, $\chi^2=0.3058$ df=1 p=0.58. High compliance rates in this sample of callers to a major PCC indicate that a sample of 147 was adequate. Reasons for non-compliance included caregiver anxiety and the need for a second opinion. Conclusions: In this group of callers, compliance rates for pediatric ingestions were high. Future studies that include other age groups, exposures and types of callers such as health care professionals will provide further support for compliance with PCC advice.

16 POISON CENTER ROTATION AS PART OF NURSING SCHOOL CURRICULUM.

Caros L, McCormick M, Bayer M. *Connecticut Poison Control Center, Farmington, CT*

Objective: To broaden their understanding and appropriate use of the poison center, a university-affiliated nursing school incorporated an orientation to the poison center as part of their senior year community health curriculum. Methods: An 11-hour, two-day program was designed by Specialists in Poison Information. This program incorporates lecture, video and hands-on computer exploration. On the first day students are placed in a classroom setting and a five question pre-test is given. A Certified Specialist then presents a brief history of the poison center, staffing patterns and educational requirements for employment, and a discussion of call sources/volume. A one-hour household products ''show and tell'' follows, then two poison prevention videos are shown. The next hour includes a review of information resources, triage, evaluation and management methodologies, and medical record/data collection form requirements. There is strong emphasis throughout on the ''managed care'' aspect of the center. Finally, the toxicity of acetaminophen, salicylates, iron and cough and cold preparations are reviewed; this includes a discussion of decontamination options. The first day concludes with the students exploring Micromedex and several ''surprise'' toxins. A post-test is given and a course evaluation is completed. The students return to the poison center for four hours during an evening shift; at this time students observe, listen to calls, and ask questions as time permits. Results: Both student and faculty response to this program has been enthusiastic. It is considered a valuable experience, with 87.5% of the 24 students rating it ''excellent''. Post-test scores improved an average of 42.5%. Students bring their knowledge into the clinical area and are more likely to access the poison center in the future. Conclusion: The program has become an integral component of the nursing school curriculum, and enhances our relationship with users of our service.

17 GEOGRAPHIC DISTRIBUTION OF HOSPITALS AND THEIR IMPACT ON POISON CENTER PENETRANCE.

Sudakin D, Horowitz BZ. *Oregon Poison Center, Portland, OR*

Background: Penetrance is a measurement assumed to reflect public awareness of poison center services. The purpose of this investigation was to utilize geographic information systems (GIS) to evaluate how the geographic distribution of hospitals affects measurements of poison center penetrance. Methods: All human exposure cases reported to the regional poison center from a single state during 1998 were analyzed using ArcView GIS. Penetrance was calculated at the county level as the number of human exposure cases/1,000 population using 1997 population estimates as the denominator. Two measurements of penetrance were calculated for each county, one including all exposure cases and the other excluding cases originating from health care facilities. Differences in average penetrance measurements using both calculation methods were analyzed statistically and visually utilizing maps which included hospital locations within counties. Results: 34,032 of 37,941 (90%) human exposure cases originated from sites that were not health care facilities. In counties with hospitals within their boundaries (n = 31), the exclusion of calls originating from health care facilities resulted in a statistically significant decrease in penetrance from 11.1 cases/1000 to 9.5 cases/1000 (paired *t*-test; t = 10.4, $p < .001$). When analyzed visually, the exclusion of calls from health care facilities resulted in less variation in penetrance measurements between counties, with most counties (92%) ranging between 7–12 cases/1000. Penetrance measurements in counties without hospitals (n = 5) were not significantly affected after eliminating calls from health care facilities. Including all call sites, the average penetrance in counties with hospitals (11.1 cases/1,000) was higher than counties without hospitals (10.0 cases/1,000) although the differences were not statistically significant. Conclusions:

Human exposures reported from hospitals contribute significantly to measures of poison center penetrance. Adjusted calculations of penetrance, which take into account the location of hospitals within geographic areas served by regional poison centers, may provide a more valid measure of general public awareness of poison center services.

18 GEOGRAPHIC INFORMATION SYSTEMS AND POISON CENTER MANAGEMENT.

Sudakin D, Horowitz BZ. *Oregon Poison Center, Portland, OR*

Background: Geographic Information Systems (GIS) refer to the automated capture, analysis, and display of spatial data. The purpose of this investigation was to use GIS to calculate poison center penetrance by zip code and county for a single state, and explore the ability of GIS to identify demographic and other features within geographic areas with low penetrance as potential targets for interventions to increase awareness of poison center services. Methods: All human exposure calls to the regional poison center during 1998 were included for analysis by ArcView GIS. Spatial elements included the zip code and county origin of the call (including detailed population demographic data), and potential sites of primary prevention (schools). Penetrance (human exposure calls/1,000 population) was calculated using both 1990 census data and 1997 population estimates as denominators to account for recent trends in population growth. Maps displaying penetrance measurements and potential sites of intervention were constructed for analysis. Results: 37,628 of 37,986 (99%) human exposure calls during 1998 were successfully geocoded. Average penetrance by county was 12.3 human exposure calls/1,000 population using 1990 census data, and 10.9 calls/1000 population using 1997 population estimates. One of 36 (3%) counties was identified as having low penetrance (<7 human exposures/1,000). Average penetrance by zip code was 11.1 calls/1000 population, 128/439 (29%) zip codes were in the low penetrance category. There was remarkable variation in penetrance between neighboring zip codes when displayed geographically, despite similar population demographics. A total of 304,625 people, including 19,681 children under the age of 5 were located in zip codes with low penetrance. 239 schools were identified within zip codes with low penetrance, which could potentially serve as sites of intervention. Conclusions: GIS can serve as a useful tool for poison centers to measure penetrance, display regional variation in penetrance, and increase understanding of population demographics that may assist in planning preventive interventions.

19 UTILIZATION OF CALLER ID TECHNOLOGY AMONG CERTIFIED POISON CENTERS.

Rose R, Carper B, Waring E, Holstege C, Cisek J. *Virginia Poison Center, Richmond, VA*

Background: The proliferation of telecommunications products and services in recent years has likely resulted in significant variation among systems used by poison centers (PC). Since information obtained via telecommunications is critical to PCs for clinical, statistical and quality assurance purposes, this study was designed to determine the prevalence and utilization of caller ID (CID) technology among certified poison centers. Methods: A one-page survey was sent to Directors of all AAPCC-certified poison centers. Initial questions addressed the use of a call sequencer, availability of CID and voice recording. Additional questions addressed the utilization of callers' phone numbers obtained via CID. Results: 47 (87%) of 53 surveys were returned. Sixteen (34%) centers utilized some type of call sequencer, thirty-one (66%) centers recorded emergency calls, and twenty-three (49%) used some type of CID. About half the centers with CID had automatic number identification, but no center had enhanced 911 capability. Two centers with CID capability did not use it. The presence or absence of CID did not appear to correspond to either call volume or method of capturing caller location. Eleven (48%) of 23 centers with CID reveal the presence of CID to callers, yet half of the centers who do not acknowledge the presence of CID stated that they (verbally) attempt to verify CID-obtained numbers. 91% of centers with CID ask callers for their phone number, 73% ask callers to verify CID numbers, and 55% attempt to reconcile differences between numbers obtained verbally with those obtained via CID. Less than 1/4 of centers with CID indicate in the call record how callers' numbers are obtained, but the majority use CID-obtained numbers to make follow-up calls (76%) or to call 911 if necessary (86%). Only one (4%) of 23 centers with CID have a written policy to guide staff on use of numbers obtained through CID. Conclusion: About half of U.S. certified poison centers have the ability to display callers' phone numbers. There appears to be no consistency in the utilization of CID data, and at least 2 centers that possess the technology don't use it. As the possibility of a national 800 number approaches, poison centers should work collaboratively to develop consistent guidelines on the use of caller ID information.

20 DEVELOPMENT OF A POISON CENTER SYSTEM INTRANET. THE NEXT STEP TOWARD A "PAPERLESS" POISON CENTER.

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Background: In January 1997, four poison centers in California were consolidated into the California Poison Control System serving more than 33 million citizens. The System operates 4 answering sites, using a single 800 number with 800 call routing, a network automatic call direction (ACD) telephone system and wide area networked use of the Dotlab® program for collecting patient case information. Standardized guidelines and protocols have been developed to improve consistency of information provided to users as primary and followup calls can be routed to any of the 4 sites depending on workload and staffing at each site. These were initially produced and distributed on paper but this was found to be extremely inefficient and difficult for staff to remember to use. Methods: A system-wide Intranet was developed to improve the use and dissemination of information within the System. The Intranet resides on a main System server and is linked to the divisions' servers by standard Internet protocol via a frame relay. Each workstation in the System has access to the Intranet using a standard Internet browser. The Intranet contains all of the System guidelines for poison information specialists, poison information provider protocols, links between the guidelines and the 3rd edition of the Poisoning & Drug Overdose Handbook written by System staff, staff directories, resource manuals with frequently called telephone numbers, announcements, cases used in the bimonthly system-wide case conference and other useful information. A demonstration of the Intranet will be provided.

21 CRITICAL INCIDENT ANALYSIS: A DECADE'S EXPERIENCES.

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Background: In the late '80s, our poison center initiated its "Critical Incident Studies" (CIS) to provide yet another needs assessment for CE functions. As we reported in 1995, the process proved effective. We sought to reassess its value as we adopted Toxicall®–electronic documentation. Method: At the end of each shift, each staff member was asked to note what–if any–"gnawing question or concern" remained with him or her. Group totals were tallied, categorized and carefully reviewed. Results: The 222 responses collected pre- and post-Toxicall were compared to prior categorizations as follows:

	1987 % (N = 330)	1994 % (N = 93)	1997 % (N = 222)
1. Treatment Issues	46	73	26
2. Administrative Matters	25	16	35
3. Facility Problems	15	2	8
4. Personal Frustrations	14	9	31

As we had first reported in 1987, staff continued to be most cooperative. In the '80s, staff members were particularly frustrated with workplace conditions–worries that plummeted after our 1993 consolidation and move. This time, computer-related issues–classified as administrative or personal frustration in nature–burgeoned, but interestingly have returned to prior levels during the 2 months following the successful implementation of Toxicall. Conclusion: The CIS affords supervisory staff insight to on-line staff concerns while providing staff with yet another opportunity to ventilate their day-to-day frustrations. Resultant data have consistently pinpointed commonalities of staff's "wants and needs" to the advantage of one and all.

22 ADOLESCENT VS ADULT OCCUPATIONAL TOXIC EXPOSURES: DIFFERENCES IN SEVERITY.

Woolf A, Garg A, Alpert H, Lesko S. *Harvard & Boston University Medical Schools; Massachusetts Poison Control System, Boston, MA*

Background: Working adolescents, because of their entry-level status, may take more risks, receive less training, and come into contact with more toxins on the job than adults. Their injuries from occupational toxic exposures are thus

hypothesized to be more severe. Objective: Compare the types and severity of adolescent vs adult workplace exposures. Methods: Secondary analysis of 1993–97 TESS data on occupational toxic exposures stratified by age and toxin. Analysis used STATA v.6 and contingency tables with the X^2 statistic; two-tailed alpha = 0.05. A validity study confirmed the exposure types and circumstances. Results: Of 283,977 US workplace toxic exposures over 5 years, 8,784 (3.1%) involved adolescents <18 yrs old, 255,655 (90%) involved adults >20 yrs old. A greater proportion of adolescents than adults were exposed to alkali (12.1% vs 7.4%; $p < 0.001$), cleaners (9.1% vs 4.1%; $p < 0.001$), and bleach (8.5% vs 2.9%; $p < 0.001$). A smaller % of adolescents than adults were exposed to gases (14.9% vs 13.2%; $p < 0.001$), hydrocarbons (9.6% vs 6.9%; $p < 0.001$), and misc chemicals (6.4% vs 2.2%; $p < 0.001$). Adolescents were less likely than adults to be exposed by inhalations (47.4% vs 35.3%; $p < 0.001$) and more often by ingestions (19.0% vs 13.7%; $p < 0.001$) and eye exposures (26.6% vs 20.6%; $p < 0.001$). 53.4% adolescent vs 45.4% adult ($p < 0.001$) exposures caused no/minor effect. 50.9% adolescents vs 43.3% adults ($p < 0.001$) were kept on site. Of the symptomatic, 81.1% adolescents vs 76.2% adults ($p < 0.001$) were ill <24 hours. Conclusion: These data do not support the hypothesis that adolescent occupational toxic exposures are more severe than those among adults. They are less likely to be triaged by poison centers to a health care facility. Age differences in types of toxins used on the job may explain these findings, with more adults than adolescents injured by gases, hydrocarbons, or potent chemicals. Adolescents are more often exposed to less injurious cleaners and bleach.

23 ADOLESCENT OCCUPATIONAL TOXIC EXPOSURES: A NATIONAL STUDY.

Woolf A, Garg A, Alpert H, Lesko S. *Harvard & Boston University Medical Schools; Massachusetts Poison Control System, Boston, MA*

Background: Job-related injuries involving adolescents <18 years old are unlikely to be reported either to OSHA or for workmen's compensation; thus there are no national data to describe early adolescent toxic exposures occurring in the workplace. Prevention of such incidents requires more information about their frequency and circumstances. The AAPCC's TESS dataset represents a unique opportunity to investigate such injuries. Objective: Describe adolescent workplace exposures occurring in the United States. Methods: Secondary analysis of 1993–97 TESS data on occupational toxic exposures in the United States, stratified by age and toxin. STATA v.6 was used for data analysis; contingency tables with the X^2 statistic and linear regression were performed; two-tailed alpha was set at 0.05. A validity study confirmed exposure types and circumstances. Results: Of 283,977 US workplace toxic exposures over 5 years, 8,784 (3.1%) involved adolescents <18 yrs old (males 64.3%). The most common adolescent exposures were to gases and fumes (13.2%), alkali (12.1%), cleaners (9.1%), bleach (8.5%), drugs (7.6%), acids (7.3%), and hydrocarbons (6.9%). Adolescents were most likely to suffer toxic exposures during the summer months (June, July, August); exposures on weekends were as common as during weekdays. Most were managed on site (51%) or treated and released from a HCF (37%); <3% of patients required admission to the hospital. 13.1% of exposures resulted in moderate and 0.3% in severe injury. There were 2 deaths. Linear regression analysis of weekly proportions suggested a trend toward increasing exposures among this age group from 1993–1997 (r^2 0.03; $p = 0.005$). Conclusion: Poison center generated data represents a valuable source of national surveillance defining occupational toxic exposures that involve young adolescents. Such injuries most often involve males exposed to fumes, caustics, cleaners or bleach. While most such exposures in this dataset were medically trivial, 13.4% had moderate/severe outcomes and there were 2 deaths.

24 PEDIATRIC ENVIRONMENTAL TOXIC ILLNESS: AN EMERGING CLINICAL SCIENCE.

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Background: Environmental toxic injuries involving children <18 years old can now be evaluated in pediatric environmental health center (PEHC) subspecialty units, cosponsored by the Association of Occupational & Environmental Clinics (AOEC) and the Agency for Toxic Substances and Disease Registry (ATSDR). These clinics present an opportunity to study the characteristics of families adversely impacted by such toxic exposures. Objective: Describe characteristics of patients referred to one PEHC from 1993–98. Methods: Retrospective medical record review. Approved by hospital IRB. Descriptive stats. Results: Records of 64 patients (55% male; mean age {excluding a mass exposure} 8.3 yrs) were analyzed. 67% of families were self-referred. Circumstances included: exposures to factory emissions

(N = 20), a mass exposure to a brominated swimming pool disinfectant (N = 16), poor indoor air quality (N = 10), home contamination (N = 7), toxic waste site (N = 5), multiple chemical sensitivity (N = 1), other (N = 5). 78 chemical categories were implicated: air pollutants (N = 31), bromine (N = 16), metals (N = 13), pesticides (N = 8), toxic wastes (N = 3), PCB (N = 2), other (N = 5). 137 symptoms were reported by 40 patients (excluding mass exposure victims): headache (37.5%), abdominal pain or rash (7.3% each), fatigue (6.6%), sore throat (5.1%), and 40 others. Only 6 of 153 (4%) laboratory tests were abnormal. A final diagnosis was evident in 37 of 64(58%) cases: bromine (N = 16), poor indoor air quality (N = 5), pesticide (N = 3), toxic fumes (N = 3), arsenic (N = 2), manganese (N = 2), carbon monoxide (N = 1), other (N = 5). In >50% of cases, the connection between the exposure and the symptoms was difficult to prove conclusively. Conclusion: Children exposed to environmental toxins are posing new diagnostic and management challenges to health care providers.

25 "THE GRANNY SYNDROME" AND MEDICATION ACCESS AS SIGNIFICANT CAUSES OF UNINTENTIONAL PEDIATRIC POISONING.

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Background: It is estimated that children ≤ 5 years, accounted for >50% of toxicant exposures according to the AAPCC. Over 80% are unintentional. In spite of the fact less than 10% of these exposures result in significant symptoms, over 50% will present to a health care facility. Objective: To characterize unintentional medication ingestions involving children, and to determine the reason for causality. Methods: All pediatric poisoning cases called into a local regional PCC between 1996 and 1997 were retrospectively reviewed. Prospectively the first 200 callers during July–August 1998, assessed as "Granny Syndrome", were asked to complete a phone questionnaire to characterize the events leading to the exposure. Analysis: Chi squared analysis of the major events (access, storage). Significance was considered as $p < .05$. Results: Grandparents are an independent risk factor for unintentional pediatric medication intoxication (Granny Syndrome), accounting for approximately 20% of all such exposures. Although medications typically are stored in non-child-resistant-containers (CRC), exposures occurred because of easy access to medications, ($p = .01$) not the type of storage container ($p = .19$). Pocketbooks are potentially a lethal weapon left on the floor, accounting for 14% of subset exposures. Analgesics, cardiovascular drugs and psychopharmaceutical preparations are the most common medication exposures. Conclusion: Unintentional medication exposures are a highly preventable public health problem affecting young children. The elderly should be counseled about the potential risk their medications pose to children. We should not rely on CRC to prevent exposures since this injury pattern is primarily caused by access.

26 POISON EXPOSURES IN THE ELDERLY: A FIVE-YEAR DESCRIPTIVE STUDY.

Martin AC, Caravati EM, Crouch BI, Diller EM. *Utah Poison Control Center, Intermountain Injury Control Research Center, University of Utah, Salt Lake City, UT*

Background: The elderly are the fastest growing segment of the US population. Previous studies of localized elderly populations have suggested a different poisoning profile compared to the general population. The purpose of this study was to characterize the epidemiology and the corresponding outcome of poison exposures in this population by using a national database. Methods: Human poison exposures involving individuals 60 years of age and older were obtained from the American Association of Poison Control Centers Toxic Exposure Surveillance System (TESS) for the period 1993–1997. Descriptive analysis using frequency distributions and cross tabulations was performed. Results: A total of 322,423 poison exposures were identified in the study population. This group represented 3.2% of all human poison exposures reported to TESS during the study period. The majority of poison exposures (273,857; 84.9%) were unintentional. Therapeutic errors occurred in nearly 20% (63,009) of cases and adverse drug reactions occurred in 13,838 (4.3%) of cases compared with 534,792 (5.3%) and 140,596 (1.4%), respectively in all TESS poison exposures. The most common substances involved in exposures were household cleaners (33,004; 10.2%), cosmetics and personal care items (27,934;8.7%), cardiovascular agents (25,915; 8.0%), bites and envenomations (24,616; 7.6%) and analgesics (18,013; 5.6%). Major effects and fatal outcomes occurred in 4,115 (1.3%) of exposures compared to 46,520 (0.5%) of the total TESS population. Conclusions: Poison exposures in older adults are more frequently due to therapeutic errors and adverse drug reactions. In addition, this population experiences more significant adverse effects following poison exposures compared with the general TESS population.

27 CLINICAL MANIFESTATIONS OF POISONINGS IN THE ELDERLY.

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Background: The frequency, type and severity of clinical effects of poison exposures in the elderly are not well known. The purpose of this study was to describe the clinical effects of poison exposures in this population. Methods: Data on poison exposures occurring in individuals aged 60 years or older were obtained from the American Association of Poison Control Centers Toxic Exposure Surveillance System (TESS) for 1997. Descriptive analysis was used to examine the documented clinical effects and the relationship with substance category and medical outcome. Results: There were 81,191 poison exposures meeting the study criteria. Medical outcomes ranged from no effect (15,036, 18.5%), to major outcome and death (904, 1.1%). A larger proportion of the elderly population had some clinical effect as a result of their poison exposure (21,492; 26.5%) as compared to the general TESS population (484,396; 22.1%) for the same time period. Major outcomes and death were most frequently associated with cardiovascular drugs (209, 23.1%), analgesics (189, 20.9%) and antidepressants (169, 18.7%). Coma was related to poison exposure in 734 (0.9%) patients. The most common substance categories associated with coma were sedative/hypnotics/anti-anxiety/anti-psychotics (276, 37.6%) and antidepressants (196, 26.7%). Multiple seizures were related to poison exposure in 30 patients. The most common substance categories associated with multiple seizures included antidepressants (10, 33.3%) and asthma therapies (5, 16.7%). The most common cardiovascular symptom reported was tachycardia (857, 1.1%). Asthma therapies (223, 26%) and antidepressants (184, 21.5%) were the most common substances associated with tachycardia in this age group. Conclusion: Clinical effects were more likely to occur in the elderly population as compared to the general TESS population. Characterizing these clinical presentations is helpful in understanding poison exposures in this population.

28 ARE DOGS LIKE PEOPLE?

Tegzes J, Herrick M. *Oregon Poison Center, Oregon Health Sciences University, Portland, OR*

Background: The AAPCC has designated a Veterinary Committee to develop protocols for animal poisonings. Over a 2 year period, one poison center handled 73 canine exposures to long-acting anticoagulant rodenticides, without a standardized protocol. This review was done to evaluate the application of a human model to this common canine poisoning. Methods: A comparison was done between the pediatric protocol developed by one regional poison center for these exposures, and treatment recommended in veterinary literature and applied in clinical veterinary practice. Results: Retrospective review of over 500 pediatric cases referred for PT (prothrombin time) screening showed that children rarely ingest large quantities of these products, and as a result, can be safely monitored at home. In contrast, a review of veterinary literature revealed that amount ingested is not a reliable criteria for treatment, based on the experience of vets that dogs ingest the entire quantity that is available to them. Neither are clinical signs of bleeding considered reliable indicators for treatment, because postmortem exams on fatal poisonings revealed that dogs died from intra-thoracic and intra-abdominal bleeding, often without external signs. It was also noted that dog owners are less likely to comply with regimen of PT screening than with administration of oral medications. For these reasons, most vets treat ingestions of these products with a minimum 4–6 week course of Vitamin K1, without PT screening. Local veterinary clinic records revealed that most dogs presenting with history of ingesting these products were treated with Vitamin K1, without PT screening. Conclusions: Pediatric protocols for management of long-acting rodenticide ingestion are not appropriate for canine exposures to these agents.

29 EPIDEMIOLOGICAL FINDINGS ON MECONIUM DRUG TESTING IN INDIANA.

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Background: Drug abuse during pregnancy is associated with a high incidence of asphyxia, premature birth, low fetal birth weight, congenital malformations, physical and developmental deficiencies. In order to more fully assess the prevalence of fetal growth deficiency associated with drug use during pregnancy, the State of Indiana recently established a program in which meconium specimens were required to be collected and tested for the presence of drugs of abuse from infants of low birth weight and small head circumference. Methods: The criteria for acceptance into the State Program required that the birth weight be less than 2500 grams and head circumference of less than the third percentile for the infant's gestational age. The drugs/drug classes included in the testing were Amphetamines, Cocaine, Opiates,

Phencyclidine, and Cannabinoids (THC). Screening was performed using radioimmunoassay followed by confirmatory testing by gas chromatography/mass spectroscopy. Results: From December 1998 through March 1999 a total of 238 specimens were submitted to AIT Laboratories from within Indiana with 23.9% meeting the State Program requirements. The overall positive rate was 20.6% (49 of 238 specimens). Consistent with overall drug use patterns, THC was the most prevalent drug found in meconium, accounting for 55.3% of all positive specimens (28 of 49). This was followed by cocaine, 42.9% (21 of 49) and opiates, 4.1% (2 of 49). No specimens tested thus far have been found to be positive for amphetamines or phencyclidine. Conclusions: These findings further support the analysis of meconium as a useful specimen for the evaluation of drug abuse during pregnancy and its incidence in infants born with low birth weight and head circumference.

30 TRENDS IN INTENTIONAL POISONING PRESENTING TO AN URBAN EMERGENCY DEPARTMENT OVER A FIVE-YEAR PERIOD.

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Background: Intentional poisonings are a frequent presenting complaint to U.S. emergency departments. The purpose of the current study was to analyze ingestion trends and changes in deliberate self-injury over the last 5 years. Methods: A retrospective chart review of all patients presenting to a regional toxicology referral center with intentional ingestions was performed for two matched time periods (1/93–6/93 and 1/98–6/98). Results: A total of 241 (1993) and 234 (1998) charts met the inclusion criteria ($p = 0.42$). Admission for medical stabilization was required for 79 patients (32.8%) in 1993 and 90 patients (38.5%) in 1998 ($p = 0.01$). The average length of stay was 2.51 days in 1993 compared to 2.91 days in 1998 ($p = 0.48$). The average intensive care unit lengths of stay were 0.67 days and 0.89 days respectively ($p = 0.56$). No patient died during either time period. In descending order of frequency, the most commonly ingested substances in 1993 were benzodiazepines, combination analgesics, non-steroidal anti-inflammatory drugs, over-the-counter cold preparations, and tricyclic antidepressants (TCA). In 1998 the 5 most common substances were benzodiazepines, acetominophen, combination analgesics, TCAs, and anti-epileptic medications. An analysis of all 33 drug categories showed no change in ingestion profile during the study period ($p = 0.37$). Conclusion: There has been no statistically significant change in the number of presenting patients, admission or ICU days, nor in profile of agents used in intentional ingestions. However, there was a statistically significant increase in the number of patients admitted between 1993 and 1998.

31 A PROSPECTIVE STUDY OF ACUTE CHILDHOOD POISONINGS IN TURKISH HOSPITAL PATIENTS, IN IZMIR.

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Background: There is no current systematic information about the causes, management and clinical course of acute childhood poisonings occurring in Turkey. We have therefore conducted a prospective survey of all children presenting with acute poisoning during one year to the emergency room (ER) of Dokuz Eylul University Hospital. Methods: This prospective study included all children with acute poisoning brought to ER during 1998. Information stored in the database includes demographics, time, route of poisoning, type of child involvement, substance, symptoms at ER referral, treatment, admission/discharge, and outcome. Results: One hundred and ninety-nine cases of acute childhood poisoning (131 females and 68 males) presented to the ER. While the majority of cases were due to accidental poisoning (63%), intentional poisoning dominated in children 10–17 years old. Suicidal attempts were more common in females (93% of intentional poisoning). Most cases involved drugs (55%), while food poisoning accounted for 14% and gas poisoning for 8%. As to drugs, analgesics (24%) and central nervous system drugs (24%) dominated. Seventy-one patients (36%) received activated charcoal. Gastric lavage was performed in 35% cases and 27% involved both gastric lavage and administration of charcoal. Of the 32 cases (16%) admitted to the hospital, one had to be managed in the intensive care unit. Most of the patients (97%) were discharged without any sequelae. There was only one fatality due to carbamazepine overdose. Conclusion: In this survey on 199 consecutive cases of acute poisoning in children, about half of the cases involved drugs. Data suggest that preventive measures such as child resistant containers for medicines in Turkey should be introduced.

32 REGULATION OF CHILD-RESISTANT PACKAGING FOR VETERINARY DRUGS.

Herrick M, Tegzes J. *Oregon Poison Center, Oregon Health Sciences University, Portland, OR*

Background: Over 30% of US households include a companion animal, mostly dogs and cats. In 1996, pet owners spent an average of $186/dog and $147/cat on veterinary care. Although no clear data could be found, it can be assumed that many of these office visits included dispensing of medications by the veterinarian. Anecdotal evidence exists that some of these drugs are dispensed in paper envelopes, plastic ziploc bags, and a variety of other containers which would allow easy access to children. This study was designed to determine what regulations exist in the Veterinary Practice Acts of each state with regard to child-resistant packaging of drugs dispensed by veterinarians. Methods: Veterinary Boards in all 50 states and the District of Columbia were surveyed to determine if the Veterinary Practice Act in each state requires that veterinarians dispense medications in child-resistant containers. Results: Forty-one states responded to the survey. The Veterinary Practice Act in nine states (21.9% of respondents) includes clear language requiring that medications be dispensed in child-resistant containers, unless the client specifically requests otherwise. Twenty-four states (58.5%) do not address the issue in their Veterinary Practice Acts. Another four states (9.8%) require that veterinarians follow the pharmacy board regulations in their state. The remaining four (9.8%) require that veterinarians follow all federal regulations covering the dispensing of medications. Conclusions: The majority of states (78%) do not have language in their Veterinary Practice Acts which specifically requires that veterinarians dispense medications to clients in child-resistant packaging. The language of the Poison Prevention Packaging Act may be interpreted to include veterinary medications, but the Act has not been routinely used to regulate this industry. More consistent regulation might reduce the incidence of accidental childhood poisoning from these drugs.

33 PERCEPTIONS OF POISON: AGES FOUR AND SEVEN.

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Background: Piaget's theory of cognitive development allows educators and parents insight on the ability of how children learn and interact with their environment. According to Piaget's theory, children ages 2 through 7 are in a stage of pre-operational thought. A primary concept of this theory which is significant to educating young children is symiotic function. Symiotic function asserts that children utilize symbols or words to classify their perceptions of objects. A cross-sectional qualitative survey was completed to assess how poison is classified in relation to symiotic function among children ages 4 and 7. Methods: A survey of 2 open-ended questions was administered to 426 children (189 age 4, 237 age 7) all of whom had no prior poison prevention education. The questions were: 1.) What is poison? and 2.) Do you have any poisons in your home? The children were read the survey verbatim to reduce bias from interpretation. Results: Of the 4 year olds surveyed, 74.6% could accurately define or provide an example of a poison; however, 86.8% stated they did not know or did not have a poison in the home. In comparison, of the 7 year olds surveyed 96.4% could accurately define or provide an example of a poison and 8.0% stated they did not know or did not have a poison in the home. Conclusions: It is notable that 74.6% of children age 4 had developed the concept of poison and only 13.2% could state they had a poison in their home. In contrast, children age 7 have developed the perception of poison and are aware of poisons in their home. We believe this survey demonstrates that while 4 year old children have developed a classification of poison they are not able to accurately account for poisons in their environment. Parents and poison center educators need awareness of children's developmental stages to facilitate poison prevention education.

34 POISON CENTERS AND HOSPITAL PHARMACY ANTIDOTE STOCKING–IMPACT OF EDUCATIONAL EFFORTS.

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Background: We have surveyed the state's acute care facilities over the last 10 years to maintain an inventory of antidotes available. In 2/96, we included the amount required to completely treat a severely poisoned adult. In 11/96, Dart *et al.* published their data in *JAMA* documenting inadequate antidote stocking of hospital pharmacies. We questioned whether stocking of antidotes is influenced by these educational efforts. Methods: A retrospective analysis of the trend in pharmacy stocking before and after educational interventions was supplemented by a survey to determine the reasons for current antidote stocking levels. Results: Current antidote stocks vary widely, with nearly all facilities

(>92%) having adequate stocks of cyanide kits, while fewer have adequate stocks of other antidotes such as protopam (<50%). Although Chi-square analysis demonstrates significant improvement in stocking a variety of antidotes after the two specific interventions mentioned, this trend began the year prior to educational efforts. The timing of an increase in stocking, particularly protopam, correlates more with events such as the sarin gas attacks in Japan and local organophosphate poisoning episodes. While pharmacists found the information provided by the PCC (79%) and the medical literature (19%) useful, their stocking decisions were determined by local utilization (26%) and cost (16%). Smaller hospitals indicate they have arranged sharing plans. Conclusions: Hospital pharmacists are aware of antidote needs, but adequate stocking is determined by utilization and cost. Poison center educational efforts should focus on encouraging adequate stocks of time-sensitive antidotes (e.g. cyanide kit, snake antivenin where indicated, and pyridoxine), maintaining information regarding hospital supplies of antidotes, facilitating treatment, and encouraging antidote stock sharing by geographically proximate facilities.

35 EFFECT OF POISON CENTER RECOMMENDATIONS ON HOSPITAL PHARMACY STOCKING OF EMERGENCY ANTIDOTES.

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Background: Insufficient stocking of emergency antidotes in hospital pharmacies is a well documented problem. Objective: Determine whether insufficient stocking of emergency antidotes can be improved by regional poison center recommendations. Methods: A written survey of pharmacy directors of all hospitals with emergency departments in Colorado, Montana, Idaho and Nevada was completed. Regional poison center antidote stocking recommendations were then distributed to all hospital pharmacies. Three months later, an identical written survey was repeated. For each survey, respondents reported the amount currently in stock of 10 different antidotes. Insufficient antidote stocking was defined as an amount inadequate to initiate treatment of one 70-kg patient: crotalid antivenin (5 vials), cyanide kit (1 kit), deferoxamine (1 g), digoxin immune Fab (20 vials), ethanol (70 g), fomepizole (2 vials), naloxone (2 mg), methylene blue (140 mg), pralidoxime (2 g), and pyridoxine (5 g). Results: Questionnaires were mailed to hospital pharmacy directors: 159 of 207 (77%) responded to the first and 122 of 203 (60%) to the second. One hospital in both surveys (<0.5%) stocked all 10 antidotes in adequate amounts. Rate of insufficient stocking for antidotes ranged from 3% (naloxone) to 98% (digoxin immune Fab) in both surveys. There was significant improvement in the stocking of only 1 antidote, fomepizole, which increased by 13.4% ($p < 0.05$ by two-tailed Fisher's Exact testing). There was no change in the stocking of the other 9 antidotes. However, the proportion of hospitals with any amount of all 10 antidotes significantly increased from 4% to 14% ($p < 0.05$ by two-tailed Fisher's Exact testing). Conclusions: Insufficient stocking of antidotes continues to be a widespread problem in Colorado, Montana, Idaho and Nevada despite the distribution of stocking recommendations by a certified regional poison center.

36 EVALUATION OF RAPID URINE TOXICOLOGICAL TESTING IN PATIENTS WITH ALTERED MENTAL STATUS IN THE EMERGENCY DEPARTMENT.

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Objective: The evaluation of patients with mental status change presents a challenge to the emergency physician. A rapid bedside urine screen may contribute to early diagnosis and management of these patients. The objective was to determine if early knowledge of drug screen results for patients with altered mental status would facilitate care. Methods: An observational, prospective study was performed on a convenience sample of 182 patients presenting to an urban tertiary care center with mental status change. We determined the impact that immediate results of the bedside Triage™ urine toxicological assay would have on patient diagnosis, management, and disposition. Examiners were required to complete pre- and post-test surveys with each drug screen. Results: Patients had an average age of 39.5 years. 50% were male, 52% were white and 48% were African-American. Survey assessment by the treating physician showed that patient diagnosis was aided in 81.9% (95% CI: 75.3–87.0) of patients and clinical management was changed in 24.7% (95% CI: 18.8–31.8) of patients. The Triage™ assay changed management seventy times in forty-two patients, (administration of naloxone 7.1%, obtaining CT of head 2.2%, performing lumbar puncture 10.4%, administration of charcoal 7.7%, performing gastric lavage 6.0% and administration of antidotes 4.9%). The assay did not result in a

significant change in disposition (p = .506). Conclusion: Early knowledge of drug screen results aids in the diagnosis and influences the management of patients with altered mental status.

37 ACTIVATED CHARCOAL ASPIRATION PRODUCES DIRECT PULMONARY INJURY.

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Background: Aspiration of activated charcoal can produce mechanical airways obstruction but intra-pulmonary charcoal is considered inert. We present a patient who aspirated charcoal and went on to develop bronchiolitis obliterans and chronic respiratory failure. Case Report: A 15-year-old girl presented to the emergency department three hours after the ingestion of 4.0 g of nefazadone. She was alert and oriented with normal vital signs but was uncooperative. A nasogastric tube was passed and its location was confirmed by auscultation during air injection. After the administration of approximately one-half of a 50 g activated charcoal in water dose, she vomited, hyperventilated and removed the tube. There was a sudden onset of respiratory distress, stridor and peri-oral cyanosis. Bradypnea (<10) and bradycardia (<60) ensued, and she was intubated with difficulty. She was admitted to the ICU and bronchial lavage removed charcoal. After recovery from the acute insult, a progressive loss of pulmonary function ensued. Over a 90-day period, her FVC and FEV_1 declined from 90% and 60% of predicted to 40% and 30%, respectively. Bronchiolitis obliterans was diagnosed and supported by a CT scan of her chest. A second scan showed deterioration. Cyclosporine over 90 days (beginning on day 18) was given with the intent of decreasing the fibrotic process within the small airways. She became dependent upon supplemental oxygen 15 weeks after aspiration, and chronic hypercarbia ensued 7 months after aspiration. She is awaiting lung transplantation. Conclusion: Aspiration of stomach contents does not lead to bronchiolitis obliterans and direct instillation of charcoal into the lungs of animals produces pulmonary injury. This, along with our patient's clinical course and a previous case report of bronchiolitis obliterans, supports the hypothesis that aspiration of activated charcoal can produce direct pulmonary injury with progressive loss of lung function.

38 PARAMEDICS' GENERAL KNOWLEDGE ABOUT TREATING POISONED PATIENTS WITH ACTIVATED CHARCOAL (AC).

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Background: Previous studies have demonstrated that paramedics receive variable amounts of training in treating poisoned patients with AC and they infrequently administer it. The objective of this study was to test paramedics' general knowledge about AC. Methods: A convenience sample of 251 ALS (advanced life support) technicians completed a 5 question multiple choice survey about AC. Questions covered general knowledge appropriate to paramedic practice as determined by a panel of emergency medicine physicians, toxicologists and paramedics. Participants were recruited at EMS (emergency medical services) stations near 4 small to medium sized US cities with poison centers. Data collection was monitored to ensure independent responses without the use of references. Names were not recorded and surveys were not examined until study-blind data entry. Results: Participants worked as ALS technicians for a mean of 9 years (Range: 1–27 years). Overall, 60% of knowledge questions were answered correctly. 47% (108/231) of respondents incorrectly believed that AC was contraindicated in a patient less than 3 years old. 37% (91/246) incorrectly identified the preferred time frame for AC administration as 4 hours, instead of 1 hour, and 25% (61/246) incorrectly believed that single dose AC causes emesis in most patients. 32% (79/246) of respondents agreed and 36% (89/246) disagreed that further training would make them more likely to administer AC in the field. Conclusions: This study demonstrates that there are deficits in some paramedics' knowledge about the indications, contraindications and side effects of activated charcoal.

39 PARAMEDICS' USE OF ACTIVATED CHARCOAL (AC) AND PERCEPTIONS ABOUT BARRIERS TO ITS ADMINISTRATION.

Hoffnung A, Cobaugh D, Wax P, Schneider S, Lawrence R. *Department of Emergency Medicine and Finger Lakes Regional Poison & Drug Information Center, University of Rochester Medical Center, Rochester, NY*

Background: Previous studies have reported that AC is infrequently administered to patients in the pre-hospital setting. The objective of this study was to describe how often paramedics administer AC and their perceptions about barriers to its administration. Methods: A convenience sample of 251 ALS technicians completed a 26 question multiple choice questionnaire about their experiences transporting patients with toxic ingestions and their perceptions about barriers to

administering AC. Participants were recruited at EMS stations near four small to medium sized US cities with poison centers. Data collection was monitored to ensure independent responses. Names were not recorded and surveys were not examined until study-blind data entry. Results: 62% (152/247) of participants reported transporting patients with toxic ingestions more often than once per month, but only 16% (38/244) administered AC more than once per month, and 72% (176/244) administered AC less than twice per year. 64% (156/243) reported that they required medical command permission to administer AC and 4% (9/243) inaccurately (as later compared to protocol) believed that they were never permitted to administer AC. The three most reported barriers to administering AC were patient resistance to ingest it (39%), risk of spillage and patient emesis (28%), and failure of medical command to advise its use (26%). 60% (147/246) of respondents agreed that the availability of a more palatable form of AC would make them more likely to administer it in the field. Conclusions: These data support previous reports that AC is infrequently administered by paramedics in the pre-hospital setting. Patient non-compliance, risk of emesis, and reliance on medical command were the three most frequently reported barriers to AC administration.

40 EFFECTIVENESS OF AN ACTIVATED CHARCOAL MEDIA CAMPAIGN.

Banach G, Kuhl H. *Central New York Poison Control Center, University Hospital, Syracuse, NY*

Background: The Poison Control Center initiated an awareness campaign focusing on the need for activated charcoal granules in the home for use after a poison emergency. The message was directed to the general public and to healthcare professionals through several media during the month of March. The purpose of the study was to determine the impact of our media efforts on the sale of activated charcoal granules for use in the home. Methods: The study ran January–April 1999. All sales of activated charcoal from 85 pharmacies of a chain reported the movement of activated charcoal granules from stores January through April. The following outreach methods were employed for promotion to begin March 1, 1999: Information cards for customers and posters on activated charcoal were given to each store. A press release went to the local television stations (5), radio stations (8) and to daily newspapers (2). Press packs were developed, a media event was held and public service announcements were sent to radio (29) stations in 14 counties, along with our newsletter. A letter with supporting information was sent to 14 Medical Societies in our 14 county area, with information for physicians. An article on activated charcoal was the lead story in our newsletter sent to 1500 readers, primarily, child related services. Results: In January and February, 102 bottles of activated charcoal granules were sold and in March and April, 271 bottles. The number of bottles of activated charcoal sold in the two months prior to the media campaign was compared to the number sold in the two months after, using Chi Square analysis ($p < 0.05$). Conclusions: It is clear that the media campaign conducted by the Poison Control Center in promotion of procuring activated charcoal granules for home use, was effective in increasing the sales of activated charcoal.

41 PEDIATRIC LAVAGE AND GASTRIC DECOMPRESSION TUBES: A NEW FORMULA EVALUATED.

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Background: At present, gastric tube depth of insertion depends on nursing estimation technique (NEX or external landmarks measurement of Nose, Ear and Xiphoid). Improper placement for lavage in poisoning cases has occurred. A pilot study of gastric tube insertion, using a formula derived graph for esophageal length correlated to patient's height (Graphic), indicated a potentially more accurate and consistent method to determine depth of gastric tube insertion depth for purposes of gastric lavage in poisoning or gastric decompression. This study formally compares these two methods. Methods: Prospective, randomized, double blinded study comparing NEX and Graphic methods for gastric tube depth of insertion. Convenience sample of pediatric emergency department patients in need of gastric lavage or decompression. Patients were block randomized to one of these two groups; all patients had the alternate depth of insertion measured. Abdominal radiographs were used to determine if tube placement was acceptable for use, distance the end of the tube was from the center of the stomach, and whether the tube was in an ideal location ($\pm$ 1.5 cm from center of stomach). Comparisons between the two methods were made using chi-square, and ANOVA. Results: Eighty-nine patients were enrolled. One patient had to be excluded. Forty-four patients each were in the NEX and Graphic groups. No statistical difference existed between the groups in terms of age, gender, weight, height, reason for tube insertion, or type of gastric tube. The Graphic method had 41 gastric tubes in an acceptable position, compared to 31 in the NEX group ($p < 0.05$). Graphic method had 31 gastric tubes in the ideal location, compared to 20 in the NEX

group ($p < 0.05$). Mean distance from the center of the stomach was -1.12 cm $\pm$ 1.3 cm for the Graphic method compared to 1.31 cm $\pm$ 3.39 cm for the NEX method ($p < 0.05$). Conclusion: The Graphic, when compared to the NEX method, shows a statistically significant ability to more consistently and accurately determine the depth of pediatric gastric tube insertion for lavage of poisonings or other indications.

42 IPECAC USE–ARE WE PRACTICING WHAT WE PREACH?

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Objective: In 1997, the AACT/EAPCCT issued a position statement regarding the use of ipecac stating it "should not be administered routinely in the management of poisoned patients." Our study was designed to see if Poison Centers (PCs) are still recommending the use of ipecac, and under what circumstances. Methods: All PCs in the US were contacted by phone and questioned about their policies for ipecac use in both the home and the Emergency Department (ED). Five factitious cases were presented to evaluate what type of ingestion by a toddler might prompt ipecac use: 1) four unidentified red berries, 2) a potentially toxic acetaminophen ingestion, 3) a suspected imipramine ingestion, 4) a potentially toxic iron ingestion, and 5) a toothpaste ingestion. Results: 58 PCs responded to the survey; 15 could not be reached. Annual calls for human exposures ranged from 11,000–135,000, with pediatric calls (age < 6 years) ranging from 33–100%. Of these 58 centers, 41 (70.7%) are AAPCC certified, 28 (48.3%) are associated with a pediatric hospital, and 56 (96.6%) have some type of physician support. Ipecac was recommended in the home always (3.4%), sometimes (89.7%), and never (6.9%); while in the ED, it was used always (0%), sometimes (29.3%) and never (69.0%). While responses to specific cases varied, iron was the most common ingestion in which ipecac was advised. 93.1% of PCs were aware of the 1997 guidelines, but only 37.9% had changed their protocols. No demographic variable was statistically able to predict the likelihood of a PC recommending ipecac for use either at home or in the ED (CI of 95%). Conclusions: Despite the AACT/EAPCCT position statement, a substantial amount of PCs still routinely recommended ipecac use. The frequency for which this occurs varied with the type of ingestion.

43 RISK ASSESSMENT AND COMMUNICATION IN THE EVENT OF A DRUG WITHDRAWAL–THE ROLE OF A POISONS INFORMATION CENTER.

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Background: On 10th March 1999, a product withdrawal notice was issued in the UK for a pediatric formulation of an acetaminophen suspension. This was because if left unshaken, layers were separating in the bottle resulting, potentially, in an acetaminophen concentration of 150 mg/5 mL in the upper layer rather than 120 mg/5 mL. Methods: The poisons center's role in the product withdrawal was as follows: a) Liaison with the drug company to advise on analysis of the product and mixing experiments. b) Liaison with other poisons centers in the UK. c) Creation of a flowchart to advise medical professionals in risk assessment and appropriate treatment. d) Liaison with liver transplant centers to alert them to the potential problem and to ascertain whether they had any suspicious cases. e) Providing press releases, with appropriate risk communication. f) Data collection on potentially affected/exposed children. Results: Initial analyses showed that the unshaken product could contain 840 mg/5 mL acetaminophen in the upper frothy layer. After advice from the poisons center, repeat analysis showed that the maximum acetaminophen concentration in the upper layer could be no greater than 150 mg/5 mL. More than 700 medical professionals telephoned the poisons center for advice regarding potentially exposed children. Only 3 cases required hospital assessment, 2 required *N*-Acetylcysteine as a precaution, none developed fulminant hepatic failure. Conclusion: The poison center's advice on analysis of the product and subsequent role in risk management had a substantial effect. Many hospital admissions were prevented as a result of the targeted advice from the poisons center. Poisons centers need to be prepared to meet such a need in the future, particularly if a commonly used pharmaceutical product is involved.

44 DRUG ERRORS: WASHINGTON STATE'S APPROACH.

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Background: Nationally, drug errors ("medication mishaps") are projected to cause some 7,000+ deaths annually; 2–3 times as many children succumb to such errors in our nation's hospitals as from "unintentional" poisonings occurring elsewhere. Last year JAMA reported the cumulative effect of drug errors and adverse reaction amounted to the 4th leading cause of death in the US. Currently, many efforts are underway to ameliorate such problems; one—specifically

aimed at the prescriber—i.e. documenting the reason for the medication on the prescription form—is trying to be implemented in Washington. Method: With the assistance of our state medical association and state pharmacy board, we gathered baseline and follow-up data—having asked all MDs to add a notation of purpose—"for cough" or "for rash"—on all prescriptions, thus alerting both the pharmacist to the intended class of drugs and the patient of its purpose with the goal of minimizing errors. Results: Among the initial 6,050 prescriptions analyzed before our educational effort, only 29% already included a "notation of purpose"—with 53% for Schedule II and III drugs, but only 6% for antibiotics. Two years later, after our "educational efforts", a second sample of 5,800 prescriptions was analyzed with only 26% showing a notation of purpose—50% for Scheduled (II-V) and 5% for antibiotics, clearly indicating that our educational efforts had been without demonstrable effect. Conclusion: We're pursuing alternative educational strategies, e.g. directed at resident trainees, family physicians and emergency doctors, and hope to be joined by the recently activated National Patient Safety Foundation—and their fiscal resources. Somehow medication errors simply must be curtailed.

45 FREQUENCY OF ADVERSE MEDICATION REACTIONS REPORTED TO MEDWATCH.

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Background: Pre-market testing often fails to detect many adverse medication reactions (ADRs) especially if the adverse effect is secondary to an overdose. The FDA, along with many organizations including the American Association of Poison Control Centers, strongly encourages its members to report suspected ADRs to Medwatch. Methods: Two-hundred and fifty abstracts from posters presented at the 1998 North American Congress of Clinical Toxicology Annual Meeting were reviewed. Presenters of those that met the Medwatch reporting criteria were contacted to determine if the case was reported to Medwatch or the product manufacturer. It is presumed that case reports of adverse effects warranting a poster would be "unique" enough to warrant a report to Medwatch. Results: Based upon our review 24/250 reports should have been reported to Medwatch or the medication's manufacturer. Of these, contact was made with 16/24 poster presenters. Medwatch or the medication's manufacturer was informed of 3/16 (18.75%) cases. Reports were not submitted for 11/16 (68.75%) cases and in 2/16 (12.5%) cases it was not known if the cases were reported. Reasons for not reporting included "no one suggested it", "we would have too many to report", "it is part of a legal case and it will be reported when it is over", and "no, it is a dietary supplement". Conclusion: Those presenting cases as part of a poster presentation should be encouraged and reminded to report all suspected adverse effects to Medwatch or the medications manufacturer.

46 REPORTING OF ADVERSE DRUG REACTIONS BY US POISON CENTERS.

Chyka PA, McCommon SW. *University of Tennessee and Southern Poison Center, Memphis, TN*

Background: Although US poison centers manage approximately 30,000 adverse drug reactions (ADR) each year, the extent of voluntary reporting of these events to the FDA MedWatch ADR surveillance program is unknown. Methods: A survey was mailed to directors of all US poison centers in April 1999 to determine their practices during 1998 and their opinions on reporting ADRs. Statistical analyses included Chi-square contingency tables. Results: A total of 56 fully completed surveys were returned (5 exclusions, response rate = 84%). Of the respondents, 30 had not directly submitted ADR reports to the FDA, 22 had submitted ten or less, and 4 had submitted a total of 47 during 1998. Reasons given for not routinely reporting ADRs included ADR reporting is not part of the regular routine (19.8%), lack of time to complete forms (15%), inability to determine causality (12.8%), most reactions are already reported and not unique (10.2%), reporting to the FDA is too much work (8.6%), and responsibility rests with the attending physician (7.0%). For the 49 poison centers that participated in the AAPCC Toxic Exposure Surveillance System (TESS), respondents indicated that a total of 27,098 ADRs were submitted to TESS. Direct reporting to MedWatch of any ADR cases was more likely when the poison center was certified by AAPCC ($p < 0.05$; odds ratio = 5.1; 95% CI, 1.1–23.5); however, this practice was not associated with documenting ADR-associated deaths to TESS during 1998, having more than 75% of the staff of Poison Information Specialists comprised of pharmacists or managing greater than 20,000 or 34,000 human exposure cases in 1998. Most poison centers (55.2%) indirectly reported some ADRs to the FDA by virtue of contacting the manufacturer or cooperating with post-marketing surveillance. Conclusion: Poison centers represent an underutilized source of reporting to MedWatch, but several internal and external obstacles limit the direct reporting of ADRs routinely.

47 CENTRAL NERVOUS SYSTEM TOXICITY ASSOCIATED WITH METRONIDAZOLE THERAPY.

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Background: Metronidazole has been occasionally reported to result in peripheral neuropathy. The effects of metronidazole on the central nervous system (CNS) are rarely documented. Case Report: A 57-year-old male with a history of hypertension, coronary arterial disease, type II diabetes mellitus was hospitalized for cellulitis and osteomyelitis of the lower extremity. The patient received numerous antibiotics for the infection, including intravenous vancomycin and gentamicin, and, subsequently, ampicillin with clavulanic acid. Because of these antibiotics, he developed pseudomembranous colitis from *Clostridium difficile*. Oral metronidazole (500 mg, QID) was started for the colitis. The patient's other medications were glyburide, furosemide, atenolol, enalapril, and dipyridamole. Approximately 3 months into the metronidazole therapy, the patient experienced increasing tiredness, numbness of the extremities, difficulty with walking and speech. Over the next 2 months, the hands felt more clumsy, the ataxia and dysarthria became pronounced. However, the patient's mentation remained normal. No other neurological abnormalities were noted except for sensory neuropathy of the extremities. A magnetic resonance scan of the brain was normal. Despite close monitoring and tight control of the serum glucose, these symptoms progressed. At this time, metronidazole toxicity was considered and discontinued. Within 48 hours, the majority of these symptoms resolved. These symptoms continued to improve over the next few months and had almost complete resolution of the ataxia and dysarthria. The sensory peripheral neuropathy still persisted (now 5 months) after the discontinuation of metronidazole. Conclusion: Prolonged administration of metronidazole can rarely cause significant CNS toxicity, such as ataxia and dysarthria. Much of these symptoms may resolve with the discontinuation of the medication. It is important for health care providers to be cognizant of these unusual adverse effects.

48 INADVERTENT INTRATHECAL ADMINISTRATION OF HYPAQUE®.

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Background: Hypaque®, diatrizoate meglumine, is a water soluble iodinated contrast media (CM) that has low toxicity administered intravenously for radiography. However, inadvertent intrathecal introduction may result in an ascending tonic-clonic seizure syndrome, rhabdomyolysis and death. A literature review revealed no previous poison center consultation on 18 cases of intrathecal introduction of iodinated CM; 6 deaths were reported. Case Report: A 49-year-old female underwent a cisternogram for evaluation of CSF rhinorrhea. Inadvertently she was administered 10 cc of Hypaque® intrathecally. Hours later she presented with a decreased LOC, muscle spasms in the lower extremities, hypertension, tachycardia, and metabolic acidosis. Upper extremity spasmodic myoclonus ensued, culminating in generalized tonic-clonic seizures. Treatment consisted of ventriculo-lumbar drainage and lavage, pharmacological paralysis, and benzodiazepines. Temporary discontinuation of paralytics 3 days post-exposure revealed continued spasmodic tonic-clonic activity. Patient was dismissed 1 week later without sequela. Discussion: The neurotoxic symptoms of intrathecally introduced iodinated contrast media are produced from direct spinal cord contact and the ensuing destabilization of neuronal membrane potentials. Previous case reports describe a sequence of symptoms a patient may experience: a latent phase of up to 6 hours followed by ascending myoclonus possibly culminating with tonic-clonic seizures, rhabdomyolysis, and death. Treatment consists of elevation of the head and trunk, removal of the CM by ventriculolumbar lavage and seizure management. Conclusion: While inadvertent intrathecal administration of iodinated CM is rare; poison center staff need awareness of this life threatening therapeutic error, toxicity and treatment guidelines.

49 ERYTHROPOIETIN OVERDOSE TREATED WITH EMERGENT ERYTHROPHERESIS.

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Background: Because of its parenteral formulation, expense and limited use, erythropoietin (EPO) overdose is rare and has only been reported in athletes and one suicidal health professional. We report the first severe complication of unintentional EPO overdose. Case Report: A 42-year-old HIV+ male presented to the hospital complaining of pain in both feet and vague abdominal pain. Several months prior to admission the patient was diagnosed with anemia and required multiple transfusions. Subsequently, he was instructed to self administer EPO 20,000 U/week, in addition to his daily interferon injections for KS. The patient became confused about the instructions and administered EPO 10,000

U/day. He subsequently missed two follow-up appointments prior to presentation. Physical examination was remarkable for plethoric facies, a BP of 128/57 mmHg with a pulse of 113/min, a diffusely tender abdomen, blackened toes bilaterally with decreased pulses and pitting edema. The patient was oriented, but slightly confused. Laboratory studies were remarkable for a hematocrit of 72.3%, a BUN of 47 mg/dL and a creatinine of 2.9 mg/dL. The patient was treated with intravenous fluids and underwent emergent erythropheresis. During hospitalization the patient underwent phlebotomy as needed to maintain his hematocrit below 55%. Pain control was given and he recovered without complication. No specific cause for his abdominal pain was ever established although low-grade mesenteric ischemia was considered. His toes improved although some skin loss occurred. Conclusions: Excessive dosing of erythropoietin can cause thrombosis from hyperviscosity. Although EPO abuse is allegedly responsible for several deaths in athletes, these events are poorly documented. Close monitoring usually prevents significant unintentional overdose. This patient's medication error resulted from confusion about the use of two outpatient parenteral medications. Emergent erythropheresis was used successfully to rapidly reduce his hematocrit. As outpatient use of parenteral medications increase, physicians and pharmacists need to assure that appropriate patient education is provided.

50 SEVERE ELECTROLYTE ABNORMALITIES AND TETANY AFTER INADVERTENT FOSCARNET INFUSION.

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Background: Foscarnet (trisodium phosphonoformate) is used for the treatment of CMV retinitis, mucocutaneous herpes simplex infection, and Varicella-Zoster infection in patients with AIDS. It complexes with DNA polymerase and prevents cleavage of pyrophosphate from nucleoside triphosphates. This effect prevents blocking further primer-template extension on the DNA. It is administered intravenously, has a serum half-life of 3 hours, and is principally eliminated by the kidneys. This is a case report of inadvertent Foscarnet administration to a child. Case Report: A 5-year-old boy was admitted to the hospital for Kawasaki's disease in order to receive immunoglobulin IV (IG IV). IG IV was premixed in a solution by the pharmacy. Shortly after the infusion ended, the patient developed tetany and hypertonia but maintained a normal mental status. Evaluation of his electrolytes revealed a serum ionized calcium of 0.6 mmol/L, a phosphorus of 1.6 mmol/L, a magnesium of 1.0 mEq/L, and a potassium of 2.8 mEq/L. The patient's electrolytes were supplemented to normal, the patient remained asymptomatic clinically, and his renal function never deteriorated. Since IG IV has never been associated with these electrolyte abnormalities further investigation revealed that the pharmacy inadvertently mixed Foscarnet into the infusion. There is evidence that Foscarnet causes electrolyte abnormalities via precipitation of divalent metal ions by phosphate which could lead to renal insufficiency. It is unclear how this error occurred. A qualitative test of the patient's blood came back positive for Foscarnet. Conclusions: Foscarnet may cause severe changes in serum electrolytes even when given as a single dose. A cause of unexplained clinical findings in relationship to drug administration may be explained by looking for extemporaneous medication contamination.

51 HYPERAMYLASEMIA IN 2 PEDIATRIC PATIENTS AFTER ACUTE INGESTION OF GRISEOFULVIN.

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Background: Griseofulvin is a medication commonly prescribed to children for the treatment of *tinea capitus* or "ringworm". Little data are available on the toxicity after acute ingestions of this drug, although it is known for its potential hepatotoxicity when taken chronically. Case Report: The Poison Control (PCC) received a call from the mother of a 2-year-old female reporting that both her child and nephew (3-year-old male) were found with an empty bottle of griseofulvin. Only 4 doses of the medication had been previously used. The bottle had contained over 200 mL of the medication, or approximately 5 g. Both children were referred to a health care facility for activated charcoal (AC). Liver function tests were also recommended. The following lab abnormalities were noted, when they were drawn the next day: 2-year-old female–amylase 280 IU/L (normal 35–116 IU/L), AST 70 IU/L (normal 5–40 IU/L), LDH 598 IU/L (normal 94–250 IU/L). 3-year-old male–amylase 185 IU/L, AST 53 IU/L, LDH 382 IU/L. CBC, electrolytes, BUN, and creatinine were within normal limits in both children. On follow up 4 days post ingestion, the 3-year-old male had an amylase of 159 IU/L and AST 34 IU/L. The 2-year-old female, who was seen 3 days post ingestion, had an AST fall to 55 IU/L. Both children remained asymptomatic. Conclusion: There are no previous reported cases of

acute griseofulvin overdose producing elevated amylase and AST. This case suggests that both amylase and hepatic transaminase be measured after large accidental pediatric ingestions of this drug.

52 A CASE OF HALOTHANE HEPATITIS FROM ORAL HALOTHANE INGESTION.

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Background: We present an unusual case of oral halothane ingestion resulting in acute liver failure. Case Report: A 29-year-old male with a history of bipolar disorder and alcoholic liver disease presented to the emergency department (ED) 5 days after hosting a party where the participants were ''huffing halothane''. He denied using halothane personally. He also denied alcohol use for the past 5 years. The following morning he awoke to take his morning dose of gabapentin, drank approximately 2.5 ounces of a clear liquid he thought was water, realized it was halothane and immediately began to vomit. Intractable emesis continued for the next 5 days. On presentation to the ED, his temperature was 103.7, heart rate 119, respiratory rate 18, and blood pressure 155/70. Physical exam revealed an obese male in mild distress from emesis and apparent abdominal discomfort. His abdomen was non-distended with no hepatosplenomegaly. He was tender to palpation over the epigastrium. Lab results showed that the ALT was 214 IU/L, with an AST of 201 IU/L. Poison Control recommended supportive care. Since the CXR showed a right middle lobe infiltrate, he was discharged on an oral quinolone. He presented for the second time 3 days later with increased nausea, vomiting, abdominal pain. He admitted to acetaminophen use for fever and discomfort. Lab results showed an AST of 2760 IU/L, ALT of 2347 IU/L, prothrombin time of 18.8 seconds, and a partial thromboplastin time of 41.4 seconds. Toxicology screens were positive for cannabinoids. The acetaminophen level was 18 mcg/mL. The patient was admitted for acute liver failure thought to be secondary to halothane ingestion. The patient left against medical advice on hospital day 2 with AST decreased to 752 IU/L and ALT decreased to 1366 IU/L. Conclusion: Oral halothane ingestion resulting in acute hepatitis is extremely unusual. Treatment is supportive.

53 FALSE POSITIVE ETHYLENE GLYCOL DETERMINATION BY ENZYME ASSAY IN PATIENTS WITH CHRONIC ACETAMINOPHEN HEPATOTOXICITY.

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Background: Serum ethylene glycol (EG) determinations are usually performed by glycerol dehydrogenase (GDH) enzyme assay or gas chromatography (GC). An advantage of the enzyme assay is that it is relatively easy to perform particularly when needed 24 hours per day. A recent report suggests that false positive results by enzymatic assay may occur in the presence of elevated LDH and/or lactate resulting in increased production of NADH, the product of the GDH enzymatic reaction. We report on 3 patients who presented with fulminant hepatic failure, a history of chronic acetaminophen abuse, and high anion gap metabolic acidosis. Ethylene glycol levels were obtained in each case. Case Series: Each patient had EG levels by GDH enzyme assay (Hitachi 900) that was $\geq$20 mg/dL. In 1 case, serial EG levels were increasing. Treatment was initiated with ethanol infusion in Case 1 and fomepizole in Case 2. The following laboratory analysis was performed:

	EG (mg/dL)	Anion gap	pH	AST (IU/L)	LDH (IU/L)	Lactate (mmol/L)
Case 1	25	31	6.8	6690	11,000	21
Case 2	50	45	7.15	12,464	39,907	14.6
Case 3	20	33	7.15	3612	12,713	26.5

In all 3 cases the elevated EG by GDH enzyme assay was later determined to be negative by GC. Conclusion: We conclude that the use of the EG assay by GDH enzyme technique in these 3 cases of fulminant hepatic failure from chronic acetaminophen abuse produced false positive results. These findings are most likely due to increases in LDH and/or lactate associated with the liver failure and acidosis. Elevated EG by GDH enzyme assay should trigger the ordering of serum LDH and lactate levels, and if they are elevated, GC confirmation for the presence of EG should be considered.

54 ABERRANT VERSIONS OF THE MATTHEWS-RUMACK NOMOGRAM.

Tominack R, Blume C, Scalzo A, Hoffman J, Patel S, Thompson M. *Cardinal Glennon Regional Poison Center, St Louis University Health Sciences Center, Saint Louis Children's Hospital, St. Louis, MO*

Background: The Matthews-Rumack acetaminophen (APAP) nomogram is a familiar and widely used tool in toxicology for assessing risk and recommending antidotal treatment. The nomogram can be accessed in journal articles, textbooks and computer versions. A clinical case highlighted an unrecognized aberration in a major textbook version of the nomogram. Method: A treating physician consulted on an APAP overdose case in which a level of 10.2 mcg/mL was obtained 19 hours after ingestion. This data point fell inside the treatment line on one nomogram in one major medical toxicology textbook, but clearly outside the treatment area on the version in another. Likewise, the DOS version of a computerized nomogram placed the data point in a different risk level than the Windows version. We surveyed the primary literature on the nomogram to investigate the discrepancy and identify the accurate version of the nomogram. Results: The original nomogram line was premised upon a 4 hour serum elimination half life decay beginning at coordinates 200 mcg/mL and 4 hours on a semilog plot of level against time. Subsequent risk level and treatment lines are parallel, originating at different 4 hour serum levels but all decaying with a slope described by a 4 hour half life. Coordinant pairs for the "300 line" are thus (300, 4), (150, 8) and (75, 12); the "200 line" (200, 4), (100, 8) and (50, 12); the "150 line" (150, 4), (75, 8) and (37.5, 12). These are inaccurately reported in an article in *Lancet* 1995; **346**: 547–552. A version of the nomogram in which the semilog scale is graphically misrepresented appears in an *Annals of Emergency Medicine* article 1991; **20**:1058–63 and is reproduced in the 2nd version of *Ellenhorn's Medical Toxicology*. It overestimates risk of toxicity as the line diverges from 4 hours. The nomogram reproduced in *Goldfrank's Toxicologic Emergencies*, originating with the manufacturer is accurate, as is the DOS version of PoisIndex®. Conclusion: Nomograms are subject to errors in description and graphic representation. Variations have been identified which may have an impact on risk assessment and antidote use. These discrepancies not only implicate the risk of error in clinical judgment but also the fiscal cost of treating a patient with NAC therapy unnecessarily. A quick check of major coordinants is recommended to validate any version of the nomogram prior to use for patient evaluation.

55 INCORRECT CALCULATION OF DOSE OF ANTIDOTAL THERAPY: CAUSE FOR CONCERN.

Marcus S, Jennis T, Ruck B. *New Jersey Poison Information and Education System; Newark Beth Israel Medical Center; University of Medicine and Dentistry of New Jersey/New Jersey Medical School, Newark, NJ*

Background: Often patients do not tolerate either the loading dose or maintenance doses of *N*-acetyl cysteine in the treatment of acetaminophen toxicity. We discovered that a patient had received an amount of drug far exceeding the recommended dose because of a calculation error. Methods: Staff attending a randomly selected pediatric grand rounds, internal medicine conference and emergency department teaching conference, were requested to calculate the dose, in ml/kg, of 20% NAC to load at 140 mg/kg and to describe the dilution into a 5% solution. Results: Both of the 2 PharmD candidates were off by a factor of 10^3. Thirty surveys were distributed at pediatric grand rounds; seventeen were returned. Forty-one percent correctly performed the calculation (7/17). Three responses by 3rd year medical students showed no correct answer; one, a graduate pharmacist, suggested that 49 liters of 20% NAC should be administered! Three 4th year students reported errors; two off by 10^3, one off by 10^2. Of the first year residents only one correctly calculated the dose; 2 of the 3 third year residents and all three attendings correctly accomplished the task. One of two, with no reported level of training, correctly calculated the dose; the other was off by a factor of 10^3. Of the twenty who attempted the calculation during internal medicine rounds, none correctly accomplished the task. Errors ranged from 10^{-1} fold to 10^3. Only thirty percent (6/20) of ED residents performed the calculation properly. Conclusion: Calculation errors are common problems in clinical medicine. The miscalculation of dose of NAC may result in excess NAC administration and a consequential intolerance to the drug. Poison centers should assure that correct calculation of dose of antidote be carried out.

56 PARATONIA (RAPID RIGOR MORTIS) IN SALICYLATE (ASA) POISONING.

Rao RB, Smiddy M, Nelson LS, Howland MA, Hoffman RS. *New York City Poison Control Center, OCME, New York, NY*

Background: ASA poisoning and death are fairly common events. In ASA poisoning, however, severe muscle rigidity, known as paratonia or rapid rigor mortis, is rarely reported and poorly documented. The following case report describes

paratonia in a severely ASA poisoned patient. Case Report: A 42-year-old female with a history of schizo-affective disorder and multiple psychiatric admissions was found disturbed and shouting. When EMS arrived she had a systolic blood pressure of 130 mm Hg and a pulse of 70/min recorded at 4:30 PM. She was brought to the hospital where she was noted to be alert with pressured speech, unable to concentrate, somewhat confused, and with alcohol on her breath. She was restrained and sent to psychiatry. At 7:05 PM, the patient was given lorazepam 2 mg and haloperidol 2 mg, both intramuscularly for agitation. At 10:00 PM, an order was written for lorazepam 2 mg PO, and diphenhydramine 50 mg PO. At 12:15 AM, the patient was transferred to the medical emergency department for respiratory distress. On arrival she was noted to have deep breaths at 12–14/min, a systolic blood pressure of 95 mm Hg, and desaturation by pulse oximeter. Skin was normal to touch, but a temperature was not reported. Shortly thereafter, two myoclonic events were noted and the patient stopped breathing. A cardiac arrest ensued. Because of a clenched mouth and severe rigidity, succinylcholine (100 mg IV) was given for intubation. However, the patient remained rigid and required cricothyrotomy. Resuscitation was unsuccessful. Blood drawn on arrival to the medical emergency department revealed a serum salicylate level of 150 mg/dL. Postmortem evaluation was notable for multiple partially digested enteric coated ASA pills throughout the gastrointestinal tract. No other toxin was identified. The failure to respond to succinylcholine excludes a diagnosis of NMS in this case, and malignant hyperthermia is unlikely given the clinical history. Rapid rigor mortis can be produced in animals when conditions of impaired glycolysis, low ATP and high lactate are present. This results in hyperthermia and irreversible myosin insolubility. Conclusions: Paratonia and the failure to respond to succinylcholine, in the absence of malignant hyperthermia, should raise a suspicion of salicylism.

Platform Session 1 — Friday, October 1 — 10:30 am–12:00 pm

Combustion Toxicology
Abstracts #57–#62

57 THE PROTECTIVE EFFECTS OF EXPERIMENTAL NEURODEPRESSORS ON LEARNING AND MEMORY FOLLOWING CARBON MONOXIDE (CO) POISONING.

Gilmer B, Thomson C, Tomaszewski C, Watts J. *Carolinas Medical Center, Charlotte, NC*

Background: Delayed neurological effects of CO poisoning are inadequately prevented by supplemental oxygen and hyperbaric oxygen therapy. Animal models show that neurodepressive agents such as NMDA-receptor antagonists prevent neurological deficits. This study examines the efficacy of L-NAME and CCPA in preventing delayed sequelae after CO poisoning. Methods: Male Swiss-Webster mice (20–30 g) were exposed to CO: 1,000 ppm for 40 min and 50,000 ppm until loss of consciousness (LOC) and were treated 15 min after LOC. The experimental groups received either CCPA (0.3 mg/kg) or L-NAME (15 mg/kg), while controls were untreated. All animals underwent passive avoidance training either 24 hours before (PRE) or 7 days after (POST) poisoning with CO. Learning and memory was assessed by measuring stepdown latency (SDL) at 7 (PRE-Memory) or 8 days (POST-Learning) subsequent to exposure. SDL was compared among groups with Kruskal-Wallis non-parametric ANOVA followed by Dunn's test for multiple comparisons. Results: Median (and range) of SDL in sec were as follows (*$P < 0.05$ versus poisoned control):

Grp.	Unpoisoned	Poisoned	CCPA	L-NAME
Pre	*145(116–185)n = 4	55(34–165)n = 10	*161(29–300)n = 10	*159(20–300)n = 10
Post	*152(15–300)n = 18	44(4–119)n = 8	*200(99–300)n = 10	*142(27–261)n = 7

Conclusions: L-NAME and CCPA were both found to be protective of both learning and memory in a mouse model of acute CO poisoning. These drugs may prevent delayed neurological sequelae by their neurodepressant effects in countering excitatory amino acids such as glutamate.

58 ARE SOOT DEPOSITS AND NEUROLOGICAL DISTURBANCES PREDICTIVE OF CYANIDE POISONING IN FIRE VICTIMS?

Borron S, Favier C, Benaissa L, Baud F. *Réanimation Médicale et Toxicologique. Hôpital Lariboisière. INSERM U26-Université Paris VII. Paris, France*

Background: Smoke inhalation is a leading cause of cyanide poisoning, however, clinical indications for cyanide antidotes in fire victims are not well defined. The aim of this study was to determine the diagnostic value of soot deposits and neurological impairment in predicting cyanide poisoning. Methods: We prospectively studied residential fire victims treated by physicians at the fire scene. Blood specimens were collected after the start of isobaric oxygen therapy, but before hydroxocobalamin. Soot deposits in the mouth and/or sputum were noted on admission. Neurological disturbances included confusion, seizures, and loss of consciousness. Blood carbon monoxide and cyanide were measured. Cyanide intoxication was defined by a blood cyanide greater than or equal to 40 μmol/L (1 mg/L). Results: 408 patients were enrolled. Soot deposits in the upper airway were noted in 168 patients. Among these, neurological disturbances were found in 138. The sensitivity of the presence of soot deposits for cyanide poisoning was 98%; the specificity was 56%, the positive predictive value 28%, and the negative predictive value was 99%. In the context of smoke inhalation with soot deposits, the sensitivity of the presence of neurological disturbances for cyanide poisoning was 98%; the specificity was 49%, the positive predictive value was 44%, and the negative predictive value was 98%. Conclusion: In fire victims, soot deposits and neurological disturbances are sensitive but nonspecific signs with regard to cyanide poisoning. Neurological disturbances may also result from other asphyxiant gases, such as carbon monoxide or volatile organic compounds. In contrast, if confirmed, the absence of neurological disturbances in victims with soot deposits may obviate the need for consideration of the administration of a cyanide antidote.

59 ARE SOOT DEPOSITS PREDICTIVE OF CARBON MONOXIDE POISONING IN FIRE VICTIMS?

Borron S, Favier C, Benaissa L, Baud F. *Réanimation Médicale et Toxicologique. Hôpital Lariboisière. INSERM U26-Université Paris VII. Paris, France*

Background: Smoke inhalation is a leading cause of carbon monoxide poisoning; clinical predictors of carbon monoxide concentrations, however, are lacking. The aim of this study was to determine the diagnostic value of soot deposits in predicting carbon monoxide poisoning. Methods: We prospectively studied residential fire victims treated by physicians at the fire scene. Blood specimens were collected after the start of isobaric oxygen therapy. Soot deposits in the mouth and/or sputum were noted on admission. Blood carbon monoxide concentration was measured. A carbon monoxide concentration ≥1 mmol/L (11% COHb) was considered toxic. Results: 408 patients were enrolled. In 2, the CO was not measured. Soot deposits in the upper airway were noted in 197 patients. The mean blood CO in patients with soot deposits was 1.61 ± 1.99 mmol/L (Range 0.04–10.7). The mean blood CO in patients without soot deposits was 0.42 ± 0.82 mmol/L (Range 0.03–8.58). The sensitivity of the presence of soot deposits for CO poisoning was 93%; the specificity was 63%, the positive predictive value 43%, and the negative predictive value was 92%.

Soot Deposits	CO ≥ 1	CO < 1
Present	85	112
Absent	17	192

Conclusion: In fire victims, soot deposits are sensitive but nonspecific signs with regard to carbon monoxide poisoning. The absence of soot deposits makes significant carboxyhemoglobinemia less likely, but does not rule out significant carbon monoxide poisoning.

60 ROLE OF VOLATILE ORGANIC COMPOUNDS IN EARLY DEATH DUE TO SMOKE INHALATION.

Houeto P, Levillain P, Borron S, Baud F. *Laboratoire de Toxicologie, Réanimation Médicale et Toxicologique–INSERM U26-Université Paris VII, Hôpital Lariboisière, Paris, France*

Background: The toxicity of fire gases is not completely explained by carbon monoxide and cyanide. This prospective study was conducted in order to assess the frequency and the quantity of volatile organic compounds (VOCs) in the blood of fire victims and their relationship to early mortality. Methods: The study group consisted of nonfatal and fatal fire victims whose blood was sampled at the fire scene. The control group consisted of acute drug poisonings and patients found dead at the scene from non-fire related causes. Blood VOCs were measured by purge and trap gas chromatography. Results: Twenty-six fire fatalities were enrolled, as well as 28 living fire victims. Fifty-five non-fire dead and 25 acute poisoned patients served as controls. Thirty-three VOCs were measured in the blood. Fifteen VOCs were statistically associated with death in fire victims. These included: ethyl acetate, acrylonitrile, propionitrile, tetrahydrofuran, toluene, benzene, *o*- and *p*-xylene, ethylbenzene, nitromethane, trichlorofluoromethane, indene, trichloroethylene, 2-pentanone, and acetaldehyde. Three products: benzene, nitromethane, and ethyl acetate are distinguished by their frequency, blood concentrations, and correlation with blood carbon monoxide. This study underscores the important role of aromatic hydrocarbons, including benzene, toluene, ethylbenzene, and *o*- and *p*-xylenes in fire death, suggesting a role for pyrolysis in fire death, as opposed to simple combustion. Certain molecules, such as trichlorofluoromethane were rarely observed but sometimes present in high blood concentrations. Conclusion: These data show that the toxic components of smoke include not only carbon monoxide and cyanide but also significant concentrations of volatile organic compounds.

61 THE NEUROPROTECTIVE EFFECTS OF GLUTAMATE ANTAGONISM ON MEMORY FOLLOWING ACUTE CARBON MONOXIDE POISONING.

Thomson C, Gilmer B, Tomaszewski C, Watts J. *Carolinas Medical Center, Charlotte, NC*

Background: Carbon monoxide (CO) poisoning causes delayed neurologic sequelae (DNS) that may be mediated by the glutamate receptor. We studied the ability of a glutamate antagonist, riluzole, to prevent DNS after CO poisoning. Methods: Controlled study in which mice were exposed to CO: 1,000 PPM for 40 min and 50,000 PPM until loss of consciousness. Treatment was administered 15 minutes later, and consisted of either IP riluzole (8 mg/kg, $n = 15$) or sham ($n = 8$). A third group ($n = 10$) received 70 minutes of air exposure and no treatment. All animals underwent learning and memory testing via passive avoidance training with a shock paradigm 7 days after exposure. On day 8, learning and memory were assessed by measuring step-down latency in the same shock box. Immediately after, animals were euthanized. Brains were paraffinized, sectioned, and stained with hematoxylin and eosin. Viable hippocampal cells were counted. Step-down latency was compared with Kruskal-Wallis nonparametric ANOVA followed by Dunn's test for multiple comparisons. Results: Step down latencies were different among all groups: unpoisoned/control 136 sec (65–300), poisoned/untreated 27 sec (4–119), and poisoned/treated 127 sec (7–300) ($H = 8.865$, $P < 0.025$). *Post hoc* analysis showed only the poisoned/untreated group ($Q = 2.758$, $P < 0.05$) and not the riluzole group ($Q = 0.4323$, NS) to be different from unpoisoned controls. Preliminary examination of viable cell numbers in the hippocampal CA1 layer, an area shown previously to be affected by CO poisoning, were similar between groups. Conclusion: Riluzole has a beneficial role in preserving learning and memory after severe CO poisoning in a murine model. We could not account for the behavioral differences based on cell numbers in the hippocampal CA1 layer.

62 EXPRESSION OF GENE BAX IN THE RAT BRAIN AFTER CARBON MONOXIDE POISONING.

Liu F, Liu Y. *Department of Emergency Medicine, China Medical University, Shenyang, Peoples' Republic of China*

Objective: The purpose of the study was to evaluate the expression of apoptosis-effector gene Bax in the rat brain after carbon monoxide exposure. Methods: Thirty male Sprague-Dawley rats were randomly divided into six groups. After exposure to carbon monoxide, blood carboxyhemoglobin levels were measured by co-oximeter. The rats were sacrificed and a cell suspension of cerebrum and cerebellum was prepared. The gene Bax was measured, using flow cytometry, immediately after sacrifice and at 24 hours, 72 hours, and 7 days after exposure. The data were analyzed using one-way analysis of variance (ANOVA) following q tests. Results:

Group	Cerebrum	Cerebellum
Control	326.2 ± 48.4*	211.4 ± 27.0*$
Blank Control	318.6 ± 53.3*#	192.3 ± 31.5*#
Immediate	368.2 ± 35.2*	240.8 ± 40.7*
24 hours	462.3 ± 45.7	358.2 ± 37.9
72 hours	419.3 ± 25.9	291.9 ± 49.0*
7 days	342.9 ± 31.3*#	199.9 ± 35.3*#

Gene Bax (MFI) * with 24 hours, $p < 0.01$, # with 72 hours $p < 0.01$, $ with 72 hours $p < 0.05$ Conclusion: Acute carbon monoxide poisoning induces the expression of gene Bax in the rat brain, which may contribute to the associated neuropsychiatric sequelae that occur after this common poisoning.

Platform Session 2 — Friday, October 1 — 1:00 pm–2:45 pm

General Clinical Toxicology I
Abstracts #63–#69

63 BOTANICALS AND OTHER DIETARY SUPPLEMENTS: ADVERSE EVENTS BY AGE.

Palmer M, Haller C, McKinney P, Tschirgi A, Klein-Schwartz W, Smolinske S, Everson G, Nelson L, Woolf A, Bartlett D, Dahl B, Dodd-Butera T. *Dietary Supplement Poison Center Study Group*

Background: Dietary supplements are exempted from pre-marketing research and post-marketing surveillance for safety. Case Series: Calls involving dietary supplements were prospectively collected by 11 poison centers in 1998. Over half are entered into a Microsoft Access database. Symptomatic cases where the ingested dietary supplements were judged to have at least 50% probability of causing the symptoms were reviewed. Symptomatic calls per total number of calls by age in years were: < 1:11/47; 1–5:31/390; 6–11:5/27; 12–17:32/79; 18–34:123/399; 35–59:94/200; ≥60: 14/50; unknown age 27/273 (total 1465). The predominant symptoms in infants were vomiting and drowsiness. 1 infant developed seizures, and another, hepatotoxicity. Four cases occurred with breast-feeding. Calls concerning elderly included a case of hepatotoxicity, as well as of coma and hypertension/tachycardia. GI symptoms (97) were most commonly reported for all ages followed by hypersensitivity (53) reactions (5 with respiratory symptoms), and CNS agitation (47) or drowsiness (39). Three deaths (2 with Ma Huang, one Pao d'arco) and severe symptoms included seizure (7, one Wormwood), coma (4), anticholinergic toxicity (5), chest pain (12), conduction abnormalities (12), hepatotoxicity (7, one Jin Bu Huan) and coagulopathy (4). Multi-ingredient products (>3 ingredients/product) predominated the high symptom group (97) followed by, and sometimes including, thermogenic products or "fuels" (50, usually containing Ma Huang), St. John's Wort (17), cultural herbal remedies (12), Kava (11), Ginkgo (10), Ginseng (9), and Melaleuca oil (6). Conclusion: These pilot data question the safety of dietary supplements, with special focus on extremes of age and symptoms.

64 KAVA'S METHYSTICIN: PROTECTION FROM STRYCHNINE AND VERATRIDINE.

Palmer M, O'Donnell R, Ye M. *New York City Poison Control Center, New York, NY; Children's National Medical Center, Washington, DC*

Background: Kava (*Piper methysticum*) is widely used in the US as a dietary supplement for its sedative or intoxicating effects, but its mechanism(s) of action remain unclear. Methysticin, one of its lactones, prevents strychnine toxicity. This property was utilized to make inferences about effects at GABA receptors using antagonists (CGP35348 for $GABA_B$; flumezenil for the benzodiazepine receptor at $GABA_A$). The effects of methysticin on tetanus and veratridine are

also explored to investigate mechanisms via pre-synaptic glycine release and Na^+ channels, respectively. Methods: Seizure/death parameters were compared in mice treated with strychnine + methysticin vs. strychnine + methysticin + CGP35348 and with strychnine + methysticin + flumazenil. Tetanospasm scores were compared in mice treated with tetanus toxin IM, with and without methysticin IP. Finally, seizure/death variables were measured in mice injected with IP veratridine vs. veratridine + methysticin. Sample size/treatment group = 10–20 mice. All controls were injected with vehicle. Survivorship analysis, Chi-square, Student's *t*-test, and power analyses were used as appropriate. Results: Injection of CGP35348 and flumazenil failed to reverse the protective effects of methysticin. There was no significant difference in the mean tetanospasm score, with or without methysticin. There was a significant difference in time to seizure and proportion of mice having seizures in groups treated with both veratridine + methysticin vs. veratridine. Conclusion: Methysticin protection against convulsions may be mediated through local anesthetic effects (but not the GABA receptors tested or glycine), since it is effective in preventing seizures produced by a Na^+ channel-opener. Methysticin may be effective therapy for strychnine or veratridine poisoning.

65 PREHOSPITAL ACTIVATED CHARCOAL: A PROSPECTIVE RANDOMIZED TRIAL.

Keyes C, DeTamble L. *University of Texas, Southwestern Medical Center, Dallas, TX*

Background: Activated charcoal (AC) has become the most important decontamination technique for oral ingestions, and time to administration of AC has been shown to be critical. Retrospective studies suggest that prehospital AC decreases administration time, and that currently very few patients receive AC in the prehospital setting. This study was designed to prospectively determine the feasibility of implementation of prehospital administration of AC in a large urban EMS and determine its impact on AC administration time. Methods: This was a prospective, randomized, non-blinded study undertaken between November, 1998 and April, 1999. Thirty four (34) overdose patients were included, each called in to the base station medical control of a large city. All subjects were randomized to receive charcoal or no-charcoal prior to arrival at the medical center. Inclusion criteria included: age >16 years old, not pregnant, not under police or jail custody, EMS response <3 hours from ingestion. Exclusion criteria included: ingestion of a caustic agent or hydrocarbon, actively seizing, unconscious or absent gag reflex, altered mental status, ipecac administration, seizure, refusal to take AC, vomiting, unstable airway, violence, or agitation. Time from dispatch of EMS to administration of AC was recorded for all patients. Results: Patients in the prehospital AC administration group were similar to those in the hospital administration group with respect to gender, age, and ethnicity. Thirty-four patients were entered into the study, with 17 in each group. The mean time for administration in the prehospital group was 28 minutes as compared with 118 minutes for the hospital group ($P < 0.001$). No aspirations were reported in patients receiving prehospital AC. Conclusions: 1) prehospital administration of AC in a large urban EMS is feasible, and 2) field administration results in clinically-significant reductions in time to AC administration.

66 HOW LONG AFTER DRUG INGESTION IS ACTIVATED CHARCOAL STILL EFFECTIVE?

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

Background: In the recent AACT/EAPCCT position statement on activated charcoal it was stated that "there are insufficient data to support or exclude its use after 1 hour of ingestion." The purpose of this study was to determine the effectiveness of activated charcoal administered 1, 2, and 3 hours after drug ingestion. Methods: This was an IRB approved, human volunteer, randomized crossover study. Ten volunteers ingested 4.0 g of acetaminophen on four occasions at least one week apart. One ingestion served as a control and the other three as experimental ingestions with charcoal being administered at 1, 2, and 3 hours after acetaminophen dosing. Eight blood specimens were obtained over the initial 8 hours for serum acetaminophen concentrations (HPLC) which were used for calculation of routine pharmacokinetic parameters. Repeated measures ANOVA and Tukey's HSD test were used for statistical analysis. Results: Pharmacokinetic parameters for acetaminophen in our volunteers were consistent with literature values. The mean AUC $\pm$ SD for the control and the 1, 2, and 3 hour groups were 221 $\pm$ 54, 154 $\pm$ 71, 206 $\pm$ 67, and 204 $\pm$ 58 mg/L.hr, respectively. The one hour group was the only one differing from control ($p < 0.01$). The decrease of bioavailibility at one hour was 30.3% which is similar to previous studies. Conclusion: Our data do not support the administration of activated charcoal at 2 hours or longer after drug overdose.

67 AN ENHANCED PALATABILITY CHARCOAL IN THE MANAGEMENT OF PEDIATRIC POISONING VICTIMS.

Smith S, Cobaugh D, Dragalin V, Wax P, Lawrence R. *Finger Lakes Regional Poison and Drug Information Center, University of Rochester Medical Center, Rochester, NY*

Background: We hypothesized that an activated charcoal powder mixed in soda pop (ACSP) has a more rapid time to dose completion in pediatric poisoned patients than to a traditional AC/H2O slurry (AC). Methods: A double-blinded, randomized, prospective study was conducted using subjects enrolled in the emergency department of a university teaching hospital. Subjects aged 1 to 18 years requiring AC as part of their ED management were stratified by age and randomized to receive either ACSP or AC. The container was weighed before and after the dose. Time to dose completion was measured with a stopwatch. Statistical analysis included a two-sided sequential design with time to completion as the end point. The measure of treatment difference was the log hazard ratio. Log hazard ratio was defined as the ratio of the chance of completion per unit time between the AC subjects and the ACSP subjects. Hazard ratio < 1 indicated a relative benefit for ACSP subjects. Results: 82 subjects were enrolled. Subjects were stratified by age: Group I (1–6 yrs) included 37 subjects and Group II (7–18 yrs) included 45 subjects. Sequential test efficacy boundary was crossed when 48 dose completions occurred (27 in the ACSP group and 21 in the AC group). In Group I, 10/37 (27%) completed at least 80% of the charcoal dose within 1 hour: 6 received ACSP and 4 received AC. In Group II, 17/23 (74%) subjects who received ACSP completed their dose by a mean time of 10 min (3.4 standard error) and 21/22 (95%) subjects who received ACSP completed their dose by a mean time of 27 min (4.7 standard error). Across age groups, the adjusted (for sequential stopping rule) hazard ratio was 0.33. That is, chance of dose completion at any point in time was 3 times greater in the ACSP group. The ACSP treatment was superior to the AC treatment with an adjusted p-value = 0.0006. In Group I, hazard ratio was 0.629 and in Group II, the hazard ratio was 0.223. Conclusion: Time to completion of an activated charcoal dose is significantly shorter with ACSP when compared to AC. Differences were more significant in children aged 7 to 18.

68 THE EFFICACY OF ORAL DEFERIPRONE IN ACUTE IRON POISONING.

Berkovitch M, Livne A, Segal M, Talmor C, Bentur Y, Klein J, Koren G. *Department of Pediatrics, Assaf Harofeh Medical Center, Israel; Division of Clinical Pharmacology and Toxicology, Health Sciences Center, Toronto, Canada*

Background: Due to its prohibitive cost and need for parenteral administration, the standard iron chelator deferoxamine is not used in many patients with acute and chronic iron poisoning worldwide. Deferiprone is the first oral iron chelator to be shown effective in chronically iron overloaded thalassemia patients. Its efficacy, by oral administration, in acute iron poisoning has not been tested. Our objective was to determine whether orally administered deferiprone can reduce the mortality of rats following acute toxic oral doses of iron. Methods: Rats were administered 612 mg/kg elemental iron orally, corresponding to LD_{50} in the species tested. Two other groups received the same oral dose of iron followed by oral deferiprone; 800 mg/kg and 800 mg/kg followed by another dose of 800 mg/kg 2 hours later. Results: Coadministration of 800 mg/kg deferiprone with the iron decreased mortality from 30% to 6.6% after 2 hours ($p = 0.02$), from 40% to 16.6% after 12 hours ($p = 0.04$), and from 53.3% to 20% after 24 hours ($p = 0.007$). Mortality was also significantly decreased among animals coadministrated two repeated doses of deferiprone of 800 mg/kg with iron, to 0%, 9%, and 18%, two, twelve, and twenty-four hours post drug administration, respectively ($p = 0.04$; 0.05; 0.04, respectively). Histologically, there was a dose-dependent decrease in iron accumulation in the gastrointestinal tract. Conclusion: Orally administered deferiprone can decrease morbidity and mortality caused by acute iron overdose. Oral deferiprone holds promise in the treatment of iron poisoning in humans.

69 IN VITRO ABSORPTION OF LITHIUM BY BENTONITE.

Ponampalam R, Otten E. *Singapore General Hospital, Department of Emergency Medicine, Singapore; University of Cincinnati, Department of Emergency Medicine, Cincinnati, OH*

Background: Lithium poisoning is currently managed using a combination of supportive care and urgent haemodialysis in severe cases. The use of kayexalate as an adsorbent has been found to be effective in some studies, however, there are reports of complications such as hypokalaemia and intestinal necrosis. Bentonite is a known adsorbent that has been used in the management of paraquat poisonings. The purpose of this study was to determine the ability of bentonite to adsorb lithium. Methods: 4.5 g of lithium carbonate was dissolved in 1.5 litres of deionized water to form the stock

solution. 50 mL aliquots of this stock solution were added to 50 mL of either distilled deionized water (pH 7) or simulated gastric fluid (pH 1.2). Bentonite of either 0.75, 1.5, or 4.5 g was then added to simulate 5:1, 10:1, and 30:1 ratio of adsorbent to drug. Controls were made with no bentonite added. The resulting mixture was placed on a shaker for 5 minutes before being filtered. The filtrate was diluted and batch analyzed for lithium using atomic absorption spectrophotometry. Results: Bentonite decreased concentration of lithium recovered from the filtrate by 20.5% in deionized water compared to 48.1% in simulated gastric fluid at a bentonite lithium ratio of 30:1 (p value 0.0001). Conclusion: This study shows that bentonite is an effective adsorbent for lithium. The effect is enhanced in simulated gastric fluid. *In vivo* studies are being planned for clinical correlation.

Poster Session 2 — Saturday, October 2 — 10:00 am–4:00 pm
Abstracts #70–#141

70 CROTALID VENOM-INDUCED HISTOPATHOLOGIC CHANGES IN MAMMALIAN JOINTS.

Hill RE, O'Malley GF, Bogdan GM, Heard K, Lear K, Huffer W, Dart RC. *Rocky Mountain Poison & Drug Center, Denver Health, Denver, CO; University of Colorado Health Sciences Center, Denver, CO*

Background: There have been anecdotal reports of intra-articular crotalid envenomation but no studies have addressed this problem or provided treatment recommendations. Objectives: 1) To establish a model for crotalid venom-induced histopathologic changes in mammalian joints; and 2) To determine if intra-articular irrigation or systemic antivenom administration will prevent those changes. Methods: New Zealand White rabbits of both sexes, weighing 1.5–4.5 kg, were given intra-articular injections of 0.25 mL (20 mg/mL) *Crotalus atrox* venom to the right knee and 0.25 mL of 0.9% normal saline to the left knee (negative control). In phase one, envenomated knees without intervention were harvested at 20, 24, 30, and 96 hours. In phase two, envenomated knees were either irrigated with 3 mL of 0.9% normal saline 5 minutes after injection of venom or systemic antivenom was administered at 10 or 60 minutes prior to the intra-articular venom injection. These joints were harvested at 48 hours. Histopathologic changes were analyzed via microscopic examination and quantified by a blinded pathologist using a modified established scale for arthritis research. Results: In phase one, histopathological effects were apparent by 20 hours in envenomated knees and included the following: necrosis of muscle and soft tissue, inflammation and hemorrhage of the subsynovium, denuded cartilage surface with 100% proteoglycan loss and increased eosinophil density. Saline control knees had normal joint architecture with no signs of disruption or proteoglycan loss. In phase two, therapeutic interventions qualitatively reduced, but did not prevent venom induced structural damage. Irrigation did reduce synovial inflammation. Joints of rabbits treated with antivenom showed reduced soft tissue and synovial hemorrhage, as well as a reduction in the percentage of proteoglycan loss (60 min). Conclusion: This is the first report of a model to study intra-articular crotalid envenomation. The preliminary results indicated that joint irrigation and systemic antivenom could help reduce damage though future studies are required to further evaluate these therapeutic interventions.

71 THE ATYPICAL TREATMENT OF A SUSPECTED CROTALIDAE ENVENOMATION.

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Objective: To describe the presentation and management of a suspected pit viper envenomation with 240 vials of crotalidae polyvalent antivenin. Case Report: A 47-year-old male presented to the ED with a suspected and severe pit viper envenomation of the right finger 2 days prior. His right hand and arm were severely swollen and ecchymotic. Bruising extended into anterior, lateral and posterior chest. He was admitted with severe pain, tachycardia, hypertension, and a history of vomiting for 2 days. A consultation was provided and our center's protocol was initiated. A follow-up call revealed that our protocol had been discontinued and a non-medical herpetologist was contacted and his recommendations were begun. An infusion of antivenin was started at 4 vials per hour for 4 hours, then 2 vials per hour for

the next 112 hours, to a total of 240 vials of antivenin. Toxicology consults were offered repeatedly by our center without success. The only significant laboratory finding was a thrombocytopenia. The patient received 6 units of platelets. Pain control and antibiotic therapy were initiated. Follow-up office visits revealed full range of motion of affected extremity with minimal swelling of right hand and arm. No evidence of serum sickness was documented within the first two months of follow-up care. Conclusion: This is an atypical treatment of a suspected crotalidae envenomation with a positive outcome and no apparent adverse effects.

72 FIRST REPORT OF A SYMPTOMATIC SOUTH AMERICAN FALSE WATER COBRA ENVENOMATION.

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Background: *Hydrodynastes gigas* is a South American, rear fanged venomous colubrid snake. It is commonly referred to as the South American False Water Cobra because of its ability to rise and spread an extended hood. Mice studies have shown the venom and saliva to be hemorrhagic and proteolytic. *H. gigas* is an imported snake legally sold in the US. This is the first reported case of a symptomatic *H. gigas* envenomation. Case Report: An 18-year-old male pet store employee was bit on the left wrist by an *H. gigas*. The snake remained attached for about 1.5 min. The bite caused a small amount of swelling for which he took diphenhydramine twice within 3 hours. He reports receiving several bites previously with no effect. Approximately 9 hours after the bite, he experienced 3 brief episodes of muscle paralysis causing him to fall, unable to move or speak. At the time of paramedic arrival the swelling progressed from the fingertips to the elbow with numbness but no pain. On arrival to the ED vital signs were: HR 105 with 10 premature atrial contractions/min, RR 18, BP 120/80. The tachycardia resolved within 2 hours. The patient was observed overnight in the hospital with the arm elevated to heart level and received ceftriaxone. Localized symptoms persisted overnight with a slight increase in edema and pain to touch. Coagulation lab results remained normal (WBC 10.1, Hgb 16.1, Hct 43.5, Plts 210,000,000, Neut. 60%, PT 14.7, PTT 22.8, INR 1.1 fibrinogen 177, d-dimer <500). The patient was discharged home with cephalexin and a sling for elevation and immobilization. On telephone follow-up the patient reported swelling persisted for 5 days, but muscle pain and weakness persisted for 2 months. Conclusion: This is the first case reported of *H. gigas* envenomation causing significant local swelling, pain, muscle paralysis, and arrhythmias.

73 SEVERE ANAPHYLAXIS FROM GILA MONSTER ENVENOMATION.

Caravati EM, Dahl B, Crouch BI. *Utah Poison Control Center, University of Utah, Salt Lake City, UT*

Background: Gila monster and Crotalidae venoms share some common antigenic proteins (hyaluronidase, phospholipase A). Envenomations by venomous lizards are very rare and anaphylaxis has been reported only once due to a Gila monster bite. Case Report: A 44-year-old man was bitten on his right ring finger by a small Gila monster. Fifteen minutes later he developed vomiting, diaphoresis, and dizziness. Facial and tongue swelling occurred en route to the hospital. Upon arrival, 45 minutes after the bite, a systolic BP 80 mm Hg and dramatic facial, lip, and tongue swelling associated with inspiratory and expiratory stridor were noted. Lung exam revealed bilateral expiratory wheezes. Skin was without rash. WBC count was 21,900 mm^3. He responded to 2 doses of subcutaneous epinephrine, IV diphenhydramine 50 mg, IV methylprednisolone 125 mg, nebulized epinephrine and avoided endotracheal intubation. Right extremity edema and lymphangitis were also noted. He was discharged to home 24 hours after admission. Twenty years prior, the patient was envenomated by a Southwest speckled rattlesnake and received 6 vials of antivenin without acute hypersensitivity reaction. Conclusion: This is only the second reported case of acute anaphylaxis associated with Gila monster envenomation. This patient's prior exposure to rattlesnake venom may have increased his risk of an acute hypersensitivity reaction due to antigenic similarities between the venoms.

74 ATROPINE USE IN *CENTRUROIDES SCULPTURATUS* ENVENOMATION.

Suchard J, Hilder R. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Samaritan Regional Poison Center, Phoenix, AZ*

Background: Systemic envenomation by the scorpion *Centruroides sculpturatus* may result in serious neuromuscular toxicity, often with associated respiratory distress from bulbar nerve dysfunction compounded by excessive oral secretions. Most published recommendations for treating scorpion stings warn against using atropine as this may exacerbate cardiopulmonary toxicity. However, serious cardiopulmonary effects are rarely seen with *C. sculpturatus* stings, in

distinction from several other dangerous scorpion species of the world. We reviewed cases of *C. sculpturatus* envenomation reported to one PCC where atropine was given, to determine if such therapy was associated with any adverse effects and to describe clinical outcome. Case Series: Five cases occurred in 4 patients where atropine was administered to victims of scorpion stings since 1996. All occurred in children with respiratory compromise from excessive oral secretions and other signs of Grade IV *C. sculpturatus* envenomation. The only reported adverse effect was an asymptomatic exacerbation of tachycardia in one patient. In 3 cases the secretions cleared and no antivenin was given; 2 of these patients were discharged home from the ED and 1 was admitted for overnight observation. Antivenin was subsequently given in 2 other cases with continued respiratory compromise; 1 patient was discharged home from the ED and 1 was admitted for treatment of pulmonary infiltrates possibly related to aspiration. Conclusion: No serious adverse effects were reported in this case series from atropine given to reverse excessive oral secretions compromising respiratory status. Atropine appears to be safe in severe *C. sculpturatus* envenomation, and may obviate the need for antivenin or prolonged observation in some patients.

75 TELETOXICOLOGY IN THE RECOGNITION AND MANAGEMENT OF EXOTIC ARTHROPOD ENVENOMATION.

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Background: Species identification is a challenge to all poison centers that assist with bites and stings by venomous creatures. It is particularly problematic when the creature in question has been inadvertently imported from another country, and when the victim is at a distance from specialized care. Telemedicine technology enables diagnostic capabilities previously unavailable in the remote setting. We present the first case of remote arthropod identification using an established, statewide telemedicine program. Case Report: A 30-year-old woman employed at a supermarket in a small US/Mexico border town contacted the poison center after being "bitten by a banana spider" as she unloaded bananas from a shipment believed to have arrived from South America. She reported immediate pain and tingling at the site of injury. Out of concern that she might have been bitten by a *Phoneutria nigriventor*, she and the arthropod were referred to the nearest emergency room, a rural site recently incorporated into a statewide telemedicine network with a terminal near the regional poison center. Examination of the patient, via remote teleconsultation, revealed minimal physical findings. Review of shipping records revealed that the bananas had come from Chiapas, Mexico, and not from South America. Examination of the arthropod revealed that it was, in fact, a scorpion and not a spider. Magnification of the scorpion further revealed anatomic characteristics suggestive of the *Centruroides* genus, indigenous to Chiapas and potentially lethal to humans. Extended observation was recommended; local symptoms resolved and the patient never developed neurologic or pulmonary complications. Conclusion: Management of envenomation in rural areas may be complicated by confusion and lack of expertise in the identification of species involved. Teletoxicology, with real-time audio and video communications, enables improved recognition of species as well as enhanced patient assessment.

76 DEATH OF A 7-YEAR-OLD BY PRESUMPTIVE BROWN RECLUSE SPIDER BITE.

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Background: Pediatric fatalities from loxoscelism are extremely rare. Three mechanisms may be lethal:1. Hemolysis, 2. Coagulopathy, and/or 3. Sepsis/necrotizing fasciitis: We report the case of a previously healthy child who died as the result of hemolysis and coagulopathy. Case Report: A 7½-year-old female, 22 kg, was seen by a school nurse at noon for fever (100°F) and backache. Two–three hours later she had 103°F, headache, nausea/vomiting, a rash, and worse pain in her back. Mid-afternoon she visited a local physician who diagnosed "a spider bite". The girl denied being aware of a bite or seeing a spider. Mid-evening she appeared jaundiced and felt worse and was taken to the local ER. Her urine was red, Hb 9.6 gm/dL, platelets 111,000/mm^3. Started methylprednisolone, furosemide and alkaline diuresis therapy, and was transferred to the regional children's hospital. She was agitated but alert and talking. Admission labs at 00:45 revealed Hb 5.8 gm/dL, platelets 61,000/mm^3, WBC 31,300/mm^3, BUN 39 mg/dL, (13.9 mmol) Cr 1.8 mg/dL, (159 µmol) AST 334 U/L, T.Bili 0.6 mg/dL. DIC was evident: PT 23 sec, APTT 47 sec, Fibrinogen 203 mg/dL, D-dimers positive. Urine was red, 1–3 RBCs, 0–2 WBCs. Direct antiglobulin was negative. She had a 10 cm in diameter irregular ecchymotic tender area on her upper back with a dime-sized pale area toward the center. Vital signs were stable from transport until the sudden onset 2 hours later of V-tach to V-fibrillation to death. A complete autopsy revealed findings consistent with loxoscelism and no other definitive cause of death. Coagulopathy was seen as

bilateral subdural hemorrhages and 350 mL of pleural and peritoneal unclotted blood. There was necrotizing eosinophilic vasculitis in the skin, and perivascular eosinophilia in the pancreas and liver. Blood culture was negative. There was no cardiac lesion. Conclusion: This child suffered rapid onset of hemolysis and DIC leading to systemic hypoxia/ischemia and death within 24 hours of a presumptive brown recluse spider bite.

77 TRANSIENT MIOSIS AND CONTACT DERMATITIS FROM *BOMBINA BOMBINA* EXPOSURE.

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Background: The European fire-bellied toad (*Bombina bombina*) has become increasingly popular as a pet in the United States. Toxicity following human exposure to the toad has not been reported. We report a pediatric exposure with dermal and ophthalmologic effects. Case Report: A previously healthy 4-year-old child removed an adult *Bombina bombina* toad from its terrarium. Mother heard the child crying, and found him holding the toad in his hands in front of his face, and a clear liquid thought to be toad secretions was noted on his face. He was taken to a local ED by family members. On examination in the ED, the child had conjunctivitis, bilateral miosis and an erythematous, painful rash on his face. Dermal and ocular irrigation were performed in the emergency department. After three hours the patient's symptoms had resolved and he was discharged to home with no further sequelae. Conclusion: Bombina species are known to contain glands that secrete polypeptide toxins. Clinical effects of these toxins in humans has not been reported. This case suggests that exposure to *Bombina bombina* secretions can produce contact dermatitis, conjunctivitis, and miosis.

78 RED-BACK SPIDER (*LATRODECTUS MACTANS HASSELTI*) ENVENOMATION REQUIRING "HIGH-DOSE" ANTIVENOM THERAPY.

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Background: Red-Back Spider (RBS) antivenom (AV) effectively reverses the symptoms and signs of latrodectism with a negligible incidence of side effects. Doses of AV do not usually need to exceed 1 to 2 vials. We report 3 cases of latrodectism where the spider was identified, requiring previously unreported, and unusually large doses of AV to treat envenomation. Case-1: A 47-year-old male presented 24 hours following a RBS bite to the right calf. He complained of severe local pain, piloerection, sweating, and pain to both lower legs and feet. P 65, BP 145/80, T 37, GCS 15, restless and distressed. One ampoule RBS-AV was administered IM with worsening of symptoms. Five further vials of AV were given IM over the next 8 hours with resolution of all symptoms and signs after the sixth vial. Case-2: 49-year-old male presented 24 hours following RBS bite to right scapular region with local pain and sweating, pain to the shoulders, axillae, groins, and both legs. There was regional sweating to both lower legs. He was unable to sleep. P 80, BP 130/80, T 35, GCS 15. No relief with 2 vials RBS-AV IM. A further six vials were given IV over the next 8 hours with resolution of symptoms and signs after the eighth vial. Case-3: 66-year-old male was bitten on the great toe of his right foot. He presented 23 hours post-bite with pain and sweating of the whole right lower leg and fasciculation of the muscles of both lower legs. Five ampoules of RBS-AV were administered IV over the next 3 hours with no relief followed by one vial IM resulting in relief over the next 20 minutes. No patient was pretreated with antihistamines or epinephrine. None suffered allergic or serum sickness complications. Conclusion: As no other effective treatment for RBS envenomation exists, multi-repetitive dosing of RBS-AV should be considered if envenomation fails to respond to the usual 1–2 ampoule dose.

79 BOTULISM-LIKE SYNDROME AFTER INJECTIONS OF BOTULINUM TOXIN.

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Background: Botulinum type A toxin (BTA) is used to treat blepharospasm, laryngeal dystonia, cervical dystonia, hemifacial spasm, strabismus, and spasmodic torticollis. It is considered to be safe and effective as small doses are administered and BTA has a high affinity for motor nerve terminal receptors, which generally results in only localized effects. We report the first case of systemic botulism-like syndrome induced by IM injections of BTA which resulted in respiratory arrest. Case Report: A 30-year-old female presented to the ED 6 hours after having her second course

of BTA injections (8 × 12.5 units) into the posterior neck muscle. She was receiving BTA treatments for neck muscle spasticity resulting from an automobile accident. The patient developed ptosis, catatonia, and collapsed approximately 5 hours after her BTA injections. She then developed shallow respirations and loss of gag reflex which progressed to respiratory arrest. The patient was intubated and mechanically ventilated. She had a complete recovery within 18 hours before BTA antitoxin was obtained from the CDC. Conclusion: The doses of BTA used in this patient were well within therapeutic limits. While systemic toxicity is uncommon, subclinical EMG abnormalities distant from the injection sites have been described with therapeutic doses of BTA. Generalized muscle weakness has been reported after initial treatment and up to 5 years after long-term treatment. This is the first reported case of *respiratory failure* associated with BTA therapy. To our knowledge, this patient did not have risk factors for BTA induced muscle weakness (i.e., myasthenia gravis, multiple sclerosis, amyotrophic lateral sclerosis). Clinicians should be advised that systemic effects, although rare, can occur with local BTA therapy and may be life-threatening.

80 HERBAL MEDICINE USAGE IN EMERGENCY DEPARTMENT PATIENTS IN THE PEOPLES' REPUBLIC OF CHINA.

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Objective: The purpose of the study was to evaluate patterns of herbal medicine use by patients in an urban adult ED in the Peoples' Republic of China (PRC). Methods: A prospective, observational study was conducted over a one-month period in a large, urban ED in Shenyang, PRC. Survey forms which included demographic data, reason for herbal medicine use, type of herb(s) used, and source of herbal preparations were completed by ED patients with unrelated complaints. Results: 1030 patients were surveyed. 65% used some type of herbal medication and 60% of those patients reported improvement in their symptoms after use. 36% denied any change in symptoms, while 4% felt that their complaint worsened after herbal medicine use. 37% of patients had used some herbal preparation within 2 weeks of their ED admission, 9.6% within 2–6 weeks, and 53.4% more than 6 weeks prior to their ED admission. The majority of patients (70%) received the herbal preparation from their physician, 16.4% from a pharmacist, 6.9% from friends or relatives, and 6.7% from a "healer." The most common use of herbal preparations was for coronary disease (18.5%). Other patient indications included stroke (12.3%), low back pain (6.2%), cough (6.2%), cold (6.2%), dysmenorrhea (3.1%), and other miscellaneous illnesses. Conclusion: Although herbal remedy usage is becoming more common in the US, the incidence in PRC has not previously been evaluated. Further data from this population will improve the understanding of the benefits, adverse effects, and toxicity of these agents.

81 POISONING DUE TO RAW *GYROMITRA ESCULENTA* (FALSE MORELS).

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Background: *Gyromitra esculenta* contains the toxin gyromitrin and others but they continue to be eaten despite warnings of toxicity and carcinogenicity. Few cases of poisoning have been reported from western North America. Great variability in symptom severity occurs and it is not known whether this is due to species variation or individual susceptibility. Hepatic, GI, neurologic, hemolytic, and renal toxicity may occur. Case report: An Asian couple consumed an unknown quantity of raw *G. esculenta* at 1400 h; they had cooked and eaten this mushroom previously without problem. Two hours post ingestion vomiting and epigastric distress developed. They went to the ER at 0300 h but due to language difficulties, the history of mushroom ingestion was not elicited and they were discharged after symptomatic treatment. The following day, 31 hours post ingestion, they were admitted to hospital with icterus, protracted vomiting, and intense abdominal pain. They were rehydrated and given pyridoxine, meperidine, and dimenhydrinate. Bilirubin (t) peaked on day 1 (63 micromoles/L) post ingestion in the female, and day 2 (46 micromoles/L) in the male. The female's INR was 1.3 on days 1 and 2 post ingestion. Peaks of LD 693 U/L and AST 431 U/L were reached on day 4 post ingestion in the female, and on day 2 (LD 236 U/L) and day 6 (AST 116 U/L) in the male. The male was discharged day 6 post ingestion, and the female day 7, both with some nausea and GI discomfort. Conclusion: Consumption of *G. esculenta* is not recommended. Toxin levels decrease with cooking but whether carcinogenicity or other toxic effects are reduced is unknown. Eating raw false morels is especially foolhardy.

82 THE RETURN OF "THE GREEN FAIRY" TO LONDON: IMPORTATION OF ABSINTH(E) TO THE UNITED KINGDOM, DECEMBER, 1998.

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Background: Absinthe was banned in most European countries in 1915 due to its association with crime and general social disruption. In the winter of 1998–99, lay press reported its importation from the Czech Republic into the UK where it was never banned. The purpose of this descriptive study was to investigate the importation of "Hill's Absinth," its use in London, to examine associated cases of poisoning or adverse effects, and to determine if it contains thujone. Methods: We traveled to London to interview the importers of "Hill's Absinth," Green Bohemia, the director of the medical toxicology unit at Guy's and St. Thomas' Hospital National Health Trust, administrators in the Ministry of Agriculture Fisheries and Food (MAFF), and local liquor dealers and bartenders. Results: "Hill's Absinth" is marketed as "the drink for the new millennium." Before legal importation, it was independently tested for purity and E.U. standards at the U of Prague. It is currently sold in over 300 bars in London, at a price of £8 ($12)/glass, marketed toward drinkers who are aware of its historical past, and made popular by "Brit Art" drinkers. Absinthe is available in off-license shops at a price of £50 ($75)/bottle. Despite its popular appeal, there have been no absinthe related reports of adverse effects or neurotoxic poisonings at the London Medical Toxicology Unit. Analysis of "Hill's Absinth" at MAFF reveals it to contain: "70% ethanol by volume, vanilla flavoring, and brilliant blue coloring." Thujone levels are non-detectable. Conclusion: The current fad of "Hill's Absinth" in the UK is of historical and sociological interest, but is likely to be of no significant toxicological danger, except for that due to its high ethanol content. At the time of this printing, drinkers in France are lobbying to repeal the ban on absinthe which their country has held since 1915.

83 ALTERNATIVE MEDICINE TOXICITY: DIGITALIS POISONING!

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Background: Foxglove is a common plant both in the wild as well as cultivated for decorative use in northern climates. The leaves can reach 3 feet in length. Digitalis is contained both in the leaves and in the stems. Case Report: A 46-year-old Asian woman without any previous cardiac history presented to an emergency department (ED) after ingesting 4 large foxglove leaves 4 hours previously. (She had mistaken the plant for comfrey leaves intended for herbal use.) She had a sudden onset of nausea, lethargy, dizziness, and weakness and came into the ED. After finding notable bradycardia she was admitted for observation and continued to have nausea and vomiting. Treated with O_2, IV fluids, and 12.5 mg of promethazine for nausea, her EKG showed sinus and junctional bradycardia with intermittent second degree AV block. Her continuously monitored heart rate transiently dipped into the 30s. Her digoxin level was 0.8ng/mL (normal levels for patients on digitalis = 1.9 or less) and her potassium was 4.5 mEq/L. While the nausea resolved shortly, junctional rhythms and a second degree block persisted. She was then given 2 vials of Digibind—approximately 24 hours after her original ingestion. Subsequently her digoxin level remained 0.8 ng/mL. When her bradycardia and AV block persisted, she was given 2 additional vials of Digibind some 48 hours after her original ingestion without notable effect. Two days later she was, however, discharged with transient AV block, but was ambulating without symptoms. Conclusion: Rarely are accidental plant ingestions of serious consequence—unless "a meal is made of the plant." In the case of foxglove ingestion, the measured digitalis level does not necessarily reflect the all cardiac alkaloids that are present in serum.

84 FATAL HYPERMAGNESEMIA FROM A DIETARY SUPPLEMENT.

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Background: Alternative health care and dietary supplements are becoming increasingly popular for adults and children. However, many patients do not report the use of these medications to their own physicians. We report a fatal case of hypermagnesemia in a child due to the overuse of supplemental magnesium oxide (MgO). Case Report: A 2.5-year-old neurologically impaired male with history of normal renal function presented to the emergency department in full cardiorespiratory arrest. He was resuscitated with 100% O_2 and epinephrine and transferred to the pediatric ICU for further management. The mother reported that the child was under the care of a nutritionist who recommended a variety of multivitamins and minerals. The mother was instructed to adjust the dose of MgO supplements according to the child's bowel patterns. She denied reporting any of these supplements to her own pediatrician. Initial labs included Mg 20.3 mg/dL (3.2 mM; normal <1.2 mM) and ionized Ca 0.9 mM (normal 1.08–1.34). During his ICU course, the patient required several catecholamine infusions and transesophageal pacing for persistent bradycardia and heart block,

presumably secondary to hypermagnesemia. After 3 hours of hemodialysis, the Mg level decreased to 7.6 mg/dL. The patient continued to have bradycardia and hypotension despite increasing catecholamine support and pacing. The patient expired within 24 hours of admission. Conclusion: In this patient with normal renal function, excessive MgO supplementation may have contributed to irreversible heart block and death. Physicians should be aware of the popularity of alternative medicines and should counsel patients on the efficacy and potential hazards of these treatments.

85 RECIPE FOR NUTMEG (*MYRISTICA FRAGRANS*) ABUSE.

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Background: Displeasurable and frightening side effects are often associated with the abuse of nutmeg and thus occasionally generate emergency departments referrals. We report a young patient's first time experiences with nutmeg and review its characteristics and mechanisms. Case Report: A 13-year-old female ingested 15–24 g of nutmeg, over a 3-hour period and smoked and shared 2 capsules and 2 joints of marijuana. 1–2 hours later, bizarre behavior and visual, auditory and tactile hallucinations developed. She slept 2–4 hours and awoke with tactile hallucinations, nausea, gagging, hot/cold sensations, and blurred vision. In the ED, 9–10 hours post-exposure, she complained of numbness, double and "triple" vision, headache, and drowsiness. Her blood pressure was 110/60, pulse 88, resp. 18, and temperature 98.4° F. Nystagmus, muscle weakness, and ataxia were present. She received 50 g of activated charcoal. The rest of the physical exam and adjunctive tests were normal. Except for complaints of dizziness and visual changes, her 2-day admission was uneventful. Discussion: To facilitate ingestion, the nutmeg was put into 00–000 gelatin capsules (0.79–1.26 g/capsule) and 19 were taken by the patient "to feel good for a long time." The method was obtained from a book and may be easily found on the *Internet*, along with users' personal experiences with nutmeg, and other suggestions for obtaining a "legal high." Although the CNS activity of nutmeg is often postulated to result from biotransformation of its chemical components to "amphetamine-like" compounds, this has not been proven and is unlikely to occur *in vivo*. Consideration must be given to the structural similarities of these compounds to substances with CNS neuromodulatory activity. Conclusion: The long known psychedelic properties of nutmeg perpetuated in publications and highlighted in resources like the *Internet*, will continue to be rediscovered, especially by first-time pleasure seekers.

86 FALSELY ELEVATED DIGOXIN LEVEL OF 45.9 NG/ML DUE TO INTERFERENCE FROM HUMAN ANTI-MOUSE ANTIBODY.

Ingels M, Rangan C, Morfin J-P, Williams S, Clark R. *California Poison Control System, San Diego, CA*

Case Report: A 77-year-old man with a history of chronic renal failure, hypertension, NIDDM, dilated cardiomyopathy, cutaneous T cell lymphoma, and B cell lymphoma presented to the emergency department with a history of 2 weeks of progressive weakness. His medications included aspirin, glyburide, potassium chloride, enalapril, ethacrynic acid, interferon, and digoxin 0.125 mg per day. He had a heart rate of 40–62 bpm, with a blood pressure of 137/50. His physical exam was unremarkable. EKG showed a sinus rhythm of 62 bpm with 1st degree AV block and occasional PVC's. Serum creatinine was 2.4 mg/dL, (212 μM/L) which did not significantly differ from previous values. The digoxin level, run on Synchron CX System™ using the DIG™ reagent kit (Beckman) was 45.9 ng/mL. A repeat blood sample had a level of 44.8 ng/mL. This sample was sent to a different laboratory, where the level was determined to be 41.2 ng/mL. The patient was admitted to the hospital. No Digibind® was given. He remained stable and was discharged the next day, off digoxin. His laboratory samples were sent to a reference lab, where it was determined that the elevated results were the result of a human anti-mouse antibody in his serum that interfered with a reagent. When his samples were retested using the DIGN™ (Beckman) and Dig II™ (Microgenics) assays, results were 1.3 ng/mL and 1.39 ng/mL, respectively. Conclusion: We present a case of a patient with an extremely high digoxin level that resulted from the presence of an anti-mouse antibody in his serum. This antibody does not cause interference in assays with newer reagents. Physicians should be aware of this potential interference with assays employing mouse monoclonal antibody reagents.

87 DEATH CAMUS: MISTAKEN IDENTITY AT AN HERB FARM.

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Background: Death camus (*Zygadenus* species) is a perennial weed often mistaken for edible plants such as wild onion and sego lily. The bulb and flower contain veratrum alkaloids which may cause gastrointestinal and cardiovascular effects. Human fatalities are rare. We report a case of 7 people ingesting death camas bulbs and developing various

degrees of toxicity. Case Reports: Seven workers at an herb farm ingested what they thought were raw sego lily bulbs. Within 4 hours of ingestion, a 50-year-old male developed abdominal pain and hematemesis. On arrival at the emergency department (ED), he was bradycardic and hypotensive. QRS widening was noted on his ECG. The plant was then identified as death camus. He was given IV fluids and admitted to the hospital overnight. Within 5 hours of ingestion, 2 more patients presented to the same ED. One of the patients, a 36-year-old female, had nausea, vomiting, sinus bradycardia (44 bpm), and QRS widening. She was admitted and required treatment with atropine and IV fluids. The third patient, a 49-year-old male, presented with hypotension and complained of dizziness. He was admitted overnight and treated with IV fluids. The symptoms of these three patients resolved within 24 hours. During the same time frame, 3 of the other patients presented to another hospital ED. All had abdominal pain but were hemodynamically stable. They were observed in the ED and discharged that evening without symptoms. The last patient remained asymptomatic and was not evaluated in a health care facility. Conclusion: These cases illustrate the spectrum of toxicity of the veratrum alkaloids found in death camus. Plant misidentification continues to be a leading reason for toxic exposures.

88 PROLONGED CARDIOTOXICITY FROM POISON LILY (*VERATRUM VIRIDE*).

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Background: Ingestions of *Veratrum Viride* are rarely reported. This case resulted from misidentification of leeks and resulted in severe prolonged cardiotoxicity. Case Report: A 51-year-old otherwise healthy male presented to the emergency department 45 minutes after ingesting a soup made with boiled "leeks." The patient reported that ten minutes after ingestion, he experienced severe nausea and vomiting, lightheadedness, and somnolence. Initial physical examination was significant for depressed mental status and sluggishly reactive equal pupils ranging 4 mm–2 mm. The patient's heart rate was 42/min and blood pressure was 90/p requiring multiple doses of atropine along with dopamine. The patient was vomiting every 10–15 minutes despite aggressive antiemetic therapy. After 2 hours, substernal chest pressure was noted concurrent with ST segment depression. Repeat ECG showed a new right bundle branch block with persistent V2-V6 ST segment depression. Initial laboratory results including CBC, SMA-7, LFTs, cardiac enzymes (CK, CKMB, Troponin I), amylase, lipase, and urine toxicology screen were all normal. At this time, the regional poison control center botanist identified the plant brought in with the patient as *Veratrum viride*. The patient improved slowly over the next 24 hours, although bradycardia and heart block persisted for approximately 48 hours. Conclusion: Although *Veratrum viride* ingestions are rarely reported in the literature, small ingestions can result in severe cardiotoxic effects. These are thought to be due to sodium channel effects and usually successfully managed supportively.

89 SEVERE UVULAR ANGIOEDEMA CAUSED BY INTRANASAL ADMINISTRATION OF *ECBALIUM ELATERIUM*.

Eray O, Tuncok Y, Eray E, Gunerli A, Guven H. *Dokuz Eylul University School of Medicine, Department of Emergency Medicine, Pharmacology and Internal Medicine, Izmir, Turkey*

Background: *Ecbalium elaterium* (Cucurbitaceae), squirting cucumber, is a plant from Mediterranean countries. Juice from its fruit has been used for the treatment of sinusitis as a folk medicine in Anatolia, Turkey by nasal aspiration since A.D. 20–79, according to the Materia Medica by Dioscorides. All parts of the plant were reported to be toxic particularly the gherkin-like green fruits, exploding when ripe. There are two cases described in the literature of ecbalium poisoning. We present a case of life-threatening uvular angioedema associated with nasal aspiration of *Ecbalium elaterium* (squirting cucumber). Case report: A 54-year-old woman presented to the emergency department with shortness of breath and sore throat after intranasal administration of *Ecbalium elaterium* as a folk remedy for her sinusitis. The patient's history included nasal aspiration of juice of the squirting cucumber (*Ecbalium elaterium*) for acute maxillary sinusitis. An airway obstruction due to severe uvular angioedema was detected by physical examination and confirmed by airway (lateral C-spine for soft tissue) X-ray. The patient was treated with 100% oxygen with mask, 0.3 mg of epinephrine subcutaneously, and 80 mg of prednisolone intravenously. Renal and hepatic function tests of the patient were found to be normal. After a 24 hour observation period, the patient was discharged consistent with her previous state of health. Conclusion: Uvular angioedema due to *Ecbalium elaterium* may be life-threatening and require emergency treatment. The use of *Ecbalium elaterium* fruit juice as a folk medicine for sinusitis should be taken into consideration with patients presenting to emergency departments with dyspnea and uvular edema.

90 TOXIC HEPATITIS INDUCED BY HERBAL MEDICINES.

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Background: With the escalating interest in alternative medicine, self-treatment with medicinal herbs has become increasingly common. We present three cases of serious hepatotoxicity that resulted from the therapeutic use of herbs. Case Series: Case 1) On the advice of her homeopath, a 42-year-old female took 3 herbal products for insomnia for 2.5 months, until she developed abdominal pain and jaundice. The peak ALT was 3386 U/L. Viral serologies were negative. Analysis of one product, An Shu Ling (*Stephaniae sinica*) detected 1-tetrahydropalmatine, the active ingredient of Jin Bu Huan, a banned Chinese herbal known to cause hepatitis and CNS depression. Case 2) A previously healthy, 39-year-old female began taking cleansing herbs to treat her liver. After becoming progressively jaundiced, she took 2 capsules of chaparral, as advised in an herbal therapy book. She became markedly jaundiced, developed fulminant liver failure and required orthotopic liver transplantation. No histologic evidence for a specific etiology for liver failure was identified. Case 3) A 46-year-old Laotian woman took 4 herbs (Kaub ib, Ncas liab, Ncas dawb, Hmab iab) to induce an abortion. Two days later, she developed nausea and vomiting, with ALT 514 U/L, bilirubin 22.5mg/dL, ammonia 93 mcmol/L, and INR 5.3. She deteriorated to grade IV hepatic encephalopathy, but the family refused liver transplantation. She eventually recovered, and reported prior use of these herbs with successful abortion and no adverse effects. Conclusions: Some herbal remedies can cause severe hepatic injury. Underlying mild liver disease may predispose an herbal user to catastrophic liver failure. Lack of product regulation, inadequate supporting scientific evidence, and ill-informed herbal practitioners make the use of herbals an unsafe practice.

91 DANGER IN THE DOCTOR'S OFFICE: TWO CASES OF SEVERE NEUROLOGIC SEQUELAE AFTER INGESTION OF PODOPHYLLIN.

Juurlink DN, Sellens C, Thompson M, McGuigan MA. *Ontario Regional Poison Centre, Toronto, Ontario, Canada*

Background: Podophyllin is a compound used topically to treat condylomata. We describe two cases of podophyllin ingestion which occurred in the offices of family physicians. Case 1: A 38-year-old male ingested 25 mL of a 25% podophyllin mixture. He was treated promptly with activated charcoal, followed by glutamic acid 650 mg tid for 1 month. A prolonged paralytic ileus ensued, as did generalized paresis with areflexia. Serial EMG and nerve conduction studies progressed from normal to showing a severe sensorimotor axonal neuropathy and diffuse myopathy, becoming maximal by 5 weeks post-ingestion. The patient remained in hospital for 5 months prior to transfer to a long-term care facility. Case 2: A previously reported 18-month-old male ingested approximately 10 mL of 25% podophyllin in benzoin tincture. No initial treatment was given. Over the ensuing 26 hours he developed metabolic acidosis and coma, followed by bone marrow depression. Serial CT scans documented progressive generalized cerebral atrophy, with evidence of demyelination at 30 days and partial recovery at 6 months. Nerve conduction studies documented an axonal and demyelinating neuropathy, which improved slightly by the second month. Over the following two years, the patient continued to demonstrate severe developmental delay, with gross motor skills improving to a greater extent than cognitive ability. Conclusions: Podophyllin ingestion can cause a variety of severe and long-lasting neurologic abnormalities. Family physicians who use podophyllin should be aware of this hazard and store the compound appropriately.

92 METHEMOGLOBINEMIA CAUSED BY CUTANEOUSLY ABSORBED ASAFOETIDA.

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Background: Extracts of asafoetida (*Ferula asafoetida*), a plant indigenous to the Middle East, is used as an herbal remedy for gastrointestinal symptoms. Case Report: A four-week-old boy of Thai heritage was brought to the emergency room with a one week history of vomiting, diarrhea, lethargy, and poor oral intake. In the emergency room, he was tachypneic and hypotensive. His blood was noted to be the typical chocolate brown color consistent with methemoglobinemia. The methemoglobin (MetHb) level was 55.6%, and he was treated with methylene blue. Twelve hours after presentation, the patient's MetHb level was down to 5.9%. Because of colic, the mother had been applying asafoetida extract every other day for two weeks to the child's abdomen after his evening bath. The extract was then left on overnight. There was no enteral administration of the extract. The patient remained in the hospital for two weeks with persistent diarrhea. However, the MetHb level never rose above 4% during the remainder of the hospitalization. Discussion: A previous case report concluded methemoglobinemia was caused by ingestion of asafoetida extract in a five-

week-old boy. This report also showed that asafoetida extract added to hemoglobin F *in vitro* produced MetHb, yet failed to induce MetHb when added to hemoglobin A *in vitro.* It is well documented in the literature that diarrheal illness in neonates can produce methemoglobinemia. However, in this specific case, the patient's diarrhea persisted for many days without recurrence of methemoglobinemia. Conclusion: Topically applied Asafoetida extract can produce methemoglobinemia in neonates.

93 PROFOUND METHEMOGLOBINEMIA INDUCED BY DERMAL AND INHALATION EXPOSURE TO ANILINE DYE.

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Background: Previous human case reports of significant methemoglobinemia (MetHb) induced by aniline have resulted from ingestion. We report 2 patients with dermal and inhalational exposure to low concentration aniline who developed significant MetHb. Case Report: Two Hispanic machine operators in a polymer factory attempted to clean a chemical spill that occurred just prior to their shift. The chemicals were identified as Nylon 66 and nigrosine (98% solvent black 7, 2% aniline). About 1.5 hours into the exposure, both workers had lightheadedness and headache, followed shortly by nausea and dyspnea, which persisted despite a 30 minute rest with fresh air. Neither worker wore any protective gear. EMS was called and medics noted that both workers had contaminated clothes and skin. Pulse oximetry was about 70% in each; both received 100% oxygen, their clothing was removed and they were decontaminated for 20 minutes in a shower on ED arrival 6 hours after exposure began. Case 1: 24-year-old male with BP 134/51, P 103, RR 22, and O_2 saturation 60% on 100% non-rebreathing mask. His initial MetHb level was 61.5%. Case 2: 37-year-old male with BP 138/65, P 106, RR 24, and O_2 saturation 74% on 100% non-rebreathing mask. His initial MetHb was 72%. Both patients received 1 mg/kg of 1% methylene blue (MB) with dramatic improvement in both symptoms and vital signs. Repeat MetHb levels 75–90 min after MB administration were 25% (case 1) and 39% (case 2). They were admitted for 23 hour observation without return of symptoms or sequelae. MetHb levels in both patients were zero the following day. Conclusion: Severe MetHb occurred in 2 previously healthy workers with a 3-hour exposure via dermal and inhalation routes to Nylon 66 and nigrosine. The only ingredient with known oxidizing potential was 2% aniline. The MSDS sheets did not identify the potential for MetHb. Potentially fatal MetHb (70% or greater) can occur from dermal and/or inhalation exposure to oxidizing chemicals.

94 EMLA-INDUCED METHEMOGLOBINEMIA (MetHb) AND LIDOCAINE TOXICITY.

Hahn I, Hoffman RS, Nelson LS. *NYC Poison Control Center, New York, NY*

Background: EMLA cream, a mixture of lidocaine and prilocaine, is a topical anesthetic used for minor procedures. Although EMLA-induced MetHb has been reported in children, toxicity in adults is unreported. We describe the first adult to develop MetHb and simultaneous lidocaine toxicity from dermal exposure to EMLA for an elective procedure. Case Report: A 30-year-old female presented to the emergency department 4 hours after a laser epilation treatment. For the procedure, 150 g of EMLA cream (5 tubes) had been applied to her lower extremities under occlusive dressing for 1 hour. About 1 hour later, she began to experience lightheadedness, dyspnea, tongue numbness, muscular twitching, and formication on her legs/back. She denied visual/auditory hallucinations. Her only medications were sertraline and an oral contraceptive. On examination she was alert, but noted to have mild respiratory distress and perioral/acral cyanosis, which did not resolve with supplemental oxygen. Her vital signs were significant for a respiratory rate of 20/min and her pulse oximeter read 84% saturation while on 100% oxygen. Her lungs were clear and her heart sounds were normal. Bilateral pretibial first degree burns were still present from a prior laser treatment 1 week ago. The remainder of her physical examination was normal. An ABG was: pH, 7.45; pCO_2, 36 mm Hg; PO_2, 385 mmHg, and her MetHb was 20% by co-oximeter. The patient received methylene blue, 50 mg IV, over 5 minutes and improved within 1 hour. A repeat MetHb level was 2.7%. Her lidocaine level (drawn at that time but measured 1 week later) was 0.68 mcg/mL confirming systemic absorption from the EMLA cream. Symptoms never recurred and the patient was discharged. Conclusion: MetHb and clinical lidocaine toxicity probably resulted from inappropriate EMLA application to damaged skin. The low lidocaine level may have resulted from the delay between acquisition and measurement. As more patients receive EMLA for minor cosmetic procedures, clinicians should be aware of the potential for adverse effects for this drug and the contraindications to its use.

95 ELEVATED SERUM COPPER LEVELS AND METHEMOGLOBINEMIA FROM RESIDENTIAL EXPOSURE TO COPPER NAPHTHENATE.

Kim S, Chomchai, S. *California Poison Control System, San Francisco Division, San Francisco, CA*

Background: Despite widespread use of copper naphthenate (CuN), reports of toxicity are rare. We report the first case of elevated methemoglobin (MetHb) level following exposure to CuN in a residential setting. Case report: A 41-year-old male, 40-year-old female, and children aged 2, 3, and 5 lived in a rented single family home. Before they moved in, CuN was sprayed heavily on the hardwood floor. During the application, the contents of the can spilled onto the floor, soaking the wood and dripping down to the crawl space below the house. The landlord put plastic sheeting in the crawl space, covered the floor with carpet, but did not alert the tenants about the incident. After moving into the house, mother developed headaches, dizziness, and nausea. The children appeared to be more tired than usual. Three months after moving into the house, the family learned of the CuN spill. At this time, lab work on the mother revealed a MetHb level of 16%. No treatment was given. The heater was shut off and the house ventilated. A repeat MetHb level 12 days later was 0.9%. None of the other family members had elevated MetHb levels. Serum copper levels of the family members were as follows: 41-year-old 100 mcg/dL, 40-year-old 145, 5-year-old 165, 3-year-old 200, and 2-year-old 220 (normal 65–145 mcg/dL). LFTs and CBCs were WNL. The family has since moved out of the house. Repeat copper levels and LFTs were recommended but have not been done. Discussion: Bluhm *et al.* reported elevated blood and urine copper levels in family members exposed to CuN sprayed on the inner foundation of their home. However, elevated MetHb level from this source of copper has not been reported previously. Conclusion: Clinicians need to be aware of the potential for elevated serum copper levels and methemoglobinemia following inappropriate use of CuN in the home.

96 VENTRICULAR DYSRHYTHMIA AND SUBSEQUENT DEATH IN A PATIENT WITH ACUTE PROMYELOCYTIC LEUKEMIA TREATED WITH ARSENIC TRIOXIDE.

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Background: Arsenic compounds have been used as medicinal agents in the treatment of psoriasis, syphilis, and at present only for trypanosomiasis involving the CNS. In addition, they are well respected poisons used for homicide and suicide. In the past three years clinical studies have reported that arsenic trioxide (As_2O_3) treatment can induce complete remission of patients with acute promyelocytic leukemia (APL) who have failed chemotherapy treatment with all-*trans*-retinoic acid. The most recent study in which 11 patients received an average cumulative dose of 360 mg of arsenic trioxide (range, 160 to 515) taken orally as outpatients reported a median duration of remission of only 5 months (range, 1 to more than 9). Acute arsenic poisoning manifests with symptoms of vomiting and diarrhea as well as ventricular dysrhythmias, myocardial dysfunction, and hypotension. We report a fatality which is suggestive of arsenic poisoning in a patient with APL who was treated with this newly approved experimental chemotherapeutic agent. Case Report: A 29-year-old man with a history of APL that had been refractory to retinoids and cytotoxic chemotherapy was treated in the hospital with As_2O_3 20 mg orally daily for 35 days (cumulative dose of 700 mg). The patient had no previous history of cardiac disease, had a normal admission ECG and electrolytes, and was not reported to be on cardiotoxic medications. Three days after the As_2O_3 regimen was stopped the patient had multiple episodes of refractory ventricular tachycardia including Torsades de Pointes. Despite treatment with lidocaine, bretylium, magnesium, and cardioversion the patient died approximately 24 hours after onset of dysrhythmia. Although a forensic autopsy was refused, a postmortem blood arsenic level of 69 mcg/L was detected in a sample that was taken during the cardiac arrest. Conclusion: The association of arsenic trioxide therapy with a lethal dysrhythmias and the absence of an alternative cause of death raises the concern of causality of arsenic in this case. Future studies using arsenic to treat APL should systematically evaluate the potential for cardiac and systemic toxicity.

97 A CASE OF SEVERE MERCURIC SULFATE INGESTION TREATED WITH 2,3-DIMERCAPTOPROPANE-1-SULPHONATE (DMPS) AND HI-FLOW HEMODIAFILTRATION.

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Background: There have been previous cases of inorganic mercury poisoning treated with hemoperfusion and hemodialysis, both with little success at removal. We report the first case treated with hemodiafiltration. Case Report: A 40-year-old man ingested an estimated l g of mercuric sulfate. He presented with hematemesis and melena, and rapidly deteriorated in

the ER requiring intubation and ventilation. He was transferred to the poisons center ICU for further management. At 4½ hours post ingestion he was commenced on IV DMPS 250 mg 4 hourly for 4 days, then 8 hourly for 7 days, followed by 200 mg 12 hourly orally for a further 8 days. He rapidly became anuric and was commenced on hi-flow hemodiafiltration (blood flow rate 150 mL/min) at 7½ hours post ingestion (for a total of 14 days) for both renal support and to improve mercury clearance. EGD revealed confluent gastritis with two gastric ulcers requiring injection. He was discharged from the intensive care unit on day 14. He required a further 8 sessions of hemodialysis for renal support, the last of these on day 39. He was discharged from hospital entirely well on day 50, with a serum creatinine of 2.3 mg/dL. (203 μM/L) Results: Initial blood mercury 1558 μg/dL. Initial filtrate mercury 138 μg/dL. Mean filtrate clearance over the first four days 5.8 mL/min, during 4 hourly IV DMPS; this dropped to 1.6 mL/min during 8 hourly IV DMPS. Total mercury cleared by hemodiafiltration 12.8 mg. Conclusion: Despite a very high initial blood mercury level full recovery occurred. The combination of hemodiafiltration and IV DMPS resulted in significant clearance, although at best this removed only 12.4 mg. Meticulous supportive care is critical to a good outcome in such patients.

98 PROLONGED CHELATION AFTER MERCURIC CHLORIDE POISONING.

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Background: Fixatives used for stool ova and parasites contain inorganic mercury. Case Report: A 2-year-old 14 kg male drank 13 mL of "Para-pak fixative" (3.4% mercuric chloride). Whole blood mercury level drawn one-hour post ingestion was 162 mcg/L (normal < 15 mcg/L). At 3 hours post ingestion the child was started on dimercaprol (4 mg/kg IM q 4 h). The child was kept NPO on maintenance IV fluids for 24 hours. Urine output remained >1 cc/kg/h. Initial 24 hour urine mercury concentration was 340 mcg/L (normal < 20 mcg/L). Endoscopy performed the second day of admission revealed no evidence of caustic injury. The hospital course was uneventful with no signs of gastrointestinal bleeding or renal failure. After 48 hours of dimercaprol, he was discharged to home on a two-week course of oral DMSA (200 mg tid). The patient was followed over the next 106 days requiring 3 courses of DMSA chelation (Table).

Days post ingestion	0–1	1–2	2–16	17	32 ~ 41	33	50	74 ~ 96	75	106
Urine Hg mcg/L	340*	110*		85		33	25		52	10
Chelation	BAL	BAL	DMSA		DMSA^			DMSA^		

* 24-hour collections, others 1st morning void. ^21 doses given irregularly.

Conclusion: This patient continued to excrete toxic mercury levels in his urine for three months after a single ingestion of a stool fixative. Prolonged chelation therapy may be necessary after ingestion of inorganic mercury.

99 MERCURY POISONING OF A NEONATE WITH MAJOR OMPHALOCELE.

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Background: The optimum treatment of omphalocele is immediate surgical repair. When this cannot to accomplished, the non-operative treatment of major omphalocele includes such techniques as painting the defect with mercurochrome, chlorhexidine, silver nitrate, povidine iodine, gentian violet, or isopropyl alcohol. None of these methods has been proven ideal because of significant toxicity. Mercurochrome, in particular, has been noted to cause mercury toxicity and death in the neonatal patient. Its use had been abandoned in most centers due to significant toxicity. We present a case of significant mercury toxicity due to the use of mercurochrome and the difficulties in diagnosis as well as treatment of this neonate. Case: The patient is a female born at a weight of 2.875 kg via elective c-section due to omphalocele. Initial measurement of the defect was 9 × 9 cm with the whole liver and most of the intestines contained in the sac. No other birth defects were noted. Primary surgical closure was unsuccessfully attempted and non-operative approach using a one time painting of the defect with 5% mercurochrome was employed. Approximately 16 hours after the mercurochrome use, the plasma mercury level was 1017 mcg/L. The toxicologist was consulted several days later when the patient became difficult to console and tremulous. Chelation, using DMSA, was started immediately (day #0). Successive levels measured were 177 mcg/L (day #1), 155 mcg/L (day #5), Pending (day #12). Conclusion: Omphaloceles are difficult to treat conservatively due to the toxicity of the agents used. The toxicity may be easily missed especially in the neonate. Therefore the clinician must have a high index of suspicion when these agents are used.

100 ELEMENTAL MERCURY INJECTED AT MULTIPLE SITES.

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Background: It has been stated that "accidental soft tissue deposition of liquid mercury appears to be an injury and not a poisoning." This may be true of small amounts of mercury, however, there are significant inflammatory local effects and in large or multiple subcutaneous dosages there may be significant elevation of mercury levels and the development of toxicity. Case Report: We report the case of a 23-year-old schizophrenic male who injected elemental mercury from thermometers at multiple body sites. These included: his earlobes, throat, both antecubital fossa, multiple fingertips, both buttocks, and scrotum. It is unclear whether this occurred at one time or over the course of a few months. Initially whole blood mercury levels were 260 ug/L, (urine 314 ug/dL). He underwent surgery to remove mercury from his left antecubital fossa, the only known injection site. He later told a fellow inpatient of another injection site which lead to a repeat exam and a metastatic X-ray survey which revealed multiple other sites. There was remarkably little evidence of vascular or lymphatic involvement. By this time he was developing significant localized inflammatory reactions and had a new tremor (complicated by the initiation of lithium although his levels were 0.6–1.2). He underwent the surgical removal of mercury from all readily accessible sites and was given DMSA (10 mg/kg 3x/d × 5 days, 10 mg/kg 2x/d × 14 days). Whole blood levels 4 months after significant debridement and chelation remain in the 40 ug/L range and the patient is asymptomatic. Conclusions: It is clear that the injection of mercury at multiple sites or in large amounts may lead to the elevation of mercury levels and possible toxicity. While history is difficult to obtain in some individuals, when intentional injection is suspected multiple other sites may also exist. Physical exam may miss some injection sites, they are, however, readily detected by radiography.

101 LARGE SUBCUTANEOUS ELEMENTAL MERCURY INJECTION.

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Background: Subcutaneous injection of elemental mercury is relatively rare. Most cases involve intentional injection of elemental mercury, either as suicide attempts, substance abuse, or occasionally machismo rites. Exposures usually require local excision, develop elevated mercury levels, and nearly half develop X-ray evidence of pulmonary emboli. We report a case of large subcutaneous elemental mercury injection without any systemic signs or symptoms. Case Report: A 26-year-old man presented to a local emergency department 1 week after a supposed accidental injection of elemental mercury into the dorsum of the left foot. He consistently denied injecting or otherwise introducing the mercury into his wound. Five days after the accident, the patient's foot was red, swollen, and painful so he attempted unsuccessfully to remove the mercury with a razor. Two days later, after progression of his local symptoms, he presented to the emergency department. X-rays showed large amounts of mercury in the subcutaneous tissues of his foot, casting doubt on accidental infiltration of the wound. He was taken to surgery where a large amount of mercury was removed using an image intensifier and suction, rongeur, curets, and NS lavage. All visible mercury was removed, and the wound was packed open and splinted with delayed primary closure of the wound occurring 3 days later. The patient was then released home with his wound healing well. A chest X-ray done during the ED admission showed no mercury emboli and he never had any respiratory symptoms. A spot urine mercury on admission was 14 ug/L (normal < 20; toxic $>$ 150). The patient never showed any other signs or symptoms of mercury toxicity such as rash, CNS changes, or GI upset. Conclusion: Despite a large subcutaneous injection of elemental mercury, and a week-long exposure, this patient did not develop any signs or symptoms of mercury toxicity.

102 ABSENCE OF RENAL FAILURE IN A MERCURIC CHLORIDE INGESTION WITHOUT CHELATION THERAPY.

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Background: Mercuric chloride is known to cause corrosive effects on the GI tract, as well as possible renal failure due to renal tubular toxicity. This is the first reported case of a patient with markedly elevated mercury level who had received no chelation and maintained a normal renal function. Case report: A 46-year-old woman was suspected of ingesting stool fixative agents containing a 15 mL vial of 4.5% mercuric chloride and a 15 mL vial of 10% formaldehyde. No GI symptoms were noted on presentation. The patient received lavage and activated charcoal; X-ray was negative for radio-opaque material in the GI tract. After 8 hours of observation, patient had two episodes of vomiting and, at ten hours, episodes of polyuria were noted. At that time, chelation therapy with succimer was recommended due to

concerns for renal toxicity. The patient refused all forms of medications, and consequently, chelation therapy was not initiated. Her renal function was normal during the 24 hours of observation in the intensive care unit, and the patient was transferred to a psychiatric facility where she was discharged after 2 days. Eight days post-ingestion, the admission serum and spot urine Hg level returned at 2,922 mcg/L and 3,517 mcg/L, respectively. Concerns were raised as to the possibility of delayed onset renal failure from such high levels of mercury. The patient was lost to follow up for four weeks, after which time her repeated renal functions were still normal and her whole blood mercury was 12 mcg/L. No urinary mercury was done. Conclusion: Despite the good outcome in our case, clinicians should be alerted to the fact that patients may initially present with minimal GI symptoms following a significant mercuric chloride ingestion. In addition, review of case reports suggested that onset of renal failure may be delayed as much as three days after presentation. Thus, an observation period of longer than 24 hours is perhaps needed to assure that renal function has been unaffected.

103 TELLURIUM INGESTION IN AN 18-MONTH-OLD MALE.

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Background: Acute human ingestions of tellurium are rare. We report a case of tellurium ingestion in a toddler. Case Report: An 18-month-old male was noted by his father to ingest an unknown quantity of silver blackening solution containing 1.7% tellurium dioxide in 60% HCl. He was noted to have several episodes of hematemesis and refused to swallow but never developed any evidence of neurological or respiratory compromise. Physical examination revealed a distinct and persistent garlic odor. Endoscopy showed erosions and exudate of the posterior oropharynx, the esophagus, and the dependent portion of the stomach consistent with a caustic ingestion. The dorsal surface of the tongue was black. Laboratory studies were unremarkable. A serum tellurium level was 200 mcg/L (33 mcg/L average) and a urine level was 56 mcg/L (normal 0.2–1.0 mcg/L). The patient's clinical symptoms of dysphagia from caustic ingestion continued to improve and he was able to tolerate p.o. intake within 24 hrs. With the exception of a persistent garlic odor, the darkened tongue, and elevated tellurium levels, no sequelae from the tellurium was noted on either physical exam or laboratory studies. Conclusion: There was no evidence of tellurium toxicity in an 18-month-old male who ingested a tellurium containing solution and had serum tellurium levels which were approximately 6 times normal.

104 ADVERSE EFFECTS OF REDUCED-DOSE d-PENICILLAMINE IN CHILDREN WITH MILD TO MODERATE LEAD POISONING.

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Background: Oral chelation with *d*-penicillamine (*d*-PCN) has proven efficacy in the treatment of mild to moderate lead poisoning. However, *d*-PCN is associated with a relatively high incidence of adverse effects (AEs) in standard doses of 25–30 mg/kg/day. Reduced-dose *d*-PCN may reduce the rate of AEs without a significant reduction in efficacy. Purpose: To examine the incidence of rash, WBC/platelet count depression, and abnormal urinalysis with *d*-PCN in a dose of 15 mg/kg/day given to children with blood lead levels <40 μg/dL. Methods: Retrospective analysis of results from all children who received *d*-PCN in 1996 at a Northeastern US lead poisoning treatment center. Results: During the study period 55 children received 66 courses of *d*-PCN. Mean age was 37.4 months. Mean pre-chelation blood lead level was 24 μg/dL (range 15–37 μg/dL) with a corresponding erythrocyte protoporphyrin (EP) level of 44 μg/dL. After 77 days of *d*-PCN blood lead level was reduced to a mean 16 μg/dL (mean fall 35%, $p = .005$) and EP was reduced to 28 μg/dL ($p = .009$). There were three episodes of rash (4.5%). During chelation, WBC fell below 5000/mm^3 in 7 cases (9.7%); there were no episodes of platelet count <150,000/mm^3. There were no cases of abnormal urinalysis. All AEs were transient and resolved during or immediately after chelation. The only patients who were prematurely terminated from therapy were those who developed rash; in all these cases the drug eruption was an isolated occurrence which resolved within 48 hours of diphenhydramine. Conclusions: Reduced dose *d*-PCN appears to maintain efficacy at reducing blood lead levels. Reduced dose *d*-PCN also appears to cause fewer AEs than previously reported. Observed AEs are benign and transient. Supported by a grant from the Agency for Toxic Substances and Disease Registry and the Association of Occupational and Environmental Clinics.

105 COMBINED SUCCIMER AND ENDOSCOPIC REMOVAL OF LEAD PELLETS.

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Background: Lead is a well-known childhood toxin that causes anorexia, malaise, and increases intracranial pressure leading to permanent brain damage. Case Report: A previously healthy 21-month-old girl was hospitalized following ingestion of two lead BB pellets. A KUB demonstrated two 0.4 cm rounded densities in the RUQ. Past medical history was unremarkable, physical and neurologic exams were normal. A blood lead level drawn prior to this exposure was 12 mcg/dL. On admission, serum lead level was 47 mcg/dL. Nasogastric polyethylene glycol (PEG) (10 mL/kg/h) was administered and repeat X-ray 24 hours later demonstrated pellets in the projection of the cecum. Succimer (DMSA) 100 mg tid was initiated. Twenty-four hours later, blood lead was 48 mcg/dL and X-ray demonstrated the position of the pellets to be unchanged in the RLQ. The patient was transferred for colonoscopy 72 hours post-ingestion. The pellets were removed endoscopically in two separate procedures following PEG (500 mL/h) colon preparation. Blood lead level the day after colonoscopy was 25 mcg/dL. The patient was subsequently discharged in excellent condition to complete the 19-day course of succimer. At follow-up ten days post-discharge, blood lead was 16 mcg/dL. Conclusion: This is the first reported case where succimer therapy was initiated while solid lead was still present in the GI tract. Succimer may have prevented further absorption of lead from the GI tract since blood lead level remained constant during the 24 hours after the initiation of therapy. It is unclear whether WBI affected the bioavailability of succimer. This combination of therapies appears to be efficacious as the patient's lead level was nearly halved in 24 hours after the initiation of therapy and the removal of the pellets.

106 LEAD POISONING IN LATE PREGNANCY DUE TO MATERNAL PICA.

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Background: Lead crosses the human placenta and placental lead levels correlate well with those in maternal plasma. Increased placental lead levels are associated with congenital abnormalities as well as neonatal lead encephalopathy. We report two cases of pregnant women with elevated peripartum lead levels due to pica. Case 1: An asymptomatic 32-week pregnant 17-year-old woman had a lead level of 130 mcg/dL and EP of 36 mcg/dL on her first prenatal visit. She admitted to eating an imported clay pot 3 days earlier. Her hematocrit was 30.7% and basophilic stippling was noted on her blood smear. An abdominal radiograph revealed lead fragments in her GI tract. The remaining pot was densely opaque radiographically and had a lead content of 22%. She received WBI with PEG-ELS at 2 L/h for 4 hours and $CaNa_2EDTA$ IV (1000 $mg/m^2/d$). After treatment with steroids for 2 days for fetal lung maturation a 2.4 kg child was delivered. The child's cord blood lead level was 78 mcg/dL and the mother's blood lead level was 48 mcg/dL at delivery. The child was treated with BAL IM (300 $mg/m^2/d$) $\times$ 1 day and $CaNa_2EDTA$ IV (1000 $mg/m^2/d$) for a total of 7 days and the lead level fell to 21 mcg/dL. The mother was treated with $CaNa_2EDTA$ for two more days and succimer $\times$ 2 days. The blood lead level decreased to 27 mcg/dL. Case 2: A 16-week pregnant 22-year-old woman had a lead level of 55 mcg/dL on routine screening tests. She stated that she had been eating soil from her yard. She was asymptomatic and fetal development appeared normal. No chelation therapy was initiated at that time. At term, the patient delivered a 5.4 kg child. A cord blood lead level of 78 mcg/dL and an EP level of 800 mcg/dL were found. The mother's lead level was 48 mcg/dL. The next day, the child's lead level decreased to 44 mcg/dL without therapy. Beginning the third day, both mother and child began lead chelation with $CaNa_2EDTA$ IV (1500 $mg/m^2/d$) $\times$ 5 days. Both appeared clinically normal. Conclusion: Pica during pregnancy may be associated with increase in both maternal and fetal lead levels. There is no consensus on therapy for pregnant mothers with elevated lead levels. Both sets of patients tolerated the treatment regimens well. Post-partum neonatal lead levels fall rapidly without treatment although the clinical implications of this fall are unclear.

107 THE TREATMENT OF LEAD POISONING—CLINICAL EXPERIENCE OF THE NATIONAL POISONS INFORMATION SERVICE (LONDON) 1997–98.

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Background: NPIS(L) is the main supplier of oral chelating agents for cases of lead poisoning in the UK. Results: From September 1997–December 1998 26 new cases presented: 16 children (10 M, 6 F; all <12 years age) and 10 adults (8 M, 2 F). Clinical features in the children (some had >1): encephalopathy (1), irritability (2), anemia (9), abdominal

pain (3), asymptomatic (4). Clinical features in the adults: encephalopathy (1), irritability (1), anemia (5), abdominal pain (3), asymptomatic (2). Levels at presentation (children): 10–29 μg/dL (3), 30–49 μg/dL (7), 50–69 μg/dL (1), 70–89 μg/dL (3), >90 μg/dL (2); range 23–194 μg/dL, mean 59.2 μg/dL. Levels at presentation (adults): 10–29 μg/dL (1), 30–49 μg/dL (0), 50–69 μg/dL (2), 70–89 μg/dL (2), >90 μg/dL (3); range 28–190 μg/dL, mean 90.6 μg/dL. Management involved identification and remediation of the source and chelation if appropriate. Sources (children): paint (10: 6 pica, 4 home redecoration), ingestion of lead weight (1), snooker chalk pica (1), unknown (4). Sources (adults): deliberate suicidal ingestion (1), organic lead (1), paint (8: 2 home redecoration, 6 professional decorators). Treatment: Calcium Disodium Edetate (with dimercaprol in the severe cases) was given to 6 children and 3 adults (with no adverse reactions). 2, 3,-dimercaptosuccinic acid (DMSA) was given to 10 children and 7 adults, a total of 34 courses of DMSA were supplied—the only adverse reaction was a spontaneously resolving rash in one adult case. The mean blood lead after treatment in January 1999 (3 lost to follow up, 5 needing further chelation) was 24.8 μg/dL in the children and 329 μg/L in the adults. Conclusion: In our experience, the main source of lead poisoning in the UK at present is leaded paint. In our clinical experience DMSA is well tolerated.

108 LACK OF BLOOD LEAD ELEVATIONS IN POLICE OFFICERS FOLLOWING SMALL ARMS QUALIFICATION ON AN INDOOR RANGE.

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Background: Airborne plumbism has been reported to occur in individuals whose work specifically involves cleaning and sweeping indoor firing ranges. Individuals who fire weapons on indoor ranges have also been reported to develop elevated blood lead levels. We report the first occupationally based study involving police officers required to fire periodically on indoor ranges for small arms qualification. This study verifies the safety of this practice with regard to exposure to lead. Methods: Blood lead levels were drawn from eight police officers and two "range masters" prior to undergoing annual, required, small arms qualification firing on an indoor range. All participants completed a questionnaire verifying that none had fired on the range within the previous 90 days. Twenty-four hours after a three hour stint on the range (nine hours for "range masters"), during which time 120, 9 mm, and 12 shotgun rounds were fired, blood lead levels were again drawn. Results: All eight officers had pre-range blood lead and post-range blood lead levels less than the lab detection threshold of 5 μg/dL. The "range masters" blood lead levels rose to 6.6 μg/dL and 9.0 μg/dL from non-detectable pre-range levels. Conclusion: Brief, occupationally required exposures for police officers on indoor firing ranges may not result in clinically important elevations in blood lead levels. Elevations in blood lead secondary to firing weapons on an indoor range is probably proportional to the time spent on the range.

109 PATIENT DISPOSITION AFTER SUSTAINED-RELEASE CALCIUM CHANNEL BLOCKER (SR CCB) INGESTION.

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Background: Delayed onset of clinical effects after SR CCB overdose has been previously described. A 24 hour observation period is suggested by most manufacturers after SR CCB overdose. The medical literature and fatal cases reported to US poison centers are reviewed to determine the necessary observation period. Case Series: In case reports of SR CCB overdoses published since 1990, 9 cases include data on the onset of clinical effects relative to time of ingestion. All 9 patients developed clinical effects within 12 hours of ingestion; 8 manifested clinical effects within 8 hours of ingestion, and 1 patient developed clinical effects 8–12 hours after ingestion. Three of those 9 reports described the onset of additional clinical effects more than 12 hours after ingestion, however, signs of toxicity were first noted before 12 hours. Fatalities: There were 168 SR CCB fatalities reported to US poison centers from 1988–1998. Data on the onset of clinical effects within the first 12 hours were provided for 56 cases. Forty-four patients developed clinical effects within 8 hours after the ingestion; 12 patients developed clinical effects 8–12 hours after the ingestion. No patient was known to develop clinical effects more than 12 hours after ingestion. Conclusion: All patients with significant SR CCB overdose and known time of onset of clinical effects, had some clinical effect within 12 hours. The majority develop toxicity in the first 8 hours. In some cases, serious toxic effects were not seen until 12–24 hours after ingestion, however, all such patients exhibited some initial less significant abnormality. Patients with any abnormality in vital signs, mental status, or ECG should be monitored for at least 24 hours and until all clinical effects resolve. After decontamination, patients without coingestants, who have normal vital signs, ECG, and mental status, may be safely discharged 12 hours after ingestion.

110 CALCIUM-CHANNEL BLOCKER OVERDOSE CAUSING MESENTERIC ISCHEMIA.

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Background: Calcium-channel blocker drug toxicity results in hypotension, bradyarrhythmias, and tissue hypoperfusion. Gastrointestinal manifestations have been limited to nausea, vomiting and intestinal hypomotility. This report describes 2 patients managed by a regional poison center within a one-month period who developed mesenteric ischemia following calcium-channel blocker overdose. Case series: A 36-year-old male with a history of hypertension ingested 1.2 g of nifedipine, plus 500 mg atenolol, clonidine, and alcohol. Lowest recorded BP was 103/53 mmHg, heart rate 57/min. He initially stabilized with calcium, glucagon, fluids and vasopressors, but 1.5 days post-ingestion developed abdominal pain, fever, and lactic acidosis. A CT scan showed evidence of bowel ischemia without perforation. Stools became melanotic and endoscopy revealed hemorrhagic gastritis and duodenitis. He dramatically deteriorated due to gastrointestinal bleeding and respiratory failure, and ultimately expired 15 days after overdose. Autopsy showed infarction and necrosis of the large intestine. The second case was a 17-year-old female who ingested 6480 mg of verapamil and BP transiently decreased to 60 mm Hg systolic with junctional bradycardia, rate 60/min. The patient rapidly stabilized with calcium, glucagon, fluids and vasopressors, but 24 hours post-ingestion developed hematemesis, melena, and abdominal pain. Laparotomy and partial colectomy revealed ischemic colitis, and the patient fully recovered. Conclusion: Calcium-channel blocker overdose can cause mesenteric ischemia and bowel necrosis in the absence of severe or prolonged hypotension. This complication should be suspected in these patients who develop gastrointestinal distress.

111 FAILURE OF MILRINONE IN A PEDIATRIC DILTIAZEM OVERDOSE.

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Background: Phosphodiestease inhibitors (PDI) are recommended as adjunctive treatment of hypotension in calcium channel blocker (CCB) ingestions. We report a case where a PDI used for CCB ingestion exacerbated hypotension in a pediatric patient. Case Report: A 13-year-old female with Williams syndrome ingested twenty-two 240 mg (105.6mg/kg) sustained release diltiazem tablets. She presented five hours later to an outlying hospital, with a pulse of 90 and a BP of 76/23. Treatment consisted of intubation with mechanical ventilation, fluid resuscitation, calcium chloride, glucagon, and dopamine and epinephrine infusions. She was transferred to our PICU, with a HR of 95 and a BP of 101/39. Milrinone was started (BP of 100/76) in attempts to further improve her circulatory status. During the bolus infusion, her BP quickly fell to 70's/P and the milrinone was discontinued. The epinephrine drip was increased and infusions of phenylephrine and insulin/glucose were initiated. She improved over the next several days and survived to be discharged. Discussion: PDIs indirectly increase intracellular calcium, which acts as a positive inotrope without increasing myocardial oxygen demand. It has been assumed that the potent vasodilatory effects of PDIs would be minimal given that the patient's peripheral vasculature is already maximally vasodilated from the CCB. Conclusion: In this pediatric case, use of a phosphodiesterase inhibitor was temporally associated with worsening of the patient's condition. We therefore advocate the cautious use of any potentially vasodilatory drug in CCB overdose.

112 NONINVASIVE HEMODYNAMIC MONITORING IMPACTING DECISION MAKING IN A CASE OF SEVERE METOPROLOL OVERDOSE.

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Background: Transthoracic electrical bioimpedance (TEB) monitoring has been demonstrated to be useful in the evaluation of critically ill patients in the emergency department. We present a case in which the use of TEB helped guide the selection of appropriate resuscitative measures. Case Report: A 61-year-old female with a history of depression was found unresponsive near empty bottles of metoprolol, Triavil®, and temazepam. She was intubated and taken to a local hospital where she had a pulse of 40 bpm and a systolic blood pressure (BP) of 50 mmHg. She was resuscitated with intravenous atropine, epinephrine, calcium chloride, and glucagon, and then placed on a dopamine infusion at 20 mcg/kg/min with a resulting pulse of 50 bpm and a systolic BP of 75 mmHg. Upon arrival to our hospital after helicopter transport, she was found to be unresponsive to deep pain, with a pulse of 48 bpm and a BP of 82/40 mmHg. An ECG demonstrated sinus bradycardia with no signs of sodium channel blockade effect, so it was inferred that her cardiovascular toxicity was primarily a result of metoprolol overdose. After no appreciable response to increasing doses of glucagon, calcium, and dopamine, the need for additional resuscitative measures (pacing and/or other vasopressors) became evi-

dent. Readings from the TEB monitor disclosed a cardiac output of 10.4 lpm, a total peripheral resistance of 5 mmHg/lpm, and an ejection fraction that was calculated to be about 65%. Bedside ultrasonography confirmed excellent cardiac wall motion. As a result, she was placed on a norepinephrine infusion with a rapid rise in her BP to 110/60 mmHg and a pulse of approximately 60 bpm. She was transferred to the intensive care unit and eventually weaned off of all support without any permanent sequelae. Conclusion: TEB monitoring technology can contribute to therapeutic decision making in cases of metoprolol overdose.

113 IOPIDINE® 0.5% (APRACLONIDINE HCL) TOXICITY IN A CHILD.

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Background: Iopidine 0.5% Ophthalmic solution contains apraclonidine, a relatively selective alpha-2 adrenergic agonist, structurally similar to clonidine. Indications include reduction in intraocular pressure prophylactically before and after ocular laser surgery and as an adjunct in the treatment of glaucoma. We report a case involving an ingestion of Iopidine® by a two-year-old child which resulted in clinical effects similar to that of clonidine toxicity. Toxicity from ingestion of this ophthalmic preparation has not been previously described. Case Report: A two-year-old child was given up to 5 cc of Iopidine® by a sibling. The estimated amount of apraclonidine ingested was 25 mg. Two hours later, the child was pale, diaphoretic and lethargic. The child was transported by paramedics to the emergency department where the child was arouseable to stimuli, had a heart rate of 60, a blood pressure of 90/60, respiratory rate of 30 to apnea and a tympanic temperature of 98.6 F. The child was given 25 g of activated charcoal by nasogastric tube. The child experienced persistent bradycardia and several episodes of apnea over three hours which eventually required intubation. The child's mental status improved and the child was extubated within 11 hours post ingestion. The child's clinical course thereafter was unremarkable. Conclusion: Ophthalmic preparations of apraclonidine, following ingestion in pediatric patients, can produce clinical effects similar to those of clonidine overdose. Patients would be expected to respond to the same management employed in clonidine overdose.

114 THEOPHYLLINE TOXICOKINETICS IN A PREMATURE INFANT.

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Background: Medication errors with theophylline may result in life-threatening overdoses in premature infants. This report describes the clinical course, successful medical management and toxicokinetics of theophylline in a premature infant after an estimated 10-fold overdose of theophylline. Case Report: A 16-day-old 34 week premature infant who was receiving theophylline for apnea of prematurity presented from an outside facility after developing sinus tachycardia with a theophylline level of 123 mcg/mL. Necrotizing enterocolitis was suspected due to a history of bilious emesis, hyperglycemia, melena, tachycardia and jitteriness. Theophylline levels were: hour 1, 123 mcg/mL; hour 5, 104 mcg/mL; hour 30, 58.4 mcg/mL; hour 52.5, 26.1 mcg/mL; hour 101, 4.8 mcg/mL. A single caffeine level at hour 30 was 13 mcg/mL. The infant exhibited first order pharmacokinetics across all theophylline levels with Kelim 0.028 1/hrs and $t_{1/2}$ 24.8 hours. Assuming an age-appropriate Vd of 0.7 L/kg, it is estimated that the infant received a dose of 179 mg. This corresponds to a dose 10 times that prescribed. Because of the infant's condition, GI charcoal, hemoperfusion or hemofiltration could not be used; the child was managed medically with phenobarbital for seizure prophylaxis and propranolol for tachycardia. The child remained without seizures throughout the hospitalization and sinus rhythm <180 was maintained. Conclusion: Based on pharmacokinetic calculations, we estimate the infant received a 10-fold overdose. The infant exhibited first order kinetics at all theophylline levels. We postulate that zero order kinetics was not observed because of the absence of saturable CYP mediated pathways at this age. We also conclude that acute severe theophylline overdose may be successfully managed in some cases without invasive maneuvers.

115 RAPID CARDIAC ARREST FOLLOWING MASSIVE CAFFEINE INGESTION.

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Background: Caffeine is a widely available stimulant. Most ingestions are inconsequential. We present 2 patients with abrupt onset of cardiac arrest following massive caffeine exposure. Case Reports: (1) A 20-year-old male ingested 18 g (287 mg/kg) caffeine on a dare. He presented to the emergency department in cardiac arrest within one hour of ingestion. He was resuscitated with standard ACLS protocol to a narrow complex rhythm after 35 minutes. He deteriorated over the

next 8 hours, developing hypotension (systolic BP 70–80 mm Hg), wide complex tachycardia, and recurrent ventricular fibrillation that were refractory to lidocaine, bretyllium, amiodarone, propranolol, and defibrillation (28×). Other premorbid manifestations included seizures, acidemia (pH 6.77), hypoxia (pO_2 < 50 mm Hg), hypokalemia (2.0–2.4 mEq/L), and hypocalcemia (6.2 mg/dL). Autopsy demonstrated a normal myocardium. His post-mortem caffeine level was 89 mcg/ml. (2) A 40-year-old male ingested 22 g caffeine and ethanol. He arrived at the emergency department awake and alert, but within 1 hour of ingestion he seized and developed pulseless ventricular tachycardia. Treatment included bretyllium, magnesium, amiodarone, metoprolol, and defibrillation (16×). Following cardiac resuscitation, he underwent 5 hours of charcoal hemoperfusion. His-pre-treatment caffeine level was 186 mcg/mL. After 3 hours of hemoperfusion, the level decreased to 123 mcg/mL, and he experienced no further dysrhythmias, hypotension or seizures. Post-resuscitation, his potassium fell to 2.0 mEq/L but was corrected over 48 hours. He was discharged 6 days post-ingestion. Conclusions: Onset of severe cardiac symptoms following caffeine overdose may occur abruptly, necessitating careful cardiac monitoring and immediate intervention. Early treatment with charcoal hemoperfusion may be beneficial.

116 PHENTOLAMINE ANTAGONISM OF PHENYLPROPANOLAMINE-INDUCED MORTALITY.

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Background: Phenylpropanolamine produces dose-related, life-threatening cardiovascular and central nervous system toxicity from alpha-adrenergic overstimulation. Although some recommend the alpha-adrenergic antagonist, phentolamine, as treatment for such toxicity, its therapeutic efficacy has not been well-studied. We sought to determine if pretreatment with phentolamine could reduce mortality in rats administered an overdose of phenylpropanolamine. Methods: Twenty-eight, unanesthetized, male Wistar rats (275–300 g; 14 animals per group) were randomized to receive an intraperitoneal injection of phentolamine (3 mg/kg) or an equal volume (1 mL) of normal saline diluent (control group). Twenty-five minutes after initial injection, all rats received an intraperitoneal injection of phenylpropanolamine (150 mg/kg) and were returned to their quarters with free access to food and water. Mortality at 24 hours was compared using the Fisher Exact test. Results: Twelve rats died, all within 6 hours of phenylpropanolamine administration. Mortality was significantly lower in the phentolamine-pretreated rats (2/14; 14%) as compared to the control group (10/14; 71%; p = 0.003). Conclusion: In this rat model, phentolamine pretreatment protected against phenylpropanolamine-induced lethality.

117 A LIFE-THREATENING HYDROXYCHLOROQUINE OVERDOSE.

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Background: Hydroxychloroquine (HCQ) overdose has rarely been reported despite the frequent use of this drug in the treatment of rheumatological diseases. A literature search found only 3 acute overdose cases, of which 2 quickly expired from cardiorespiratory arrest. The third case had ventricular tachycardia that responded to lidocaine and bretylium. We report an acute on chronic overdose in a patient who developed severe symptoms but survived. Case Report: A 16-year-old female presented to the ED with a BP of 63 mm Hg by palpation, P 110 beats/min, slurred speech, and drowsiness. History revealed she had taken a handful of her HCQ 200 mg, thyroxine, aspirin, and ibuprofen, 30 minutes prior to presentation. Fluid boluses brought her BP to 76/32 mm Hg and dopamine was begun at 10 mcg/kg/min. She was gastrically lavaged with evidence of pill fragments and 50 g of charcoal instilled. The initial ECG showed a QRS .14 msec and a left BBB. Initial labs: Na 138 mEq/L, K 2.1 mEq/L, Cl 111 mEq/L, HCO_3 17 mEq/L, Glucose 57 mg/dL, BUN 9 mg/dL, Cr 0.7 mg/dL, Ca 8.4 mg/dL, PO_4 2.5 mg/dL. A central line was placed and KCL 20 mEq/h for 2 hours was given. ABG was pH 7.34, $paCO_2$ 37.7, paO_2 232, HCO_3 19.7, sats 99% on 15 liters of O_2. Drug screen was positive for HCQ, and an unidentified substance. The BA <.01 mg/dL, APAP <10 mcg/mL, ASA = 17 mg/dL at 6 hours post ingestion. At 4.5 hour post ingestion, the dopamine was stopped and the BP was 100/74 mm/Hg, P 110 beats/min, and QRS .108 msec. The patient was awake and oriented, but drowsy. She complained of nausea and vomited charcoal. At 8 hours post ingestion, her K was still 2.2 mEq/L and 60 mEq KCl was given. Another 40 mEq KCl was given 20 hours post ingestion. The K stabilized at 3.8–4.2 mEq/L by 24 hours post ingestion. Her P continued to be 120 beats/min, gradually decreasing to 90–95 beats/min over 3 days. Conclusion: Although HCQ overdoses are very rare, life-threatening hypotension, conduction problems, and hypokalemia can occur within 30 minutes of ingestion.

118 EIGHTEEN MONTH RETROSPECTIVE EVALUATION OF SEROQUEL® EXPOSURES.

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Background: In 1997 the dibenzothiazepine antipsychotic drug Seroquel® (quetiapine fumarate) was approved for use in the United States. Quetiapine is structurally similar to clozapine, and is classified as an atypical antipsychotic since less extrapyramidal side effects are noted with its use. There are limited data detailing quetiapine overdose or adverse drug reactions. Methods: A retrospective analysis was conducted of the American Association of Poison Control Centers Toxic Exposure Surveillance System data of quetiapine human exposures from 1997–1998. Data with coingestants was not evaluated. Results: A total of 871 quetiapine exposures to humans were reported; 459 without coingestants. Unintentional exposures accounted for 36% of the reports, 52% were intentional, 12% were adverse drug reactions and 1% were an unknown reason. The majority (76%) of unintentional exposures experienced no symptoms or minor effects consisting of drowsiness (22%), dizziness or tachycardia (5% each), and ataxia or agitation (4% each). Of the 209 suicide exposures, 46% reported drowsiness, 25% tachycardia, 6% each for agitation, hypotension or coma, 4% slurred speech, and 2% each for respiratory depression or miosis. There were 2 cases with single seizures and one case with respiratory arrest. 26% of suicides reported no effect, 37% minor effect, 19% moderate effect, 4% major effect and no deaths. Symptoms lasted between 2 and 24 hours in 43% of suicide exposures. Of the 55 adverse drug reactions, 42% reported minor neurological symptoms such as drowsiness, confusion, dizziness, agitation and slurred speech. Significant adverse effects included 3 cases with seizures, 2 cases with renal failure and one major outcome and one death. Hypotension, coma, muscle rigidity, status seizures, and respiratory arrest were noted in the death. Duration of symptoms for adverse drug reactions varied from less than 2 hours up to one week. 48% of adverse drug reactions and unintentional exposures received no therapy or only observation, while 78% of suicides received gastric decontamination and 35% some form of treatment. Conclusion: Most exposures to quetiapine experience minor neurological and cardiovascular symptoms, although serious toxicity may occasionally be seen in suicide exposures and adverse drug reactions.

119 INTENTIONAL QUETIAPINE (SEROQUEL®) OVERDOSE.

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Background: Quetiapine (Seroquel®) is a new dibenzothiazepine antipsychotic that was introduced in the US in 1997. It is similar to clozapine, and indicated for psychotic disorders. The proposed mechanism of action is through dopamine (D2) receptor and serotonergic type 2 (5-HT2) receptor antagonism. The effects of quetiapine in overdose have not yet been established. Since the patients prescribed this medicine are at high risk for intentional overdose, the following study was designed to help elucidate the effects of quetiapine in overdose. Methods: Records of all patients with a history of quetiapine ingestion reported to a regional poison center from 1997–1999 were reviewed. Clinical and demographic data, co-ingestants, and interventions were recorded. Outcomes were scored according to accepted AAPCC criteria. Results: 22 cases were reviewed. 17 were intentional ingestions in adults. The 5 cases excluded comprised 4 home calls regarding adults, and an ADR in a 10-year-old boy. Ingested doses were poorly reported, but ranged to a potential maximum of 8 grams. 13/17 patients had a history of co-ingestants which included benzodiazepines, SSRI, antipsychotics, divalproate sodium, Ritalin, terazosin, acetaminophen, ethanol, and codeine. These patients presented most commonly with sedation (10) and tachycardia (4). In the 4 patients with isolated quetiapine ingestion, symptoms included agitation (1), tachycardia (3), sedation (2). One of these 4 patients had QRS prolongation on the ECG and PVCs were noted in 2 patients. Decontamination measures included: orogastric lavage followed by activated charcoal and a cathartic (6); activated charcoal and a cathartic alone (8); none (2); syrup of ipecac (1). All patients recovered without complications. Outcomes were scored as minor or moderate (12), severe (3), none (2). Conclusion: Central nervous system depression and tachycardia seem to be the most common findings in patients with quetiapine ingestions. Some potential for cardiotoxicity was also noted. Although these preliminary data can not exclude more serious toxicity following a massive ingestion, this early experience suggests a relatively benign outcome. Further evaluation including increased sampling and blood levels is required to completely categorize the toxicity of quetiapine.

120 TRAMADOL AND FLUOXETINE INDUCED SEROTONIN SYNDROME WITH SUBSEQUENT SEVERE HYPERPYREXIA DESPITE CYPROHEPTADINE.

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Background: Only 2 cases of serotonin syndrome (both mild) have been described in the setting of tramadol and SSRI use. We describe a case of severe serotonin syndrome after a polydrug overdose including tramadol and fluoxetine.

Profound hyperpyrexia occurred despite an initial dose of cyproheptadine. Case Report: A 32-year-old female presented to the ED 2 hours after an intentional overdose of her own medications that included tramadol (1.6 g), fluoxetine (80 mg), metaxalone (2.4 g), acetaminophen-oxycodone (20 tabs), etodolac (800 mg), and naproxen (30 g). Vital signs on presentation were BP 181/112 mmHg, HR 100 bpm, and T 100.1 F. A urine drug screen was negative for drugs of abuse. The patient denied other ingestions including MAOIs. Her physical exam and mental status were normal, she received an initial dose of activated charcoal and was admitted for observation. 12 hours after ingestion her mental status declined, and she developed diaphoresis, muscle stiffness and twitching, and required intubation. At this time vital signs were BP 130/70 mmHg, HR 135 bpm and T 98.8 F. Serotonin syndrome was suspected and cyproheptadine 4 mg was given. The patient's symptoms did not improve and less than 2 hours after cyproheptadine, she became more diaphoretic, developed a temperature of 108.8 F, HR 188 bpm, and BP of 86/52 mm Hg. Her vital signs improved with aggressive cooling, lorazepam, and fluid resuscitation. The patient went on to develop ARDS and rhabdomyolysis, but recovered and was discharged 3 weeks after admission. Analysis of the blood from admission showed fluoxetine 143 ng/mL (therapeutic 47–469 ng/mL) and tramadol 800 ng/mL (therapeutic 230–770 ng/mL). Conclusion: Given the relative lack of serotonergic properties of metaxalone, acetaminophen-oxycodone, etodolac, and naproxen, we concluded that the excessive ingestion of tramadol and fluoxetine in a patient already on these medications resulted in the development of severe serotonin syndrome. Single dose cyproheptadine clearly did not prevent deterioration in this case. Because of the late deterioration in this patient, prolonged observation should be considered after ingestion of multiple serotonergic agents.

121 A PROSPECTIVE EVALUATION OF ANTIDEPRESSANT POISONING.

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Background: Although non-cyclic antidepressant agents (NCADs) are thought to be much safer than cyclic-antidepressant agents (CADs) in overdose, little comparative data exists on the natural history of poisoning with these two broad classes of drugs. Methods: Prospective, comparative study of all patients admitted to the hospital toxicology service with antidepressant (AD) poisoning from 24/2/97 to 31/8/98. Prospective data collection in a poisoning database including: demographic details, co-ingestants, vital signs, ECG parameters, disposition, details of management and complications of poisoning, and length of hospital stay. Statistical significance if $p \leq 0.05$. Results: 123 patients (CAD 57, NCAD 66). No age or gender differences between groups. Length of stay was comparable between groups. Patients ingesting CAD's had lower admit GCS (12 vs 14, $p < 0.001$), were more likely to require intubation and ICU admission (26.3 vs 4.5%, $p \leq 0.001$), develop seizures (12 vs 2%, $p \leq 0.02$), have longer QRS (96.1 vs 85.2 msec, $p < 0.001$) and were less likely to have co-ingestants present (9 vs 36%, $p \leq 0.001$). No cardiac arrhythmias or deaths were seen in either group. Patients in the NCAD group were more likely to be discharged from the emergency department (58 vs 37%, $p \leq 0.03$) and develop serotonin syndrome (12 vs 0%, $p \leq 0.01$). Most frequent agent ingested was dothiepin (23.2%) followed by sertraline (19.4%). During 1997/98 dothiepin represented only 11% and sertraline 20.2% of all antidepressant prescriptions in the state of New South Wales. Conclusion: Despite the availability of many newer antidepressant agents, CADs continue to be involved in almost half of antidepressant poisonings in western-Sydney. Dothiepin appears to be over-represented in its hospital presentation frequency when compared to antidepressant prescribing patterns in the state of New South Wales.

122 A NATIONWIDE SURVEY COMPARING THE 1999 TO 1998 MANAGEMENT OF ASYMPTOMATIC CHILDREN WHO INGESTED A TRICYCLIC ANTIDEPRESSANT.

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Background: The triage of asymptomatic, unintentional pediatric TCA exposures (≤6yr) has been based upon single cases or small studies. Walsh in describing 2 cases involving 15–20 mg/kg ingestions recommended hospitalizing all children ingesting TCA for 24 evaluation. In 1998 newer information about the safety, pharmacology, and therapeutic use of TCA has emerged, suggesting that not all asymptomatic pediatric TCA exposures need referral to a health care facility (HCF). Objective: To evaluate the patterns of triage practiced by regional PCC nationwide for asymptomatic pediatric TCA exposures, and compare them to the 1998 patterns. Would the newer information about pediatric exposures to TCA result in changing management practices? What amount ingested (mg/kg) resulted in referral to the

emergency department (ED), as well as the recommended hours of observation in a HCF? Lastly, to evaluate the role of activated charcoal in these exposures. Method: We sent a survey to the 30 certified regional PCC that responded to our survey in 1998. Results: 22 PCC responded (73%). Fourteen (64%) referred to a HCF based upon mg/kg, compared to 6 (20%) in 1998. Of the 14, 6 (45%) referred at doses >5 mg/kg, compared to 2 in 1998. All PCC recommended 6 hours observation in a HCF compared to 90% in 1998. If referred to the ED, 18 PCC (82%) recommend giving AC. Discussion: The lowest toxic dose reported in the literature is 6.7 mg/kg. This is consistent with our PCC data during the past 6 years where no child was toxic at doses less than 5 mg/kg. Conclusion: This survey demonstrates significant changes in PCC triage patterns for asymptomatic pediatric TCA exposures have occurred during the last 12 months. Additional prospective study is needed to determine the optimum triage for pediatric TCA ingestions.

123 ANTI-DEPRESSANT OVERDOSES PRESENTING TO A REGIONAL TOXICOLOGY REFERRAL EMERGENCY DEPARTMENT OVER A FIVE YEAR PERIOD.

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Background: Intentional tricyclic anti-depressant (TCA) overdoses were the leading cause of ingestion death in 1994. The purpose of this study is to determine the impact that the increased utilization of serotonin-specific reuptake inhibitors (SSRI) had upon the demographics of deliberate anti-depressant ingestion. Methods: A retrospective chart review of all patients presenting to a regional toxicology referral emergency department with intentional ingestions was performed. Preliminary analysis was performed on two similar time periods (1/93–6/93 and 1/98–6/98). Results: TCA ingestions were the fifth most common overdose in 1993 and the fourth most common in 1998. There was no significant change in the number of either deliberate SSRI ($p = 0.26$) or TCA ($p = 0.36$) ingestion over the study. In patients requiring admission for medical stabilization, TCAs represented the third most commonly ingested substance in 1993 and the most commonly ingested substance in 1998. Individuals who ingested TCAs were significantly more likely to be admitted ($p < 0.01$) than were those who ingested SSRIs ($p = 0.26$). No patient died during either study period. Conclusion: The increased utilization of SSRIs did not produce an expected decrease in the number of deliberate TCA ingestions. Surprisingly, TCA overdoses presenting to our institution increased as a percentage of all ingestions over the five year period. Furthermore, the associated need for admission and medical stabilization increased over the study period.

124 THE EFFECT OF IMIPRAMINE-INDUCED CARDIAC DYSFUNCTION ON SARCOPLASMIC RETICULUM CALCIUM CONTENT.

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Objective: To determine if imipramine-induced (IMIP) cardiac dysfunction is mediated by sarcoplasmic reticulum (SR) calcium content. Methods: Isolated rat hearts were perfused with Krebs-Henseleit-Bicarbonate (KHB) at a constant coronary flow of 10 mL/min and paced at 300 bpm for 65 min. Left ventricular (LV) pressures were measured with a balloon-tipped catheter placed in the LV via the mitral valve. LV generated pressure (LVGP) was used as an index of cardiac function and was calculated by subtracting LV end diastolic pressure from LV peak systolic pressure. Experiments included the following sequence: 15 min of basal, 30 min of treatment, 5 min of 0 mM calcium, and 15 min of KHB. Caffeine (40 mM) was perfused from 1 to 5 min during the 0 mM calcium. Caffeine induced LV contractions and the sum of the LVGPs was used as an index of SR calcium content. Initial experiments included treatment as either KHB ($n = 5$) control, or IMIP at concentrations (ng/mL) 1000 ($n = 5$), 2000 ($n = 3$), and 3000 ($n = 3$). In subsequent experiments isoproterenol (ISO, 1 uM) was used to increase SR calcium content. Following ISO perfusion, 0 mM calcium and caffeine exposure were conducted in the same manner as the initial experiments. Reported values represent the mean of the percentage of control. Results: 1) IMIP decreased LVGP in a dose dependent manner. 2) SR calcium content after 30 min of IMIP exposure decreased from control at increasing IMIP concentrations (66% at 1000 ng/mL; 38% at 2000 ng/mL; 29% at 3000 ng/mL). 3) ISO alone ($n = 3$) increased LVGP and SR calcium content to 177% and 235% of KHB control, respectively. ISO + IMIP (1000 ng/mL) ($n = 3$) increased LVGP and SR calcium content to 192% and 134% of IMIP (1000 ng/mL) control, respectively. Conclusion: IMIP decreases left ventricular contractile response and SR calcium content.

125 HYPERTONIC SALINE IN SEVERE TRICYCYCLIC ANTIDEPRESSANT CARDIOTOXICITY.

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Background: Hypotension and cardiac conduction abnormalities following TCA overdose are treated with crystalloid, serum alkalinization and catecholamines. The use of hypertonic saline in rat and swine models has also shown promise in reversing these adverse effects. We report a case of severe cardiovascular toxicity following TCA overdose that was refractory to standard therapy but improved following hypertonic saline infusion. Case Report: A 29-year-old female presented to the ED in a coma 90 minutes after ingesting approximately 8 g of nortriptyline. She was intubated, lavaged, and received activated charcoal. The patient became hypotensive and developed a widened QRS (124 ms) both of which improved with crystalloid, hyperventilation, and $NaHCO_3$. Despite massive fluid resuscitation, maximizing serum alkalinization (pH 7.51–7.55), and high dose catecholamine therapy (dopamine @20 mcg/kg/min and norepinephrine @22 mcg/min) the patient developed refractory hypotension (78/42 mmHg) and worsening cardiac conduction abnormalities (QRS 139 ms, QTc 621ms). A 200 mL bolus of hypertonic (7.5%) saline was infused over 3 minutes. Immediately following infusion, the QRS narrowed to 120 ms and blood pressure improved to 104/60 mmHg. These effects were sustained and the patient survived without complication. Discussion: The use of hypertonic saline for severe TCA associated cardiovascular toxicity is supported in animal models and isolated case reports. In this case, hypotension and worsening cardiotoxicity that were refractory to current standard therapies responded to hypertonic saline infusion and the effect was sustained and without complication. Conclusion: We propose that the use of hypertonic saline be considered in cases of hypotension and cardiac conduction abnormalities that are refractory to standard therapies in the setting of TCA overdose.

126 RECURRENT SEIZURES FROM SUSTAINED-RELEASE BUPROPION.

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Background: Bupropion is a structurally unique, monocyclic anti-depressant marketed since 1988. A sustained-release (SR) preparation (Zyban®) was recently introduced. Seizures are common in bupropion overdoses. Described is a case where a patient took an overdose of Zyban® which caused seizures on two occasions, ten hours apart. Case report: A healthy 39-year-old male took up to 30, 150 mg Zyban® SR tablets. In the ER, he experienced a tonic-clonic seizure controlled by IV lorazepam. His EKG was remarkable for sinus tachycardia only and he was admitted to the ICU. The usual co-ingestants were ruled out and other labs were within the normal limits, the patient was not acidotic or hypoglycemic (HCO_3 26; Glu 114). Whole bowel irrigation was recommended but never initiated, because the KUB was unremarkable for any bezoars or aggregations. Approximately ten hours after the initial seizure, the patient experienced another tonic-clonic seizure lasting about 90 seconds, again controlled by IV lorazepam. By the next day, the patient was medically stable, had complete neurologic recovery, and was admitted to a psychiatric unit. Conclusion: Seizures are often reported in overdoses of Zyban®, but never occurring ten hours apart. Theoretically, the second seizure could have been prevented with aggressive bowel irrigation, thereby preventing bupropion absorption. Typically, a KUB is not sensitive enough to rule out the presence of a bezoar. In all large overdoses of Zyban® SR tablets, aggressive whole bowel irrigation should be considered. This could prevent systemic absorption of bupropion and the development of seizures. The risks and cost of treatment are minimal. This may shorten the length of hospitalization and reduce morbidity.

127 STATUS EPILEPTICUS SECONDARY TO BUPROPION OVERDOSE.

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Background: Bupropion is a noncyclic antidepressant reported to cause seizures in overdose as well as in patients with therapeutic blood levels of the drug. To date there have been no reports of status epilepticus associated with overdose of this drug. We report the first patient with status epilepticus secondary to bupropion overdose. Case report: A 44-year-old female presented to the emergency department (ED) in an obtunded state after a generalized seizure at home. On presentation, her vital signs, serum glucose, and pulse oximetry were all normal. Naloxone was administered intravenously with no response. A head CT scan was normal. In the ED, the patient had 4 additional 30 second generalized tonic-clonic seizures with interposed post-ictal periods consisting of lethargy and inability to follow commands. The patient was treated with benzodiazepines, phenytoin, and supportive care. Her recovery was uneventful and she admitted

to taking 60 bupropion tablets (150 mg) in a suicidal gesture. Conclusion: Bupropion, in overdose, is capable of inducing status epilepticus.

128 DELAYED CARDIOTOXICITY AFTER HALOPERIDOL AND PAROXETINE OVERDOSE.

Graeme K, Curry S, Kunkel D. *Department of Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: Previous cases of cardiotoxicity following haloperidol overdose report torsades de pointes or polymorphic ventricular tachycardia occurring within 18 hours of overdose. We present a case of recurrent, nonsustained polymorphic ventricular tachycardia beginning 31 hours after co-ingestion of haloperidol and paroxetine. We propose that inhibitors of the P450-2D6 isoenzyme, such as paroxetine, may delay the cardiotoxic effects of haloperidol after combined overdose. Case report: A 42-year-old man, without previous cardiac history, presented immediately after a witnessed suicidal ingestion of haloperidol and paroxetine. Urine drug screen also revealed ethanol, cocaine, and methadone. Following 11 hours of ED observation without cardiotoxicity, he was admitted for persistent somnolence. Neurological examination was nonfocal. 24 hours post-ingestion he was conversant. His ECG revealed a NSR at 69 bpm with a QTc of 445 msec. 31 hours post-ingestion he developed 30 beats of self-limited polymorphic ventricular tachycardia, followed by a similar episode, of 50 beats, within 20 minutes. His ECG revealed a NSR at 66 bpm with a QTc of 457 msec. K^+ 3.9 mmol/L. Ca^{2+} 8.1, Mg^{2+} 2.1, and phosphorus 2.4 mg/dL. Serum haloperidol 18.6 ng/mL (2–15 ng/mL). He received 2 g IV magnesium. 3 hours later, rhythm strips revealed 18 beats of self-limited polymorphic ventricular tachycardia. He remained hemodynamically stable. Rhythm strips revealed premature atrial complexes on the tail-ends of the T-waves preceding each episode. Despite normal electrolytes, echocardiogram and serial cardiac enzymes, 5 hours later he became transiently unconsciousness, with recurrent, nonsustained polymorphic ventricular tachycardia, lasting 35 seconds. ECG revealed a NSR at 64 bpm with QTc of 499 msec. He received magnesium sulfate 2 g IV and isoproterenol by IV drip for 36 hours. No further polymorphic ventricular tachycardia was noted. 5 days post-ingestion, ECG revealed a NSR at 77 bpm and a QTc of 420 msec. Conclusions: Cardiotoxicity following co-ingestion of haloperidol and paroxetine may be delayed.

129 BUPROPION TOXICITY CAUSES WIDE COMPLEX TACHYCARDIA.

Fresh L, Donovan W, Burkhart K, Kramer G. *Pennsylvania State University College of Medicine, Hershey Medical Center, Hershey, PA*

Background: In overdose, buproprion (Wellbutrin®, Zyban®) has been reported to cause tachycardia, and one report includes a cardiac conduction delay (QRS 132 ms). Buproprion is an antidepressant which has weak norepinephrine, dopamine, and serotonin reuptake effects. We report a case of wide complex tachycardia which reverted to normal over a period of 4 days. Case Report: A 36-year-old female presented to the ED 12 hours after ingesting 4.5 g of buproprion. Her initial heart rate and blood pressure were 136 and 90/60, respectively. She was given NSS with an increase in her blood pressure 100/60, but without a change in the heart rate. On ECG the QRS was 166 ms (QTc 587ms) with a left bundle branch block pattern. She also chronically took paroxetine, but she only ingested 60 mg of this medicine. She had no history of cardiac problems. Her treatment included 50 g activated charcoal, intubation, $NaHCO_3$ infusion, and transferral to our facility. On arrival, her ECG was essentially unchanged despite the $NaHCO_3$ infusion (ABG: pH 7.56, pCO_2 26.4, pO_2 308, HCO_3 23.3). Comprehensive drug screening by TLC, enzyme-linked assays, and HPLC from two different labs revealed only paroxetine, buproprion metabolites, trace acetaminophen, benzodiazepines, and metoclopramide (the latter two given therapeutically). The serum buproprion level was 0.44 mg/L (therapeutic 0.025–0.2 mg/L) was obtained at 16 hours post-ingestion. Serum paroxetine level was undetectable. Serial ECGs showed progressive normalization of QRS width and rate. An echocardiogram was normal as was an ECG upon discharge (rate 75, QRS 100 ms, QTc 438 ms). Conclusion: This is the first case of buproprion overdose demonstrating severe cardiac conduction abnormalities with a measured toxic level and no other detectable coingestants.

130 ELECTROCARDIOGRAPHIC CHANGES ASSOCIATED WITH CARBAMAZEPINE TOXICITY IN THE PEDIATRIC POPULATION.

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Background: Carbamazepine (CBZ) toxicity has been associated with a high incidence of QTc and QRS prolongation in adults. CBZ cardiotoxicity is rarely reported and poorly characterized in the pediatric population. Methods: A retro-

spective chart review of all pediatric patients with the discharge diagnosis of CBZ poisoning at 3 urban teaching hospitals (1988–1998) was conducted to identify those that had documented ECG during the intoxication. Patients with preexisting cardiac disease, reported co-ingestion of other agents known to affect the ECG, abnormal serum calcium or potassium values or serum CBZ level <12 μg/mL were excluded. Heart rate, QRS duration and QTc duration were measured by the ECG computer and a medical toxicologist and were compared to established age-appropriate values. Results: 56 patients met the entry criteria. Group 1: 35 cases of children ≤6 years of age, mean age of 3.2 ± 1.4 years, mean peak CBZ level 27.2 ± 8.6 μg/mL and mean time to ECG 6.8 ± 5.3 hours. Group 2: 21 cases of children >6 but <18 years of age, mean age 12.0 ± 3.3 years, mean peak CBZ level 30.7 ± 12.9 μg/mL and mean time to ECG 11.0 ± 7.3 hours. Group 1: 5.7% had tachycardia, 37.1% had QRS prolongation, 2.8% had QTc prolongation. Group 2: 0% had tachycardia, 33.3% has QRS prolongation, 0% had QTc prolongation. No one developed clinically significant arrhythmias. Conclusion: Pediatric CBZ intoxications are associated with QRS prolongation. No cases of clinically significant cardiac arrhythmias were reported.

131 MARKEDLY ELEVATED VALPROATE LEVELS DO NOT SERVE AS AN INDICATION FOR HEMODIALYSIS OR HEMOPERFUSION.

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Background: Previous reports suggest that markedly elevated valproate (>950 mg/L) may be an indication for hemodialysis and/or charcoal hemoperfusion. We questioned whether markedly elevated serum or plasma valproate should be used as criterion for hemodialysis or hemoperfusion. Methods: A retrospective review of inpatients admitted to or seen in consultation by the toxicology service at a tertiary referral center from April 1, 1995 to March 31, 1999 was conducted. Charts of patients with markedly elevated valproate levels (peak levels >950 mg/L) were further assessed. Results: 50 cases of valproate exposure, out of 1627 inpatients, were found. Four patients had documented peak valproate levels of greater than 950 mg/L. Case 1, 2, 3, and 4 demonstrated peak valproate levels of 1440, 1188, 1089, and 980 mg/L, with peak levels measured at 14, 8.5, 13, and 12.5 hours post-ingestion, respectively. Cases 1, 3 and 4 demonstrated elevated ammonia levels, with peak levels measured at 307, 114, and 131 umol/L, respectively (not measured in case 2). Case 2 demonstrated metabolic acidosis, with an initial serum bicarbonate of 14 mmol/L; this resolved over 6 hours with intravenous hydration. Case 3 demonstrated a transient low SBP of 80 mm Hg, with a pulse of 80 bpm, which did not require treatment. All became unconscious; cases 1 and 3 required mechanical ventilation. None demonstrated persistent hemodynamic instability or metabolic acidosis. Cases 1, 3, and 4 received carnitine. None received hemodialysis or hemoperfusion. All recovered without sequelae from their valproate toxicity within 24–48 hours of ingestion. Conclusion: In the absence of persistent hemodynamic instability or metabolic acidosis, comatose patients with extremely elevated valproate levels do well with supportive care, without hemodialysis or hemoperfusion.

132 UTILIZATION OF HEMODIALYSIS TO ENHANCE VALPROIC ACID (VPA) ELIMINATION.

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Background: VPA is highly protein bound and therefore not amenable to hemodialysis in the therapeutic range. In overdose, saturation causes more unbound drug to be available for removal by hemodialysis. We describe the use of hemodialysis to enhance VPA elimination in 2 cases. Case Report #1: A 23-year-old female was brought to the emergency department 1.5 hours after ingesting an unknown quantity of VPA. She progressed quickly from drowsiness to obtundation. Activated charcoal (50 g) and sorbitol (108 g) were administered. The absence of bowel sounds prevented further charcoal administration. The VPA serum level peaked at 791 mcg/mL 7.5 hours post-ingestion. The patient was intubated and comatose at this time. Upon initiation of hemodialysis (11.5 hours post-ingestion) the serum level reported was 752.7 mcg/mL. One hour into hemodialysis her mental status dramatically improved. The serum level dropped to 269 mcg/mL following 3.5 hours of hemodialysis. The calculated half-life during hemodialysis was 2.48 hours; post-dialysis was 16.64 hours. Case Report #2: A 37-year-old female ingested an unknown quantity of VPA 3 hours prior to her emergency department presentation. Initially, she was alert and oriented but became obtunded over the next 3 hours. The initial VPA serum level 4 hours post ingestion was reported as 93 mcg/mL. The patient received 2 doses of AC before she developed hypoactive bowel sounds preventing its further administration. The VPA serum level peaked at 1290 mcg/mL 11 hours post-ingestion. At that time, she was unresponsive to deep pain. The calculated

half-life of VPA at this time was 14.51 hours. Hemodialysis was instituted 23 hours post-ingestion. VPA serum levels taken 1.5 hours prior to and 4 hours into hemodialysis were 910 mcg/mL and 417 mcg/mL respectively. The patient was arousable but confused at the completion of hemodialysis. The calculated half-life during hemodialysis was 3.99 hours, which increased to 15.45 hours post-dialysis. Both patients were discharged 3 days after presentation. Conclusion: Based on our experience, hemodialysis may be a viable modality to enhance the elimination in patients presenting with very high (>700 mcg/mL) VPA levels.

133 MULTICENTER CASE SERIES OF VALPROIC ACID INGESTION.

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Background: Valproic acid (VPA) ingestions reported to poison centers have increased more than 4 fold over the last 4 years. There are no large case series published on VPA ingestion. Method: Multicenter case series of all patients reporting an ingestion of VPA. Data collected included: age, gender, dose ingested, co-ingestants, symptoms and vital signs, lab values, length of hospital stay and medical outcome. Entrance into the study required a VPA level above the therapeutic threshold of 100 mcg/mL. Statistical analysis was by Fisher's exact test. Results: 134 patients were reported to participating centers of which 80 (60%) had VA levels in the toxic range. 23 cases of the 80 cases were polydrug ingestions leaving 57 cases of pure VPA ingestion for evaluation. 45 patients (79%) were female. Age ranged from 11 years to 56 years with a mean of 29.5 years (±10.9). Peak VPA levels ranged from 110 mcg/mL to 1840 mcg/mL with a mean of 361 mcg/mL (±337 mcg/mL). Time post-ingestion to the peak measured VPA level was from 1 to 18 hours, with a mean of 7.5 hours (±3.4). Symptoms included lethargy (n = 41), coma (n = 8), tachycardia (n = 6), aspiration (n = 5), metabolic acidosis (n = 3) and hypotension (n = 2). A peak level of >450 mcg/mL was more likely to be associated with a moderate or major outcome ($p < 0.05$). A peak level >850 mcg/mL was more likely to be associated with coma ($p < 0.05$) and acidosis ($p < 0.05$). Six patients experienced transient thrombocytopenia (platelets < 150,000) and all had peak VPA levels >850 mcg/mL. Two patients experienced transient leukopenia (WBC < 3.5). The mean hospital stay for all patients was 44.7 hours (±28). A hospital stay of >48 hours was more likely to be associated with a peak VPA level of >450 mcg ($p < 0.05$). All patients recovered with supportive care. Conclusions: In this case series patients with peak VA levels >450 mcg/mL were more likely to be associated with significant clinical effects and have longer hospitals stays.

134 VALPROIC ACID OVERDOSES: A RETROSPECTIVE STUDY COMPARING SERUM DRUG LEVELS AND THE INCIDENCE OF ADVERSE OUTCOMES.

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Objective: To study the relationship between serum valproic acid levels and adverse outcomes in overdose patients. Case Series: This is a retrospective review of the 505 valproic acid related calls received by the California Poison Control System, San Francisco Division in 1997 and 1998. A total of 108 cases had a documented peak serum drug level ≥120 μg/mL, with 81 patients having a peak drug level <450 μg/mL (Group 1; average 207, median 188, range 120–420) and 27 having a peak drug level ≥450 μg/mL (Group 2; average 722, median 688, range 450–1,600). The average age and female/male ratio of the two groups were similar. A total of 57 adverse outcomes were documented:

	n	Group 1	Group 2
Hypernatremia (Na ≥ 150 mEq/ml)	7	2 (29%)	5 (71%)
Hypocalcemia (Ca ≤ 8.5 mg/dl)	6	1 (17%)	5 (83%)
Anion gap (Na − Cl − CO2 > 15)	7	0	7 (100%)
Unresponsive to painful stimuli	17	3 (18%)	14 (82%)
Endotracheal intubation	17	0	17 (100%)
Cerebral edema	2	0	2 (100%)
Death	1	0	1 (100%)

Overall 11% of the documented adverse outcomes occurred in Group 1 and 89% in Group 2. Conclusion: In this study population, the vast majority of adverse outcomes associated with valproic acid overdoses occurred at peak drug levels ≥450 μg/mL.

135 BULLOUS SKIN LESIONS ASSOCIATED WITH SEVERE VALPROIC ACID OVERDOSE IN A FOUR YEAR-OLD CHILD.

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Background: Superficial skin bullae have been reported in association with barbiturate and benzodiazepine overdoses but not with valproic acid (VPA) overdose. We report such a case. Case Report: A four-year-old Black female was found comatose approximately 12 hours after being put to bed. Unresponsive to all stimuli and hypotensive, she was intubated, placed on a dopamine infusion, and air evacuated to our hospital. Upon arrival, apparent bruising was noted on the left wrist and in the left inguinal area. A solitary, clear fluid-filled bulla was also noted in the inguinal area. History from the family revealed patient had access to VPA and carbamazepine. Laboratory evaluation obtained approximately 22 hours after estimated ingestion time showed a serum VPA level of 804 mcg/mL, a serum ammonia level of 131 umol/L, a lipase of 403 IU/L and mild transaminase elevation without prolongation of the prothrombin time. Serum carbamazepine, serum ethanol, and rapid urine drug of abuse screen were all negative. Rhabdomyolysis was present, with a serum CPK >32000 U/L. Sixteen hours after initial laboratory data were obtained, the serum VPA level was 281 mcg/mL, serum ammonia level was 23 umol/L, and the patient was awake and following commands. The patient continued to have elevated CPK levels >32000 U/L for the next 4 days and exhibited progressive bone marrow suppression requiring transfusion after reaching a nadir on hospital day 4. Other lab abnormalities had normalized. During this time period, she displayed progression of the bullous skin lesions initially noted in her inguinal area. In addition, new regions of ecchymosis subsequently developed on her extremities, evolving into clear fluid-filled bullae. Conclusion: We present a case of superficial bullous lesions associated with severe VPA overdose in a child.

136 STATUS EPILEPTICUS FOLLOWING ACUTE TIAGABINE OVERDOSE.

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Background: Tiagabine (TGB), is a lipophilic analog of nipecotic acid that crosses the blood-brain barrier where it selectively inhibits GABA uptake by presynaptic neurons; its prevention of synaptic impulse transmission inhibits the propagation of epileptic activity within the brain. We describe the first reported case of TGB ingestion overdose. Case Report: One hour postingestion of an unknown quantity of 4 mg TGB tablets which belonged to a relative, a 24-year-old male with no significant past medical history presented to the ED with tonic clonic seizures which reportedly began thirty minutes postingestion. VS: T 99.4, BP 128/65, HR 110 bpm and ineffective respirations. His exam was otherwise unremarkable. Intubation with 20 mg diazepam and 200 mg succinylcholine was followed by the cessation of his clinical seizures with the total seizure time estimated to be 2.5 hours. Subsequent to EEG demonstration of continued subclinical seizure activity, a continuous midazolam infusion stopped the seizures. OGL produced pill fragments and a single dose of AC was given within 2 hours postingestion. ECG demonstrated ST (110 bpm). A noncontrast head CT and routine laboratory data, including urine drug screen, were normal. The patient was discharged at 48 hours following an uneventful course without further seizure activity. Serum tiagabine levels measured by HPLC were 710 ng/mL, 310 ng/mL, 270 ng/mL, and 110 ng/mL at 4.5 hours, 12 hours, 13.5 hours, and 19 hours postingestion respectively. Conclusion: This represents the first reported case of TGB overdose and TGB-induced status epilepticus. We calculated an estimated half-life for TGB to be 4.5–6.3 hours. Back extrapolation to time zero yielded an estimated peak plasma TGB in this patient of 1180–1974 ng/mL. Utilizing the volume of distribution for TGB from the literature, we would estimate that the patient may have taken between 13 and 21 four mg tablets of TGB.

137 SEVERE LACTIC ACIDOSIS FROM ACUTE METFORMIN OVERDOSE.

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

Introduction: Metformin is an oral hypoglycemic agent used in the management of Type II Diabetes. Lactic acidosis is an uncommon (0.03 cases/1000 patient years), but serious (50% mortality rate) complication associated with therapeu-

tic dosing. There are few reports of overdose. We report 2 patients with severe lactic acidosis and death due to metformin overdose. Cases: In unrelated incidents, two patients, a 29-year-old male and a 32-year-old female each with Type II diabetes overdosed with metformin. They each developed severe lactic acidosis, pH 6.73 lactate 28 mmol/L; 6.93 and 23 mmol/L respectively. Treatment included bicarbonate, vasopressors and extracorporeal removal. Both patients died. Serum metformin concentration was 90.3 μg/mL in the male. Cyclobenzaprine was also found in the serum of the male, and diphenhydramine, acetaminophen and trimethoprim were found in the urinary drug screen of the female. Discussion: Metformin enhances lactate production and reduces its clearance. Metformin associated lactic acidosis (MALA) is caused by decreased gluconeogenesis from alanine, pyruvate and lactate leading to lactate accumulation. MALA is often associated with an increase in plasma metformin concentration. Elevated plasma concentrations associated with overdose can also result in severe lactic acidosis. The management of these patients includes supportive care, vasopressors, and aggressive bicarbonate therapy. Extracorporeal methods may be indicated to remove the large sodium load associated with the bicarbonate therapy. Conclusion: Metformin overdose may result in severe lactic acidosis. Despite aggressive therapy, the mortality is high.

138 MULTICENTER CASE SERIES OF ADULT METFORMIN INGESTION.

Spiller HA, Weber J, Hofman M, Sollee DR, Winter M, Klein-Schwartz W, Stork C, Krenzelok E. *Kentucky Regional Poison Center, Louisville, KY*

Background: There are no large studies or case series of metformin ingestion in adults. Case reports suggest a risk of lactic acidosis without hypoglycemia in massive ingestion. Method: Case series of all ingestions reported to eight regional poison centers with age ≥18 years. Data collection included age, gender, dose ingested, medical history, co-ingestants, symptoms and vital signs, lab values, length of hospital stay and medical outcome. Entrance into the study required at least 24 hour of follow-up. Statistical analysis was by Fisher's exact test and ANOVA. Results: 65 patients were reported with a mean age of 46.4 (±18). 49 patients (77%) were female. 16 cases were polypharmacy and 42 patients (65%) had a history of diabetes. There was no difference between non-diabetic vs diabetic patients in dose ingested, gender or percentage of patients evaluated in a HCF, but diabetic patients tended to be older. Reported dose ingested ranged from 500 mg to 30 g, with a mean of 4.3 g (±6.6). Thirty-three patients (51%) ingested a maximum of ≤2000 mg. 31 patients were evaluated in a HCF, and 52 had regular glucose measurements. Two diabetic patients with metformin ingestion experienced lactic acidosis and coma of which one case was fatal. Severe toxicity was evident within six hours of hospital arrival. Hypoglycemia (n = 7) was only seen in diabetic patients with polydrug overdoses associated with concomitant sulfonyurea or insulin overdose. Other symptoms in diabetic patients attributed to metformin included lethargy (n = 1), and DIC (n = 1). Symptoms attributed to metformin in non-diabetic patients were minor: GI complaints (n = 3), headache (n = 1) and dizziness (n = 1). Conclusion: In this case series the majority of metformin ingestions in non-diabetic and diabetic patients resulted in minimal toxicity. However, two diabetic patients developed severe toxicity, which was evident within 6 hours.

139 MULTICENTER CASE SERIES OF PEDIATRIC METFORMIN INGESTION.

Spiller HA, Weber J, Winter M, Klein-Schwartz W, Hofman M, Gorman S, Stork C, Krenzelok E. *Kentucky Regional Poison Center, Louisville, KY*

Background: There are no large studies or case series of metformin ingestion in children. Case reports in adults suggest risk of lactic acidosis without hypoglycemia in massive ingestion. Method: Case series of all ingestions reported to eight regional poison centers with age <18 years. Data collection included age, gender, dose ingested, co-ingestants, symptoms and vital signs, lab values if performed, length of hospital stay and medical outcome. Entrance into the study required at least 24 hour of follow-up. Results: Forty-six cases were collected. Age ranged from 15 months to 17 years, with a mean and median of 4 (±4.4) and 2 years, respectively. Dose ingested by history ranged from 250 mg to 16.5 g, with a mean and median of 1808 mg (±3,573) and 500 mg, respectively. Thirty-three children (72%) ingested a maximum of two tablets (≤1700 mg). Dose/weight ratios ranged from 9-196 mg/kg, with a mean and median of 61 mg/kg (±45) and 41 mg/kg, respectively. Thirty-two children were evaluated in a HCF, and 35 had regular glucose measurements. Reported symptoms included nausea (2) and dizziness (1). No child experienced hypoglycemia. Arterial blood

gas and electrolyte measurements were done in 3 and 15 children, respectively. No evidence of acidosis was seen. Two children had lactate levels drawn and were normal. Twenty-three patients received activated charcoal. Five patients received parenteral glucose (D5W) and one adolescent with a history of diabetes received insulin for hyperglycemia. Conclusion: Unintentional ingestion of ≤1700 mg in the healthy pediatric population does not appear to pose a significant health risk of hypoglycemia or detrimental outcome. In the seventeen total children with either ABG, electrolytes or lactate level determination no evidence of lactic acidosis was seen.

140 A REEVALUATION OF CAUSTIC INGESTIONS IN PEDIATRIC PATIENTS.

Nichols M, Monroe K, King W. *University of Alabama Birmingham, Department of Pediatrics, Birmingham, AL*

Background: Management of caustic ingestions has been debated over the last decade. No case series in the U.S. has been published since limiting the use of steroids and antibiotics was recommended. We reviewed a current series of caustic ingestions in our institution. Case Series: A chart review (1992–98) of caustic admissions at a tertiary pediatric hospital was conducted. Wilks' log likelihood test was used to analyze symptoms versus endoscopic results. 136 patients were evaluated. 105 (77%) patients were African American, 31 (33%) Caucasian with 74 males (54%) and 62 females (46%) with average age of 2.2 years. Insurance coverage was 79 patients (58%) Medicaid, 18 (13%) private insurance, 39 patients (29%) no insurance. 90 (66%) patients underwent endoscopy within 24 hours of exposure. 6 patients received steroids and 8 received antibiotics. Average length of stay was 1.2 days (<1–12 days). Among patients who had endoscopy, 76 (84%) were normal, 7 (8%) esophagitis and 7 (8%) 2nd or 3rd degree esophageal burns. Drooling as a presenting symptom was more common among patients with esophageal burns ($G^2 = 9.99$, $p = 0.001$) and esophagitis ($G^2 = 4.30$, $p = 0.04$) than those with normal endoscopy. Neither oral burns nor swollen lips were associated with esophageal burns ($G^2 = 1.51$, $p = 0.22$ and $G^2 = 1.15$, $p = 0.28$ respectively). For esophageal burns, 2 patients had strictures requiring multiple admissions. Caustics ingested (''*'' denotes caustics causing esophageal burns) include: hair products (48)*, lye (20)*, drain openers (18), oven cleaner (6), detergents (5), cleaner fluid (4), bleach (5), ammonia (3)*, nail prods (3), batteries (2), carpet cleaner (2), industrial solvents (2)*, mixed (2), and other (9). Conclusion: Drooling was associated with esophageal burns and esophagitis, while oral burns were not. The most common caustic ingestant was hair relaxer. Antibiotics and/or steroids were used in 8 cases with esophageal burns or esophagitis.

141 RENAL FAILURE AND CORROSIVE AIRWAY AND GASTROINTESTINAL INJURY FOLLOWING INGESTION OF DILUTE DIQUAT SOLUTION.

Tanen DA, Curry SC. *Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: Diquat dibromide is a nonselective herbicide, structurally similar to paraquat. Only 13 cases of isolated diquat ingestions with systemic toxicity have been reported. All involved the ingestion of concentrated solutions (e.g., 20% diquat cation). The reported mortality rate approached 70% with death resulting from gastrointestinal complications, pneumonia, pontine infarction and renal failure. We report the first case of systemic toxicity resulting from the ingestion of a dilute solution of diquat. Case Report: A previously healthy 66-year-old man swallowed 200 cc of Dexol Weed and Grass Killer® (1% diquat cation) in a suicide attempt. Eight hours later, he experienced a sore throat and non-bloody emesis that prompted him to seek medical care. His initial physical exam was remarkable only for mild pharyngeal injection. His initial metabolic panel, ECG, and CXR were unremarkable. He was hospitalized for observation. 24 hours post ingestion, laboratory values revealed a rise in serum creatine to 2.2 mg/dL that peaked at 4.0 mg/dL on day 4. Physical exam revealed lower lip and tongue swelling along with mucositis of his tongue and posterior pharynx. Endoscopy revealed significant burns throughout the length of his esophagus and proximal stomach. The patient's acute renal failure, mucositis and esophagitis resolved over a month. Conclusion: Acute ingestion of large amounts of relatively dilute diquat dibromide solutions, may lead to delayed renal failure, gastrointestinal corrosive injury, airway obstruction, acute lung injury, brainstem infarction and death.

Platform Session 3 Saturday, October 2 4:30 pm–6:00 pm
Acetaminophen Poisoning and *N*-Acetylcysteine
Abstracts #142–#147

142 α-GST AS A BIOMARKER OF ACETAMINOPHEN-INDUCED HEPATOTOXICITY.

Sivilotti MLA, Burns MJ, Linden CH, Aaron CK. *University of Massachusetts, Worcester; Harvard University, Boston, MA*

Background: Serum alpha glutathione *S*-transferase (α-GST) has been shown to be superior to aspartate aminotransferase (AST) as a biomarker of hepatic injury after a variety of insults, including acetaminophen overdose. Normally present at low concentrations in the serum (median < 1 μg/L, 95%ile < 7.5 μg/L), it is both released into and cleared from the circulation more rapidly than AST. The slow turn-around-time of α-GST radioimmunoassays (over 24 h) has limited the clinical utility of this test. An α-GST enzyme immunoassay (HEPKIT™-Alpha, Biotrin, Dublin) with a turn-around-time of <3 hours has recently become available. Methods: Serum α-GST concentrations were measured by HEPKIT™ in a convenience sample of patients presenting after potentially toxic acetaminophen ingestion. Patients with pre-existing liver disease or known risk factors for acetaminophen hepatotoxicity were excluded (e.g. alcoholism). Results: Mean (SD) acetaminophen concentration 4 hours after ingestion in the 27 patients studied to date was 282 (164) μg/mL (time of ingestion unknown in 8 cases). The 5 patients with elevated AST at presentation [3230 (3400) U/L] all had initial α-GST >100 μg/L. α-GST concentrations fell more rapidly than AST in each case. 17 other patients had acetaminophen concentrations above the possible hepatotoxicity line on the Rumack-Matthew nomogram. Two of these patients had AST concentrations rise above 100U/L during treatment, whereas 11 (65%) demonstrated elevated α-GST concentrations. The remaining 5 patients with acetaminophen concentrations below the possible hepatotoxicity line all had persistently normal AST concentrations, but two exhibited elevated α-GST concentrations. The time of the first elevated α-GST ranged from 3 to 14 hours post-ingestion, with all but one of these patients demonstrating an elevated α-GST at presentation. All patients recovered fully. Conclusions: α-GST appears to be more sensitive than AST for subclinical hepatic injury following acetaminophen overdose. Its rapid rise shortly after acetaminophen ingestion and subsequent clearance suggest potential use as a biomarker to guide early treatment decisions as well as to follow resolution of traditionally defined hepatic injury. Measurement of α-GST in a larger number of patients will be necessary to establish the clinical role of this test.

143 EVALUATION OF HEPATOTOXICITY IN ALCOHOLIC PATIENTS FROM THERAPEUTIC DOSING OF ACETAMINOPHEN.

Kuffner E, Dart RC, Bogdan GM, Mlakar P, Casper E, Darton L. *Rocky Mountain Poison & Drug Center and Department of Psychiatry, Denver Health, Denver, CO*

Background: A preliminary study of 60 alcoholic patients showed no difference between patients treated with acetaminophen (APAP) and those treated with placebo. Our study sought to increase the power to detect hepatotoxicity. Methods: A prospective, double-blind, randomized, placebo-controlled trial of alcoholics was conducted at an alcohol detoxification center. Alcoholism was confirmed by two methods. Baseline serum APAP, AST, ALT and INR were determined. Exclusion criteria included a recent history of ingesting >4 grams of APAP, serum APAP >20 mcg/mL, AST or ALT >120 IU/L or INR >1.5. Patients were randomized to receive APAP, 1 g every four hours for 4 doses on day 1 and day 2 or placebo on the same schedule. AST, ALT and INR were measured on days 2 and 4. Results: A total of 200 patients completed the trial: 102 received APAP and 98 received placebo. Patient groups were not different based upon demographics, severity of alcoholism, nutritional status or baseline AST, ALT or INR. Four patients in the APAP group and three patients in the placebo group developed an AST or ALT >120 IU/L. There was no statistical difference between the two groups at any time with respect to AST, ALT or INR means. Fisher's Exact test ($p > 0.05$ two-tailed) was used to compare categorical data and an Unpaired Student *t* test ($p > 0.05$ two-tailed) was used to compare continuous numerical data. There was a 95% probability of detecting a 15 IU/L difference between AST or ALT means. Conclusion: Alcoholic patients treated with the maximal therapeutic doses of acetaminophen did not develop hepatotoxicity. Our study does not support the concept of reducing acetaminophen dose in the alcoholic patient.

144 INCREASED INR AND REDUCED FUNCTIONAL FACTOR VII IN ACETAMINOPHEN POISONING WITHOUT EVIDENCE OF HEPATOTOXICITY.

Whyte IM, Buckley NA, Dawson AH, Reith DM, Goodhew I, Seldon M. *Department of Clinical Toxicology, Mater Hospital, Newcastle, Australia*

Background: Acetaminophen may increase the International Normalized Ratio (INR) prothrombin time in anticoagulated patients. Acetaminophen poisoning without hepatic damage may also cause an increased INR. Objective: To describe, in a large series of acetaminophen overdoses not complicated by hepatotoxicity, the effect on the INR and, in a smaller group of acetaminophen overdoses, to investigate the mechanism. Methods: A retrospective review of INR in 143 admissions with acetaminophen overdose without hepatotoxicity was performed. Factors VII, VIIIc and IX were also measured prospectively in 30 patients and 8 controls. Results: The INR showed a time-dependent increase with 50% having an abnormal INR at some time. Both the dose ingested ($P = 0.01$) and the nomogram-based risk (P for trend $= 0.005$) were correlated with the effect. The odds of an abnormal INR with an acetaminophen concentration above the 150 mg/L line were 3.8 (95% Confidence Interval, 1.9–7.5) when compared to non-toxic exposures. *N*-acetylcysteine had a protective effect. Functional Factor VII fell compared to control ($P = 0.005$) and was less than antigenic Factor VII in cases ($P = 0.03$) but not controls. There was a small fall in factor IX ($P = 0.02$) but no effect on factor VIIIc. Conclusions: An isolated, small rise in INR is common after acetaminophen overdose and does not necessarily indicate hepatotoxicity. It appears to be due to inhibition of Vitamin K dependent activation of coagulation factors. This effect suggests a possible mechanism for the observed interaction between acetaminophen and warfarin.

145 INTRAVENOUS *N*-ACETYLCYSTEINE FOR ACETAMINOPHEN OVERDOSE: AN ABBREVIATED PROTOCOL.

Donovan JW, Mancuso E, Burkhart KK. *Central Pennsylvania Poison Center, Pennsylvania State University College of Medicine, Hershey, PA*

Objective: Describe the clinical experience after adopting an abbreviated intravenous *N*-Acetylcysteine (IV NAC) protocol for the treatment of acetaminophen (APAP) poisoned patients. Our center has previously reported that normal transaminase values at the time of initiation of IV NAC are an excellent predictor of good outcome. Therefore, our center has shortened the length of treatment from 48 to as little as 24 hours when transaminase values have remained normal and APAP is undetectable. Method: This is a retrospective chart review of patients treated for APAP overdose between January 1997 through June 1998, at a university hospital regional poison treatment center. APAP overdose was determined by the Rumack-Matthew nomogram. Toxicity for this analysis was defined as development of a transaminase value greater than 100 IU/L. IV NAC was given as 140 mg/kg loading dose over 1 hour, followed by 70 mg/kg every 4 hours for at least 24 hours. Therapy was continued only if detectable APAP remained or transaminase values were elevated. Patients who did not develop hepatotoxicity during hospitalization were later contacted by telephone to assess any development of jaundice or illness. Results: Eighty-eight patients met the inclusion criteria. Twenty-eight (32%) developed hepatotoxicity, but 10 of these were not treated until greater than 24 hours post-ingestion. Forty-nine (82%) of the 60 non-toxic patients received less than 48 hours of IV NAC. Twenty-five (51%) of those were reached in follow-up and none reported jaundice or any liver problem since their hospitalization. Six (7%) of those treated had minor adverse reactions. Conclusions: An abbreviated IV NAC protocol of less than 48 hours is safe and effective for selected APAP overdoses.

146 *N*-ACETYLCYSTEINE IS NOT EFFECTIVE IN RAPIDLY REDUCING METHEMOGLOBINEMIA.

Tanen D, LoVecchio F, Curry S. *Good Samaritan Regional Medical Center, Phoenix, AZ*

Objective: To determine if intravenous *N*-acetylcysteine administered to human volunteers with nitrite-induced methemoglobinemia produces a clinically significant decline in blood methemoglobin concentrations. Methods: Randomized, controlled crossover trial with each subject serving as their own control. Methemoglobinemia was induced with IV sodium nitrite (4 mg/kg) over 10 minutes starting at time 0. At time 30 minutes, subjects were randomized to be treated with IV *N*-acetylcysteine (150 mg/kg) for 1 hour followed by 14 mg/kg for 40 minutes, or to be treated with equal volumes of 5% dextrose in water. The experiment was repeated no less than one week later when each subject received the alternative treatment. Blood methemoglobin concentrations were measured by multiwavelength cooximetry at times 0, 15, 30, 50, 70, 90, 110, and 130 minutes. Area under the [methemoglobin]-time curve (AUC) between 30 and 130

minutes was compared between groups using a two-tailed paired *t*-test. Results: Baseline methemoglobin concentrations were not statistically different between control and treatment groups (.04 ± .01 g/dL, .08 ± .03 g/dL, respectively; p = .12). There was also no statistical difference between control and treatment groups with regard to methemoglobin concentrations at the beginning of treatment at time 30 minutes (.85 ± .30 g/dL, .88 ± .31 g/dL; p = .31). Mean AUC for the control group (77.1 ± 5.7 g·min/dL) was actually lower than the mean AUC for the treatment group (84.5 ± 4.7 g·min/dL); p = .01. Conclusion: *N*-acetylcysteine was not useful in the acute treatment of methemoglobinemia in this model.

147 A VARIABLE DURATION NAC TREATMENT PROTOCOL FOR ACETAMINOPHEN OVERDOSE.

Parker SJ, Bizovi K, Smilkstein M. *Oregon Poison Center, Portland, OR*

Background: The ideal duration of NAC treatment remains controversial and settings in which shorter courses of NAC are appropriate remain undefined. Most NAC protocols are defined by a predetermined duration of NAC therapy. This retrospective study describes our experience with a protocol with varying lengths of treatment based on the time of ingestion and clinical course. Methods: Poison center charts of all patients treated under the variable duration protocol between January 1, 1997 through December 31, 1997 were reviewed and data abstracted. In these cases, NAC was recommended to be continued until 36 hours after APAP ingestion. At that time if AST was normal and APAP non-detectable, discontinuation of NAC was recommended, regardless of the duration of therapy. Post discharge outcome was determined by contacting hospitals where the patient was initially admitted and by checking the tertiary care/liver transplant center records for readmission. Results: 171 patients were identified as APAP ingestions treated with NAC. Discontinuation of NAC at 36 hours post ingestion was recommended in 112 (65%). The majority of people were treated as recommended for 36 hours or less (58%). Of these patients 40% were considered "high risk" with APAP levels above the 300 mcg/mL nomogram line. Duration of treatment ranged from 6 to 69 hours (median 36 hours). Mean time to initial NAC treatment was 11 hours (range 2–45 hours). Maximum AST level was 475 with median value of 22. The majority of patients were female (77%). Age ranged from 3 to 86 years old. Hospital follow up was established on 94%. No patients were readmitted for liver injury. Conclusion: This variable duration NAC protocol was safe in this subset of patients with APAP toxicity, including patients traditionally considered high risk. Analysis of patients not treated with or non-compliant with the protocol will be needed to better determine the applicability of this approach.

Poster Session 3 — Sunday, October 3 — 10:00 am–4:00 pm
Abstracts #148–#202

148 ACCURACY OF DRUG ABUSE CALL PATTERNS IN PREDICTING PRESCRIPTION DRUG ABUSE.

Krummen K, Tsipis G, Siegel E, Bottei E. *Cincinnati Drug & Poison Information Center, Cincinnati, OH*

Background: The Cincinnati Drug & Poison Information Center (DPIC) monitors the substances cited in drug abuse calls each year. The list is disseminated to local law enforcement, counselors and health professionals. As the list is used to increase awareness and direct intervention activities, the accuracy with which it reflects drug abuse patterns in the Cincinnati area is important. Methods: The top 10 prescription drugs diverted, according to the Cincinnati Police Division of Pharmaceutical Diversion Squad, were compared with the top 20 prescription drugs cited in DPIC calls for the year of 1997. Results: The top 10 drugs diverted were (in order of greatest use): hydrocodone, diazepam, oxycodone, APAP/codeine, carisoprodol, fentanyl, propoxyphene, tramadol, alprazolam, butalbital. The top 20 prescription drugs cited in DPIC drug abuse calls were (in order of greatest use): hydrocodone, oxycodone, diazepam, alprazolam, methocarbamol, propoxyphene, butalbital, carisoprodol, cyclobenzaprine, lorazepam, tramadol, clonazepam, morphine, codeine, methylphenidate, pentazocine, clorazepate, phentermine, metaxolone, chlorozoxazone. 70% of the top

10 diverted pharmaceuticals were cited in the DPIC top 10 list of prescription drugs abused. 90% were cited in the DPIC top 20 list. Conclusion: As the DPIC abuse calls closely reflect the prescription drugs diverted in Cincinnati, it is probable that prescription drug abuse trends can be predicted by monitoring DPIC calls. A developing interagency system of substance abuse treatment agencies may give further documentation of correlation. Toxicovigilance efforts, such as awareness and intervention activities, are appropriately directed.

149 DOES RECORDING POISON CENTER CALLS DETER LAY PUBLIC REQUESTS FOR IDENTIFICATION OF DRUGS OF ABUSE?

Alsop J, Sands T. *California Poison Control System-Sacramento Division, Sacramento, CA*

Background: An automatic call distribution (ACD) system was installed in the poison center in 1998. The new ACD system incorporated a recorded message informing each caller that the conversation would be recorded for quality assurance purposes. Determining if the recording message would decrease the number of calls from the lay public for identifications of drugs of abuse was undertaken. Methods: The number and type of drug identification (ID) calls both before and after ACD installation were compared. All drug ID calls from one month without ACD were compared to all ID calls with ACD in the same month one-year later. The data was sorted into source of caller (lay public, HCF staff, tox lab staff or law enforcement), drug class, drug status (controlled, prescription, OTC or unknown), abuse potential of the drug, and time of day of the call. Results: The total number of drug identification calls increased 29% after ACD was installed. Total calls on drugs of abuse from all caller sources increased from 31.3% to 37.7%. There was a small increase in calls from health care professionals (6.4% from 5.1%) and a small decrease in calls from the lay public (18.4% from 19.0%). Calls on drugs of abuse from law enforcement almost doubled (11.8% from 6.5%) in the same time span. Conclusion: The use of an ACD system with a message stating that calls will be recorded does not significantly impact drug ID call patterns from the lay public who request identification of drugs of abuse.

150 SPIRAL CT IMAGING OF INGESTED FOREIGN BODIES WRAPPED IN PLASTIC: A PILOT STUDY DESIGNED TO MIMIC COCAINE BODYSTUFFERS.

Hibbard R, Wahl M, Kirshenbaum M, Nellamattiathil G, Aks S. *Illinois Masonic Medical Center, Illinois Poison Center, Mercy Hospital and Medical Center, Chicago, IL*

Background: Bodystuffing is the practice of hurriedly ingesting poorly wrapped packets of drugs in an attempt to avoid discovery by the police. This is not an uncommon presentation at many urban Emergency departments. There have been case reports of various imaging modalities such as CT, KUB without contrast, KUB with Gastrografin, in an attempt to visualize these packets, but very little consensus has been reached on the modality of choice. A pilot study was designed using a non-toxic placebo (rock sugar) and five healthy volunteers to determine if non-contrast spiral CT is of potential utility in these patients. Methods: In a previous study, crack cocaine was described as having an attenuation value of 91 hounsfield units *in vitro*. Using the same *in vitro* technique it was determined that our placebo samples had an attenuation of 78 hounsfield units. The samples were felt to have sufficient correlation in which to proceed to an *in vitro* study. Five healthy volunteers swallowed pieces of rock sugar wrapped in the corner of plastic bags; the total size of the packet was approximately 7.5 mm including the sample, bag and knot. The volunteers received non-contrast spiral CT exams with 5.0 mm cuts two to four hours after ingestion. An attending radiologist then read the CT scans to determine if the placebo was visible. Results: Three out of five CT scans (60%) revealed a small intraluminal radiopaque density consistent with placebo ingestion. In the other CT scans, no definite foreign body was visible. Conclusion: This pilot study demonstrates that spiral CT scan is capable of identifying plastic wrapped placebo designed to mimic crack cocaine. Future studies to determine clinical correlation with crack cocaine will be needed in the future.

151 CONTRAST CT SCAN FAILS TO DETECT THE LAST HEROIN PACKET.

Hahn I, Hoffman RS, Nelson LS. *New York City Poison Control Center, New York, NY*

Background: ''Body packers'' or ''mules'' smuggle narcotics internationally by ingesting large numbers of packets filled with illicit drugs. An initial plain abdominal radiograph is often used to confirm the presence of multiple packets. Clinical criteria or radiographic studies may evaluate subsequent adequacy of GI decontamination. No study has confirmed whether computed tomography (CT) scan is superior to traditional contrast radiography. This case report suggests that the conventional radiograph with contrast may be more sensitive for detecting a single packet after decontamination.

Case Report: A 28-year-old female stated she had swallowed 55 packets of heroin 2 days earlier. She passed 50 packets spontaneously prior to incarceration and arrival in the emergency department. She was asymptomatic. On physical examination, she was calm and alert with normal vital signs and physical examination, including reactive mid-size pupils and a soft non-tender abdomen with normoactive bowel sounds. Laboratory studies and 12-lead ECG were normal. Radiographs of the abdomen revealed 3 diagnostic densities and 2 possible densities. The patient received whole bowel irrigation (WBI) with polyethylene glycol (PEG-ELS) at 2 liters/h for 2 hours and evacuated 4 packets. Failure to pass the fifth packet raised questions about the patient's history. A helical abdominal CT with oral contrast, interpreted by the CT radiology attending, did not reveal any abnormal densities even though the contrast filled the entire small and large intestine. An upright abdominal radiograph was subsequently obtained that clearly demonstrated the last packet in the right lower quadrant. The patient received more WBI and passed the last packet within one hour. She remained asymptomatic and was discharged. Subsequently, the CT radiology attending reviewed the CT with the plain radiograph and remained unable to detect the last packet. Conclusions: Computed tomography with contrast missed the single body packet whereas the plain radiograph with contrast did not. Until further study, we suggest using oral contrast plain radiography with bowel follow-through to evaluate the GI tract after WBI.

152 LIMITATIONS OF WHOLE BOWEL IRRIGATION AND LAPAROTOMY IN A COCAINE "BODY-PACKER."

Olmedo RE, Hoffman RS, Nelson LS. *New York City Poison Control Center, New York, NY*

Background: Illicit drugs are smuggled by the intentional ingestion of drug packets. Current recommendations for cocaine "body packers" include treatment with activated charcoal, whole bowel irrigation (WBI), and surgical removal if any symptoms of cocaine toxicity develop. This case report highlights the limitations of WBI and surgery. Case Report: A 30-year-old woman stated that two days prior to presentation she had swallowed approximately 40 packets of cocaine in an attempt to smuggle them into the US. She was asymptomatic and cooperative and had a normal physical exam. Initial abdominal radiograph revealed multiple packs in the bowel with no signs of obstruction. A urine toxicology screen was positive for cocaine. The patient received whole bowel irrigation with polyethylene glycol (PEG) at 2 L/h for 3 days and passed 25 packets. Due to the failure to pass additional packets, an abdominal CT without contrast was performed which identified remaining packets in the sigmoid colon. Several hours after the CT, the patient developed precipitous hypotension and coma. Following resuscitation, the patient underwent laparotomy and removal of 29 drug packets via an enterotomy. Two of the extracted packets had ruptured. Careful inspection and palpation of the entire GI found no abnormalities. Post-operatively the patient had persistent tachycardia and fever. On post-op day four, a repeat abdominal CT performed to exclude intra-abdominal abscess, located two additional packets in the sigmoid colon. These packets were removed by colonoscopy and appeared to be intact. After treatment for wound dehiscence and cellulitis, the patient was discharged with residual memory deficits. Conclusion: Prolonged whole bowel irrigation may not be effective for total evacuation of drug packets. Although intra-abdominal surgery is the gold standard of diagnosis and treatment of "body packers," it may also fail to detect intraluminal drug packets. History obtained from the patient and the associated packet count are unreliable.

153 PROLONGED QRS DURATION DUE TO COCAINE INTOXICATION.

Goto CS, Delaney KA. *The University of Texas Southwestern Medical Center, Dallas, TX; The North Texas Poison Center, Dallas, TX*

Background: Animal studies demonstrate that high doses of cocaine result in direct myocardial depression with prolonged QRS and QT interval. These effects are similar to those caused by type I antidysrhythmics that inhibit the fast inward sodium current. Despite the common occurrence of cocaine poisoning in humans, conduction disturbances are rarely reported. We report a case of massive cocaine ingestion with prolonged QRS and QT intervals. Case report: A 30-year-old male ingested at least 2 g of crack cocaine following apprehension by law enforcement officers. He seized during transport and arrived in the emergency department with BP 100/39, pulse 90, temperature 37.2°C. Normal saline was given IV with improvement in BP to 120/56. Gastric lavage was performed with instillation of activated charcoal. ECG showed QRS 136 ms, QT 522 ms, right bundle branch block and rightward axis deviation. Laboratory data showed Na 138, K 5.1, Cl 98, CO_2 4, glucose 160, Cr 1.5, Ca 9.3. ABG showed pH 6.81, pCO_2 38, pO_2 103, BE -28. An ECG 1½ hours after arrival showed QRS 114 ms, QT 444. The pH improved to 7.26. He was intubated and given 100 mEq of sodium bicarbonate IV. An ECG 3 hours after arrival showed QRS 88 ms, QT 438 ms and a normal axis. pH

improved to 7.46. 4 liters of normal saline had been administered IV. A urine toxicology screen showed only cocaine and THC. CKMB and troponin levels were not elevated. Conclusion: This case adds to the existing literature describing myocardial depression and prolonged QRS and QT interval in human cocaine overdoses, presumably due to myocardial sodium channel blockade.

154 RECOVERY FROM SEVERE HYPERTHERMIA (45°C) AND RHABDOMYOLYSIS AFTER METHAMPHETAMINE BODY-STUFFING.

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Background: We report a case with complete recovery from severe hyperthermia (45°C) and rhabdomyolysis resulting from methamphetamine body-stuffing and physical exertion. This is the highest reported core body temperature in a case with laboratory confirmed sympathomimetic toxicity. Case Report: A 23-year-old man ingested approximately 1 g of methamphetamine following a minor motor vehicle accident with a police cruiser and fled the scene on foot. The patient was chased (ambient temperature ~22°C), apprehended, and required physical restraint to be transported for medical evaluation. The patient's agitation and combativeness prompted sedation, neuromuscular blockade, and endotracheal intubation upon arrival to the ED. Initial vital signs were rectal temperature 45°C (113°F), HR 164/min, and BP 152/70 mmHg. Physical examination was remarkable for mydriasis and diaphoresis. Initial laboratory work-up was significant for gross myoglobinuria and serum creatinine kinase 2083 IU/L. Additional cooling measures included ice packs to the groin and axillae and a cooling blanket, reducing the patient's temperature to 38.1°C (100.6°F) within 90 minutes. The patient remained comatose for 26 hours, but still exhibited signs of sympathomimetic toxicity for 2 more days. Creatinine kinase peaked at 119,901 IU/L without evidence of compartment syndrome, but with hydration and urinary alkalinization the patient never developed renal insufficiency. He was discharged home in stable condition on hospital day 5. Conclusions: Severe hyperthermia may occur from the combination of physical exertion and methamphetamine body-stuffing. Aggressive cooling measures, intravenous hydration, and urinary alkalinization resulted in complete recovery, despite rhabdomyolysis and prolonged sympathomimetic toxicity.

155 SEVERE ASTHMA ASSOCIATED WITH HEROIN INSUFFLATION.

Prachand N, Krantz A, Franklin C, Gummin D, Hryhorczuk D, Hershow R. *University of Illinois at Chicago School of Public Health/Cook County Hospital/Toxikon Consortium, Chicago, IL*

Background: In the past decade, the primary route of heroin administration has shifted dramatically from injection to insufflation; this has occurred in association with an increase in drug purity. During the last 3 months of 1998, 3 cases of *status asthmaticus* associated with heroin insufflation were seen by the toxicological consult service in the intensive care unit (ICU) at an urban public hospital. This study explores the rate and impact of heroin use among ICU asthma patients. Methods: A retrospective medical chart review of all adult ICU asthma admissions under age 50 (n = 113) during 1997 and 1998 was conducted. Results: 107 (95%) charts were reviewed [mean age: 35 years, range: (17–50)]. 61 (57%) were male, 95 (89%) were African-American. 105 (98%) had previous diagnosis of asthma. 106 (99%) reported urban residence. 65 (61%) had medical histories and/or urine toxicological screens indicating recent opiate use. For 15 patients (14%), there was a history or clinical impression recorded indicating a temporal relationship between heroin insufflation and precipitation or exacerbation of symptoms leading to the current admission. Several patients identified drug adulterants (diphenhydramine, vitamin B_{12}) which reportedly were particularly associated with symptom exacerbation. Conclusions: The results suggest a relationship between heroin insufflation and subsequent acute asthma symptoms, based on reported temporal patterns. Clinicians should consider heroin insufflation as a possible precipitating factor for patients with severe asthma.

156 HEROIN-INDUCED PRIAPISM.

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Background: Priapism is rare. Although frequently idiopathic, it is associated with some systemic diseases. Drug-induced priapism has been described. Contrary to verbal communications, heroin-induced priapism is unreported. Case Report: A 41-year-old African American male presented with a complaint of 12 hours of painful erection unassociated with sexual stimulation. It began 4 hours after using IV heroin. He admitted to similar episodes in the past uniquely after heroin use which required surgical therapy. His PMH was not contributory except for heroin abuse. He denied taking any prescribed or over-the-counter medications, penile injection, or trauma. His physical exam disclosed bilateral rigid

corpora cavernosas with a flaccid glans and spongiosum. No constricting device was noted. The blood tests revealed an Hgb of 12.6 g/dL (Hct: 37.7%), WBC of 5.4, and a negative sickle screen. His UA was normal. The UDS was tested for opiates, PCP, benzodiazepines, cocaine, amphetamines, THC, barbiturates, ethanol, methadone, and propoxyphene and was positive for opiates. The cavernosa gases were pH 6.85, pCO_2 135, pO_2 6. Medical therapy failed. Both cavernosas were drained and then irrigated with a lidocaine-epinephrine solution. Detumescence occurred within 1 hour. Of interest, he had an episode of priapism 4 days later after heroin which required surgical therapy. Conclusions: Mechanism of action may resemble apomorphine ($C_{17}H_{17}NO_2$) which is structurally similar to heroin ($C_{17}H_{17}(O\text{-}C_2H_3O)_2ON$) and heroin-metabolite, morphine ($C_{17}H_{19}NO_3$). Phase III studies show that apomorphine acts on the post-synaptic dopamine receptors of the hypothalamus and induces erection (stimulating NO-pathways). Opiates, as CNS sympatholytic agents may induce an unopposed cholinergic state that may result in Ach-induced vasorelaxation entailing NO pathways. Alpha-blockade has been postulated. All these facts highlights the importance of an autonomic system dysregulation which seems to be not dose-specific. In this case, the odds of an adulterant were considered but it was not excluded.

157 TRAMADOL ABUSE IN THE CINCINNATI AREA.

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Background: Tramadol is an alternative to traditional opioids for the treatment of pain. Although abuse has been reported, it is thought to be uncommon. The Cincinnati Drug & Poison Information Center (DPIC) has found abuse of tramadol to be a relatively significant problem. Methods: The number of DPIC calls since the introduction of tramadol was assessed. In addition, the top 10 prescription drugs diverted, according to the Cincinnati Police Division Pharmaceutical Diversion Squad, were evaluated during the same time period. Counselors, probation officers, callers and health professionals were queried for possible explanations. Results: The DPIC documented 362, 107, 515, and 326 tramadol abuse calls in 1995, 1996, 1997, and 1998, respectively. The Cincinnati police documented diversion of 7,258 doses of tramadol from 9/97–12/97 and 11,385 doses in 1998. Tramadol was listed in both the Cincinnati Police top 10 diverted prescription drugs and the DPIC top 20 prescription drugs abused lists for 1997. Reasons for abuse include: prevention of withdrawal from other opioids when unable to get the narcotic of choice, easier availability due to lack of control status, exclusion from urinalysis screening for drugs of abuse. Conclusion: Tramadol abuse is not rare in the Cincinnati area. Physicians need to consider the abuse potential and monitor patients for dependence. Closer control of distribution, at least locally, would be advisable.

158 SEX ON THE STREETS OF CINCINNATI.

Krummen K, Bottei E, Whiteman P. *Cincinnati Drug & Poison Information Center, Cincinnati, OH; Hamilton County Coroner's Laboratory, Cincinnati, OH*

Background: Our Drug & Poison Information Center (DPIC) interacts with many agencies in order to disseminate high quality information about local drug abuse trends to counselors, law enforcement, and health professionals. These interactions were especially helpful in defining a new trend of abuse of methylenedioxymethamphetamine (MDMA) and other amphetamine-related compounds in our local area. Recently, multiple calls requesting information about ingestion of unknown tablets imprinted with "SEX" were made to the DPIC. Local police also found several "SEX" tablets in a plastic bag in a cigarette pack that contained marijuana. Methods: The coroner's lab used a Marquis Test, Nitroprusside Test, UltraViolet/Visible Spectrometry and GC/MS to identify the active component of the "SEX" tablets. The coroner's laboratory also provided information about similar substances identified in recent cases of drug possession and trafficking. DPIC MDMA abuse calls were evaluated for an increase in volume. Results: The "SEX" tablets contained MDMA. The coroner's lab identified similar amphetamine-like substances in 29 local cases of drug possession or trafficking from October, 1998 to March, 1999: 13 unidentified substances contained MDMA (3 powder, 8 tablets, 1 capsule, 1 crystal), 8 methylenedioxyamphetamine (4 powder, 1 tablet, 3 capsules), 7 methamphetamine (5 powder, 1 solid, 1 liquid) and 1 methamphetamine/ephedrine (powder). The number of calls to DPIC about MDMA quadrupled from 1996 to 1998. We forwarded information about this new trend to local law enforcement, counselors, and health professionals. Conclusion: This cooperative effort between drug and poison information centers, the coroner's office, and law enforcement increases awareness among police agencies, counselors, and physicians of the local drug abuse trends.

159 DRIVERS UNDER THE INFLUENCE OF DRUGS (DUID) ARE USUALLY UNDER THE INFLUENCE OF MORE THAN ONE DRUG.

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Background: Forensic toxicologists must often determine the psychoactive effects of drugs on motor vehicle operators. Research to date has often focused on the effect of a single drug, but this may not accurately reflect the true sociologic phenomenon of DUID, since many drug users often take a variety of substances, including alcohol. Case series: All drivers in a major metropolitan area suspected of operating a motor vehicle under the influence of drugs are subjected to blood testing and forensic toxicology evaluation. These are special determinations apart from the usual tests for alcohol alone. An anonymous prospective data bank was generated by computer from this reporting system which contained age, gender, reason for arrest, and the results of blood laboratory evaluations. Serum or plasma were subjected to immunochemical assay and retested with gas chromatography/mass spectrometry (GC/MS) to report drug concentrations. The first 196 specimens in this series contained only two specimens with no drugs or alcohol. The driver arrested for DUID was more often male (86%) and averaged 32.6 years of age (SD 12.1). The arrests were 54% motor vehicle code violation, 32% motor vehicle accident (MVA), 13% MVA with injuries, and 1% MVA with fatalities. Active compounds by drug class were detected in the following proportions: ethyl alcohol =68%, THC and cannabinoids =44%, benzodiazepines =30%, cocaine =16%, phenylidine =13%, opiates and opioids =6%. Ethyl alcohol was the sole drug detected in 28% of specimens; without ethanol, 36% of specimens still contained two or more classes of drugs. One drug class was detected in 41% of specimens, two drugs in 41%, three drugs in 17%, and four drugs in 1%. Conclusion: If our experience is reproduced elsewhere, then forensic toxicologists must be prepared to evaluate reports with an understanding of the interactions of drugs of abuse and alcohol, as this is the most likely scenario encountered.

160 5 YEARS OF 3,4-METHYLENEDIOXYMETHAMPHETAMINE (MDMA) TOXICITY.

Rella JG, Nelson LS, Hoffman RS. *New York City Poison Control Center, New York, NY*

Background: MDMA, XTC, or "Ecstasy," has been a popular drug of abuse for two decades. Until recently, cases of MDMA exposure in our region had been relatively uncommon. As the use of MDMA became widespread in our area, and since the majority of published data on clinical toxicity consists mainly of isolated case reports, we designed the following study to record systematically our experience with MDMA toxicity. These data now represent the largest collection of MDMA cases reported to date. Methods: Our poison center database was searched for the years 1993 to 1999 inclusive. Patient demographics, coingestants, and symptoms were recorded. Outcome was scored according to AAPCC criteria. The annual incidences of MDMA cases were compared using linear regression and calculation of a Pearson's correlation coefficient. Results: There were 137 cases reported in this 5.25-year period. 86 males and 50 females were exposed; the gender of one patient is unknown. The median age was 22 years (IQ_{25-75} = 17–26 years). 43 cases (31%) experienced moderate to major toxicity, including 1 death due to hyperthermia. 94 cases (69%) experienced minor or no toxicity. The most commonly reported symptoms were agitation (25%), tachycardia (25%), and nausea and vomiting (13%). Hyperthermia and hyponatremia, the most serious complications of MDMA use, were reported in 6 patients and 1 patient, respectively. Urinary retention was reported in 3 patients, all of whom also reported concurrent ketamine use. Coingestants included cocaine, ethanol, heroin, marijuana, phencyclidine, and psilocybin-containing mushrooms. Beginning in 1997, ketamine and GHB were newly reported as common coingestants. MDMA reports increased with time ($r = 0.85$, $p < 0.05$). Conclusion: Increasing MDMA exposures in our region occurred predominately in a young population, of whom nearly a third met AAPCC criteria for moderate to severe effects. Although hyperthermia and hyponatremia occurred infrequently, we advocate rapid initial screening for these potentially devastating complications, while providing appropriate supportive care.

161 METHYLPHENIDATE EXPOSURES: 5 YEARS OF POISON CENTER EXPERIENCE.

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Background: Methylphenidate is widely used for ADD. The purpose of this study was to evaluate the toxicity of exposures reported to poison centers. Methods: AAPCC Toxic Exposure Surveillance System was queried for all methylphenidate exposures reported between 1993 and 1997. Using a relational database, information on exposures involving methylphenidate alone was extracted and analyzed using descriptive statistics. Results: Of 19,521 exposures reported

to the AAPCC, 18,151 involved methylphenidate alone. There were 5,520 (29%) exposures in children 6 years and under. Major effects occurred in 3 children and there were no deaths. Of the 3,721 cases with known outcomes in this population, no effects (2,468), minor effects (1,002), and moderate effects (248), were reported in 99.9% of the cases. In exposures involving patients over 6 years, 5,356 were followed to a known outcome. Two fatalities, both intentional ingestion in adults, were reported. Major effects were observed in 26 cases. The majority of the incidents (99.4%) involved no effects (2,720), minor effects (1,760), or moderate effects (848). In patients with major effects the most frequently reported symptoms included: tachycardia [12 (41%)], hypertension [4 (13%)], and seizures [34% (1 status, 8 multiple-discrete, 1 single)]. A total of 5,027 (27.7%) patients were treated in a healthcare facility of which 22% (1,113) were admitted for medical care. The most common methods of GI decontamination utilized included single dose activated charcoal (2,980), lavage (1,001), and ipecac (571). Conclusion: The majority of patients with exposures to methylphenidate alone exhibited minimal toxicity. However, methylphenidate overdose can cause serious toxicity and death with those at greatest risk being adults with intentional exposures.

162 A PROFILE OF METHYLPHENIDATE EXPOSURES.

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Introduction: Methylphenidate is prescribed commonly for children with Attention Deficit Disorder. An estimated 2.8% of US youths aged 5 to 18 years use it for the management of this disorder. Despite the widespread use of methylphenidate, the demographics and outcome of intentional and unintentional exposures to methylphenidate have not been described. Methods: To profile human exposures to methylphenidate, a retrospective review of all reports to a certified regional poison information center for 1998 was conducted. Data analysis included patient demographics, reason for the exposure, dose ingested, clinical effects and patient outcome. Results: There were 103 methylphenidate human exposures. The following table summarizes the values for selected parameters that were investigated:

AGE GROUP	<6	6–12	13–19	>19
Mean Age ± SD	2.9 ± 1.4 years	8.5 ± 2.1 years	15.0 ± 1.6 years	33.0 ± 10.1 years
Mean Dose ± SD	13.6 ± 8.2 mg	26.8 ± 18.7 mg	106.8 ± 177 mg	70.0 ± 73.9 mg
Unintentional	100%	80.8%	26.7%	44.5%
Intentional	0%	19.2%	73.3%	54.5%
Symptomatic	16.0%	30.8%	50.0%	54.5%
Asymptomatic	84%	69.2%	50.0%	44.5%
Sample Size	25	26	30	22

Conclusions: The majority of exposures in children ≤12 years of age involved unintentional ingestion of a sibling's medication, self-administration of an excessive therapeutic dose or the administration of an inadvertent dose given by a caregiver. Abuse was common in adolescents and adults. Regardless of the reason for the exposure the amount ingested or treatment, all exposures had a favorable outcome. Pediatric doses of less than 1 mg/kg were not associated with adverse events.

163 ADVERSE EVENTS REPORTED WITH THE USE OF GAMMA BUTYROLACTONE PRODUCTS MARKETED AS DIETARY SUPPLEMENTS.

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Background: Gamma butyrolactone (GBL) [2(3H)-furanone dihydro] has been marketed as a dietary supplement in the U.S. with a variety of claims (e.g., stimulation of growth hormone release, facilitation of sleep, sexual enhancement, "relief" from addictions). GBL is rapidly metabolized to gamma hydroxybutyrate (GHB), a drug of abuse known for its euphoric effects and associated with coma and severe respiratory depression. Because of the severity and number

of adverse event reports to FDA involving GBL-containing products, FDA took regulatory action and issued a talk paper requesting the voluntary recall of these products. Case Series: From April 1998 until April 15, 1999, FDA received 119 adverse event reports related to the use of GBL (age 11–71 years, median 27 years; 63% male/37% female). The majority of reports were submitted from Poison Control Centers (PCCs) (60%), the remainder being from health care providers and consumers. Two deaths were reported in association with the ingestion of GBL. Typical symptoms of acute GBL intoxication were: CNS depression, respiratory depression [at least 19 patients (17%) required intubation for ventilation or airway protection], bradycardia, myoclonus, vomiting, confusion, and combativeness. Symptoms of "addiction" and/or withdrawal were reported in 9 patients (7.5%). Conclusions: GBL toxicity is similar to GHB. Adverse event reporting by PCCs to FDA is critical in the monitoring of GBL-associated adverse events due to treatment of these patients in emergency rooms.

164 SEIZURE ASSOCIATED WITH BUTANEDIOL INGESTION.

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Background: γ-hydroxybutyrate (GHB) is a CNS depressant with rapid onset and offset. Common patient populations include weight lifters, participants in rave parties, and in victims of drug associated date rape. 1,4-butanediol (pine needle oil) is converted by alcohol dehydrogenase to γ-hydroxybutyraldehyde, which is then converted to GHB by aldehyde dehydrongease. Butanediol is not a controlled substance and can be easily purchased from chemical supply companies. Case Report: Patient is a 39-year-old male with a history of depression that was well managed on bupropion 200 mg bid. He occasionally had insomnia and was active in several Internet "chat groups" where he learned about butanediol. He purchased a 1-liter container of butanediol directly from a chemical supply company and began ingesting approximately 10-mL aliquots each night to enhance sleep. On the seventh night, he had a single, witnessed 3-minute tonic episode associated with an altered mental status and incontinence of stool. The seizure resolved spontaneously. When seen in the emergency department immediately afterward, he was asymptotic with a normal neurological examination. The complete blood count, electrolytes, calcium, and drug screens were negative. An EEG performed in the Emergency Department was negative and he remains well 5 months post-ingestion. Conclusion: GHB has occasionally been associated with seizure activity. This is the first reported case of butanediol associated seizures. It is uncertain as to whether butanediol or GHB induced the seizure. Health care providers must be aware of the availability of information via the Internet and the ease of legally obtaining chemical precursors to potentially dangerous illicit drugs.

165 GHB WITHDRAWAL SYNDROME: EIGHT CASES.

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Background: Gamma Hydroxybutyrate, GHB, an unapproved drug in the United States, has been responsible for numerous cases of acute poisoning in the past 9 years. The acute symptoms; coma, myoclonus and bradycardia, resolve rapidly in the absence of complications. We are reporting a more prolonged symptom course that occurs after discontinuing chronic use of GHB or its precursors. Case series: Eight patients ranged in age from 22–38 years of age, most (88%) were employed. Bodybuilding was frequently (63%) the motivation for drug use. Urine toxicology screens were negative for common drugs of abuse except for two patients. One patient with a fractured leg had opiates and THC present. Another patient's urine contained benzodiazepines that were prescribed therapeutically for early withdrawal symptoms. All patients reported frequent ingestion of GHB, every 1–3 hours around-the-clock with nighttime awakening to take doses. The duration of GHB use was over 2 months to 3 years. Some cases had recent escalations in their dose. In 3 patients the product was available and analyzed allowing estimation of the dose which ranged 43–144 g/d. The onset of withdrawal symptoms, insomnia, tremor and confusion began within 1–6 hours after the last dose of GHB. All the patients experienced the central nervous system symptoms of tremor, auditory and visual hallucinations, and delirium requiring restraints. Autonomic stimulation with tachycardia was present in all of these patients. The duration of symptoms ranged from 5–15 days with one death occurring on hospital day 13 as symptoms were resolving; the direct mechanism is unclear. Conclusion: The GHB withdrawal syndrome is similar to other sedative, hypnotic and alcohol withdrawal syndromes. Chronic ingestion with short dosing intervals is required for dependence due to the unusually rapid elimination of GHB.

166 γ-BUTYROLACTONE (GBL) WITHDRAWAL PRESENTING AS ACUTE PSYCHOSIS.

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Backround: GBL, a precursor of γ-hydroxybutyrate (GHB), is a naturally occurring metabolite of γ-amino butyric acid (GABA) and is a common drug of abuse. Once ingested GBL is metabolically converted to GHB and alters the regulation of GABA, dopamine, 5-hydroxytryptophan, and acetylcholine. While acute intoxication with GBL or GHB is well described in the literature, GBL withdrawal is not well characterized. This report details acute psychiatric manifestations of GBL withdrawal in the absence of metabolic disturbances. Case Report: A 36-year-old woman using 10–15 cc/d of "Invigorate™" (3.5 g GBL/oz) for a 90 day period presented to the ED 48 hours after her last use with an acute psychosis. She had no past medical or psychiatric history and denied alcohol or other drugs. Physical exam: T 98.2F, RR 20, BP 130/84, P 64. Psychiatric findings included thought-blocking, tangential cognition, delusions, and visual and auditory hallucinations. Remainder of the exam was unremarkable. CT scan, ECG, and serum chemistries were unremarkable. CBC was significant only for WBC of 13,400/mm^3. Urine drug, hallucinogen screen, and GHB were negative. Her mentation improved with a single dose of lorazepam (1 mg) and haloperidol (2 mg). Collateral confirmation of return to baseline mentation occurred 48 hours after admission. Conclusion: GBL/GHB withdrawal has been described as alcohol/benzodiazepine-like with resultant sympathetic hyperactivity, fully absent in this case. GBL/GHB may produce pure psychiatric withdrawal symptoms without autonomic dysfunction or metabolic derangement seen with alcohol or sedative hypnotic withdrawal.

167 EARLY PREDICTORS OF SEVERITY IN RESISTANT ALCOHOL WITHDRAWAL (AW).

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Background: While most patients in AW normalize their vital signs with small doses of benzodiazepines (BZ), a subgroup is refractory to standard therapy. These resistant patients require excessive doses of BZ, additional sedatives, and extended ICU stays. Hypotension, prolonged unconsciousness and ventilatory insufficiency complicate their course. This observational study was designed to identify early predictors of resistant AW (RAW). Methods: 19 adults admitted to an inner city hospital for AW who required 50 mg of diazepam (DPM) IV in the first hour of treatment were prospectively enrolled and followed for 24 hours. Age, sex, EtOH level, and maximal CIWA-Ar score (when available) were recorded. Hourly and total doses of IV DPM with concurrent vital signs were recorded at specified intervals. Patients were classified as having RAW if they required barbiturates (Barbs) to control their AW symptoms, or had persistent vital sign abnormalities at 24 hours, or as having non resistant AW (NRAW) if their vital signs normalized within 24 hours with BZ only. Results: 4 patients had NRAW, and required DPM in an hourly (and total) mean dose of 62 mg (62 mg), 32 mg (95 mg), 31 mg (128 mg), 20 mg (146 mg), 28 mg (174 mg), 10 mg (184 mg) at 1, 2, 3, 6, 12, and 24 hours, respectively. The 15 RAW patients required DPM in an hourly (and total) mean dose of 60 mg (60 mg), 45 mg (105 mg), 115 mg (220 mg), 156 mg (376 mg), 189 mg (565 mg), and 113 mg (678 mg) at 1, 2, 3, 6, 12, and 24 hours respectively. 14/15 of the RAW patients required Barbs, 7 were intubated and there were 5 episodes of hypotension. EtOH levels, CIWA-Ar scores, signs and symptoms, and vital signs were similar on admission. Neither hourly nor total BZ doses were statistically different at 1, 2, 3 hours. Both hourly and total BZ doses were significantly different at 6 and 12 hours. At 24 hours only the total dose was different (all by ANOVA). All NRAW patients had normal vital signs by 3 hours with a total DPM dose <200 mg. 14/15 RAW patients had abnormal vital signs at 3 hours despite DPM (range: 60–440 mg). Conclusion: At 3 hours, persistently abnormal vital signs or requiring a total DPM dose >200 mg may be early predictors of RAW. RAW patients receiving Barbs may have hypotension, require intubation, and therefore need ICU monitoring. Prospective validation is warranted.

168 NALTREXONE MISADVENTURES IN OPIOID-ADDICTED INDIVIDUALS: PROLONGED OPIOID-WITHDRAWAL WITH DELIRIUM.

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Background: Naltrexone is a long-acting, opiate antagonist used for rapid-opioid detoxification and to prevent recurrence of opioid dependence. Naltrexone was recently made available on general prescription to treat alcohol and opioid dependence in Australia. We report 3 cases of naltrexone-induced opioid withdrawal with associated delirium in patients attempting self-detox without medical supervision. Case-1: 44-year-old M, on 130 mg of methadone daily, presented

soon after ingestion of 2 × 50 mg naltrexone tablets obtained from a friend. Exam revealed piloerection, vomiting, agitation, yawning, and confusion. P 70/min, BP 140/80 mmHg, T 36°C, RR 30/min. Symptoms resolved over the next 12 hours. Case-2: A 27-year-old F ingested 50 mg naltrexone obtained from her primary care physician (PCP). Last heroin use was 10 hours previously. She complained of nausea, vomiting, diarrhea and abdominal pain. Pulse 90/min, BP 100/50, T 36.5°C, RR 25/min, GCS-15. Two hours later disorientation and confusion developed. Symptoms resolved over the next 12 hours. Case-3: 22-year-old M presented 1 hour following ingestion of 50 mg naltrexone prescribed by his PCP. Last heroin use 10 hours earlier. On arrival nausea, vomiting, diarrhea, muscle cramps, yawning, piloerection, disorientation, confusion, and agitation noted. P 60/min, BP 110/60 mmHg, T 36°C, RR 30/min, mydriasis. Symptoms persisted for 16 hours. Treatment: All 3 patients received IV fluids, ondansentron, clonidine, and octreotide. Conclusion: Acute withdrawal resulting from naltrexone misuse may be prolonged and severe in opioid-dependent individuals and may include an acute confusional state. Hospital admission will be required to treat prolonged withdrawal symptoms. Widespread availability of naltrexone will likely result in further presentations of severe withdrawal to hospital.

169 DEATH FROM INTENTIONAL IV ADMINISTRATION OF BENZONATATE.

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Introduction: Benzonatate, chemically similar to tetracaine, is a nonnarcotic oral antitussive agent formulated as a perle. While the literature reports at least 3 deaths from ingestion, we present the first death from IV administration of benzonatate. Case report: Hearing a loud noise in the bathroom, the patient's spouse discovered this 21-year-old male lying on the floor pulseless, breathless, and unresponsive to verbal or physical stimulation. A tourniquet, syringe, 2-3 aspirated benzonatate perles, and a small amount of blood at the antecubital injection site were also noted. The spouse performed CPR until the paramedics arrived. After intubation, the patient was found to be in ventricular fibrillation which progressed to asystole after two rounds of defibrillation. In the ED resuscitation for continued asystole resulted in VT with a pulse, then sinus tachycardia (120 bpm) with a BP 100/70 on dopamine. NG tube placement yielded bloody drainage. Examination was initially remarkable for dilated pupils, absent heart tones, poor capillary refill, unresponsiveness to all stimuli, absence of corneal reflexes, hypoactive DTRs, GCS 3, and absence of head trauma. The initial ECG demonstrated sinus tachycardia (150 bpm), QRS 0.94 and ST depression diffusely over all limb and chest leads. CXR revealed acute pulmonary edema. Lab data were remarkable for initial ABG 6.64/119/35/12, PT 15.8, PTT 90.4, and INR 1.9. Head CT revealed diffuse brain edema, a subarachnoid hemorrhage and no evidence of midline shift. EEG several hours later was consistent with brain death. Conclusion: We report the first case of intentional IV administration of benzonatate. The mechanism of death included dysrhythmias, coagulopathy and subarachnoid hemorrhage.

170 SUBSTANCE ABUSE USING TELAZOL®: AN ANIMAL TRANQUILIZER.

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Background: Telazol® (Tiletamine Hydrochloride 50 mg/ml Zolazepam Hydrochloride 50 mg/ml) is utilized in veterinary medicine as an animal anesthetic. A MEDLINE search revealed no human exposures reported, even though the manufacturer reports 2 exposures over the last two years. We report here a woman who used Telazol as a drug of abuse. Case Report: A 30-year-old female employee at a local zoo was found "down" by fellow workers in a clean animal treatment room. Initial reports were that she may have taken intravenously veterinary grade diazepam, and Telazol, a small animal anesthetic. On scene paramedics reported her GCS = 10, BP = 90/palp, O_2 sat normal, gag intact. She was arousable to deep painful stimuli. A fresh track mark was present on her right arm; nearby were a syringe, tourniquet, and bottles of each drug. ED treatment included ABC, IV access, lavage, activated charcoal with a cathartic. Shortly after arrival she became alert and oriented. Family members insisted this was not an OD. Patient had been previously evaluated for reported syncope, "only in the evening, while at work," and prescribed diazepam for anxiety. Product information on Telazol was not available, except from the Veterinary Drug PDR. A urine toxic screen was positive for benzodiazepines and cannabis. In the ICU the patient revealed a history of recreational use of Telazol. She was discharged to an in-patient detoxification facility, 12 hours post-admission. Conclusion: Telazol, used in veterinary medicine as an anesthetic agent, is structurally related to ketamine. Telazol causes almost immediate anesthetic effects; and sudden alertness is not uncommon as the effects of the drug subside. Telazol has abuse potential among workers in veterinary medicine.

171 MULTIPLE SMALL-VOLUME SUBCUTANEOUS WD-40 INJECTIONS WITH SEVERE LOCAL AND SYSTEMIC TOXICITY.

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Background: WD-40 is a commonly used lubricating agent consisting of petroleum distillates and oils. Hydrocarbon injections are rare compared to oral, inhalational, or cutaneous exposures. Most reports focus on high-pressure injection injuries. We present a case of local and systemic toxicity after a low-pressure, subcutaneous injection of a small amount of WD-40. Case Report: 24-year-old Hispanic male presented with bilateral thigh and buttock pain and fever approximately 24 hours after 11 subcutaneous injections with a total of 0.5 mL of WD-40. The purpose was to induce "weight loss." He had no significant past medical history and denied the use of medications, tobacco, ethanol, and intravenous drugs. Vital signs included HR 145, BP 126/77, RR 20, and T 37.8°C. Physical exam was benign except for multiple 6–10 cm^2 areas of cellulitis with central induration but no fluctuance. Some areas coalesced. Photographs are available. His WBC was 24,100 with 86% segmented cells. He was treated with IV antibiotics and analgesics. Blood and tissue cultures were sterile. He improved and was discharged on oral antibiotics after the third hospital day. Skin lesions and pain were completely gone within 2 weeks of the exposure. Conclusion: Previous reports reflect that subcutaneous and intramuscular injections of hydrocarbons commonly cause cellulitis, tissue necrosis, abscess formation, fever, and leukocytosis; with sterile cultures. This case is unique because it is the smallest amount of a hydrocarbon reported to be injected per site. Despite the very small volume injected per site (0.05 mL), hydrocarbons can cause significant local and systemic toxicity. The lack of tissue necrosis or abscess formation likely resulted from the small volume injected.

172 A CASE SERIES OF ACCIDENTAL EPINEPHRINE AUTO-INJECTOR INJURIES: INVASIVE TREATMENT NOT ALWAYS REQUIRED.

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Background: Numerous case reports in the literature describe individuals with accidental parenteral exposures to epinephrine auto-injectors, although no large case series has been published. These individual reports represent the worst scenarios giving the impression that all exposures must be treated in an emergency facility with invasive procedures. We present 28 cases of exposures to epinephrine via autoinjector. Case Series: All accidental parenteral epinephrine auto-injector exposures reported to two RPIC's over a one year period were included. Four of these involved Epipen Jr while 24 patients were exposed to Epipen. Injection sites included digits in 23 cases, the palm in four cases and one thigh injection. Symptoms included swelling, pallor, pain, and erythema. Four patients reported no effect and nine required no treatment, three had nitroglycerin paste applied, one had nitroglycerin paste and phentolamine, one nitroglycerin paste and terbutaline and one had phentolamine alone. Ten patients had relief with warm soaks, compresses and massage, 1 patient had massage only and 2 were lost to follow-up. Fourteen patients were examined in the emergency department. Only 5 were referred by the poison center. One of those examined in a HCF was a pregnant female who developed mild contractions. Fourteen were treated at home. Of the 28 suspected accidental injections, only 6 (21%) required medical treatment that could not have been given at the site of exposure. Conclusion: Although some injection injuries must be treated in an emergency facility, many can be treated at home. Immediate referral to a health care facility is not needed in all cases and at times is unwarranted.

173 THE ATTITUDES OF US REGIONAL POISON CENTERS TOWARD PHYSOSTIGMINE FOR ANTICHOLINERGIC DELIRIUM.

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Background: Controversy exists surrounding the role of physostigmine (PHY) in managing patients with anticholinergic delirium. Although extremely effective as an antidote, concerns about the safety of indiscriminate use of PHY have led to widespread discouragement of its use, even for cases when it would be of therapeutic benefit. Methods: We conducted a structured telephone survey of certified US regional poison centers (PCs) to determine their standard recommendations for an 18-year-old male with severe agitation (unresponsive to 30 mg diazepam) after ingesting diphenhydramine. If asked for, peripheral manifestations of the anticholinergic syndrome were present, and contraindications to PHY (e.g. ECG abnormalities) were absent. To assess bias due to the survey methodology, several PCs were randomly selected to receive a scripted call designed to simulate an actual case, and were thus blinded to the survey nature of

the call. Results: Only 6 of 19 PCs (32%) advised the use of PHY. No PCs (0/7) recommend PHY when called with the "real" case (blinded PCs). Of these, 2/7 mentioned PHY, but both discouraged its use; after the caller asked specifically about PHY in the remaining 5 cases, each PC tried to dissuade the caller from its use. On the other hand, PCs given the hypothetical case (unblinded PCs) advised a trial of PHY in 6/12 cases (50% vs 0%, p = 0.034). Among calls transferred from the PC to a toxicologist, 0/5 recommended PHY when blinded to the survey, compared to 6/9 unblinded (0% vs 67%, p = 0.028). 7/9 toxicologists stated they had used PHY in their practice in the recent past. Conclusions: Although divided in their willingness to recommend physostigmine for a hypothetical case of anticholinergic delirium, poison centers invariably recommended against its use in actual practice. This discrepancy in recommendations also raises important methodological issues regarding the validity of using unblinded hypothetical scenarios to assess current treatment practices.

174 SEIZURE FOLLOWING INADVERTENT APPLICATION OF A NICOTINE PATCH.

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Background: Nicotine is present in a wide variety of tobacco products. The majority of exposures to nicotine result in no toxicity, but serious incidents do occur. Ingestion of tobacco usually induces emesis shortly after ingestion, thus limiting the absorption of the toxic moiety. Case Report: The mother of an 11-month-old stated that her child had seemed very fussy after his morning bath. He vomited three times approximately one hour and thirty minutes after bathing. Within fifteen minutes he experienced violent, jerky muscle movements lasting 1 minute and his eyes "rolling around in his head." The mother called 911, and EMS arrived approximately eight minutes later. The paramedics noted a very lethargic child. The emergency department physicians noted a child who appeared pale, post-ictal, afebrile, with a blood pressure of 131/79 and a heart rate of 110. His respiration's were 24 per minute. Blood gasses were drawn and a pH of 7.29 was noted. Pupils were equal and reactive. The child was monitored with little change over the next hour. At that time, the child's diaper was removed, and it was discovered that the child had a Nicoderm® 21 mg/24 hour patch adhered to his buttocks. The patch was removed and the area was cleansed at that time. Within fifteen minutes of removing the patch, the child began emerging from the lethargic state which had persisted since arrival. The child was admitted to the pediatric intensive care unit where his symptoms completely resolved within four hours. He was discharged the next day with no subsequent complications noted. Investigation revealed an attempt was made to discard the used patch in the trashcan at home, but it landed instead on the floor. The parent stated that after his bath the child was placed naked on the floor where he liked to scoot around on his behind, apparently leading to the unintentional placement of the nicotine patch on his buttocks. Conclusion: We present a case of a pediatric seizure as a result of an unintentional dermal exposure to a nicotine patch. Care should be utilized when nicotine is present in any form in an environment where children may have access. This case illustrates the potential for severe toxicity that can result from exposure to nicotine.

175 UNEXPLAINED OSMOL GAP FOLLOWING LACQUER THINNER INGESTION.

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Background: The osmol gap is commonly used as a marker for toxic alcohol poisoning. We recently treated a patient who ingested lacquer thinner. No toxic alcohol was detected but over an 8 hour period the osmol gap increased from 15 mmol/kg to 31 mmol/kg. Case Report: The patient presented after ingesting ~250 mL of lacquer thinner. He had a solvent odor to his breath and was drowsy with slurred speech and nystagmus. Vitals were normal. Ethanol, salicylates, and acetaminophen were not detected. Electrolytes and blood gases were normal. The anion gap was 4 mmol/L. The osmol gap was 15 mmol/kg. An ethanol infusion was started. Three hours later methanol, ethylene glycol, acetone, and isopropanol were reported as negative but the osmol gap (accounting for ethanol) had increased to 20.5 mmol/kg. Ethanol was continued and serum reanalyzed. At 9 hours the osmol gap had increased to 31 mmol/kg but no toxic alcohols were detected and the patient had regained his normal mental status. Ethanol was stopped and the patient was discharged to psychiatry. Laboratory Methods: Three serum samples were analyzed by gas chromatography with head space analysis. No toxic alcohol was detected but volatile substances later identified as methyl ethyl ketone, toluene and xylene were present. We were unable to quantify these substances but the toluene and xylene peaks increased with time. Conclusion: We have presented a patient with an elevated osmol gap following lacquer thinner ingestion. Methyl

ethyl ketone, toluene and xylene appear to have contributed to the osmol gap and should be considered when confronted with an unexplained osmol gap. Ongoing absorption and inhibition of hepatic metabolism likely contributed to the observed increase in osmol gap.

176 INTRAVENOUS MEDICATION ADDITIVES: A REFINEMENT IN THE INTERPRETATION OF THE ELEVATED OSMOL GAP.

Velendzas D, McKay C. *University of Connecticut Medical Center, Farmington, CT*

Background: Medical toxicologists are often consulted on patients with unexplained osmol gaps. These laboratory calculations can be effected by many things. Among these are the osmol contributions from IV medication additives, including propylene glycol (PG) and glycerol (G). Methods: We reviewed many common IV infusions utilized in our ICU to determine the presence of osmotically active additives and evaluated the contribution of PG in one patient on high-dose infusions of lorazepam and nitroglycerin. Serum PG levels were measured by gas chromatography with flame ionization detection. Total osmol gaps were calculated and compared with the calculated PG osmol contribution. Results: Several commonly used infusions contain significant amounts of PG and G including lorazepam (PG), diazepam (PG), etomidate (PG), pentobarbital (PG), phenytoin sodium (PG), nitroglycerin (PG), and propofol (G). The following table summarizes the osmol contribution in the patient described:

Sample	Total osmol gap	Serum [PG] mg/dL	PG osmol contribution
1	24	97.7	13
2	32	168.7	22
3	53	406.4	53
4	55	348.8	46

Conclusions: Unexplained osmol gaps in a patient on multiple IV infusions may well be caused by the accumulation of propylene glycol or other osmotically active additives. Confirmation of their osmol contribution is most accurately determined by quantitative gas chromatography. When laboratory confirmation is not available, their osmol contribution can be estimated from the infusion rate and the calculated elimination half life.

177 DERIVATION OF A FORMULA TO CALCULATE THE CONTRIBUTION OF ETHANOL TO THE OSMOLAR GAP.

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Objective: To evaluate the relationship between osmolar gap and serum ethanol level and derive a formula which can be utilized clinically to calculate the expected osmolar gap in the presence of ethanol. Some investigators have noted that the osmolar gap appears to increase as ethanol level increases but we were unable to find references relating the osmolar gap and the ethanol level. Methods: In part A, a convenience sample of Emergency Department patients undergoing serum ethanol determination had Na, BUN, glucose and osmolality determined on the same blood sample and prospectively recorded. Predicted osmolality was calculated using the formula 2Na + BUN + Glucose + ETOH and the gap was determined. In part B we repeated this experiment *in vitro*. We added known amounts ethanol to serum. Data was entered into SPSS (v.6.1 MacIntosh) for analysis. Results: Fifty observations were made on 43 patients. The mean ethanol level was 44.3 mmol/L (range 0–105), the mean osmolar gap was 55.6 (range 0–130), and the relationship between these two variables was linear (Pearson's coefficient of correlation 0.99). Linear regression generated a model with the formula Predicted Gap = 1.51 + (1.22 × ethanol level). The 95% confidence interval for the additive constant for this formula was −1.07 to 4.10 (SE 1.28). The 95% confidence interval for the multiplication factor was 1.17 to 1.27 (SE 0.024). Eight *in vitro* experiments with 3–6 observations per experiment produced a similar result and provided strong evidence that the increasing gap with increasing ethanol level is not due to unmeasured osmoles. Conclusion: There is a linear relationship between serum ethanol level and osmolar gap. The formula we derived has potential clinical applications and warrants prospective validation.

178 BITREX AND PEDIATRIC TOXIC ALCOHOL POISONINGS: INTERIM REPORT.

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Background: In 1995, Oregon became the first state in the US to mandate the addition of a bitter aversive agent to consumer automotive products containing ≥10% ethylene glycol (EG) or ≥4% methanol (MetOH). The recommended agent was denatonium benzoate (Bitrex™, MacFarlan Smith Ltd) at a concentration of 30–50 ppm. The intent of the Toxic Household Products statute was to reduce the severity and lethality of accidental pediatric poisonings by EG and MetOH. Methods: Retrospective review of 1) poison center records from 1987 through 1998 with respect to pediatric (<6 yrs) exposures to automotive antifreeze (EG) and windshield washer fluid (MetOH), 2) poison center charts of all children treated with ethanol infusion or hemodialysis for EG or MetOH poisoning in 1987–1998, 3) medical examiner reports of poisoning related deaths for a 4 year period (1994–1997) to identify EG or MetOH deaths not reported to the PCC, and 4) AAPCC TESS fatality data for 1984–1997. Results: Our PCC recorded 229 EG exposures and 80 MetOH exposures among preschool children from 1987–1998 with no trend in total annual frequency before or after 1995. No cases resulted in death or "major" effects. Seven children received EtOH infusion for suspected EG or MetOH poisoning (all before 1995). However, 6 had no detectable level of the suspected agent, and only 1 child had an [EG] = 9 mg/dL in the ED (infusion was stopped when the EG level was known). No child underwent HD. Coroner reports from the state health division detected 1 MetOH death (deliberate suicide in an adult) and no EG deaths in 1994–1997. AAPCC TESS fatality reports detected 4 preschool age deaths attributed to EG or MetOH in 14 years. The only EG death was a misdiagnosis of methylmalonic acidemia. All 3 MetOH deaths were attributed to actions of a parent or babysitter. Conclusion: Because deaths and serious illness rarely occur among children with accidental EG or MetOH exposures, we were unable to demonstrate any effect of Bitrex on morbidity and mortality from pediatric EG or MetOH exposures.

179 FOMEPIZOLE TREATMENT OF ETHYLENE GLYCOL POISONING IN AN INFANT.

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Background: Fomepizole has not been studied adequately in the pediatric population. Case Report: An 8-month-old 7.7-kg boy drank up to 120 mL ethylene glycol 95% (EG). He was taken to a hospital, where he appeared lethargic and was intubated. Ethanol was loaded and infused intravenously. After transfer to a children's hospital, ethanol was discontinued (level = 40 mg/dL) and fomepizole was administered intravenously. Hemodialysis was begun 5 hours post ingestion for approximately 4 hours.

Time post Ingestion (h)	pH	Base deficit	Osmol gap (mOsm/kg)	EG (mg/dL)	Urinalysis	Fomepizole Dose (mg/kg)
3	7.32	7	60	384		
4					3+ crystals	15
10	7.36	2	8			10
15	7.37	2		54	90–100 rbc	
20					TNTC rbc	
22						10
33	7.42	1	3	11		
34						10
45			0		0–2 rbc	

Conclusion: We believe this is the first report of fomepizole use in an infant. Fomepizole effectively blocked the production of EG toxic metabolites after dialysis was completed. He recovered within 48 hours.

180 FOMEPIZOLE ELIMINATION KINETICS IN FULMINANT HEPATIC FAILURE.

Wax P, McMartin K. *University of Rochester, Rochester NY; Louisiana State University Medical Center, Shreveport, LA*

Background: Fomepizole (4MP) is predominantly eliminated by hepatic metabolism. We present a patient in fulminant

liver failure who had an ethylene glycol (EG) level of 50 mg/dL and was treated with a single dose of fomepizole. Serial serum fomepizole levels were obtained over the next 7 days to investigate the elimination of fomepizole in this virtually anhepatic patient. Case Report: A 38-year-old male presented to the ED with fulminant hepatic failure from chronic acetaminophen (APAP) and ethanol abuse. Upon presentation he was unresponsive and had a pH of 7.15, AST of 8580 IU/L, T Bili of 2.5 mg/dL, ammonia of 119 μmol/L, INR of 6.1, creatinine of 1.8 mg/dL, APAP level of 31 mcg/mL, and ethylene glycol level of 50 mg/dL. The patient was treated with NAC and a single intravenous dose of fomepizole 15 mg/kg (total 1050 mg). Subsequently, the EG level was discovered to be a false positive and no further fomepizole was given. The initial fomepizole level 1 hour after administration was 204 μmol/L. The rate of elimination was 2.9 μmol/L/h. In the recent clinical trial on fomepizole in EG poisoning the rate of elimination was 16.5 ± 3.1 μmol/L/h. Conclusion: Therapeutic doses of fomepizole (4MP) are eliminated normally by nonlinear or zero order kinetics. These doses produce blood levels in the range of 100–300 μmol/L. Studies in healthy humans have shown that fomepizole is eliminated from the body primarily by hepatic metabolism. The nonlinear elimination probably results because of a saturation of this metabolic pathway. In this patient, the elimination of a single IV dose of 4MP was not saturated, but followed classic first order kinetics, with a half-life of 17.8 hours. In comparison, Phase 1 studies in healthy humans showed that the elimination of 4MP, after the initial saturation phase, was first order, with half-lives about 2 hours. These data suggest that in this patient the metabolism of fomepizole was completely blocked by the loss of liver function. Hence, the elimination occurred by excretory processes (most likely in the urine), which are first order processes.

181 BRAINSTEM INFARCTION AND QUADRIPLEGIA ASSOCIATED WITH ETHYLENE GLYCOL INGESTION.

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Background: The brainstem and tegmentum are extremely resistant to ischemic and toxic insults. CT scans and post mortem studies of patients that have ingested ethylene glycol have demonstrated cerebral edema, infarcts of the internal capsule and basal ganglia, and diffuse cerebral infarcts. Cranial nerve deficits after ethylene glycol ingestion with normal imaging studies have been well documented. There is no description in the literature of brainstem infarction associated with ethylene glycol exposure although a case of tetraplegia has been reported. Case report: A 17-year-old white male with a history of depression was brought to an outside hospital with slurred speech and decreased level of consciousness. The outside medical facility attributed his symptoms to benzodiazepines and administered flumazenil with no response. The patient continued to deteriorate and was noted to have a profound acidosis with a bicarbonate of 2.8 mmol/l and was subsequently transferred to our hospital approximately 18 hours after his initial presentation. The patient was started on an ethanol infusion during transport and then changed to 4-methylpyrazole after arrival. He was intubated and remained acidotic with a pH of 7.1 and a HCO_3 of 7 mmol/L. Further history from his parents revealed a profound upper extremity weakness and lower extremity paresis prior to arriving at the initial hospital. Due to these symptoms, a head and cervical spine MR scan was obtained which revealed bilateral basal ganglia and brainstem infarcts. The ethylene glycol level was 29 mg/dL at 18 hours post-ingestion. The following day the patient's pupils became fixed and dilated. Repeat MR scan with perfusion images revealed no cerebral blood flow, diffuse cortical ischemia, and bilateral uncal herniation in addition to the previous findings and life support was subsequently withdrawn. Conclusions: This case demonstrates a possible association of ethylene glycol causing direct neurotoxic effects at the brainstem.

182 PULMONARY SYMPTOMS FOLLOWING DELAYED EXPOSURE TO TOLUENE DIISOCYANATE BASED VARNISH.

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Background: Toluene diisocyanate (TDI) is a respiratory tract irritant, a potent sensitizer, and a common component of varnish. When varnish dries and the area of use is ventilated, the risks of developing upper respiratory irritation and bronchospasm should decrease. We report the occurrence of irritative symptoms and bronchospasm three days after TDI containing varnish had dried and the area was ventilated. Case series: Thirteen workers moving voting machines into a school gym developed symptoms of upper respiratory irritation including cough and sore throat. The gym's hardwood floors had recently (3 days previously) been re-finished with TDI containing varnish. The floors were dry and the space was ventilated with fans before the workers entered the area. Three of the thirteen workers were dyspneic

with wheezing. Two of those with bronchospasm resolved with inhaled steroids. One worker was hospitalized and eventually diagnosed with reactive airway dysfunction syndrome (RADS). The ten workers not wheezing on presentation resolved their irritative symptoms following cool misted oxygen and intravenous fluid hydration. Conclusions: TDI induced upper respiratory symptoms and bronchospasm in workers can be caused by residual TDI fumes despite the passage of significant time after application and despite ventilation.

183 CHLORAMINE GAS EXPOSURE FROM HOUSEHOLD CLEANERS REQUIRING TRACHEOSTOMY AND VENTILATORY SUPPORT.

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Background: Liquid ammonia and bleach are two of the most common cleaning agents available. Mixing of the two may result in the release of chloramine gas which can produce respiratory irritation and pneumonitis. We found no previous reported cases of tracheostomy being required due to upper airway compromise and vocal cord swelling induced by chloramine gas. Case Report: A previously healthy 53-year-old woman was cleaning a walk-in freezer at her workplace using over-the-counter liquid ammonia and bleach. The door to the freezer was closed and there was no air exchange with the outside. Approximately 30 minutes after beginning, she noted increasing shortness of breath and called 911. Paramedics noted audible wheezes and administered nebulized albuterol. In the ED, the patient noted increasing tightness of her throat and developed aphonia. She received intravenous steroids and continued nebulized albuterol and vaponephrine without improvement. Rapid sequence intubation was attempted but unsuccessful secondary to swelling of her upper airway and tracheostomy was performed. Radiologic evidence of pneumonitis developed over the next four hours. Arterial blood gas revealed a pH 7.23, pCO_2 49 mm Hg and pO_2 102 mm Hg on 100% oxygen, with assisted ventilation. The patient received supportive care and her tracheostomy removed within 5 days and was discharged from the hospital within 7 days. Conclusion: Although infrequent, chloramine gas exposure represents a significant risk when household cleaners containing bleach and ammonia are mixed. Rarely, upper airway swelling is severe enough to compromise the airway and require a surgical airway.

184 CARBON MONOXIDE POISONING: UTILIZING A NATIONAL MEDIA CLIPPING SERVICE FOR SURVEILLANCE OF CO DETECTOR IMPACT ON SURVIVAL.

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Objective: We utilized a novel surveillance approach to explore the role of CO detectors in the prevention of CO-related deaths in the US. Methods: Using a national media clipping service, all cases of CO poisoning in the US cited in media reports between September 1994 and February 1998 were categorized according to time of year, site, and etiology of exposure; the impact of CO detectors was investigated. Results: 4564 total CO exposures resulted in 406 (8.9%) fatal and 4158 (91.1%) nonfatal outcomes. 3571 (78.2%) exposures occurred during October to March. 2617 (57.3%) CO exposures occurred in the home, accounting for 374 (92.1%) CO-related deaths. Faulty heating systems constituted 2540 (55.6%) CO exposures and 186 (45.8%) deaths with alternate heating sources responsible for 389 (8.5%) exposures and 104 (25.6%) deaths (26.7% case fatality rate). Other etiologies (% CO exposures/% case fatality rate) include cars running in garages (2.8%/63.5%), generators (1.4%/53.1%), grills in enclosed spaces (1%/26.7%), water heaters (4.8%/9.2%), stoves/ovens (3.0%/8.8%) and blocked chimneys (6.9%/6.6%). During the study period, cities with CO detector ordinances (Chicago and St. Louis) had significantly lower case fatality rates than those cities without ordinances ($p < 0.001$). 1008 (24.2%) survivors attributed their survival directly to the presence of a CO detector. CO exposure category and percent survival attributed to CO detectors were tabulated: faulty heating systems (56%), grills (42.4%), blocked chimneys (32.5%), cars in garages (28.3%), water heaters (21.7%), stoves/ovens (20%), generators (13.3%), single family dwellings (34.6%), schools/daycare (33%) and multiple family dwellings (12.2%). Conclusions: A media clipping service, an innovative investigational tool, provides meaningful insight regarding the impact of CO detectors on the prevention of CO-related deaths: 1008 (24.2%) survivors attributed their survival to CO detectors, and cities with detector ordinances have significantly lower case fatality rates than cities without the ordinances.

185 CARBON MONOXIDE DETECTORS: A GUIDE FOR SPIS.

Mrvos R, Krenzelok EP. *Pittsburgh Poison Center, Children's Hospital of Pittsburgh, University of Pittsburgh, Pittsburgh, PA*

Background: Carbon monoxide detectors have gained well-deserved popularity in American households and are recommended by a variety of both government and private agencies. The detectors are designed to sound an 85 decibel alarm when CO is detected and reaches a specific threshold, alerting the residents of the building before a tragedy occurs. The poison center may be the first resource contacted when a CO detector alarms, making it important for the SPI be knowledgeable about CO detectors and be prepared to evaluate each situation. To assist SPIs in managing these inquiries, a RPIC developed a CO detector response protocol. Methods: A flow chart based on CO detector standards and the clinical toxicology of CO was developed with clear triage and treatment guidelines. Results: If anyone in the home is symptomatic the caller is advised to evacuate the residence and contact 911. If no one in the home is symptomatic, the caller is instructed to ventilate the premises, turn off all combustion sources and contact a trained repair service. The SPI asks a series of questions to assist the caller in locating the problem such as: were combustion appliances (stove furnace, hot water heater, etc.) or fireplace in use, is there an attached garage and was a gas powered vehicle running in the garage prior to the alarm, and the placement of the detector in relation to these events. If there is no indication of the source, the caller is told to contact their HVAC contractor for further assistance. Conclusion: When a SPI receives a call regarding an alarming CO detector they must rapidly assess the situation and recommend appropriate response, referral or treatment. By following the CO Detector Alarm Flow Chart, they can be assured the caller is getting complete and adequate instructions without putting undue stress on first responders, utilities or other agencies.

186 ASPHYXIATION BY CARBON DIOXIDE DUE TO SUBLIMATION OF DRY ICE IN A CLOSED SPACE.

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Background: Simple asphyxiation due to CO_2 is uncommon despite the widespread use of dry ice. Dry ice, or compressed carbon dioxide snow, sublimates under standard conditions and liberates CO_2. Since CO_2 is denser than air, it descends upon release and displaces oxygen from the ground upward. In addition, the ambient concentration of CO_2 rises. Symptoms in exposed patients are related to both a reduced fraction of inspired oxygen (FIO_2) and an elevated fraction of inspired CO_2 ($FICO_2$). Case Report: A 50-year-old medical researcher was discovered dead in a refrigerated room (8′W × 14′D × 8′H) that contained 15 new blocks (10″ × 10″ × 10″) of dry ice. The dry ice had been stored in the refrigerator (4°C) at approximately 9 a.m. that day to reduce their sublimation. The researcher had last been seen at approximately noon, indicating that at least three hours had elapsed between storage and exposure. It appeared that at the time of death the descedent was crouching to store samples in a container several inches from the ground. There were no signs of struggle and the descedent had no history of psychiatric disorders, recent personal crises, or medical illnesses. The external ventilation system in the cold room was nonfunctional, although internal air movement occurred via the cooling fan. The blocks of ice appeared grossly intact. Post-mortem examination of the descedent was unrevealing, as was the toxicologic evaluation. A blood pCO_2 was not performed due to its well described rapid post-mortem rise. In order to confirm the cause of death, the conditions at the time of the event were reproduced exactly using the same cold room. Air was sampled serially at several heights; the O_2 concentration fell and the CO_2 concentration rose within 20 minutes, and peaked by 3 hours. The FIO_2 three hours after dry ice storage was 13.6%, and the CO_2 concentration was 27.6%, both at a height of 9 inches. The temperature of the room had fallen to −15°C, Conclusions: Cold storage at 4°C does not prevent the sublimation of dry ice. Closed space storage of dry ice may lead to rapid incapacitation and death due either to a deficiency of ambient oxygen or an excess of CO_2.

187 THE POISON CENTER ROLE IN BIOLOGICAL AND CHEMICAL TERRORISM.

Krenzelok EP, Allswede MP, Mrvos R. *Pittsburgh Poison Center, Children's Hospital of Pittsburgh, University of Pittsburgh, Allegheny University of Health Sciences, Pittsburgh, PA*

Introduction: Nuclear, biological and chemical (NBC) terrorism countermeasures are a major priority with municipalities, healthcare providers and the federal government. Significant resources are being invested to enhance civilian domestic preparedness by conducting education at every response level in anticipation of a NBC terroristic incident. The key to a successful response, in addition to education, is integration of efforts as well as thorough communication and understanding the role that each agency would play in an actual or impending NBC incident. Methods: In anticipation

of a NBC event, a regional counter-terrorism task force was established to identify resources, establish responsibilities and coordinate the response to NBC terrorism. Members of the task force included first responders, hazmat, law enforcement (local, regional, national), government officials, the health department and the regional poison information center. Response protocols were developed and education was conducted, culminating in all members of the response task force becoming certified NBC instructors. Results: As a recognized resource and task force participant, the poison center participated actively in three incidents of suspected biologic and chemical terrorism: an alleged anthrax-contaminated letter sent to a women's health clinic; a possible sarin gas release in a high school; and a potential anthrax/ebola contamination incident at an international airport. All three incidents were determined to be hoaxes. The regional response plan establishes the poison information center as the common repository for all cases in a biological or chemical incident. Conclusions: The poison center is one of several critical components of a regional counter-terrorism task force. It can conduct toxicosurveillance and identify sentinel events. To be responsive, the poison center staff must be knowledgeable about biological and chemical agents. The development of basic protocols and a standardized staff education program is essential. The use of the RaPiD-T (R-recognition, P-protection, D-detection, T-triage/treatment) course can provide basic staff education for responding to this important but rare consultation to the poison center.

188 EMERGENCY DEPARTMENT PREPAREDNESS FOR CHEMICAL, RADIOLOGIC OR BIOLOGICAL CASUALTIES.

Gummin DD, Lee MS, Stein-Spencer L, Lee TE, Glushak C, Aks SE. *Illinois Emergency Health Care Team (Illinois Department of Public Health), Cook County Hospital, Toxikon Consortium, Chicago, IL*

Background: Use of non-conventional weapons by terrorist groups poses an increasing societal concern. National and local efforts are underway to assess risk and preparedness. We conducted a survey of all emergency departments (EDs) in our state to assess preparedness for managing patients after release of a biological, radiologic, or chemical agent. Methods: A seventeen-item questionnaire was distributed to the directors of all 188 EDs in the state in January 1999. Additionally, those same EDs were contacted by telephone in February or March 1999. The physician in charge or charge nurse was presented the same set of questions. Responses were tabulated. Results: Forty-one percent of ED directors responded. Complete telephone survey data was obtained for 66%. Eighty-six percent of all respondents confirmed hazardous materials exposure in their facility's disaster plan. Similarly, written protocols, protocols for contaminated patients and for spills were confirmed in 76–88% of responses. "Don't know" responses from ED staff regarding protocols outnumbered those from directors 2:1. Most directors and staff stated an ability to manage 1–5 casualties per institution. However, only 17% of directors and 23% of staff stated they'd used their decontamination facilities in the past year. Regarding decontamination capabilities, only a shower stall was consistently present with greater than 50% frequency. Directors acknowledged absence of decontamination facilities in 17%. Conclusions: Most institutions surveyed have protocols in place for handling these complicated patients in the event of a disaster. Facilities for decontamination appear less available than the protocols. Efforts to improve disaster preparedness in our state are indicated.

189 THE EXPANDING ROLE OF THE POISON CENTER IN DOMESTIC TERRORISM INCIDENTS.

Sawyers B, Orletsky P, Hilder R, Thomas, R. *Samaritan Regional Poison Center, Phoenix, AZ*

Background: The troubling new wave of domestic terrorism incidents is challenging poison centers to meet a need for information about agents of mass destruction. This is due in part to increased access to information about biological and chemical agents and technical information. The poison center has been called upon for information in several anthrax threats over the past year. Methods: Toxicologists and CSPIs have actively pursued training in all areas of biological, chemical, and nuclear domestic terrorism incidents, including a course developed by the US Department of Defense. We participated in a disaster drill simulating a chemical release inside a commercial airplane. This was the largest nonmilitary disaster drill in the history of the state and involved working together with a multitude of agencies including: several municipal fire departments, law enforcement agencies, hospitals, and airport personnel. Results: Based upon the evaluation of the disaster drill and experiences from anthrax threats, the poison center recognized the need for readily available information resources for area hospitals and emergency responders to assist them in handling these situations. The poison center developed one and two page rapid reference treatment monographs for responders and

providers who may be called upon to treat victims of domestic terrorism. Conclusions: Future incidents may be better managed with the additional information now available through the regional poison center.

190 POISON CENTER PREPAREDNESS FOR TERRORIST EVENTS.

Rodgers G, Matyunas N. *University of Louisville, Louisville, KY*

Background: There has been a surge in concern about the potential for nuclear, biological and chemical (NBC) terrorist events in the US. Poison centers can be expected to be important sources of public and professional information if such an event were to occur. Method: In late 1997 a survey was conducted of US poison centers to assess their degree of preparedness to respond to such an event. Results: Responses were received from 54 centers covering 40 states. Responding centers included 44/51 centers certified by the AAPCC at the time. All but one center had 24 h/d access by telephone although only 14/54 had radio connection to local or regional emergency services. 25/54 centers had call stacking capability allowing them to readily handle large call volumes. Only 10/54 centers had disaster plans. Centers were best prepared to deal with chemical events: 40/54 had some relationship with local/regional hazmat units and 42/54 had either a staff person or consultant with chemical expertise. Most centers had some reference materials relevant to chemical disasters: 21/54 could identify national/governmental resources in this area. Most centers had limited expertise or resources in the area of biological events: 20/54 had access to either in-house expertise or a consultant with expertise in biological terrorism and only 16/54 could identify a national/governmental source of information. 15/54 centers reported some in-house expertise with nuclear/radiation events, although few had any reference materials or could identify a national/governmental source of information. Conclusions: Most poison centers are poorly prepared to deal with an NBC terrorist event. Most centers expressed strong interest in expanding their knowledge and capabilities. This survey has resulted in a contract between the CDC and the AAPCC to improve the preparedness of centers.

191 THE POISON CENTER'S ROLE IN RESPONDING TO ANTHRAX THREATS.

Tharratt S, Alsop J. *California Poison Control System-Sacramento Division, Sacramento, CA*

Background: With the increasing risk of potential terrorist acts dealing with biological and chemical agents, it is vital that poison centers have an organized means of responding to requests for information. Case Report: Between December 17–23, 1998, there were four alleged threats of anthrax release in business and government buildings in the Los Angeles area. The threats arrived via letter and telephone. The threats claimed to have contaminated the air-conditioning and heating systems. Emergency Medical Services, Fire, HazMat, Police, FBI, County and State Departments of Health Services and CDC representatives responded to the scene. There was little contact between the Poison Control Center (PCC) and the on-scene responders. At the 1st threat, without input from the PCC, all potentially contaminated victims were decontaminated on-site and started on antibiotics. At the 2nd threat, all victims were started on antibiotics but not decontaminated. At the 3rd and 4th threat, no decontamination was done and no one was started on antibiotics. Area HCFs calling the PCC asked if all the victims needed to come in for further decontamination and treatment. They also wanted information on symptoms of anthrax and the potential risk to HCF staff caring for these patients. As additional anthrax threats continued to be called in across the State, a clear, organized guideline was needed to assist PCC staff in responding to inquiries that could involve mass numbers of victims. Results: A guideline was developed cooperatively between the PCC Medical Directors and Managers. The guideline integrates California's Standardized Emergency Management System (SEMS) and positions the role of the PCC as providing support and information to the on-scene management team. A chain of command and step-wise directions were developed stating the responsibilities of various PCC staff when responding to a terrorist act or a threat. Conclusion: With planning and clearly stated guidelines, PCC staff can be better prepared to handle terrorist threats and incidents and contribute to the overall mitigation of the threat.

192 DELAYED LIFE-THREATENING REACTION TO ANTHRAX VACCINE.

Swanson-Biearman B, Krenzelok EP. *Pittsburgh Poison Center, Children's Hospital of Pittsburgh, University of Pittsburgh, Pittsburgh, PA*

Background: Anthrax is an acute infectious disease caused by the spore-forming bacterium *Bacillus anthracis*. Due to the current world threat of unpredictable biological terrorism, the Department of Defense has mandated the systematic vaccination of all U.S. military personnel against this warfare agent. Many may experience a mild flu-like illness and soreness at the injection site, but systemic reactions are rare. We report a delayed and potentially serious life-threatening

adverse reaction after the administration of the third dose of anthrax vaccine. Case Report: A previously healthy 34 year old male was transported to the emergency department with dyspnea, diaphoresis, pallor and urticarial wheals on his face, arms and torso. Oxygen, hydrocortisone and diphenhydramine were administered enroute. Initial vital signs: temp 96.5°, HR 48, BP 124/68, RR 16. An audible wheeze continued and the patient was treated with bronchodilators. All symptoms resolved and the patient was discharged on prednisone and diphenhydramine. The only aberration in the patient's routine was at noon the previous day, the military had administered the third in a series of six anthrax vaccinations. He had no complaints with the first injection except mild tenderness at the injection site. At 24 hours after the second injection, the patient described feeling diaphoretic, weak and was pale. The anaphylactic-like symptoms developed approximately 20 hours after his third injection. Conclusion: Pharmaco-epidemiological data indicate that 30% of anthrax vaccine recipients experience mild local reactions. A moderate local reaction can occur if the individual has a history of anthrax infection. Severe systemic reactions occur in less than 0.2% and are characterized by flu-like symptoms. With large numbers of military personnel being vaccinated, it is imperative that we understand and recognize the full dynamic of delayed adverse reactions.

193 ORAL PROPHYLAXIS OF ORGANOPHOSPHATE POISONING IN MICE.

Meggs WJ, Freeman JM Jr. *East Carolina University, Greenville, NC*

Objective: To determine the relative efficacy of oral pretreatment with pralidoxime, diazepam, atropine sulfate, and pyridostigmine bromide in a murine model of organophosphate poisoning. Methods: Female balbc mice weighing 17 to 22 g were given either pralidoxime (100 mg/kg), diazepam (20 mg/kg), atropine sulfate (4 mg/kg), or pyridostigmine bromide (0.5 mg/kg) by oral gravage. Doses were chosen from experiments in the literature reporting effects for other uses without toxicity. One hour after gravage, mice were intoxicated with the organophosphate pesticide paraoxon (8 mg/kg) by intraperitoneal injection. Lethality was scored at 4 hours and 24 hours. Chi-square analysis was used to compare outcomes between groups. Results: 5 of 10 mice given pralidoxime survived to 4 hours, while 4 of 10 mice given atropine sulfate, 1 of 10 mice given diazepam, and 0 of 10 mice given pyridostigmine bromide survived to 4 hours. With chi-square analysis, pralidoxime was superior to both diazepam and pyridostigmine bromide, with $p = 0.05$ and $p = 0.009$, respectively. Pralidoxime was not significantly different from atropine sulfate in preventing lethality. All animals surviving to 4 hours survived to 24 hours. Conclusion: Oral pretreatment with pralidoxime was superior to diazepam and pyridostigmine bromide in preventing lethality in female balbc mice poisoned with the organophosphate paraoxon at the doses used in this experiment. Extension of this result to other doses of antidotes, other organophosphate compounds, and other species will require further investigation.

194 AFFINITY OF ORGANOPHOSPHATE INSECTICIDES TO ACETYLCHOLINESTERASE COMPARED TO PLASMA CHOLINESTERASE.

Nutenko I, Taitelman U. *Rambam Medical Center, Haifa, Israel*

Background: The level of acetylcholinesterase in erythrocytes correlates with tissue cholinesterase, and is a reliable way of measuring the severity of the exposure to organophosphates. Pseudocholinesterase is a more convenient way of checking the exposure because of the lower cost of the test and its availability. Some organophosphates have a higher affinity to acetylcholinesterase and others to pseudocholinesterase. We have measured the affinity of the two organophosphates. Dichlorvos and Paraoxon, to cholinesterases and the dynamics of the inactivation process. Methods: This study was performed *in vitro* using the blood of healthy volunteers. We performed IC_{50} measures (concentration of organophosphates in which the enzyme activity is 50% of its baseline level) for both Dichlorvos and Paraoxon. We also measured the dynamics of the process of inhibition. Results: 1. There was a higher affinity for pseudocholinesterase in both organophosphates. The affinity was higher in Paraoxon, as compared to Dichlorvos. 2. Using IC_{50} concentration, maximum inhibition of cholinesterase activity was reached within one hour for Dichlorvos, and 2 hours for Paraoxon. 3. After the inhibition of cholinesterase activity by dichlorvos, its activity gradually increased. The inhibition of cholinesterase activity in Paraoxon remained stable. Conclusions: Because the inhibition of pseudocholinesterase activity is more significant, and occurs first, the sensitivity of this method must be very high. The correlation with the level of inhibition of acelylcholinesterase activity is not permanent. Consequently, the measure of pseudocholinesterase activity is not a reliable way of estimating the severity of the intoxication.

195 INTERMEDIATE SYNDROME AFTER MALATHION INGESTION AND CONTINUOUS INFUSION OF 2-PAM.

Sudakin D, Mullins M, Horowitz BZ, Abshier V, Letzig L. *Oregon Poison Center, Portland, OR*

Background: Intermediate syndrome (IS) refers to the delayed onset of proximal and diaphragmatic muscle paralysis after the resolution of the cholinergic signs of organophosphate (OP) poisoning. Controversy exists regarding its etiology, with some investigators proposing that it results from inadequate oxime therapy. We describe a case of intentional malathion ingestion complicated by IS despite the continuous and therapeutic administration of 2-PAM. Case report: A 33-year-old female ingested an unknown quantity of malathion (50% solution) in a suicide attempt. Cholinergic signs consistent with severe OP intoxication developed and were treated within 6 hours of ingestion. The patient received a single dose of activated charcoal, and did not undergo gastric lavage. Prolonged depression of plasma and RBC cholinesterases were documented. Atropine and a continuous infusion of 2-PAM (400 mg/h) were administered IV for 5 consecutive days. Plasma 2-PAM concentrations drawn at 5, 10, and 18 hours after initiation of the 2-PAM infusion ranged from 11.6–13.7 mcg/mL (minimum therapeutic concentration 4 mcg/mL). Despite clinical improvement and favorable ventilatory weaning parameters at 48 hours post-ingestion, on day 3 the patient developed flaccid motor paralysis consistent with IS necessitating mechanical ventilation until day 12. Occasional cholinergic signs (miosis, secretions, diarrhea) were intermittently observed and treated with IV atropine. The patient fully recovered and delayed neurological sequelae did not develop. Conclusions: Intermediate syndrome can develop despite the continuous and therapeutic administration of 2-PAM. Other factors, including ongoing absorption and redistribution of active OP, may reduce the effectiveness of 2-PAM and contribute to the development of IS.

196 LESSONS FROM A UNIQUE "CARBAMATE" INCIDENT.

Jones AL, Murray VSG. *Chemical Incident Response Service, Guy's and St Thomas' NHS Trust, London, United Kingdom*

Background: The chemical incident response service (CIRS) advises on the management of chemical incidents for a population of approx. 31 million people and works closely with the NPIS (London), clinicians and the toxicology laboratory. Results: On 5th October 1998 at 1030 am CIRS was called by a police officer who said that a lorry with sacks of white powder labeled "Herculite A" had spilt its load on a main road. Using a commercially available database the substance was identified by CIRS as a carbamate insecticide. Our pre-prepared carbamate advice sheet was faxed to the police station and the officer received verbal advice to stay away from the lorry until the fire service arrived with breathing apparatus. At 1055 CIRS was contacted to say that a policeman without protective clothing had moved the bags to the side of the road and was now short of breath and covered in white powder. He was advised to attend the local ER for decontamination and when he arrived at 1115 am, the hospital staff did not know how to do this and needed detailed advice from CIRS. At 1120 am the police station phoned CIRS to say their floor was covered in white powder from the boots of two police officers and asked how they should clean it up. Later analysis of the substance involved revealed it to be Plaster of Paris. Conclusions: The police need to improve communication to aid hazard identification and staff in CIRS/ poisons centers need to be aware of the potential for mistakes in recognition and reading of labels on containers until a spilt chemical is properly identified, and appropriate protection available, no handling should take place. Training needs to be provided to ER departments. Greater awareness is needed to avoid secondary and tertiary contamination!

197 PLAYING WITH "MIRACULOUS" CHALK.

Michels JE, Marchbanks BL, Lockman PR, Shum S. *Texas Panhandle Poison Center, Northwest Texas Healthcare System, Amarillo, TX*

Background: Miraculous Insecticide Chalk and Pretty Baby Chinese Chalk are illegal pesticide products that resemble ordinary blackboard chalk. The chalk is imported illegally to the US and is used to draw a line on the floor to kill insects by contact. The EPA has indicated that samples of pesticide chalk contain deltamethrin as the active ingredient; Micromedex® also reports the active ingredients as pyrethrins. We report a case of serious toxicity from ingestion of a pesticide chalk containing an organophosphate. Case Report: A 4-year-old male was brought to a rural hospital after chewing on a chalk substance with "poison" stamped on it. While in the ED, the patient became unresponsive, had a seizure, and was intubated. Treatment included 3 g activated charcoal and 1 mg atropine IV. The patient was transferred to a tertiary care hospital. Symptoms upon arrival included diaphoresis, increased salivation, and diarrhea. He was

flaccid and hyporeflexic with occasional spontaneous respirations. VS: BP 180/110 mmHg P 180 beats/min. Non-contrast head CT was negative. Based on symptoms, atropine 3.3 mg IV and pralidoxime 600 mg IV for 2 doses were included in the patient's treatment. The patient responded favorably, extubating himself and responding to commands within 12 hours. The patient made a full recovery and was discharged on day 3. Chemical analysis of the chalk substance revealed Di-syston® 3.6% as the active ingredient. Conclusion: This patient developed significant acute cholinergic symptoms from a pesticide chalk which reportedly contains pyrethrins. Patient symptoms and toxicological analysis provided information of actual ingredients. Pesticide chalks may contain organophosphates, representing a serious toxicity hazard. Poison Center staff should consider not only available product information, but also patient symptoms when treating illegally imported products.

198 PESTICIDE CONTAMINATION OF MUSEUM ARTIFACTS RETURNED TO A NATIVE AMERICAN TRIBE THROUGH NAGPRA LEGISLATION.

Seifert SA, Boyer LV, Odegaard N, Smith DR. *University of Arizona, Tucson, AZ*

Background: There are 771 federally recognized tribes that have or will be receiving funereal, ceremonial and religious artifacts from museums through the Native American Graves Protection and Repatriation Act (NAGPRA). Such objects may number in the hundreds of thousands. Museums have routinely treated perishable artifacts with pesticides, including arsenic, mercury, polychlorinated hydrocarbons, organophosphates, carbamates, volatiles and others. This is the first report of a chemical analysis of repatriated artifacts. Methods: Three ceremonial masks, from different museums, that had been returned to a tribe in the Southwestern United States were analyzed. Masks were primarily made of leather, with attachments of grasses, cornhusks, feathers, horsehair, yarn, and paint. Masks were sampled by collection of surface materials as well as air from head-space collection. Analysis for heavy metals was by energy dispersive x-ray analysis. Analysis for organics was by pyrolysis gas chromatography-mass spectroscopy. Results: Mask 1 had highly elevated concentrations of arsenic on all surfaces. Total mask arsenic was extrapolated to be up to 2.6 g. Mask 2 had trace amounts of naphthalene. Head space analysis for naphthalene was below detection limits. Mask 3 contained moderate amounts of arsenic, primarily on the exterior surface paint, extrapolated to be up to 100 mg total mask arsenic. No other pesticides were detected. Conclusions: Many museum artifacts of Native American origin may have been treated with pesticides. Significant residue may remain on repatriated artifacts, posing a health risk during storage, handling, disposal or cultural use of these objects.

199 A PROSPECTIVE STUDY OF PYRETHROIDS EXPOSURES.

Ramón MF, Ballesteros S, Martinez-Arrieta R, Torrecilla JM. *Servicio de Informatión Toxicológica, Spanish Institute of Toxicology, Madrid, Spain*

Background: Pyrethroids are very commonly used during the warm summer months in Spain. Insecticides plug-in electric are very accessible to children and intoxications with allethrin, biollethrin, permethrin, and prallethrin are very frequent. Due to a controversy in Spain about the security of this product we designed a prospective study of patients exposed who contacted our poison center in Madrid. Methods: All human exposures, between January 1 and October 31, 1998, documented to the Spanish Poison Center were recorded. Data including type of product, sex, age, route, pathological and clinical symptoms, were recorded of 166 exposures; telephone follow-up was obtained in 68 of them in whom alergical antecedents were inquired. Results: Pediatric exposures were 90.3% (mean age 1.7 year, range 0–7 years). Routes of exposure were as follows: oral 89.7%, dermal 3%, ocular 0.6%, respiratory 0.6%, and several routes 6%. Self-referral calls were 62.6% and calls from a health care professional, 34.4%. Common symptoms were allergic dermatitis (7.2%), vomiting (6%), diarrhea (5.4%), cough (3.6%), edema and pruritus (2.4%), drowsiness and paresthesia (1.2%). Liquid preparations caused symptoms in 29.3% of patients and solid ones in 9.5%. Patients with previous allergic pathology had more symptoms (61%) than the rest (21%) specially with liquid products (86%). Unique clinical features with the latter were vomiting and cough probably due to the hydrocarbons. The majority of allergic reactions were associated with liquid compounds (14 versus 5%) specially in allergic patients (71 versus 33% with solid ones). Conclusions: Acute exposure to pyrethroids can cause several allergic effects mainly with liquid preparations and in patients with previous hypersensitivity reactions. Although outcome was favorable in 100% of cases, it seems reasonable that measures to avoid exposures of children with allergic antecedents should be implemented.

200 TOPICAL USE OF DEET INSECT REPELLENT AS A CAUSE OF SEVERE ENCEPHALOPATHY IN A HEALTHY ADULT MALE.

Hampers L, Oker E, Leikin J. *Children's Memorial Hospital, University of Illinois, Cook County Hospital/Toxikon Consortium, Chicago, IL*

Background: *N,N*-diethyl-m-toluamide (DEET) is a commonly used insect repellent. Clinical cases of toxicity after topical use are rare and have been reported most often in children. Case Report: A 27-year-old previously healthy male developed paresthesias and altered mental status after applying Off! Deep Woods Spray (DEET 20%) several times while fishing on a hot (>34°C) and humid afternoon. His symptoms progressed to auditory hallucinations and severe agitation. On presentation to the emergency department he was afebrile, tremulous, confused and combative. Following heavy sedation, he required mechanical ventilation. All standard laboratory tests, including serum electrolytes, urine and serum toxicologic screens, lumbar puncture and computed tomography were unremarkable. Over 24 hours, his mental status improved and he was weaned from the ventilator. He was discharged 3 days later with no sequelae. Analysis of a sample of the patient's serum 16 hours after presentation revealed a DEET concentration of 1.6 mcg/mL. Conclusion: Environmental heat and humidity may have increased transdermal absorption of the compound in this patient. To our knowledge, this case represents the first instance in which DEET toxicity has been confirmed in a healthy adult male who was using the product in a manner consistent with the manufacturer's instructions.

201 SUCCESSFUL ORGAN PROCUREMENT AND TRANSPLANTATION FROM A VICTIM OF MALATHION POISONING.

Dribben W, Cappiello H, Kirk M. *Indiana University School of Medicine, Indianapolis, IN*

Background: One of the major limitations to organ procurement and donation is the lack of suitable donors. As the demand for suitable organs exceeds the supply, identification of potential donors continues to evolve. Due to perceived risks of transmittable agents and insufficient understanding of toxicological fate, poisoned patients are often overlooked as organ donors. Case report: A 17-year-old white male was found having a seizure in bed by his mother. A strong odor of pesticides was noted and an empty container of malathion was found. He was transported to an outlying hospital and underwent prolonged cardiopulmonary resucitation. The patient exhibited symptoms consistent with cholinergic poisoning and received a total of 12 mg of atropine and a pralidoxime bolus of 1 gram followed by an infusion at 500 mg/h. Initial plasma cholinesterase was 1433 IU/L (normal 4100–10400) and the red blood cell cholinesterase was 13199 IU/L (normal 7500–14600). The patient developed an aspiration pneumonia and worsening neurological status. No further treatment for cholinergic toxicity was needed 5 days after admission and a cerebral blood flow scan confirmed brain death. After review of the available literature on the disposition and fate of malathion in human tissues, the decision was made to harvest the patient's organs for transplantation. His liver and kidneys were transplanted and the recipients were all doing well 6 months post transplantation. Conclusions: This case of successful transplantation after organophosphate exposure underscores the fact that poisoned patients should not be overlooked as transplant candidates. Decisions should be based on the clinical presentation and knowledge of the properties of the toxin.

202 FULMINANT HEPATIC FAILURE AFTER SUCCESSFUL LIVER TRANSPLANTATION: UNMASKING THE CULPRIT.

Wax P, Linden E. *University of Rochester Medical Center, Rochester, NY*

Background: At times liver transplantation (TXP) may be performed in cases of fulminant hepatic failure (FHF) without a clear understanding of the underlying precipitant. A case of FHF is presented in a patient who 11 weeks earlier had received liver TXP for FHF of unclear etiology. Case Report: A 42-year-old previously healthy female was transferred to a liver TXP center with FHF. She had no history of ETOH abuse, solvent exposure, or mushroom ingestion, and her only medication was thyroxine. Admission labs were AST 4754 IU/L, INR 3.9, ammonia 194 mcmol/L, and lactate 9.1 mmol/L. Although her acetaminophen (APAP) level was 21 mcg/mL, there was no history of APAP OD, and the level was thought to be from occasional APAP use and not the culprit of her FHF. Due to her precarious hemodynamic state, the patient underwent emergency liver TXP. Histology on the native liver demonstrated centrilobular necrosis involving 60% of the liver with no evidence of chronic liver disease. Hepatitis serology was negative. After a stormy postoperative course, the patient recovered and went home. Besides the usual post-TXP medications, she was started on phenytoin for tacrolimus-induced seizures. She was told that she could safely take prescription doses of APAP-containing products for pain. 11 weeks after the initial TXP, she again presented to the hospital with FHF. At this time

she had AST 3612 IU/L, INR 3.3, ammonia 195 mcmol/L, and lactate 16.9 mmol/L. Liver biopsy again showed centrilobular necrosis involving 50% of the transplanted liver without evidence of acute rejection. Immunologic and viral studies were all negative. Toxicologic consultation recommended obtaining an initial APAP level from her current admission. The APAP was 68 mcg/mL. Serial APAP levels revealed a 10-hour half-life despite FHF. The patient was started on IV NAC and recovered. Upon awakening the patient admitted to taking at least 3-4 gms of APAP per day both before and after her TXP. Conclusion: APAP use should be strongly discouraged after liver TXP in cases of apparent idiopathic FHF. The combination of APAP and phenytoin may be particularly problematic due to p450 enzyme induction.

Platform Session 4 Sunday, October 3 10:15 am–12:00 pm
General Clinical Toxicology II
Abstracts #203–#208

203 EFFECT OF COMBINED IRON DEFICIENCY AND LEAD POISONING ON *IN VIVO* EXTRACELLULAR EXCITATORY AMINO ACID CONCENTRATIONS.*

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Objective: To determine whether combined iron deficiency and lead poisoning alter the release of excitatory amino acids *in vivo* using microdialysis. Methods: 32 weanling rats were divided into four groups: 1) iron deficient (ID), 2) iron deficient & lead poisoned (IDLP), 3) iron replete & lead poisoned (IRLP) and 4) iron replete (IR). Animals receiving the iron deficient diet were fed <20 PPM iron in food. Iron replete animals received a control diet containing 200 PPM iron. Animals receiving lead were given water *ad libitum* with 50 PPM lead. Non-lead poisoned animals received deionized, distilled water. Animals were fed their respective diets for 4 weeks, anesthetized with urethane, stereotaxically implanted with microdialysis probes in the hippocampus and infused with artificial CSF (aCSF). Dialysates were collected at baseline during perfusion with normal aCSF and during isotonic high K+ (30 mM) perfusion to determine depolarization-evoked neurotransmitter levels. Amino acid levels were measured by HPLC with electrochemical detection. Results: Basal aspartate levels were different between groups (1-way ANOVA, $F = 5.3$, $p = 0.0019$). In two group comparisons, ID animals had higher aspartate levels than IR ($p = 0.017$); IDLP animals had higher aspartate levels than both IR animals ($p = 0.0002$) and IRLP animals ($p = 0.006$). Aspartate levels also differed after K+ evoked depolarization by 1-way ANOVA ($F = 3.126$, $p = 0.04$). In two group comparisons the same three groups differed. Glycine levels did not differ at baseline (1-way ANOVA, $F = 2.67$, $p = 0.067$) and post K+ ($F = 0.449$, $p = 0.72$). Glutamate levels were also not significant at baseline ($F = 0.52$, $p = 0.67$) or after K+ ($F = 0.794$, $p = 0.51$). Conclusion: Effects of iron deficiency and lead poisoning on excitatory amino acid levels appear to be independent and not synergistic. Aspartate appears to be most sensitive to the effects of lead and iron deficiency.

* Winner of the American Academy of Clinical Toxicology Research Award.

204 WORKSITE SIMULATION IN THE EVALUATION OF CHILDREN EXPOSED TO ENVIRONMENTAL/OCCUPATIONAL TOXINS—A DAYCARE CENTER AND 7 DEAD BIRDS.

Shannon M. *The Pediatric Environmental Health Center, Children's Hospital, Harvard Medical School and the MA Poison Control System, Boston, MA*

Background: The evaluation of children exposed to environmental toxins is often complicated by uncertainty of whether a significant exposure has truly occurred. Investigation of a site which has been "cleaned up" presents a significant

diagnostic challenge. In these cases, simulation of the environment at the time of exposure may provide useful data. Case Report: A daycare center with 40 children had its playground adjacent to a garage undergoing a resurfacing project. Concern about exposure to resurfacing chemicals emerged as the project was being completed. Chemicals in use included diethylene triamine, triethylene tetramine, petroleum distillates, phenol, toluene, xylene, β-chloroprene, silica and coal tar pitch volatiles. The children had no health complaints; however, parents insisted on a risk assessment for potential exposure/harm. Fear of exposure was heightened when 7 dead sparrows were found on the daycare center playground. Methods: An environmental assessment was conducted via the contractor returning to the garage and simulating the resurfacing process. Air monitoring was conducted using personal sampling pumps and collection tubes. Chemical analyses were performed using gas chromatography (modified NIOSH Method 2007). Results: During the simulation, ambient concentrations of all chemicals except respirable silica fell below 10% of occupational exposure limits. Soil analysis disclosed no contamination. Necropsy of the birds revealed avian botulism with no evidence of fatal poisoning. On this basis a conclusion of no significant harm to the children from the resurfacing project was conveyed. Conclusions: In performing risk assessments of children after possible exposure to environmental pollutants, simulation of the environment at the time of exposure is a method for obtaining valuable objective data. (Supported by a grant from the AOEC and the ATSDR)

205 ENVIRONMENTAL REMEDIATION: LAG TIME TO NORMALIZATION CHILDHOOD BLOOD LEAD.

Angle CR, Manton WI. *University of Nebraska Medical Center, Omaha, NE; University of Texas at Dallas, Richardson, TX*

Background: Epidemiologic investigations of the effect on childhood blood lead (B Pb) of environmental remediation and soil abatement typically employ follow ups of 6–12 months, consistent with the $t^1/_2$ of 15 days for B Pb. Objective: Determine the $t^1/_2$ of B Pb after remediation. Methods: Prospective, longitudinal observational study for 2–4 y of 22 children 2–3 yo. Monthly duplicate diet (F Pb), hand wipe (HW Pb) and urine (U Pb); quarterly B Pb and floordust (FD Pb), carpet dust (CD Pb), window sill (S Pb) and door mat (DM Pb). Samples collected under clean conditions and analyzed by a Finnigan MAT 261 thermal ionization-mass spectrometer (TI/MS) with ^{205}Pb spike for total Pb and isotopic ratio $^{206}Pb/^{207}Pb$. Results: 6 children had increases in Pb B from concentrations <4 μg/dL to 7–12 μg/dL related to remodeling projects: 3 were to short term exposures; 3 were to lengthy projects over many months. $^{206}Pb/^{207}Pb$ of blood and urine reflected the primary contribution of HW Pb to B Pb. HW Pb was significantly correlated with FD Pb and CD Pb. Food Pb made no detectable contribution to B Pb. The apparent $t^1/_2$ of Pb B after short term exposure was 8–11 mo; the $t^1/_2$ after prolonged exposure was 20–38 mo. The Pb B isotopic ratios reflected continued recycling of remodelling Pb from bone to blood for ≥4 y. Conclusions: Examination of the effects on childhood Pb B of interventions such as home remediation and soil abatement should be designed to compare the blood lead of the next generation of children growing up in the modified environment with that of the historical controls. Longitudinal pre- and post-abatement studies of the target population should be extended to 2–4 years post-abatement. Supported by National Institute of Health Grant #ESO4762.

206 NEUTRALIZATION OF *AGKISTRODON SAXATILIS* [*GLOYDIUS SAXATILIS*] VENOM WITH CROTAB® IN A MURINE MODEL.

McNally J, Boyer L, Hare T, Consroe P, McClure T. *Arizona Poison and Drug Information Center, University of Arizona Health Sciences Center, Tucson, AZ*

Background: Pit viper envenomation has been successfully treated in the United States using affinity purified polyvalent crotalid antivenom, ovine FAb (CroTAb®), which is formulated using venoms of North American pit vipers of genera *Crotalus* and *Agkistrodon*. In Russia and the Newly Independent States, envenomation by Asian pit vipers of the genus *Agkistrodon [Gloydius]* is a common clinical challenge, for which no antivenom is in routine use. The clinical syndrome associated with envenomation in Asia includes local and systemic signs and symptoms directly analogous with those seen with American pit viper bite; but without antivenom local physicians are limited to supportive care and to administration of human blood products for treatment of coagulopathy. We sought to demonstrate cross-reactivity of the North American antivenom with Asian venoms, in a murine model. Methods: The lethal toxicity of *Agkistrodon saxatilis*

[Gloydius saxatilis] was determined by injecting venom into the tail vein of mice. Six mice were used at each of six dose levels of the venom. The LD_{50} and 95% confidence limits (CL) were calculated by the method of Litchfield and Wilcoxon as incorporated into a computer program by Tallarida and Murray. The quantal endpoint of lethality or survival of the mice was determined at 48 hours for calculations of LD_{50}'s. Rescue with CroTAb® antivenom was performed in mice injected with twice the LD_{50}. Results: The calculated LD_{50} was 5.1 mg/kg with 95% confidence limit range of 3.4–7.5. In mice given twice the LD_{50} (10 mg/kg) we observed 100% fatality. In a second group of 6 mice given twice the LD_{50} and whole CroTAb® in a 1:15 ratio, we observed 100% survival at 48 hours. In all groups volume injected was controlled to 0.2 mL. Conclusion: CroTAb® antivenom neutralizes lethality of *Agkistrodon saxatilis [Gloydius saxatilis]* venom in a murine model.

207 PREVENTION AND TREATMENT OF RECURRENT LOCAL SYMPTOMS IN CROTALID ENVENOMATION TREATED WITH OVINE FAB.

Seifert SA, Dart RC, Boyer LV, Clark RF, Hall EL, Kitchens CS, Bogdan GM, McNally J, and the CroTAb Investigative Group. *Arizona Poison Center, Tucson, AZ; Rocky Mountain Poison Center, Denver, CO*

Objective: To illustrate the prevention and treatment of recurrence of local symptoms in patients with crotalid envenomation treated with an initially controlling dose of affinity purified, mixed monospecific crotalid antivenom, ovine Fab for injection. Methods: Patients (n = 31) were enrolled in a multi-center clinical trial. Patients received up to 12 vials of antivenom for initial control and then were randomized into one of two groups: 1) Scheduled doses of 2 vials at 6, 12 and 18 hours after initial control, with additional antivenom as needed (SCHED-AV); or 2) 2 vials as needed (PRN-AV). Local recurrence after initial control (local recurrence) was defined as increasing local symptoms requiring non-scheduled antivenom. Results: Of 31 patients, 16 were in the PRN-AV group and 15 in the SCHED-AV group. Eight of 16 patients (50%) in the PRN-AV group developed local recurrence, (mean time to first recurrence = 9.72 hours, range 3.5–23.5). Zero of 15 patients (0%) in the SCHED-AV group developed local recurrence ($p = 0.0024$, Fisher's Exact, two-tailed). Additional antivenom was effective in re-achieving control of symptoms, at least temporarily, in each instance. Six of the eight had additional recurrences after re-achieving local control. There were no local recurrences beyond 24 hours. Conclusions: Local recurrence of symptoms was seen in 50% of patients with crotalid envenomation treated with an initially controlling dose of ovine, Fab antivenom. Re-dosing of antivenom in the first 24 hours was effective in re-achieving control of local symptoms. Scheduled antivenom in the first 24 hours prevented local recurrence.

208 *IN-VITRO* EVALUATION OF THE MEIXNER TEST FOR AMATOXIN.

Lee DC, Beuhler M. *North Shore University Hospital, Manhasset, NY*

Background: Identifying a poisonous mushroom or diagnosing a potentially toxic mushroom ingestion can be very difficult. Meixner reported a simple bedside colorimetric spot test for detecting the presence of amatoxin, an organic toxin found in mushrooms such as *Amanita phalloides.* Although this test has been described in several major medical textbooks, there are little data on the reliability of this test. This study is an *in-vitro* evaluation of the specificity, sensitivity and interrater reliability of the Meixner test. Methods: Three sets of paper were embedded with five different amounts of amatoxin (0, 0.8, 1, 2, 4 mcg) in a standardized area. All specimens were prepared as per the protocol described by Meixner. The first 2 sets were shown within 10–20 minutes of preparation. The third set was allowed to sit for 1 hour after preparation before being observed. Five blinded physicians were asked to determine if specimens had a bluish discoloration (positive result). Amatoxin was purchased from Boehringer-Manheim Chemical Company (Indianapolis, IN). Results: All testers were able to observe positives on all 2 mcg or greater specimens. Sensitivity increased at the 1 hour mark whereupon all testers verified positives on all 0.8 mcg specimens. Interrater reliability had an average kappa value of .54 (with a range of .33 to 1). Conclusions: Although Meixner noted a cut-off value of 10 mcg per specimen, this study noted a much lower threshold for detecting amatoxin with good interrater reliability. *In-vitro*, the Meixner test appeared to be reliable for noting the presence of amatoxin.

Platform Session 5 — Sunday, October 3 — 1:00 pm–3:00 pm

Acute Poisonings
Abstracts #209–#215

209 FORMATE KINETICS IN METHANOL POISONING.

Kerns W, Tomaszewski C, McMartin K, Ford M, Brent J, META Study Group. *Carolinas Medical Center, Charlotte, NC; Louisiana State University, Shreveport, LA; and University of Colorado Health Sciences Center, Denver, CO*

Objective: We sought to describe the kinetics, dialysis clearance, & clinical markers of formate (FA), the toxic metabolite of methanol (MeOH). Methods: Data were obtained from a prospective, multicenter treatment protocol using fomepizole ± hemodialysis (HD) for MeOH poisoning. HD indications included: MeOH >50 mg/dl, pH <7.1, refractory acidosis, or altered visual acuity. Blood samples were drawn for serial FA, MeOH, pH, & bicarbonate measurements. Plasma MeOH & FA were determined by gas chromatography. Elimination half-life ($t_{1/2}$) was calculated as $t_{1/2} = 0.693/Kd$, where Kd, the elimination rate constant, was determined from the slope of ln[FA] vs time. HD clearance was calculated using the relationship HD flow × [(inlet FA − outlet FA)/inlet FA]. Pearson correlation coefficients were determined for initial FA vs initial pH, serum bicarbonate, & anion gap (AG). Data were analyzed using SAS statistical software ($p < 0.05$ significant). Results: 11 patients (10 males) with mean age 39.9 years (range 19–61) were included. 7 patients underwent HD. The mean initial MeOH was 196.3 mg/dL (range 28–768) & FA, 11.0 mmol/L (range 0–34.8). Non-HD $t_{1/2}$ was 205 min ± 25 sd and HD $t_{1/2}$, 185 min ± 62.7 sd (NS by Student's t-test). Overall HD FA clearance was 223 mL/min ± 24.5 sd. Correlation coefficients were: pH vs FA $r^2 = 0.929$ ($p < 0.05$); bicarbonate vs FA $r^2 = 0.804$ ($p < 0.05$); & AG vs FA $r^2 = 0.760$ ($p < 0.05$). Conclusions: Even though HD clears FA, HD does not decrease the FA elimination $t_{1/2}$. Low pH, low bicarbonate, and elevated AG correlate with FA presence.

210 ACCURACY AND RELIABILITY OF URINE FLUORESCENCE BY WOOD'S LAMP EXAMINATION FOR ANTIFREEZE INGESTION.

Wallace K, Suchard J, Curry S, Reagan C. *Department of Medical Toxicology, Samaritan Research Institute, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: Bedside Wood's lamp examination for urinary fluorescence has been advocated for detecting ingestion of ethylene glycol (EG)-containing automotive antifreeze. A prior study may have overestimated accuracy of such testing based on study design. We tested the hypothesis that simultaneous comparison of control and test urine specimens enhances test accuracy and reliability. Methods: Healthy adult men taking no medications provided urine specimens prior to and at 1–2 and 4–6 hours after ingesting 0.6 mg sodium fluorescein, an amount consistent with a toxic ingestion of EG-containing antifreeze. Blinded ED physician observers characterized randomly ordered specimens as fluorescent or not by Wood's lamp exam, according to two methods of specimen presentation: sequential (i.e. one at a time) versus simultaneous (i.e. all test samples and controls together). Results: 28 subjects and 6 observers (in 3 pairs) were enrolled. Sequential presentation had 34.8% sensitivity, 75.0% specificity, and 48.2% accuracy for detection of urinary fluorescence; simultaneous presentation had 42.7% sensitivity, 66.7% specificity, and 50.9% accuracy. Conditional logistic regression analysis revealed a slight, but statistically significant ($p = 0.043$) improvement in interrater reliability with simultaneous presentation of samples compared to sequential presentation. There was no significant difference in accuracy ($p = 0.517$) between the two methods. Conclusion: Under optimal experimental conditions, Wood's lamp determination of urine fluorescence is not accurate for detecting sodium fluorescein ingestion in an amount associated with a toxic ingestion of EG-containing antifreeze.

211 A MODEL TO PREDICT ETHYLENE GLYCOL ELIMINATION DURING FOMEPIZOLE MONOTHERAPY.

Sivilotti MLA, Burns MJ, McMartin KE, Brent J. *University of Massachusetts, Worcester, MA; Harvard University, Boston, MA; Louisiana State University, Shreveport, LA; University of Colorado, Denver, CO*

Objective: With the availability of the potent ADH inhibitor fomepizole, the absolute blood ethylene glycol (EG) concentration may no longer be a primary indication for hemodialysis. The decision to institute hemodialysis may largely depend on the predicted time course of EG elimination in the non-acidemic patient treated with fomepizole. We sought

to develop such a predictive model based on parameters readily available at presentation, since single or serial EG concentrations are not immediately available at many centers. Methods: Using data from the META trial, we analyzed a single compartment pharmacokinetic model during fomepizole monotherapy, defined to be any interval during which fomepizole concentrations were above 10 μM, ethanol below 10 mg/dL, and hemodialysis was not in progress. Thus, the only available elimination routes for EG were renal excretion and non-ADH metabolism and elimination. The rate of renal excretion was postulated to vary as a function of creatinine clearance (Cl_{creat}), which was calculated from patient age, sex, and serial, non-steady state serum creatinine concentrations. Results: Data were drawn from 19 EG-poisoned patients with concentrations ranging as high as 210 mg/dL. A roughly linear relationship between EG elimination rate and Cl_{creat} was identified ($r^2 = 0.53$), with the first order elimination constant $k_{elim} = 0.375kg/L \times Cl_{creat} + 0.0204h^{-1}$. The y-intercept of $0.0204 \pm 0.0070h^{-1}$ represents the non-ADH mediated, non-renal elimination, while the slope multiplied by the V_d yields a fractional excretion of EG by the kidneys of 25.5 ± 9.4%. Conclusions: Initial creatinine clearance can be used to predict the rate of EG elimination. This model can help establish whether hemodialysis will be needed to expedite EG removal in patients presenting before the accumulation of toxic acid metabolites. The model can also serve as an element in the cost-benefit analysis of extracorporeal removal for the EG poisoned patient treated with fomepizole.

212 SERUM AND URINE CONCENTRATIONS OF TRICYCLIC ANTIDEPRESSANTS (TCA) FOLLOWING ADMINISTRATION OF TCA IMMUNE FAB (OVINE).

Heard K, O'Malley G, Bogdan GM, Dart RC, Burkhart KK, Donovan JW, Brown BL, Ward SB, Porter RS. *Rocky Mountain Poison & Drug Center, Denver Health, Denver, CO; Central Pennsylvania Poison Center, Hershey, PA; Therapeutic Antibodies, Inc., Nashville, TN*

Background: TriTAb® TCA immune Fab (ovine) is effective in neutralizing and redistributing TCA in a mouse poisoning model. We report the serum and urine concentrations of free and total TCA for 4 patients treated with Fab for TCA intoxication. Methods: Patients with TCA overdose and a QRS interval of >100 msec were treated within a forced titration dose finding study. Patients 1–3 received 7 g (as 1, 2 and 4 g infusions), while patient 4 received 14 g (as 2, 4 and 8 g infusions). Serum samples were collected at baseline, after each Fab infusion and 0, 0.5, 1, 2, 4, 8, 12, and 24 hours following final Fab infusion. Urine samples were collected at baseline and 0, 4, 12, and 24 hours following final Fab infusion. Samples were analyzed for free and total levels of each TCA using HPLC. Results: Total and free drug levels (parent + active metabolite) for serum and urine obtained at baseline and after final Fab infusion (0 hr) are shown. Conclusions: 1) TCA immune Fab effectively binds TCA in overdose patients. 2) Following Fab administration, total TCA serum and urine levels increase while free TCA levels show inconsistent and inconclusive reductions. 3) This suggests that Fab administration redistributes TCA from tissue into serum.

	Serum TCA Concentrations (ng/ml)		Urine TCA Concentrations (ng/ml)	
	Total (Free)		Total (Free)	
Patient No.–TCA	Baseline	0 Hr	Baseline	0 Hr
1–Imipramine	450 (ND)	4,500 (ND)	Not obtained	10,100 (112)
2–Amitriptyline	510 (20)	3,100 (15)	11,800 (10,800)	49,000 (9,100)
3–Nortriptyline	1,600 (130)	5,300 (120)	36,000 (34,000)	250,000 (47,000)
4–Amitriptyline	1,330 (59)	7,200 (27)	6,600 (4,700)	290,000 (11,200)

213 THIORIDAZINE-INDUCED CARDIAC DYSFUNCTION AND NECROSIS.

Wang R, Derevianko A, Raymond R. *Department of Emergency Medicine, Brown University School of Medicine, Providence, RI*

Objective: To evaluate the direct effects of thioridazine (TDZ) on cardiac function and tissue necrosis. Methods: Isolated rat hearts were perfused with Krebs-Henseleit-Bicarbonate (KHB) at a constant coronary flow of 10 mL/min and paced at 300 bpm for 60 min. Left ventricular (LV) pressures were measured with a balloon-tipped catheter placed in the LV via the mitral valve. LV generated pressure (LVGP) was used as an index of cardiac function and was calculated

by subtracting LV end diastolic pressure (LVEDP) from LV peak systolic pressure (LVPSP). Coronary perfusion pressure (CPP) served as an index of coronary resistance. The experimental trial lasted 60 min and included: 15 min of basal, 30 min of treatment, and 15 min of KHB for recovery. Treatments included: KHB (n = 4), and TDZ at 8 mcg/mL (n = 3), 16 mcg/mL (n = 4), 24 mcg/mL (n = 4). Creatine phosphokinase (CPK) was measured in heart effluent at basal, treatment (1, 2, 5, 15, 20, 30 min) and upon recovery. Reported values represent the mean of the percentage of control. Results: 1) TDZ decreased LVGP, increased LVEDP, and increased CPP in a dose dependent manner. 2) CPK at 5 min of treatment were 187% (TDZ 24 mcg/mL), and 137% (TDZ 16 mcg/mL) of KHB control. At 30 min of treatment with TDZ at 8 mcg/mL, CPK was 162% of KHB control. 5) Hearts did not return to basal function upon return to KHB after treatment with TDZ at 16 and 24 mcg/mL. Conclusions: 1) TDZ directly decreases left ventricular contractile response and increases coronary vascular resistance. 2) TDZ causes irreversible cardiac dysfunction that is associated with early CPK release.

214 HOMOCYSTEINE LEVELS IN COCAINE ABUSERS.

Williams RH, Maggiore JA, Shah SM, Negrusz A, Brown N, Johnson V, Erickson TB. *Department of Emergency Medicine, Department of Pathology and the Toxikon Consortium, Chicago, IL*

Background: Cocaine abusers have an increased risk for cardiovascular and CNS complications. Homocysteine has been recognized as a potential risk factor for atherosclerosis and cardiac and cerebrovascular disease. Measurement of free homocysteine (fHcy) along with total homocysteine (tHcy) may prove useful in predicting risk factors for morbidity in cocaine use. Methods: Plasma fHcy and tHcy levels were analyzed using an HPLC method in patients (n = 29) presenting to an urban Emergency Department who quantitatively screened positive (COC+) for cocaine metabolite, benzoylecgonine (BE). These patients were compared to healthy subjects (n = 34) who screened negative (COC−) using fluorescence polarization immunoassay. Also analyzed were plasma and urine for cocaine, norcocaine, BE, and ecgonine methyl ester using GC-MS. Plasma levels for fHcy and tHcy from cocaine abused patients and normal subjects were statistically evaluated using one way analysis of variance (ANOVA). Medical records from 27 of the 29 COC+ were retrospectively reviewed for clinical signs and symptoms consistent with acute cocaine toxicity. Results: COC+ had significantly higher levels of plasma fHcy and tHcy ($p < 0.001$) compared to COC−. The mean and 95% confidence intervals for fHcy and fHcy in μmol/L was 3.5 ± 0.7 and 9.3 ± 1.1 for COC+ and 1.9 ± 0.2 and 7.1 ± 1.3 for COC−, respectively. Also noted was a direct relationship between plasma BE and tHcy levels ($p < 0.01$). Retrospective review of patient records testing positive for cocaine demonstrated no consistent clinical correlation with levels of fHcy and tHcy. Conclusion: Measuring fHcy and tHcy may be valuable in the assessment of patients who abuse cocaine. However, no clinical correlation was noted in those who had elevated levels. A prospective trial noting other confounding variables including chronicity and frequency of cocaine use is indicated.

215 USE OF MULTIPLE LOGISTIC REGRESSION ANALYSIS OF PLASMA PARAQUAT CONCENTRATIONS TO PREDICT OUTCOME.

Jones AL, Dargan PI, Flanagan RJ. *National Poisons Information Service (London), Guy's Hospital, United Kingdom*

Background: It is important to be able to predict who may survive from paraquat poisoning, so that inappropriately aggressive techniques such as hemodialysis are not employed in those who have little hope of survival and minimally poisoned patients can be protected from unnecessarily aggressive treatment. Methods: Case records of patients admitted to the poisons ward over the last 5 years and all the literature on paraquat poisoning were examined. Data were recorded from all patients where outcome and timed plasma paraquat concentrations were present. Results: 375 cases (113 M: 62 F: 200 unknown) of mean age 38.3 years (range 1–87 years) were identified. 49 patients had evidence of renal toxicity and 41 received hemodialysis or charcoal hemoperfusion. 61 cases developed pulmonary sequelae and 44 had lesions in the upper gastrointestinal tract. The median time from ingestion to death in the 241 deaths reported was 270 hours (minimum 3 h and maximum 720 h). The logarithm of the plasma paraquat concentration was plotted against the logarithm of time since ingestion. The predicted probability of survival for any specified time and concentration = exp(logit)/[1 + exp(logit)] where logit = 0.58 − 2.33 × log (plasma conc of paraquat) − 1.15 × log (no. of hours since ingestion). Conclusions: We report a new equation which may be helpful in predicting who will survive after ingestion of paraquat up to at least 200 hours after ingestion. This can now be used as a research tool for studies on efficacy of treatment of paraquat poisoning.

Platform Session 6 — Sunday, October 3 — 3:15 pm–5:15 pm

Poison Center Issues
Abstracts #216–#220

216 CLONIDINE: A PEDIATRIC CASE SERIES. CAN DOUBLE DOSES IN PEDIATRIC PATIENTS BE SAFELY MANAGED AT HOME?

Matthews L, Courtemanche L. *New Hampshire Poison Information Center, Lebanon, NH*

Background: Clonidine is an imidazoline derivative hypotensive agent which is gaining wide acceptance as an adjunct to psychostimulants such as methylphenidate in the treatment of behavioral disorders in children. As little as 0.1 mg has produced toxic symptoms (sx) in children. Resources state that all children ingesting clonidine should be referred to a health care facility (HCF) for management; however, some children are on a maintenance dose of 0.1 mg therapeutically. These children are likely to be more tolerant to toxic effects. We examined the hypothesis that children who are maintained on clonidine therapeutically can tolerate a double dose. Methods: A prospective study was designed to confirm this hypothesis. We collected data on all pediatric clonidine tablet exposures reported to us over a 24 month period. Cases were categorized as: A) ≤2 times maintenance dose; B) >2 times maintenance dose; and C) acute ingestions by children not on clonidine. Results: Out of 41 total cases, A) 8 (dose range 0.2–0.4 mg) fit this category and none developed sx. B) Of 8 (dose range 0.075–0.5 mg), 2 were asymptomatic, 4 had minor sx (drowsiness, hyperactivity) and 2 had moderate sx (marked lethargy, hypotension). C) Of 25 cases (dose range 0.0125–2.6 mg), 4 were asymptomatic; 14 had drowsiness, dizziness, slurred speech, and/or ataxia; 7 had moderate to major sx including coma, hypotension, bradycardia, and/or respiratory depression. Conclusion: None of the 8 children who were on clonidine therapeutically and who received no more than a double therapeutic dose developed sx. In the remaining 33 cases, 27 (82%) developed sx. Children who are therapeutically on clonidine and receive no more than double their therapeutic dose can be safely managed at home with close follow-up by a poison center; all other pediatric exposures should be referred to a HCF.

217 THE IMPACT OF TELEPHONE ACCESS ON POISON CENTER "PENETRANCE".

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

Background: Regional PC certification criteria include "penetrance" (calls per 1000 population) as evidence of a PC's accessibility. PC services are inherently linked to a telephone delivery system, yet telephone availability is not figured into this formula. This study was conducted to determine the role that household telephone availability (HT) may play in determining PC penetration rates. Method: Data from the Federal Communications Commission's (FCC) 1999 report *Trends in Telephone Service* on telephone subscribership was compared to 1998 census population figures, 1998 state PC human exposure and info call data, and traditionally calculated penetrance. Results: Inclusion criteria were PC services provided by only 1–2 PCs which were in-state, and provided services to the whole state. PC data was collected for 28 states of which 20 were served by certified regional PCs. State population ranged from 591,000–12,045,000; PC call volumes from 5,114–92,745 human exposures, and 1,677–60,526 info calls. Penetrance as measured by human exposures per 1000 population ranged from 2.98–16.89. HT ranged from 88–97%. The FCC study found white households had HT rates of 95.2%, blacks =87.7%, and Hispanics =88.9%. Younger households (<25 yrs) had HT rates of 87.3%; elderly households (>70 yrs) =96.3%. Households with annual earnings <$5,000 had HT rates of 77%; those >$75,000 had rates of 99%. Human exposures/1000 HT ranged from 5.18 to 53.65. There was no correlation between PC calls/human exposures per 1000 population and the percentage of HT. Conclusion: HT data may be useful on a smaller (county) scale. Statewide data, however, does not support a link between telephone accessibility in the home and utilization of state wide PC services.

218 POISON CENTER UTILIZATION PATTERNS OF PEDIATRICIANS.

Goto CS, Hodge D III, Wiebe RA. *University of Texas Southwestern Medical Center, Dallas, TX; Washington University, St. Louis, MO*

Objective: To determine the Poison Center (PC) utilization patterns of pediatricians for toxic and nontoxic exposures. Methods: Attendees of an interactive seminar at the American Academy of Pediatrics 1999 Spring Session were sur-

veyed. They were presented with 14 case scenarios of pediatric patients with common toxic and nontoxic exposures, of which 8 cases could be safely managed at home and 6 cases required emergency department (ED) evaluation. After each case was presented, attendees were asked to choose from the following management responses: (1) Don't worry, Junior will be fine; (2) I'll call you back. I need to check with the PC; (3) Call the PC and follow their instructions; (4) Give ipecac. Let me know if Junior does not vomit; (5) Give ipecac and come right to the office; (6) Come right to the office; (7) Give ipecac and go right to the ED; (8) Go right to the ED; (9) Call 911. Results: Attendees reported that they called a PC daily to monthly (30%), once every 2–6 months (47%), once per year (8%) or almost never (16%). There were 629 responses to the 8 cases which could be safely managed at home. The PC was utilized in 175 (28%). The ED was unnecessarily utilized in 241 (38%), and "911" in 35 (6%). There were 501 responses to the 6 cases which required ED evaluation. The PC was utilized in 236 (47%). Home management was incorrectly chosen in 53 (11%). Conclusions: (1) The PC is significantly utilized by pediatricians. (2) When the PC is not utilized by pediatricians, discrepancies in management occur which may adversely affect patient care or result in unnecessary utilization of health care resources.

219 PIPs vs SPIs: RESULTS OF A NATURAL EXPERIMENT.

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

Background: There currently is debate among providers of poison center services whether Poison Information Providers (PIPs) can provide the same level of care as Specialists in Poison Information (SPIs). To date, little evidence has been generated to address this question. Funding changes at this institution provided an opportunity to compare outcomes between PIPs and SPIs. PIPs had been used until 1996. After 1996, SPIs were used exclusively. This study was done to evaluate differences in outcomes between cases managed using PIPs and those using SPIs. Methods: Data were collected from annual reports for 1993, 1994, 1997, and 1998. The data from years 1995 and 1996 were excluded because of variances in staffing while PIPs were being phased out. Variables assessed included % cases with ipecac use coded, % of cases managed on site, % of cases managed in a HCF, and outcomes of either no effect; minor effect; not followed, no effect expected; not followed, minimal toxicity expected. Results were analyzed using ANOVA. Results: Despite an increase in the total number of cases managed each year, the % of cases managed using ipecac and the % of cases managed at a HCF decreased significantly ($p < 0.01$). % of cases coded with either no effect; minor effect; not followed no effect expected; not followed, minimal clinical effects increased significantly ($p < 0.01$). The difference in the percentage of patients managed at home from 1993 to 1998 was 7.29%. Assuming an average of $500 saved for each case managed at home instead of an emergency department, use of specialists in 1998 resulted in overall savings of $1,400,000. Conclusions: There are significant differences in poisoning patient outcomes between PIPs and SPIs. The differences in outcomes offset the cost of providing higher level personnel.

220 THE USE OF FORMATIVE RESEARCH TO MARKET POISON CENTER SERVICES.

Cucchi P, Grubbs T, Deibler K, Williams P. *Georgia Poison Center, Atlanta, GA*

Background: Effective marketing campaigns to increase awareness of poison center services, are based on an understanding of the knowledge, attitudes and behaviors of the target audience. Formative research tools, including focus groups and surveys, can be used to gather information about a target audience. A regional poison center (RPC) conducted formative research to assess awareness and perceived importance of its services and to identify unmet needs in a selected target audience. Methods: The RPC conducted a focus group and self-administered mail survey with pediatricians. During the focus group, a moderator asked the 3 participating pediatricians open-ended questions about the RPC and its services. The qualitative results of the session were interpreted from the session's transcript. 330 randomly selected pediatricians were mailed the 16-item multiple choice and open- ended question survey. 81 (24.5%) pediatricians returned the survey. Descriptive statistics were used to analyze the surveys. Results: More than half of the focus group and survey participants were aware of the RPC's 24-hour poison emergency telephone service. However, less than half of the participants knew you could call the RPC for food poisonings, insect/snake bites, animal bites, animal poisonings and prevention services. The services identified as most important were: treatment advice for human poisonings, insect bites, and animal bites; and public education services. Conclusion: The results of the formative research provided insight into the level of awareness and perceived importance of the RPC's services among pediatricians in the state. The results of the research are being used to design a campaign to market poison center services to pediatricians.

Saturday, October 2 6:00 pm–7:30 pm
Toxicology History Society
Abstracts #221–#223

221 COITUS RIGOR MORTIS OR POISONOUS PASSION.

Paloucek FP, Leikin JB, Hoppe J. *University of Illinois and the Toxikon Consortium, Chicago, IL*

Avicenna (''*Prince of Physicians* 980–1037) describes in his 20 volume encyclopedia a ''Poison Maiden.'' This was ''A young girl fed poisons (in increasing doses) so she became very venomous. A kiss or sexual intercourse would prove fatal to her lover.'' The girl was reportedly so venomous, she killed biting insects. Poison kisses have been prominently described in causing cyanide-associated deaths in 1910 and 1923. In India, the Queen of Ganore killed the Rajah Bukht by impregnating his marriage robes with poison. Described in the Indian Manual of Medical Jurisprudence, this lead to copycat killings of other historical figures. The concept of a poisonous bed was a prominent feature of the 1908 Boston murder trial of Mary Kelliher. In the early 18th century, a Frenchman was hung following the murder of a series of young beautiful wives. He wished the experience to be sweet so he ''pleasured'' them with lethal arsenic doses on his goatskin condoms. Modern medical literature yields 20 articles describing 33 cases of sexually transmitted poisonings. Only three drugs/toxins have been described more than once (Ciguatoxin, Penicillin/s, and Nitroglycerin). Searching for cases is very difficult due to the varied terms. No standard definition exists although commonly used terms included paramour, connubial, consort, or conjugal poisoning. For simplicity, Sexually Transmitted Poisoning (STP) is proposed. ''Love potions,'' aphrodisiacs or sensory enhancers, disinhibitors (Date Rape drugs), contraceptives, and reproductive poisons were excluded. Sex, though described historically as a method of poisoning, is not currently considered a site, route or cause of poisoning. ''Those who cannot remember the past are condemned to repeat it,'' Santayana.

222 WICCA OR MYCOTOXIN? THE SALEM WITCH TRIALS.

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The autumn of 1691 was not a good harvest year for the New England Puritans. A wet, warm planting season was followed by a hot, stormy summer. Thomas Putnam, a well-regarded Salem farmer whose swampy land supplied much of the colony's rye flour, donated grain regularly to the Reverend Samuel Parris's household. By the October after the harvest, 11-year-old Abigail Williams, the Reverend's niece, was spending time with their two Caribbean servants, Tituba and John Indian, who told hair-raising, yet seductive voodoo stories to Abigail and three other 9–17-year-old girls. The girls were soon talking magic and baking 'witch pies' made out of dog urine mixed with ergoty rye. Then they started writhing in pain, insensate with convulsive twitching, occasionally accusing witches in their midst of tormenting them. Court-approval spectral evidence provided by the girls, visions of wicca-practicing townsfolk, defined the 'proof' of such preternatural mischief. The march to the gallows on witches' hill began the next spring and did not end until September 1692, with 20 'witches' convicted, sentenced, and executed. The colony had sown distrust, jealousy, superstition, and moldy grain; it reaped death and despair.

Some historians have postulated that the girls responsible for the travesty suffered from ergotism. The *Claviceps purpurea* fungus thrives in damp, cool conditions and improper storage, growing on rye, corn, and other grains. Its alkaloids are potent mycotoxins; isoergine (lysergic acid amide) is similar to LSD. Gangrenous ergot poisoning is characterized by vasoconstriction and gangrenous injury to the extremities. Convulsive ergotism is associated with vertigo, headaches, painful muscular contractions, mania, delirium, and visual and auditory hallucinations. Skeptics of this toxicological explanation abound. Yet we are left with the question of Salem: was it wicca? hysteria? religious insularism? The X-files? or just bad sandwiches made with molding bread!

223 THE PHILADELPHIA ARSENIC GANG: MURDER AND MAYHEM IN THE CITY OF BROTHERLY LOVE.

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Background: In 1939, a series of articles appeared in the *Philadelphia Inquirer* concerning a crime syndicate responsible for the poisoning murders of over 100 individuals in the tri-state Pennsylvania, New Jersey, New York area. An extensive

police investigation of these murders uncovered a bizarre tale of conspiracy, murder, insurance fraud, witchcraft and satanism based in Philadelphia's Italian community. This murder syndicate was unofficially dubbed "the arsenic murder ring." Methods: The archives of various Philadelphia newspapers housed at Temple University were searched and original newspaper articles of the day and original photographs were assembled and reviewed. These accounts were pieced together to re-create the story of the Philadelphia Arsenic Gang. Results: Beginning in the late 1920s, until formal prosecutions commenced in 1938, certain prominent individuals in Philadelphia's Italian community posed as magical faith healers and as such, preyed upon many vulnerable and superstitious Italian immigrants. This crime syndicate acted primarily by persuading these immigrants to buy life insurance policies for spouses or other family members, with syndicate members as named beneficiaries. Eventually, the syndicate would arrange the "accidental" death of the insured by providing arsenic to the family member with instructions to administer to the unsuspecting insured. Conclusion: The escapades of the Philadelphia Arsenic Gang not only documents an interesting historical use of arsenic as a homicidal agent but it also provides a fascinating insight into the sociology and criminology of Philadelphia in the 1920s and 1930s.