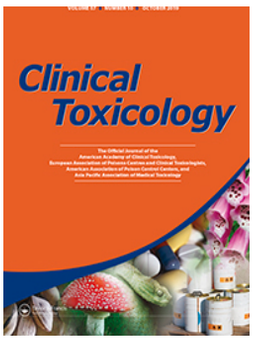

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North American Congress of Clinical Toxicology (NACCT) Abstracts 2019

1. Assessment of microRNA-122 for Drug-Induced Liver Injury in Patients with Muscle Injury

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Background: Drug-induced liver injury (DILI) caused by medications or recreational drugs is a leading cause of morbidity and mortality. Aminotransferases are currently considered the gold standard biomarkers for the diagnosis of liver injury regardless of the etiology. However, due to their lack of specificity, elevations of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) can also be observed in other clinical conditions, such as rhabdomyolysis. Therefore, developing more liver-specific biomarkers for DILI is more clinically meaningful, especially for drug-abuse patients who have high risk to develop rhabdomyolysis and/or DILI. MicroRNA-122 (miR-122) is predominantly (~70% of total liver miRNA content) and specifically expressed in the liver, making it an ideal DILI biomarker candidate. We investigated the diagnostic value of miR-122 for DILI in patients with rhabdomyolysis.
Subject Recruitment: 22 patients (18 males, 21–73 year-old; 4 females, 39–80 year-old) with creatine kinase (CK) > 5 x upper limit of normal (ULN) due to rhabdomyolysis were recruited at San Francisco General Hospital. 13 out of 22 patients had a positive urine screen for abused drugs, including amphetamine, cocaine, opiates etc., 1 took atorvastatin, and the other 8 had muscle injury caused by trauma, surgery, seizures, or excessive exertion etc. Serum samples (69 in total) were collected at 2–6 different time points for each patient. Serum samples from 22 healthy subjects and 6 patients with liver injury were included as reference samples and positive controls respectively and tested at the same time.
Methods: RNA was purified with TRIzol LS from 350 ml serum and followed by reverse transcription; and right before RNA purification, cel-miR-39 (5 fmol, 5 ml) was sparked into each serum sample as the internal control. The miR-122 level was estimated with quantitative real-time PCR (Taqman MicroRNA assay, miR-122-5p) and calculated relatively to cel-miR-39 at Arbitrary Unit (A.U.).
Results: In the rhabdomyolysis patient samples, AST and ALT closely correlated with the increase of CK (Figure 1A–C). However, miR-122 levels in 71% of the serum samples (49 out of 69) were within the reference range (Figure 1D), and only 16% (11 out of 69) of the serum samples from 6 out of 22 patients had miR-122 level greater than the ULN of 3.00 A.U. (calculated as Mean +2 x SD of reference samples). In serum samples with liver injury, miR-122 significantly increased (p
Conclusion: Serum miR-122 is largely not increased in the presence of muscle injury only, indicating it is more liver specific than the aminotransferases. We found few patients with rhabdomyolysis who also had elevated miR-122, suggesting concomitant liver and muscle injury.

KEYWORDS Drug-induced liver injury, micro-RNA, muscle injury

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2. Efficacy of oral administration of sodium thiosulfate and glycine in a large, swine model of oral cyanide toxicity

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Background: Cyanide is a deadly poison, particularly with oral exposure where larger doses can occur before any symptoms develop. Multiple governmental agencies highlight oral cyanide as an agent that can be used in a terrorist attack. Cyanide remains a major threat to civilian and military populations worldwide as it can be easily weaponized and is readily available. Currently there are no FDA approved antidotes specifically for oral cyanide. Current therapies for cyanide are not ideal for use in a mass casualty situation. An oral countermeasure that can neutralize and prevent absorption of cyanide from the GI tract after oral exposure is needed.
Materials and Methods: We conducted a randomized controlled trial comparing oral sodium thiosulfate and glycine to no treatment control animals following oral cyanide exposure using a swine model (female Yorkshire swine weighing 45-55kg). All experiments were approved by the University of Colorado's Institutional Animal Care and Use Committee (IACUC). Animals were instrumented, sedated and stabilized. Potassium cyanide (8mg/kg of KCN) (Sigma Aldrich, St. Louis, MO) was diluted in saline and delivered as a one-time bolus via the orogastric tube. Animals in the treatment group received sodium thiosulfate (508.2mg/kg, 3.25M solution) and glycine (30mg/kg using 3.5M solution) (Sigma Aldrich, St. Louis, MO) via orogastric tube after cyanide exposure. Survival at 60 minutes was the primary outcome and was analyzed by comparing survival between groups by log-rank, Mantel-Cox analysis. Hemodynamic variables and laboratory studies were trended and analyzed post hoc.
Results: At baseline and time of treatment all animals were similar. Survival at 60 was 100% in treated animals compared to 0% in the control group ($p = 0.0027$). By the study end, there was a significant difference in the lactate concentration between treatment and control group (9.43 + 4.08 [Control]; 1.66 + 0.82 [Treatment]; 7.69 + 2.07 [Difference between means]; -14.05,-1.32 [95% CI difference]). Mean arterial pressure was significantly different between treatment and control group (26 + 6.7 [Control]; 81 + 14 [Treatment]; 55.2 + 7.1 [Difference between means]; 37.8, 72.6 [95% CI difference]). pH and oxygen saturation were also significantly different between treatment and control group.
Conclusion: The combination of oral sodium thiosulfate and glycine significantly improved survival and clinical outcomes in a large animal model of oral cyanide toxicity.

KEYWORDS Cyanide, antidote, countermeasure

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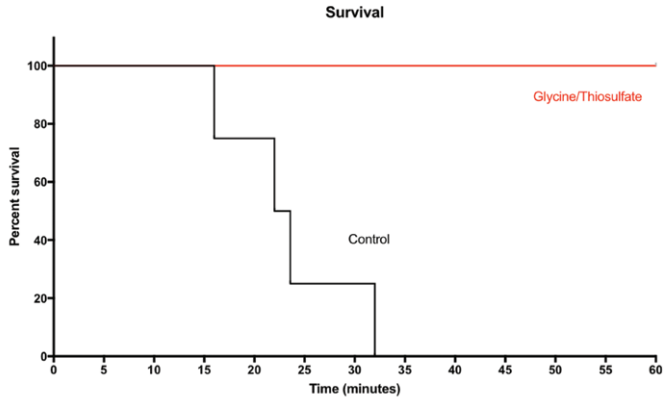
This article was originally published with errors. Please see Correction (http://dx.doi.org/ 10.1080/15563650.2019.1662112)



Median observation time in control group: 22.7 minutes (IQR 38.8)
Median observation time in treatment group: 60 minutes (IQR 0), all treatment animals survived to 60 minutes

3. Accuracy of patient exposure history compared with analytical findings for those reporting use of synthetic cannabinoid receptor agonists

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Objective: The United Kingdom (UK) Identification Of Novel psychoActive substances (IONA) study is linking clinical features with analytical findings in patients (≥16y) presenting with severe toxicity (according to specific definitions) associated with suspected novel psychoactive substance (NPS) use. The current research has compared patient histories of exposure to synthetic cannabinoid receptor agonists (SCRA) with analytical findings from patient samples to assess the accuracy of the patient-provided history of exposure.
Methods: With ethical approval, patients presenting to participating hospitals across the United Kingdom have been recruited with informed consent or with the agreement of an appropriate relative/representative (with subsequent confirmation/refusal of their own consent on recovery). Patient history of exposure, clinical features, treatments used and outcomes are recorded using a structured data collection sheet. Blood and urine samples are collected and analysed for novel and conventional drugs of misuse by liquid chromatography-tandem mass spectrometry. The current analysis was restricted to patients reporting use of a single novel or conventional illicit drug only and excluded patients where no history was obtained or drug use was otherwise unknown
Results: Clinical and analytical data were available for 206 study participants reporting use of a single illicit drug recruited between March 2015 and January 2019. Their median age was 33 years (range 16-72 years) and 175 (85%) were male. Of 114 patients who reported using a SCRA or SCRA-containing product (including 'spice'), a SCRA was detected in one or more samples from 75 ('true positives') and not detected in 39 ('false positives'). For 92 patients reporting use of other drugs (but not SCRAs), a SCRA was detected in one or more samples in 9 ('false negatives') and not detected in 83 ('true negatives'). As an indicator of SCRA exposure, the patient history of SCRA use had a sensitivity of 89.3% (95% confidence intervals 80.6 to 95.0%), specificity of 68.0% (95% CI 59.0 to 76.2%), positive predictive value (PPV) of 65.8% (95% CI 59.0 to 71.6%) and negative predictive value (NPV) of 90.2% (95% CI 83.1 to 94.5%). Taking into account smoking as the route of exposure improved specificity (77.6%, 95% CI 68.5% to 85.1%) and PPV (73.9%, 95% CI 66.4% to 80.3%) without affecting sensitivity (88.4%, 95% CI 78.97 to 94.51%) or NPV (90.2%, 95% CI 83.2% to 94.5%). Of note, non-SCRA drugs of misuse were also detected in samples from 87 (76.3%) of the 114 patients reporting SCRA use only.
Conclusions: As an indicator of analytically confirmed exposure, a patient history of SCRA use is moderately sensitive but lacks specificity, perhaps because patients use products without considering their chemical constituents or are misled by the packaging of products or the information provided by vendors. Taking into account use by smoking improves specificity. Clinicians should also be aware that in those reporting use of a SCRA alone, blood and/or urine samples often contain other drugs of misuse.

KEYWORDS Synthetic cannabinoid receptor agonists, patient history, sensitivity and specificity

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4. The Effect of 4-Methylpyrazole on Oxidative Metabolism of Acetaminophen in Human Volunteers

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Background: Acetaminophen (APAP) is commonly ingested in both accidental and suicidal overdose. Most APAP is converted to non-toxic metabolites in the liver, but a portion undergoes oxidative metabolism to NAPQI by cytochrome P4502E1 (CYP2E1). Normally, glutathione (GSH) conjugates with and detoxifies NAPQI, but in overdose, GSH stores become depleted, allowing NAPQI to accumulate and cause liver injury and death. There has been interest in preventing liver injury by inhibiting CYP2E1 using 4-methylpyrazole (4-MP), as has been demonstrated in mice, but its efficacy has not been evaluated in living human beings.
Methods: This randomized crossover trial examined the ability of 4-MP to inhibit cytochrome P450 2E1 metabolism of APAP in five human volunteers, as measured by the main outcome parameter, 24-hour urinary excretion of APAP oxidative metabolites. Participants received a single oral dose of APAP 80 mg/kg with and without intravenous 4-MP (15 mg/kg; 10 mg/kg 12 hours later), in two study arms separated by at least 2 weeks. 24-hour urine collections and serial plasma samples were analyzed for APAP and its metabolites (APAP-glucuronide, APAP-sulfate, APAP-cysteine, APAP-glutathione, and APAP-N-acetylcysteine).
Results: Co-treatment with 4-MP significantly decreased the percentage of ingested APAP that was recovered as oxidative metabolites in 24-hour urine (median ± IQR = 4.75 ± 1.86% vs. 0.41 ± 0.19%, p = .043). Plasma area under the curve (AUC) of oxidative metabolites also decreased with 4-MP cotreatment, from 27.4 ± 4.5 mM*hr to 5.2 ± 0.7 mM*hr for APAP-cysteine, and from 4.4 ± 0.9 mM*hr to 0 mM*hr for APAP-N-acetylcysteine (none detected in any plasma sample). No APAP-glutathione was detected in any urine or plasma sample. In plasma, peak APAP concentrations and time to peak were similar in both study arms with only minimal concentrations still detectable at 24 hours. By inspection, peak plasma levels of non-oxidative metabolites appeared greater and more sustained in the APAP +4-MP condition, but similar by time 24 hours. The percent of administered APAP that was recovered in 24-hour urine as APAP and all measured metabolites was similar in both study arms.
Conclusions: 4-methylpyrazole effectively decreased oxidative metabolism of APAP in human volunteers ingesting a supratherapeutic APAP dose.

KEYWORDS Acetaminophen, Fomepizole, Humans

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5. Effectiveness of antivenom for definite red-bellied black snake (Pseudechis porphyriacus) envenomation

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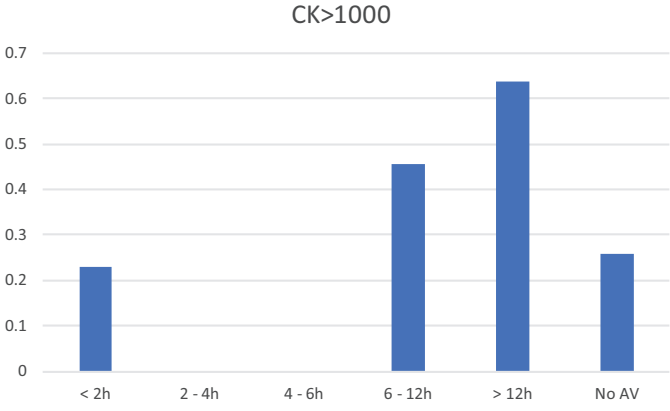
Objective: To investigate the effectiveness of antivenom in patients with definite red-bellied black snake (RBBS; Pseudechis porphyriacus) bites.
Methods: Prospective cohort study of patients with definite RBBS bites, recruited to the Australian Snakebite Project from January 2002 to March 2019. Data on all snakbites were collected prospectively, including patient demographics, clinical effects, laboratory results, complications and treatment. Blood samples were collected and then analysed by venom specific immunoassay for RBBS venom to confirm the snake species and measure venom concentrations before and after antivenom.
Results: There were 222 definite RBBS bites: the median age was 41 y (interquartile range [IQR]: 24-59 y) and 159 (72%) were males. Systemic envenomation occurred in 161 patients (68%). Systemic features included non-specific systemic symptoms (nausea, vomiting, headache, abdominal pain, diarrhoea) in 145 patients (90%), elevated activated partial thromboplastin time (aPTT) in 70 (43%) and myotoxicity with a creatine kinase (CK) > 1000U/L in 38 (24%). There were no deaths. Median peak venom concentration in 107 systemically envenomated patients with blood available was 20 ng/mL (IQR, 7–49 ng/mL; range, 0–360 ng/mL). In 101 patients with a white cell count [WCC] measured, 79 (78%) had a WCC >11x10-9), 75 envenomated patients (47%) were given antivenom: either tiger snake, black snake or polyvalent antivenom. Earlier antivenom was associated with a decreased incidence of myotoxicity, with only 3/53 patients (6%) given antivenom within 6 h developing myotoxicity, compared to 12/22 (55%) given antivenom >6 h post-bite and 22/86 (26%) not given antivenom (Figure 1). Administration of antivenom was association with normalising of the aPTT. Venom was undetectable after antivenom in all but one case. 26/75 envenomated patients (35%) given antivenom had an early systemic hypersensitivity reaction. Four non-envenomated patients got antivenom, one had a hypersensitivity reaction.
Conclusion: RBBS envenomation was characterised by non-specific symptoms, abnormal aPTT and WCC, and myotoxicity, the latter occurring in a quarter of patients. Early antivenom appeared to decrease the incidence of myotoxicity if given within 6 h and bound free venom. However, antivenom was associated with early systemic hypersensitivity reactions in over one third of patients.

AV Timing	CK >1000
< 2h	0.230769
2 - 4h	0
4 - 6h	0
6 - 12h	0.454545
> 12h	0.636364
No AV	0.258824
Non Envenomed	0



KEYWORDS Snakebite, venom, antivenom
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6. Severe delayed QT prolongation: a novel risk factor for adverse cardiovascular events from acute drug overdose

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Background: In ED patients who present with acute drug overdose, severe QTc prolongation (>500 ms) has been shown to be a predictor of adverse cardiovascular events (ACVE). However, it is unclear what clinical factors are associated with delayed severe QTc prolongation (dsQTp), and it is unknown whether dsQTp can predict ACVE. This study aims to: (1) Define clinical factors associated with dsQTp, and (2) test whether dsQTp is an independent predictor for ACVE.
Methods: This was a prospective cohort study at 2 urban tertiary care EDs. Data was collected by trained research assistants, and included demographics, drug exposure, medication administration, initial and repeat ECG data, lab data, and outcome measures. dsQTp was defined as presence of initial QTc 499. The primary study outcome was the composite of ACVE defined as in-hospital occurrence of any of the following: MI, shock, ventricular dysrhythmia, and cardiac arrest. Univariate statistics and multivariable logistic regression calculations were made using SPSS version 24. With a fixed sample size of 1670, we calculated that we would have 99% power to show a 3-fold increase in risk with 0.05 alpha.
Results: Out of 2311 patients screened, 641 were excluded (age 500) leaving 1670 patients for analysis. The dsQTp group (N = 27) was found to be older than the control group (N = 1643)(40.1 vs 51.6, P
Conclusion: This cohort study reveals a subset of ED patients who are at greater risk of overdose-related ACVE but not immediately apparent by the initial ECG. Further study is needed to identify which patients are at risk for dsQTp, as they may require prolonged observation and repeated ECGs. Limitations include missing repeat QTc measurements, and inability to control for overdose severity as a surrogate for repeat ECGs. Future study is warranted to further characterize patients at risk for dsQTp to evaluate other exposure-related factors as yet undiscovered.

KEYWORDS Overdose, QTc, adverse events
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7. Associations of Prenatal Manganese with Visual-Motor Skills in Adolescence

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Background: Manganese (Mn) is an essential nutrient required for normal development, found naturally in various foods and as an environmental contaminant resulting from industrial emissions. While an essential nutrient, Mn is known to be neurotoxic in adults with high-level occupational exposure. Epidemiologic studies suggest that early life or childhood exposures to lower-level Mn may be associated with declines in cognitive and motor function in infants and school-age children. In some cases, these associations are modified by sex or other

exposures. Few studies have examined whether Mn associations persist through adolescence. We assessed hypothesized associations between biomarkers of prenatal Mn exposure and visual-motor skills in adolescents using the Wide Range Assessment of Visual Motor Abilities (WRAVMA), a standardized test of visual-motor performance.
Methods: 788 mother-infant pairs were recruited for the New Bedford Cohort (NBC) study, a prospective birth cohort examining associations between prenatal chemical exposures and neurocognitive development. Cord blood and maternal peripartum toenails were collected and analyzed for metals, including Mn, lead, and arsenic. The WRAVMA, comprised of visual-motor (VM), visual-spatial (VS), and fine motor (FM) subscales and a composite score, was administered to NBC children at approximately age 15 years. Covariates were obtained from questionnaires and medical record reviews administered after birth and in periodic follow-up visits. The current study includes 518 NBC participants with both 15-year WRAVMA testing and at least one Mn exposure biomarker in cord blood or maternal toenails. Multiple linear regression was performed with each Mn biomarker on each WRAVMA score. Regression diagnostics were performed to ensure the assumptions of linear regression were met. Regression model covariates and potential confounders were selected based on a priori and empiric (model fit, change in Mn exposure effect estimates) considerations.
Results: The studied population was predominantly white (69%), 52% female, and socioeconomically diverse with a mean (range) age at WRAVMA testing of 15.6 (14.3 to 17.9) years. Biomarker levels of Mn were consistent with exposures in other birth cohort studies. There was no adverse association of cord blood or toenail Mn levels with 15-year WRAVMA scores (Table 1).
Conclusions: In this initial analysis, findings were null. Future analyses will assess potential differential sensitivity to Mn effects by sex or other exposures such as lead.

Table 1 Multiple linear regression models associating an interquartile range (IQR)* increase in cord blood or maternal toenail Mn levels with WRAVMA scores at age 15 years.

	Cord Blood Mn** (n = 413):	Nail Mn** (n = 254):
Score	β (95% Confidence Interval)	β (95% Confidence Interval)
Composite	0.91 (-0.33 to 2.15); p = 0.15	0.02 (-1.30 to 1.35); p = 0.97
Visual Motor	0.78 (-0.45 to 2.01); p = 0.21	-0.95 (-2.31 to 0.41); p = 0.17
Visual Spatial	0.80 (-0.62 to 2.22); p = 0.27	0.45 (-1.00 to 1.91); p = 0.54
Fine Motor	0.47 (-0.92 to 1.85); p = 0.51	0.50 (-1.04 to 2.04); p = 0.53

*Cord blood Mn IQR = 17.0 ng/mL; toenail Mn IQR = 0.37 µg/g.
**Models adjusted for: child age and school grade at exam, sex, cord blood lead; maternal IQ, education, age at birth, and depression; parental marital status; examiner and HOME score. Cord blood Mn models additionally adjusted for child race, video game usage, household income and paternal education. Toenail Mn models additionally adjusted for maternal toenail arsenic.

KEYWORDS Manganese, Visual-motor abilities, Prenatal exposure

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8. Antiemetics Effectiveness for Cannabinoid Hyperemesis Syndrome (CHS) Emergency Department (ED) Encounters

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Background: CHS is a cyclic vomiting syndrome associated with heavy cannabis use. CHS-related ED encounters have increased since cannabis policy liberalization in Colorado. We evaluated antiemetic effectiveness for CHS treatment in ED patients.
Methods: This was a retrospective chart review of CHS-ED visits from a large urban academic ED between 1/1/2012-12/31/2014. An antiemetic was considered effective if it was the last drug administered before ED discharge home, with documented symptom improvement. Patients admitted to the hospital, transferred to intensive care (ICU) or to the observation unit after antiemetic administration were considered to have unresolved symptoms. Antiemetic effectiveness was compared to all other administered antiemetics using chi square and odds ratios (OR) or Fischer exact test for drugs/combinations with
Results: 247 CHS visits were abstracted. The median age was 30 (IQR: 24, 36); there were 123 females, 124 males and the race/ethnicities were: African-American/Black (n = 103, 44.13%), White/Caucasian (n = 102, 41.30%), Other (n = 37, 14.98%), Asian (n = 2, 0.81%), unspecified (n = 2, 0.81%), and Hawaiian/Pacific-Islander (n = 1, 0.40%). 645 antiemetics were administered from five drug classes. The most frequently administered antiemetics were: ondansetron (n = 249, 38.60%), haloperidol (n = 107, 16.59%), lorazepam (n = 70, 10.85%), diphenhydramine (n = 62, 9.63%), promethazine (n = 58, 8.99%), metoclopramide (n = 55, 8.53%), droperidol (n = 21, 3.26%), prochlorperazine (n = 16, 2.48%), olanzapine (n = 4, 0.62%), and diazepam (n = 3, 0.47%). 142 (59.49%) visits had effective antiemetics, 54 (21.86%) were admitted to the hospital, 48 (19.43%) were transferred to the observation unit, and 3 (0.12%) were admitted to the ICU. In 34 effectively treated visits, 2-3 antiemetics were co-administered. Relative effectiveness of each antiemetic or combination are summarized in Tables 1-2.
Ondansetron was the most frequently administered overall and most frequently administered as a first antiemetic (n = 249, 38.6%, n = 156, 63.2% respectively). Ondansetron was effective in only 17.27% of administrations (OR = 0.62; 95% CI = 0.42-0.93). Ondansetron was effective when administered as the first drug only 14.74% of the time (OR = 1.40; 95% CI = 0.63-3.09). Droperidol's overall effectiveness was nearly 48% (OR = 3.39; 95% CI = 1.41-8.15), yet it was only administered as the first drug 4 times (p = 0.19). Haloperidol was effective in 25% of first administrations (OR = 2.53; 95% CI = 1.03-6.25) and was the only effective first administered antiemetic. The most effective overall combinations were diphenhydramine/metoclopramide (OR = 18.32; 95% CI = 2.12-158.13) and haloperidol/ ondansetron (OR = 9.95; 95% CI = 2.60-38.02); they were only given first 4 and 13 times, respectively.
Adverse reactions to antiemetics administered intravenously were reported in three visits; one had akathisia after haloperidol, another had dysphoria following droperidol, and the third returned to the ED with a dystonic reaction after receiving 15 mg haloperidol with 25 mg diphenhydramine. Each was discharged from ED with resolved symptoms.
Conclusions: Antipsychotics were the most effective antiemetics for CHS treatment. Ondansetron was the most frequently administered but was ineffective for CHS treatment.

KEYWORDS Cannabinoid Hyperemesis Syndrome, Antiemetic, Effectiveness

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9. National assessment of loperamide abuse awareness and ability to restrict purchase at retail pharmacies

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Background: Reports of loperamide abuse and subsequent cardiac dysfunction have increased through various national surveillance systems. In January of 2018 the Food and Drug Administration (FDA)

stated they will ask manufacturers to limit package sizes of loperamide in an effort to reduce inappropriate use. No restriction exists on the quantity of loperamide packages that can be purchased. The goal of our study is to characterize pharmacist knowledge of loperamide abuse at the point of retail access. Secondly we aim to determine what pathways pharmacist have throughout the United States to limit the sale of loperamide when abuse is suspected.

Methods: Random sampling was done using a list of zip codes compiled from the United States Postal Service and Internal Revenue Service. A Random Number Generator (RNG) was used to identify three zip codes within each state and a list of pharmacies was created from the google maps "nearby" function within each identified zipcode. Pharmacies to contact from the list were identified again using the RNG. The self-reported pharmacists at contacted pharmacies were asked a three question survey regarding prior knowledge of loperamide abuse, knowledge of dose or co ingestants needed for abuse, and ability to restrict suspicious sales. Responses to "ability to restrict sale" were then characterized into themes. Data collection will continue until three pharmacies from each state are included (n = 150).

Results: A total of 225 pharmacies were contacted and 106 pharmacies from 50 states participated in the survey. Chain pharmacies comprised 58.5% (n = 62), 40.5% (n = 43) were independent, and 1.0% (n = 1) were hospital associated pharmacies. In total 74.5% (n = 79) of pharmacists reported being aware that loperamide was being abused or had abuse potential. Only 27.4% (n = 29) were aware of the need for supratherapeutic dosing or co ingestion with a P-glycoprotein (PGP) inhibitor to achieve a euphoric effect. Further, only 31.1% (n = 33) felt they had the ability to deny suspiciously large sales or restrict purchasable quantity if they suspected abuse. The majority of pharmacies (n = 73, 68.9%) believed they had no ability to restrict the sale. Common themes from free text responses included inability to oversee purchases from other departments (n = 29, 27.8%) and concerns regarding ability to deny Over-The-Counter (OTC) purchases (n = 8, 7.7%). In the sample 4.7% (n = 5) of pharmacists stated they could or already have placed the product behind the counter. Only 1.0% (n = 1) had a sale quantity restriction in place.

Conclusions: In this sample, many pharmacists knew of loperamide abuse, though one quarter of pharmacists were unaware. Close to three quarters of survey respondents were not aware of the larger doses or co ingestion with PGP inhibitor needed for abuse. The majority of pharmacists felt they had no ability to reduce the quantity of loperamide sold or deny suspicious sales. Common reasons for inability to regulate sale were multiple avenues of purchase, and questions regarding ability to deny the purchase of OTC drugs. The FDA initiative to decrease the package sizes of loperamide may be ineffective without further regulation to increase pharmacist ability to monitor sale.

KEYWORDS Loperamide, Abuse, Policy

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Table 1 Pharmacist Survey Answers.

	Chain (N = 62)	Independent (N = 43)	Hospital (N = 1)	Total N = 106
Aware of loperamide abuse, n (%)	47 (75.8%)	29 (72.1%)	1 (100.0%)	77 (72.6%)
Aware of higher doses or PGP inhibitor needed for euphoria, n (%)	20 (32.2%)	9 (20.9%)	0 (0%)	29 (27.3%)
Able to restrict sale of loperamide, n (%)	14 (22.6%)	18 (41.8%)	1 (100.0%)	33 (31.1%)

10. Impact of ASTM Safety Standard on Accidental Exposures to Liquid Laundry Packets in Children

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Background: Accidental exposures to liquid laundry packets have been reported to regional poison centers since liquid laundry packets became available in 2012. Emergency department visits, injuries, and

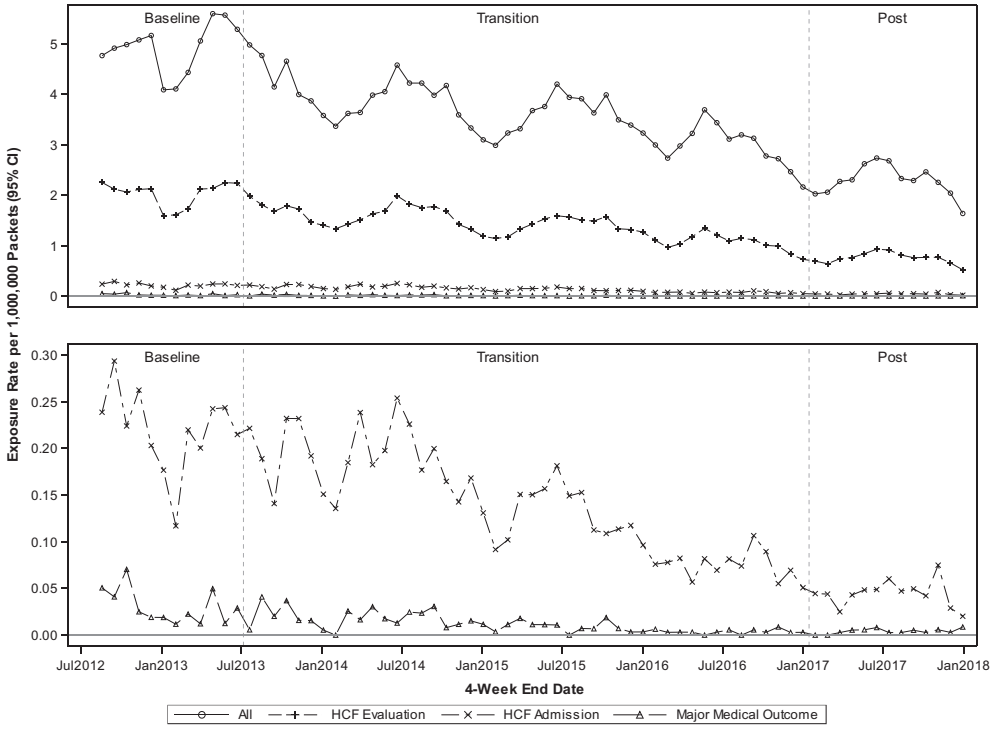

Figure 1 Cumulative Sales-Adjusted Rates of Accidental Exposures to Liquid Laundry Packets in Children <6 Years Old by Level of Care.

some deaths have also been reported. In response to concerns about liquid laundry packet safety, ASTM International developed a voluntary safety standard to reduce the risk of accidental exposures. The standard details product, packaging, and labeling requirements and was finalized in 2015. This study aimed to compare liquid laundry packet exposures in children
Methods: NPDS was queried for unintentional general exposures in children
Results: A total of 64,532 accidental exposures in children
Conclusions: The morbidity of accidental exposures to liquid laundry packets in children decreased after implementation of the ASTM safety standard as measured through decreases in clinically significant exposures (HCF evaluations, HCF admissions, major medical outcomes). These findings are consistent with reports from the National Electronic Injury Surveillance System which showed emergency department visits involving liquid laundry packets decreased after ASTM standard implementation. As exposures and HCF encounters continue to occur, ongoing monitoring should be performed to determine if additional safety measures are required.

Table 1 Cumulative Sales-Adjusted Rates of Accidental Exposures to Liquid Laundry Packets in Children <6 Years Old by Period of ASTM Standard Implementations.

	Baseline Period Cumulative Rate per 1,000,000 Packets Sold	Post Period Cumulative Rate per 1,000,000 Packets Sold	% Change(95% CI)
All Exposures	**4.920**	**2.291**	**-53.4% (-54.7%, -52.1%)**
HCF Evaluation	2.026	0.758	-62.6% (-64.3%, -60.9%)
HCF Admission	0.218	0.044	-79.6% (-82.8%, -76.0%)
Major Medical Outcomes	0.030	0.004	-86.4% (-91.9%, -77.1%)

KEYWORDS Liquid laundry packets, ASTM standard, NPDS evaluation

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11. Modern Lethality Rate of Suspected Cyclopeptide Mushroom Poisoning in the US: The Power of Supportive Care!

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Objective: This study was designed to estimate the mortality of suspected cyclopeptide-containing mushroom ingestions reported to the National Poison Data System.
Background: Although silibinin reportedly improves survival in suspected cyclopeptide-containing (hepatotoxic) mushrooms, the untreated mortality rate often cited as greater than 20% is based on decades old data. An ongoing open-label trial of silibinin seeks US Food and Drug Administration approval. Our recent analysis of data from a single poison control center (PCC) suggested a lower mortality rate of 8.3% following suspected cyclopeptide-mushroom ingestion. This new study was designed to validate our PCC's findings in the National Poison Data System (NPDS) by increasing the sample size, and accounting for geographical and local practice variations.
Methods: A 10-year (1/1/2008-12/31/2018) retrospective review of suspected cyclopeptide-containing mushroom ingestions reported to NPDS used the same inclusion/exclusion criteria as an ongoing silibinin trial (NCT00915681): Age >2; history of eating foraged mushrooms; gastrointestinal symptoms (cramping abdominal pain, nausea, vomiting, and/or watery diarrhea) within 48 hours of mushroom ingestion; and aminotransferases (AST or ALT) above the upper limit of normal value for the lab reference range within 48 hours after mushroom ingestion. We systematically followed up on all cases that met inclusion criteria with their respective PCC to confirm, eligibility, diagnosis, treatment, and mortality or transplant outcomes. De-identified data were gathered by survey using a standardized abstraction tool.
Results: 8,953 mushroom exposures were reported to NPDS, of which 296 were retained based on study inclusion criteria. The survey response rate was 57% (27/47 PCCs), as was the case response rate (168/296). Based on survey responses, 33 cases were subsequently excluded, largely because of the absence of confirmed foraging, or a non-mushroom diagnosis, leaving 135 included cases. The geographic distribution was: Northeast, 34%; Southeast, 15%; West coast, 31%; Midwest, 16%; South Central, 4%. The overall mortality rate was 9% (12/135). Mortality in silibinin/silymarin-treated vs untreated cases was 10% (4/39), vs 8% (8/96), respectively. Mycologist identification occurred in 17.8% of cases (24/135), of which 70.8% (17/24) were cyclopeptide-containing mushrooms. Among identified cases, the mortality rate was; 13% (1/8) vs 11% (1/9), in silibinin/silymarin-treated vs untreated cases, respectively.
Conclusion: In this retrospective review of NPDS data, the mortality rate was 8% in patients with presumed cyclopeptide-mushroom poisoning who met enrollment criteria for the ongoing silibinin trial but did not receive silibinin. This modern fatality rate is much lower than the greater than 20% previously reported and likely represents improved supportive care. Using the same criteria for cyclopeptide-mushroom poisoning, a well-designed randomized controlled trial would require more than 300 patients to find a statistically significant 50% reduction (from 8% to 4%) in mortality with an alpha of 0.05 and beta of 0.2. As the current open-label trial plans to enroll 50 patients, it is likely underpowered to demonstrate evidence of silibinin's efficacy.

KEYWORDS Amanita, Mushroom, Silibinin

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12. Rates of adequate reversal and symptoms of opioid withdrawal syndrome (OWS) in patients treated initially with low dose naloxone or high dose naloxone

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Background: When managing opioid overdose patients, the optimal naloxone regime should rapidly reverse respiratory depression while avoiding opioid withdrawal syndrome (OWS). Patients with OWS may leave hospital against medical advice and self-medicate. Published naloxone administration guidelines have not been empirically validated and most were developed before fentanyl overdose was common.

Methods: In this retrospective matched cohort study, we reviewed prehospital and emergency health records of opioid overdose patients treated in two urban emergency departments in British Columbia, Canada during an ongoing fentanyl overdose epidemic (January 1, 2013 to Dec 31, 2017). We compared the proportion of patients with adequate reversal and symptoms associated with OWS when treated initially with low dose naloxone (LDN; ≤ 0.15 mg) or with high dose naloxone (HDN; > 0.15 mg) administered either prior to hospital arrival or in hospital. Adequate reversal was defined as Glasgow Coma Scale (GCS) > 10 and either respiratory rate >11, or O2 Saturation >91% within 30 minutes of naloxone administration. Symptoms associated with OWS included the following: new or worsening nausea requiring treatment, new or worsening agitation, aggressive behavior, restlessness, pulse >100, diarrhea, tremor, flushing, sweating, gooseflesh skin, piloerection, bone or joint aches, rhinorrhea, lacrimation, and yawning. Four binary outcomes were examined: adequate reversal within 30 minutes of initial dose before another dose of naloxone was administered, adequate reversal at any time, symptoms associated with OWS after the initial dose but before another dose of naloxone was administered, and symptoms associated with OWS at any time. For the analysis, LDN patients were matched to HDN cases based on initial respiratory rate as a proxy for overdose severity. Odds ratios (OR) for adequate reversal and for symptoms associated with OWS were obtained via logistic regression stratified by matched sets and adjusted for initial naloxone dose (LDN or HDN), age, sex, pre-naloxone GCS, and presence of other drugs or alcohol.
Results: Eighty LDN patients were matched up to 4:1 with 299 HDN patients. Compared to LDN patients, HDN patients were more likely to have adequate reversal after initial dose (OR = 2.73; 95%CI:1.19-6.26, p = 0.0181) and adequate reversal at any time (OR = 6.07; 95%CI:1.81-20.32, p = 0.0034). Twelve (15.0%) LDN and 114 (38.1%) HDN patients had adequate reversal after a single dose of naloxone. LDN patients were treated with a mean total dose of 0.22(SD 0.41) mg of naloxone before adequate reversal. HDN patients were treated with a mean total dose of 0.66(SD 0.43) mg of naloxone before adequate reversal. Compared to LDN patients, HDN patients were also more likely to have symptoms associated with OWS after the initial dose (OR =8.43; 95%CI:1.96-36.27, p = 0.0042) and at any time (OR =2.56; 95%CI:1.17-5.6, p = 0.0187). Symptoms associated with OWS occurred after initial naloxone dose in 5.0% of LDN patients and 19.7% of HDN patients and at any time in 15.0% of LDN patients and 29.8% of HDN patients.
Conclusions: HDN patients had a higher incidence of adequate reversal after initial naloxone dose but were also more likely to have symptoms associated with OWS. Further prospective research should compare different naloxone dosing regimens to determine the rates of adequate reversal and OWS.

KEYWORDS Opioid, Overdose, Naloxone

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13. Efficacy of Intravenous Hydroxocobalamin Versus Control for Treatment of Severe Methanethiol Toxicity in a Swine (Sus scrofa) Model

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Background/Objectives: Methanethiol (CH4S) is a poison which occurs naturally in crude oil and natural gas and is used to produce methionine, a dietary component in poultry and animal feed. It is also used in mining operations. There have been reports of deaths, accidental and intentional, with this toxin. According to the Occupational Health and Safety Administration and the US Environmental Protection Agency (EPA), CH4S is highly toxic and has been associated with workplace injury or deaths. Significant exposure to CH4S can cause metabolic acidosis, unconsciousness, seizures, myocardial infarction, and death. Moreover, the Occupational Safety and Health Administration (OSHA) lists CH4S as a potential terrorist weapon. CH4S inhibits cytochrome c oxidase of the electron transport chain which compromises ATP production. This can lead to the development of metabolic acidemia and hyperlactatemia. Given this agent causes toxic effects similar to cyanide and hydrogen sulfide (H2S), treatment for toxicity is postulated to be similar, although there is no specific treatment for CH4S. The objective of this study is to directly evaluate the impact of IV Hydroxocobalamin (HOC) versus saline/vehicle control on mortality in a methanethiol induced apnea large animal model.
Methods: We administered intravenous sodium methanethiolate (an IV alternative to inhaled methanethiol for safety of laboratory personnel) to 32 anesthetized swine to a standardized endpoint (20 seconds of apnea confirmed by capnography) and then randomly assigned each animal (16 control, 16 treatment) to receive either intravenous HOC (150mg/kg)or saline control. Physiologic parameters monitored included heart rate (HR), MAP, mixed venous saturation (SVO2), cardiac output (CO), and pulmonary artery pressure. Laboratory values monitored included mixed venous oxygenation, pH, lactate, base excess, serum bicarbonate, urinary/blood toxin concentration, and inflammatory markers.
Results: The two groups were similar in size and other baseline characteristics at study start and at treatment time. Compared to the HOC group, the control group had significantly lower HR, SBP, DBP, and MAP at 5-10 minutes post-apnea, lower SVR at 5 minutes post-apnea, and lower TVE 10-15 minutes post-apnea. The control group also had lower etCO2 and higher etO2 at 10-15 minutes post-apnea. None of the animals survived to the end of the study period (60 minutes). The Kaplan-Meier survival curves of the two groups were significantly different (log-rank p = 0.0321), with the HOC group surviving longer than the control group (32.4 ± 7.3 minutes vs. 25.8 ± 1.0 minutes).
Conclusions: While hydroxocobalamin administration results in a transient improvement in vital signs and a brief delay in death, it is not effective in improving survival in a swine model of methanethiol poisoning.

KEYWORDS Methanethio, hydroxocobalamin, methyl mercaptan

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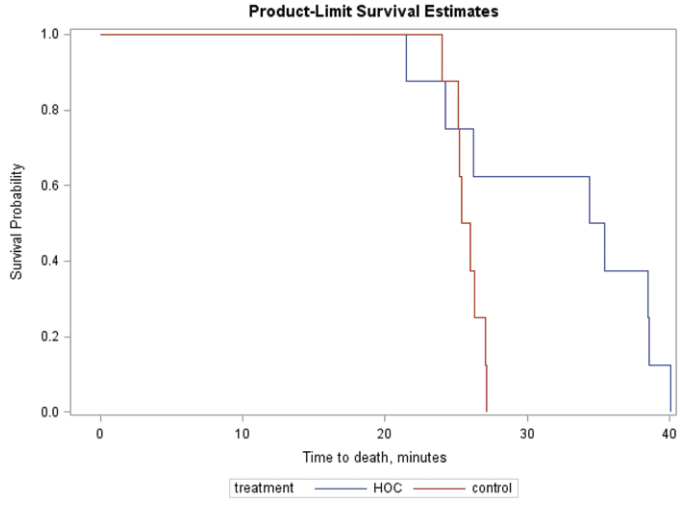

Figure 1 Survival curves for HOC (blue) and control (red). Log-rank p-value =0.0321.

14. Intravenous phytonadione administered orally for warfarin-related coagulopathy

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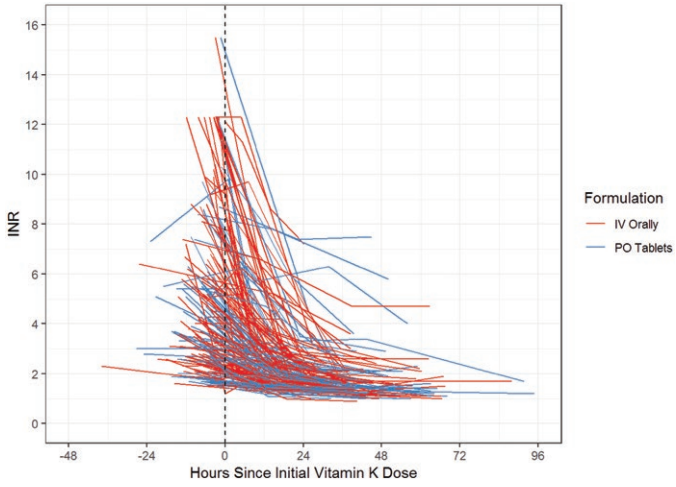
Background: The long-acting anti-coagulant, brodifacoum, has been identified in contaminated synthetic cannabinoid products. Like warfarin, oral phytonadione has been used to treat coagulopathy associated with brodifacoum ingestion; however, the doses required are significantly higher. Additionally, the treatment duration is substantially longer (weeks – months). The cost of phytonadione tablets tripled from 2014 to 2016 and is a significant barrier to treating patients after hospital discharge. In response to these cost barriers, our institution adopted the practice of compounding the intravenous (IV) phytonadione solution into an oral solution for the treatment of warfarin-related coagulopathy. This formulation, given by this route, has not been evaluated for efficacy in comparison to the commercially available tablet. The objective of this study was to evaluate the efficacy of phytonadione intravenous solution given orally compared to phytonadione tablets for reversal of coagulopathy related to warfarin.
Methods: We conducted a retrospective, observational study of adult patients admitted to UK HealthCare who received oral phytonadione for warfarin-related coagulopathy. Patients were excluded if they were less than 18 years of age, received phytonadione via intravenous or subcutaneous route, or had known liver dysfunction. The international normalized ratio (INR) was measure before and after phytonadione administration. The primary outcome was INR ≤1.4 at 24 hours after phytonadione administration.
Results: From January 1, 2015 to August 1, 2018 a total of 200 patients were identified. In total, 58% (n = 116) patients received IV phytonadione solution given orally and 42% (n = 88) patients received the tablets. The baseline median INR (IQR) prior to phytonadione administration was 4.3 (2.7-7.2) in the IV solution orally group and 3.4 (2.3-5.6) in the tablet group (p = 0.020). The primary outcome was not significantly different between groups; the primary outcome was met in 20.7% (n = 24) of patients in the IV formulation group and in 17.9% (n = 15) of patients in the tablet group (p = 0.750). The median post-phytonadione INR at 24 hours was 1.4 in the IV solution orally group and 1.4 in the tablet group (P = 0.773). The absolute reduction in INR at 24 hours was greater in the IV solution orally group; mean reduction in INR from baseline was 2.5 and 1.7 (p = 0.043) in the IV formulation and tablet groups, respectively.
Conclusions: There was no significant evidence of a difference in achievement of INR ≤1.4 at 24 hours between the IV phytonadione solution given by mouth and the tablet. The findings from this study can be used to inform recommendations for the use of intravenous phytonadione by mouth as an alternative for patients after brodifacoum ingestion.



KEYWORDS Phytonadione, brodifacoum, long-acting anti-coagulant

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15. Five Decades of Global Chemical Terror Attacks: Data to Inform Training and Preparedness

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Objective: To review a large publicly-available database containing five decades of worldwide incidents of terrorism to inform the training and preparedness of healthcare providers and policy-makers for future chemical terror attacks.
Methods: We conducted a descriptive study of chemical agent terror attacks to understand historical trends in agents, targets and routes of exposure utilizing The Global Terrorism Database (GTD), maintained by the National Consortium for the Study of Terrorism and Responses to Terrorism (START). We downloaded the full dataset of 180,000 incidents from 1970-2017 onto Microsoft Excel. We eliminated cases without chemical agents as the primary weapon, and included 383 cases in which a chemical weapon was employed for analysis. We measured the following factors over time: target type, morbidity and mortality, region, and method of attack. Where available, the identity of the agent was recorded. The attacks were classified by route of exposure and traditional chemical weapon categories (choking, blister, blood, nerve, or riot control).
Results: We included 383 terror incidents involving chemical weapon use from 1970 to 2017. There has been an increase in the absolute number of chemical attacks from 2011 to 2017, particularly associated with armed conflicts in Afghanistan, Syria, and Iraq. We identified increasing chemical weapon incidents in South Asia, the Middle East and North Africa. Twenty-one percent of chemical attacks involved explosives or bombs. The most common targets were private citizens and property, police, and educational institutions. We were able to identify the target agent in 40 percent of cases. The most frequent routes of exposure were dermal and mucosal (acids were used in approximately 17 percent of all attacks while tear gas accounted for 21 percent of attacks where the specific agent was known) and inhalational (with chlorine accounting for 26 percent of all attacks where the agent could be identified). Where the agent could be classified, blood agent incidents declined from 33 percent of attacks before 2001 to 14 percent after 2001, while nerve agent attacks declined from nine to one percent over the same period. In contrast, across this period, choking (chlorine) and vesicant agent (mustard) use increased from 7 to 48 percent and from 2 to 6 percent of attacks, respectively. The most commonly employed agents after 2001 were chlorine, which increased from 2 percent before 2001 to 42 percent of attacks after 2001; tear gas, which decreased from 33 to 13 percent; and cyanide, which decreased from 23 to 11 percent.
Conclusions: These findings can inform future chemical weapon preparedness and training. South Asia and the Middle East and North Africa appear to be most at risk for chemical weapon use in terror activities and preparedness efforts should prioritize these regions. Training activities should focus on educational facilities, police, and private citizens. As approximately one-fifth of attacks involved explosives, training must also include blast injury prevention and response. First responder training and pre-deployment of assets should focus on choking agents, namely chlorine; riot-control agents; mucosal or dermal acids; and cyanogenic agents.

KEYWORDS Terrorism, Chemical Weapons, Preparedness

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16. Drug-Specific Anti-Xa Levels Before and After Infusion of Coagulation Factor Xa (recombinant), inactivated-zhzo (Andexxa®) for Bleeding in Patients Anticoagulated with Apixaban or Rivaroxaban: A Quality Improvement Project

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Background: Coagulation factor Xa (recombinant), inactivated-zhzo (CFXa), known as Andexxa®, was approved for use by the Food and Drug Administration (FDA) in 2018 for reversal of bleeding from direct Factor Xa inhibitors (DFXIs) based on data from the ANNEXA-4 study demonstrating hemostatic efficacy as well as reduction in medication-specific anti-Xa levels (mean reduction at end of CFXa infusion =92%). CFXa is the first in class antidote for reversal of bleeding due to DFXIs and is currently priced at approximately $30,000 (U.S. currency) for the low-dose regimen and $60,000 for the high-dose regimen. Due to the high cost and scarce nature of CFXa, our institution created restriction criteria for use of CFXa (see Table 1). We sought to determine if anti-Xa levels changed in a similar nature to the ANNEXA-4 study after introduction of CFXa to our hospital's formulary.

Methods: This was a medication utilization quality improvement (QI) project assessing the use of CFXa after its introduction at a level I Adult and Pediatric U.S. Trauma Center. This project was deemed exempt by the local IRB. Shortly after FDA-approval of CFXa in 2018, CFXa was added to the hospital formulary. Cases were identified by querying the electronic medical record (Epic, Verona, WI) for any administered doses of CFXa. The study hospital's laboratory is capable of performing real-time drug-specific anti-Xa levels, which were incorporated into the clinical criteria for CXFa utilization. Cases were abstracted by clinical pharmacists for pre/post drug-specific anti-Xa levels, DFXI dosing, CFXa indications, initial Glasgow Coma Scale (GCS) and demographic data. Descriptive statistics were used for analysis.

Results: CFXa was administered to six patients over a four month time period. The indication for anticoagulation for all patients was atrial fibrillation; and one patient was also using for secondary prophylaxis after a stroke. Median age 85.5 years old. All patients received CFXa due to an intracranial hemorrhage (ICH). GCS scores on admission ranged from 6-15. Additional clinical data are displayed in Table 2. Mean decrease in drug-specific anti-Xa levels drawn at the end of the two-hour CFXa infusion was 15.2% (range, +17% to -42.9%). CFXa dosing and pre/post-infusion medication-specific anti Xa-levels are displayed in Table 3. One patient died (initial GCS =6); the remaining five patients were discharged from hospital. Three patients are residing at the same living facilities where they were living prior to their ICH. One patient receives care at an outside facility; their current living and neurological status are unknown. One patient discharged from a rehabilitation unit to a skilled nursing facility as his abilities for self-care, mobility, and cognitive-linguistics were below baseline.

Conclusions: In a small series of six patients we observed drug-specific anti-Xa level reductions after the completion of CFXa infusions were smaller than those observed in the ANNEXA-4 trial, and in one case increased. These data suggest a more thorough external validation of the laboratory outcomes in ANNEXA-4 is warranted as CFXa is introduced into practice in the U.S.

KEYWORDS Coagulation factor Xa inactivated, anticoagulant, reversal

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Table 1 CFXa Use Criteria.

Patient must meet all of the following criteria:	Patients with the following should not be considered candidates for CFXa administration:
1. Intracranial hemorrhage or life-threatening hemorrhage unresponsive to the massive transfusion protocol and/or standard procedural interventions 2. Be considered survivable per staff physician 3. Have a medication-specific anti-Xa level above 50 ng/mL (if timing of last dose is definitely known, would not have to wait for level)	1. Expected survival of less than 1 month 2. Patients with normal renal function whose last dose of apixaban or rivaroxaban was greater than 48 hours ago

Table 2 Patient Characteristics.

Patient	Age	SCr (mg/dL)	Oral Anticoagulant Dose	Reason for Anticoagulation	Last Dose Prior to Emergency Department Arrival	Chief Complaint	Injuries	Initial GCS
1	≥ 89	0.98	Apixaban 2.5 mg BID	Atrial fibrillation	Morning or evening day of admission	Mechanical fall	Subdural hematoma	15
2	72	1.07	Apixaban 5 mg BID	Atrial fibrillation	2 hours	Syncope vs. fall (on ice)	Subarachnoid hemorrhage	15
3	88	1.05	Apixaban 5 mg BID	Atrial fibrillation	8.5 hours	Mechanical fall on ice	Subdural and subarachnoid hemorrhage	15
4	84	1.21	Apixaban 5 mg BID	Atrial fibrillation; stroke	8 hours	Hemorrhagic conversion of R cerebellar stroke	Intraparenchymal hemorrhage	13
5	52	1.21	Rivaroxaban 20 mg Daily	Atrial fibrillation	Unknown	Motor vehicle crash at highway speeds	Intraparenchymal hemorrhage	6
6	87	1.26	Rivaroxaban 15 mg daily	Atrial fibrillation	16 hours	Fall from standing	Subarachnoid and subdural hemorrhage	15

Table 3 Medication-Specific Anti-Xa Levels.

Patient	Pre-infusion Level (ng/mL)/ Time after ED arrival (minutes)	High or Low Dose CFXa	Time of CFXa Administration after ED arrival (hours)	Post-infusion Level (ng/mL)/ Time after ED arrival (hours)	Percent Change from Baseline	Post-infusion Level (ng/mL)/ Time after ED arrival (hours)
1	85.1/17	Low	2-4	46.4/5	-45.5%	44.5/12.5
2	218.3/23	Low	2.5-4.25	255.4/5	+17%	145.2/10
3	136.7/6	Low	1.75-3.75	135.5/3.5	-0.01%	None available
4	280.6/2	Low	1.75-4	224.9/4.5	-19.9%	135.7/13.75
5	53.2/37	High	1.5-4	30.4 /3	-42.9%	None available
6	127/13	Low	2-4.5	137.1/4.5	+0.08%	None available

17. Clinical Characteristics of Thallium Poisoning Reported to the Baghdad Poison Control Center Between 2008-2015

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Background: Thallium is a metal found in the environment as a constituent of granite, shale or volcanic rock. It forms toxic univalent or trivalent salts that are odorless, tasteless and often used as a rodenticide outside the United States where its sale was banned in 1972. Case reports and series of thallium poisonings have previously been described from eating different laced food items, dermal exposure and from the suicidal ingestion of thallium salts.
Methods: This is a retrospective review of de-identified case report forms of thallium poisonings that were managed by the Baghdad Poison Control Center during 2008–2015. Data regarding demographics, clinical manifestations, treatment and outcome were extracted by the research team. The frequency of various variables was analyzed using IBM SPSS 22. Secondary analysis of association of various variables with outcome was performed.
Results: A total of 67 cases were analyzed out of which 67% (n = 43) occurred in 2008. The majority of cases (53.7%, n = 36) were reported from the city of Baghdad. The reason of exposure was not uniformly confirmed but was deemed homicidal or criminal by the poison center director. The routes of exposure were ingestion (80.6%, n = 54) and dermal (19.4%, n = 13). The mean age of cases was 24 ± 15.6 year and 70.1% (n = 47) were male. Most of the cases had a delayed presentation to the hospital. The median (IQR) time of presentation from exposure was 14 (7-30) days. The most frequently reported symptoms were neurological (97%, n = 65) and consisted of severe ascending pain in lower extremities (88%, n = 59), coma (6%, n = 4), lower limb weakness (4.5%, n = 3), delirium (3%, n = 2), seizure (3%, n = 2), slurred speech (3%, n = 2), ptosis (1.5%, n = 1) eye pain, (1.5%, n = 1) and bilateral leg paralysis (1.5%, n = 1). The dermal symptoms were reported in 77.6 % (n = 52) of cases including alopecia (77.6%, n = 52), rash (6%, n = 4), hyperpigmentation (1.5%, n = 1) and mees lines (1.5%, n = 1). The gastrointestinal symptoms occurred in 52.2% (n = 35) of cases. They included abdominal pain (43.3%, n = 29), constipation (16.4%, n = 11), vomiting (10.4%, n = 7), dysphagia (7.5%, n = 5), and diarrhea (6%, n = 4). Thallium blood and urine levels were not available. A semi-quantitative colometric urine test was positive for all cases. This test uses cyanide reagent with chloroform and dithizone along with thallium standards that were prepared at poison center. Fifty-nine cases (88.1%) were treated with Prussian blue oral capsules at a regimen of 100-200 mg per Kg per day. Four patients had died by the 30-day follow-up (mortality rate 6%). No additional follow up information was available. There was a statistically significant association between presence of dermal manifestations and lower mortality rate ($p = 0.009$, $\chi^2 = 6.777$).
Conclusion: Thallium poisoning can occur from ingestion or dermal exposure to thallium salts. Neurological manifestations were the most common followed by dermal and gastrointestinal manifestations. In resource-limited settings, a semiquantitative analytical test was performed successfully. Mortality seems to be low and may be related to a low dose of exposure or to the ready-availability and use of Prussian blue at the Baghdad poison center.

KEYWORDS Emory University, Atlanta, Georgia, USA, Georgia Poison Center, Atlanta, Georgia, USA, Baghdad Poison Control Center, Baghdad, Iraq

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18. Home Brew: A systematic approach to the evaluation of autobrewery syndrome

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Background: Autobrewery syndrome (ABS), also called "gut fermentation syndrome," is believed to be caused by the endogenous production of ethanol by gut microbes that ferment dietary carbohydrates in the gastrointestinal tract. Elevated ethanol levels and symptoms of intoxication can occur after the consumption of carbohydrates, even in the absence of alcohol consumption. Currently, there is no established criteria for identifying or verifying the presence of this syndrome. We present a case that lead to the development of a systematic evaluation of ABS under controlled conditions.
Case Report: A 47-year-old male presented to our toxicology clinic with episodic ataxia, slurred speech and agitation occurring several times a month over two years. Episodes started in the afternoon, lasting through the evening. When the patient awoke the next day, he would be back to baseline and amnestic to the night's events. His wife described episodes as the patient "looking drunk." The patient denied drinking alcohol on any of these occasions. His wife purchased a home breathalyzer machine and noted levels up to 0.2% BAC, coinciding with symptoms. During one episode, a BAC of 0.13% on breathalyzer corresponded with a plasma ethanol obtained within 2 hours of 227 mg/dL. His diet consisted of meat and carbohydrate eaten in moderation and 1-2 glasses of soda daily. He drank 3-4 gallons of milk a week. ABS was suspected but there are no definitive tests. Thus, we developed a 24-hour protocol in a continuously observed environment devoid of ethanol containing products. Our test consisted of an overnight stay with 8-hours of fasting prior to an intense 12-hour carbohydrate challenge. Throughout the challenge, he had hourly breathalyzer tests, plasma ethanol and glucose drawn every 2 hours, and lactate every 4 hours. Clinical intoxication was tested by using the Hack-Impairment Index (HII). During the 713-gram carbohydrate challenge, the breathalyzer peaked at 0.012%. His plasma ethanol remained undetectable.
Case Discussion: ABS is rare and difficult to diagnose. Though surreptitious drinking may be suspected, there have been true cases of ABS with high ethanol concentration measurements in a controlled environment. Saccharomyces cerevisiae and Candida species are among the implicated fermenters. Our patient did not grow yeast on stool cultures. His carbohydrate challenge revealed breathalyzer readings that minimally trended up, but his plasma ethanol remained undetectable. Though our evaluation did not make a definitive diagnosis for ABS, our patient had been doing well on a ketogenic diet, and we feel his results may represent a test of cure.
Conclusion: This case demonstrates a feasible, systematic approach to ABS evaluation in a controlled environment.

KEYWORDS Autobrewery, ethanol, protocol

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19. Seniors and SUDS (Single-Use Detergent Sacs): A review of the National Poison Center Database

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Background: The dangers of exposure to single-use detergent sacs (SUDS), or laundry pods, has been well documented in pediatrics. Elderly adults, especially those with dementia, have an increased risk of unintentional exposure to household products. In addition, comorbidities may predispose the geriatric population to more adverse outcomes. This study aims to describe SUDS exposures in adults greater than 65-years-old.
Methods: This is a review of SUDS exposure in cases 65 years of age or older that were reported to the National Poison Data System (NPDS) from 2012 to 2017. Inclusion criteria were: cases greater than age 65, and exposure substance coded as single-use laundry detergent packs. The distribution of cases was analyzed for demographics, exposure circumstances, management, and outcome.
Results: There was an increasing trend in the number of exposures over time, with an average age of approximately 70-years-old. Females comprised the most exposures (n=485, 63.7%). Unintentional exposures occurred in 93.9% (n=714), and intentional exposures in 4.1% (n=31). Exposure routes were oral, ocular, dermal or rectal. In some cases, there were multiple routes of exposure, typically dermal and ocular. The most common route, ingestion, comprised of 68.2% of the cohort (n=518), of which 94.6% (n=490) were unintentional ingestions. Many of the unintentional ingestions reported confusion and/or mistaking the SUDS for a pill or food. Respiratory depression or arrest occurred in 1% (n=6) of ingestions, however intubation occurred in almost 2% (n=10). Positive x-ray findings, such as infiltrates indicating aspiration, were reported in 2.5% (n=13) of ingestions. Of the overall cohort, 56.5% (n=430) were managed on site, 39.7% (n=302) were treated at a health care facility (HCF), and 3.7% (n=28) were listed as unknown or other. Only 25.5% (n=194) were treated, evaluated and released. In those cases that were admitted, 36 (4.7%) were placed in non-critical care units and 1.7% (n=23) went to critical care units. A large number of cases had no or minor effects (n=606, 79.7%) or moderate effects (n=87, 11.4%). Major effects were seen in 9 cases (1.2%) and death ensued in 4 cases (0.5%). All deaths occurred after an ingestion, with subsequent respiratory compromise and cardiac arrest.
Conclusions: Laundry detergent pack exposures are increasing among the elderly despite industry changes such as opaque packaging, a child resistant lid, and the addition of bittering agents. The number of cases reporting confusion or mental incompetence emphasizes the poisoning dangers that geriatric populations with dementia face. While most cases were managed at home with minimal symptoms, some were admitted to the ICU or died. All deaths had respiratory compromise, without reported lethargy, which preceded cardiac arrest. We feel that these results suggest aspiration and/or respiratory depression as the cause of death. These cases indicate that older adults and their caretakers may be a good target for additional poison prevention education.

KEYWORDS Detergent, Elderly, NPDS

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20. Beware Of What Is In The Mixture: Calculation Error in Compounded GI Cocktail

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Background: It is not uncommon for physicians to prescribe medications that require a pharmacy to compound the prescription to meet a specific patient's needs. One such compounded product is the gastrointestinal (GI) cocktail, which is comprised of a mixture of a liquid antacid, viscous lidocaine, and an anticholinergic agent to treat dyspepsia. We present a case in which a pharmacy-compounded GI cocktail resulted in anticholinergic toxicity.
Case Report: A healthy 65-year-old female experienced abdominal discomfort that was associated with acute gastritis and gastric reflux disease. To help alleviate the discomfort, the patient was prescribed daily omeprazole and ranitidine, as well as a pharmacy- compounded GI cocktail formulated with the following ingredients: atropine, a liquid antacid (Maalox®) and viscous lidocaine with the direction to take 10-15 mL by mouth 3 times a day as needed for stomach pain. The pharmacy compounded the prescription with an attached label stating "Atropine Sulfate 1mg/Lido2%/Antacid" for a total volume of 240 mL and dispensed the bottle to the patient. Shortly after the first dose, the patient started to experience difficulty moving, confusion, hallucinations, and vomiting. Upon EMS arrival, the patient became combative and was given 2mg of lorazepam IV and 4mg of ondansetron. The patient was intubated in the ED; her admitting vitals were HR 104, BP 150/76, RR 19 and Temp 99. On exam, her pupils were mydriatic and sluggish in responsiveness to light. The CT imaging of the head did not indicate any abnormal findings. Even though she was sedated via a propofol drip, the patient was spontaneously moving all of her extremities. It was concluded by the emergency team that the patient was exhibiting signs and symptoms associated with a strong anticholinergic. The patient was treated with supportive care only and was discharged on Hospital Day 3. While the prescription only called for 1 mg of atropine total per bottle (1 mg/240 mL), per compounding logs, 24 mLs of an Atropine Sulfate 1% solution were added to the mixture, totaling 240mg of atropine total per bottle, thus making it a 1 mg/mL concentration. An outside laboratory tested the cocktail to further confirm that the final concentration of atropine dispensed was 1 mg/mL.
Discussion: Atropine, a muscarinic receptor antagonist, when added to GI preparations can decrease GI motility to alleviate gastric distress. As the dose increases, muscarinic antagonistic activity increases which can lead to central and peripheral nervous system toxicity resulting in delirium, agitation, hallucinations, hyperthermia, and cardiac instability. The typical amount of oral atropine, when found in GI preps, is 0.3 – 1.2mg per dose. If taken as directed, it is likely that our patient ingested 10-15 mg per dose.
Conclusion: Pharmacy-compounded GI cocktails containing anticholinergic agents remain a popular compounded prescription, however, if compounded incorrectly can lead to anticholinergic toxicity. Accurately performing pharmaceutical calculations is a "must do" function. To minimize compounding errors, organizations such as the ASHP recommend that all calculations be double-checked prior to dispensing.

KEYWORDS Pharmacy, anticholinergic, toxicity

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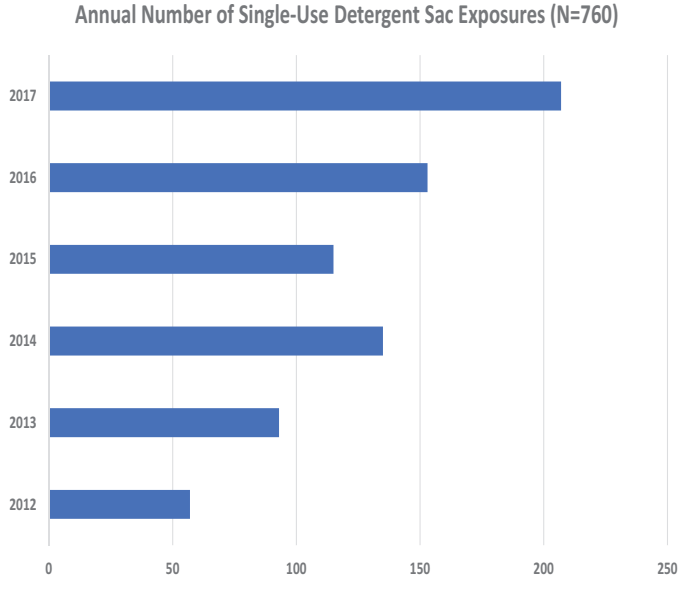

21. Grapefruit Seed Extract; A Case of Oral Burns in an Infant

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Background: Grapefruit seed extract is marketed as a treatment for thrush due to its antifungal properties. As a dietary supplement, it has not been evaluated by the FDA. Below, to our knowledge, is the first case of grapefruit seed extract causing oral burns in a pediatric patient.
Case Report: The mother of a 6 week old girl called the Poison Center as she was using Citricidal grapefruit seed extract to treat thrush. The mom swabbed the concentrated liquid on the inside of the girl's cheeks before realizing that it needed to be diluted in water. Shortly after the exposure, the child started to foam at the mouth with thickened secretions and gagging. Her tongue also appeared to be white and discolored. The child did not show signs of respiratory distress. Mom attempted to rinse the child's mouth but she refused to drink anything. The Poison Center recommended the child be brought to the nearest emergency room (ER) for evaluation. In the ER, the child had obvious oral burns although was able to drink some of her bottle. She was then transferred to the nearest children's hospital. The patient was taken to the operating room for an endoscopy which revealed burns to the mouth and tongue only, no deeper tissue burns. She was observed overnight and in the morning was able to tolerate oral intake without any problem and was subsequently discharged.
Case Discussion: Citricidial grapefruit seed extract is a natural quaternary compound synthesized from the seed and pulp of grapefruit and during the manufacturing process is converted into an extremely potent compound. The suggested usage for any indication is "mix 1-5 drops in a glass of water or juice, 1-3 times daily or as directed. Use diluted only". According to the Safety Data Sheet (SDS), the active ingredients include grapefruit seed extract and Vitamin C which make up 60% of the chemical formula. The additional 40% is glycerin. The product has a pH of 1.5-3.0 In this case, the child developed oral irritation and burns within minutes of having the undiluted product rubbed in her mouth. Although the outcome for this child was favorable, there is the potential for more significant symptoms including esophageal burns, inability to tolerate secretions leading to choking and compromised airway.
Conclusion: This case is important as it raises awareness of the dangers of Citricidial grapefruit seed extract which is not otherwise reported in the literature.

KEYWORDS Grapefruit seed extract, oral burns, Citricidial
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22. Total Bilirubin: Not a Total Waste of Time in Naproxen Toxicity

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Background: Naproxen is a readily accessible non-steroidal anti-inflammatory drug that is frequently encountered in cases of overdose. Unlike acetaminophen, aspirin, and many drugs of abuse, serum naproxen quantification is not routinely available or performed. Naproxen and its primary metabolite O-desmethylnaproxen (ODMN) have been suspected of causing interference with certain bilirubin analyzers.[i] We present a case of a patient with altered mental status suspected of drug overdose with an abnormal bilirubin pattern that was suspicious for analytical interference leading to a diagnosis of naproxen toxicity.
Case report: An 18-year old girl with a history of depression was involved in an argument and found the following morning by her parents with a decreased level of consciousness. The patient was unresponsive to verbal stimuli with a blood pressure of 103/54 mmHg, pulse 101/min, Temp 36.6 °C, Respirations 17/min. Lab studies were significant for a pH of 7.28, PCO2 28 mmHg, WBC 30,000/ml, HCO3 11 mEq/l, Total Bilirubin (T-Bili) 7.9 mg/dL, unconjugated bilirubin 0.0 mg/dL, conjugated bilirubin 0.0 mg/dL, AST 16 U/L, ALT 57 U/L, with albumin and alkaline phosphatase that were within normal limits. T-Bili trended upward peaking on hospital day 2 at 11.4 U/L with no significant change in the other liver function tests. On the next blood draw serum naproxen and bilirubin were measured simultaneously; T-Bili 6.3 mg/dL, naproxen 100 mcg/ml, unconjugated bilirubin 0.0 mg/dL, conjugated bilirubin 0.0 mg/dL. The patient's mental status continuously improved and the metabolic acidosis and leukocytosis resolved. Microbiology studies included cerebrospinal fluid but demonstrated no infectious organisms. On Hospital Day 4, labs studies continued to normalize, naproxen level decreased to 28 mcg/ml and T-Bili to 0.6 mg/dL. The patient returned back to baseline health and admitted purposefully ingesting "three to five", 500 mg naproxen tablets in a self-harm attempt.
Case discussion: Drug testing is infrequently necessary to provide care for the poisoned patient. Serum measurement of the majority of xenobiotics require reference laboratory analysis. The lack of in-hospital quantification results in delays that limit the clinical utility of certain drug leveling. Although quantitative testing may be unnecessary, qualitative testing that could suggest a toxicologic cause of metabolic acidosis and altered mental status may help direct therapy and reduce invasive testing. An isolated number of case reports have suggested that naproxen and its metabolite ODMN may interfere with diazo reaction-based bilirubin measurement leading to spurious elevations. [ii],[iii] Because fractionated assessment does not suffer the same impedance, the conjugated and unconjugated bilirubin levels are accurately reported as undetectable. If T-bili analysis using the diazo reaction proves to reliably cross react with ODMN, then the incongruous pattern of total bilirubin elevation with undetectable conjugated and unconjugated bilirubin may serve as a surrogate biomarker suggestive of naproxen toxicity.
Conclusion: When confronted with the discordant combination of elevated total bilirubin yet undetectable fractionated bilirubin, clinicians should be aware of the possible presence of naproxen.

KEYWORDS Naproxen, Bilirubin, overdose
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23. Hydrogen Sulfide Induced Respiratory Failure Treated with Veno-Venous Extracorporeal Membrane Oxygenation (VV ECMO)

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Background: Hydrogen Sulfide (H2S) is a gas, typically encountered in agriculture or industries such as transport, petroleum manufacturing, etc. While its most notorious presentation is "drop attacks" secondary to decoupling oxidative phosphorylation, it may also cause devastating pulmonary injury. We present a case of a hydrogen sulfide induced respiratory failure that was successfully treated with VV ECMO.
Case Report: At 00:40, six employees were working inside an underground vault, characterized as a cave-like enclosure, 20-50 feet deep. They were removing a section of pipe approximately 10 feet from the bottom of the vault. The pipe unexpectedly shifted, releasing a combination of H2S gas and coal ash slurry. This prompted an immediate evacuation. Two employees were unable to escape the vault and drowned. Four managed to climb out, including our patient, a 31

year-old male. Upon arrival of emergency medical services, patient was unresponsive and hypoxic. He was intubated in the field, and emergently transported to our institution via helicopter. On arrival at our institution, his oxygen saturation was persistently in the 70's, with acidosis and hypercapnia (Table 1) His saturations stabilized sufficiently with prone positioning and manual bagging to allow CT imaging (Image 1), which showed significant pulmonary injury. Cardiothoracic Surgery was consulted for ECMO cannulation for refractory respiratory failure. Patient was admitted to the surgical ICU, with plans for cannulation if he decompensated. At 05:00, he developed a worsening acidosis, hypoxia and hypercapnia. He was placed on VV ECMO at 06:00, with rapid improvement in his respiratory and hemodynamic status. After VV ECMO was initiated, pt exhibited rapid improvement. Bronchoscopy on HD #0 showed erythema and hemorrhage, primarily in the right middle lobe. His hospital course was complicated by pneumonia, eustachian tube dysfunction and bilateral subconjunctival hemorrhages. He was decannulated on HD#4, extubated on HD#5 and successfully discharged. Pt followed with Pulmonary 40 days after his initial presentation, where he had a normal CT chest, and pulmonary function testing that showed only a mild decrease in diffusion capacity. At that time, patient had minimal pulmonary complaints. An OSHA investigation of the incident found 11 violations.

Time	pH	PaCo2	pO2
3:32	7.203	66	71
5:01	7.181	49.4	74.2
6:06	7.430	36.7	493
7:00	7.298	46.7	193
9:47	7.518	30.4	243
14:33	7.482	22.6	225
15:51	7.491	30.5	179

Case Discussion: This case highlights the dangers of hydrogen sulfide exposure. Despite immediately recognizing the danger, two healthy men were physically unable to self-extricate and drowned. Our patient was able to rapidly escape the source of the before losing consciousness. Despite this, he suffered a severe pulmonary injury, which did not respond to conventional ventilation. VV ECMO was required to maintain this patient's respiratory status.
Conclusion: There is no specific antidote for hydrogen sulfide exposure. H2S can have severe complications beyond its decoupling mechanism. As in other toxic exposures, ECMO should be considered to bridge patients to recovery.

KEYWORDS Hydrogen Sulfide, ECMO (Extracorporeal Membrane Oxygenation), Pulmonary

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24. Daily tuna consumption resulting in higher than expected mercury levels

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Background: Mercury is a heavy metal that has no physiologic role in the human body. It exists in three forms: elemental, inorganic, and organic. Bioaccumulation of organic mercury can occur through consumption of fish. Studies in pregnant women who consume fish and cultures with predominantly fish diets have shown significantly higher mercury blood levels than their non- or lower fish-eating counterparts. However, these elevated levels are in the range of 1-10 mcg/L. We report a case of a significantly elevated blood mercury level, likely due to consumption of tuna.
Case Report: A 72 year-old male with no reported past medical history presented to a dermatologist for a resolved rash on his ankles and was concerned for mercury toxicity. He was on a diet for the past year and a half that included daily 6-ounce tuna steaks. His initial blood mercury level was 126.2 mcg/L (normal
Case Discussion: In population studies, the mean concentrations of whole blood and urine mercury are 1-8mcg/L and 4-5mcg/L, respectively. The average blood mercury level in non-fish-eaters is 2mcg/L. Previous studies have shown that blood mercury concentrations increase as the frequency of seafood ingestion increases. Tuna specifically shows a statistically significant positive correlation. In one population-based study, adults who consumed seafood >5 times per month had a mean blood mercury concentration of 1.70 mcg/L. Our patient with a low urine level and a high blood level suggests an organic type of mercury accumulation. His only identifiable source was daily consumption of tuna. Interestingly, his mercury level was orders of magnitude higher than has previously been reported. The downward trend after abstaining from fish intake also suggests lack of ongoing exposure and would lend credence to the source being dietary. It is unclear why his levels were significantly higher than expected. It would be interesting to obtain further sampling of the fish he eats and further details regarding the tuna, as it is possible that others may be at risk if there is fishing from a mercury-contaminated body of water. Fortunately, he remained asymptomatic and his rash was felt to be related to a contact dermatitis and not mercury poisoning.
Conclusions: This case suggests that, while rare, daily tuna consumption may result in significantly elevated levels of blood mercury.

KEYWORDS Mercury poisoning, fish, elevated levels

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25. Cosmetic or Coma? Pentobarbital masquerading as an herbal product

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Background: Pentobarbital (brand name, Nembutal) is a barbiturate that is not commonly used in the United States due to its high abuse potential and controversies related to capital punishment. However, it is still used in veterinary medicine and has gained an online following for use in human suicides and assisted suicides. We report a case of pentobarbital masquerading as an herbal product, a worrisome practice which has not previously been reported.
Case report: A 44 year-old female presented to the emergency department after drinking an unknown amount of pentobarbital in a suicide attempt. She reported ordering Nembutal from a veterinarian in Mexico off the "dark web" as a plan for self-harm. She was somnolent with slurred speech but arousable to voice with a blood pressure of 119/72 mmHg, pulse 91bpm, respirations 12/min, temperature 36.6oC, and oxygen saturation 97% on room air. Her metabolic panel was normal and ethanol, acetaminophen, and salicylate levels were undetectable. Other than sedation, she had a normal physical exam and was admitted to the intensive care unit for monitoring of her mental status. She had an uncomplicated course with improvement in her mental status the following day. Her urine drug screen was positive only for barbiturates, with a pentobarbital confirmation concentration of 2,347 ng/mL (normal
Case Discussion: The patient's clinical effects and laboratory data are consistent with intoxication due to pentobarbital. The product came in two 100mL bottles that were labeled "Natura Skin Cleanser Face Masque" with the listed ingredients being water 26%, olive oil 45%, hazelnut oil 18%, chamomile 9%, and alcohol 2%. It has previously been reported that due to lack of stringent regulations in the supplement industry, pharmaceutical xenobiotics have been found in "herbal products," a fact which is often not known to the buyer. However, in this case, the patient volitionally ordered this product, knowing it was pentobarbital. The herbal packaging was presumably a means to surreptitiously avoid legal detection, similar to the tactics of "bath salts" and "spice" marketing, before there was familiarity with these products.

We tested the residue in the bottles and found that the concentration of pentobarbital was so high that it suppressed the internal standard, precluding concentration quantification, but confirming that this was the source. This deceitful labeling has implications for potential pediatric exploratory ingestions, as well as unexpected clinical effects based on the unassuming label contents.
Conclusions: Clinicians and the public should be aware that pentobarbital may be disguised as an herbal product to avoid legal detection, in a way that is marketed to facilitate suicide.

KEYWORDS Pentobarbital, herbal product, assisted suicide

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26. Lead Toxicity 30 Years After Sustaining a Gunshot Wound

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Background: Morbidity and mortality due to lead toxicity has been reported as far back as the ancient Greek era. Currently the most common cause of lead toxicity in adults is due to industrial exposure. Lead toxicity in adults from retained bullet fragments is a known but rarely reported event. Here we report a case of chronic lead toxicity from a retained bullet fragment obtained 30 years prior to clinical toxicity.
Case Report: A 55-year-old man sent to a tertiary care center from his primary care physician for a hemoglobin (Hb) of 6.8 gram/deciliter (g/dL). The patient describes a one-year decline of worsening weakness, cachexia, numbness and tingling, and joint pain in both the upper and lower extremities. Six months prior to presentation patient was treated for lead concentration of 150 microgram/deciliter (ug/dL) with succimer in another county with some subjective improvements. Treatment was stopped due to cost. Patient presented to our hospital several hundred miles away with severe normocytic anemia (Hb 6.8), worsening weakness, abdominal pain, numbness and tingling in the distal extremities, and new encephalopathy. An in-depth history revealed patient had been shot in the right hip 30 years prior in another country. The patient's exam showed cachexia, extensor muscle weakness and atrophy, blue-grey lines of the gingiva, and encephalopathy. A pelvic xray and subsequent computed tomography (CT) showed extensive metallic fragments embedded in the right femoral head and right acetabulum. An initial whole blood lead concentration of 84 ug/dL was received several days later due to the lead concentration needing to be evaluated at another lab. Due to no access to sodium calcium ethylenediaminetetraacetic acid (EDTA) and significant encephalopathy he was started on intramuscular British anti-lewisite (BAL) and oral succimer. The patient required red blood cell transfusions complicated by him having several antibodies. Orthopedics was consulted for foreign body removal which resulted in full right hip arthroplasty. Over the next few days the patient's encephalopathy and strength improved. He was discharged on succimer. His lowest whole blood lead concentration dropped to 14 ug/dL one month later. Succimer was stopped by his primary care doctor due to cost and patient's clinical improvement. Five months after initial evaluation, patient was found to have increasing weakness, anorexia, and fatigue at a routine orthopedic follow up. His whole blood lead concentration was 45 ug/dL. Oral succimer was restarted with subjective clinical improvement. Patient is actively being followed by the orthopedic and toxicology team.
Discussion: Chronic lead toxicity is rare but serious disease in adults. Significant symptoms can include neuropsychiatric disorders, paresthesias, weakness, cachexia, nephropathy, anemia, hypertension, and death. Delayed clinical symptoms despite likely having elevated lead concentrations such as this case have been reported. Prolonged exposure results in significant deposition of lead into bone matrices. With an excretion half-life of 25 – 30 years from the bone, recurrent toxicity may occur despite chelation efforts. Treatment in these scenarios should be targeted at symptom mitigation rather than whole blood concentrations.
Conclusions: Chronic lead exposure may result in significant symptoms and require prolonged treatment strategies.

KEYWORDS Lead, Gunshot, Chelation

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27. Epidemiology of Snakebites for a Large Poison Center from 2007 - 2017

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Background: There are approximately 9,000 venomous snake bites in the United States every year resulting in approximately 5 deaths per year. The large majority of bites are by native pit vipers with a small percentage due to coral snakes and exotic pets. With a small percentage of hospitals having access to an inpatient toxicology service or dedicated snakebite services, the majority of venomous snakebites receive consultation from poison centers to help guide appropriate care. Here we report the epidemiology of snakebites to a large poison control center over an 11-year period.
Methods: All calls to a large regional poison center for snakebite exposures from January 1st, 2007 through December 31st, 2017 were extracted and analyzed. During calls, specialists in poison information (SPI) recorded data during based on the poison center's standardized snakebite protocols There were no exclusion criteria. A standardized database was established using the REDCap data management system. These data were analyzed using descriptive statistics.
Results: One-thousand seven-hundred ninety-two cases were categorized as snakebites during this time. The majority of calls were due to venomous snakes (N = 1217, 67.9%) followed by non-venomous snakes (N = 351, 19.6%) and unknown (N = 224, 12.5%). The mean patient age was 33 years old (standard deviation =20.1). Most bites occurred in the summer months. Males (N = 1217, 67.9%) represented the majority of bites compared to women (N = 536, 29.9%). More bites occurred in the upper extremities (N = 837, 46.7%) than the lower extremities (N = 725, 40.1%). Bites to the hand occurred in 747 cases (41.6%). Rattlesnakes accounted for the majority of the venomous snakebites (N = 818, 67.2%). Other venomous snakes included copperheads (N = 127, 10.4%), unknown pit vipers (N = 71, 5.8%), water moccasins (N = 15, 1.2%), exotic snakes (N = 9, 0.7%) and coral snakes (N = 2, 0.2%). Local symptoms (N = 1385, 77.3%) were the most common documented symptoms followed by systemic (N = 427, 23.8%) and hematologic (N = 94, 5.3%) symptoms. Hypotension (N = 98, 23.0%), nausea (N = 142, 33.3%), and perioral paresthesias (N = 125, 29.3%) were the most common systemic symptoms. Sixty four percent (N = 782) of recorded venomous snakebites received antivenom. Twenty-six (3.0%) patients received antivenom for a non-venomous snakebite. Three (0.2%) deaths occurred. None of the reported deaths received antivenom.
Conclusions: Our data is consistent with previous data illustrating more bites occur in males. A smaller percentage of venomous snakebites received antivenom than other recently published data. Many of the nonvenomous snakebites were managed at home and did not require medical evaluation. Deaths were rare. Our data is limited by what is documented by the SPI while actively managing a case.

Demographics

Table 1 Demographics (n = 1792).

Characteristic	N	Percent (%)	p-value
Age			
<6 years	251	14.01	
6-10 years	138	7.70	
11-18 years	205	11.44	
19-30 years	289	16.13	
31-60 years	738	41.18	
>60 years	171	9.54	
Missing/Not Documented	*738*	*41.18*	
Sex			
Female	536	29.91	
Male	1217	67.91	
Unknown/Missing	*39*	*2.18*	

Mean age= 33.88 (20.12).

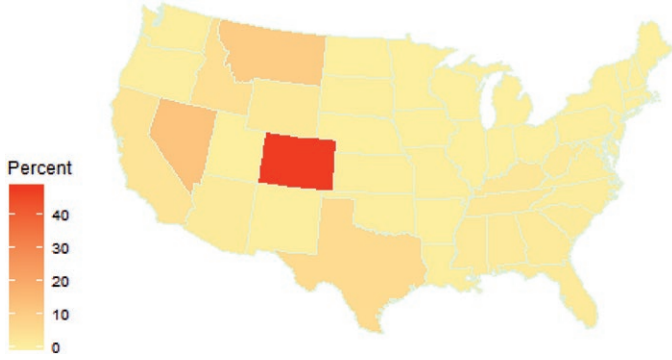

Figure 1 Percentage of Snakebites in the United States (2007-2017).

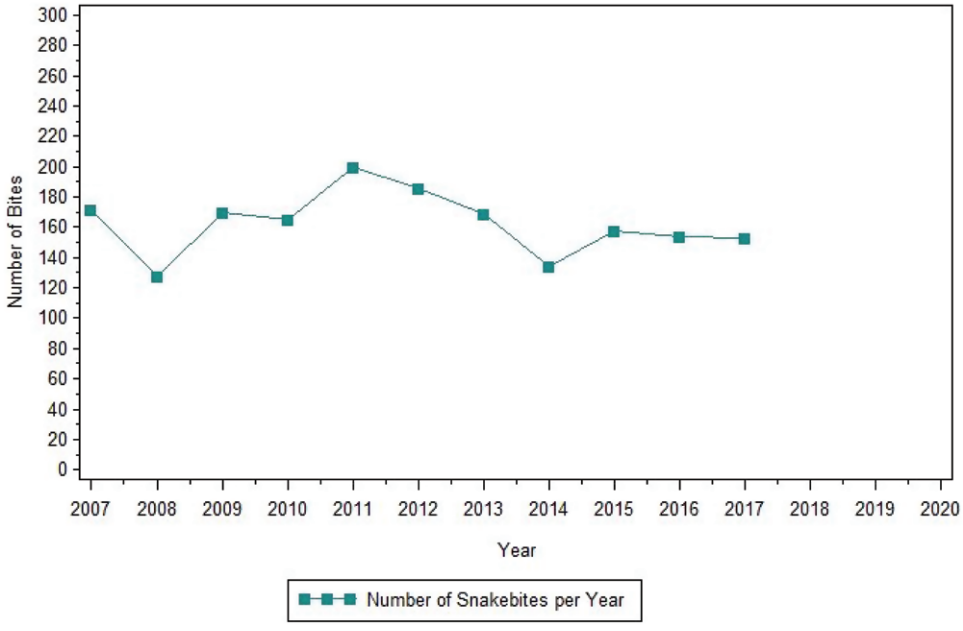

Figure 2 Number of Snakebites Reported by Year (2007-2017).

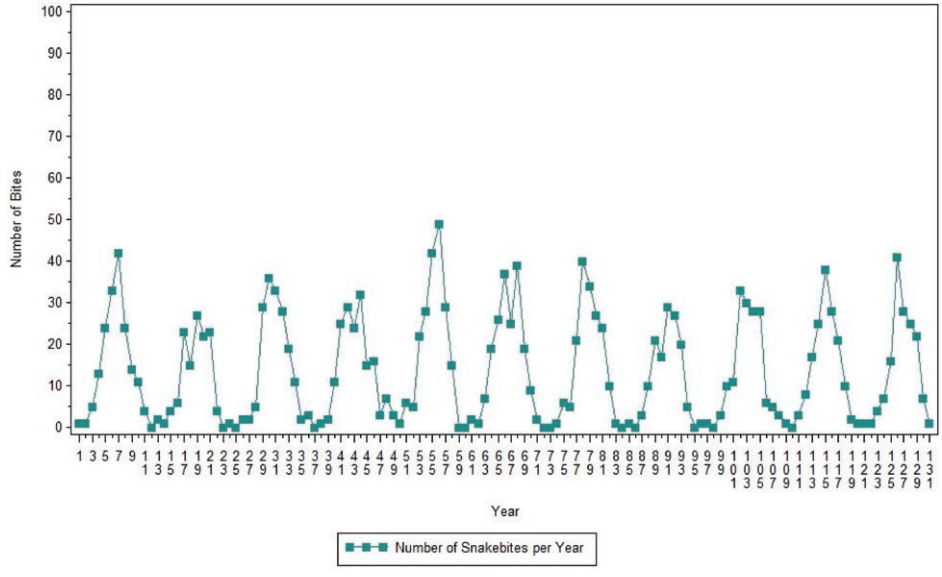

Figure 3 Number of Snakebites Reported by Month & Year (2007-2017).

Patient Information

Table 2 Medical History (n = 405).

Characteristic	N	Percent (%)	p-value
Medical history: Yes			
Hypertension	57	14.07	
Diabetes	14	3.46	
Asthma	17	4.20	
Eczema	0	0.00	
Coronary artery	9	2.22	
Peripheral vascular	0	0.00	
Other (~110)	155	38.27	
Mental health	21	5.19	
Pregnant	6	1.48	
Not documented/missing	*1387*	*77.40*	
Comorbidity total			
0	203	50.12	
1-2	176	9.83	
3 or more	26	1.46	
Allergy history			
No	263	15.10	
Yes	55	3.16	
Not documented/missing	*1474*	*84.62*	
Concomitant medications			
No	1634	91.18	
Yes	103	5.75	

KEYWORDS Snakebites, Envenomation, Epidemiology

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28. Association of Hyperglycemia with Calcium Channel Blocker Exposures Reported to United States Poison Centers

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Background: Calcium channel blocker (CCB) toxicity has been known to lead to significant morbidity and mortality. CCB antagonize the L-type calcium channels, primarily on myocardial and vascular smooth muscle cells. L-type calcium channels of the pancreas can also be blocked, preventing the release of insulin and leading to hyperglycemia. The objective of this study was to determine the association between hyperglycemia in CCB exposures and major effect or death.
Methods: A retrospective chart review was performed of a cohort of CCB exposure cases using data from the National Poison Database System (NPDS) from January 1, 2007 to December 31, 2017. Inclusion criteria included all patients CCB exposure. Exclusion criteria included cases with co-ingestions and patients lost to follow up. Hyperglycemia was defined as glucose >150mg/dL not attributable to pre-existing diabetes. Descriptive statistics were performed.
Results: Over the 10-year study period, there were a total of 49,576 CCB exposures without co-ingestions reported to US poison centers. There were a total of 1,253 exposures that resulted in major effect or death. Of the those exposures, 274 (21.9%) had documented hyperglycemia. Comparatively, 48,323 exposures had no effect, mild effect, or moderate effect and 352 (0.72%) of those exposures had hyperglycemia. Of the total number of CCB exposures, 626 (1.26%) had hyperglycemia, 274 (43.7%) of which resulted in major effect or death. Compared to the 48, 950 cases without hyperglycemia, of which there were 979 (2.00%) cases with major or effect or death, there was relative risk of a major outcome or death in cases with hyperglycemia was 21.
Conclusion: In this large retrospective review of CCB exposures reported to U.S. poison centers, hyperglycemia is positively associated with major effect or death but was not found in a majority of those outcomes. Further study is needed to determine dangerous threshold of hyperglycemia as well as the effect of other variables such as age or intentionality of exposure. In addition, it would be helpful to differentiate risk of specific CCBs.

KEYWORDS CCB, hyperglycemia, effect

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29. Face to Face: Atypical Copperhead Envenomation

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Background: a 73-year-old man who was working in his yard, was bitten on the left side of his face by a Copperhead. Initially, the bite was thought to be dry, but facial and neck swelling led to respiratory compromise requiring endotracheal intubation.
Case Report: initial BP 130/70 mm Hg, pacemaker set at 70 bpm, respiratory rate of 15 and oxygen saturation was 96%. Forty-five minutes later, the patient was diaphoretic, nauseated and became hypotensive with syncope. Dopamine drip was initiated and six vials of CroFab® were administered. While in the CT scanner, patient stated that he felt something in his throat. His face began to swell and soon airway swelling required sedation and intubation. Patient was then transferred to a tertiary level of care. Two more vials of CroFab® were administered and Toxicology service was consulted. Six addition vials of CroFab® were recommended due to airway compromise. Swelling progressed into the left shoulder. Fourteen more vials of CroFab® were administered and maintenance dosing was scheduled. Three hours later, BP dropped to 85 systolic and 500cc Lactated Ringers was infused. At this time, hemoglobin was 11.6 (14.2), hematocrit 35(42), pH 7.22 and bicarb 17. Two amps of bicarb and six more vials of CroFab® were administered. Post infusion the hgb/hct were 13/39 respectively, ph 7.36 and platelets 206 (platelet count had not decreased). Five hours later, the patient developed an intermittent junctional rhythm; however, the patient had an extensive cardiac history of atrial fib/flutter and did have a pacemaker. An additional 14 vials of CroFab® were administered. Labs then revealed hgb/hct 13.6/40.2 and INR was 1.4. Patient was extubated and placed on oxygen. Swelling decreased, vital signs remained stable and all labs remained within normal limits.
Case Discussion: Copperhead bites are prevalent in the South. Snake bites range from dry bites (no envenomation) to severe bites requiring antivenom. Snake venom is a mixture of complex proteins and peptides which can cause local tissue destruction (which may be evidenced by bleb or blister), pain, ecchymosis and edema. Systemic effects include nausea, vomiting dizziness, fasciculation, hypotension, and coagulopathy. Respiratory compromise requiring intubation is rare. CroFab® is indicated for hemodynamic instability, coagulopathies and progression of swelling. True compartment syndrome is rare. Patients with papaya allergy need to be monitored for allergic reaction. Initial dosing is four to six vials in 250 cc NS over an hour. Control must by established as evidenced by reversal of coagulopathy as well as no further progression of swelling or tissue necrosis.
Conclusion: Copperheads envenomation usually causes pain, swelling and some tissue necrosis. Coagulopathy is rare. Endotracheal intubation is rarely required, but in this patient, the massive swelling resulted in airway compromise. This patient received 34 vials of CroFab® and recovered without sequelae.

KEYWORDS Envenomation, Intubation, CroFab®

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30. Exposures to pramipexole and ropinirole reported to US poison control centers

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Background: Ropinirole and pramipexole are selective dopamine agonists used in the treatment of Parkinson disease and restless leg syndrome. Adverse effects in therapeutic use include orthostatic hypotension, somnolence, dyskinesia, nausea, and hallucinations. However little is known about the clinical effects of these agents in overdose. The objective of this study is to characterize exposure to pramipexole and ropinirole reported to US poison control centers.
Methods: This is a retrospective review of single substance exposures to pramipexole or ropinirole reported to the American Association of Poison Control Center's National Poison Data System (NPDS) between January 1, 2005 and December 31, 2014. Only cases followed to a known outcome were included for analysis.
Results: During the study period, there were 5,753 exposures to ropinirole and pramipexole reported to NPDS. There were 3,109 (54.0%) cases followed to a known outcome. Ropinirole accounted for 1641 cases (52.8%) and pramipexole accounted for 1468 cases (47.2%). The median age for the population was 4 years old (mean: 24 years, range: 7 months – 97 years) and females were most commonly exposed (1662, 53.5%). The reasons for exposure reported most frequently reported were unintentional – general (1707, 54.9%), unintentional – therapeutic error (795, 25.6%), intentional – suspected suicide (294, 9.5%) and adverse reaction – drug (151, 4.9%). Most patients were managed in a health care facility (1886, 60.7%), with many patients treated evaluated and released from the emergency department (1265, 67.1%). Medical outcomes are summarized in Table 1. Death was reported following pramipexole exposure in 2 cases, and there were no deaths reported from ropinirole. In pediatric patients 5% of the study population include: drowsiness (1066, 34.3%), vomiting (911, 29.3%), nausea (387, 12.4%), "other" (201, 6.5%), tachycardia (183, 5.9%) and dizziness/vertigo (179, 5.8%). More severe symptoms occurred less frequently following exposure: hypotension (75, 2.5%), hallucinations (52, 1.7%), dystonia (38, 1.2%) respiratory depression (10, 0.3%), coma (7, 0.2%) and seizure(s) (6, 0.2%).
Conclusions: Clinical effects in overdose of the dopamine agonists pramipexole and ropinirole are similar to the adverse effects experienced in therapeutic use. Death and major effects were rare. However, a significant portion of exposures required emergency department management or admission to the hospital. Thus, symptomatic adults may require medical evaluation. Though serious outcomes in pediatric patients were rare, further research is needed to determine which exposures require referral to a healthcare facility.

KEYWORDS Dopamine agonists, exposures, general toxicity

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Table 1 Medical outcome by drug.

Medical Outcome	Ropinirole N	Ropinirole (%)	Pramipexole N	Pramipexole (%)	Total N	Total (%)
Death	0		2	(0.1)	2	(0.1)
Major effect	9	(0.5)	4	(0.3)	13	(0.4)
Minor effect	757	(46.1)	776	(52.9)	1533	(49.3)
Moderate effect	234	(14.3)	192	(13.1)	426	(13.7)
No effect	641	(39.1)	494	(33.7)	1135	(36.5)
Total	1641	(100.0)	1468	(100.0)	3109	(100.0)

31. The ToxIC Sodium Bicarbonate Subregistry: Treatment Recommendations and Clinical Outcomes on Behalf of the ToxIC Investigators Consortium (ToxIC)

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Objectives: There is significant practice variation in the use of sodium bicarbonate to treat sodium channel blockade in the setting of acute poisoning. Given the history of sodium bicarbonate shortages, our objective was to evaluate criteria for initiation and discontinuation, dosing regimens, and complications of sodium bicarbonate therapy.
Methods: This is a prospective cohort study of cases entered in the ToxIC registry where additional data was collected in a subregistry regarding sodium bicarbonate use for sodium channel blockade in a 21 question data collection tool. Cases were collected from April 2015 through December 2017 with 12 ToxIC collaboration groups participating.
Results: There were 44 patients with completed data. The mean age was 33 years with 26 male and 18 female patients. Primary agents listed were amitriptyline, nortriptyline, carbamazepine, bupropion, duloxetine, chlorpheniramine, doxepin, diphenhydramine, flecainide, diethylene glycol, tramadol, cocaine, venlafaxine, dextromethorphan, acetaminophen, tizanidine, alprazolam, lithium, flecainide, sotalol, loperamide, and three unknown agents including one unspecified rodenticide. Medical Toxicology services recommended initiating sodium bicarbonate therapy for a QRS duration greater than 100-120 msec. All services used 150 mEq/L as the concentration of sodium bicarbonate infusion. In the current study 50.0% (22) received both sodium bicarbonate bolus and infusion, 40.9% (18) received boluses only, and 9.1% (4) received infusion only. Duration of infusion ranged from 3-95 hours though two cases had an unknown duration. Mean improvement in QRS duration from initiation of sodium bicarbonate to minimum QRS was 19.7 +/- 23.6 msec. Five patients developed ventricular tachycardia and adjunct therapies given to the patients with ventricular tachycardia included: lipid emulsion (2), hypertonic saline (1), amiodarone (1), cardioversion (1), defibrillation (2), CVVH (1), glucagon (1), magnesium (1), and temporary pacer (1). One patient was defibrillated prior to administration of sodium bicarbonate. Eleven cases (25.0%) had recurrent widening of the QRS after the cessation of sodium bicarbonate (range 106-202 msec occurring up to 24 hours after stopping therapy). Sodium bicarbonate was re-initiated in five cases due to QRS re-widening. Complications occurred in six cases and included hypokalemia, hypernatremia, significant alkalemia, hypocalcemia, mild hypophosphatemia, and QTc prolongation with 63.6% of those classified as electrolyte abnormalities. There were two deaths (one attributed to amitriptyline/clonazepam, unknown agent in other case) and no life support was withdrawn.
Conclusions: On average, sodium bicarbonate resulted in a clinically meaningful reduction in the QRS interval in patients with sodium channel blockade. Complications occurred in 13.6% of patients but the majority were electrolyte abnormalities. Treatment regimens varied, and as seen by QRS re-widening in 25.0% of cases, further studies are needed to determine the optimal dosing and duration of the use of sodium bicarbonate to treat sodium channel blockade in the setting of acute poisoning.

KEYWORDS Sodium Bicarbonate, QRS widening, Sodium Channel Blockade

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32. Systematic Review of Succinylcholine Induced Cases of Malignant Hyperthermia

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Background: Malignant Hyperthermia (MH), a skeletal muscle disease that presents with a hypermetabolic response, can be fatal if untreated. Treatment generally includes immediate dantrolene administration, cooling measures, and supportive care. MH susceptibility is genetically inherited, most commonly with mutations in skeletal muscle ryanodine receptors. The most common triggers of MH are volatile anesthetics; however, succinylcholine alone has also been implicated. Given MH's high fatality rate without treatment, it is recommended that dantrolene be readily available when using potential triggers. Succinylcholine is often used for emergency tracheal intubation; in United States emergency departments, 346,000 endotracheal intubations were performed in 2016. Thus, MH is a potential concern for emergency physicians. A systematic review was conducted to examine the risk of MH from succinylcholine administration alone.

Methods: We searched articles published from inception through 2018 on Medline, Embase, and Cochrane library (Review and Central), using search terms: (succinylcholine OR suxamethonium) AND (malignant hyperthermia OR hyperpyrexia OR dantrolene). Two independent reviewers screened articles for relevance by title and abstract. There was no restriction based on publication language. Articles deemed relevant were read in full to determine if they met inclusion criteria: case reports or series of MH or possible MH in which succinylcholine was administered without any use of inhaled anesthetics (review articles that did not discuss individual cases were excluded).

Results: The initial search identified 1,834 articles; 1,451 were excluded based on review of the abstract alone. The remaining 383 articles were read in full, with 31 articles containing a total of 51 case reports meeting inclusion criteria. Twelve articles (39%) were published after the year 2000 (Figure 1). All case reports either specifically stated there was no family history of adverse anesthetic reactions (35%; n = 18) or family history was not discussed (65%; n = 33). Median patient age was 24 years and 29% (n = 15) were female. Symptom onset after succinylcholine administration was 30 minutes or less in 33% (n = 17) of cases. Using MH clinical grading score criteria, likelihood of each case actually representing MH was calculated, with only 58% (n = 29) scoring as "somewhat greater than likely" or higher (scores for 6 case reports not calculated due to lack of information). The average temperature rise was 2.3 °C and 42.6 °C was the highest temperature reached by a patient. Diagnostic testing for MH susceptibility was performed in 66% (n = 34) of cases and was positive in 88% (n = 29).

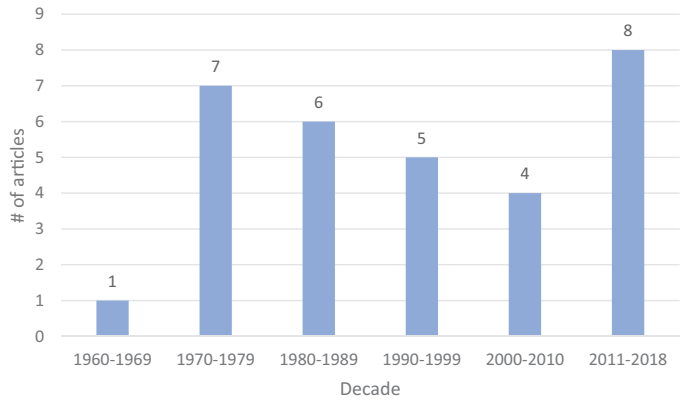

Figure 1 Publication year of analyzed articles.

Table 1 Case report data.

Variable	Mean or % of total	
Age	27 years (n = 51)	Median: 24 years
Gender, female	29% (n = 15)	
Dose of succinylcholine	81.88mg (n = 42)	SD: 50.71; Range: 15-290mg
Time to symptom onset after succinylcholine administration	≤30mins: 33% (n = 17) ≥60mins: 67% (n = 10)	
Location of Exposure	OR: 78% (n = 40) ICU: 2% (n = 1) ED: 4% (n = 2) Other: 16% (n = 8)	
MH Score	0 = Almost never: 2% (n = 1) 3-9 = Unlikely: 0% (n = 0) 10-19 = Somewhat less than likely: 29% (n = 15) 20-34 = Somewhat greater than likely: 22% (n = 11) 35-49 = Very likely: 24% (n = 12) 50+ = Almost certain: 12% (n = 6)	
Temperature rise	2.3 °C (n = 16)	SD: 1.7; Range: 0-4.4 °C
Temperature max	39 °C (n = 30)	SD: 1.74; Range: 35.7-42.6 °C
Muscle rigidity present	81% (n = 41)	
Cardiac Arrest	12% (n = 6)	
CK value	23,242.2 U/L (n = 25)	SD: 21,140.15; Range: 18-60,530
Dantrolene administered	22% (n = 11)	88% (n = 45) given other therapies (ex: cooling, hyperventilation, IV fluids, other medications)
Died	8% (n = 4)	
Diagnostic testing performed	66% (n = 34)	Positive: 88% (n = 29) Equivocal or Inconclusive: 9% (n = 3) Negative: 3% (n = 1)

Conclusions: Although succinylcholine is considered a MH trigger, a systematic review of case reports of succinylcholine induced MH identified just 51 cases, of which only 29 scored as "somewhat greater than likely" or above to represent a case of MH. While conclusions cannot be drawn as to the strength of the link between succinylcholine and MH from this review alone, the lack of cases linked to succinylcholine as the sole trigger in the last few decades may suggest this risk is less severe than previously believed. This could have implications for dantrolene stocking in emergency departments.

KEYWORDS Succinylcholine, malignant hyperthermia, emergency intubation

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33. Point-of-care Ultrasound to Enhance the Assessment of Crotaline Envenomation

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Background: Evaluation of a patient after crotaline envenomation requires assessment of local and systemic signs and symptoms in conjunction with laboratory data. With variability of envenomation severity, therapeutic decision-making is ideally founded on objective

indices. Point-of-care ultrasound (POCUS) has been used to describe sonographic findings of crotaline envenomation but more data is needed to evaluate the utility of POCUS. In this study we used POCUS to evaluate the depth of soft tissue injury, the involvement of the fascia and the most proximal edge of envenomation.
Methods: This was a prospective observational study evaluating the sonographic characteristics of crotaline envenomation. Inclusion criteria were patients older than six years with history and physical examination consistent with crotaline envenomation. The copperhead species (Agkistrodon contortrix) are responsible for the majority of envenomations in our region. This was a convenience sample based on the incidence of snake bites and investigator availability. After informed consent, an ultrasound examination was performed. Images were taken directly over the bite site, at the same location on the contralateral limb (for comparison) and at the proximal edge of envenomation. The edge of envenomation was determined by the proximal line demarcated by pain and edema on palpation. Image acquisition was performed in both longitudinal and transverse planes using a 13-6 MHz linear array transducer on initial assessment and when feasible, prior to discharge. The POCUS endpoints included evaluation of the subcutaneous findings of crotaline envenomation which included: the depth of edema, muscle, fascia or tendon involvement at the bite site, and at the proximal edge of envenomation. Demographic, clinical and laboratory data were also collected.
Results: In the last 29 months, eight patients were enrolled. Of these, 75% were male and the median age was 45 (range 21-71) years. All cases were treated with a median of four vials (4-12) of Crotalidae polyvalent immune fab (AV). One case was a Western diamondback (Crotalus atrox), the others were copperheads (Agkistrodon contortrix). Sixty-three percent of the bites involved digits. The median hours post-envenomation until initial POCUS was 6.5 hours (range 3-21) and only three had a documented discharge POCUS. All of the patients had initial subcutaneous cobblestoning on POCUS at the bite site. All digit envenomations had edema in the tendon or tendon sheath. Of the non-digit envenomations, there was no edema below the fascia or muscle. Fifty-percent of patients had POCUS findings of cobblestoning proximal to the edge of envenomation with a median distance of 3 cm (range 0-13). The nadir of hematological indices was unremarkable. One patient underwent hand debridement and small tissue flap five weeks post-envenomation for a non-healing wound.
Conclusion: On envenomated extremities, subcutaneous edema was universally present. Digit envenomations showed edema of the tendon and tendon sheath with none needing acute surgical intervention. Edema was not observed below the fascia or within the muscle. POCUS identified signs of envenomation proximal to the physical exam findings, however, the clinical significance of this is unknown.

KEYWORDS Envenomation, Ultrasound, Crotaline

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34. National Poison Data System Fatalities Involving Pediatric Exposures to Single Ingredient Acetaminophen, 2007-2017

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Background: Acetaminophen is the most commonly used antipyretic and analgesic therapy for children. When used as directed, acetaminophen is generally recognized as safe and effective. In overdoses acute liver failure and death can occur. Due to its widespread use and the risk of serious adverse events, safety monitoring efforts for acetaminophen are ongoing.
Methods: The National Poison Data System (NPDS) was searched for pediatric (<12 years old) exposures involving single ingredient acetaminophen products reported between 2007 and 2017. Fatality abstracts for all deaths were obtained. Each fatality is assigned a relative contribution to fatality (RCF) based on all substances reported (undoubtedly responsible, probably responsible, contributory, probably not responsible, clearly not responsible and unknown). When cases are deemed to have a RCF of at least contributory, all substances are given an individual cause rank that describes the contribution of each substance to the fatality. Details of each fatality are described.
Results: A total of 415,873 pediatric exposures to single ingredient acetaminophen products were reported to NPDS, of which eight cases (0.002%) were fatalities. The fatalities involved the following reasons for exposure: unintentional-general (n = 3), unknown (n = 2), adverse reaction (n = 1), intentional-abuse (n = 1) and unintentional-unknown (n = 1). There were four females and four males with a median age of 3.0 years (range 25 days to 11 years). Two cases reported acetaminophen as the only substance involved in the exposure (undoubtedly responsible n = 1; clearly not responsible n = 1). The one fatality that was deemed undoubtedly responsible occurred in a 7 year old male who took an unknown amount of acetaminophen several days prior to presentation while visiting a friend. In the six polysubstance fatalities, the substances were determined to be undoubtedly responsible in two, probably responsible in one, and clearly not responsible in one; two were unknown. In the three at least contributory fatalities, acetaminophen was determined to be the primary substance in one of the cases (probably responsible in 5 year old female). In another case (18 month female), hydrocodone was the primary substance, followed by acetaminophen, alprazolam, and dihydrocodeine. The third case occurred in an 11 year old male and involved paint (aerosol), cigarette lighter fluid, ethanol, oxycodone, oxymorphone and midazolam with acetaminophen's contribution to the fatality ranked fourth (Table 1). History of ingestion was unclear in all eight fatalities.

Table 1 Contribution of Substances to Fatalities.

Case	Age	Year	Relative Contribution to Fatality (RCF)	Reason	Acetaminophen cause rank	Other substances (cause rank)
Single Substance	7 years	2007	Undoubtedly Responsible	Unintentional-General	1	N/A
	8 months	2007	Clearly Not Responsible	Unintentional-Unknown	N/A	N/A
Polysubstance	18 months	2013	Undoubtedly Responsible	Unknown Reason	2	Hydrocodone (1), Alprazolam (3), Dihydrocodeine (4)
	11 years	2011	Undoubtedly Responsible	Intentional-Abuse	4	Paint (aerosol) (1), Cigarette lighter fluid (2), Ethanol (3), Oxycodone (5), Oxymorphone (6), Midazolam (7)
	5 years	2014	Probably Responsible	Unknown Reason	1	"Cold medicines"*
	7 years	2013	Clearly Not Responsible	Unintentional-General	N/A	Valproic acid†
	25 days	2011	Unknown	Adverse Reaction-Drug	N/A	Lidocaine Hydrochloride (1%)†
	3 years	2016	Unknown	Unintentional-General	N/A	Xanax, Klonopin, Beta-blocking agents, Atenolol, Amlodipine, Elavil, Vistaril, Sertraline, Pravastatin, Triamterene, Loratadine and related agents, Pristiq, Tenormin†

*Acetaminophen was the only coded substance but additional substances were identified in the text. Only coded medications are given a cause rank.
†Only cases with a RCF of at least contributory are given cause ranks for substances.

Conclusion: Pediatric fatalities involving single ingredient acetaminophen are reported infrequently (0.002% of all reported acetaminophen exposures). Of the eight fatalities reported to NPDS involving acetaminophen, the coded substances were determined to be at least contributory in four cases, three of which involved substances other than acetaminophen. This report highlights that deaths related to single ingredient acetaminophen in children are very uncommon and often include multiple substances or other contributors with unclear history of ingestion.

KEYWORDS Acetaminophen, Fatalities, Pediatric

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35. Review of recommendations for use of intravenous sodium bicarbonate to treat salicylate poisoning

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Objectives: The treatment of salicylate poisoning remains challenging. Published authorities offer various recommendations regarding the formulation and concentration of sodium bicarbonate (NaHCO3) treatment. There are no randomized trials examining patient outcomes using different NaHCO3 infusion solutions for serum and urine alkalinization in the setting of salicylate poisoning. Our objective was to examine the disparate recommendations for NaHCO3 therapy for the treatment of salicylate poisoning, and our goal was to determine the most sensible treatment based on current published consensus, ease of administration, and principles of intravenous pharmacy.
Methods: A MEDLINE literature search was performed using the key words and terms: ["sodium bicarbonate" OR "alkalinization,"] AND ["salicylate toxicity" OR "salicylate overdose" OR "salicylate poisoning."] Our search strategy was limited to contemporary human articles, published from January 1, 2008 to December 31, 2018, written in English. Additionally, the most recent editions of commonly used emergency medicine and medical toxicology textbooks were assessed. A total of seven articles were found, and nine key textbook chapters were identified, delineating specific NaHCO3 regimens for the treatment of salicylate overdose.
Results: A majority of the published recommendations, including six textbooks and four articles, endorsed a simple method of adding three ampules (150 mL total) of 8.4% NaHCO3 (150 mEq total) into 850 mL of 5% dextrose (D5W) to produce an isotonic but hyperosmolar source of NaHCO3. Two textbooks describe adding 150 mEq of NaHCO3 into 1L of D5W (resulting in a more dilute, hypotonic solution). One resource recommended placing 100 mEq of NaHCO3 into 1L D5W and 0.25% normal saline. A 2015 position statement written by the American College of Medical Toxicology described using a solution of 1L D5W containing three 50 mL ampules of 7.5% or 8.4% sodium bicarbonate. Two pediatric references illustrated dosing by weight, recommending 1-2 mEq/kg NaHCO3 in 1L D5W, but did not specify solution composition. Lastly, one pediatric case report proposed a modified NaHCO3 regimen which added 500 mL D5W to 40 mEq NaHCO3. Generally, recommendations were consistent for potassium repletion prior to or during NaHCO3 infusion. The rate of sodium bicarbonate infusion for alkalinization was generally suggested as either 1.5-2 times the maintenance rate in adults or 2-3 mL/kg/hr in children, with adjustments made based on fluid tolerance, renal function, and urine output to prevent pulmonary edema.
Conclusions: Intravenous infusion osmolarity is an important factor in vascular tolerance, and tonicity is an important factor in in vivo fluid compartmentalization. Dextrose supplementation is considered important in the prevention of neuroglycopenia due to salicylate poisoning. Of the sixteen resources reviewed, the most commonly recommended NaHCO3 formulation was to add 150 mEq of NaHCO3 (3 ampules of 8.4%) into 850 mL of D5W. We support this recommendation as this solution is simple, isotonic, within acceptable osmolarity for pediatric intravenous infusion, provides dextrose, provides an ample dose of alkaline reserve, and supported by the most authors. There remain disparate instructions from different authorities, so further scientific review and research evaluating the optimum NaHCO3 solution for achieving alkalinization in salicylate toxicity is warranted.

KEYWORDS Salicylate Poisoning, Alkalinization, Sodium Bicarbonate

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36. Possible Buprenorphine Toxicity in a Breastfeeding Neonate

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Background: Buprenorphine is an acceptable alternative for medication-assisted treatment (MAT) of opioid use disorder during pregnancy and is associated with lower risk of neonatal abstinence syndrome (NAS). Buprenorphine and the metabolite nor-buprenorphine are transmitted via breastmilk but to our knowledge have not previously been associated with adverse events in breastfeeding neonates.
Case Presentation: A full term, previously healthy 2-week-old male was brought to an Emergency Department (ED) for somnolence and decreased feeding. Upon arrival he was lethargic with pinpoint pupils and initial Glasgow Coma Score 5. His blood glucose was 79mg/dL. He received Naloxone 0.15mg twice after which he cried and had improved tone. His mother was undergoing MAT and had been taking buprenorphine 8mg twice daily for several months. She denied illicit substance use. Patient was exclusively breastfeeding. Neither the patient nor his mother was taking other medications. The patient was transferred to our hospital where he had negative infectious studies and extended toxicologic screen. He had recurrent hypoglycemia requiring intravenous dextrose. He again received Naloxone 0.15mg for pinpoint pupils, bradycardia and lethargy, with some improvement noted by the treating provider. Urine buprenorphine metabolites were sent approximately 16 hours after symptom onset (Table 1). Breastfeeding was stopped. Over the next 3-5 days he developed mild NAS however morphine rescue was not required.
Discussion: Buprenorphine is metabolized to active metabolites norbuprenorphine, norbuprenorphine-glucuronide and buprenorphine-glucuronide. Nor-buprenorphine is particularly associated with respiratory depression in an animal model. A previous study of 7 asymptomatic breastfeeding infants of mothers on therapeutic buprenorphine found urine buprenorphine. Possible explanations for this are variability in maternal or neonatal CYP3A4 activity, or impaired neonatal P-glycoprotein and glucuronidation activity. Future work could correlate presence of metabolites and symptoms in neonates. Providers should be educated regarding the potential for buprenorphine to have adverse effects in breastfeeding neonates. Breastfeeding mothers using buprenorphine should remain vigilant for signs of opioid intoxication in infants.

Buprenorphine	< 2ng/mL
Norbuprenorphine	**13** (< 2ng/mL)
Buprenorphine glucuronide	< 5ng/mL
Norbuprenorphine glucuronide	**11** (< 5ng/mL)
Naloxone	< 100

KEYWORDS Buprenorphine, breastfeeding, neonate

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37. Evaluating Medical Outcomes of Exposures to Propellants Among All Age Groups from 2006 to 2017

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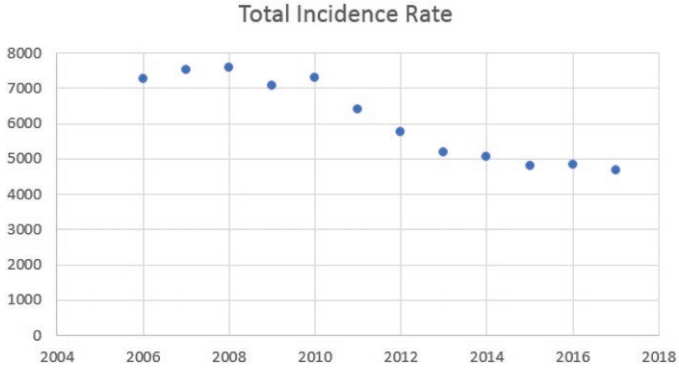
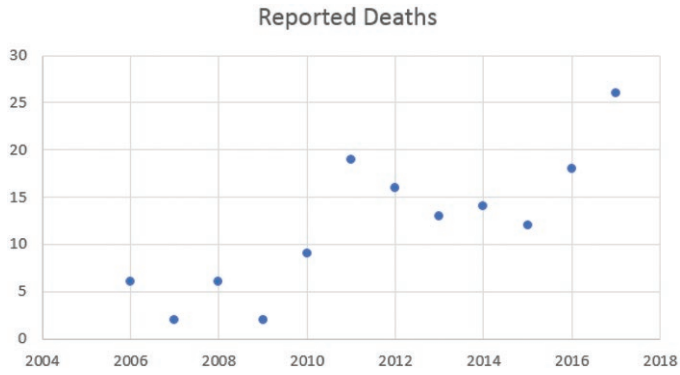
Background: The objective of this study was to evaluate recent trends in fluorinated hydrocarbon and propellant exposures reported to United States (US) poison centers utilizing the National Poison Data System (NPDS). A previous study showed that the overall prevalence of inhalant abuse cases reported to US poison centers decreased 33% from 1993 to 2008, but cases that involved propellants had a significant increase after the year 2000. [1] The purpose of this data review was to evaluate all propellant exposures to see if this trend has continued and the implications this has had on medical outcome.
Methods: The National Poison Data System (NPDS) compiles data in real time provided by 55 US poison centers. Cases of "Freon and Other Propellants" exposures from 2006 to 2017 were evaluated to determine if these exposures are on the rise or decreasing in occurrence. We also evaluated the data to determine the severity of medical outcomes experienced by reported patients and the number of patients who required treatment in a healthcare facility (HCF).
Results: There were a total of 73,372 propellant exposures reported to poison centers from 2006 to 2017. The number of exposures decreased by 36% over the course of 12 years (Figure 1), but the number of deaths slowly increased over time and were 4.3 times higher in 2017 than in 2006 (Figure 2). The increase in deaths could be related to a phenomenon known as "sudden sniffing death" that causes fatal cardiac dysrhythmia. NPDS data includes those patients who seek medical treatment at a HCF and exposures in patients who do not require medical treatment. This data is essential in evaluating exposures that are managed outside of a healthcare facility. According to NPDS data, unintentional exposures have decreased by 42.7%, and therefore the number of patients managed outside of a HCF has decreased as well. Conversely, intentional, primarily abuse or suicide, exposures have stayed the same, therefore the number of patients treated in a HCF has stayed constant over the last 12 years. Inhalant exposures occur more commonly in patients >19 years of age. When specifically looking at this population there was a peak in inhalant exposures in 2010 that has steadily decreased by 25.7% over time.
Conclusions: Even though we have seen a decrease in inhalant exposures over the last 12 years, deaths related to these exposure has continued to rise, therefore these exposures should raise some awareness amongst healthcare professionals evaluating and treating these patients.





KEYWORDS Poison center, propellants, National Poison Data System (NPDS)

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38. Metformin Toxicity Cases Stratified by Lactic Acid level: Management and Outcome

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Background: Metformin is a biguanide that remains a safe and effective cornerstone of diabetes treatment despite well-documented instances of lactic acidosis during chronic therapy and after overdose. This study looks at how stratifying lactic acid (LA) level in our reported cases can identify risk and the need for treatment.
Method: Our PC metformin cases occurring during the years 2008 and ending April 2019 were included if metformin was either the only substance listed, if listed as the first (primary) substance or if a fatality case. Non-fatality cases were included if at least one lactic acid level was reported. When available the following secondary variables were collected: lowest venous or arterial pH, bicarbonate, total carbon dioxide, and highest serum creatinine, Managements recorded were the use of vasopressors, IV alkalinization, mechanical ventilation, and any method of excorporeal treatment such as dialysis. Outcome also included the time to resolve most metabolic abnormalities in survival cases. Other demographics were the estimated dose of metformin, reason for exposure, age and gender. Cases were stratified into the following six categories of LA level (mmol/L): [6 ≤ 14] and [>14]. These intervals were chosen to provide a similar distribution of cases. Two cases with LA level above the limit of lab detection were rounded to 20mmol/L.
Results: Metformin exposure cases were 2355 of which 765 (33%) were potentially evaluated at a health care facility. Of these, 347 (45%) were metformin as single or primary (first) substance listed and 126 (36%) had a LA level recorded (except in 2 fatalities). Table 1 compares baseline characteristics and outcomes in the six categories of LA level. A suspected suicidal intent was noted in 92 cases including 5 out of 11 fatalities. Of the remaining 6 fatalities, 4 were judged as adverse drug reactions and 2 were of unknown reason. Severe outcomes were absent when LA levels were 6 mmol/L. Vasopressors, bicarbonate, mechanical ventilation, excorporeal treatment were rarely used in cases of LA 14 mmol/L. Generally, duration of metabolic abnormalities in survival cases was usually short (1-4 days) but was probably influenced by excorporeal elimination strategies such as dialysis. Excorporeal membrane oxygenation (ECMO) was used in one case with survival.
Conclusion: Metformin lactic acidosis after overdose in this poison center study was usually the result of a suicidal intention. Most cases required minimal intervention when the LA level <6 mmol/L. Recovery was usually rapid but fatalities in this study occurred when the LA level

Table 1 128 Metformin case characteristics by intervals of reported lactate.

	<2	2 < 3	3 < 4	4 ≤ 6	>6 ≤ 14	> 14
N (126 with lactic acid levels)	21	18	21	21	22	23
Fatalities (2 with no lactate reported)	0	0	0	0	3	6
Average Age (years)	31	35	36	40	56	56
Female : Male ratio	2 : 1	2 : 1	1.6 : 1	1 : 1	0.8 : 1	1.3 : 1
Average gram Dose (n)	14 (16)	19 (13)	15 (16)	22 (17)	36 (10)	29 (3)
Average maximum Creatinine (n)	1.1 (7)	1 (5)	1.3 (5)	1 (9)	5.7 (13)	3.9 (19)
Average serum pH (n)	X	X	7.35 (7)	7.33 (14)	7.23 (21)	6.89 (21)
Average serum bicarbonate (n)	X	X	22.5 (11)	19 (8)	14.5 (18)	7.6 (21)
Excorporeal Treatment	0	0	0	1	6	21
Vasopressors	0	0	0	0	7	12
Mechanical Ventilation	0	0	0	1	7	16
Correction of acidemia with IV bicarbonate	0	0	0	2	9	20
Median duration (days) of metabolic abnormalities or need for treatment in survival cases (n)	X	1 (12)	1 (20)	1 (20)	2 (18)	4 (16)

n = the number of cases with available information; X = insufficient data collected.

exceeded 6 mmol/L and could occur despite the use of dialysis, vasopressors and mechanical ventilation. Severe acidemia was often managed with sodium bicarbonate but dialysis was usually used when a LA level exceeded 6-14 mmol/L.

KEYWORDS Metformin, Lactic acidosis, Risk stratification

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39. Successful treatment of a potentially fatal 5-Fluorouracil overdose.

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Background: 5-Fluorouracil (5FU), a commonly used antineoplastic agent, may have devastating adverse effects such as thrombocytopenia, neutropenia, myelosuppression, mucositis, cardiac arrhythmia, cardiac arrest, acute cerebellar syndrome, and sudden death in overdose. We present a case of a potentially fatal 5FU overdose successfully treated with uridine triacetate (UTA).
Case report: A 58-year-old male with a past medical history of Stage IIIB (cT3, cN1a, CM0) status post resection adenocarcinoma of the descending colon, non-insulin-dependent diabetes mellitus, hypertension, benign prostatic hypertrophy, cerebrovascular accident, splenomegaly, iron deficiency anemia, Wegener'sgranulomatosis, depression, agranulocytosis, leukocytosis, sleep apnea, and morbid obesity presented to the emergency department (ED) following a 5FU overdose. At a cancer center, for his 8th cycle of chemotherapy, the patient received 900mg of leucovorin, 900 mg of 5FU, 16 mg of ondansetron, 20 mg of dexamethasone intravenously (IV), and had an infusion pump set up with 5495 mg of 5FU to be delivered over 46 hours. One hour after being discharged from the cancer center, the patient noticed that the pump was beeping, he returned to the cancer center, where it was discovered that his pump delivered the full dose over one hour (for a total of 6395 mg of 5FU).He complained of having "foggy" mentation as well as transient numbness over his left arm and face which completely resolved after a brief period of time. On examination, vital signs were within normal limits. With the exception of binocular horizontal nystagmus, the rest of the physical examination was unremarkable. The patient was admitted to the floor for treatment using UTA, and supportive treatment included IV fluids, ondansetron, pantoprazole, and misoprostol. During the patient's hospital stay, he developed leukopenia [WBCs nadir 1.3] for which he received filgrastim and thrombocytopenia [Platelets nadir 62] for which he received romiplostim. He also required vancomycin for a Clostridiumdifficile infection. Follow up with the patient's primary care provider 1 month following discharge revealed full recovery with no residual effects.
Case discussion: 5FU is commonly used to treat colorectal cancer, but carries the risk of having a narrow therapeutic dose range, further complicated by great individual variability in toxicity and efficacy. Deaths have been reported from cases receiving doses ranging from 1000 mg to 27200 mg. UTA, a pyrimidine analog, is a specific antidote for 5FU indicated in overdose or unusually severe adverse effects. The recommended dose is 10 g orally every six hours for 20 doses, starting as soon as possible within 96 hours. This regimen has a reported 30 day survival rate of 96%. At the time of writing this case report, there is only one supplier of UTA in the United States. It is important for any center using 5FU to familiarize themselves with the process of obtaining UTA given the time sensitivity of the matter.
Conclusion: Early administration of UTA and control of adverse effects using supportive treatment are key in managing 5FU overdose.

KEYWORDS Fluorouracil, Pump, uridine triacetate

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40. Title: Titanium - Tolerable or Terrible?

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Background: Although numerous studies on titanium (Ti) toxicity exist, titanium's systemic effects and effects on individual organs, toxic dose range, and genotoxic activity have not been well elucidated. We present a case with severe elevation in serum Ti levels in a patient with a prosthetic Ti alloy hip.
Methods: This is a case report of a 59-year-old female with a past medical history of Poland syndrome, depression, migraines, anemia and GERD, who had her primary hip replacement surgery 4 years prior to presentation. Preoperative labs were drawn for cobalt and chromium which were within normal limits. The prosthetic hip was a Ti alloy (DePuy Pinnacle Cup) that was discovered to have failed and required removal. Intra-operatively, there was excessive particulate metal debris in the tissue surrounding the prosthetic joint. Post-operatively serum Ti levels were ordered and her serum Ti was found to be 412 mcg/L, 271 mcg/L, 224 mcg/L, 246 mcg/L, and 327 mcg/L on 9/15/18, 10/5/18, 10/31/18, 11/1/18 and 2/13/2019 respectively. The patient did not have any systemic symptoms pre- or post-operatively, but developed septic arthritis following surgery.
Discussion: Titanium is a naturally occurring metal that is used as an alloy in surgical implants due to its high biocompatibility. Reports on Ti toxicity associated with prosthetic implants are very rare. One study reported that powdered Ti was more cytotoxic to osteoblast-like cells than bulk metal. This was attributed to the release of a much higher ion concentration into the media, which perhaps explain the extremely high levels found in this patient. Ti implants may corrode, releasing ions, bind to cell-membrane proteins, and present as neoantigens, inducing autoimmune reactions and allergies. When bound to intracellular proteins, it may alter normal cell physiology, and promote cytokine release from macrophages. Furthermore, Ti dioxide is implicated in causing adverse effects via induction of oxidative stress resulting in cell damage, genotoxicity, inflammation, and immune response.

Conclusion: To our knowledge, these are the highest serum Ti levels to be reported in a patient. More research is warranted to address guidelines for laboratory follow up or requirement for chelation therapy for patients with elevated serum Ti levels.

KEYWORDS Titanium, Prosthetic, implants

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41. Nicotine content from cigarettes submerged in soda

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Background: Cigarettes and other tobacco products may be extinguished by submersion in liquids in beverage cans or bottles. Cases of nicotine poisoning in children have been reported following ingestion of such liquids. The aim of this study is to analyze variability in nicotine concentration over time in liquids in which cigarettes have been submerged, as well as variability with the number of cigarettes submerged.

Methods: One unsmoked cigarette (Newport ®) was immersed in a soda can containing 55 milliliters (mL) of a cola beverage. Samples of the liquid from the mixture were obtained at 1, 2, 6, 12, and 24 hours, and 1 week. Three separate samples were obtained per chosen time period, and as a control, we used soda without a cigarette. A total of 21 samples were studied.To assess for nicotine concentration variation with increased number of cigarettes, 4 cans containing 100 mL of a cola beverage, each having either 1,2,3 or 4 full, unsmoked cigarette(s) immersed in them were prepared, and samples of the liquid from the mixture were obtained after 6 hours of submersion. This was repeated 3 times for a total of 12 samples. In both experiments, the samples were analyzed using liquid chromatography-mass spectrometry (LCMS). Data were analyzed using linear regression in SPSS software.

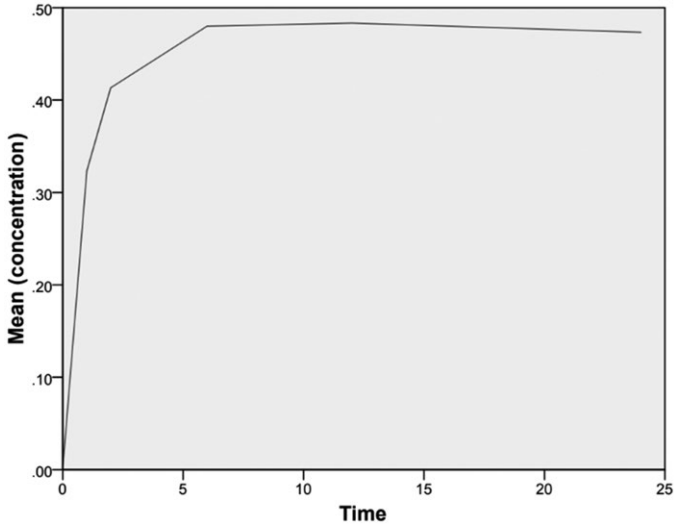
Table 1 Nicotine Concentration (mg/ml) and Time.

SODA			55 mL	Nicotine/Soda	Absolute Level
Key	Time	Cigarette	Soda	mg/ml	mg x 55 mL
1A: control	0 hours	no cigarette	soda alone	0.00	0.00 mg
1B: control	0 hours	no cigarette	soda alone	0.00	0.00 mg
1C: control	0 hours	no cigarette	soda alone	0.00	0.00 mg
7A	1 hour	1 cigarette	soda	0.23	12.6 mg
7B	1 hour	1 cigarette	soda	0.21	11.5 mg
7C	1 hour	1 cigarette	soda	0.53	29.2 mg
3A	2 hours	1 cigarette	soda	0.45	24.8 mg
3B	2 hours	1 cigarette	soda	0.40	22.0 mg
3C	2 hours	1 cigarette	soda	0.39	21.4 mg
6A	6 hours	1 cigarette	soda	0.41	22.5 mg
6B	6 hours	1 cigarette	soda	0.51	28.1 mg
6C	6 hours	1 cigarette	soda	0.52	28.6 mg
5A	12 hours	1 cigarette	soda	0.48	26.4 mg
5B	12 hours	1 cigarette	soda	0.50	27.5 mg
5C	12 hours	1 cigarette	soda	0.47	25.8 mg
4A	24 hours	1 cigarette	soda	0.46	25.3 mg
4B	24 hours	1 cigarette	soda	0.49	26.9 mg
4C	24 hours	1 cigarette	soda	0.47	25.85 mg
2A	1 week	1 cigarette	soda	0.48	26.40 mg
2B	1 week	1 cigarette	soda	0.45	24.75 mg
2C	1 week	1 cigarette	soda	0.55	27.50 mg

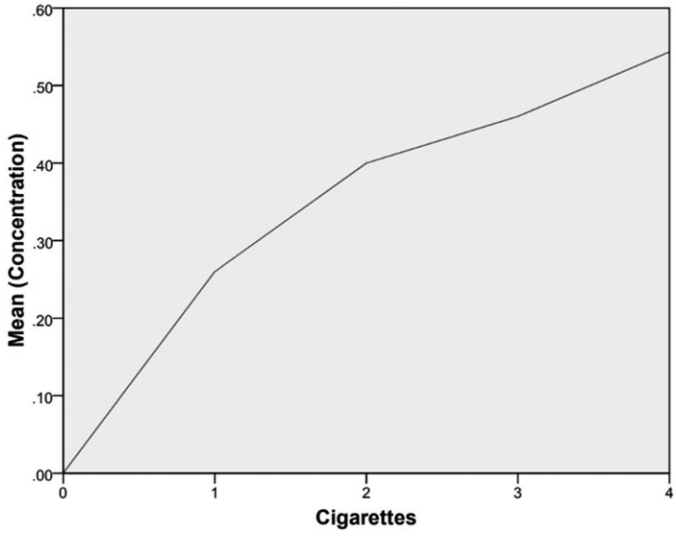
Results: The mean concentration of nicotine measured over the course of 6 hours from one full cigarette in 55 mL of a cola beverage was found to be 0.48 mg/mL. Nicotine concentrations steadily increased in the first 6 hours following submersion, after which, the levels plateaued. There was a positive correlation between nicotine

Table 2 Nicotine Concentration (mg/ml) and Number of Cigarettes.

SODA			100 mL	Nicotine/Soda	Absolute Level
Key	Time	Cigarette	Soda	mg/ml	mg x 100 mL
1A	6 hours	1 cigarette	soda	0.28	28 mg
1B	6 hours	1 cigarette	soda	0.25	25 mg
1C	6 hours	1 cigarette	soda	0.25	25 mg
2A	6 hours	2 cigarettes	soda	0.38	38 mg
2B	6 hours	2 cigarettes	soda	0.41	41 mg
2C	6 hours	2 cigarettes	soda	0.41	41 mg
3A	6 hours	3 cigarettes	soda	0.45	45 mg
3B	6 hours	3 cigarettes	soda	0.43	43 mg
3C	6 hours	3 cigarettes	soda	0.50	50 mg
4A	6 hours	4 cigarettes	soda	0.52	52 mg
4B	6 hours	4 cigarettes	soda	0.55	55 mg
4C	6 hours	4 cigarettes	soda	0.56	56 mg



Line Graph 1. Mean concentration of nicotine (mg/ml) with time(hours).



Line Graph 2. Mean nicotine concentration (mg/mL) with the number of cigarettes submerged.

concentration and time of submersion of cigarettes, r = 0.530, n = 18, P = 0.024 when the one-week levels were excluded. With one week levels included, the correlation ceased to exist (r = 0.328, n = 21, P = 0.147), possibly because of the plateauing of nicotine concentration levels with time after 6 hours. There was also a strong positive correlation between nicotine concentrations and the number of cigarettes. (r = 0.967, n = 12, *P*<.001).
Conclusions: The mean concentration of nicotine measured over the course of 6 hours from one full cigarette in 55 mL of a cola beverage was found to be 0.48 mg/mL. After six hours, the concentration of nicotine per sample did not differ with increasing time. The concentration of nicotine increased with the number of soaked cigarettes in the liquid and with the duration of submersion. Severe toxicity has been reported with ingestion of less than 2 mg of nicotine in children with oral LD50 in humans being in the range of 6.5 - 13 mg/kg. Therefore, drinking a few ml of leftover beverage containing cigarettes in them could prove clinically significant especially in children.

KEYWORDS Nicotine, soda, cigarettes

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42. Methylene blue use in medical toxicology as reported through the ToxIC registry

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Objectives: Methylene blue (MB) has been traditionally used for the treatment of methemoglobinemia. However, there is considerable literature describing its effectiveness in the management of distributive shock from various causes including sepsis, anaphylaxis, and calcium channel blocker poisoning. The objective of this study is to understand the characteristics of MB use in clinical toxicology since 2010 as reported to the Toxicology Investigators Consortium (ToxIC) Registry, a North American database.
Methods: The ToxIC registry was probed from January 1, 2010, to December 31, 2018, to identify all reported cases for which MB was used as an "antidotal" agent. The xenobiotic agents involved, specific clinical indications to start MB, clinical parameters of the cases involved, and other forms of treatment received were obtained.
Results: We identified 109 patients reported to the registry, in whom MB was used as a therapeutic agent. Roughly 40% of cases received MB to treat hypotension or shock that was unresponsive to vasopressors. On linear regression analysis of the usage of MB from 2010 to 2018, there was a statistically significant overall increase in the use of MB for any therapeutic purpose (p =0.047) and for methemoglobinemia (p = 0.02), but no statistically significant increase in its use for hypotension/shock (p = 0.1). The highest reported usage is in the years 2015 and 2018. Calcium channel blocker was the most common agent for which MB was used, in the treatment of hypotension and shock, followed by antibiotics, local anesthetics, and analgesics for the management of methemoglobinemia. Other notable toxic agents were Ifosfamide for its neurotoxicity (n = 3) and hypotension and shock resulting from insecticide poisoning (n = 2), metformin (n = 2) and snake bite envenomation (n = 1). MB was most commonly administered in the ICU more than 50% of the time, followed by in the emergency department, about 35% of the time. When MB was given for indications other than methemoglobinemia, 77.3% of the patients were having hypotension or shock, 75% of them had required multiple vasopressors, 27.3% of them required CPR, 13.6% received ECMO and approximately 40% of them eventually died.
Discussion: MB has traditionally been used as a reducing agent to treat methemoglobinemia. However, animal studies, case reports, and observational studies have described how MB prove to be effective in the management of distributive shock from causes like sepsis, anaphylaxis and cardiovascular medication toxicity like calcium channel blockers and beta blockers. This can be explained by its inhibitory action on nitric oxide-cyclic guanosine monophosphate (NO-cGMP) pathway, which plays a significant role in the pathophysiology of distributive shock by the production of NO that leads to vasodilation and hypotension. Our data from the toxic registry suggest that over the years from 2010 to 2018, MB is being increasingly used for both methemoglobinemia and hypotension/shock, although no statistically significant increase was found for the latter.
Conclusion: MB is being increasingly used in clinical toxicology as reported to the ToxIC registry, particularly for methemoglobinemia and hypotension/shock. Calcium channel blocker was the most common xenobiotic.

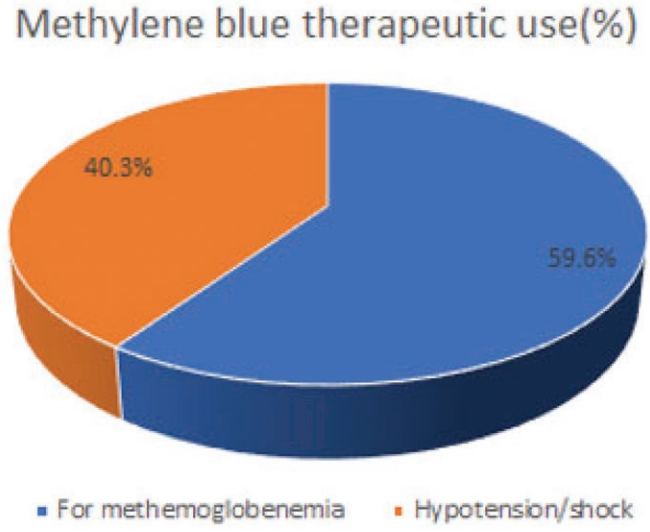

Figure 1.

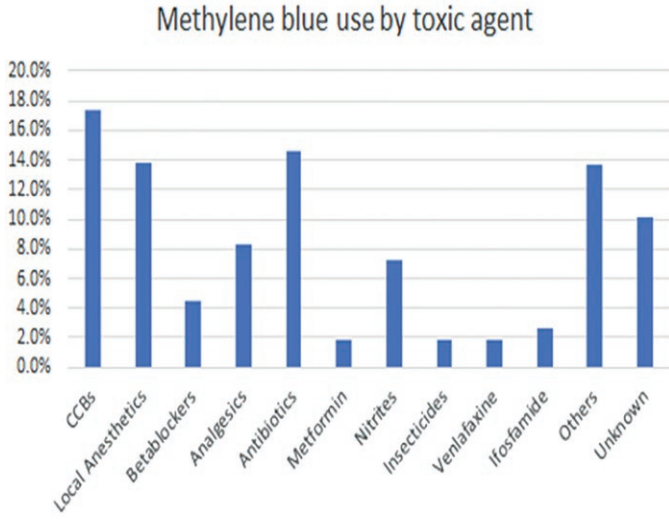

Figure 2.

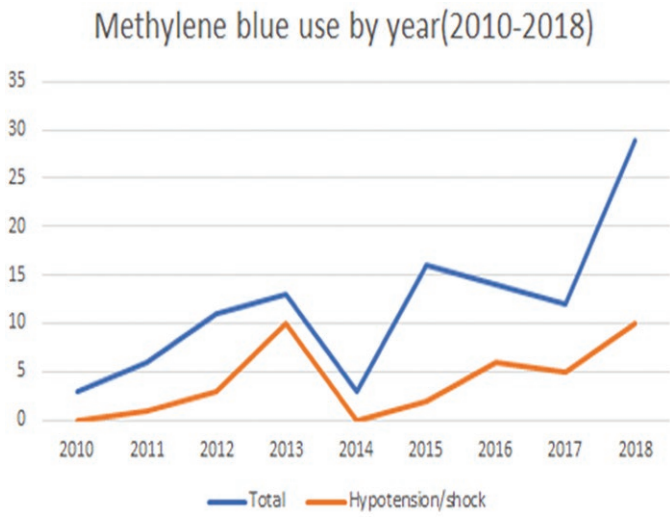

Figure 3.

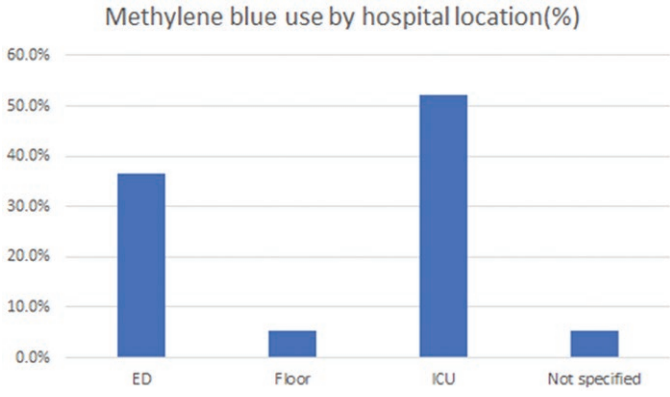

Figure 4.

KEYWORDS Methylene blue, distributive shock, calcium channel blockers
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43. A simple approximation of the QT nomogram

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Background: The QT nomogram is superior to most formulae that attempt to "correct" QT for heart rate. In comparison to the Bazett, Fredricia, and most other QT corrections, the QT nomogram provides similar sensitivity for arrhythia events while having superior specificity. The use of the QT nomogram has potential to decrease unnecessary resources and monitoring for patients at low risk of arrhythmia. The nomogram line is a graph of a polynomial equation. Clinicians may have difficulty in applying the nomogram when a graphic depiction is unavailable.
Objectives: To propose a linear approximation for easy calculation of the value of the QT nomogram (X) for any heart rate (HR) above 65 bpm, and to compare the linear approximation to the derived QT nomogram.
Methods: We used data from Isbister et al originally describing the QT nomogram, the QT nomogram and the Linear Approximation appear on the same graph (Figure 1). The Linear Approximation is X = 484 ms when HR 65 bpm. For every heart rate between 30 and 155 bpm, we calculated the difference (expressed in percent of the QT nomogram value) between the derived and approximated lines.
Results: The QT nomogram is linear for rates below 65 bpm and nearly linear at rates above the inflection point near 65 bpm. The Linear Approximation has maximum deviations of +0.6% to – 7.5%. All deviations with an absolute value >1% occur at heart rates >130.
Conclusions: The linear approximation closely fits the derived QT nomogram line. A simple arithmetic calculation can quickly discern whether a QT-HR pair is above or below the nomogram line without reference to the graph or an online calculator. The linear calculation estimates within 1% of the QT nomogram for all heart rates up to 130 bpm.

KEYWORDS QT nomogram, risk prediction, arrhythmia
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44. Brazil nuts from the "Amazon": A case report of digitoxicity in the digital age

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Background: Digoxin-like cardioactive steroids (CAS) found in plants, herbal preparations, and topical aphrodisiacs cause cardiac and gastrointestinal toxicity. Cases of toxicity are often related to exposures to substances known to have CAS. We present a case of CAS toxicity secondary to supplements not known to contain such substances.
Case report: A 27-year-old man presented to an emergency department after a suicide attempt. He ingested an entire package of "Semilla de Brazil" his wife had purchased online, which contained 30 nuts. One hour later, the man developed dizziness, fatigue, vomiting, and diarrhea. Initial vital signs were: heart rate, 40 beats/min; blood pressure, 135/95 mmHg. An electrocardiogram demonstrated complete heart block. The patient was externally paced and Poison Control was consulted. We recommended atropine, discontinuing external pacing, and adding a digoxin concentration to laboratory tests. The patient was given 1.5mg of atropine and converted to a normal sinus rhythm at 90 beats/min shortly afterwards. Laboratory results demonstrated a serum digoxin concentration of 0.9 ng/mL via a monoclonal digoxin assay. This patient denied taking digoxin nor did he have access to it. The patient was monitored in an intensive care unit for approximately 48 hours. His electrocardiogram remained stable and symptoms had resolved.
Case discussion: Brazil nuts have been used as a source of nutrition for centuries and do not contain any substances that cause cardiac toxicity. They are harvested from the wild and difficult to mass produce due to their extensive environmental requirements. There are reports of mislabeling of other products of similar appearance and sold as brazil nuts. "Codo de fraile", a seed from the thevetia tree known commonly as yellow oleander, has been sold under the name of "Semillia de Brazil" and various others and marketed as a weight loss supplement. Yellow oleander is associated with cardiotoxicity as it contains several CAS. Identifying specific CAS in serum presents its own inherent challenges given the size of the molecule and low concentrations that are present in serum. Fortunately, these substances often cross react with commercial digoxin assays. In this case, the serum digoxin concentration resulted after the patient clinically improved and digoxin-Fab was withheld. We believe CAS to be the culprit given the elevated digoxin concentration although we were not able to identify the product as there was none left to examine. It was subsequently removed from Amazon.com where it was obtained. Interestingly, several reviews from verified purchasers reported severe illness after ingestion. A particular review, prior to our case, reported severe bradycardia and persistent vomiting requiring hospitalization.

Conclusion: Supplement use is often discouraged in western medicine given lack of regulation and little demonstration of either safety or efficacy. This case demonstrates the dangers of obtaining supplements from unverified sources. These cases are infrequent and warrant serious consideration when there is little history to support the clinical presentation. Consideration for digoxin-fab in these patients with significant bradycardia and/or hemodynamic instability is advised.

KEYWORDS Digoxin, yellow oleander, supplements

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45. Lipid Emulsion (Intralipid®) Treatment of Flecainide Overdose Associated with Acute Respiratory Distress Syndrome

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Background: Intravenous Lipid Emulsion (ILE) was pioneered as rescue for local anesthetic overdose, but has also been used for a variety of other overdoses. It is proposed that ILE alters drug distribution by functioning as a lipid sink, and also acts as a source of fatty acids for poisoned myocytes. Lipid Emulsion (Intralipid® 20%) is a buffered sterile non-pyrogenic fat emulsion prepared for intravenous administration composed of soybean oil, egg yolk phospholipids, glycerin, and water. Major component fatty acids are linoleic (44-62%), oleic (19-30%), palmitic (7-14%), linolenic (4-11%) and stearic (1.4-5.5%). Acute Respiratory Distress Syndrome (ARDS) results from acute hypoxic lung injury and is characterized by pulmonary edema and normal cardiac filling pressures. Fatty acid chemistry has been demonstrated to contribute to the development of ARDS. Free fatty acid is more toxic than neutral fat, and is typically bound to albumin in circulation. Oleic acid is the most common free fatty acid in mammals and has been associated with acute lung injury secondary to fat emboli. The proposed mechanism is threefold: 1) damages epithelial and endothelial cells, increasing membrane permeability resulting in edema and denuded areas in alveoli causing formation of a hyaline membrane, 2) Induces apoptosis in alveolar type 1 and 2 cells, and 3) alveolar macrophages trigger an inflammatory cascade. A case of flecainide overdose treated with lipid emulsion, triggering the ARDS cascade is presented with supporting microanatomical sample results.
Case Discussion: A 35 year old presented in asystolic cardiac arrest, intubated by EMS and given 5 doses of epinephrine prior to arrival. With further resuscitation, patient had intermittent wide complex arrhythmias with short-lasting returns of spontaneous circulation (ROSC). EMS provided her medication list which included flecainide, and a review of her chart showed history of cardiomyopathy, atrial fibrillation, depression and suicidality. Further resuscitation involved continued compressions, epinephrine, 450mEq of sodium bicarbonate, 4g of calcium gluconate, 4g of magnesium, and amiodarone with intermittent ROSC with a wide complex bradycardia. With little information as to quantity or duration of possible exposure, a 1.5mL/kg bolus of ILE was given twice with near instantaneous resolution of wide complex into sinus rhythm. An infusion of ILE was started along with maximized vasopressor support, and the patient was admitted to cardiac intensive care unit. She remained profoundly hypotensive. Unfortunately, the patient had suffered an anoxic brain injury and care was withdrawn the next day. Flecainide concentration from time of presentation resulted at 2.01mcg/mL, twice the upper limit of normal. Family noted 10-15 tablets of flecainide missing from the bottle, suggestive of the drug's narrow therapeutic window. Histologic results on autopsy reveal incipient development of ARDS. Lung Microscopy showed "congested lungs with well aerated alveoli, patchy collections of intraalveolar neutrophils, and focal hyaline membrane formation. Non-occlusive intravascular lipid deposits noted, as highlighted in one osmium fixed section." Slides are available for review. Hyaline membranes are the earliest of findings in ARDS.
Conclusion: The evidence presented supports the concept that lipid emulsion rescue therapy may contribute to development of ARDS in critically ill patients.

KEYWORDS ARDS, Intravenous Lipid Emulsion, flecainide

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46. A systematic review and meta-ananlysis of alternative dosing to the FDA approved 3-bag regimen.

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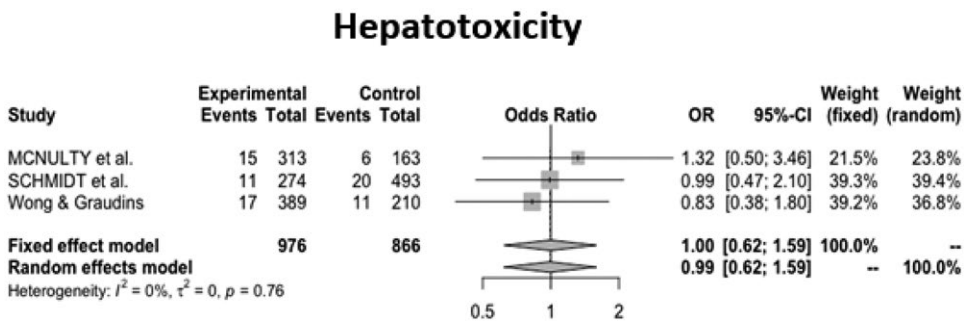
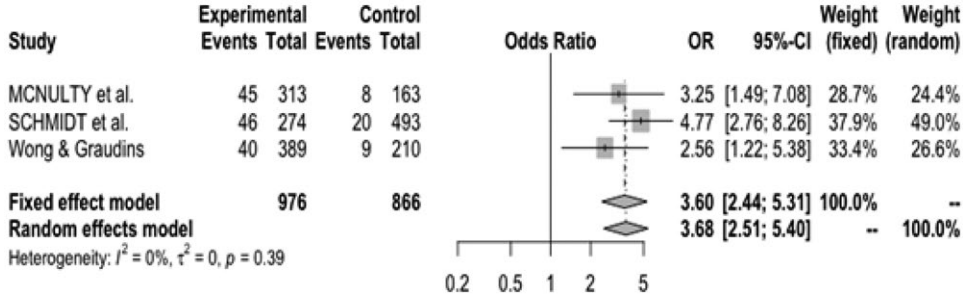
Background: Acetaminophen (APAP) toxicity is the most common cause of acute liver failure in the United States. Acetylcysteine has long been the mainstay of treatment, but recent literature suggests a simplification of the FDA approved "three-bag" method may decrease administration errors and adverse effects. We performed a systematic review of the literature and meta-analysis to compare efficacy and safety of a simplified acetylcysteine administration compared to the FDA 3-bag method.
Methods: We followed PRISMA guidelines.
Search Strategy: On September 1, 2018 we used the MESH terms: NAC, acetaminophen toxicity, acetyl-cysteine, N-acetylcysteine, paracetamol, APAP, 2-bag, and 3-bag. We searched Medline/Pubmed, Google, Google scholar, Cochrane library, EMBASE and Toxnet.
Inclusion/Exclusion criteria: All languages were included. We excluded articles not containing data on simplified or 3-bag method or lacked data on adverse reactions. Case reports, review articles, and animal studies were excluded. Two authors independently reviewed each study using Rayaan QCRI to determine if studies met search criteria blinded to the selection of the other. Disagreements were discussed until agreement was met.
Data Analysis: We conducted a fixed-effect meta-analysis using R package meta. The software first computes the log OR and its variance for individual studies. The summary log OR is then computed as a weighted average of the log ORs, where weights are the inverse of the estimated variances. To visually summarize the meta-analysis results, we also produced forest plots. We used the Q test to assess for heterogeneity between the studies. Primary outcomes of our meta-analysis were hepatotoxicity and a secondary outcome of anaphylactoid reactions.
Results: Database MESH search resulted in 636 unique citations. Of the 636 studies, 38 met criteria for full text review, and six met study criteria. Of the six studies, which investigated a simplified acetylcysteine regimen, three studies shared the same 2-bag treatment iteration with three also utilizing some form of a modified 2-bag infusion regimen but with primary focus being on varying the duration or dosing of infusions. Using the three studies that shared the same 2- bag regimen we conducted our meta analysis. For hepatotoxicity, there was no heterogeneity between the three studies, $Q(2) = 0.54$, $p = 0.8$,





indicating the non-significant difference between the three-bag versus two-bag treatment which remained consistent across the studies, OR =0.996, 95% CI: [0.623; 1.593]. For anaphylactoid reactions, there was not heterogeneity Q(2) = 1.89, p = 0.4. The result of meta-analysis indicates that three-bag compared to two-bag treatment consistently increased the odds of anaphylactoid reactions, OR =3.5967, 95% [2.4372; 5.3078], p<.0001.

Conclusion: Following analysis, a simplified acetylcysteine is non-inferior to the 3-bag method, but is associated with less administration errors and anaphylactoid reaction. Next steps would be to validate this further with a randomized control trial, as well it may be beneficial to further investigate the benefit of early termination of acetylcysteine therapy in low risk patients.

KEYWORDS Acetaminophen Toxicity, N-acetylcysteine, Simplified Dosing Regimen

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47. Methamphetamine-Induced Pseudovasculitis

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Background: Pseudovasculitis includes a variety of disorders that simulate systemic vasculitis. These conditions present with clinical, radiological, and laboratory features resembling those of systemic vasculitis. However, the pathogenesis, therapeutic approach, and prognosis are different. Cocaine and levamisole have been both implicated as the cause of vasculitis and pseudovasculitis. However, this entity has been rarely reported with methamphetamines.

Case Report: A 44-year-old man was admitted to the intensive care unit for agitated delirium, rhabdomyolysis and acute kidney injury. He admitted to frequently smoking methamphetamines, including on the morning of admission. He denied ever knowingly using cocaine siting his preference of methamphetamine and the lower cost. On hospital day five, the patient was noticed to have several small bullae on his right arm; which, progressed into extensive retiform purpura and bullae on all extremities, buttocks, back and scrotum. Extensive serological testing, including p-ANCA and c-ANCA, was negative. Liquid chromatography-mass spectroscopy analysis of or blood and urine showed methamphetamine and benzoylecgonine (evidence of cocaine use). A skin biopsy showed necrosis of eccrine coils and full thickness necrosis without vascular inflammation. These results were not consistent with levamisole-induced vasculitis. The patient improved clinically with just continuous veno-venous hemodialysis for his renal injury and, one week later, a second skin biopsy showed only mild abnormalities. As serology was negative and histopathology did not show the typical microthrombi with adjacent inflammatory cells found in levamisole-induced vasculitis, a diagnosis of methamphetamine-induced pseudovasculitis was made.

Case Discussion: Vasculitis is a challenging diagnosis for many physicians, including specialists. In this case, a multidisciplinary team including medical toxicology, rheumatology, dermatology, pathology, nephrology and critical care collaborated to rule out potential causes of the patient's findings and ultimately diagnose him with methamphetamine-induced pseudovasculitis. Though cocaine has a greater historic association with vasculitis, methamphetamine is increasingly being recognized as a cause. Methamphetamine has similar vasoactive properties to cocaine which are postulated to cause tissue injury by vasoconstriction. Unlike classical vasculitis, cases of vasculitis and pseudovasculitis caused by substances of abuse are best treated by removal of offending agents and not high dose corticosteroids.

Conclusions: Methamphetamine-induced pseudo-vasculitis may clinically mimic vasculitis but it resolves with cessation of the offending agent. Physicians should be aware of this diagnosis in patients presenting with suspected vasculitis as it may spare the patients from unnecessarily receiving corticosteroids or immunosuppressants.

KEYWORDS Pseudovasculitis, Methamphetamine, Dermatology

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48. Canine Medication Apoquel® in Human Exposure: An Observational Study from a Single Poison Center

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Background: Oclacitinib maleate (Apoquel®) is a synthetic janus kinase (JAK) inhibitor indicated for allergic dermatologic diseases in dogs. Approved in 2013, it inhibits JAK1-dependent and JAK3-dependent cytokines as well as interleukin (IL)-2, IL-4, IL-6, IL-13, and IL-31. In humans, JAK inhibitors are widely used in the treatment of disease states ranging from atopic dermatitis to rheumatoid arthritis. Tofacitinib is a similar drug used in humans; it binds to JAK1 and JAK3 like oclacitinib. While the mechanism of action of these two drugs is similar, the specific effects of oclacitinib in humans remain unknown. A single poison center reviewed their Toxicall® records for the last six years. This study characterizes the data collected from oclacitinib ingestions reported to a single poison center.
Methods: This study was designed as a retrospective observational study. The patient data used was generated from a single poison center's Toxicall® data from 2013 to 2018. Data was collected using the product codes for Apoquel® 3.6mg (7834959), Apoquel® 6.4mg (7835345), Apoquel® 16mg (7834892), and oclacitinib (7834793) in the product code filter of the advanced search reporting tool.
Results: The total number of patient exposures was 123 with 110 (89.4%) managed on site/non-health care facility, 10 (8.1%) treated/evaluated and released from a healthcare facility, 1 (0.8%) admitted to a noncritical care unit, 1 (0.8%) lost to follow-up/left against medical advice, and 1 (0.8%) managed at an unknown site. Patient age was as follows: 101 (82.1%) > 20 years, 11 (8.9%) < 5 years, 2 (1.6%) 6-12 years, 1 (0.8%) 13-19 years, and 8 (6.5%) unknown/invalid. Seven cases reported clinical effects as follows: 4 (3.3%) reported gastrointestinal effects, 7 (5.7%) reported neurologic effects, 1 (0.8%) reported miscellaneous effects, and 1 (0.8%) reported respiratory effects. Most cases, 98 (79.7%), were ruled unintentional therapeutic error, and 24 (19.5%) were unintentional general. Intentional exposures accounted for no exposures. For patient outcomes, 112 (91.1%) of cases were reported as nontoxic/minimally toxic and not followed; 2 (1.6%) were potentially toxic and not followed; 8 (6.5%) were followed to outcome in a health care facility. Of those 8 cases, 7 (87.5%) had no effect, and 1 (12.5%) had a moderate effect.
Conclusion: The results of this study suggest that our understanding of oclacitinib is limited. Of the 8 patients followed to outcome in a health care facility, the majority had no symptoms, with one report of respiratory difficulty resulting in a moderate effect. This study affirms the need to investigate the toxic effects of oclacitinib in the human body.

KEYWORDS Apoquel, Oclacitinib, Canine

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49. Can a Stand-Alone Emergency Department Manage a Toxic Patient Efficiently?

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Background: Stand-alone emergency departments (SAED) have been operational since 2010 and number in the hundreds. According to Ticci et. al, (2017) the SAEDs are not equipped to manage the toxic patient due limited staffing, lack of antidotes, restricted laboratory studies capability, and limited ancillary support. Toxic patients may require elaborate testing and support. According to the Medicare Payment Advisory Commission report, SAEDs basically treat patients with lower acuity than hospital emergency departments. Triage patients are assigned an emergency severity index (ESI) number between 1 and 5, with 1 being the most critical. Patients with an ESI score of 1 can be unstable, require immediate evaluation by the provider, and may require higher level of resources to include staffing, materials, energy, services, and knowledge. Toxic patients are categorized as ESI 1.
Method: Review the number of toxic cases reported to a PCC by SAEDs from January 1 to November 13, 2018. Note the number of patients treated and released, and the number of patients that required a higher level of care that were transferred to a tertiary hospital or mental institution.
Results: A regional poison control center received 859 calls from SAEDs involving 501 patients, 157 were treated and discharged, 203 were transferred to a tertiary hospital for a higher level of care; 110 were admitted to a critical care unit, 93 were admitted to a noncritical care unit, 98 were admitted to a mental health institution, and 43 left against medical advice.
Conclusion: The results of this survey suggest that the stand alone emergency departments are not efficient in the management of the toxic patient because of their limitations in staffing, antidotes and laboratory studies. The transfer process to a tertiary hospital may take hours based on bed availability. We believe that development of rapid procedures for identification, stabilization, and transfer of the toxic patient to a tertiary hospital can improve morbidity and mortality.

KEYWORDS Stand-Alone, efficiency, limitations

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50. Poison Control Center's "Rolled Out No Answer" (RONA) Calls and the Financial Ramifications

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Background: The following data of six poison control centers (PCC) was reviewed to determine the number and financial effects of calls that were made to the PCCs in 2018 that went unanswered. Calls to the PCC that are not answered, lost, or rolled over to another center are called rolled out no answer (RONA). Each call that is lost had the potential to spare health care resources and can be costly for callers who did not get help from the PCC and went to the Emergency department for treatment.
Method: A retrospective data review was conducted on six PCCs and 40 specialists in poison information (SPI) to appraise the number of telephone calls to the PCC that were not answered while SPIs were on duty. Incoming calls may not be answered for a variety of reasons to include breaks, case work, special assignment, or just missed. The review will include the amount of time logged on, the number of calls that were not answered while the SPIs on duty and in the ready mode, and the projected number of callers that could have been helped at home.
Results: PCC- A: 4 SPIs answered 7195 and had 356 RONAs, 5% of the calls, PCC-B: 11 SPIs answered 24,735 and had 275 RONAs, 1% of the calls, PCC-C: 5 SPIs answered 10,929 calls and had 289 RONAs, 3% of calls PCC-D: 7 SPIs, answered 13,708 and had 1143 RONAs, 8% of the calls, PCC-E: 9 SPIs answered 17,953 calls and had 752 RONAs, 4% of the calls PCC-F: 6 SPIs and answered 13,184 calls and had 1547 RONAs, 10% of the calls. The 6 PCCs answered 87,704 calls and had 7042 RONAs.

Conclusion: According to the AAPCC's estimation, 69%, 4859 of the documented RONAs could have been managed at home. A toxic workup in an emergency department can range between $2,000-$10,000 thousand dollars. The cost for treating 7042 people in the ED could range between $14,084,000 and $70,420,000. The six PCCs answered 87,704 calls in 2018 and spared $1,174,356.56 in health care dollars.

Table of six poison center's RONA data.

PCC	Number of SPIs	Calls answered	RONAs	Percent of calls
PCC- A	4	7195	356	5%
PCC-B	11	24.735	2745	1%
PCC-C	5	10,929	289	3%
PCC-D	7	13,708	1,143	8%
PCC-E	9	17,953	752	4%
PCC-F	6	13,184	1547	10%

KEYWORDS RONA, ramifications, resources

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51. Insect repellent band exposures reported to a statewide poison center network

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Background: Currently on the market, there are non-DEET (N, N-diethyl-meta-toluamide) insect repellent bands infused with plant-derived essential oils such as lemongrass oil. Vapors from the wristbands are meant to produce a protective shield around the immediate area and serve as an alternative to conventional DEET products. The objective of this study was to describe insect repellent band exposures reported to a statewide poison center network.
Methods: Cases were insect repellent band exposures reported to a statewide poison center network during 2003-2018. Cases were identified by reviewing the substance description fields and notes field for "mosquito," "insect," or "bug" in association with "band" or "bracelet." Case distribution was determined for factors related to patient demographics, exposure circumstances, management, and outcome.
Results: Of 152 total insect repellent band exposures identified, 85 (55.9%) were reported during May-July. The annual number of exposures ranged between 1-10 during 2003-2014 and then increased to 23-26 during 2015-2018. The patient age distribution was 146 (96.1%) children (=20 years), and 1 (0.7%) unknown age; 85 (55.9%) of the patients were female. The exposure route was 133 (87.5%) ingestion alone, 13 (8.6%) ingestion and dermal, 3 (2.0%) dermal alone, 2 (1.3%) ocular, and 1 (0.7%) unspecified other route. Of the 152 exposures, 146 (96.1%) were reported as unintentional, 3 (2.0%) intentional, 2 (1.3%) adverse reaction, and 1 (0.7%) contamination/tampering. Most (n=147, 96.7%) of the exposures occurred at the patient's own or another residence and 5 (3.3%) at other locations. The management site was 141 (92.8%) on site, 9 (5.9%) already at or en route to a healthcare facility, 1 (0.7%) referred to a healthcare facility, and 1 (0.7%) at an unspecified other location. The medical outcome was 32 (21.1%) no effect, 6 (3.9%) minor effect, 22 (14.5%) not followed-judged nontoxic, 87 (57.2%) not followed-minimal clinical effects possible, 2 (1.3%) unable to follow-potentially toxic, and 3 (2.0%) unrelated effect. The most frequently reported clinical effects were oral irritation (n=5, 3.3%), vomiting (n=5, 3.3%), and abdominal pain (n=4, 2.6%); other clinical effects reported in 1-3 exposures were edema, erythema/flushed, hives/welts, rash, dysphagia, nausea, throat irritation, fever/hyperthermia, drowsiness/lethargy, ocular irritation/pain, and red eye. The most common treatments were dilution/irrigation/washing (n=118, 77.6%) and food/snack (n=23, 15.1%).
Conclusions: Insect repellent band exposures reported to this poison center network have increased in recent years. The majority of cases were reported during May-July and occurred at the patient's residence. Most patients were children that mainly involved exposures by ingestion, followed by dermal contact. The most frequently reported clinical effects were gastrointestinal and dermatological. In most cases, exposures managed on site resulted in minimal effects, and none resulted in serious outcomes.

KEYWORDS Repellent, insect, essential oil

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52. Cannabidiol exposures reported to a statewide poison center network 2000-20018

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Background: Cannabidiol (CBD) is the main nonpsychoactive cannabinoid found in the cannabis plant and is classified as a Controlled Substance Schedule I substance by the Drug Enforcement Administration (DEA). Since the DEA Schedule reclassification of CBD with THC content below 0.1% from I to V in 2018 due to the Food and Drug Administration (FDA) approval of CBD indicated to treat two rare forms of childhood epilepsy, there has been a growing interest for its perceived beneficial pharmacological effects. The preliminary clinical research in CBD has focused on treating conditions such as epilepsy, anxiety, insomnia, and pain as a result of reported anti-inflammatory and relaxing effects. This study intended to describe cannabidiol exposures reported to a statewide poison center network.
Methods: Cases of CBD exposures reported to one statewide poison center network during 2000-2018. Case distribution was determined for various factors related to patient demographics, exposure circumstances, management, and outcome.
Results: Fifty-one total CBD exposures were identified during this study period. No exposures were reported before 2015; the annual number afterward was 2 in 2015, 5 in 2016, 6 in 2017, and 38 in 2018. Thirty-three (64.7%) of the patients were male, and 18 (35.3%) female. The median patient age was 28 years (range 1-89 years), and 46 (90.2%) of exposures occurred at the patient's residence. Forty-one (80.7%) of the exposures were by ingestion alone, 7 (13.7%) by inhalation alone, 1 (2%) ingestion and inhalation, 1 (2%) ingestion and dermal, and 1 (2%) by an unknown route. Of these 51 exposures, 10 (19.6%) cases involved additional substances. Reasons for exposure included 18 (35.3%) unintentional, 18 (35.3%) intentional, 11 (21.6%) adverse reaction, 3 (5.9%) unknown, and 1 (2%) malicious intent. The management site was 26 (51.0%) already at or en route to a healthcare facility, 17 (33.3%) on site, 5 (9.8%) at other sites, and 3 (5.9%) referred to a healthcare facility. The distribution by medical outcome was 15 (29.4%) not followed-minimal clinical effects possible, 8 (15.7%) minor effect, 8 (15.7%) moderate effect, 7 (13.7%) unable to follow-potentially toxic, 6 (11.8%) no effect, 2 (3.9%) major effect, 1 (2.0%) not followed-judged nontoxic, and 4 (7.8%) unrelated effect. The most common clinical effects reported were drowsiness/lethargy (n=8, 15.7%), tachycardia (n=7, 13.7%), hypertension (n=6, 11.8%), vomiting (n=6, 11.8%), and agitated/irritable (n=6, 11.8%). The most frequent treatments were IV fluids (n=12, 23.5%), dilution (n=8, 15.7%), and benzodiazepines (n=5, 9.8%).

Discussion: CBD exposures reported to this statewide poison center network increased by over 500% in 2018. Unintentional and intentional exposures to CBD were proportional. Most exposures involved adults and male patients by ingestion alone at their residence. More than half of the patients were managed at a healthcare facility. Most commonly reported clinical effects were neurological, cardiovascular, and gastrointestinal.
Conclusions: The majority of medical outcomes resulted with minor-moderate clinical effects. CBD exposures were increasingly reported in this statewide network over the last five years as a result of regulatory changes and clinical research.

KEYWORDS Cannabidiol (CBD), cannabinoid, cannabis

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53. Shotgun pellet embolism to the right atrium with elevated blood lead concentrations (BLL)

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Background: There is no consensus on whether asymptomatic patients with retained extra-articular bullets or shotgun pellets should have blood lead surveillance monitoring. Routine BLL monitoring may be warranted in cases with concomitant bony fractures or those patients with numerous retained fragments. It is unknown whether embolized intracardiac bullets portend an increased risk of elevated BLLs and also warrant monitoring. Intracardiac bullet embolization presents a unique problem as therapeutic options for removal, such as surgical extraction or endovascular retrieval, are invasive and carry significant risk of complications. We present a case of a shotgun injury followed by venous pellet embolism to the right heart with subclinical elevated serial BLLs.
Case report: A 22-year-old woman sustained a shotgun wound to her left thigh. Upon ED arrival, the patient was tachycardic and hypotensive. Physical examination revealed multiple small entrance holes in the anteromedial left thigh and groin region with no pellet wound sites of the chest or abdomen. Distal pulses to the lower limbs were normal with no evidence of impaired perfusion. Thoracoabdominal and pelvis CT was performed, revealing numerous shotgun pellets in the medial compartment of the left thigh without active extravasation. A single shotgun pellet was also noted within the right atrium with no evidence of intraabdominal, intrathoracic, or retroperitoneal injuries. The patient received four units of packed RBCs and was taken to the OR for wound exploration and washout. Intraoperative angiography of the left lower extremity demonstrated no evidence of arterial or venous injury. The poison center was contacted for recommendations on BLL monitoring. We suggested interval monitoring at the time of hospital admission, followed by weekly intervals until 1-month post-injury, then monthly until 4 months post-injury, and then again at 1-year post-injury. Birth control was also strongly encouraged. A BLL on hospital day 2 was 12.3 mcg/dL. During hospitalization, the patient experienced no dysrhythmias, chest pain or shortness of breath and she was discharged home after an uneventful 3-day hospital course. The patient experienced no cardiopulmonary complications or symptoms of lead toxicity as of 3-months post-injury. BLLs are shown in Figure 1. A rapid rise in BLL occurs by 2 weeks post-injury. BLLs stabilize over the next 30 days then gradually increase at 90 days post-injury.
Discussion: No consensus exists on whether asymptomatic patients with retained extra-articular bullets or shotgun pellets should have serial BLL monitoring. In our case, the intracardiac location of the embolized pellet prompted BLL surveillance monitoring with the theoretical concern that continuous contact with blood may liberate more lead from the pellet. However, it is possible that a large number of pellets were unable to be removed during operative washout. If so, the retained pellets in the patient's thigh may be a more important factor in this patient's elevated BLL and repeat imaging could assist in determining the current location of the embolized pellet as well as any retained pellets not removed during operative washout.
Conclusion: Routine monitoring of BLLs should be considered in patients with retained intracardiac shotgun pellets.

KEYWORDS Retained bullets, Lead toxicity, shotgun pellet embolization

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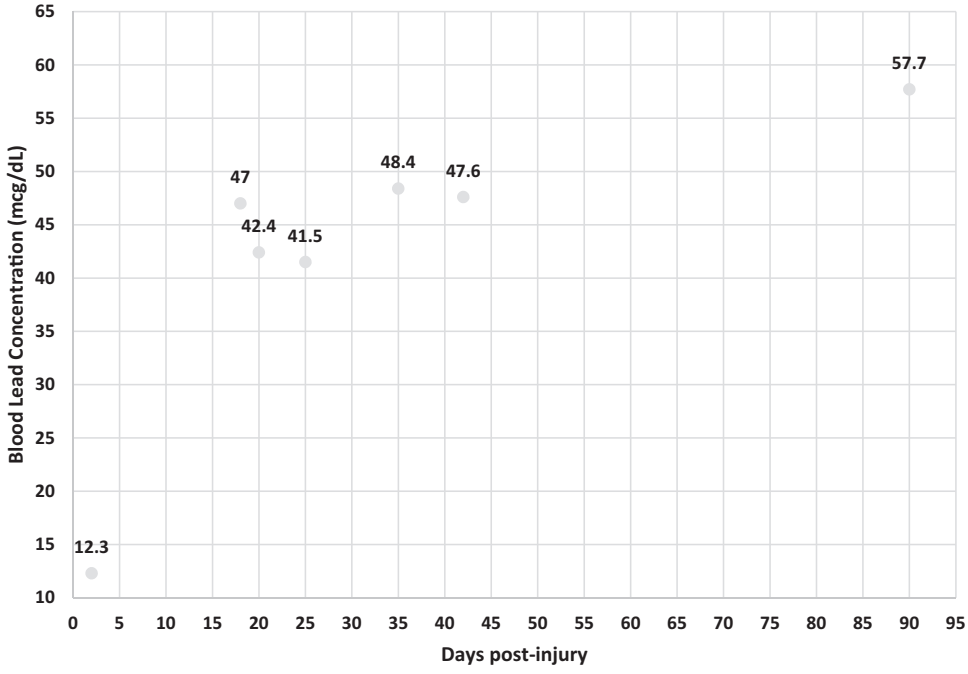

Figure 1 Serial whole blood lead concentrations (mcg/dL). Testing performed by Inductively Coupled Plasma-Mass Spectrometry (ICP-MS). Tests performed by Mayo Clinic Laboratories- Rochester Superior Drive 3050 Superior Drive NW, Rochester, MN 55901.

54. Kratom Fatality: A Case Report

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Background: Kratom, Mitragyna speciosa, is a tropical tree in the coffee family native to Southeast Asia. Recently it has gained popularity as a natural supplement for many uses including increasing energy and as an analgesic. Though scientific studies of this plant are limited, it is known to contain mitragynine and 7-hydroxymitragynine (7-HMG), natural alkaloids with some affinity for mu & kappa opioid receptors. These properties suggest promise for possible treatment of opioid addiction. In August of 2016, the DEA temporarily place kratom into its Schedule I category due to high potential for abuse, but after further consideration rescinded this categorization in December of 2016. Previous cases of death from kratom toxicity have been reported in combination with other illicit substances. We believe this is one of the first reported deaths in the United States solely from kratom toxicity.
Case Report: A 32 year old male with a history of anxiety and illicit substance use (not further specified) was found unresponsive at home approximately 30 minutes after he was last seen alive. He could not be resuscitated. Subsequent autopsy disclosed no significant injury or natural disease. Routine post-mortem drug screens performed on urine, and repeated on peripheral blood, were presumptively positive only for diphenhydramine, quantitation of which was below detectable limits (50 ng/mL). Blood volatile screens were also negative for acetone, ethanol, isopropanol, and methanol. Given the circumstances of death, additional testing for novel psychoactive substances and designer opioids was pursued on postmortem blood using high performance liquid chromatography (HPLC) and time of flight mass spectrometry (TOFNS). These studies revealed a mitragynine level of 23 ng/ml. No other prescribed or non-prescribed substances were identified during post mortem fluid studies. The death certificate listed cause of death as "Acute Mitragynine Toxicity".
Discussion: With varying levels of legal status of this product in the United States and its advertised "natural" safety, many users perceive kratom to be a "safe" substance. Anecdotally, clinicians have reported serious morbidity and occasional mortality associated with use of this substance, typically in combination with other abusable substances. Furthermore, withdrawal syndromes have been observed, but not scientifically studied, with sometimes significant clinical effects including nausea, diarrhea, agitation and seizures.
Conclusion: M. speciosa use is rising as evidenced by increasing calls to poison centers nationally. Pharmacologically, this substance is presumed to partially agonize opioid receptors. It is purportedly used as a home remedy to lessen opioid withdrawal effects. Further study is necessary to fully understand pharmacological properties and effects of this substance in humans.

KEYWORDS Kratom, mitragynine, poisoning

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55. Seizure in a 3-month-old following ingestion of homeopathic skin tag remover

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Background: Thuja occidentalis is a native European tree often used as a flavoring agent in food and drink products, as a fragrance in cosmetics, and in homeopathic remedies for conditions ranging from joint pain to heart ailments and inflammatory disorders. Reports of toxicities related to Thuja are primarily anecdotal and include headache, convulsions, diarrhea, liver and renal damage, and arrhythmia. It is thought that Thuja may lower the seizure threshold in those with a seizure disorder.
Case Report: A 3-month-old female infant was exposed to a homeopathic skin tag remover when her mother mistook the bottle for that of a colic remedy. The infant was immediately transported to the ED at which time the poison center was called. The mother reported she had "poured some" of the product into the baby's mouth. The product, Provent Skin Tag Remover, was labeled as containing 0.34 fluid ounces of a mixture of Thuja occidentalis 6X (HPUS) (noted as the active ingredient), cedar leaf oil, Melaeuca aternifollia leaf oil, and Ricinus communis seed oil (each listed under "other ingredients). Upon the initial call to the poison center, the infant could be heard crying persistently, but no other symptoms were noted. During a period of observation for signs or symptoms of aspiration, the infant developed "uncontrollable seizures" and subsequent possible aspiration and was transferred to a tertiary care facility. Vital signs remained "stable" throughout. Levetiracetam was administered as well as agents to facilitate intubation for protection of the airway. On hospital day two, vital signs were reported as: HR 181, RR 52, T 38.1C, BP 80s/50s, SpO2 99% (FIO2 40%). EEG had been performed for 5-6 hours and was "negative". No further seizures occurred and the infant was receiving only acetaminophen for fever and lorazepam. She was subsequently extubated and was doing well. On hospital day three, the infant was eating and drinking; receiving only 2 LPM oxygen by nasal cannula. She was discharged home that evening. The initial hospital's laboratory was asked to retain the child's blood specimen for potential analysis; however, when a facility was identified for this testing, the hospital reported the specimen was no longer available. The original product container had also been discarded in the ED.
Case discussion: Homeopathic products are often marketed as 100% natural; as was seen with this product. This may be interpreted by the lay public as safe. Although the seizures occurring in this patient cannot be definitively attributed to ingestion of the Thuja occidentalis-containing homeopathic product, an association may be present. Potentially, the infant had an as-yet-unidentified seizure disorder that was unmasked by the use of this product.
Conclusions: Use of homeopathic products should be approached with caution as their safety and efficacy have not been tested. Many tested homeopathic products have been found to have been diluted incorrectly or contain contaminants and have potential to cause significant harm. Ingestion of this Thuja occidentalis-containing skin tag remover may have resulted in seizures in a 3-month-old infant.

KEYWORDS Thuja occidentalis, seizure, skin tag remover

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56. Lipoid Pneumonia from Carbon 60 Oil

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Background: Exogenous lipoid pneumonia is an uncommon condition characterized by the aspiration or inhalation of fat-based substances into the alveoli. While acute presentations can occur, the presentation is typically insidious and non-specific with the most common symptom being progressive shortness of breath. Similarly, the radiological findings on chest computerized tomography (CT) are also non-specific and varied.

Case Report: A 59-year-old previously healthy male presented to hospital with a seven-day history of progressive shortness of breath, cough and subjective fevers. He was admitted to the intensive care unit for hypoxemia requiring high flow nasal cannula and treated empirically for bacterial and viral pneumonia. Chest CT on presentation demonstrated multiple pulmonary emboli and non-specific diffuse ground glass opacities. Therapeutic anti-coagulation was started with resolution of pulmonary emboli on repeat imaging at day eight, however, ground glass opacities persisted. Pseudomonas aeruginosa pneumonia was treated for seven days. A presumptive diagnosis of cryptogenic organizing pneumonia was made and a 14-day course of methlyprednisone started. Despite these treatments, there was no improvement. On day 23, a bronchoscopy was negative for infection. Autoimmune, malignant and vasculitis workup was unremarkable. The patient was concerned that he possibly could have been poisoned due to the nature of his work. The poison centre was consulted and a detailed history and medication review was conducted. The patient admitted to using several vitamins and minerals including Carbon 60 oil liquid suspension orally daily for the past six months every morning typically on an empty stomach or with coffee. Because of this revelation, additional assessment of the previously collected bronchoalveolar lavage was requested to assess for lipoid material. Oil red O staining for lipids was positive; a diagnosis of lipoid pneumonia was confirmed. This patient was treated with a pulse and then tapering course of prednisone with improvement in oxygen requirements and symptoms over the next several weeks.
Case Discussion: This is the first documented case of lipoid pneumonia associated with Carbon 60 oil. This marketed health supplement is a hydrophobic symmetric carbon nano-structure discovered by biomedical engineers in 1985. To date, only animal studies have been conducted to examine the oxidant and free-radical scavenging properties. It is not approved for human consumption in North America, however, is available for purchase on the internet suspended in olive oil. Lipoid pneumonia is a rare pulmonary complication of inhalation or aspiration of fat- or oil-containing substances. It has classically been reported as a complication of chronic oil-based laxative use, but has also been reported due to a number of exposures including hydrocarbons, vaping e-cigarette liquid, butane hash oil, and a number of natural remedies and supplements. The diagnosis requires a high degree of suspicion as clinical and radiological findings are non-specific. BAL or lung biopsy with a specific request for lipoid staining are required for confirmation.
Conclusion: This case demonstrates the importance of taking a detailed exposure and medication history in the context of assessing non-resolving airspace disease; recognizing that exposure to alternative supplements including Carbon 60 oil may induce lipoid pneumonia.

KEYWORDS Lipoid, Pneumonia, C 60

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57. A case of senna-induced skin burns

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Background: Glycoside sennosoids are a component of a number of effective over the counter laxatives. An under-appreciated complication of sennosoids are dermal chemical burns. In young children this may prompt evaluation for child abuse.
Case report: A healthy 3 yo male was admitted to the burn service with extensive second degree burns to his buttocks. His mother reported the child had consumed several of his 12 year old brother's chocolate laxatives the day prior and had experienced 5 loose stools before bedtime. She discovered the next morning the child had been in a soiled diaper overnight. When cleaning the child she noted blistering and sloughing of skin on his buttocks. This prompted an Emergency Department visit for further evaluation and management of his injury. Extensive sloughing and peeling of skin consistent with a second degree burn was noted and the burn service consulted. The burn service did not feel the injury was consistent with mother's story. Pediatric and child protective services were consulted for a social and non-accidental trauma (NAT) evaluation. Toxicology was also consulted.
The older brother used a chocolate flavored senna laxative for chronic constipation. Based on the mother's history and the appearance of the injury the toxicology service was able to corroborate the mother's history and expedite the child's discharge home with mother. When seen one week later in the burn clinic the child's skin was healing well.
Case discussion: The use of senna laxatives became more common after the Food and Drug Administration to removed phenolphthalein from its list of products considered safe in over the counter (OTC) laxatives in 1999. Soon afterwards the first reports of senna-induced skin burns were reported. Since then, there have been several more reports describing similar injury. Senna-induced skin burns are not uncommon. One study found that 11% of pediatric patients with accidental ingestion of senna-containing OTC laxatives developed skin blisters and sloughing. All of these children were still using diapers. Diaper usage with prolonged skin contact time appears to be an important prerequisite. Like our case, some of these previous cases had initially been evaluated for potential child abuse. The location and shape of the burn, consistent with the location of the diaper has been proposed as a way to differentiate this from a scald injury. Senna laxatives are not recommended for children under two years of age without a doctor's approval. Skin burns are not listed as a warning on these products. Although it seems that this type of injury could also occur in the debilitated elderly patient with incontinence we are unaware of any reports of this.
Conclusion: Senna induced skin burns in children appears similar to scald injuries associated with neglect and abuse. Although important to still consider child abuse, awareness of this type of injury can expedite social service and NAT evaluation in cases such as this.

KEYWORDS Senna, Skin burn, laxative

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58. Phenibut withdrawal treated with a baclofen taper

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Background: Phenibut was first developed in Russia in the 1960s. Structurally similar to baclofen, it likewise has predominant GABABactivity. It is used for anxiety and insomnia in Russia. Although not available as a prescription drug in the United States, it is legal to buy online or at vitamin shops. Despite being promoted as a safe alternative to prescription drugs for anxiety, insomnia, and as a nootropic, phenibut can lead to dependency and withdrawal.
Case Report: The patient is a 19 year-old male with a past medical history of substance abuse who presents to the Emergency Department with anxiety, palpitations, and insomnia. The patient states the symptoms began shortly after stopping phenibut two days prior to arrival. The patient had been buying this supplement online and using it to self-medicate for insomnia and anxiety. However, he has been steadily increasing the dose – now taking as much as 5 grams a day. About one year prior to arrival, he had been hospitalized for phenibut withdrawal that was associated with severe agitation, hallucinations, delirium, and rhabdomyolysis. He required admission to the ICU at that time and was managed with dexedetomidine and benzodiazepines. The patient had been off of phenibut since this event until about 2 months prior

to this presentation. For management of his withdrawal toxicity this hospital admission, we recommended baclofen, titrating the dose to symptoms. At a dose of 45 mg divided into 3 doses daily, he experienced significant symptom relief and required only several prn doses of hydroxyzine for insomnia. The baclofen was tapered over 4 weeks, decreasing by 5 mg per day every 4 days. The patient was monitored in the hospital during the initial stages of the taper with notable improvement in symptoms. He was able to be discharged three days after presentation to complete the baclofen taper. The patient was seen in the toxicology clinic thirteen days after initial presentation. He had been doing well on the baclofen taper and states that this process is much smoother that last time.
Case Discussion: This patient demonstrated phenibut dependency, with tolerance marked by increasing dosage and withdrawal. Phenibut and baclofen are structurally similar, and baclofen has been used to manage phenibut withdrawal. The optimal duration of a baclofen taper is not known. Previous cases have described tapers of 6 days to 24 weeks. It has been previously described that approximately 10 mg of baclofen are needed for each gram of baclofen in a taper regimen. This information was used to structure the taper for this patient. The optimal duration and dose of a baclofen taper regimen requires further study to elucidate.
Conclusion: Regular phenibut use can lead to dependency and withdrawal. We described a 4 week taper that successfully managed this patient's withdrawal and avoided a repeat ICU admission.

KEYWORDS Phenibut, Withdrawal, Baclofen

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59. Intracerebral Hemorrhage Associated with Phenylethylamine adulterated Kratom

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Background: "Kratom" from the plant Mitragyna speciosa is a tropical evergreen tree native to Southeast Asia. It has long been used for its opioid and mild stimulatory effects. Mitragynine is believed to be the active alkaloid with activity at both the mu and delta opioid receptors. In recent years it has been marketed in the United States as an herbal supplement to treat multiple maladies including chronic pain and opioid withdrawal. Some preparations are marketed as having more stimulatory properties than others.
Case Report: A 54-year-old man presented with altered mental status after ingestion of kratom. He had a reported history of using a product called Kratom Crazy® regularly for about one year. He had recently run out and began using a new product called Vivzen Maeng Da Kratom®. Twenty minutes after first ingestion of new brand he developed severe headache and emesis, shortly thereafter fell asleep. The patient's wife had also used the product at the same time, and noted that it had a much stronger stimulatory effect than their previous product, but suffered no ill effect. The following day, family noted mental status changes and the patient was brought to the hospital. On presentation, approximately 16 hours after ingestion, he was altered but had normal vital signs and no focal neurological deficits. A CT scan revealed a large right frontal lobe intraparenchymal hemorrhage with intraventricular extension with 7 mm of subfalcine herniation. The patient underwent cranioplasty and hematoma evacuation and was discharged on hospital day 12 without neurological sequelae. A comprehensive urine drug screen was negative for any drugs not administered by the treating team. Analytical testing revealed a serum concentration of mitragynine 340 ng/mL on day 3. A collaborative effort between department of health and treating toxicology team resulted in the confirmation of mytraginine in both Kratom Crazy® and Vivzen®, however the Vivzen® product was adulterated with phenylethylamine (PEA).
Case Discussion: Although used for decades in Southeast Asia, published investigations of mitragynine have been limited to case reports and small trials. In addition to its opioid effects, Mytraginine is thought to blunt stimulation of 5HT receptors and post-synaptic alpha-2 receptor agonist effects. It is by this mechanism that Mytraginine is thought to have its stimulatory effects. PEA is an unscheduled compound different from amphetamine by a single methylation at the alpha carbon. It has the potential to increase sympathetic tone resulting in a dangerous rise in blood pressure that could predispose one to intracerebral hemorrhage. We hypothesize that ingestion of the PEA adulterated product resulted in a transient episode of hypertension that resulted in intracranial hemorrhage.
Conclusion: We report a case of close temporal relationship with PEA adulterated Kratom ingestion and intraparenchymal hemorrhage.

KEYWORDS Kratom, Phenylethylamine, hemorrhage

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60. Development of Methemoglobinemia after Hydroxocobalamin Administration

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Introduction: Smoke inhalation during an enclosed-space fires is the most common cause of cyanide poisoning in the US. Hydroxocobalamin(OHCob) is the primary antidote for cyanide toxicity with the advantage that it does not induce methemoglobinemia. Although methemoglobin has a higher affinity for cyanide, it also decreases oxygen carrying capacity in patients who may have concomitant carboxyhemoglobinemia. There is a single case report of a burn patient with suspected methemoglobinemia associated with the administration of OHCob. We similarly report a patient from a housefire who experienced significant smoke inhalation and developed methemoglobinemia after receiving OHCob.
Case: A 62-year-old male patient with a medical history of coronary artery disease and aortic valve replacement, presented to the Emergency Department (ED) after prolonged smoke exposure from a house fire. Initial vital signs showed: blood pressure 123/76 mmHg, heart rate 100 bpm, temperature 34.1oC, respiratory rate 20 rpm, and oxygen saturation 100% on 100% FiO2. Physical exam revealed an awake male in moderate respiratory distress, soot in his mouth and posterior pharynx, cool skin, a 1% TBSA burn on the left ear. He was intubated for worsening respiratory distress and hypoxia. ED VBG showed: pH 7.12, pCO2 55mmHg, pO2 53mmHg, carboxyhemoglobin 1.3%, methemoglobin 0.3%. Within three hours, the patient clinically deteriorated and was started on vasopressors, antibiotics, continuous renal replacement therapy, and was admitted to the Intensive Care Unit. Blood cyanide analysis, taken 6 hours after smoke inhalation, was undetectable. Serum lactic acid concentration, taken 11 hours after ED arrival, was 9mmol/L. At 14 hours, the patient received OHCob 5g IV for presumed cyanide toxicity and underwent serial ABG measurements. Immediately prior to OHCob administration, the methemoglobin measured 0.7%. Following administration, methemoglobin concentrations increased to 4.2%; 14.3%, and 16.3%, at 2, 16, and 47 hours, respectively. He received methylene blue 50mg IV at 60 hours with minimal response. The patient's hospitalization was complicated by multi-system organ failure and he expired on hospital day five with a methemoglobin of 6.4%.
Discussion: Definitive laboratory diagnosis of cyanide toxicity is not readily available and empiric treatment is indicated for sick patients

with smoke exposure. Our patient developed a significant methemoglobinemia 14 hours after smoke inhalation despite no medical history suggestive of an acquired or genetic predisposition. He received topical sulfadiazine cream, which has been linked to methemoglobinemia, but is an unlikely etiology in this case. Several studies have evaluated the spectrographic absorption of OHCob and potential interference with co-oximetry interpretation. However, this is not consistently reported on all machines or at therapeutic doses of OHCOb. The patient may have also developed sulfhemoglobinemia since most co-oximeters do not distinguish methemoglobin and sulfhemoglobin, but this was not evaluated. The link between OHCob and methemoglobinemia is supported by an in-vitro study that documented formation of a methemoglobin variant after OHCob administration in swine, indicating a potential mechanism in some humans. Further studies are warranted to investigate the link between OHCob and methemoglobin, and we caution providers to monitor for methemoglobinemia after OHCob administration, especially if the patient lacks clinical improvement.

KEYWORDS Hydroxocobalamin, Methemoglobin, Cyanide

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61. Fatal Dysrhythmia Associated with Subcutaneous Ropivacaine Infusion in a Child

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Background: Local anesthetic systemic toxicity (LAST) is most commonly associated with inadvertent intravascular administration of an agent during regional nerve block. We present a case of extended subcutaneous treatment in a pediatric patient where route of administration and pharmacokinetic factors may have contributed to toxicity and lack of response to conventional cardiopulmonary resuscitation (CPR) measures combined with intravenous lipid emulsion (ILE).
Case Report: A 4-year-old 15.3 kg male with a history of bilateral nephroblastomas, diagnosed three months previously, experienced a good response to chemotherapy other than a large residual mass involving the right kidney. He underwent elective surgery for resection of renal masses. Post-operatively he was started on continuous renal replacement therapy and a subcutaneous ropivacaine infusion at 6 mg/h (6.5 mcg/kg/min) via an On-Q pump for pain control. Other medications included ceftriaxone 50 mg/kg q 24 h, pantoprazole 0.5 mg/kg IV daily, fentanyl 1 mcg/kg/h, pediatric TPN 45 mL/h, and propofol 120 mcg/kg/min. On post-operative day four the child had increasing ventricular ectopy followed by wide complex tachycardia. Ropivacaine was discontinued, an esmolol infusion was started, and titrated to 50 mcg/kg/min. Because of the persistent abnormal rhythm, a 1.5 mL/kg ILE bolus was given, but within minutes the patient had an asystolic cardiac arrest. Chest compressions were begun, the child was intubated, and resuscitation was continued with pediatric cardiology at the bedside. During this resuscitation, the child received several doses of epinephrine, sodium bicarbonate, magnesium, and attempts at electrical cardioversion. External pacing was attempted without success. Return of spontaneous circulation was achieved and the child was started on an epinephrine infusion. The patient remained hemodynamically stable on the epinephrine infusion for approximately three hours but then developed a wide complex rhythm that degenerated to ventricular fibrillation. Resuscitation was attempted with amiodarone, bicarbonate, dextrose and insulin (due to hyperkalemia), and additional ILE without success. After ultrasound showed no cardiac activity, resuscitative efforts were discontinued. Postmortem serum ropivacaine concentration from inferior vena cava blood was 1.4 mcg/mL.
Case Discussion: ILE is most effective in treating systemic toxicity due to inadvertent rapid intravascular injection of a local anesthetic. In this case, continuous ropivacaine infusion for several days may have exceeded the child's capacity to metabolize the drug, resulting in development of LAST. Concurrent illness and medications, as well as mode of medication delivery may have also played a role. Ropivacaine is metabolized by CYP1A2 and CYP3A4 to inactive metabolites that are cleared renally. An in vitro study demonstrated an inhibitory effect of propofol on ropivacaine metabolism by CYP3A4 (Osaka, J Anesth 2006; 20: 60-63). Co-administration of propofol to this child may have saturated enzymatic ropivacaine metabolism, contributing to systemic toxicity.
Conclusion: We present an unusual case of LAST associated with prolonged subcutaneous ropivacaine administration rather than accidental intravascular injection. Conventional CPR measures combined with ILE bolus administration were not effective treatments for this child.

KEYWORDS Ropivacaine, Local anesthetic systemic toxicity, Intravenous lipid emulsion

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62. Recovery from Copperhead Snake Envenomation: Role of Age, Sex, Bite Location, Severity, and Treatment

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Background: Few data exist to understand the recovery phase of pit viper envenomation and whether specific patients recover more quickly, or respond differently to antivenom therapy, than others. This study examines time course of recovery from copperhead snake (Agkistrodon contortrix) envenomation patients managed with and without the use of antivenom, stratified by age, sex, anatomic site of envenomation, initial severity of envenomation, and geographic region.
Methods: This is a post hoc subgroup analysis of data from a multi-center double-blinded clinical trial of Fab antivenom (FabAV) vs. placebo. Outcomes were the Patient-Specific Functional Scale (PSFS) score at 3, 7, 10, and 14 days after envenomation. Least-squares mean PSFS score curves were calculated for each subgroup, and repeated measures ANOVA was used to estimate between-group comparisons.
Results: Seventy-two subjects were included, of whom 44 received FabAV. Men demonstrated better overall recovery than women (model predicted PSFS score 6.18 vs 4.99; difference: 1.19; 95% CI: 0.12 to 2.25; $p = 0.029$). No sex difference was found in response to FabAV. Overall recovery and effect of FabAV were similar in adult vs adolescent patients, patients with upper vs lower extremity envenomation, and patients with initially mild vs moderate envenomation signs. Analysis by geographic location was not successful due to ANOVA model instability. This study was limited by small numbers of subjects in some subgroups.

Conclusions: Male victims of copperhead snake envenomation demonstrate slightly better recovery than females during the first 14 days after treatment, but response to Fab antivenom overall is similar across all subgroups studied.

KEYWORDS Envenomations, snake, Agkistrodon, Antivenoms

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63. Where the Texas coral snakes are: Comparison of Micrurus tener geographic distribution with the location of bites reported to Texas poison centers

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Background: Micrurus tener (Texas coral snake) is one of approximately 80 coral snake species found in the Western hemisphere and the only one of the three indigenous U.S. coral snake species native to Texas. According to the Herps of Texas website (www.herpsoftexas.org), M. tener can be found in 121 of 254 Texas counties. The intent of this study was to compare the reported geographic distribution of M. tener with the location of bite calls received by Texas poison centers.
Methods: Cases were M. tener bites reported to Texas poison centers during 2000-2018 where the caller's county was known. The distribution of the cases was compared to the known geographic distribution of M. tener.
Results: There were 521 M. tener bites reported from 76 counties. Of these 76 counties, 69 (90.8%) were known to have M. tener. Of the 521 M. tener bites, 514 (98.7%) were from counties where M. tener is located. Seven counties accounted for 293 (56.2%) of the M. tener bites: Harris (n= 89, 17.1%, 1.16 per million population), Travis (n= 58, 11.1%, 3.04 per million population), Bexar (n= 56, 10.7%, 1.76 per million population), Nueces (n= 24, 4.6%, 3.77 per million population), Montgomery (n= 23, 4.4%, 2.71 per million population), Cameron (n= 22, 4.2%, 2.86 per million population), and Fort Bend (n= 21, 4.0%, 1.95 per million population).
Discussion: The majority of both the counties where M. tener bites were reported from and of the bites themselves were counties where M. tener is known to live. The fact that M. tener bites came from seven counties not known to have Texas coral snakes suggests that M. tener may have a wider distribution than previously recognized. Limitations include potential snake misidentification and the county from where the call originated may not be the county where the bite occurred.
Conclusion: M. tener bites were reported from 76 of 254 Texas counties, including several not previously known to have a coral snake population. Knowing where M. tener bites occur may be useful in allocating resources, e.g., antivenom and healthcare provider training.

KEYWORDS Micrurus tener, coral snake, geographical distribution

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64. Clozapine-induced myocarditis

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Background: Myocarditis is a rare but potentially life-threatening complication of therapeutic clozapine use, a second-generation antipsychotic drug that is the cornerstone of therapy for refractory schizophrenia. A high index of clinical suspicion is critical to preventing mortality in these patients. We describe a case of myocarditis shortly after initiation of clozapine treatment for refractory schizophrenia. Our report focuses on the characterization, detection, and management of clozapine-induced myocarditis.
Case Report: A 22-year-old male was started on clozapine with slow titration to 300 mg daily. An initial high-sensitivity troponin-T (HST-T) was <6 ng/L on day 3 of treatment. He had no history of alcohol, tobacco, or illicit-substance use. On day 20 of treatment, he developed difficulty breathing and pleuritic chest pain associated with tachycardia at 135 beats per minute, hypotension at 90/59 mmHg, fever of 101.9 °F, and hypoxia at 89% on room air. A HST-T was found to be elevated at 1086 ng/L (normal <6-14 ng/L). He was transferred to the emergency department (ED) with a presumptive diagnosis of clozapine-induced myocarditis. His physical exam was significant for right-sided diffuse pulmonary crackles. His laboratory workup was significant for mild leukocytosis of 10.8 K/µL, elevated pro-brain natriuretic peptide (Pro-BNP) of 8764 pg/mL and ECG showing sinus tachycardia at 104 beats per minute, normal QTc and QRS intervals, and new T-wave inversions in leads V5-V6. A non-contrast computed tomography scan showed a multifocal pneumonia in the right middle and lower lobes and transthoracic echocardiography revealed moderate global left ventricular systolic dysfunction with an ejection fraction of 30-35%. A coronary catherization showed no occlusive coronary artery disease. Blood and urine cultures were negative for growth after 48 hours and his respiratory viral panel was negative. The patient was monitored on telemetry and started on carvedilol and lisinopril. A HST-T at discharge was 55 ng/L. A plasma trough clozapine level taken on day 19 of clozapine therapy was found to be 881 ng/mL with a norclozapine level of 261 ng/mL (total clozapine level of 1142 ng/mL). Clozapine therapy was discontinued.
Case Discussion: The development of clozapine-induced myocarditis typically occurs within the first few weeks of initial therapy. Supportive evidence for detecting myocardial damage includes elevated cardiac enzymes, elevated Pro-BNP, ECG showing signs of ischemia, and chest radiography ruling out other etiologies. An echocardiogram demonstrating reduced ventricular function in the absence of ischemic coronary disease further confirms a diagnosis of myocarditis. However, the gold standard for diagnosing myocarditis is an invasive endomyocardial biopsy.
Conclusion: Our case highlights a rare, yet potentially fatal cardiovascular complication related to therapeutic clozapine use. Development of chest discomfort, cough, shortness of breath, fever, or unexplained tachycardia in a patient taking clozapine should be aggressively investigated with screening laboratory tests looking for myocardial damage, such as a high-sensitivity troponin-T.

KEYWORDS Clozapine, Myocarditis, Adverse Drug Effects

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65. How to "Poison" an Inpatient Medical Toxicology Consult Service

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Background: Inpatient bedside consultation is a crucial component to the clinical practice and curriculum of Medical Toxicology. However, with declining reimbursement and alternative sources of expertise (i.e. Poison Centers, Addiction Medicine), threats to the growth of inpatient consultations have never been greater. We describe the greatest threats to the sustainability of the inpatient consultation service: reimbursement trends and the replacement of a consult service by order sets.

Methods: Our inpatient Medical Toxicology consultation service has existed since July, 2001. Since its inception, we have tracked the number of consultations billed by fiscal year (FY) using Current Procedural Terminology (CPT). The same provider (with limited back-up) provided inpatient consultation services at four hospitals, with no change in availability over this time period. We also investigated financial data and electronic medical record (EMR) order set usage. There was no change in billing or collection procedure over this time period.
Results: In 2006, EMR generated toxicology-based order sets were initiated at our site. Since then, they have been used 10,034 times; the alcohol withdrawal order set (accessed 8,367 times) and the acetaminophen overdose order set (accessed 381 times) have been most utilized. The number of Medical Toxicology inpatient CPT billing encounters and number of order sets used from FY 2002-2018 are shown on Figure 1. At the start of the practice (FYs 2002 and 2003 combined), the medical toxicology service billed approximately $39,000 for alcohol/drug withdrawal consults, with 267 individual CPT bills. By FY 2006 (the year before order set use was widely used), the service billed $40,696. In FYs 2016-2018 combined, the total amount billed had decreased to $15,189, with a total of 61 individual instances billed. For acetaminophen overdoses, the total billed in FY 2002-2003 was almost $69,000, with 417 individual instances billed; from FY 2016-2018, the total billed was $46,415, with a total of 123 individual instances billed. We also note two important decreases in coding charges. In 2018, the contractual charge for critical care code 99291 was $655, compared to $1,033 in 2003. In addition, CPT codes 93042 (rhythm strip interpretation) and 99358 (non-face-to-face physician contact time) have been functionally eliminated; these billed for $30 and $340 per CPT bill. Overall annual charges and revenues between FYs 2010-FY 2014 averaged $183,353 (± $6,598) and $90,328 (± $6,169), respectively; in FY 2015-2018, these averaged $124,016 (± $22,438) and $61,909 (± $14,287). However, our gross collection rate improved from approximately 30% in FY 2002 to 45% in FY 2018 and the days in Account Receivable decreased from 74 days in 2010 to 31 days in 2018.
Conclusion: Our medical toxicology service noted a substantial decrease in total revenue, despite more efficient billing practices. This is attributable to a lack of longitudinal care, lack of procedures, downward reimbursement trends, and especially increased order set usage in lieu of formal inpatient toxicology consultations. An inpatient consulting service can no longer be the primary financial support for a toxicology service-based business plan.

KEYWORDS Reimbursement, Ordersets, Inpatient Consultations

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66. Nicotine pod ingestions, a clinical conundrum: a case series

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Background: Recently, the FDA has expressed concerns about an increase in nicotine use through vaping electronic cigarettes (e-cigarette). JUUL, an e-cigarette manufacturer, is currently the top selling e-cigarette brand in homes making them accessible to small children. JUUL use amongst school age children is wide spread, again posing a risk of ingestion. There is limited data available to guide management of ingestion of e-cigarette nicotine containing cartridges. These exposures can be dangerous as these liquids are highly concentrated and currently have limited regulation. We present 3 cases of ingestion of JUUL nicotine cartridge ingestions.
Case Reports: Case 1: A 17-year-old male presented to the emergency department (ED) reporting swallowing a JUUL pod on a dare 2 hours 15 minutes before presentation. He stated the pod was empty, but there was no way to confirm. He was clinically stable on admission with his only abnormality being bigeminy that was unlikely related to the exposure. An abdominal X-ray confirmed the presence of the pod in the stomach. He was admitted to a telemetry unit for observation and screening of stools. The patient remained asymptomatic and the pod passed via stool approximately 26 hours after ingestion.
Case 2: A 23-year-old male unintentionally swallowed a JUUL pod with his morning medications. He arrived to the ED 33 minutes after the ingestion asymptomatic. An abdominal X-ray confirmed presence of the pod, distal to the pylorus. The JUUL pod was removed via endoscopy and he was discharged home.
Case 3: A 13-year-old male accidentally ingested a JUUL Pod 2.5 hours prior to arrival to the ED. He had taken polyethylene glycol 3350 (Miralax®) prior to his arrival in attempt to assist with the passage of the pod. On arrival to the ED, he was tachycardic, thought related to anxiety. An abdominal X-ray confirmed the pod in the stomach, so he was made NPO and transferred to a hospital with pediatric gastroenterology for endoscopic removal. When he arrived at the referral center, a repeat X-ray identified the presence of pod in the small intestine. Prior to re-consulting with the poison center, he was taken to the operating room and the pod was removed via exploratory laparoscopy from the right colonic flexure. He was discharged home two days later without complications.

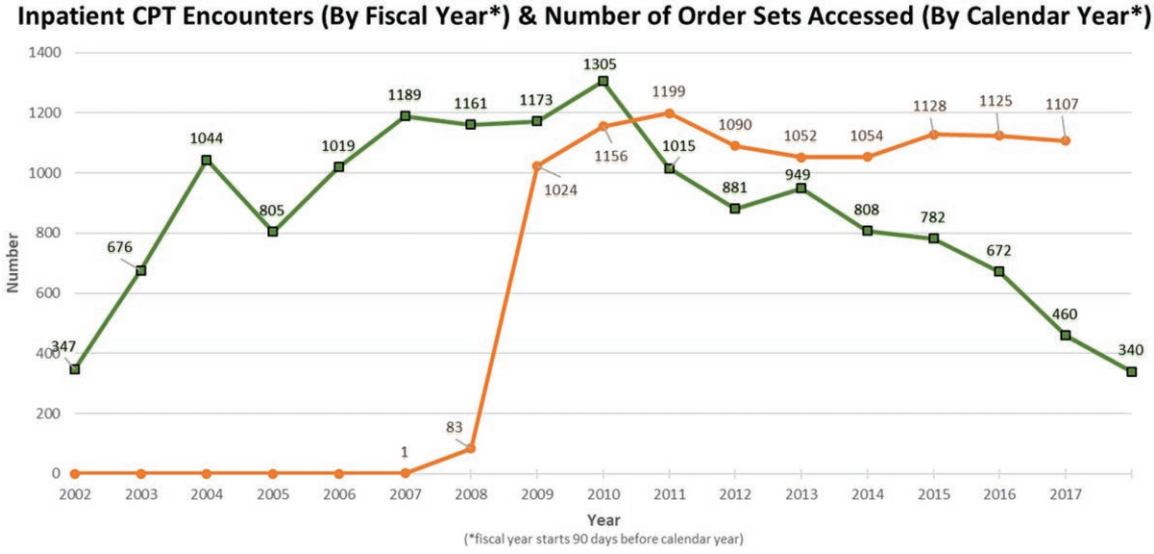

Figure 1.

Case discussion: These cases present a clinical conundrum. JUUL pods contain either 23 mg or 40 mg of nicotine, a quantity likely to cause significant toxicity in a small patient. The pods are fairly small, measuring approximately 1.51cm x 0.76cm and can be easily swallowed. We are unaware of available data on the integrity of the pods, but the website offers returns if the pod is leaking, suggesting there is some risk of pods leaking.
Conclusion: The increasing popularity of e-cigarettes and the limited data on the risk of nicotine leakage pose a clinical conundrum. Until more data are available, clinicians must weigh the risks of nicotine poisoning with more aggressive management (e.g., whole bowel irrigation, endoscopic removal, or surgical intervention).

KEYWORDS Nicotine, ingestion, foreign body

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67. Sure Jell drug test detox method – positive and negative results

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Background: Random mandatory urine drug screening is a common practice by probation and parole officers, especially those convicted of a drug- or alcohol-related offense. Subjects may attempt to take any number of substances to detoxify the body to pass their urine drug screen. One such method is called the Sure Jell Drug Test Detox which involves the ingestion of fruit pectin and a large amount of water to produce a false-negative on a cannabinoid screen. We report the first case, to our knowledge, of acute water intoxication as an adverse consequence of this drug screen evasion method.
Case Report: A 45-year-old male presented to the emergency department due to acute mental status change and seizure activity 30 minutes prior to arrival. It was reported he took Sure Jell in order to pass his urine drug screen. He was post-ictal on exam with BP 154/70, HR 822, RR 20, T 98.1, and O2 99% on room air. Subsequently, the patient became extremely combative and required three staff members to restrain him. He was intubated and sedated with propofol, cisatracurium, and midazolam. Computed tomography of the head was normal. Laboratory studies revealed significant hyponatremia (Na 112, K 3.9, Cl 75) and a presumptively negative urine cannabinoid screen. He was transferred to a tertiary care center for further hospitalization. Laboratory studies the following morning showed Na 116, K 4.1, Cl 81, CO2 24, Glu 118, BUN 3, Cr 0.73, serum osmolality 250 mOsm/kg; urine sodium was undetectable; urine specific gravity and osmolality were not performed. Over the next two days his hyponatremia resolved with fluid restriction and furosemide, and his mental status became appropriate enough for extubation on hospital day 4. A urine drug screen performed on hospital day 4 for presumptively positive for cannabinoids and benzodiazepines. On hospital day 5, his urine specific gravity was 1.031. At this point the patient admitted to being on probation and subject to routine drug screening but denied any recollection as to what he did that resulted in his hospitalization.
Case Discussion: The Sure Jell Drug Test Detox method is advertised on the Internet as a safe way to easily pass a cannabinoid urine drug screen. Ingestion of the fruit pectin is thought to trap circulating cannabinoids in the bile and prevent them from being excreted into the urine. This claim is without any scientific or medical evidence. Our patient did initially present with a false-negative urinary cannabinoid screen, most likely due to dilute urine, which resolved along with his fluid imbalance.
Conclusion: The Sure Jell Drug Test Detox method can produce a false-negative urinary cannabinoid screen at the risk of life-threatening neurological dysfunction from acute water intoxication. This case also highlights the potential harm posed by medical misinformation on the Internet.

KEYWORDS Cannabinoids, urine drug screen, hyponatremia

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68. Laboratory-confirmed thallium poisoning with no alopecia

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Background: Alopecia is considered a hallmark of thallium poisoning and aids in the diagnosis of a condition that otherwise presents with non-specific findings, especially early in the course. Interestingly, this case highlights a patient with laboratory-confirmed thallium poisoning and severe peripheral neuropathy that never developed alopecia.
Case Report: A 21 year-old college student with no significant past medical history presented to toxicology clinic with a chief complaint of "I've been poisoned by my roommate." He reported three weeks of tingling in his bilateral hands and feet. The patient had been well until, on returning to campus after spring break, he had multiple episodes of vomiting and diarrhea lasting two days. In the ED, the patient was described as "delirious" with heart rate 120 and blood pressure 110/76. Labs were notable for AST 118 and ALT 120. Approximately 3 days later, the patient developed tingling in the bilateral hands and feet. During this time, the patient's roommate, a chemistry major, exhibited bizarre behavior and made vague statements suggesting the patient had been poisoned. As part of the police investigation, the patient was referred to a forensic laboratory for testing. Patient presented to toxicology clinic 4 weeks after onset of symptoms. His vital signs were heart rate 74, blood pressure 112/65, respiratory rate 16, temperature 99 °F and oxygen saturation 99% on room air. Exam was notable only for subjective altered sensation in his bilateral feet, especially with walking. On follow up two days later, patient had developed severe pain in his bilateral feet that subsequently ascended his calves. Nerve conduction studies were consistent with axonal degeneration. A qualitative urine heavy metal screen returned positive for thallium. Patient was started on activated charcoal. Prussian blue was ordered from a compounding pharmacy in California and the patient commenced treatment 3 days later. Quantitative analysis prior to onset of therapy revealed plasma thallium concentration 2.4 ng/mL and urine thallium concentration 15.8 µg/L. Laboratory studies ordered on completion of three days of activated charcoal and three days of Prussian blue revealed plasma thallium concentration was zero and urine thallium concentration was 4.2 µg/L. Throughout his course, the patient never developed alopecia. On follow up 9 months later, patient continued to have bilateral foot pain.
Case discussion: Initial symptoms of thallium poisoning are non-specific; consequently, delays to diagnosis and initiation of therapy are common. While there is no antidote for thallium poisoning, treatment with Prussian blue to prevent absorption and enhance elimination is thought most effective if initiated promptly after exposure. Alopecia typically develops around 10 days after thallium exposure and is often the first distinguishing feature. In this case, diagnosis of an already rare condition – thallium poisoning - was further hindered by the atypical clinical course – absence of alopecia.
Conclusion: Diagnosis of thallium poisoning requires a high index of suspicion and recognition of characteristic findings; absence of alopecia is an unusual finding that may further delay diagnosis and treatment.

KEYWORDS Thallium, poisoning, alopecia

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69. Sources of Severe Unintentional Carbon Monoxide Exposures Admitted to the Hospital or Transferred for Hyperbaric Oxygen Therapy

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Background: Unintentional carbon monoxide (CO) poisoning is a common preventable environmental poisoning throughout the United States. Studies have shown that home CO detectors have been associated with small decreases in hospital admissions for CO poisoning. The goal of this study is to compare characteristics of unintentional, non-fire CO exposures from home appliances, to those from other non-home appliance sources.
Methods: We performed a retrospective chart review of CO poisoning cases reported to a regional poison center (RPC) between January 1, 2015 and December 31, 2017. Eligible cases included those coded as CO poisoning in the RPC's database who were admitted to a hospital or transferred for HBO therapy during the study period. Exclusion criteria were poisoning due to house fires, intentional poisoning, patients discharged from the emergency department without admission or HBO therapy, or cases with no source identified in the case narrative. The included cases were divided into two groups based on exposure source: home appliances; including ovens, stoves, furnaces, and water heaters/boilers; and non-home appliance sources including all other sources. The two groups were analyzed for differences in age, sex, site of exposure and indicators of severe poisoning: carboxyhemoglobin level, syncope, elevated troponin and HBO therapy. Statistical analysis was performed to evaluate the differences between groups using a student's t-test to compare mean CO levels and a chi-square test to compare all other variables analyzed.
Results: 81 cases met inclusion criteria. There were 38 poisonings from home appliances: stoves (2), water heaters (2), ovens (10) and furnaces/boilers (24). There were 43 poisonings not from home appliances: a gas-powered vacuum (1), concrete mixer (1), hookah (1), concrete saws (3), power washers (9), generators (12), and automotive vehicles (16). The details of these exposures are summarized in Table 1.
Conclusions: Less than half of cases admitted for carbon monoxide poisoning in the RPC database were from home combustion appliances such as a stoves, ovens, furnaces, or water heaters. Non-home appliance portable combustion sources accounted for the majority of admitted cases for which a source had been documented in the RPC record. Patients admitted for CO exposure from a home appliance were more likely to be female while those admitted due to poisoning from a non-home portable appliance were more likely to be male. We observed a higher rate of syncope and transfer for HBO therapy in exposures from non-home appliance sources than in those from home appliance sources. Further research is needed to identify public health prevention measures in addition to home CO detectors to decrease severe CO poisoning.

KEYWORDS Carbon Monoxide, Environmental Toxicology, Unintentional Poisoning

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70. GorillaTM Glue Guy Gets Gastrostomy

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Background: Poison control centers (PCC) frequently manage glue ingestions, but most are inconsequential or result in minor symptoms. Severe outcomes have been to reported following ingestions of cyanoacrylate glues in humans and di-isocyanate glues in dogs. GorillaTMGlue (GG) contains urethane prepolymer and 4,4'diphenylmethane di-isocyanate. It is a unique non-toxic adhesive sold commonly for household repairs, and is known for its capacity to expand. From 2002-2019, our PCC received 166 calls regarding GG ingestions, with all outcomes being coded as either "minor" or "no effect". However, case reports previously describe the development of gastric outlet obstruction required surgical removal in dogs ingesting GG. Here we describe the first known human case of a GG ingestion that resulted in an exploratory laparotomy and gastrostomy.
Case Report: A 47-year-old-man with a history of bipolar II disorder presented to the emergency department 5 days after ingesting 3.5 oz of GG in an attempt to relieve several days of heartburn. Post-ingestion, he developed abdominal pain, decreased oral intake, nausea, and 7 episodes of hematemesis. He had no bowel movement for four days. Physical exam was notable for abdominal guarding and rebound tenderness noted maximally in the epigastrium. Laboratory investigations revealed a white blood cell count of 24.7k/uL. Computed tomography of the abdomen demonstrated a large, solid gastric mass along with wall thickening and inflammatory changes involving the gastric body and antrum (Figure 1). He underwent emergent exploratory laparotomy and gastrostomy and the foreign body was removed (Figure 2). The glue formed a gastric cast bezoar that was not adherent to the gastric mucosa (Figure 3). His post-operative course was complicated by fascia dehiscence requiring revision of fascial closure. He was discharged on post-admission day 13 and scheduled for surgery clinic follow-up 3 weeks later, but failed to present. The patient subsequently died of unknown cause.
Discussion: To our knowledge, this is the first reported case of a human ingestion of GG resulting in exploratory laparotomy and gastrostomy. The 4,4'diphenylmethane di-isocyanate, when mixed with gastric acid, expands more than 8-fold within a 2-hour period post-ingestion via a polymerization reaction. The resultant mass is firm, indigestible, and may obstruct the gastric outlet. Unlike cyanoacrylate glue ingestions, di-isocyanate ingestions do not adhere to the gastric mucosa. Operative removal of the foreign body is often required, and has been described in dogs ingesting as little as 2 oz. In our patient, 3.5 oz resulted in surgery. Signs reported in dogs include vomiting, hematemesis, abdominal distension and pain within 8 hours of ingestion. Radiographs are recommended as an initial screening test as they may detect the bezoar. If undetected, abdominal CT may reveal the etiology of symptoms, as in our patient. Surgical gastrostomy is often recommended for foreign body removal following di-isocyanate ingestions, as endoscopy removal is likely to fail.
Conclusion: Small ingestions of di-isocyanate glues, such as GG, can result in severe adverse events including gastric outlet obstruction, and may require surgical intervention. All symptomatic patients should be referred to a healthcare facility for imaging and possible surgical consult.

KEYWORDS Gorrilla Glue, Obstruction, Gastrostomy

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Table 1.

	Site of Exposure	Age	Sex	CO Level	Syncope	Elevated Troponin	HBO Therapy
Home Appliance	38 residential	43 ± 26	15 Male 23 Female	22 ± 9	10/38 patients (26%)	12/38 patients (32%)	11/38 patients (29%)
Non-home Appliance	27 residential 16 other	37 ± 19	32 Male 11 Female	23 ± 7	25/43 patients (60%)	8/43 patients (18%)	24/43 patients (56%)
P-value		0.86	0.001	0.27	0.004	0.18	0.01

71. Hemolytic crisis in non G6PD deficient patient after ingestion of Acalypha indica: a case report

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Objective: Acalypha indica is a tropical plant, which is widely used as a traditional remedy in Asia. However, information about its adverse effects have been rarely reported. All reports were acute hemolytic crisis in Glucose-6-phosphate dehydrogenase (G6PD) deficient patient, caused by oxidative stress from ingestion uncooked leaves. We report a case with normal G6PD status, who presented with acute severe hemolysis after ingesting Acalypha indica leaves.
Case report: A 55-year-old Thai male with history of high blood pressure and dyslipidemia, presented with fever, headache and myalgia after ingesting approximately one handful of Acalypha indica' s uncooked leaves as a health supplement 2 days before presentation. One day later, he developed dyspnea on exertion and started to pass dark brown urine. On physical examination, he had low graded fever and mild tachycardia. Pallor and marked jaundice were also noted. His liver span was below right costal margin without tenderness. Urinalysis revealed microscopic hematuria. He was primarily diagnosed with left ureteric stone complicated with urinary tract infection and was treated with an oral antibiotic. Laboratory results on the day 1 included acute anemia from severe intravascular hemolysis with suspected G6PD deficiency (hematocrit 20%, reticulocytes 7%, normochromic normocytic erythrocytes with basket cells, bite cells, ghost cells and blister cells); indirect hyperbilirubinemia (total bilirubin 8 U/L, direct bilirubin 1.48 U/L); and acute renal failure (BUN 56 mg/dL, serum creatinine 3.56 mg/dL). Blood transfusion, aggressive hydration and forced diuresis were given. On day 2, he developed respiratory failure and was intubated and transferred to the ICU. Progressively worsening of renal function with uremia, volume overload and severe metabolic acidosis were also documented. He, therefore, received intermittent hemodialysis on the fourth day and improved gradually later on. Two months later, G6PD activity was tested and yielded normal result. Patient was lost to follow-up at 3 months after the event.
Case discussion: There are various types of phytochemical in Acalypa indica including seven cyanogenic compounds may cause cyanide intoxication in livestocks. Cyanogenic glycoside is a potent catalase inhibitor which prevents cell injury from reactive oxidant species. Accumulation of oxidative species lead to intravascular hemolysis, especially in G6PD deficient patients who lack NADPH, an antioxidative mechanism. To our knowledge, we report the first case demonstrating intravascular hemolysis after ingesting uncooked Acalypa indica in a patient without G6PD deficiency.
Conclusion: Ingestion of uncooked Acalypha indica leaves can cause severe intravascular hemolysis in patient without G6PD deficiency. This could be explained with cyanogenic glycoside, a potent catalase inhibitor, and other oxidative stressors contained in this plant.

KEYWORDS Acalypha indica, Traditional remedy, Intravascular hemolysis

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72. Homemade slime exposures reported to poison centers

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Background: Homemade slime is promoted as a do-it-yourself project that can be done at home or at school. One popular version of homemade slime uses water, borax (sodium tetraborate), and Elmer's glue. Although the recipe for homemade slime and its use have been around for years, in 2016 and 2017 its promotion on social media, including YouTube and Instagram, increased its popularity. Concomitant with this increased interest in homemade slime have been reports of injuries when making or playing with the substance. The objective of this study was to characterize homemade slime exposures reported to poison centers.
Methods: Case were all exposures involving homemade slime reported to the statewide poison center system during January 2003-December 2018 where the patient age was 0-19 years. In order to identify potential cases, the Notes field was reviewed for any mention of "slime," "borax," "boric," or "borate." These records were reviewed to determine whether the record appeared to be an exposure to homemade slime, and if so, whether borax was a component of the homemade slime. The distribution of homemade slime exposures was determined for various factors.
Results: A total of 415 exposures were identified. During 2000-2015, the annual number of exposures ranged 1-9 per year; there were 16 exposures in 2016, 172 in 2017, and 172 in 2018. Borax was specifically mentioned in 266 (64.1%) exposures, not mentioned in 99 (23.9%), and unknown in 50 (12.0%). The patient age was 219 (52.8%) 0-5 years, 150 (36.1%) 6-12 years, and 46 (11.1%) 13-19 years; 249 (60.0%) of the patients were female, 163 (39.3%) male, and 3 (0.7%) unknown gender. The exposure route was 365 (88.0%) ingestion, 54 (13.0%) dermal, 9 (2.2%) inhalation, and 5 (1.2%) ocular. The exposure site was 359 (86.5%) own residence, 50 (12.0%) school, and 6 (1.4%) other or unknown locations. Most (n = 353, 85.1%) of the patients were managed on site, 50 (12.0%) were already at or en route to a healthcare facility, 2 (0.5%) were referred to a healthcare facility, and 10 (2.4%) were managed at other or unknown locations. The medical outcome was 63 (15.2%) no effect, 40 (9.6%) minor effect, 6 (1.4%) moderate effect, 43 (10.4%) not followed-judged nontoxic, 248 (59.8%) not followed-minimal clinical effects possible, 4 (1.0%) unable to follow-potentially toxic, and 11 (2.7%) unrelated effect. The most common clinical effects were vomiting (n= 37, 8.9%), abdominal pain (n = 18, 4.3%), dermal irritation/pain (n = 17, 4.1%), erythema/flushed (n = 15, 3.6%), and nausea (n = 15, 3.6%).
Conclusion: This poison center system observed a slight increase in the number of homemade slime exposures in 2016 and a much greater increase in 2017. Most of the exposures involved borax, but this may be related to the manner in which cases were identified. The majority of patients were 0-5 years and female. Most of the exposures occurred by ingestion and occurred at the patient's home or school. The majority of exposures tended not to have serious outcomes and were managed outside of a healthcare facility.

KEYWORDS Slime, homemade, boric acid

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73. When smoothies don't go smoothly: Cherry pit ingestions reported to poison centers

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Background: Cherry pits contain amygdalin, a cyanogenic glycoside hydrolyzed to cyanide in the gastrointestinal system and absorbed. Intact cherry pits do not release cyanide. Ingestion of one-two cherry pits is nontoxic, and toxicity is uncommon after accidental ingestion.

However, serious adverse effects after ingestion of cherry pits have been reported in the news. The objective of this study was to describe cherry pit ingestions reported to poison centers.
Methods: Cases were all cherry pit ingestions reported to a statewide poison center system during 2003- 2018. All records involving cherry ingestions were reviewed to determine whether cherry pits alone or whole cherries including the pits were ingested. The distribution of cherry pit ingestions was determined for various factors related to patient demographics, ingestion circumstances, management, and outcome.
Results: A total of 698 cherry pit ingestions were identified. The record mentioned ingestion of only the pits in 511 (73.2%) of the ingestions and the cherries and pits in 187 (26.8%); 93 (13.3%) of the ingestions involved a smoothie, shake, or juice. Of the 592 ingestions where the number of pits was mentioned, 308 (52.0%) involved one pit (mean 4.4 pits, range 1-50 pits). The annual number of ingestions increased from 9 in 2000 to 130 in 2018 with 405 (58.0%) of the ingestions occurring in 2015-2018. Most (n = 613, 87.8%) of the ingestions occurred in May-August. The patient age distribution was 359 (51.4%) 0-5 years, 121 (17.3%) 6-12 years, 22 (3.2%) 13-19 years, 152 (21.8%) 20 years or more, and 44 (6.3%) unknown age; 392 (56.2%) of the patients were female and 306 (43.8%) male. The ingestion site was 681 (97.6%) patient's own residence, 11 (1.6%) another residence, and 6 (0.9%) at other or unknown sites. The ingestion reason was 681 (97.6%) unintentional, 12 (1.7%) intentional, 3 (0.4%) adverse reaction, and 2 (0.3%) unspecified other reason. Most (n = 651, 93.3%) of the patients were managed on-site, 33 (4.7%) were already at or en route to a healthcare facility, 12 (1.7%) were referred to a healthcare facility, and 2 (0.3%) were managed at an unknown location. The medical outcome was 165 (23.6%) no effect, 9 (1.3%) minor effect, 2 (0.3%) moderate effect, 156 (22.3%) not followed-judged nontoxic, 349 (50.0%) not followed-minimal clinical effects possible, 7 (1.0%) unable to follow-potentially toxic, and 10 (1.4%) unrelated effect. The most commonly reported clinical effects were abdominal pain (n = 12, 1.7%), vomiting (n = 11, 1.6%), and nausea (n = 7, 1.0%). The most frequent treatments were dilute/irrigate/wash (n = 260, 37.2%) and food/snack (n = 92, 13.2%).
Conclusion: There was an increase in cherry pit ingestions over the 19-year period. The majority of the ingestions involved a single cherry pit; 13% involved cherry pits inadvertently included when preparing a smoothie, shake, or juice. Most of the ingestions were reported during May-August. The majority of patients were children and female. In spite of the warnings about the danger of cherry pits in the news and on the Internet, most cherry pit ingestions were managed outside of a healthcare facility and did not result in a serious outcome.

KEYWORDS Cherry pit, cyanogenic glycoside, cyanide

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74. Acids as casual agent of pediatric burns

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Introduction: Burns can result in incapacitating injuries independently of the causative agent. chemical burns, although not so common, are associated with systemic toxicity. The American Burn Association (ABA) report that chemical burns represent 3% of burns in USA. Chemicals can cause direct cell injury through multiple mechanisms such as oxidation, reduction, denaturation, and dehydration depending on the substance. These reactions are often exothermic (a chemical reaction that releases energy via light or heat), which can add thermal injury to the chemical injury. among the main groups of corrosive chemicals play an important role The Acids, these are chemical agents that release hydrogen ions when added to water with a pH below 7 as Sulfuric, nitric, hydrofluoric, hydrochloric, acetic acid, formic, phosphoric, phenols, chloroacetic and methacrylic acid among others. Due to its characteristic it can produce coagulation necrosis, limiting the depth and penetration of the burn but causing more systemic toxicity than other chemicals, including metabolic acidosis which was observed in all these patients studied.
Method: A retrospective review of all burn injuries observed in children under the age of 21 years evaluated by the Pediatric Surgery and Burn Service of a pediatric tertiary hospital during 2010 – 2017. Information collate included age, sex, site, extension and type of burn, substance, mechanism of injury, associated morbidity and complications.
Results: 1,237 patients were evaluated due to burns, 41 cases were chemical burns of which in 20 acid was the causal agent. All cases were consulted with the local Poison Center. Acids were the most common substance causing chemical burns (48.8%), specifically nail primer with methacrylic acid as main compound (42.5%), sulfuric acid (2.4%), nitric acid (2.4%) and an unspecific acid (2.4%). All patients presented metabolic acidosis. Most cases were male patients, all patients were ages between 1-3 years old, 100% of patients required hospitalization; (15.0%) were severe burns – more than 20% TBSA –. admitted to intensive care unit. 90% of patients had second degree burns, the one caused by nitric oxide had third degree burns and required mechanical ventilation due to respiratory failure by inhalation injury. The length of stay was 6 ± 5 days. All patients were managed following the guidelines of burn care by ABA with closed advanced dressings without need of skin graft.
Conclusion: By extension, depth of injuries and complications as metabolic acidosis and respiratory failure all patients required hospital admission for care. 73% of burn injuries including pediatric chemical burns occurred in the house. The children are curious, and all chemical products must be unreachable because these corrosive substances like cleaning and cosmetics can cause short-long-term and lifelong health complications mainly if are treated inadequately cutaneous, inhaled or if ingested. The most common acid was methacrylic acid a compound of nail primer which is widely used by female adults, as such increasing the chance of exposure to children and risk of burns with severe sequels; this is why more alarming information about the dangers of this product should be more available to people.

KEYWORDS Acid burns, pediatric burns, chemical burns

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75. Toxic brain death mimics

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Background: Certain xenobiotics, including baclofen and amitriptyline, are reported to mimic brain death in overdose. Loss of brainstem reflexes often triggers a physician to consider brain death. This leads to extensive testing, monitoring, and the introduction of further stress to the family of a patient. No consensus exists on which xenobiotic toxicities may obscure the diagnosis of brain death. The aim of our study is to provide a summary of the toxicologic brain death mimics currently reported in the literature in order to provide guidance for physicians assessing brain death in a patient after a toxic exposure.
Methods: We established an a priori definition of "brain death mimic" as an unresponsive patient requiring mechanical ventilation with fixed pupils, absent corneal reflexes, and absence of two additional

Primary Xenobiotic	Drug Class	Cases	Dose Range	Age Range, years	Brain death criteria met	Brain death diagnosis failed	Duration of symptoms	Discharge condition	Miscellaneous
Baclofen	Gaba B agonist	8	450-2000mg	25-59yo	Pupils fixed Absent corneal and two additional reflexes Intubated Miscellaneous additional findings	Miscellaneous	12 hours - 5 days	Baseline – 7 Unknown - 1	
Secobarbital/ phenobarbital	Barbiturate	1	unknown	69yo	Pupils fixed absent corneal, DTR No response to stimuli intubated		48 hours	Unknown	Serum barbiturate concentration 5mg/100ml
Diazepam	Benzodiazepine	1	unknown	24yo	Pupils fixed and dilated, Absent corneal reflex, gag, no response to painful stimuli; Intubated/apneic	Occasional gag reflex	~ 2 days	Baseline health	Additional OD: Ethylene glycol
Bretylium	Antidysrhythmic	1	12mg/kg/hr infusion	6 day old	Pupils fixed and dilated Absent corneal, oculocephalic, gag, oculovestibular, reflex. No motor activity No respiratory effort on vent	Occasional random movement of extremities	24 hr	Discharged home, developing well	Bretylium concentration 17ug/ml (Therapeutic ~1.3ug/ml)
Bupropion	Dopamine/ Norepinephrine Reuptake inhibitor	1	unknown	29yo	Pupils fixed and dilated Absent corneal, oculocephalic, gag, oculovestibular, plantar reflex. Intubated/apneic	CT head wnl, DTR present but diminished	24 hr	Baseline health	Additional OD: gabapentin 2.1g Bupropion concentration 1441ng/ml (reference 50-100ng/ml)
Poison Hemlock	Nicotinic	1	unknown	28yo	Pupils fixed Absent corneal, oculocephalic, gag reflex, Intubated/no spontaneous respirations	Occasional sluggish pupils	6 days	Baseline health	
Lidocaine	Sodium channel blockade	1	Variable bolus and drip	60yo	Pupils fixed Absent corneal, gag, oculocephalic, plantar reflexes. Absent DTR except 1 bicep Intubated	2/4 reflex in one bicep	5 hours	To rehab; Cognitive impairment, bilateral leg amputations	
Magnesium		1	50 gram bolus	27yo	Pupils fixed and dilated Absent corneal, oculocephalic reflexes, "areflexia" Intubated		6 hour	Baseline health	
Organophosphate	Organophosphate	2	"50ml" of phorate;	28yo; 72yo	Pupils fixed Absent corneal, oculocephalic, and DTR Intubated/Apnea		4-5 days	Baseline health	
Snake Bite	Envenomation; neurotoxic	8	N/A	6-45yo	Pupils fixed and dilated Absent motor responses Absent corneal, and oculocephalic reflex Intubated/apneic	Miscellaneous	12 hours – 4 days	Baseline health – 7 Residual dilated pupils - 1	All cases occurred in India
Tricyclic antidepressant	Tricyclic antidepressant	2	Doxepin Unknown; Amitriptyline 9g	24yo; 46yo	Pupils fixed Absent corneal reflexes "Areflexia" Intubated/ apneic		8-24 hours		
Lagocephalus scleratus fish ingestion	Tetrodotoxin	1	N/A	80 / M	Pupils fixed/dilated Absent corneal, oculocephalic, caloric "areflexia" Intubated/apneic		18 hours		
Valproate	Antiepileptic	1	unknown	61 / F	"absence of brainstem reflexes, including pupillary light reaction"		"within hours"		Valproate 12,430umol/L (therapeutic 350-700umol/L)
Zolpidem	Nonbenzodiazepine Sedative	1	unknown	40 / F	Pupils fixed Absent corneal, vestibulo-ocular reflex unresponsive to painful stimuli, Intubated		72 hours		

brainstem reflexes, but who did not otherwise meet full brain death criteria. We began a systematic review of the literature following Cochrane guidelines using MEDLINE, EMBASE, and Google Scholar to find case reports and/or case series in English, French, and Spanish that met criteria, and hand searched the results. The MEDLINE search from years 1970-present provided preliminary results. Once we collected the cases, we recorded xenobiotic dose, duration of physical exam mimicking brain death, and how the cases failed to meet full brain death criteria, if available.
Results: MEDLINE results identified 393 hits of which 30 cases from 16 different substances met the a priori definition of "brain death mimic:" baclofen (8 cases), secobarbital/phenobarbital (1 case), diazepam (+ ethylene glycol) (1 case), bretylium (1 case), bupropion (+ gabapentin) (1 case), poison hemlock (1 case), lidocaine (1 case), iatrogenic magnesium overdose (1 case), organophosphates (2 cases), snake envenomation (8 cases), amitriptyline (1 case), doxepin (1 case), ingestion of tetrodotoxin producing fish (1 case), valproate (1 case), and zolpidem (1 case). Nearly all patients failed to fulfill brain death criteria by virtue of having normal neuroimaging or intermittent trace presence of at least one brainstem reflex.
Discussion: Preliminary search results of this systematic review identified 16 different substances from 14 classes of xenobiotics that met a priori definition of brain death mimic, leading physicians to consider or pursue brain death evaluation. All patients survived to discharge home or a psychiatric facility, and almost all at their baseline physical health. There are several limitations to our search. A physician who recognizes drug intoxication/effect as an exclusion criterion for brain death diagnosis would not report a case as a "brain death mimic". We found other cases in which a physician considered brain death but did not meet our a priori definition. Lastly, definition of brain death has evolved over the 40-year search period, which may have influenced reported data.
Conclusion: Prompt and accurate identification of brain death is important for appropriate utilization of intensive health care resources for living patients, organ donation, and counseling to families. The xenobiotics in this study should be considered in cases of poisoning resulting in loss of brainstem reflexes.

KEYWORDS Brain Death, Overdose, Coma

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76. Prevalence and Characteristics of Hydrofluoric Acid Ingestions as reported to the National Poison Data System: 2007-2017

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Background: Hydrofluoric acid (HF) ingestions are classically thought of as invariably fatal. The risk of serious systemic toxicity is directly proportional to both the concentration and the affected surface area. Ingestions pose a higher risk of morbidity and mortality given the potential extensive involvement of the esophagus and stomach. However, aside from case reports, we are only aware of one published retrospective study that reported a 2% fatality rate and 9% systemic toxicity rate. These results appear to directly contradict the traditional belief about the risks associated with HF ingestions.
Methods: This is a retrospective chart review of HF ingestions reported to the National Poison Data System (NPDS) between 2007-2017. We describe the patient demographics, intent, signs and symptoms, treatment modalities, and case outcomes. Case narratives were requested from all poison centers to further evaluate if an ingestion took place, the severity of the ingestion, the scenario, as well as specific electrolyte abnormalities and treatments provided. A logistic mixed model regression was performed to determine the effect of route of exposure on the severity of patient outcome.
Results: A total of 7,740 total HF exposures and 893 HF ingestions were recorded in the NPDS during the study period. We excluded 239 cases due to non-exposure or no known outcome, leaving 654 ingestions. Fourteen cases were from closed poison centers, and 640 case narratives were requested. We received 396 (60.6%) narratives from 33/55 (60%) poison centers. Of these narratives, the median age was 31.5 (IQR =9.0 – 48.5) years and 71.4% (95%CI =65.9 – 76.4) were male. After reviewing the narratives, an additional 58.1% were excluded as they were deemed to not be an actual ingestion and approximately 33.8% of the cases involved a mouthful or less of ingestion. Eight percent (95%CI =5.1 – 12.5) involved an ingestion of more than a mouthful. The majority of cases (59%) involved HF concentrations less than 20% and 24% involved concentrations greater than 50%. The most common exposure scenario (42.2%) was that the HF was stored in a drinking container (95%CI =33.7 – 51.7). Death occurred in 5.8% (95%CI =3.1 – 10.8) with an odds ratio of death as severity of ingestion increased of 2.36 (p = 0.002). Hypocalcemia occurred in 34.2% (95%CI =21.7 – 49.2) and hypomagnesemia in 27.1% (95%CI =16.4 – 41.4). Oral calcium was administered in 46.6% (95%CI =36.6 – 56.8) of patients.
Conclusion: The mortality associated with HF ingestions is higher than previously described although it represents a relatively small percentage of all ingestions, despite commonly finding electrolyte disturbances. The NPDS coding remains an issue as a large number of cases were miscoded as an ingestion.

KEYWORDS Hydrofluoric Acid, Ingestion, Mortality

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77. Valproic acid-induced thrombocytopenia-related spontaneous hemorrhage

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Background: Valproic acid (VPA) is an antiepileptic utilized for management of a variety of disease states. While benefits of therapy have been well described, VPA-induced coagulation changes can limit use. Thrombocytopenia (TPA) occurs in 12-18% of patients, but rarely requires discontinuation. We report a case of VPA-induced TPA-associated spontaneous subdural hemorrhage (SDH).
Case: A 57 year old female presented with bleeding from the mouth and vaginal area and multiple atraumatic contusions and ecchymosis all throughout the chest, abdomen, back, upper and lower extremities and left preorbital area. She had a past medical history of generalized anxiety disorder and choreiform movements. Approximately seven weeks prior to arrival, patient was admitted for a traumatic subdural hematoma (SDH) with a complicated hospital course characterized by bacteremia. During hospital stay, three weeks prior to admission, patient was initiated on VPA for management of choreiform movements. On arrival, patient was found to be anemic, thrombocytopenic and CT scan revealed bilateral SDH with a midline shift. Notable labs at admission include: red blood cells 2.75 x 106/µL, platelets 4,000/µL, hemoglobin 7.3 g/dL, hematocrit 23.1%, prothrombin time (PT) 15.7 secs, partial thromboplastin time (PTT) 30.4 secs, fibrinogen 467 mg/dL, VPA level 26.3 µ/mL. Thromboelastography

(TEG) releveled normal values, with the exception of decreased maximum amplitude of 33.4 mm (reference range 52-71 mm). At previous hospital discharge, 15 days prior to arrival, her hemoglobin and platelets were within the reference range. Despite transfusion of platelets on hospital days one and two (Table 1), the patient's platelet decreased on day four and was initiated on steroids and administered two doses of immunoglobulin. After discontinuation of VPA, transfusions and administration of steroids and immune globulin, the patients platelets continued to rise after day four (Figure 1). During the hospital stay, the patient was worked up for all other causes of TPA, including disseminated intravascular coagulation (DIC), which were all negative.

Discussion: Many studies have reported hematologic abnormalities associated with VPA administration, however, clinical relevance is still debated as there are few case reports of significant bleeding related to VPA. In general, the adverse effects associated with the hematologic abnormalities are mild at presentation (i.e. bruising, petechia, hematoma and epistaxis) or found in laboratory assays. Hemostatic abnormalities reported with VPA include TPA, platelet dysfunction and decreased fibrinogen and von Willebrand factor levels. Previous reports of systemic bleeding and intracranial hemorrhage associate with VPA are in the setting of decreased fibrinogen levels. The absence of any other cause of TPA, as well as the temporal relationship of the initiation of VPA, support the diagnosis of VPA-induced TPA. To our knowledge, this is the first case report of spontaneous systemic bleeding and intracranial hemorrhage due to VPA-induced thrombocytopenia in the setting of normal fibrinogen levels.

KEYWORDS Adverse drug event, valproic acid, intracranial hemorrhage

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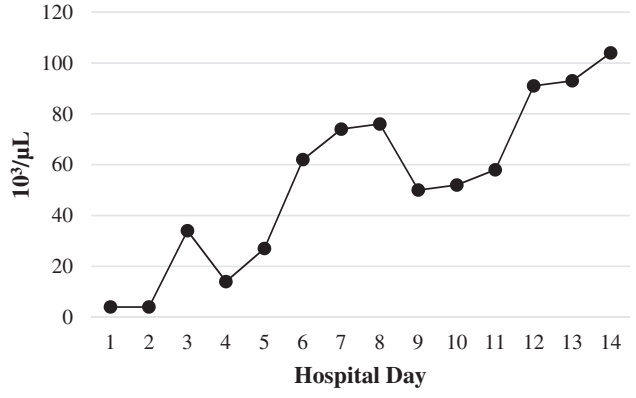

Figure 1 Platelet Count.

Table 1 Blood Product Administration.

Hospital Day	Units	Description	Volume (mL)
1	1	O + Platelet; random donor, leukocyte reduced, CMV -, irradiated	486
1	1	O + Platelet; random donor	232
2	1	O + PRBC leukocyte reduced, CMV negative	342
2	1	O + PRBC leukocyte reduced	350
2	1	O + Platelet; random donor, leukocyte reduced	241
2	1	O + Platelet; single donor, leukocyte reduced	294
2	1	A + Platelet; single donor, leukocyte reduced	251
10	1	O + PRBC leukocyte reduced	250

78. CNS Explosion: Seizure after C-4 ingestion in active duty service member

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Background/Objectives: C-4, also known as Composition 4 is a common explosive used by the military. Service members, particularly those involved with demolition and in the explosive ordnance disposal (EOD) teams may handle the material often for training and day to day missions. The explosive properties of C-4 primarily come from a nitroamine termed "Research Department Explosive" or "RDX" for short. C-4 is a mixture of RDX, a plasticizer (dioctyl sebacate) and a binder (polyisobutylene and oil).

Case Report: Our poison center was consulted on a previously healthy, 22 year old, active duty service member who presented with seizure while on a training exercise. The member was training as part of a military EOD team. Seizure was aborted with 2 mg of lorazepam, and the patient was observed in the emergency department. He was given IV fluids, and his work up to include CBC, Head CT, EKG were unactionable. Lab analysis revealed acute kidney injury (AKI) which resolved with fluids. He was prescribed antiepileptics and then discharged back to duty. Upon follow up 24 hours later, the patient admitted that he, and 3 other service members had eaten an unknown quantity of C-4 explosive material while out in training. The three other service members, all in their early 20s and previously healthy, were referred for evaluation in the emergency department. Two of the service members revealed that they had GI symptoms shortly after the ingestion and were found to have AKI. One member was asymptomatic and had normal laboratory evaluation. All were admitted for observation and serial laboratory work. Those that had AKI had subsequent resolution following IV fluid administration. All service members were discharged and returned to duty after 24 hours of medical evaluation.

Case Discussion: Few reports describing ingestion of C-4 and the characteristics of human toxicity exist. It is reported that individuals can experience seizure, nausea and vomiting, hematuria and elevation of transaminases. Motivation for the consumption has been reported to potentially be a rite of passage and C-4 may also have a euphoric effect. In this case, 4 individuals with varying presentation admitted to consuming C-4. Liver enzymes were normal. Limitations of this report include the lack of confirmatory testing detecting RDX concentrations in the serum. Samples were collected but due to the remote nature of the evaluation and presentation, there were challenges in getting the samples sent for analysis. However, given the history of ingestion from the patients as well as the timing of presentation, it is plausible that the ingestion of C-4 was contributory to the presentation. Furthermore, since these service members were out in the field during training, other environmental exposures that were not recognized or accounted for could have been responsible or contributory to their presentation.

Conclusion: RDX is a nitroamine, and is a primary ingredient in C-4 explosives. Consumption can lead to seizure, acute kidney injury and elevated transaminases.

KEYWORDS RDX, nitroamine, C-4 Ingestion

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79. Characterization of acute exposures to cannabis resins

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Background: As more states legalize its use, poison centers and healthcare providers should expect to treat increasing exposures to cannabis, including relatively novel high-potency concentrated products like resins.
Objective: To characterize the products associated with and clinical effects of acute exposures to cannabis resins.
Methods: This is a sub-analysis of data from a previously published study (Acute Cannabis Toxicity, Clinical Toxicology, Jan 2019) of 253 subjects for whom the Oregon Poison Center was contacted about an acute cannabis exposure between 12/4/2015 - 4/15/2017. A data collection instrument prospectively captured cannabis exposure and product data including route of exposure, cannabis type, and clinical information (vitals, signs and symptoms of toxicity, treatments received, and disposition). Categories of heart rate and blood pressure values were defined according to published guidelines. Subjects were included in this sub-analysis if they were exposed to a cannabis resin product. Resin was defined as a type of concentrated cannabis typically characterized as a solid or semi-solid product whereby tetrahydrocannabinol is extracted using high heat and pressure or a hydrocarbon solvent (e.g. butane). Any mention of "dab" or "dabbing" was considered to be an exposure to resin. Products were classified as liquids (not resins) if they were intended to be used in an electronic vaporizer. When combined into a food item, any resins or liquid concentrates were classified instead as edible products.
Results: Table 1 lists subject demographics, product details, vital signs, and neurologic clinical effects of acute exposure to cannabis resin, by route of exposure. Dose of exposure was infrequently reported: 5/26(19.2%) overall, 3/7(42.8%) ingestions and 2/19(10.5%) inhalations. In 2 ingestions, quantitative doses were reported, including "less than ½ gram" of cannabis wax in a 2-year-old and "1 gram" of dab material in a 57-year-old. In the other 3 exposures, non-quantitative doses were reported (e.g. "3 dabs"). One 9-month-old (ingestion) required intubation for respiratory and mental status depression. The teenager (inhalation) became agitated, received ketamine, and was admitted to an intensive care unit. One 13-month-old (ingestion) and a 16-year-old (inhalation) were admitted to a hospital ward. Otherwise, most subjects were discharged after evaluation in an emergency department (ED) (3/6 ingestions, 13/16 inhalations). Four subjects (1 ingestion, 3 inhalations) were managed at home.
Conclusion: In our study, exposures to resin occurred mostly in adolescents and young adults. As with other non-resin cannabis exposures, inhalation most commonly resulted in tachycardia and neuroexcitation. The only reports of bradycardia were in two pediatric ingestions. In all resin exposures, any reported excitation was more likely in adults and any reported sedation was more likely in children and adolescents. Hypertension may be more common after resin ingestion. Not enough dose information was available to analyze dose-dependent effects. Most subjects were discharged following ED evaluation or managed at home. Although more study is needed to fully characterize adverse effects of acute exposure to cannabis resin, our data may be useful to clinicians and poison centers hoping to better understand these exposures.

Table 1 Subject demographics, product details, vital signs, and neurologic clinical effects of acute exposure to cannabis resin, by route of exposure.

	Ingestion exposures (N = 7)	Inhalation exposures (N = 19)
Age range	9 months – 57 years	13 – 34 years
Median age	15 years	18 years*
Average age	18.6 years	19.6 years*
Male %	5 (71.4%)	14 (73.7%)
Manufacture source	2 (28.6%) homemade/ homegrown	3 (15.8%) dispensary/retail
	5 (71.4%) unknown	16 (84.2%) unknown
Ownership source**	3/4 (75.0%) family member/ caretaker	1/11 (9.1%) family member/caretaker
	1/4 (25.0%) friend	4/11 (36.4%) friend
		6/11 (54.5%) unknown
Heart rate	1 (14.3%) tachycardia	7 (36.8%) tachycardia
	2 (28.6%) bradycardia	3 (15.8%) normal
	1 (14.3%) normal	9 (47.4%) unknown
	3 (42.9%) unknown	
Blood pressure	3 (42.9%) hypertension	2 (10.5%) hypertension
	1 (14.3%) normal	4 (21.1%) normal
	3 (42.9%) unknown	13 (68.4%) unknown
Neurologic effects	2 (28.6%) excitation	8 (42.1%) excitation
	2 (28.6%) sedation	5 (26.3%) sedation
	1 (14.3%) excitation and sedation	2 (10.5%) excitation and sedation
	2 (28.6%) none	2 (10.5%) other
		2 (10.5%) none
Any excitation, by age	25.0% children/adolescents	37.5% children/adolescents
	66.7% adults	77.8% adults
Any sedation, by age	75.0% children/adolescents	62.5% children/adolescents
	0% adults	22.2% adults

*Resin inhalations included one "teenager" and one "adult." Both were excluded from median and average age calculations.
**For subjects <21 years (the legal minimum age for retail cannabis use/ possession), the ownership source was identified as to whom the cannabis belonged or from whom it was obtained.

KEYWORDS Cannabis, resins, exposures

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80. Reduced-Dose Intramuscular Ketamine for Severe Agitation in an Academic Emergency Department: A Case Series

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Background: Ketamine is a dissociative anesthetic used increasingly in pre-hospital and emergency department (ED) settings to safely sedate severely agitated patients. It is an attractive option due to its rapid onset and unique mechanism of action. However, the adverse effects associated with the use of ketamine include sialorrhea, apnea, laryngospasm, hypertension, and tachycardia, and may be related to dose or rate of administration. Studies to date report conflicting results on whether a dose-dependent association with intubation following IM ketamine for agitation exists, though this is likely difficult to establish given the numerous factors that may lead to patient intubations. Most studies evaluating IM ketamine for agitation have utilized a dose of 4-6mg/kg with maximum doses 500-600mg. However, this dose has not been validated via a dose-finding study to ensure it is not in excess of what a patient requires to be properly sedated. Therefore, the purpose of this case series was to describe the efficacy and safety of a reduced-dose (2mg/kg) IM ketamine protocol.
Methods: A protocol was implemented at our institution utilizing IM ketamine at a dose of 2mg/kg (maximum 200mg) for patients ≥18 years old with severe agitation, excited delirium, or agitation following a trauma. This dose can be repeated with an additional 2mg/kg (maximum 200mg) dose after 5 minutes, if necessary, to a cumulative maximum dose of 4mg/kg (400mg). All patients receiving IM ketamine for agitation in the emergency department via this reduced dose protocol were included. Successful sedation of the agitated patient was defined as either documentation from a healthcare provider or a lack of additional sedating medication administration for 30 minutes following administration of IM ketamine.
Results: There were 15 patients who met the inclusion criteria for this case series. Of these patients, 12 (80%) were male with a median age of 33 years and median weight of 75 kg. Agitation was controlled in 13 of 15 patients (87%) following a single dose of reduced-dose ketamine. The median total dose administered was 157.5mg and the median weight-based dose was 2mg/kg. The median total dose was 157.5mg

(IQR 150-200) and the median weight-based dose was 2mg/kg (IQR 1.9-2.1). No patients had any documented adverse effects associated with the use of ketamine. We also found no instances of intubation due to over-sedation, apnea, or clinically significant vital sign variations during this time-period.
Conclusions: Based on our institutional protocol (2mg/kg as the initial dose), our case series included patients who received less than half the commonly studied dose. Almost 90% of patients (13 of 15) were properly sedated following the first ketamine dose with no instances of adverse effects documented. The limitations of this report include the small sample size, absence of randomization, potential deficiency of documentation due to retrospective nature, and lack of comparator group. Based on this case series, it appears that a lower dose of 2mg/kg of IM ketamine could be utilized to safely sedate most agitated patients in the ED to minimize the risk of adverse effects and intubation.

KEYWORDS Ketamine, Agitation, Safety

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81. Late Thrombocytopenia After Treatment with Crotalidae Polyvalent Immune Fab and Crotalidae Immune F(ab')2

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Background: Two FDA-approved antivenoms (AV) are available for treatment of rattlesnake envenomation (RSE) with variable elimination half-lives: Crotalidae Polyvalent Immune Fab (Fab) (Crofab) 12-23 hr t1/2; and the newer equine-derived Crotalidae Immune F(ab')2 (F(ab')2) (Anavip) 133 hr t1/2. A clinical trial by Bush et al reported the newer antivenom conferred a decreased incidence of delayed or recurrent hemotoxicity. In the Bush study the 6 subjects in the F(ab')2 treated arms that developed delayed hemotoxicity were from a single center in southern California. We now report a case in Arizona with delayed thrombocytopenia in a patient that received both antivenoms.
Case Report: A 44-year-old man with a RSE to the right thumb presented to an outside ED and received 6 vials Fab for progressive upper extremity pain and edema. No systemic signs of envenomation were reported; however, an episode of hypotension (160/95 to 74/41 mmHg) occurred 2.5 hours after the bite. Although closely timed, a temporal relationship to antivenom infusion could not be definitively established. Initial lab results prior to antivenom and ~1.5 hours after the bite revealed platelets (plt) 217 K/mm^3 (130-450 K/mm^3); PT 13.2 s (9.4-12.5 s); and fibrinogen 288 mg/dl (200-465 mg/dl). The patient was transferred to our center where a history of previous RSE treated uneventfully with Fab 14 years prior was obtained. Exam revealed normal vital signs, puncture marks proximal to the nail bed of the right thumb with oozing, marked edema and tenderness extending to the elbow with erythematous streaks along the medial upper arm, along with right axillary tenderness. An additional dose of 4 vials Fab was begun for severe edema and pain, repeat lab studies were: plt 201 K/mm^3 (130-450 K/mm^3); PT 14.3 s; and fibrinogen 233 mg/dl. During Fab infusion the patient developed an urticarial rash (see photos) and recurrent hypotension without dyspnea, wheezing, or other complaints. The Fab infusion was stopped (~25% infused); famotidine, diphenhydramine, and 1L lactated ringers bolus were given with good response. Due to the adverse reaction, F(ab')$_2$, 10 vials were obtained and administered without problems based on severity and progressive signs of envenomation. In total the patient received 30 vials of F(ab')$_2$ and estimated 7 vials of Fab. Thrombocytopenia (Plt 111 K/mm^3) occurred 62 hours after the envenomation with a plt nadir of 67 K/mm^3 (141 hours post-envenomation) which gradually resolved without complications or re-treatment.
Conclusion: Despite availability of a F(ab')2 antivenom with a longer elimination half-life, continued monitoring for late or delayed thrombocytopenia or coagulopathy appears warranted at present, and this may be most important in patients partially treated with Fab.

KEYWORDS Rattlesnake envenomation, delayed thrombocytopenia, antivenom adverse reaction

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82. Midwest Mushroom Madness: A Regional Poison Control Center's Experience

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Background: Mushrooms exposures are frequent, though difficult to characterize. They are classically associated with wooded areas and moist climates such as Eastern Europe, Asia, and the North American Pacific coast. Exposures from other regions are poorly described. We sought to characterize mushroom exposures reported to a North American Midwest regional poison control center (PCC).
Methods: The database of a North American Midwest regional PCC was queried for all mushroom exposures from 1/01/2013 to 12/31/2018. Only human exposures were selected. After training to ensure inter-rater reliability, the following data points were abstracted: age, sex, date of exposure, reason for exposure, management site and disposition, laboratory values, medical outcome, presence and timing of GI symptoms, interventions, mycologist consultation, presence of mushroom picture, and identification of the mushroom. All data was analyzed using Microsoft Excel (Redmond, WA).
Results: 441 cases were identified. The average age was 13.2 years (SD 18.5) with 259 cases (59%) being 5 years or younger. A majority of cases were male (n = 245). The most cases were reported in 2013 (n = 89) and May and Aug were the most common months of exposures with 101 and 111 cases, respectively. Unintentional ingestion was the most common reason for exposure (n = 307) though there were 38 cases of misuse/abuse. Most cases were managed at home (n = 279) and resulted in no clinical effect (n = 257). Vomiting or diarrhea occurred in 135 cases (30.6%). The time to onset of these symptoms was 3.5 hours (SD 4.49) from mushroom ingestion. Most cases (n = 304) received either no intervention or basic dilution with PO liquids. Intravenous fluids were given in 76 cases and anti-emetics were given in 59 cases. N-acetylcysteine was given in 5 cases. AST was reported in 55 cases but in only 3 cases was it over 100 u/L (max 227 u/L). Seven cases had a creatinine kinase (CK) reported and 3 were over 1000 u/L, including a case with a max CK of 27,853 u/L after an unidentified mushroom ingestion. Fifty-six cases were admitted to the hospital, including 9 to a critical care. There were no deaths reported. A picture of the mushroom was available in 120 cases (27.2%) and a mycologist was consulted in 101 cases (22.9%). When the mycologist was consulted, the mushroom was identified in 75% of the cases (n = 76). Otherwise the mushroom was identified in 2.9% (n = 10) of cases. Chlorophyllum molybdites was the most commonly identified mushroom (n = 29). Table 1 lists the six most commonly identified mushrooms. In addition, 13 cases were reported as exposures to a hallucinogenic mushroom (Psilocybe sp.)
Conclusions: Mushroom exposures reported to this North American Midwest PCC were most common in summer months and typically

Table 1 Six Most Commonly Identified Mushrooms.

Mushroom	Number of Cases
Chlorophyllum molybdites	29
Amanita theirsii	6
Chlorophyllum sp.	5
Pleurotus ostreatus	4
Coprinopsis variegata	2
Agaricus campestris	2

involved unintentional exposures in young children. While vomiting and diarrhea occurred in approximately a third of cases, basic supportive care measures were typically sufficient and morbidity was minimal. In most cases the mushroom was never identified.

KEYWORDS Mushroom, Poison Control Center, mycologist

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83. A simple table to estimate hemodialysis dose for treatment toxic alcohol poisoning

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Background: Ethylene glycol (EG) and methanol are toxic alcohols amenable to hemodialysis (HD) for correction of acidosis and toxin removal in cases of poisoning. The lack of EG or methanol concentrations can complicate not only the diagnosis but also the management regarding duration and timing of HD and fomepizole. The dose of HD can be calculated for methanol or EG based on the equation t= [-V ln(20/A)/0.06k] where t is time, V is the Watson estimate of total body water, A is the initial toxin concentration in mg/dL, and k is 80% of the manufacturer specified dialyzer urea clearance. Unfortunately, patient data for the variables in this calculation are often unavailable to consulting poison centers, but also represent potentially dynamic variables for the bedside toxicologist or nephrologist. Further, it is the author's experience that many nephrologists dose HD in 3-4 hour blocks similar to outpatient renal replacement therapy resulting in the need for serial EG or methanol levels and subsequent delay in definitive toxin removal. These cases demonstrate the utility of a simple table (Figure 1) based on estimations of the variables above to guide the HD dose in the setting of toxic alcohol poisoning.

Case reports: Case 1: 73 yo 74 kg male presented with suspected antifreeze ingestion and altered mental status a pH of 7.14, bicarbonate 7, anion gap (AG) 25 and osmolar gap (OG) of 35. He was loaded with fomepizole and an EG concentration returned at 118mg/dl. A continuous 7 hour dose of HD was recommended with a repeat EG concentration of 9mg/dl post HD and normalization of acid base abnormalities.

Case 2: A 42 yo M 140 kg male presented after being found obtunded and initial laboratory findings of pH 7.1, bicarbonate of 4, AG 25 and OG 60. He was loaded with fomepizole and EG concentration returned at 143mg/dl and a continuous 13 hour HD dose was recommended. The patient received a 12 HD dose and immediate post HD EG concentration was 21 mg/dl.

Case 3:70 yo ~70kg M presented after being found with altered mental status and a prior history of EG ingestion. Initial findings pH 6.86, bicarbonate 6, AG 28, and an EG concentration of 532 mg/dl. Emergent dialysis was planned for 4 hours but upon poison center consultation 12 hours of HD was recommended and performed. Post HD EG concentration was 16mg/dl.

Case discussion: Estimation of the HD dose allows for an initial continuous HD dose and follow up EG concentration(s) rather than divided HD doses with serial EG concentrations. These cases demonstrate that a consulting poison center is able to gather needed information to make recommendations on HD dose utilizing this table with success in case series.

Conclusion: We present three cases of successful HD dose estimation by approximating a previously published method into a more practical representation in table format.

How to use this table:

1. Find your patient's weight in the first column.
2. Find your patient's toxic alcohol (ethylene glycol or methanol) concentration along the top row.
3. The intersection of that row and column give a crude estimate of dialysis time rounded to whole hou
4. This is a gross estimate rounded to the nearest hour. Very obese patients, extremely high levels, or slow functioning catheters will have the least accurate estimates (high or low depending).

Level / Weight	50-60 mg/dL	60-70 mg/dL	70-100 mg/dL	100-150 mg/dL	150-200 mg/dL	200-250 mg/dL	250-300 mg/dL	300-350 mg/dL	350-400 mg/dL	400-450 mg/dL	450-500 mg/dL	500-550 mg/dl	550-600 mg/dL	600-650 mg/dL	650-700 mg/dL
50 Kg	3	3	4	4	5	6	6	7	7	8	8	8	8	9	9
55 Kg	3	3	4	5	6	7	7	8	8	8	9	9	9	10	10
60 Kg	3	4	4	6	6	7	7	8	9	9	9	10	10	10	11
65 Kg	3	4	5	6	7	8	8	9	10	10	10	11	11	11	11
70 Kg	4	4	5	6	7	9	9	10	10	11	11	11	12	12	12
75 Kg	4	4	5	7	8	9	9	10	11	11	12	12	13	13	13
80 Kg	4	5	6	7	9	10	10	11	12	12	13	13	14	14	14
85 Kg	4	5	6	8	9	10	10	12	13	13	13	14	14	15	15
90 Kg	5	6	7	8	10	11	11	12	13	14	14	15	15	16	16
95 Kg	5	6	7	9	10	11	12	13	14	14	15	16	16	16	17
100 Kg	5	6	7	9	11	12	12	14	15	15	16	16	17	17	18
120 Kg	6	7	9	11	13	15	15	17	18	18	19	20	21	21	21
140 Kg	7	8	10	13	15	17	17	19	21	21	22	23	24	24	25

*Assumptions: Hemodialysis is being run at 300ml/min and the manufacturer's estimated urea clearance for that cartridge at that rate is 250ml/min. Total body water is estimated by weight Kg * 0.6L/Kg.

If Height, weight, age, sex, dialysis flow rate, and manufacturers estimated urea clearances are known then the following equation can be utilized for a more accurate estimate. t= [-V ln(20/A)/0.06k]

t is time, V is the Watson estimate of total body water, A is the initial toxin concentration in mg/dL, and k is 80% of the manufacturer specified dialyzer urea clearance.

KEYWORDS Toxic Alcohol Poisoning, Hemodialysis, Alcohol Poisoning

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84. Do apple seeds keep the doctor away? Apple seed ingestions reported to poison centers

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Background: Apple seeds contain amygdalin, a cyanogenic glycoside hydrolyzed to cyanide in the gastrointestinal system and absorbed when the seeds are chewed or crushed. Apple seeds contain approximately 0.6-0.8 mg of hydrogen cyanide per gram of seed (average lethal dose of cyanide: approximately 50 mg orally for an adult). Swallowing whole apple seeds is not expected to cause symptoms because the seed coat protects the seeds from the digestive enzymes. Because of information on the Internet warning about the hazards of apple seed ingestion, poison centers may receive calls. The objective of this study was to describe apple seed ingestions reported to poison centers.
Methods: Cases were all apple seed ingestions reported to a statewide poison center system during 2003- 2018. All records involving apple ingestions were reviewed to determine whether apple seeds alone or whole apples or their cores including the seeds were ingested. The distribution of apple seed ingestions was determined for various factors related to patient demographics, ingestion circumstances, management, and outcome.
Results: A total of 329 apple seed ingestions were identified. Of the 106 ingestions where the number of seeds was mentioned, 46 (43.4%) involved one seed and 24 (22.6%) two seeds (mean 3.8 seeds, range 1-100 seeds). Of the 179 ingestions where the number of whole apples or cores was mentioned, 171 (95.5%) involved one apple/core (mean 1.2 apples/cores, range 1-15 apples/cores). There were no clear annual or seasonal trends. The patient age distribution was 273 (83.0%) 0-5 years, 20 (6.1%) 6-12 years, 6 (1.8%) 13-19 years, 28 (8.5%) 20 years or more, and 2 (0.6%) unknown age; 184 (55.9%) of the patients were male and 145 (44.1%) female. The ingestion site was 324 (98.5%) patient's own residence and 5 (1.5%) at other or unknown sites. The ingestion reason was 322 (97.9%) unintentional and 7 (2.1%) intentional. Most (n= 322, 97.9%) of the patients were managed on-site, 1 (0.3%) was already at or en route to a healthcare facility, 4 (1.2%) were referred to a healthcare facility, and 2 (0.6%) were managed at other or unknown locations. The medical outcome was 88 (26.7%) no effect, 3 (0.9%) minor effect, 91 (27.7%) not followed-judged nontoxic, 138 (41.9%) not followed-minimal clinical effects possible, 4 (1.2%) unable to follow-potentially toxic, and 5 (1.5%) unrelated effect. Vomiting was reported in two cases and diarrhea, nausea, fever/hyperthermia, agitated/irritable, dizziness/vertigo, drowsiness/lethargy, and muscle weakness each in a single case. The most commonly reported treatments were dilute/irrigate/wash (n= 107, 32.5%) and food/snack (n= 33, 10.0%).
Conclusion: The majority of apple seed ingestions involved 1-2 seeds or a single whole apple/core. Most patients were children age 0-5 years and male. The majority of the ingestions occurred at the patient's own residence and were unintentional. In spite of the warnings about the danger of apple seed ingestion on the Internet, most apple seed ingestions were managed outside of a healthcare facility and did not result in a serious outcome.

KEYWORDS Apple Seed, Plant ingestions, seed

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85. Two Scorpions, One Gun: Simultaneous Hottentotta Tamulus and Androctonus Australis Envenomation in a Self-Harm Attempt

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Background: Envenomations in the United States (US) account for 16,000 poison center calls a year – more than snakes and spiders combined. Envenomations in the US are largely from Centruroides species, contributing to about 1 death every 2.5 years. Exotic scorpions kept as pets or in zoos can be more deadly.
Case Report: A 30-year-old male presented to a suburban trauma center with scorpion envenomations to both hands and a self-inflicted gunshot wound to the left shoulder. The patient had intentionally allowed two of his pet scorpions, Hottentotta tamulus (Indian Red) and Androctonus australis (African Fat Tail) to sting him on the dorsum of the right hand and the left middle finger, respectively. He then attempted to shoot himself, but due to the pain in his hands, he missed his chest, grazing his left shoulder. He presented to the emergency department shortly thereafter with tourniquets placed on bilateral arms by emergency medical services. On arrival, he was hypertensive to 183/88 mm hg and tachycardic to 123 bpm. The tourniquets were removed. His main complaint was superficial pain at both of the envenomation sites and the gunshot wound, which was treated with oral and intravenous opiates. Six hours after presentation he was started on oral prazosin at one milligram every 12 hours for four doses. His hypertension improved to 155/83 one hour after the first dose of prazosin. He received two milligrams of lorazepam 19 hours after presentation. He did not require parenteral hemodynamic medications or respiratory support. His lactate peaked at five mmol/L and his highest recorded creatinine kinase was 851 U/L. The patient had persistent but gradually improving hypertension and tachycardia that was monitored until complete resolution of autonomic symptoms 36 hours post presentation.
Case Discussion: The envenomation toxidrome of nearly every species is similar: severe pain (grade 1), autonomic stimulation (grade 2), cardiopulmonary collapse (grade 3), and multiorgan failure (grade 4). This case progressed to clinical grade two. Prazosin is a mainstay of therapy to block autonomic excitation that characterizes grade 2 envenomation. Each species' toxin is different, necessitating species specific antivenom. Hottentotta antivenom has been shown to reduce the duration of symptoms and to stop progression to more severe symptoms. The benefit of the Androctonus antivenom is less clearly defined. In the US, antivenom to non-native species is not as widely stocked as Centruroides antivenom. Using the Antivenom Index we were able to locate expired Androctonus antivenom at an aquarium 800 miles away. Given the improvement of the patient's symptoms with prazosin and the time it would take to transport the antivenom, it was decided to forgo antivenom therapy.
Conclusion: To our knowledge, this is the first time that simultaneous envenomation by scorpion species from two distinct continents has been reported. Our patient responded well to oral prazosin therapy and did not progress to a higher grade envenomation. Given the prevalence of exotic pets, it is important for poison centers to have a protocol for mobilizing antivenom such as use of the Antivenom Index.

KEYWORDS Hottentotta tamulus, Androctonus australis, Prazosin

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86. Ingestion of Foraged Toxicoscordion venenosum (Death Camas)

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Background: Foraging for wild foods seems to be regaining popularity in recent years. Many poisonous plants and mushrooms resemble edible varieties, and foragers are at risk of toxicity from misidentification. The bulbs of death camas (Toxicoscordion venenosum) are oval in shape and resemble onions. Although they do not smell or taste like members of the Allium genus, death camas bulbs can easily be mistaken as edible wild onions, and ingestion may result in significant toxicity.
Case Report: The patient is a 60-year-old female who presented to the Emergency Department (ED) with complaints of oral tingling and nausea after eating a meal made with foraged roots she believed to be wild onions. The patient and her husband both reported a bitter taste to the meal. The patient's husband did not swallow any of the meal, but the patient completed it despite the bitter taste. She subsequently developed paresthesias and nausea, so she re-examined the plant and identified it as death camas. Her presenting vital signs (VS) were notable for a heart rate (HR) of 30 beats per minute and a blood pressure (BP) of 60/30 mmHg. Electrocardiogram demonstrated sinus bradycardia with normal intervals. The patient was given two doses of 0.5 mg atropine for bradycardia and hypotension, 3 liters of 0.9% normal saline, and ondansetron for nausea. Her nausea and paresthesias resolved. Her HR and BP transiently improved, but she was ultimately placed on a dopamine drip at 6 mcg/kg/minute for pressure support. She was weaned off the dopamine drip on the second day of admission. Her VS normalized, she remained symptom-free, and she was discharged home about 48 hours following her time of ingestion.
Case Discussion: Toxicoscordion venenosum is widely distributed throughout western North America. It was formerly classified under the genus Zigadenus, but recent molecular phylogenetic studies lead to the reclassification of many former Zigadenus species. All parts of the plant are poisonous and contain zygacine, the main toxic compound. Zygacine is a steroidal alkaloid that causes cardiac and neurotoxicity by altering the permeability of voltage-gated sodium channels, essentially acting as a sodium channel opener. The main target organ systems for toxicity are cardiac and neurologic. Reported signs and symptoms include bradycardia, arrhythmias, hypotension, paresthesias, tremor, hyperreflexia, and seizures. The treatment is mainly supportive, and several cases of successful treatment using atropine and dopamine have been reported.
Conclusions: We report a case of Toxicoscordion venenosum exposure with significant effects successfully treated with atropine and dopamine. This case highlights the risks posed by foraging and the importance of proper plant identification as well as the utility of medical toxicology expertise in the treatment of poisonings from wild plants.

KEYWORDS Toxicoscordion, Zygacine, Foraging

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87. Paracanthurus hepatus (Blue Tang) Envenomation

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Background: Paracanthurus hepatus is a species of Indo-Pacific surgeonfish and is a popular aquarium pet. The distinctive characteristic of the surgeonfish family is the scalpel-like spines found on either side of the tail, which are extremely sharp and may be used to inflict deep wounds when the fish are handled. Since the appearance of the blue tang, Dory, in Disney Pixar's films Finding Nemo and the sequel Finding Dory, sales of Paracanthurus hepatus have steadily increased.
Case Report: The patient is a 72-year-old female who presented to the Emergency Department (ED) with a complaint of hand pain and swelling following an injury from her blue tang (Paracanthurus hepatus) aquarium fish. The patient was noted to have a small abrasion to the palmar aspect of her left fourth finger with surrounding erythema and edema of her hand. The treating ED physician, who is also a medical toxicologist, noted the degree of erythema and edema and the patient's pain were greater than would be expected from the wound itself. A plain film of the hand showed focal soft tissue swelling but no radiopaque foreign body. She was treated with hot water immersion for one hour without improvement in symptoms. The patient had relief of pain following an injection of bupivacaine. She was prescribed ciprofloxacin and cephalexin for prophylaxis and discharged home. The patient was contacted several months following the incident, and she reported complete resolution of symptoms and normal use of her hand.
Case Discussion: There are scattered and poorly-characterized envenomations by blue tangs reported in the toxicologic literature. Online, non-academic forums have discussed the potential venomous nature of this increasingly-common exotic pet. A morphological study identified an anterolateral glandular groove with an associated venom gland in Paracanthurus hepatus, and phylogenetic data has suggested than many more fish species in the Class Actinopterygii, of which the blue tang is a member, are likely to be venomous.
Conclusions: We present a case of apparent envenomation by Paracanthurus hepatus. Coupled with the known morphologic and phylogenetic data, this report lends credence to the characterization of blue tangs as venomous fish. While warm water immersion did not provide significant relief in this case, it is a low-cost, non-invasive, and low-risk intervention. Given its demonstrated benefit in other fish envenomations, it should be the first-line treatment for blue tang envenomation. This patient has significant improvement with local anesthetic, which is a reasonable second-line intervention. There is no consensus regarding antibiotic prophylaxis for marine envenomations. A risk-benefit discussion with the patient and shared decision-making is likely a reasonable approach. Given the Disney-induced popularity of blue tangs, envenomations may become more common, and the classification of Paracanthurus hepatus as a venomous fish is important knowledge for both toxicologists and the public.

KEYWORDS Blue tang, envenomation, Paracanthurus hepatus

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88. Rare Side Effects of Initiating Antihypertensive Therapy

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Background: Beta blockers are among some of the most widely used antihypertensives. Metoprolol is one of the most commonly prescribed beta blockers and it has central nervous system effects with psychiatric symptoms that may present insidiously or abruptly [1,2]. This case report documents a rare neuropsychiatric adverse reaction to metoprolol that began with paranoid ideation and progressed to delirium with hallucinations.
Case report: A 66-year-old woman with a past medical history significant for Parkinson's disease and hypertension presented to the emergency department with new onset auditory and visual hallucinations. One week prior to admission, the patient was started on amlodipine

for hypertension, developed muscles spasms as an adverse reaction to the medication, and discontinued use after 2 days. Following a 3-day amlodipine washout period, the patient was started on metoprolol succinate ER, 25 mg once daily. A half-day after starting metoprolol, the patient's husband reported she thought the television was talking to or ridiculing her. She developed an olfactory hallucination stating the carpets smelled like carpet cleaner the day after starting the metoprolol. However, her husband reported the carpets had not been cleaned for months. On the third day of treatment with metoprolol, the patient was brought into the emergency room with self-reported auditory and visual hallucinations. The patient reported seeing and hearing multiple people pointing at her and mocking her in her home. Therefore, she ran out of her house without shoes or a jacket on a winter night. The patient was found at a neighbor's house four hours later, banging on the neighbor's front door, and was unable to account for whereabouts in the preceding 4 hours. Evaluation in the emergency department revealed normal vital signs; an unremarkable physical exam; extensive, normal laboratory data; normal cerebral computed tomography, a normal EKG; and neurology and psychiatry consultations that revealed no other cause for her symptoms than the new prescription for metoprolol. She was observed for 8 hours and her symptoms resolved. She was not re-challenged with metoprolol to see if these symptoms recurred. Follow-up over a three-month period revealed no recurrence of symptoms.
Summary: Serious reactions, such as vivid nightmares, dreams, hallucinations, and delirium have been reported following treatment with beta-blockers, including metoprolol [3]. This case report illustrates visual, auditory, and olfactory hallucinations and persecutory delusions associated with initiating metoprolol therapy.

KEYWORDS Metoprolol, Side Effect, Delirium

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89. Dramatically Improved Hemodynamics following Hydroxocobalamin in Hypothermic Cardiac Arrest

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Goal: Cardiac arrest due to hypothermia can be challenging to treat particularly as warming measures may initially prove to be ineffective. Yet, standard teaching argues against ceasing cardiopulmonary resuscitation (CPR) until normothermia has been obtained. We present a case of hypothermia -induced cardiac arrest and resultant shock that eventually demonstrated improvement in core body temperature and hemodynamics following rescue therapy with hydroxocobalamin.
Case: A 40 year-old male with a history of HIV presented to the emergency department in full cardiac arrest. History obtained later confirmed that the patient was abusing heroin and ethanol before becoming unconscious outside where the environmental temperature was 27 Celsius. Initial rhythm was pulseless electrical activity and initial core body temperature was 27 Celsius. Epinephrine, calcium, naloxone, and sodium bicarbonate therapy were administered and return of spontaneous circulation was obtained after 10 minutes of CPR; osborne waves were subsequently visualized on electrocardiogram (EKG). He was started on maximum doses of epinephrine, norepinephrine, phenylephrine, and vasopressin infusions, as well as warm water gastric lavage and application of a non-invasive targeted temperature management device. These therapies were unable to increase systolic blood pressure (SBP) greater than 50mmHg and core body temperature remained less than 30 Celsius. Five grams of hydroxocobalamin was administered with immediate temporal improvement of SBP to 190mmHg and temperature to 33 Celsius. Vasopressin and phenylephrine infusions were discontinued while epinephrine and norepinephrine infusions were rapidly titrated down. He sustained rapid improvement of hemodynamics and became normothermic over the next 36 hours but was ultimately removed from ventilator support secondary to severe anoxic injury seen on neuroimaging.
Discussion: This case demonstrates a patient with severe refractory shock and hypothermia that did not respond to aggressive external and internal warming measures with concurrent maximum vasopressor therapy until a dramatic and temporal improvement was noted after administration of hydroxocobalamin. In severe hypothermia, cardiovascular collapse occurs largely to impediment of ion currents during all stages of the cardiac action potential. At the same time, systemic vascular resistance falls with severe hypothermia as catecholamine release is blunted, a process similar to vasoplegia pathophysiology. Recently, hydroxocobalamin has been the focus of much investigation for mechanisms independent of its role in treating cyanide poisoning. Ex vivo models have focused on its ability to scavenge nitric oxide (NO) and to potentially inhibit nitric oxide synthase, thereby decreasing endogenous NO and increasing systemic vascular resistance. Based on these investigations, several case reports have also focused on hydroxocobalamin's role in vasoplegic syndrome with similar temporal improvement in patients' hemodynamics. However, optimal dosing remains ambiguous and evidence based trials are lacking in human populations.
Conclusion: Hydroxocobalamin could be considered in refractory severe hypothermia to increase systemic vascular resistance and to potentially increase efficacy of warming measures. Further high evidence based research is needed.

KEYWORDS Hydroxocobalamin, nitric oxide, hypothermia

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90. Envenomation of a South Carolina Man by A Pope's Green Pit Viper

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Background: Trimeresurus popeorum, the Pope's Green Pit Viper, is a venomous snake responsible for many accidental envenomations in Southeast Asia. Given the Pope's Pit Viper is not endemic to the United States, envenomation and subsequent treatment here have not been reported in the western literature. This case report describes the presentation, hospital course and long-term effects of envenomation by this species in a South Carolina man.
Case report: 46-year-old male presented to the emergency department 30 hours after envenomation by a Pope's Green Pit Viper complaining of local tissue swelling and decreased grip strength in his right hand. Laboratory results revealed hematologic changes including coagulation dysfunction. Pertinent laboratory values included a D-dimer of 1312 (0-230 ng/mL), platelet count 112 k/mm3, and white blood cell count of 13.9 k/mm3. Despite repeated

Table 1 Laboratory values.

Hrs since attack	Hour 30	ǂ	Hour 36	Hour 42	Hour 49	Approx. 6 days later♦
Hematology						
WBC (3.7-10.1 K/mm3)	13.9 H		13.7H	10.2H	8.7	
Hgb (14.0-16.4 gm/dl)	14.1		12.5 L	12.8 L	13.2 L	13.4♦
Hct (40.0-47.2%)	42.4		37.1 L	37.9 L	39.7 L	
MCV (81.8-94.6 fL)	80.8 L		79. 9			80♦
MCH (27.9-33.1 pg)	26.9 L		26.9 L			27.1♦
Plt count (150-400 K/mm3)	112 L		98 L	87 L	83 L	186♦
Neut % (auto)(40.1-81.3%)	85.4 H		72.1	63.4	59.0	

ǂAntivenom administered.
♦Laboratory values from outside lab (LabCorp), ranges as follows: MCV (79-97 fL),MCH (26.6-33.0 pg), Hgb (13.0-17.7 gm/dl), plts (150-379 K/mm3).

Table 2 Laboratory values. TNP (test not performed).

Time	Hour 30	Hour 31	ǂ	Hour 36	Hour 42	Hour 49	Approx. 1 week♦	Day 23♦	Day 47♦
Coagulation									
PT (9.8-13.9 sec)	TNP	TNP		16.0 H	13.9	12.6	11.4♦	♦	♦
APTT (24.9-37.9 sec)	TNP	TNP				21.8		♦	♦
INR	TNP	TNP		1.44			1.1♦	♦	♦
Fibrinogen (224-424 mg/dl)	TNP	TNP		<26 L			238♦	♦	♦
D-dimer (0-230 ng/ml)	1312H						2650H♦	4108♦	3500H♦

ǂAntivenom administered.
♦Laboratory values from outside lab (LabCorp), ranges as follows: INR (0.8-1.2), PT (9.1-12.0), D-dimer (0-490 ng/ml), fibrinogen antigen (180-350 mg/dL).

attempts, studies including fibrinogen, PT and PTT did not give formal results. This was due to a profound abnormality in fibrinogen level and markedly prolonged PT/PTT. Anti-venom was transported from an outside supplier, mixed and administered onsite. Laboratories were collected every six hours and included complete blood cell count, fibrinogen, and coagulation studies. Nineteen hours after the initial studies were collected the patient's laboratory results had improved, with the exception of his D-dimer. After 24 hours of observation in the emergency department and intensive care unit, the patient was discharged home in stable condition. Three months following the event the patient continues to suffer from mild residual symptoms and persistent markedly elevated D-dimer.

Case Discussion: The alterations in our patient's laboratories were the result of direct venom effects as well as steroid and host stress response factors. His response to antivenom therapy was as predicted with rapid improvement of his coagulopathy. The exception is his persistently elevated D-dimer, which suggests the possibility of lingering coagulopathy. The clinical relevance of this is not known at this time.

Conclusion: This case describes the clinical course of a man bitten by a Pope's Green Pit Viper in South Carolina.[Table 1][Table 2]

KEYWORDS Snake Bite, Pope's Green Pit Viper, Coagulopathy

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91. Candlenut Ingestions Reported to Poison Centers

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Background: The candlenut (Aleurites moluccana) is a flowering tree in the spurge family Euphorbiaceae found in the tropics worldwide. It grows to a height of 15-25 m with pale, green, ovate, 3-5-lobed leaves. It produces round, 4-6 cm nuts with a shell surrounding a white, oily, fleshy kernel or seed. The seeds contain saponin and phorbol. Adverse gastrointestinal effects such as nausea, abdominal pain, and diarrhea, and dehydration and electrolyte imbalances have been reported with ingestion of the seeds. A. moluccana seeds have been promoted in the media and on the Internet for weight loss and the treatment of other health problems. A. moluccana seeds are sold whole or in capsules in markets in Mexico under the names of "nuez de la India" (India nut) or "semilla de Brasil" (Brazil seed), and the products are also available for sale via the internet in the United States. The objective of this study was to describe A. moluccana ingestions reported to poison centers.

Methods: Cases were all A. moluccana ingestions reported to a statewide poison center system during 2000-2018. The distribution of A. moluccana ingestions was determined for various factors related to patient demographics, ingestion circumstances, management, and outcome.

Results: Fifty A. moluccana ingestions were identified. Forty-five (90.0%) of the ingestions involved no other substances. The patient age distribution was 10 (20.0%) 0-5 years, 3 (6.0%) 6-19 years, and 37 (74.0%) 20 years or more; 39 (78.0%) of the patients were female and 11 (22.0%) male. All of the ingestions occurred at the patient's own residence. Twenty-one (42.0%) of the exposures were reported from counties that border Mexico. The ingestion reason was 20 (40.0%) unintentional, 6 (12.0%) intentional, 22 (44.0%) adverse reactions, and 2 (4.0%) unknown reason. Thirteen (26.0%) of the patients were managed on-site, 31 (62.0%) were already at or en route to a healthcare facility, 5 (10.0%) were referred to a healthcare facility, and 1 (2.0%) were managed at an unspecified other location. The medical outcome was 10 (20.0%) no effect, 6 (12.0%) minor effect, 10 (20.0%) moderate effect, 2 (4.0%) major effect, 1 (2.0%) not followed-judged nontoxic, 12 (24.0%) not followed-minimal clinical effects possible, 3 (6.0%) unable to follow-potentially toxic, 5 (10.0%) unrelated effect, and 1 (2.0%) death. The most common clinical effects were vomiting (n = 22, 44.0%), diarrhea (n = 14, 28.0%), nausea (n = 10, 20.0%), abdominal pain (n = 7, 14.0%), and dizziness/vertigo (n = 7, 14.0%). The most common treatments were IV fluids (n = 14, 28.0%), antiemetics (n = 11, 22.0%), and dilute/irrigate/wash (n = 10, 20.0%).

Conclusion: The majority of A. moluccana ingestions involved adults and females. The highest proportion of cases were adverse reactions. Most of the patients were managed at a healthcare facility and 32.0% had serious outcomes, including one death. While the most frequently reported clinical effects were gastrointestinal, A. moluccana ingestions should be considered dangerous in symptomatic patients and referred for evaluation.

KEYWORDS Candlenut, Aleurites moluccana, Brazil Seed

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92. QTc Prolongation in poison center exposures to CredibleMeds list of substances with "Known Risk of TdP"

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Background: In the setting of an acute overdose, many drugs carry some risk of QT prolongation. The Credible Meds QT Drug List identifies 58 medications listed under the category of "Known Risk of Torsades de Pointes (TdP)" that prolong the QT even at therapeutic dosing. This list does not address risk in the setting of an acute overdose nor does it stratify the risk of TdP within this category.
Objective: We sought to determine the proportion of cases exhibiting QTc prolongation, ventricular tachycardia (VT)/ventricular fibrillation (VF), TdP, and cardiac arrest in patients exposed the 58 agents listed on the Credible Meds QT Drug List with a "Known Risk of TdP" reported to our Regional Poison Center (RPC).
Methods: This was a retrospective chart review of all cases reported to our RPC from May 7th, 2018 through March 7th, 2019 that were treated at a health care facility and were coded as exposures to one or more of the 58 substances on the Credible Meds QT Drug List. Exposure demographics, treatments provided by healthcare facilities, clinical effects, and medical outcome for each case were characterized.
Results: During this 10-month period there were 220 exposures reported to our RPC to substances with "Known Risk of TdP" per the Credible Meds QT Drug list that were treated at a health care facility. Of these exposures, 75 (34%) had QTc prolongation as defined by NPDS as QTc >450msec for males and QTc >470msec for females. In 26 (34.7%) of these cases "QTc prolongation" was not coded as a clinical effect for the exposure despite meeting this definition. Of the cases that had QTc prolongation, the average age was 44, and 35 (46.7%) of the patients were male. QTc >500msec was reported in 25 (33.3%) cases. The most common substances were citalopram (n = 22), cocaine (n = 16), and escitalopram (n = 16). Although most exposures included other co-ingestants (n = 58; 77.3%), only 4 of the cases involved more than one substance with "known risk of TdP". There were no cases in which TdP was documented, 2 cases where VT/VF was reported, and one case in which asystole was reported.
Conclusion: In our review of 10 months of data involving 220 exposures to medications on the CredibleMeds list with "Known Risk of TdP", we had no documented cases of TdP, but other arrhythmias were reported including VT/VF and asystole with QTc prolongation. Limitations include voluntary reporting, data from only one poison center, no confirmation of exposure, no EKGs available for QTc verification, no standard documentation of long QT syndrome, and frequent miscoding of QTc prolongation as a clinical effect. Further studies with a larger data set would be helpful to provide better correlation between QTc prolongation and risk of TdP. QTc Prolongation in poison center exposures to CredibleMeds list of substances with "Known Risk of TdP"

Tables:

Substance	Number of Cases	Percent of 220 Cases	Number of Cases with QTc Prolongation per substance	Percent of Cases with QTc Prolonation per substance
Amiodarone	4	1.82%	2	50%
Azithromycin	1	0.45%	0	0
Chlorpromazine	5	2.27%	3	60%
Ciprofloxacin	2	0.91%	0	0
Citalopram	59	26.82%	22	37.29%
Cocaine	56	25.45%	16	28.57%
Dofetilide	2	0.91%	0	0
Donepezil	4	1.82%	1	25%
Dronadarone	1	0.45%	1	100%
Escitalopram	41	18.64%	16	39.02%
Flecainide	6	2.73%	2	33.33%
Haloperidol	11	5%	6	54.55%
Levofloxacin	1	0.45%	0	0
Methadone	13	5.91%	4	30.77%
Ondansetron	9	4.09%	2	22.22%
Sotalol	9	4.09%	3	33.33%

Substance	Number of Cases	Longest QTc Recorded	Average of Longest QTc per substance
Amiodarone	2	496	489.5
Chlorpromazine	3	496	474.6667
Citalopram	22	662	508.8636
Cocaine	16	700	504.375
Donepezil	1	480	480
Dronadarone	1	482	482
Escitalopram	16	662	504.375
Flecainide	2	606	535
Haloperidol	6	487	474
Methadone	4	701	576.25
Ondansetron	2	527	526.5
Sotalol	3	476	472.3333

KEYWORDS QT prolongation, TdP, arrythmia

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93. Buprenorphine Microinduction During Continuous Fentanyl Infusion

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Background: Buprenorphine is a high-affinity partial mu receptor agonist with long duration of action indicated for analgesia and opioid use disorder. Transition from full opioid agonists to buprenorphine typically requires a washout period to avoid precipitated withdrawal after buprenorphine administration. This may be particularly challenging in patients with difficulty stopping opioid use, even for brief periods. We report a case of buprenorphine microinduction in a critically ill patient receiving continuous fentanyl infusion.
Case Report: This is a 39 year-old male with history of craniopharyngioma resection and complex hospital course complicated by stroke, blindness, and respiratory failure requiring tracheostomy with prolonged ICU stay. The patient received continuous fentanyl infusion averaging 250 mcg/hr for over a month due to severe agitation with

dose adjustments. Additional sedation included trials of dexmedetomidine, ketamine, and antipsychotics at various times that were limited by bradycardia in setting of hypothyroidism and prolonged QT interval; fentanyl dose was not influenced by these adjunctive agents. Due to difficulty weaning fentanyl, buprenorphine was initiated at 30 micrograms IV every 6 hours during continuous fentanyl infusion. Over the next 3 days buprenorphine was administered sublingually and increased from 150 mcg every 6 hours to 450 mcg every 6 hours and 1 mg every 12 hours while fentanyl infusion was maintained. On Day 4 the fentanyl infusion was decreased by 50 mcg/day and buprenorphine was doubled daily until reaching 8 mg twice daily. Fentanyl infusion was then stopped and the patient continued buprenorphine without signs of opioid withdrawal identified during induction. By the end of transition the patient was back to baseline mental status, able to work with physical therapy, transition from bed to chair, and started working with speech therapy without agitation.
Case Discussion: This case illustrates a novel buprenorphine induction technique used to transition a patient with iatrogenic opioid dependence from high dose fentanyl infusion to buprenorphine. We used a microinduction with small doses of buprenorphine titrated over several days while gradually tapering the full agonist. Utilizing lower dose formulations available for pain provides flexibility in this process and potentially allows for concomitant use. Two cases were previously described of buprenorphine induction utilizing the Bernese method with administration of microdoses of buprenorphine during full agonist use. A similar technique was reported using low doses of buprenorphine shortly after methadone cessation before effects completely dissipated. This technique is theorized to cause buprenorphine accumulation at the receptor with gradual displacement of the full agonist, easing transition for the patient. This may be considered for individuals with evidence of significant physical dependence unable to tolerate a standard induction. Potential applications include transitioning from full opioid agonist analgesia, sedation weans in critically ill patients, and induction for traditional application in the treatment of opioid dependence.
Conclusion: Buprenorphine microinduction may be used to assist with challenging sedation weans of full agonist opioids in critically ill patients.

KEYWORDS Buprenorphine, Microinduction, Fentanyl

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94. Lytes Out: Massive Melatonin Overdose with Resulting Profound Hyponatremia

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Background: Melatonin is a hormone that contributes to sleep and circadian rhythms. It is sold as a dietary supplement for sleep aid. Melatonin overdose typically results in mild sedation. Significant toxicity in overdose has not been previously reported in literature.
Case Report: A 72-year-old man with history of opioid use disorder presented with altered mental status, ataxia, and slurred speech. He had been self-treating symptoms of withdrawal from tramadol with melatonin, taking a total of 570 mg over the course of 2 days. Vital signs were within normal limits. Computed tomography of his head did not reveal any abnormalities. He was found to be hyponatremic (Na 113 mMol/L), hypokalemic (K 2.7 mMol/L), and hypochloremic (Cl 77 mMol/L). He had no clinical signs of dehydration and had a BUN of 15 mg/dL and creatinine of 0.9mg/dL. Glucose was 127 mg/dL. A random prolactin level was elevated (45.2 ng/mL, normal range 0.6-19 ng/mL), ACTH and cortisol were within normal limits (11 pg/mL and 16.8 ug/dL, respectively). He was admitted to the intensive care unit (ICU) due to electrolyte abnormalities. His electrolytes normalized and mental status returned to baseline after 3 days. No other cause of his electrolyte abnormalities was identified.
Discussion: This is the first reported case of melatonin overdose associated with significant electrolyte abnormalities. Typical dosing of melatonin is 0.3mg-5mg taken prior to bedtime. This patient took a total of 570mg of melatonin over the course of 48 hours, representing a 50-2000 fold overdose. Melatonin receptors are known to play a role in hormonal signaling of the pituitary-hypothalamic-gonadal axis, and melatonin has been shown to antagonize glucocorticoid receptors in animal models and reduce cortisol response to ACTH in limited human studies. While significant electrolyte abnormalities have not been reported in therapeutic dosing, such a massive overdose could cause profound derangement of neuroendocrine pathways and lead to such biologic effects.
Conclusion: In massive overdose, melatonin may cause disrupt neuroendocrine hormonal pathways and lead to electrolyte disturbances.

KEYWORDS Melatonin, Hyponatremia, Opioid withdrawal

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95. Methylphenidate Associated Peripheral Vasculopathy in a Teenager with Rapid Recrudescence after Dosing Error

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Introduction: Methylphenidate is a piperidine containing phenylethylamine analog with stimulant properties and is not commonly associated with peripheral vasculopathy.
Case: A 16-year boy with a history of attention deficit hyperactivity disorder (ADHD) on atomoxetine and methylphenidate (72 mg daily, up from 54mg one month earlier) presented with two weeks of painful swelling and discoloration of his hands and feet. On exam, he had progressive edema extending from his elbow to his hands and from his mid thigh to his feet. His rash was characterized by a blanching, erythematous, reticular pattern on his proximal arms and legs that progressed distally. His hands and feet were dusky, edematous, and tender. His white blood cell (WBC) count was 13.1 x 109/L with 17% eosinophils. Given unclear etiology of the rash, he underwent rheumatologic work-up showing normal C3 and C4 complement levels, CRP.
Discussion: Methylphenidate was the most likely cause of the painful peripheral vasculopathy given the patient's negative rheumatologic work-up and temporal recrudescence of symptoms after reintroduction of methylphenidate. The high dose of methylphenidate and the concomitant use of atomoxetine, a norepinephrine reuptake inhibitor, may have led to increased vasoconstriction, which predisposed this patient to the development of peripheral vasculopathy. Complete cessation of the inciting agent was the most effective therapy.
Conclusion: Methylphenidate is rarely associated with peripheral vasculopathy, but high doses of the medication and co-administration with sympathomimetic xenobiotics may predispose individuals to the development of this rare side effect.

KEYWORDS Methylphenidate, Peripheral vaculopathy, Adverse drug effect

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96. Mamma Mia! Acute exposure to apricot kernels resulting in macular ganglionic loss

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Background: Apricot kernels are not commonly found in grocery stores in the United States, but are frequently sold in other countries. Ingestion of apricot kernels may result in significant toxicity from amygdalin, a cyanogenic glycoside. We report a rare case of macular ganglionic loss and persistent visual changes after apricot kernel ingestion.
Case Report: A 25-year-old female, employed as a nurse, purchased what she believed to be almonds from a grocery store while vacationing in Greece. After sampling two types of almonds, she purchased the most bitter of the two. Approximately 45 minutes after eating up to 100 pieces, she developed weakness, dizziness and blurred vision in both eyes. She discovered the product was actually apricot kernels and immediately sought care at a local hospital. She underwent gastric lavage and was admitted for two nights. Three months later, she still experienced blurry vision despite the use of reading glasses, and her primary care physician referred her to a medical toxicology clinic for further evaluation. Upon initial medical toxicology evaluation, the patient reported persistence of blurry vision along with emotional lability and speech difficulties. A brain MRI was unremarkable, and ophthalmologic consultation revealed normal retinas. An optometry exam revealed severe ganglionic loss in the maculae, a finding unusual for the patient's age. The patient's visual acuity had deteriorated when comparing tests from before the trip, after she returned and two months later (Right eye 20/25, 20/30, 20/20 and Left eye 20/40, 20/60, 20/80, respectively). The patient was prescribed progressive lenses, and her visual acuity remained stable over the following several months.
Discussion: The Greek government has warned citizens not to eat more than 10 apricot kernels per day, due to concerns for toxicity. The European Food Safety Authority has also warned about amygdalin content in apricot kernels. These warnings are not posted on packaging or in grocery stores, and apricot kernels are sold without regulation in stores and online. Apricots kernels, such as the seeds ingested by our patient, contain more amygdalin than bitter almonds, also commonly available.
Conclusion: Cyanogenic glycoside poisoning after acute apricot kernel ingestion can result in permanent sequelae, including macular injury and visual deterioration. Increased public awareness of the dangers of apricot kernel ingestion may reduce the incidence of cyanogenic glycoside toxicity from these products.

KEYWORDS Apricot kernel, cyanide, macular ganglion loss

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97. Measuring Consumers' Satisfaction with a Poison Control Chat Service

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Background: Gauging customer satisfaction is a required metric for the poison control accreditation process. Traditional efforts of measuring satisfaction often involve outbound calls to low acuity patients. As an increasing number of healthcare agencies offer electronic approaches to managing care, patients have the opportunity to rate cyber experiences. Healthcare agencies also have the opportunity to gain insight into these interactions by measuring the satisfaction of consumers who use electronic methods of contact. As such, a regional poison control center sought to evaluate consumers' experiences utilizing a poison control chat service for medical assistance.
Methods: Closed chat case records were extracted over a calendar year period totaling 1,256 cases. At the end of each chat encounter, a survey was deployed prompting chatters to evaluate the service they received. Competency of the person providing the help, efficiency of the interaction, and probability to use traditional telephonic means for receiving care were domains of measurement. The survey applied a Likert scale star-rating system. Four and five-star ratings were grouped affirmatively; one, two, and three-star ratings were grouped neutrally or negatively. The survey was completed by 23% of chatters (n = 290). Chatters could also type additional comments about their experience through a text-box field. Sixty-four chatters provided additional comments.
Results: Chat survey respondents indicated a high level of confidence in the medical professionals providing the help. Ninety-five percent of respondents felt confident or very confident in the abilities of the person helping them. In addition, 95% of respondents felt that their chat was answered in a timely way. Average wait time to chat with a medical professional was 47.7 seconds. When asked how receptive respondents were to calling poison control about the same issue, most were receptive, but one in five (20%) indicated they were not as willing or wouldn't call. Respondents named a variety of factors contributing to their preference to chat including convenience, perceived speed of response, and social angst.
Conclusions: Virtual medical consultation is becoming more of a reality for patients. Poison control centers have begun to offer complementary electronic options for consumers to receive poison control advice. However, little is known about the overall satisfaction level of patients who choose to receive online care from poison control. Furthermore, advising patients online for suspected poisonings is still a fairly novel concept for both patients and poison control centers. More research is needed to determine the long-term significance of managing patients' poison exposures electronically. In light of these limitations, this study found several implications for consideration: 1) people showed a willingness to contact poison control via chat; 2) online chat technology offers a way to attain a customer satisfaction pulse; 3) regional poison control chat users report high levels of satisfaction using the chat service; 4) some people who seek online help from a poison control center may not be willing to contact poison control via phone.

"I feel fine chatting just as good as if I call..."
"So helpful and convenient, and saves an ER visit with such a quick response."
"Chatting was way faster…within 5 minutes I have all the answers I needed."
"I loved the chat feature considering I was at work and didn't want others to hear my conversation."
"Very easy to use this and the response was fast but I didn't have an actual emergency - I would have called had it been urgent."
"So thankful for the online chat…[it] took me straight to a person."
"I would call but running around with an angry infant chat was definitely more convenient."
"The person who helped me was awesome. They were very helpful. Thank you for offering the chat option. I have major social problems so for me it is much easier to text than talk."

KEYWORDS Online chat, patient satisfaction, medical management

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98. Uridine Triacetate Outside of the 96-Hour Window

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Background: Uridine triacetate (UT) is indicated for the treatment of fluorouracil (5-FU) or capecitabine overdose, and for early-onset, severe adverse reactions (gastrointestinal toxicity and/or neutropenia) within 96 hours following 5-FU or capecitabine administration. UT is a pyrimidine analog that is deacetylated in the body, yielding uridine. Uridine competitively inhibits cell damage and cell death caused by 5-FU, and reduces incorporation of fluorouridine triphosphate (a 5-FU metabolite) into RNA of hematopoietic progenitor cells and gastrointestinal mucosal cells to reduce 5-FU toxicity in normal tissues. Safety and efficacy of UT initiated more than 96 hours after completing 5-FU or capecitabine administration have not been established.
Case Report: A 68 year old male with squamous cell carcinoma of the tonsil was hospitalized for his first cycle of chemotherapy. On day 1 he received 100mg/m2 cisplatin, 75 mg/m2 docetaxel and 1000mg/m2 5-FU. Dosing of 5-FU was repeated on days 2-4, finishing on hospital day 5. On day 8 he developed watery, bloody diarrhea which progressed over the next 3 days to severe mucositis, with a 2 foot section of intestinal mucosa sloughed off in a stool sample on day 9. He was also neutropenic, with an absolute neutrophil nadir of 960 k/cm3 measured on the same day. Imaging strongly suggested ischemia, presumed due to hypoperfusion in the setting of volume depletion, renal impairment and cardiac insufficiency, and infection, with stool studies positive for norovirus. Toxicity remained the strongest consideration given onset of symptoms shortly following completion of chemotherapy; 5-FU was the most likely culprit given colitis, bloody diarrhea, severe oral mucositis, and myelosuppression. The patient was outside the 96-hour treatment window, but symptom severity, onset within the first 4 days post-exposure, and concern for further loss of bowel and worsening mucositis compelled consideration of UT therapy. UT was obtained at a cost of $80,000 and started on day 10: 10g every 6 hours for 20 doses. Symptoms improved after initiation: bloody diarrhea resolved and stool frequency decreased over the next 5 days. Patient was discharged on day 15. Genotyping revealed 2R/3RC genotype, predicting a low thymidylate synthase (TYMS) expression and an increased risk for 5-FU toxicity.
Discussion: 5-FU is frequently used to treat a variety of gastrointestinal cancers, and is associated with severe diarrhea, mucositis, stomatitis, and esophagopharyngitis. 5-FU is an irreversible inhibitor of the enzyme TYMS, an underexpression of which leads to the manifestation of toxic drug effects. Genotyping revealed that this patient had low TYMS expression, supporting 5-FU as the cause of his severe mucositis and neutropenia. Although only approved for use within the first 96 hours following completion of 5-FU therapy, this patient's symptoms improved and ultimately resolved after completing the course of UT, despite initiation 120 hours after 5-FU therapy.
Conclusion: Although UT is only indicated for the treatment of 5-FU toxicity within 96 hours of exposure, there is potential benefit in giving the antidote outside of the 4 day window, though the high cost of the medication may be prohibitive.

KEYWORDS Uridine, fluorouracil, capecitabine

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99. Characterizing trends in synthetic cannabinoid use from structured patient interviews during medical toxicology consultation

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Background: Synthetic cannabinoid receptor agonists (SCRAs), currently among the most prevalent new psychoactive substances, have seen considerable growth in popularity over the past two decades. Data on subjective experience as well as behaviors surrounding the use of SCRAs has been mainly limited to case reports and series with few studies examining larger trends. The present study sought to characterize current trends in SCRA use by using standardized interviews in a population of individuals receiving medical toxicology consultation due to SCRA intoxication.
Methods: Patients receiving medical toxicology consultation at 10 designated sites within the Toxicology Investigators Consortium in the United States with reported or suspected exposure to a new psychoactive substance were interviewed by treating medical toxicologists. Investigators collected clinical data as well as expanded qualitative data including knowledge, attitudes and beliefs, as well as practices related to the use of novel psychoactive substances. Interviewers utilized a primarily open ended style of questioning and answers were reviewed for data quality and completeness by the study's program manager. At the completion of the data collection phase, responses were categorized and enumerated to determine the presence of trends.
Results: Of the124 cases entered into the study between October 2014 and early 2016, the majority of cases (N = 86) involved exposure to SCRAs. The majority of identified SCRA users were single (72.1%), white (45.3%) and African American (43%), males (93%) with a mean age of 31.1 years, and incomplete high school education (47.7%). Over half (58.1%) reported at least one psychiatric comorbidity, the most common being depression (28%), anxiety (26%), and bipolar disorder (22%). The majority of patients (68.8%) had used SCRAs at least once before the presenting episode with most patients reporting extensive prior use. Patients were evenly divided between using SCRAs with friends and alone (40.7% vs 41.9%). Nearly half of patients (47.7%) considered SCRAs to be very easy to obtain, reporting that they are generally acquired free from a friend or acquaintance (39.4%) or by purchasing from a gas station or a convenience store (22.1%) (Table). Nearly half (48.8%) of patients reported that their primary reason for use was the high they experienced from smoking it while only a small proportion mentioned using it to avoid testing positive on drug screening (6.9%), current addiction (5.8%), as an alternative to marijuana (4.6%), or for experimentation (4.6%). Approximately one third (34.9%) of users described their experience as primarily positive while the remaining patients found it either primarily negative (24.4%) or mixed positive and negative (19.8%). Under half (44.2%) reported paying for the substances while nearly a third (32.6%) acquire it for free, with a small proportion (3.5%) reporting to barter for it with other drugs.
Conclusion: Of 86 reported SCRA exposures, most patients reported having used it extensively prior to presentation and using it primarily for the subjective effects associated with the drug. These results support the view that an independent and stable culture is developing around the use of SCRAs separate from their appeal as an "undetectable" alternative to marijuana.
The above table lists various the sources which patients mentioned acquiring SCRA products from.

Table Sources of SCRAs.

Source	N (%)
Free from friend or acquaintance	34 (39.5%)
Purchased from Gas Station/Tobacco Shop/ Convenience store	19 (22.1%)
Purchased from friend	7 (8.1%)
Purchased from Drug Dealer	6 (6.9%)
Found it	3 (3.5%)
"At school"	2 (2.3%)
Ordered Online	2 (2.3%)
Given by relative	2 (2.3%)
Traded for Prescription Drug	1 (1.2%)
Response Missing	10 (11.6%)

KEYWORDS Synthetic cannabinoid receptor agonist, Toxicology Investigators Consortium, Psychoactive drug intoxication

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100. Epinephrine auto-injectors exposures reported to poison centers

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Background: Epinephrine auto-injectors, such as EpiPen®, administer epinephrine intramuscularly and may be carried by patients at risk for anaphylaxis. Since at least 2007, the price of EpiPen® auto-injectors has increased greatly. Moreover, since November 2017, EpiPen® auto-injectors have been in short supply. On May 9, 2018, the United State Food and Drug Administration (FDA) posted a supply shortage alert for the product. The objective of this study was to describe epinephrine auto-injector exposures reported to poison centers.
Methods: Cases were all epinephrine auto-injector exposures reported to a statewide poison center system during 2000- 2018. The distribution of epinephrine auto-injector exposures was determined for various factors related to patient demographics, exposure circumstances, management, and outcome.
Results: A total of 3,743 epinephrine auto-injector exposures were identified. The annual number of exposures increased from 24 in 2000 to 359 in 2016 before declining to 354 in 2017 and 317 in 2018. June-August accounted for 1,130 (30.2%) of the exposures and December-February for 727 (19.4%) of the exposures. The patient age distribution was 1,340 (35.8%) 0-5 years, 1,049 (28.0%) 6-12 years, 243 (6.5%) 13-19 years, 919 (24.6%) 20 years or more, and 192 (5.1%) unknown age; 1,940 (51.8%) of the patients were male, 1,797 (48.0%) female, and 6 (0.2%) unknown gender. The most common routes of exposure were injection (n = 2,795, 74.7%), dermal (n = 546, 14.6%), and ingestion (n = 327, 8.7%). The exposure site was 3,431 (91.7%) patient's own residence, 85 (2.3%) other residence, 80 (2.1%) workplace, 66 (1.8%) school, and 81 (2.2%) other or unknown sites. Most (n = 2,555, 68.3%) of the patients were managed on-site, 828 (22.1%) were already at or en route to a healthcare facility, 302 (8.1%) were referred to a healthcare facility, and 58 (1.5%) were managed at other or unknown locations. The medical outcome was 317 (8.5%) no effect, 1,382 (36.9%) minor effect, 314 (8.4%) moderate effect, 10 (0.3%) major effect, 91 (2.4%) not followed-judged nontoxic, 1,413 (37.8%) not followed-minimal clinical effects possible, 201 (5.4%) unable to follow-potentially toxic, and 15 (0.4%) unrelated effect. The most frequent clinical effects were puncture/wound/sting (n = 1,953, 52.5%), dermal irritation/pain (n = 1,183, 31.6%), pallor (n = 504, 13.5%), edema (n = 228, 6.1%), ecchymosis (n = 152, 4.1%), numbness (n = 144, 3.8%), erythema/flushed (n = 116, 3.1%), and tachycardia (n = 115, 3.1%). The most commonly reported treatment was dilute/irrigate/wash (n = 1,831, 48.9%).
Conclusion: The annual number of epinephrine auto-injector exposures increased during 2000-2016, suggesting that the increase in EpiPen® auto-injector price did not adversely impact the number of epinephrine auto-injector exposures reported to these poison centers. The annual number of exposures then declined slightly during the next two years. It is not possible to attribute this decline to the recent EpiPen® auto-injector shortage. Most patients (63.8%) were children age 0-12 years. Although the majority of exposures occurred by injection, a portion also occurred by dermal route and ingestion. Most of the exposures occurred at the patient's own residence. Epinephrine auto-injector exposures tended to be managed outside of a healthcare facility and did not result in a serious outcome.

KEYWORDS Epinephrine, EpiPen, auto-injector

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101. Turpentine ingestions reported to poison centers

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Background: Turpentine is a fluid produced by the distillation of resin from live trees, mainly pines. Turpentine is composed of terpines. It is used as a solvent and as a raw material for the production of organic compounds. The Internet, including YouTube, contains a number of sites advocating drinking turpentine for health reasons, particularly the treatment for intestinal parasites. This study characterized turpentine ingestions reported to poison centers.
Methods: Cases were turpentine (Generic code 0039509) ingestions reported to a large, statewide poison center network during 2000-2018. The distribution of cases was determined for various factors related to patient demographics, exposure circumstances, management, and outcome.
Results: There 400 turpentine ingestions; the annual number declined from 36 in 2000 to 9 in 2013, then increased to 25 in 2018. There was no seasonal trend. The patient age distribution was 176 (44.0%) 5 years or less, 17 (4.3%) 6-12 years, 15 (3.8%) 13-19 years, 188 (47.0%) 20 years or more, and ; 4 (1.0%) unknown age; 209 (52.3%) were male, 187 (46.8%) female, and 4 (1.0%) unknown gender. The ingestion was unintentional in 332 (83.0%) of the exposures, intentional in 53 (13.3%) (including 17 or 4.3% intentional misuse), adverse reaction in 4 (1.0%), other in 7 (1.8%), and unknown in 4 (1.0%). The ingestion occurred at the patient's own residence in 362 (90.5%) of the cases, another residence in 25 (6.3%), the workplace in 7 (1.8%), and other or unknown sites in 6 (1.5%). The patient was managed on site in 239 (59.8%) of the cases, already at/en route to a healthcare facility in 98 (24.5%), referred to a healthcare facility in 56 (14.0%), and at other or unknown locations in 7 (1.8%). The distribution by medical outcome was 128 (32.0%) no effect, 84 (21.0%) minor effect, 19 (4.8%) moderate effect, 2 (0.5%) major effect, 11 (2.8%) not followed-judged nontoxic, 107 (26.8%) not followed-minimal effects possible, 40 (10.0%) unable to follow-potentially toxic, and 9 (2.3%) unrelated effect. No deaths were reported. The most common reported clinical effects were vomiting (n= 44, 11.0%), cough/choke (n= 36, 9.0%), nausea (n= 21, 5.3%), abdominal pain (n= 19, 4.8%), oral irritation (n= 15, 3.8%), and throat irritation (n= 14, 3.5%). The most common treatments were dilute/irrigate/wash (n= 257, 64.3%), food/snack (n= 22, 5.5%), and IV fluids (n= 20, 5.0%).
Conclusions: Patients who ingested turpentine were most often adults followed by young children. Most of the ingestions were unintentional and occurred at the patient's home. The majority of ingestions were not serious and were successfully managed outside of a healthcare facility.

KEYWORDS Turpentine, Terpine, Ingestion

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102. Lack of Systemic Toxicity after the Bite of the Cape Coral Cobra (Aspidelaps lubricus)

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Background: Aspidelaps lubricus, known as the Cape coral cobra or South African coral snake, is a small elapid native to the southern aspects of Africa. Like all elapids, it is venomous but has

relatively small front fangs. Due to its bright colors it is popular among venomous snake collectors. Its threat to humans is not well described but a recent publication reports respiratory failure after an envenomation by a captive A. lubricus. We describe an A. lubricus bite which resulted in local effects but no systemic toxicity.
Case report: A 25-year-old man was bitten by his captive Aspidelaps lubricus which he had bought online several years ago from South Africa. He was bitten on his left hand. He transported himself to a nearby emergency department, arriving approximately 20 minutes after the bite. Two bite marks were noted on the dorsum of his left hand over the middle metacarpals. His vital signs on arrival were a blood pressure of 113/83 mmHg, pulse 98 bpm, respiratory rate of 18 breaths per minute, and a temperature of 36.3° C. Erythema and swelling of the hand was noted but the patient denied any pain or tenderness. He also denied any weakness, numbness or difficulty breathing. Laboratory evaluation was essentially normal, including a hemoglobin of 15.2 g/dL, platelets of 219x103/mm3, white blood cell of 3.8x103/mm3, creatinine 1.1 mg/dL, PT 10.4 seconds and a fibrinogen of 230 mg/dL (normal 210-360 mg/dL). His tetanus status was updated but no other medications were given. A regional AZA accredited zoo was contacted regarding possible need for antivenom. The zoo carried Inoserp Pan Africa antivenom but did not feel it would be effective in this case and would not release it. The patient was admitted to the hospital for observation. The poison control center advised neostigmine if any neuromuscular symptoms developed. Overnight he did develop mild pain in the left hand but the swelling improved. Repeat labs remained normal. He never developed any neuromuscular symptoms and was discharged approximately 25 hours after the bite.
Case discussion: Human envenomations from Aspidelaps lubricus are rarely described in the medical literature. Animal studies demonstrate the venom is neurotoxic and there is a human case report of neurotoxicity after envenomation by a captive A. lubricus. There is no commercially available A. lubricus specific antivenom but a recent study suggests that South African Institute for Medical Research polyvalent antivenom would be effective. There is no evidence on the utility of Inoserp Pan Africa antivenom for A. lubricus envenomations but considering it is effective for other elapids, it would likely have some cross reactivity. Neostigmine could also be used in the setting of neurotoxicity, similar to recommendations for its use with South American coral snakes. Otherwise, supportive care with emphasis on respiratory support is the standard of care. Fortunately, though this patient had local effects, he never developed evidence of neurotoxicity.
Conclusion: In this case, a bite from an Aspidelaps lubricus resulted in local symptoms but no systemic toxicity.

KEYWORDS Snake, Elapid, Aspidelaps

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103. Acute Opioid Withdrawal Precipitated by Contrave®: A Novel Weight Loss Medication Containing Naltrexone and Bupropion

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Background: Increasing rates of obesity in North America have led to the development of pharmacologic agents for weight loss. Contrave®, an oral sustained release formulation of naltrexone 8mg and bupropion 90mg, has demonstrated modest ability to induce clinically relevant weight loss. We present a case of acute opioid withdrawal precipitated by Contrave®.
Case: A 55-year-old woman with a history of fibromyalgia and chronic lower back pain was prescribed Contrave® by her family physician for weight loss. Within an hour of her first dose, she became agitated, diaphoretic, and experienced severe nausea and vomiting. On arrival to the Emergency Department, she was noted to have dilated pupils with the following vitals: BP: 163/102, HR: 99, RR: 20, SpO2: 99% on room air. Upon further questioning, the patient revealed that she had been taking Tylenol #3 (acetaminophen 300 mg, caffeine 15 mg, codeine phosphate 30 mg) 1 tablet, up to 4 times daily for several years for pain control. She was diagnosed with acute opioid withdrawal and on the recommendation of the local poison centre was treated with IV fluids, lorazepam, ondansetron, and clonidine. Within 4 hours of presentation the withdrawal symptoms had improved, and by 12 hours she had returned to her clinical baseline and was discharged with instructions to discontinue Contrave®.
Discussion: Naltrexone and bupropion act synergistically in the hypothalamus and the mesolimbic dopamine pathway to increase satiety, reduce food intake, and increase energy expenditure. Both drugs have unique mechanisms of action and the potential for drug interactions that must be considered prior to use.Naltrexone – and its active metabolite 6β naltrexol – is a competitive opioid antagonist at the μ and κ opioid receptors. It is recommended that patients discontinue opioids at least 7-10 days prior to use of Contrave® because of the potential to precipitate acute opioid withdrawal. Bupropion is a dopamine, norepinephrine, and serotonin reuptake inhibitor, and is a potent cytochrome P450 2D6 (CYP2D6) inhibitor. Codeine is a prodrug that requires conversion to its active metabolite morphine by CYP2D6. Bupropion can potentially exacerbate opioid withdrawal in the setting of codeine use by inhibiting the conversion of codeine to morphine by CYP2D6.
Conclusion: We report a case of accidental opioid withdrawal in a woman with chronic opioid use after starting Contrave®. The patient was not counselled about the potential drug interactions between Contrave® and codeine or other opioids. To prevent opioid withdrawal with the use of Contrave®, healthcare providers should be aware of potential drug interactions, perform thorough medication reviews, and counsel patients about discontinuing other opioids prior to initiation.

KEYWORDS Opioid withdrawal, Contrave, Iatrogenic

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104. Comparative Toxicity of Venlafaxine and Duloxetine Utilizing Data Reported to the National Poison Data System

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Background: Venlafaxine (VLF) is a serotonin and norepinephrine reuptake inhibitor (SNRI). Its toxicity is characterized by neurologic and cardiovascular effects. These effects include agitation, tremors, seizures, tachycardia and conduction disturbances. Duloxetine (DUL) is another SNRI. Clinical effects for DUL exposures are characterized as causing minor toxicity and rarely result in serious morbidity or mortality. However, we were not able to identify any comparative studies between VLF and DUL characterizing their clinical effects and medical outcomes of exposures. The objective of this study was to compare the clinical effects and medical outcomes between VLF and DUL exposures reported to the National Poison Data System (NPDS).

Table 1 VLF vs DUL Symptom Comparisons (>1% incidence).

	VLF (%) n = 8309	DUL (%) n = 5481	P value	RR (95% CI)
Clinical Effects				
Abdominal pain	136 (1.64)	139 (2.54)	0.0003203	0.6454 (0.5108, 0.8155)
Acidosis	89 (1.07)	20 (0.36)	0.000002497	2.935 (1.81, 4.762)
Agitated/irritable	689 (8.29)	328 (5.98)	0.000000388	1.386 (1.22, 1.573)
Ataxia	88 (1.06)	58 (1.06)	>0.9999999	1.001 (0.7197, 1.392)
Conduction disturbance	335 (4.03)	91 (1.66)	<0.0000001	2.428 (1.931, 3.054)
Confusion	334 (4.02)	148 (2.70)	0.0000345	1.489 (1.23, 1.801)
CPK elevated	98 (1.18)	18 (0.33)	<0.0000001	3.591 (2.175, 5.93)
Diaphoresis	219 (2.64)	113 (2.06)	0.03486	1.278 (1.021, 1.6)
Diarrhea	57 (0.69)	72 (1.31)	0.0003046	0.5222 (0.3696, 0.738)
Dizziness/vertigo	416 (5.01)	273 (4.98)	0.9796	1.005 (0.8662, 1.166)
Drowsiness/lethargy	1344 (16.18)	897 (16.37)	0.7668	0.9884 (0.9148, 1.068)
Electrolyte abnormality	167 (2.01)	57 (1.04)	0.000008225	1.933 (1.434, 2.605)
Fever/hyperthermia	103 (1.24)	37 (0.68)	0.001267	1.836 (1.263, 2.669)
Hallucinations/delusions	130 (1.56)	47 (0.86)	0.0002988	1.825 (1.309, 2.543)
Headache	239 (2.88)	170 (3.10)	0.4757	0.9274 (0.7641, 1.126)
Hypertension	815 (9.81)	535 (9.76)	0.9266	1.005 (0.906, 1.115)
Hyperventilation/tachypnea	161 (1.94)	34 (0.62)	<0.0000001	3.124 (2.161, 4.515)
Hypotension	134 (1.61)	58 (1.06)	0.007286	1.524 (1.122, 2.07)
Muscle rigidity	83 (1.00)	23 (0.42)	0.0001181	2.38 (1.502, 3.773)
Mydriasis	451 (5.43)	154 (2.81)	<0.0000001	1.932 (1.614, 2.312)
Nausea	712 (8.57)	584 (10.65)	0.00003989	0.8042 (0.7248, 0.8923)
Seizure (single)	313 (3.77)	26 (0.47)	<0.0000001	7.941 (5.331, 11.83)
Seizure (multi/discrete)	128 (1.54)	5 (0.09)	<0.0000001	16.89 (6.916, 41.24)
Tachycardia	2200 (26.48)	977 (17.83)	<0.0000001	1.485 (1.389, 1.589)
Tremor	508 (6.11)	209 (3.81)	<0.0000001	1.603 (1.37, 1.877)
Vomiting	543 (6.54)	550 (10.03)	<0.0000001	0.6513 (0.5813, 0.7296)

Methods: A retrospective cohort study was conducted analyzing data from the NPDS for single agent exposures (VLF or DUL) reported to the American Association of Poison Control Centers (AAPCC) from August 2013 through December 2018. Excluded cases were those with multiple drug ingestions, non-ingestion route, outcomes that were unrelated, confirmed non-exposures, and no follow-up. Case outcomes as defined by AAPCC criteria and symptoms were compared. Chi-square or Fisher exact test, relative risk and 95% confidence intervals were calculated between VLF and DUL for non-significant (no effect/minor) and clinically significant (moderate/major/death) adverse outcome severity and common symptoms.
Results: There were 8309 VLF and 5481 DUL cases that met inclusion criteria. The majority of patients were female, 65.3% for VLF and 67.0% for DUL. Median age was 22 years old for VLF, and 20 years old for DUL. The odds of having a clinically significant (moderate/major/death) adverse outcome was significantly higher for VLF (RR =1.414, 95% CI =1.326-1.508). In the VLF patients, there was significantly greater risk of developing acidosis, agitation, conduction disturbances, confusion, CPK elevation, diaphoresis, electrolyte abnormalities, fever, hallucinations, tachypnea, hypotension, muscle rigidity, mydriasis, seizures, tachycardia and tremors (Table 1). DUL patients had significantly greater risk of having abdominal pain, nausea, vomiting and diarrhea (Table 1).
Conclusion: In this comparison of single agent ingestions of VLF or DUL, VLF patients had greater odds of having significant outcome severity coded as a moderate, major or fatal outcome. VLF exposures also had significantly greater incidences of neurologic and cardiovascular clinical effects. The results of this study demonstrate the relative safety of DUL when compared to VLF utilizing data reported to the NPDS. Importantly, these comparative study results are consistent with previous VLF or DUL reviews.

KEYWORDS Venlafaxine, duloxetine, comparative toxicity

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105. Phenibut exposures reported to a statewide poison center network

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Background: Phenibut (beta-phenyl-gamma-aminobutyric acid) is structurally similar to gamma-aminobutyric acid (GABA) and acts primarily at GABA-B receptors. It has many uses and is sold online as an anti-anxiety supplement, a nootropic or "smart drug," as a sleep aid, and is used recreationally. Phenibut is used legally in Russia to treat anxiety, insomnia, depression, post-traumatic stress disorder, and stuttering. Adverse symptoms reported with phenibut use alone included agitation, confusion, drowsiness/lethargy, hypertension, hypotension, hallucinations, delusions, nausea, vomiting, tachycardia, bradycardia, convulsions, insomnia, muscle rigidity, respiratory depression and tremors. The objective of this study was to describe phenibut exposures reported to a statewide poison center network.
Methods: Cases were phenibut exposures reported to a large, statewide poison center network during 2000-2018. Case distribution was determined for factors related to patient demographics, exposure circumstances, management, and outcome.
Results: Sixty-two phenibut exposures were identified. No exposures were reported prior to 2007; the annual number after that was 2 in 2007, 1 in 2010, 1 in 2011, 1 in 2012, 2 in 2013, 2 in 2014, 8 in 2015, 10 in 2016, 14 in 2017, and 21 in 2018. Twenty-eight (45.2%) of the exposures involved additional substances. Forty-six (74.2%) of the patients were male. Mean patient age was 29 years (range 15-58 years). Fifty-five (88.7%) exposures were by ingestion alone, 3 (4.8%) ingestion and inhalation, 1 (1.6%) inhalation alone, and 3 (4.8%) by an unknown route. Forty-three (69.4%) of the exposures were intentional (14

suspected attempted suicide, 11 abuse, 10 misuse, 8 intentional unknown), 4 (6.5%) unintentional, 5 (8.1%) withdrawal, 2 (3.2%) adverse reaction, and 8 (12.9%) unknown reason. Most (n = 53, 85.5%) exposures occurred at the patient's own residence, and 9 (14.5%) at other or unknown locations. The management site was 1 (1.6%) on site, 52 (83.9%) already at or en route to a healthcare facility, 7 (11.3%) referred to a healthcare facility, and 2 (3.2%) at an unknown location. The medical outcome was 6 (9.7%) no effect, 7 (11.3%) minor effect, 27 (43.5%) moderate effect, 7 (11.3%) major effect, 3 (4.8%) not followed-minimal clinical effects possible, 11 (17.7%) unable to follow-potentially toxic, and 1 (1.6%) unrelated effect. The most frequent clinical effects for phenibut and co-ingestants were drowsiness/lethargy (n = 22, 35.5%), agitated/irritable (n = 14, 22.6%), tachycardia (n = 12, 19.4%), hypertension (n = 11, 17.7%), confusion (n = 11, 17.7%), vomiting (n = 8, 12.9%), and bradycardia (n = 7, 11.3%). Results were similar for Phenibut-alone exposures: drowsiness/lethargy (n = 10, 29.4%), agitated/irritable (n = 8, 23.5%), tachycardia (n = 7, 20.6%), hypertension (n = 7, 20.6%), confusion (n = 6, 17.6%), vomiting (n = 5, 14.7%), and bradycardia (n = 4, 11.8%). The most frequent treatments were IV fluids (n = 36, 58.1%), benzodiazepines (n = 20, 32.3%), and oxygen (n = 9, 14.5%).
Conclusions: With a 10-fold increase in reported cases since 2007, this gabapentinoid may be another drug to watch for its abuse potential. Most phenibut exposures reportedly occurred by ingestion, were intentional, and resulted in moderate or major outcomes. Awareness of this exposure may arm healthcare providers with information on what to expect with Phenibut exposures.

KEYWORDS Phenibut, GABA, Nootropics

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106. An assessment of urine THC immunoassay in healthy volunteers receiving an oral proton-pump inhibitor

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Background: Urine drug-screening (UDS) is used in the healthcare setting to detect drugs of abuse. A limitation of UDS is the occurrence of false-positive results, triggering unnecessary testing and potentially negatively impact patient care. Pantoprazole, a proton-pump inhibitor (PPI), is reported to cause false positive tetrahydrocannabinol (THC) results on urine immunoassay. A warning was subsequently added to the package insert, describing the potential for cross-reactivity with THC urine immunoassays. However, this warning is based on limited case report data. Additionally, it is unknown whether this phenomenon is specific to pantoprazole or if it is a drug class effect. We conducted a prospective healthy volunteer study to determine if use of oral PPIs causes false-positive THC results on the One Step Marijuana Single Drug Test®.
Methods: The study was comprised of two phases, during which healthy volunteers completed a 5-day course of a PPI and underwent urine immunoassay testing. Healthy volunteers were defined as having no chronic medical conditions, not currently taking medications, not currently taking PPIs, and with normal vitals and physical exam. Female subjects underwent urine testing to confirm negative pregnancy status. All subjects underwent baseline urine testing with the One Step Marijuana Single Drug Test® to confirm a negative THC. Baseline urine samples underwent specific gravity testing for comparison to subsequent samples provided. All study materials and methods were approved by our Institutional Review Board and all subjects provided written informed consent at the time of enrollment. During phase one, subjects were given a 5-day supply of oral pantoprazole 40 mg and instructed to take the medication at the same time once daily. Subjects received a daily text message reminder to ensure medication compliance. Study subjects returned on day 5 to provide a urine sample. All samples were tested for specific gravity and compared to baseline urine. Samples were then tested using the One Step Marijuana Single Drug Test®. During phase two, subjects were randomized to a 5-day supply of once daily oral esomeprazole 20 mg, lansoprazole 15 mg, or omeprazole 20 mg. All study methods and testing mirrored phase one of the study.
Results: A total of 3 subjects were recruited for phase one and 9 subjects for phase two, with 3 subjects in each PPI group. All subjects completed the study protocol and reported 100% compliance with their medication. All baseline THC results were negative. Specific gravity did not significantly vary between samples. Urine samples collected on day 5 were negative for THC in all subjects.
Conclusions: This prospective healthy volunteer study provides data for assessing the ability of oral PPIs to cause a false-positive THC on a urine immunoassay. Our results demonstrate that PPIs did not cause a false-positive THC using the One Step Marijuana Single Drug Test®. Limitations of this study include a small sample size, use of a single immunoassay product, and the inability to directly confirm medication compliance. Further research assessing other urine immunoassay tools is needed to evaluate the potential for false-positive THC results in patients taking pantoprazole or another PPI.

KEYWORDS Proton-pump inhibitor, Tetrahydrocannabinol (THC), False-positive

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107. Elevated Osmol Gaps Not Explained by Toxic Alcohol Exposure: A Retrospective Review

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Background: Readily available toxic alcohol measurements with a short turnaround time are not routinely available in most clinical settings. Although the osmol gap is used as an aid in the evaluation of a possible toxic alcohol exposure, the osmol gap has many limitations and is commonly calculated when a toxic alcohol exposure is considered. However, not all elevated osmol gaps are explained by toxic alcohols. Previously, reports have associated an elevated osmol gap with diagnoses including ketoacidosis, lactic acidosis, and renal failure. Our objective was a descriptive review of diagnoses associated with an elevated osmol gap and negative assay for ethylene glycol and methanol at a University Hospital.
Methods: An Institutional Review Board waiver of consent was obtained for this study. We reviewed data of patients presenting with an elevated osmol gap and negative ethylene glycol and methanol assay from Jan 1, 2014 to Dec 31, 2018. Baseline demographic and clinical lab data were extracted and the final diagnosis recorded for each patient.
Results: The total number of patients over the study timeframe were 45 (n = 45) with negative ethylene glycol and methanol concentrations. Of these 45 patients, 11 had an elevated osmol gap (> 10 mOsm). The diagnoses were ketoacidosis in 7 of 11 (3 diabetic, 3 alcoholic and one undefined), pancreatitis in 2 (both with ketoacidosis), and ethanol intoxication, isopropanol intoxication, acute liver failure (secondary to

massive acetaminophen overdose), and unexplained lactic acidosis in one each. Five of the 11 had acute kidney injury (AKI), 2 of these 5 were acute-on-chronic renal failure. Only one patient (acute liver failure) had a lactate >10 mmol/L. Nine of the 11 had an anion gap acidosis (range 23 to 43) while 2 had an anion gap ≤15. The two without an anion gap were ethanol (530 mg/dL) and isopropanol intoxication, one each. Osmol gap range was 12 to 65, with only two > than 30 (isopropanol in one, DKA in one).
Conclusion: The most common cause of an elevated osmol gap in our small series was ketoacidosis. Acute kidney injury often accompanied this. The two cases without an elevated anion gap were due to either ethanol or isopropanol intoxication. The osmol gap in the case of ethanol intoxication (530 mg/dL) would have been 0 had we used one of the alternate proposed ethanol correction factors, 4.0 rather than 4.6 for calculation of osmolarity. Our results are consistent with previous studies that found ketoacidosis, lactic acidosis, and renal failure to be the most common causes of an elevated osmol gap when a toxic alcohol was excluded. Our study was limited by the small number of cases and as well by exclusion of cases lacking a contemporary ethanol measurement with the measured osmolality. Our 5-year review of cases with negative ethylene glycol and methanol assay revealed that an osmol gap >10 mOsm with an anion gap >15 was most commonly associated with ketoacidosis, with AKI a common co-morbidity.

KEYWORDS Osmol gap, Toxic Alcohol, Ketoacidosis

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108. A systematic review of second-line therapies in toxic seizures

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Background: Seizures are a common symptom of toxic exposures and require immediate and possibly ongoing management. Guidelines recommend benzodiazepines as first line therapy for toxic seizures, however, there is paucity of literature regarding secondary treatment selection. We aim to systematically evaluate the available literature on the treatment regimens utilized for second line treatment of toxic seizures.
Methods: We searched PubMed, Embase, PsychINFO, Cochrane Library, Web of Science, Google Scholar, and International Pharmaceutical Abstracts from inception through August of 2018, following PRISMA Guideline. The MESH terms focused on identifying treatments for toxic seizures. We excluded articles if they involved animals, had seizures resulting from alcohol, were case reports, or not peer reviewed. Next, two investigators screened citations, then four authors reviewed all papers for eligibility. To minimize bias, screening and eligibility phases utilized Rayaan, a systematic review application, so the authors were blinded to the results of others. Once the review was completed, we discussed discrepancies as a group, and we had no disagreements. Then we abstracted the included papers into an Excel file for demographics, first line agent used, second line agent used, effectiveness of treatment and outcomes. Our primary outcome was seizure termination and/or suppression of the second line agent as determined by the primary author. We used descriptive statistics for analysis.
Results: We identified 2594 studies following the initial search. After screening and removal of duplicates, there were 62 articles remaining. Following eligibility, we identified six studies, which were all case series. The most common reasons for study exclusion were non-predefined outcomes and deficient study design. Included case series contained nine to 235 patient cases each. The age range for all patients discussed was between two and 82 years. Most exposures were single agents, and the most common xenobiotics were bupropion, isoniazid, and anti-psychotics. The description of seizures was diverse, but mostly complex tonic-clonic seizures or status epilepticus. First-line treatments were primarily benzodiazepines, but also included phenobarbital, propofol, and phenytoin. Secondary treatments included propofol, barbiturates, physostigmine, phenytoin, valproic acid, and levetiracetam. Patient outcomes differed, attributable to any combination of mixed toxic substances, drug-drug interaction, inability to control seizures, or toxicity of the AED's themselves. More severe ingestions resulted in coma, recurrent seizures, and/or death. However, few cases specifically discussed the success of seizure termination and/or suppression due to secondary treatment administration. Two of the six studies evaluated treatments for toxic substances no longer routinely seen (e.g. tetramethylenedisulfotetramine and maprotiline).
Conclusions: Available literature discussing second-line treatment for toxic seizures is poor quality with high heterogeneity. Although similar second-line agents were used in the majority of articles, it is difficult to compare the efficacy of these agents. Additional studies are necessary to identify the most efficacious second-line therapies in toxic seizures.

KEYWORDS Refractory, epilepticus, treatment

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109. Crotalidae Immune F(ab')2 Antivenom Effectively Treats Neurotoxic Manifestations of Mohave Rattlesnake (Crotalus scutulatus scutulatus) Envenomation

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Background: In the southwestern United States, rattlesnake envenomation causes predominately local cytotoxic and hemotoxic effects. Neurotoxicity may also occur, and the Mohave rattlesnake (C. s. scutulatus) is known for its neurotoxic effects due to the presence of phospholipase A2, known as Mohave toxin. The Mohave rattlesnake has three venom phenotypes: Type A (neurotoxic), Type B (hemorrhagic), or a combination Type A + B. Mohave rattlesnakes inhabit a large range within the desert southwest, but their venom components and phenotypes vary geographically. Signs and symptoms of neurotoxicity may include dizziness, vision changes, emesis, diarrhea, paresthesias, fasciculations, and seizures. Airway edema and endotracheal intubation rarely have been reported. To our knowledge this is the first reported case of severe neurotoxicity with angioedema requiring intubation in a child who was envenomated by a Mohave rattlesnake and was treated with crotalidae immune F(ab')2 antivenom.
Case Report: A 6-year-old boy was riding his bicycle outside his home in the desert, when he crash landed atop a rattlesnake. It was expertly identified as C. s. scutulatus. The snake bit him on the right patella causing immediate dizziness. His mother rushed him to the local fire station, where EMS noted vomiting, loose stool, and tongue swelling. He was hypotensive at 60/30 mmHg with tachycardia up to 180 bpm. He became somnolent responding only to pain and was intubated for airway protection. He was airlifted to a tertiary care pediatric emergency department. There he was given two doses of epinephrine, as well as diphenhydramine, famotidine, and dexamethasone. Ten vials of crotalidae immune F(ab')2 antivenom were then administered. Physical exam was remarkable for prominent angioedema involving the tongue, and his right lower extremity had moderate edema that extended from the distal thigh to the proximal calf. It did not progress

after antivenom administration. He was extubated the following day with resolution of angioedema. Initial lab results were WBC 58.6 k/mm3, platelets 504 k/mm3, fibrinogen 209 mg/dL, PT 13 seconds, INR 1.1, PTT 28.1 seconds, and d-dimer 2539 ng/mL. After antivenom WBC decreased to 18.8 k/mm3, platelet nadir 246 k/mm3, fibrinogen nadir 182 mg/dL, and peak PTT 28.1 seconds. Approximately 36 hours later, his PT/INR rose slightly to 14.6 seconds/1.3, and the patient was administered another four vials of crotalidae immune F(ab')2 antivenom. Additionally the patient exhibited myotoxicity (peak creatine kinase 7629 IU/L) without acute kidney injury and at time of abstract submission is expected to be discharged home approximately 72 hours after hospital arrival.

Discussion: Mohave rattlesnake envenomation may result in cytotoxic, hemotoxic, myotoxic, and neurotoxic effects. Neurotoxic effects occur rarely, however the clinical presentation may be severe. Severe angioedema occurs even less commonly than other neurotoxic manifestations, but it is important to be aware of the possibility for critical airway compromise in the rattlesnake-envenomated patient. Crotalidae immune F(ab')2 antivenom is effective in treating severe C. s. scutulatus bites with neurotoxic effects including angioedema.

KEYWORDS Rattlesnake, neurotoxic, venom

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110. Chronic Bromide Toxicity Superimposed on Ethanol Withdrawal Syndrome

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Background: Reports of bromide toxicity in the literature have declined since the removal of bromide salts from most medications in 1975. Products containing bromide are still available for purchase and used for epilepsy in animals. Chronic bromide toxicity may cause "bromism," a neurologic and psychologic syndrome which can be confused with alcohol withdrawal. We present a case of chronic, intentional use of potassium bromide resulting in bromide toxicity with confirmatory levels treated by hemodialysis.

Case Report: A 51-year-old male presented to the emergency department with altered mental status and rectal pain. He was confused, agitated, and tremulous with a past medical history of ethanol withdrawal. Initial chemistries were normal except for a chloride >145mEq/L, creatinine 1.21mg/dL and anion gap -28. The patient was admitted for ethanol withdrawal treatment and monitoring of his profound hyperchloremia with initial concern for a prolonged bromide exposure. The poison center recommended crystalloid resuscitation, a bromide level, and indicated the potential need for hemodialysis. Early nephrology consultation was obtained, but hemodialysis was deemed not necessary by the onsite nephrologists. By hospital day eight, the chloride level was consistently >145 mEq/L, the patient remained altered and a bromide level was ordered. The bromide resulted at 259.1 mg/dL on hospital day eleven and hemodialysis (HD) was initiated. Immediately post HD, the patient was reported to be alert and oriented with no agitation. Post-HD labs reported a chloride level >145mEq/L and bromide level of 40.3 mg/dL. A second HD session was performed the following day with a post-HD chloride level of 112 mEq/L (see graph). On day 13, family brought in laboratory grade potassium bromide purchased from the internet. The patient had been taking 0.5 - 1 teaspoon twice daily for perceived health benefits for the "past couple of months." The patient was discharged at baseline health on day 14.

Case Discussion: This case highlights associated symptoms of chronic bromide toxicity causing bromism. Hemodialysis was a definitive treatment for our patient and has been reported to be an effective enhanced elimination technique. Toxic bromide concentrations are considered >50 mg/dL with severe toxicity at concentrations >200 mg/dL and death at >300mg/dL. Our patient displayed signs of bromism with initial bromide level consistent with previously reported cases. The profound hyperchloremia and negative anion gap are consistent with bromide toxicity and may be used to differentiate bromism from ethanol withdrawal. Elevated chloride levels are due to interference of laboratory instrumentation by the bromide ion.

Conclusion: Hemodialysis is an effective treatment for bromide toxicity. Elevated chloride levels and a low or negative anion gap should heighten suspicion for this diagnosis. In patients with mental status changes and marked hyperchloremia and low anion gap, bromide toxicity should be considered.

KEYWORDS Bromism, Potassium Bromide, Hemodialysis

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111. From Surgeonfish to Surgery

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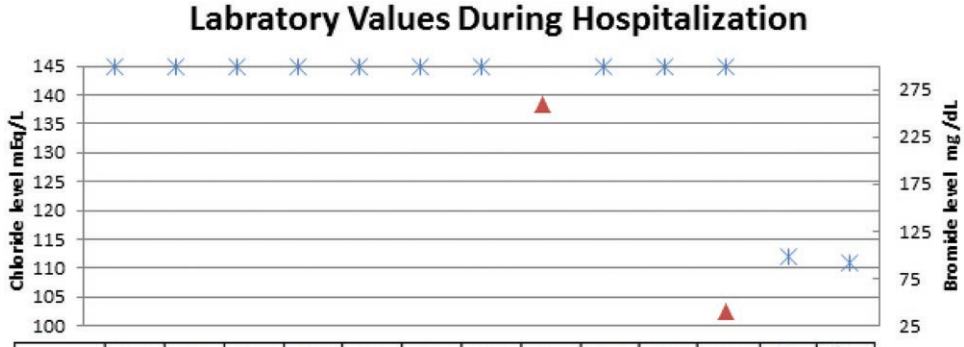
Einstein Healthcare Network

Background: The clown surgeonfish Acanthurus lineatus is prized in saltwater aquariums for its bright colors and stripes. Unlike most surgeonfish, A. lineatus is territorial and aggressive. It is a spiny-rayed fish



	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Day 12	Day 13
Chloride Level	> 145	> 145	> 145	> 145	> 145	> 145	> 145		> 145	> 145	> 145	112	111
Bromide Level								259			40.3		

with unretractable caudal spines containing venom sacs. Like most spiny-rayed fish, the venom apparatus remains poorly understood and the venom has not been characterized. Although few published case reports exist of envenomations by A. lineatus, reported symptoms include severe pain and swelling at or adjacent to the envenomation site. Reported systemic symptoms include nausea, vomiting, and lightheadedness. To our knowledge, this is the first published report of flexor tenosynovitis (FTS) from A. lineatus envenomation.
Case report: A healthy 35-year-old aquarium hobbyist presented to the emergency department with severe left hand pain after cleaning his fish tank, believing that he was stung by his A. lineatus. He presented mildly tachypneic, which improved with morphine and hydromorphone, although he continued to report severe pain. Examination was significant for a puncture wound of the volar surface of the left middle finger with circumferential swelling. He was neurovascularly intact. He was treated empirically with ceftriaxone and doxycycline and admitted to the hospital for observation. Adjunctive gabapentin improved his pain. Over the next 12 hours, his pain changed from burning to throbbing in quality with minimal improvement in severity. His erythema, swelling, pain, and tenderness migrated proximally, consistent with FTS. He underwent surgical incision and drainage 24 hours after presentation, revealing cloudy subcutaneous fluid extending into the flexor tendon sheath with an injury to the A3 pulley, without tendon laceration. A drain was placed then removed after two days. He was continued on outpatient cephalexin. Operative samples of tissue and fluid grew no aerobic or anaerobic organisms. Follow up at one month revealed persistent extensor and flexor contractures and stiffness. Physical/occupational therapy frequency was increased, and near-complete range of motion was observed at two months post-injury.
Discussion: This is the first reported case of envenomation by A. lineatus resulting in FTS. The negative cultures suggest a toxic or inflammatory etiology of this rapidly progressive FTS. Atypical marine bacteria, such as Mycobacterium marinum, cause delayed, chronic FTS. There has never been a case with symptom onset earlier than two weeks after initial injury, making atypical bacterial infection unlikely. Inflammatory FTS is usually treated conservatively with non-steroidal anti-inflammatory drugs, ice, and splinting, while infectious FTS benefits from early surgical intervention. Thus, distinguishing toxic FTS from marine envenomation from infectious FTS may direct management. More research on the pathophysiology of the toxin is needed.
Conclusion: Most marine toxins remain undescribed. Though most A. lineatus stings cause self-limited symptoms, deeper complications such as FTS can occur. Despite general clinical awareness of toxic FTS, there remains a paucity of literature about the phenomenon. Thus, best practices regarding management remain unknown.

KEYWORDS Marine, envenomation, tenosynovitis

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112. Isoxazoline exposures reported to a statewide poison center network

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Background: Isoxazolines are a Food and Drug Administration (FDA)-approved class of pesticides for the treatment and prevention of flea and tick infestations in animals. Isoxazolines inhibit insects' GABA-gated chloride channels, thereby causing death from uncontrollable central nervous system stimulation. Reportedly, isoxazolines are low in toxicity in mammals due to a lower sensitivity of mammalian GABA receptors. Most isoxazoline products are available as pills or chews, while one form is a topical spot-on product. In September 2018, the FDA issued an alert for potential neurologic adverse reactions including muscle tremors and seizures in dogs and cats treated with these products. The study aim was to describe human isoxazoline exposures reported to a statewide poison network.
Methods: Cases were isoxazoline exposures reported to a statewide poison center network during 2000-2018. Cases were identified by reviewing the substance description field for any of the specific isoxazolines and their brand names: afoxolaner (Nexgard®), fluralaner (Bravecto®), sarolaner (Simparica®), and lotilaner (Credelio®). Case distribution was determined for patient demographics, exposure circumstances, management, and outcome.
Results: Of 83 isoxazoline exposures, 42 (50.6%) were afoxolaner, 32 (38.6%) fluralaner, 8 (9.6%) sarolaner, and 1 (1.2%), lotilaner. No exposures were reported prior to 2014. The annual number of exposures was 3 in 2014, 5 in 2015, 17 in 2016, 27 in 2017, and 31 in 2018. Patient age distribution was 41 (49.4%) 0-5 years, 5 (6.0%) 6-12 years, 1 (1.2%) 13-19 years, and 36 (43.4%) 20 years or more; 43 (51.8%) of the patients were male and 40 (48.2%) patients were female. Seventy-seven (92.8%) exposures were by ingestion alone, 4 (4.8%) dermal alone, and 2 (2.4%) by ingestion and dermal route. Eighty-one (97.6%) exposures were unintentional, 1 (1.2%) contamination/tampering, and 1 (1.2%) adverse reaction. All of the exposures occurred at the patient's own residence. The management site was 68 (81.9%) on site, 13 (15.7%) already at or en route to a healthcare facility, 1 (1.2%) referred to a healthcare facility, and 1 (1.2%) at an unspecified other location. Medical outcomes were 19 (22.9%) no effect, 3 (3.6%) minor effect, 6 (7.2%) not followed-judged nontoxic, 52 (62.7%) not followed-minimal clinical effects possible, 1 (1.2%) unable to follow-potentially toxic, and 2 (2.4%) unrelated effect. The reported clinical effects were vomiting (n = 4, 4.8%), nausea (n = 2, 2.4%), dermal irritation/pain (n = 2, 2.4%), abdominal pain (n = 2, 2.4%), confusion (n = 1, 1.2%), and unspecified other (n = 2, 2.4%). The reported treatments were dilute/irrigate/wash (n = 48, 57.8%), food/snack (n = 19, 22.9%), antiemetics (n = 2, 2.4%), and unspecified other (n = 4, 4.8%).
Conclusions: Few isoxazoline exposures were reported and involved mostly young children. Most exposures involved ingestion, were unintentional, and occurred at the patient's own home. Most patients were managed outside of a healthcare facility and experienced only minor gastrointestinal effects.

KEYWORDS Isoxazoline, Seizures, Animals

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113. Houston, we have a "cycad" problem: Geographic distribution of sago cycad (Cycas revoluta) reported to Texas poison centers

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Background: Sago cycad (Cycas revoluta) is a gymnosperm that thrives in warm temperate and subtropical regions of the United States. The popularity of C. revoluta as an ornamental plant has increased significantly in recent years due to its hardiness and ease of care. The whole plant of C. revoluta, including the seeds, contains toxins such as cycasin, beta-methylamino L-alanine, and cyanogenic glycoside. These toxins are harmful to humans and animals if ingested. Even a young plant contains enough toxin to be lethal to animals. This study identified the distribution of one state's counties where C. revoluta exposures and information calls originated and were reported to a

statewide poison center network and made comparisons between the types of exposure/call (human exposure, animal exposure, or information call).
Methods: Cases were C. revoluta human exposures, animal exposures, and information calls reported to a poison center network during 2000-2018 where the caller county was known. The distribution of cases was determined for caller county and Public Health Region (PHR). A PHR is a cluster of counties grouped together for public health administrative purposes. We compared the origin of exposures/calls by county and PHR with the type of call (human exposure, animal exposure, information).
Results: A total of 455 cases met study criteria including 183 (40.2%) human exposures, 113 (24.8%) animal exposures, and 159 (34.9%) information calls. Of the 183 human exposures, 157 (85.8%) were from PHRs 6-8 (Central and Southeast Texas), 113 (61.7%) from PHR 6 (Southeast Texas), and 72 (39.3%) from Harris County (city of Houston). Of the 113 animal exposures, 96 (85.0%) were from PHRs 6-8, 74 (65.5%) from PHR 6, and 54 (47.8%) from Harris County. Of the 159 information calls, 137 (86.2%) were from PHRs 6-8, 101 (63.5%) from PHR 6, and 82 (51.6%) from Harris County.
Discussion: Over 80% of the C. revoluta exposures and information calls reported were from Central and Southeast Texas with over 60% reported from Southeast Texas; 40-50% were from Harris County (Houston). The same pattern was observed for human and animal exposures and information calls. The geographic pattern of the calls may reflect geographic differences in the abundance of C. revoluta or the tendency to contact poison centers.
Conclusions: Most human exposure and information calls on C. revoluta originated from Central and Southeast Texas. Information on the geographic distribution of C. revoluta exposures and information calls may be useful to target education and prevention activities.

KEYWORDS Sago cycad, plant toxicity, Texas

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114. Can the FDA's Adverse Event Reporting System (FAERS) Data Be Used to Assess the Impact of interventions Such As Risk Evaluation and Mitigation Strategies (REMS)?

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Background: The FDA's Adverse Event Reporting System (FAERS) is a publicly available database of voluntary reporting for adverse drugs events by both the public and health care professionals designed to support the FDA's post-marketing safety surveillance program. It is difficult to fully determine the safety profile of a medication before market approval. For this reason ongoing post-market safety surveillance is paramount to monitoring safety of new medications. For drugs with concerning safety profiles the FDA has the opportunity to institute REMS, an approach to track and assure safe use of medications. We sought to evaluate whether FAERS data could provide an accurate reflection of adverse drug event timing.
Methods: Adverse Event Open Learning through Universal Standardization (AEOLUS) database provides a curated de-duplicated, and cleaned version of FAERS that is standardized and linked to RxNorm to consolidate brand names for the years 2006-2014. The AEOLUS database was downloaded and MySQL queries were created to pull drugs and associated adverse outcomes. AEOLUS was linked to FAERS in order to get temporal information of "Event Date" and "Report Date."
Results: The highest number of records 38, 363 demonstrated a time lag of 13 days between "Event Date" and "Report Date." The average time lag between the "Event Date" and "Report Date" averaged 520 days with a median of 512.5 days. The minimum time from "Event Date" to "Report Date" was 0 days. Maximum time of "Event Date" to "Report Date" was 6,477days. Roughly 50% of all records have no "Event Date" at all.
Conclusion: Given the wide variability and substantial time lag between event and report date it may be challenging to track changes in adverse events before and after an incident or initiation of a harm reduction program such as REMS using FAERS. Outliers skewed the average time lag from event to report. Lack of "Event Date" in 50% of FAERS reports further limits the ability to accurately date reported events. Despite these limitations the majority of adverse event reports stated an "Event Date" within weeks of the FAERS report with the maximum count of reports occurring at a time lag of 13 days. This suggests that most reports without an "Event Date" listed likely occurred close to the report date. Further evaluation of FAERS reports without an "Event Day" is warranted to better determine the accuracy of timing for adverse events in FAERS.

KEYWORDS FDA, adverse drug event, post-marketing surveillance

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115. Bupivicaine Toxicity After Greater Occipital Nerve Block in a Patient with a Skull Defect

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Background: Occipital nerve blocks by local anesthetics are being used for management of chronic headaches. Toxicity of local anesthetics is directly related to quantity of the drug that spreads systemically. The onset and severity of toxicity is amplified if intracirculatory injection occurs. A relatively unreported cause of toxicity is direct central nervous system (CNS) exposure.
Case report: A 32-year-old female (weight =54.4 kg) consented to bilateral greater occipital nerve blocks for intractable headaches. The patient had had previous occipitial nerve blocks with no complications. She had a previous medical history of a medulloblastoma as an infant resulting in a posterior fossa craniotomy with a resulting left sided skull boney defect and a ventriculoperitoneal (VP) shunt. Bupivicaine 0.75% injection 5 mL was injected around the right greater occipital nerve and 4 mL around the left greater occipital nerve near the VP shunt tubing. Within a few minutes after the left sided injection, the patient described increased pain in the area and then became unresponsive and cyanotic with no pulse. Compressions were initiated and the initial rhythm was reported as pulseless electrical activity. She was intubated and transferred to the Emergency Department where she was found to have a pulse; however, she developed seizure like activity that was treated with intravenous lorazepam and propofol. CT of the head and CT angiography did not demonstrate any obvious lesions other than the known left sided skull defect. Shunt series demonstrated intact tubing. She continued to have seizure like activity and a lipid emulsion 20% 1.5 mL/kg was loaded over an hour followed by an infusion 0.25ml/kg/hour for 4 hours. Subsequent electroencephalogram demonstrated diffuse slowing. She was moving all extremities the next day and extubated hospital day #3.
Case discussion: Bupivicaine is known to exert cardiovascular and neurologic toxicity at dosing that is less than other local anesthetics. The typical recommended ceiling dose is 2 mg/kg. The maximum dose documented in the medical record for this patient was

9mL of 0.75% solution which would total 67.5 mg. The temporal response to the injection in the area of the skull defect to the collapse to the patient resulted in concern for inadvertent CNS injection. In one case report, cardiopulmonary collapse after retrobulbar anesthesia with bupivicaine occurred in patient who had an orbital roof defect.
Conclusion: We present a case of a 32 year-old-female who has a skull defect and developed CNS toxicity likely due to bupivacaine injection into the CNS during a greater occipital nerve block.

KEYWORDS Bupivicaine, neurotoxicity, therapeutic misadventure

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116. Grade IV Centruroides Envenomation in an Adult Male Treated with Antivenom

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Background: Scorpion envenomation is a common call to poison centers with the majority occurring in adult patients who generally sustain minimal and localized symptoms. This is in contrast to pediatric envenomations which more frequently present with systemic symptoms. This is reflected in the medical literature which largely focuses on pediatric envenomations resulting in a dearth of literature describing adult envenomations especially those requiring antivenom. We present here a case of severe scorpion envenomation in an adult treated with antivenom.
Case: A 47 year old man with a past medical history of cerebrovascular accident, hypertension, high cholesterol, and alcohol abuse presented to a rural emergency department following a scorpion envenomation to the left shoulder presumed to be due to the Centruroides sculpturatus based on symptomatology and geography. He presented with pain, hypertension, hypersalivation, involuntary motor activity, and opsoclonus which qualifying him as a grade IV envenomation based on previously defined criteria. No antivenom was available at his presenting healthcare facility so he was treated supportively with 3 mg IV lorazepam, 320 ug IV fentanyl, 0.5 mg atropine, and 0.1 mg glycopyrrolate with reported improvement in his symptoms prior to being transferred by air ambulance to a regional academic tertiary care facility. On arrival, his vital signs were notable for tachycardia and his exam continued to demonstrate pain, hypersalivation, involuntary motor activity, and opsoclonus. A trial dose of 1 vial of Centruroides Immune F(ab')2 antivenom was administered along with 1 mg IV lorazepam and 1 mg IV midazolam with subsequent improvement in tachycardia and resolution of opsoclonus. An additional 2 vials of antivenom were administered with subsequent normalization of the patient's vital signs and involuntary motor activity. A complete blood count, complete metabolic profile, urine drug screen, creatine kinase, ethanol level and electrocardiogram were normal. Prothrombin time was minimally elevated above normal though felt to be unrelated. On reassessment, the patient had mild confusion and there was concern for possible alcohol withdrawal given his history of alcohol abuse and undetectable ethanol level. He was hospitalized for observation, required no additional treatments, and was discharged in the morning with normal vital signs and no ongoing symptoms.
Case Discussion: This case describes an adult male with Grade IV Centruroides sculpturatus envenomation successfully treated with 3 vials of Centruroides Immune F(ab')2 antivenom. Supportive care with benzodiazepines, opioids, atropine, and glycopyrrolate was only somewhat effective in this case. A component of alcohol withdrawal was initially suspected as a contributing factor in this case but the lack of any additional treatment after antivenom administration argues against this. Availability of antivenom at the initial healthcare facility may have saved the cost and risk of transfer.
Conclusions: Severe scorpion envenomation among adults in North America is rare but under-described in the medical literature. This case contributes our experience to the medical literature and reaffirms the utility of antivenom therapy in select cases of adult envenomation as well as the potential utility of atropine and glycopyrrolate as a temporizing measure if antivenom is not available.

KEYWORDS Scorpion, Envenomation, Antivenom

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117. Motrin induced myotomy: The case of the broken heart

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Background: Ibuprofen is a very common exposure, but severe toxicity is rare. Invasive cardiac procedures are rarely needed for drug related toxicity. We discuss a massive ibuprofen overdose that resulted in severe left ventricular failure, requiring a septostomy for treatment.
Case Report: A 17-year-old female presented to the emergency department about two-hours after an acute ingestion of 100 grams of Ibuprofen (1,466 mg/kg dose). On presentation, she was vomiting, confused, and agitated. Over the next few hours she developed CNS depression and hypotension with systolic blood pressures in the 80s. Initial labs showed lactic acidosis, which deteriorated to an arterial pH 6.99 and lactate 9.3 mmol/L within 16 hours. She had continued hypotension despite multiple vasopressors. On hospital day 1, the patient was intubated and transferred to another hospital where she was immediately placed on venous arterial extracorporeal membrane oxygenation (VA-ECMO). After ECMO initiation, she needed less pressor support but still required multiple agents. Due to continuing acidosis and developing acute kidney injury, she was placed on continuous veno-venous hemodialysis. On hospital day 2, she had multiple transesophageal echocardiograms which revealed worsening heart failure with left atrial hypertension and a dilated left ventricle. As such, an atrial septostomy was performed to decompress the left atrium on hospital day 2. She was weaned off of ECMO on hospital day 5. During her hospitalization, the patient had severe upper GI bleeding that required endoscopy with cauterization and transfusion of 23 units of packed red blood cells (PRBCs). She was extubated on hospital day 6 and eventually discharged neurologically intact. She has had subsequent cardiology visits and evaluations with no indication of long-term limitations from this procedure, and has even been able to run a marathon without issue.
Case Discussion: Ibuprofen ingestions are frequently reported to poison control centers, but severe effects occur infrequently. We believe our patient's heart failure was likely caused by ibuprofen precipitated myocardial dysfunction from hypotension associated lactic acidosis and worsening anion gap metabolic acidosis. While ECMO is a life-saving intervention for critically ill poisoned patients, our patient's severe ventricular failure required a more aggressive measure. A myotomy, more specifically an atrial septostomy, is performed via cardiac catherization using transseptal piercing and balloon dilation. Unlike other ventricular offloading treatment options, such as an intraaortic balloon pump, a septostomy has the advantage of not requiring additional procedures for removal once the patient has been weaned from ECMO. There are reports of septostomy in patients requiring ECMO due to other lactic acidosis etiologies, such as sepsis. However, to our knowledge, there are no reports of septostomy as a treatment for toxicity from overdose.

Conclusion: We are unaware of published reports of severe ibuprofen overdose treated with septostomy and of only one case report of an ibuprofen overdose successfully treated with ECMO. Given our patient's excellent recovery, this novel treatment could be a viable option in overdoses with similar cardiovascular collapse.

KEYWORDS Ibuprofen, ECMO, Septostomy

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118. Is that really the level? On the imprecision of commonly ordered toxicologic assays

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Background: Many interventions in medical toxicology are initiated once the serum drug or metabolite concentration exceeds a threshold value. However, toxicologists may be uninformed or misinformed about the precision of their laboratory assays. Differences in instrument precision may be an underrecognized source of harm when it comes to managing the poisoned patient.

Methods: We accessed the Food and Drug Administration's (FDA) Clinical Laboratory Improvement Amendments database and manufacturer websites to obtain data on the analytical precision of all instruments which measure the following: acetaminophen, salicylate, iron, lead, lithium, and lactate. These measurands were chosen because there exist agreed-upon serum threshold concentrations which trigger costly or high-risk clinical actions (e.g. hemodialysis for serum [salicylate] > 100 mg/dL, chelation for serum [lead] > 50 ug/L). All instrument manufacturers must perform precision testing and provide the "total run" standard deviation (SD) of an instrument before seeking FDA approval. The total run SD is obtained for multiple measurand concentrations by analyzing a reference standard of known concentration over multiple days on the same instrument,

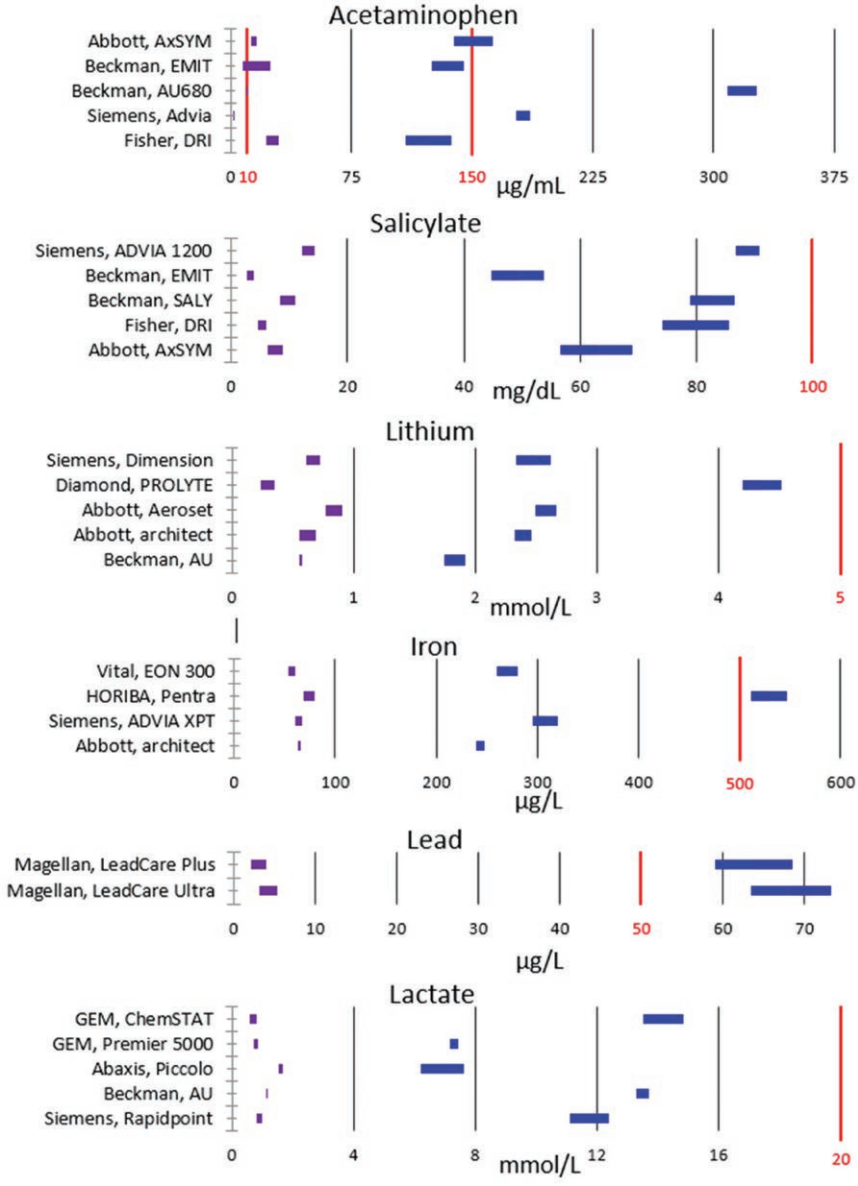

Figure 1 Analytical precision of commercial toxicologic laboratory assays for acetaminophen, lactate, lead, lithium, iron, and salicylate. Bars represent 95% confidence intervals based on a single patient measurement. Thresholds values for critical therapeutic actions are indicated by red lines.

then aggregating the error. We systematically collected the total run SD at the highest and lowest measurand concentrations reported for each instrument. We used these data to calculate 95% confidence intervals (95% CI) based on a single patient measurement to render them clinically meaningful.
Results: Precision data were available for between 2-5 instruments per measurand (Figure 1). Precision decreased with increasing measurand concentration for all instruments. Near the 10 ug/mL threshold for acetaminophen, the most precise instrument (Siemens, Advia) had a 95% CI range of 0.7 ug/mL while the least precise instrument (Beckman, EMIT) had a 95% CI range of 18 ug/mL. We observed that many manufacturers did not validate instrument precision at measurand concentrations near threshold values: all five salicylate assays failed to report precision data for serum concentration above 100 mg/dL. It is likely that measurand concentrations near these thresholds in clinical practice are less precise than even our data suggest. Low precision in this context will lead to both systematic overtreatment and undertreatment of patients (Figure 2). Reassuringly, instrument precision has improved with time. The Abbott AxSYM and Fisher DRI platforms were the oldest and consistently had the lowest precision while the current generation Abbott ARCHITECT platform had the highest precision across multiple measurands.
Conclusions: Our study highlights the importance of contextualizing laboratory test results within the overall evaluation of the poisoned patient, particularly when those results are near a threshold value which requires high-risk interventions. Although results are reported without confidence intervals in the clinical setting, all measurements are inherently imprecise. We recommend that toxicologists collaborate with institutional laboratory services to determine the instrument-specific precision of frequently ordered toxicologic tests.

KEYWORDS Precision, Analytical toxicology, Clinical Chemistry

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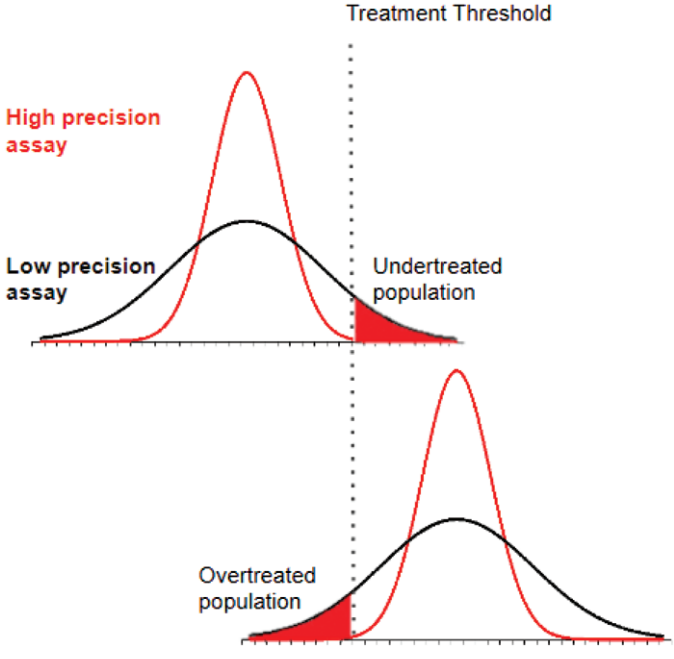

Figure 2 In a population of patients with the same assay result, a low precision instrument leads to the undertreatment of patients with results below the threshold and overtreatment of patients with results above the threshold.

119. Parental vs Enteral N-acetylcysteine in Adolescent Acetaminophen Poisoned Patients

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Background: US poison centers experience a high rate of adolescent acetaminophen intentional ingestion. Despite IV n-acetylcysteine's availability for more than 15 years, many hospitals continue to utilize oral NAC due to its favorable cost profile compared to the IV formulation. The goal of this study is to identify treatment failures with oral administration of NAC compared to the IV formulation in adolescents presenting to the emergency department following toxic acetaminophen overdose.
Methods: A large US poison center's database was queried for all cases of acetaminophen overdoses in adolescents beginning in 1/1/2006 and ending on 12/31/2016. Inclusion criteria was: 1. adolescent age (defined as age 12-19) and 2. intentional ingestion. Patients were excluded for: 1. Polypharmacy 2. Missing baseline or follow up liver function tests 3. Hepatic injury at time of ED arrival (defined as AST or ALT >50 IU/L) 4. Unknown time of ingestion 5. Medication error/Incorrect administration of NAC 6. Pregnancy or 7. Transfer to a different facility. The primary outcome of this study was treatment failure, which was defined a priori as having occurred if the patient either 1. developed hepatotoxicity (defined as AST or ALT >100 IU/L) or 2. failed to complete original treatment regimen. Data collected included route of NAC administration, age, gender, initial acetaminophen concentration, and time to treatment.
Results: Over the study period, 235 adolescents were included for intentional acetaminophen overdose. Of these patients, 190 were excluded, leaving 45 patients for analysis. Of the 45 included patients, 26 (57.8%) received IV NAC while 19 (42.2%) received oral NAC. The mean age in the cohort was 15.2 years (SD: 1.74). The majority of patients were female (n = 37, 82.2%). The baseline acetaminophen level was 183.77 mcg/mL (SD: 104.52). Baseline AST and ALT levels were 20.39 IU/L (SD: 8.19) and 18.05 (SD: 7.90), respectively. Mean time to treatment was 6.87 hours (SD: 4.97). There were no significant differences in the demographics receiving IV and PO NAC at baseline. Of the 26 patients receiving IV NAC, only four (15.4%) had a treatment failure. Of these four failures, three (75.0%) failed for hepatotoxicity while one failed for failure to complete the treatment regimen (25.0%). Of the 19 patients receiving PO NAC, 12 (63.2%) had a treatment failure. Eight (66.7%) of the 12 failed for inability to complete the regimen, one (8.3%) failed for hepatotoxicity, and three (25.0%) failed for both failures to complete the regimen and hepatotoxicity. The difference in treatment failure between the groups was significant (15.4% vs. 63.2%; $p < 0.001$). Peak AST (IV: 56.12 IU/L, SD: 125.52; PO: 263.79 IU/L, SD: 969.69, $p = 0.2938$) and ALT (IV: 57.84 IU/L, SD: 125.52; PO: 332.74 IU/L, SD: 1,276.70, $p = 0.2896$) levels were similar between groups.
Conclusion: In our cohort of adolescents with intentional acetaminophen ingestion covered by a regional poison center, IV NAC was associated with fewer treatment failures than oral NAC. Health care practitioners should consider the incidence of treatment failure and liver injury when deciding between oral and IV NAC.

KEYWORDS Acetaminophen, N-acetylcysteine, pediatric toxicology

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120. Delayed Ototoxicity Following a Fatal Ingestion of Diethylene Glycol

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Background: Diethylene glycol (DEG) poisoning has been reported to cause various neurologic deficits, particularly cranial nerve seven palsies. We present a case of delayed ototoxicity following a fatal ingestion of DEG that resulted in delayed bilateral hearing loss.
Case Report: A 36-year-old woman presented to a community emergency department with chest pain following intentional ingestion of unknown amount of "Chafing Dish Fuel – Safe Heat" (diethylene glycol 100%). Initial vital signs were notable for a respiratory rate of 40, otherwise within normal limits. Five hours after ingestion, she developed a severe anion gap acidosis with a pH of 7.1, pCO2 of 8 mmHg, serum bicarbonate of 2.6 mmol/L, and an anion gap of 24. Her initial creatinine was 1.2. She was started on fomepizole, and 11 hours after ingestion she underwent intermittent hemodialysis. On day 3 the patient developed anuric renal failure with creatinine rise to 7.9 mg/dL. She also displayed evidence of hepatotoxicity with an AST of 990 units/L and ALT of 998 units/L. Her initial acetaminophen concentration was undetectable. She then developed pulmonary edema requiring non-invasive ventilatory support and two more dialysis sessions. On day 8 she complained of ringing in her ears bilaterally that then deteriorated to total hearing loss. Neurology was consulted for this new finding and an MRI brain without contrast was done which did not reveal any acute abnormality. On day 11 the patient's respiratory and mental status decompensated requiring intubation. Her severe encephalopathy persisted as she underwent continuous renal replacement therapy and intermittent blood transfusions for anemia of unclear etiology. She was declared brain dead on day 15 and she expired after support was withdrawn.
Discussion: It is generally understood that DEG's toxic properties are caused by its metabolites, namely 2-hydroxyethoxyacetic acid (HEAA) and possibly diglycolate (DGA). Ototoxicity is an uncommon manifestation of DEG toxicity, however, there are various case reports of cranial nerve deficits after DEG ingestion, including cranial nerve III, IV, and VI – X palsies. Interestingly, most of the reports of cranial nerve deficits appear to be bilateral, though the mechanism by which this occurs has not been well elucidated. We postulate that our patient's ototoxicity was likely secondary to bilateral vestibulocochlear nerve (cranial nerve VIII) palsies Nerve conduction studies have previously revealed a demyelinating sensorimotor neuropathy in the extremities, manifesting as limb weakness following DEG ingestion. A similar demyelinating process may also be the cause of cranial nerve palsies. The delayed timing of her ototoxicity is consistent with previous reports involving DEG toxicity which describe cranial nerve deficits, particularly cranial nerve seven, presenting around day 5-10 post-ingestion.
Conclusions: DEG and its metabolites HEAA and DGA are reported to cause severe toxicity in overdose with uncommon cranial nerve deficits in addition to severe acidosis and renal failure. Delayed bilateral hearing loss, possibly due to direct toxicity of the vestibulocochlear nerve, may occur in severe DEG poisonings.

KEYWORDS Diethylene Glycol, Ototoxicity, Hearing Loss

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121. Online Training vs In-Person Training for Opioid Overdose Prevention Training for Medical Students, a Randomized Controlled Trial

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Background: The growing opioid overdose epidemic has grappled the nation with the CDC now reporting that drug overdose deaths have become the most common cause of death for young people. Medical education has historically ignored substance use disorders, and though they generally require all medical students to learn basic life support, they have not taught how to respond to opioid overdoses. Further, medical education is moving towards modalities which utilize adult learning theory. One such modality are online modules. However, there are few studies comparing their outcomes with traditional lectures. Previously, the authors compared in-person and online training of medical students to respond to opioid overdoses using naloxone in a non-randomized controlled setting, which showed no meaningful differences in knowledge, attitudes, and preparedness outcomes for students. In this paper, the authors attempt to use a randomized controlled trial to compare the two educational modalities at a second urban medical school.
Objectives: The author's primary objective was to demonstrate non-inferiority of online compared to in-person training for knowledge. Our secondary objective were to show non-inferiority of online compared to in-person training attitudes, and preparedness.
Methods: Our study received IRB exemption as an education intervention. As a part of a transition to clinical clerkships curriculum used for second year medical students, second year medical students in an urban medical school were randomized into training sessions by the office of medical education without foreknowledge of the planned study. Students taking the online training were provided with a link to online modules with pre- and post-tests and video based lectures. Students randomized to the in-person training group took a pre-test just prior to receiving an oral lecture, and then immediately completed a post-test. Paired student's t-tests were used to compare measurements for each group in knowledge, attitudes, and preparedness, and Cohen's D was used to measure the effect size of the change. We calculated 99% confidence intervals for each measure and utilized a margin of non-inferiority of 5%.
Results: The in-person group demonstrated a statistically significant increase in knowledge, a non-statistically significant decrease in self-reported preparedness, and a small non-statistically significant increase in attitudes, see Table 1. The online group demonstrated a statistically significant increase in knowledge and self-reported preparedness, without a statistically significant change in attitudes, see Table 1. 99% CIs were [-0.20, 1.09] for knowledge, [6.51, 10.93] for preparedness, and [-2.32, 1.59] for attitudes, see Figure 1.
Conclusions: Online training for opioid overdose prevention training provided non-inferior outcomes for knowledge, preparedness, and attitudes. This study supports the use of online opioid overdose prevention training as a non-inferior alternative to in-person training.

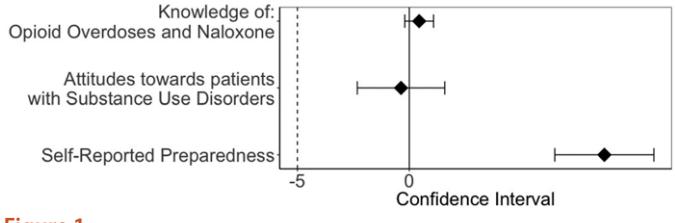

Figure 1.

Table 1.

Modality	Measure	Pre-Mean(SD)	Post-Mean(SD)	p	Cohen's d
In-Person	knowledge	4.86(1.59)	8.05(1.72)	<0.01	1.76
	preparedness	38.07(3.30)	37.49(1.97)	0.11	0.18
	attitudes	47.93(6.61)	48.33(7.79)	0.41	0.09
Online	knowledge	5.69(2.05)	8.86(1.90)	<0.01	1.56
	preparedness	31.49(7.09)	44.35(6.82)	<0.01	1.58
	attitudes	46.94(7.84)	47.16(7.90)	0.70	0.04

KEYWORDS Opioids, Overdose, Naloxone

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122. Antecedent Activities, Exposure Site and Demographics of Crotalid Envenomations Reported to One Southeastern United States Poison Center, 2014-2018

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Background: Crotalid envenomations are common, potentially dangerous exposures that occur in a significant part of the United States; there were 4071 exposures reported to the American Association of Poison Centers in 2017. Studies linking rattlesnake envenomations with human behaviors have been published, but the applicability of these associations in areas with predominantly copperheads is less clear as copperhead behavior is significantly different than that of rattlesnakes. Education and preventative efforts should be directed using high-quality data for efficient use of resources.
Methods: Retrospective review of human pit viper bites (rattlesnakes, copperheads, cottonmouths, unknown crotalids, and unknown snake bite) cases reported to one United States Poison Center from 1/1/2014-12/31/2018. All case records were reviewed, with free text notes taking precedence over coded fields. Case demographics, body part bitten, exposure site, time, and the presence of pre-determined activity and location categories were abstracted. Cases were excluded if it was not a pit viper envenomation, the patient was determined to be a drug seeker, the exposure site was in an out-of-state county not adjacent to the state or the poison center was not the primary center.
Results: Of 3384 cases reviewed, 606 were excluded, leaving 2778 cases. Copperheads were responsible for most cases (81.2%), with unknown crotalid being the second most common (15.3%); rattlesnake bites were rare (1.9%). Adults averaged 46.4 years old with 58.3% males, and pediatric cases (age <18 years) averaged 9.6 years old with 57.3% males; 18.8% of patients were aged 60 years or more. The anatomic location most often bitten in pediatric cases was the lower extremity (81.1%), whereas the upper extremity was most often bitten in adult males (50.6%). For cases where an activity was documented (n = 2382), the most frequent activities associated with envenomation were walking (36.7%), gardening (24.9%), and reaching where they couldn't see (19.8%). There were 225 cases where the patient reported being aware of the snake prior to being bitten with only 22 of those attempting to avoid the snake prior to the bite. Most bites occurred during the day, but a significant number occurred at night (32.9%). For cases with known exposure site (n = 2656), the most frequent location was own residence (77.5%) with public area (14.2%), other residence (4.0%) and workplace (3.6%) being less common. Urban counties reported a greater number of envenomations (81.5%) than rural counties. There were 382 (16.0%) of bites that occurred in close proximity to a living space, with 25 cases occurring indoors; none of those cases were pets and about half of those occurred in urban counties.
Conclusions: This is the one of the largest case series detailing antecedent activities and conditions for predominantly copperhead envenomations. These characteristics appear different than previously reported crotalid series from other geographical areas. Prevention efforts should focus on activities associated with getting bitten, safety around the home and individuals at the extremes of age as 38% of patients in this series were less than 18 or greater than 59 years old.

KEYWORDS Copperhead, Prevention, Pit Viper

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123. Selling Poison by the Bottle: Availability of Dangerous Substances Found on eBay®

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Background: During the 19th century, many new poisonous substances came onto the market to control plants and vermin, for use as surface cleaners, or as medicines. To prevent mishaps, poison bottles were often given distinctive colors, patterns or marked with certain raised shapes. Many of these bottles still hold their original contents and may be purchased online by collectors. This common practice places people at risk for serious poisonings following unintentional or intentional exposures. Our objective was to quantify the wide variety of dangerous poisons for sale to the general public on a popular online auction Web site.
Methods: Over an 8-month period, weekly listings on the online auction Web site eBay® were searched using the term "poison bottles". Products advertised as containing any of their original contents were identified. Product name, toxic ingredient(s), the amount of the product in the container, and relative toxicity rating were recorded using structured abstraction forms. Toxicity ratings were based on known median lethal dose (LD50) of each ingredient. Descriptive statistics (frequency tables, confidence intervals) were used to summarize the data.
Results: A total of 283 individual products were identified during the study period; 140 (49%) were liquids, 84 (30%) were in solid/tablet form, and 59 (21%) were powders. Bottles were full for 136 items (48%) and partially full for the remaining 147. At least 31 (11%) of the containers were described by the seller as cracked or poorly sealed. Overall, 93 (33%) contained 21 ingredients rated as extremely toxic; 132 products (47%) were rated as moderately toxic. Poisons for sale included heavy metals (arsenic, mercury), controlled substances (codeine, phenobarbital), pharmaceutical grade toxins (strychnine, pilocarpine), insecticides (nicotine), herbal extracts (oleander, hemlock) and beauty aids (belladonna, scopolamine).
Conclusions: While the products we identified were being sold for the nostalgic appeal of their containers, there is no guarantee that purchasers of these products would not attempt to discard the contents or use them in some way. Before antique bottles are purchased, the hazardous contents should be carefully removed and disposed of in a manner that is consistent with local, state, and federal legal guidelines.

KEYWORDS eBay®, poisonous substances, antique bottles

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124. Acute Fluoride Toxicokinetics Modelled Via Electrolyte Disturbance Velocity in a Single Patient

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Background: Hydrofluoric acid is a weak acid with a variety of uses in industrial and household settings. Due to its nature as a weak acid it can cross lipid membranes into tissue more easily than stronger acids. The fluoride anion binds divalent cations such as calcium and

magnesium, lowering their serum concentrations, and raises serum potassium through an unclear mechanism.
Case Report: We report a case of a 58-year-old man with accidental ingestion of a gulp of an initially unidentified liquid stored in a ginger ale container in an autobody shop. Shortly after ingestion, the patient began to experience nausea and hematemesis. During EMS transport patient developed lethargy, abdominal pain, and desire to use the bathroom. On arrival to a health care facility vitals were HR 100, BP 90/68, Temp 36.3, resp 20, SpO2 92%. Initial ECG QRS 84 msec, QTc 532 msec with no acute ischemic changes. Initial labs showed hypokalemia (3.1 mmol/L), Ca 8.8 mg/dL, low ionized calcium of 0.71 mmol/L, hypomagnesemia (0.9 mg/dL), pH 7.14 and pCO2 of 64mmHg. The patient had progressive obtundation and was intubated for airway protection. During RSI the patient was given a total of 216 mg succinyl choline in two divided doses. The patient experienced post intubation PEA arrest requiring chest compressions, then obtained ROSC with an initially wide QRS complex rhythm which narrowed minutes later. Labs repeated 106 minutes after initial showed worsened ionized hypocalcemia (0.41 mmol/L), worsened hypomagnesemia (0.6 mmol/L), pH 7.22, and pCO2 44mmHg, and 240 minutes after showed hyperkalemia (5.7 mmol/L), hypocalcemia (<4.0 mmol/L, below laboratory detection threshold). During course of hospitalization patient experienced recurrent torsades de pointes refractory to more than 25 defibrillations, attempted electrolyte repletion, and overdrive pacing with isoproterenol. Ionized calcium nadired at 0.41 mmol/L, magnesium at 0.6 mg/dL, and total calcium <4.0 mg/dL. Patient received a total of 4 grams of calcium gluconate, 21 grams of calcium chloride, and 18 grams of magnesium sulfate before dying approximately 21 hours post ingestion. Qualitative sampling of the ingested fluid confirmed fluoride content, and autopsy showed no major anatomic lesions.
Case Discussion: Accounting for minimally changed pH and administration of succinylcholine and exogenous calcium, we calculate the following electrolyte disturbance velocities:Serum Potassium increased by up to +0.65 mmol/L per hour.Total Calcium decreased by greater than or equal to -1.20 mg/dL per hour.Ionized Calcium decreased by greater than or equal to -0.17 mmol/L per hour.Magnesium decreased by -0.17 mg/dl per hour.Limitations to these calculations include the possibility of a non-linear slope were more data points available, administration of succinylcholine hours prior to repeat potassium potentially changing the value, and administration of exogenous calcium in between concentration measurements which may have attenuated the natural course of decreasing calcium.
Conclusion: Fluoride toxicity may be characterized by rising potassium and falling magnesium and ionized calcium concentrations. Conversely, this pattern of electrolyte disturbance velocity may aid in the empiric diagnosis of fluoride toxicity. Calculation of electrolyte disturbance velocity may aid in determining electrolyte repletion in future cases.

KEYWORDS Hydrofluoric acid, Torsades de pointes, Calcium

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125. Feasibility of Incorporating Medical Simulation into a Poison Center's Medical Toxicology Curriculum

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Background: Medical simulation is a useful educational tool which trains learners to methodically evaluate a patient, work as a team, and think through a patient's care in real time. Simulation provides a unique educational format in that learners must actively employ all six core competencies set forth by the Accreditation Council for Graduate Medical Education (ACGME): practice-based learning and improvement, patient care and procedural skills, systems-based practice, medical knowledge, interpersonal and communication skills, and professionalism. Poison center toxicology curricula often utilize conference-table discussions and lectures. This study aimed to address the feasibility of incorporating low overhead high quality medical simulation into one poison center's didactics. Learners at our poison center include medical toxicology fellows, resident physicians, medical students, pharmacists, pharmacy students, and specialist in poison information (SPI) trainees.
Methods: An internet search was performed to identify free peer-reviewed medical toxicology simulation cases. Several sources were discovered, notably emsimcases.com and acep.org/tox, from which cases were reviewed and added to the curriculum. The affiliated university hospital provided surplus educational, expired, and opened/unused materials. We attained a Laerdal ResusciAnne(TM) manikin torso, bag valve mask, endotracheal tubes with stylets, intubation blades, laryngeal mask airway, oxygen tubing, pacer pads, syringes, triple lumen central venous catheters, saline vials as surrogate medications, stethoscope, and other basic medical supplies. Two simulation sessions are performed each week in the poison center conference room. A medical toxicology fellow administrates the simulation which is structured as follows: 1. A 10-minute lecture is given on simulation mechanics, teamwork, communication, primary and secondary survey, and diagnosis, 2. The manikin is placed on the conference table and the team leader assigns roles to team members, 3. The case begins with hands-on assessment and management of the patient i.e., primary & secondary survey, resuscitative procedures, diagnostic test selection, therapy administration, disposition, and communication with consultants and admission team. There is verbal back-and-forth between the simulation administrator and team during case progression, 4. Stimulus materials, (electrocardiograms, radiographs, lab results) are displayed on a screen at the team's request, 5. Following the case conclusion, there is a structured debriefing regarding crucial decision points, facts of the case, and learners' self-assessments, 6. An anonymous quality assurance feedback survey is sent to all participants.
Results: Medical simulation has been a sustainable weekly aspect of our poison center's educational curriculum for over 12 sessions. Feedback from learners has been universally positive with comments such as, "the scenario itself was well acted and facilitated, giving it a mostly real feel despite being in a conference room," and "it was a high-yield tox case that unfolded in a realistic way, so was great practice for caring for a similar real patient in the future," and "it was great to have some applied learning. Debrief was thorough and very educational."
Conclusions: Incorporating high fidelity medical simulation into a poison center curriculum is an effective, low cost, and low resource method of educating.

KEYWORDS Medical Simulation, Toxicology Education, Poison Center Didactics

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126. "No lye in my beer, please." Partnering with Public Health After Noting an Increase in Beverage Contamination with Drink-Dispenser Cleaners at Commercial Food Establishments.

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Background: An unusual cluster of beer-line cleaner exposures in customers at various commercial food establishments prompted us to characterize our poison center's experience of beverage contamination with drink-dispenser cleaners.
Methods: Toxicall was searched from 2010 to 2019 for cases with either an exposure reason of "contamination / tampering" or an exposure location of "restaurant / food service." All cases that involved ingestion of a cleaning product that was dispensed from a beverage machine at a commercial establishment were included.
Results: Twenty-seven cases met criteria: 2 in 2011, 1 each in 2013 and 2014, 8 in 2017, 14 in 2018, and 1 in 2019. Eleven exposures were from tap beer, 10 of which occurred at either a bar or restaurant, while the eleventh occurred at a golf course. Seven exposures were from coffee, two at convenience stores and the other five in a single incident at one coffee shop. The other nine exposures were: iced tea (4), soda (2), soft-serve ice cream (1), a milkshake (1), and unknown (1). These nine exposures occurred at a restaurant (7), a convenience store (1) and an unspecified business (1). In 9 cases, a specific beer-dispenser cleaner with a pH ≥13.5 was identified. One case each involved a "beer keg cleaner," sodium carbonate, or a chlorinator (pH 10.4). In the remaining 15 cases, the unspecified cleaners were described as tasting like bleach, a cleaner, soap or perfume. All patients developed symptoms. Eight cases had only "a bad taste," 5 cases had only nausea and 4 cases had only oral irritation and/or sore throat. In the other 10 cases, symptoms included oropharyngeal burns, vomiting, diarrhea, excessive oral secretions, blisters or burns to the lips or mouth, esophageal or chest pain, abdominal pain, and dyspnea. Thirteen patients sought health care, 11 at an ED and 2 at a clinic. Nine were treated and released and 4 were hospitalized (length of stay 2-4 days). Six had an EGD and 1 was intubated. Three of the six EGDs found no evidence of corrosive injury. The fourth patient's EGD had grade 2 esophageal burns and grade 3A gastric burns. The fifth patient's EGD found severe burns with fibrinous exudates in the esophagus and upper stomach. The sixth patient's intubation was difficult because of significant caustic burns and edema down to the vocal cords. This patient's EGD revealed minimal esophageal damage and gastritis without necrosis.
Discussion: Five cases, beginning in late 2018, prompted our epidemiological investigation. The significant increase in cases starting in 2017 was reported to our state health department, along with our epidemiological information. The state health department, partnering with other state agencies, sent a reminder to commercial food establishments about the importance of rinsing beverage dispensers after cleaning, and the health consequences that can occur if they are not adequately rinsed.
Conclusion: While most of these exposures resulted in minor symptoms, serious injury has occurred. By partnering with public health, the poison center's findings led to an important reminder being sent to food establishments across the state.

KEYWORDS Beverage dispenser cleaner, Caustic ingestion, Pulbic health alert

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127. Evaluation of cutoff values in acute paracetamol overdose following the guideline of the United Kingdom.

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Background: In 2012, the United Kingdom guideline for acute paracetamol (AAP) overdose was revised, which recommends that the treatment threshold should be lowered to '100-treatment line' without risk stratification of hepatotoxicity. AAP serum level plays a key role but emergency departments (ED) in some developing countries do not have laboratory resources providing drug level in time. The primary aim of the study is to evaluate the cutoff AAP doses for N-acetylcysteine (NAC) antidote treatment in the indigent laboratory environment.
Methods: Data were collected retrospectively from two ED that more than 60,000 patients visit annually, between 2010 and 2017. Inclusion criteria were acute single AAP intoxication with dose ≥75 mg/kg, visited ED in 15 hours after overdose and over 14-year old. The trend of change in the frequency of toxic level with increasing dose was determined by $\chi 2$ test for trend. The correlation with risk factors of hepatotoxicity and toxic concentration was evaluated by multiple logistic regression and sensitivity, specificity, positive and negative predictive value of 100, 125, 150 and 175 mg/kg for the level above 100-line were calculated.
Results: A total of 297 patients over 14-year old visited ED with AAP overdose ≥75 mg/kg. Excluding 32 patients presented after 15 hours and 77 patients with missing dose or weight record, 196 patients were enrolled in the primary analysis. 40 patients (20.4 %) were male and 156 (79.6 %) female. The median age was 24 years old (14-83). 166 patients (84.7 %) were overdosed with extended-releasing or compound preparation. 56 subjects (28.6 %) showed toxic level above 100-line in the first test. There was positive trend in the frequency of toxic level with increasing dose ($\chi 2 = 7.63$, p-value =0.0057). In logistic regression, only time from intoxication to ED was useful for predicting toxic concentration (OR =0.911, 95 % CI 0.833-0.997, p-value =0.042). Sensitivity for toxic level of 100, 125, 150 and 175 mg/kg were 85.7 %, 76.8 %, 69.6 % and 60.7 % respectively.
Discussion: Only time from ingestion to ED were useful for predicting toxic concentration, the use of lowered threshold might offset the role of risk factors used in the past. There were 4 subjects showed line-crossing serum level through the 100-line. All of 4 subjects got NAC treatment, only 1 subject showed elevated alanine aminotransferase with the peak of 442 IU/L. None of them had any risk factor of line-crossing except 1 subject with late ED presentation (>8 hours). These cases suggest that lowering threshold could significantly reduce the risk of hepatotoxicity from delayed toxic concentration. Considering possible fatal outcome, it is essential to set the dose with the highest sensitivity as a cutoff value. For this reason, the dose of 100 mg/kg is judged to be the threshold for antidote therapy in this study.
Conclusion: For the patient of acute single AAP overdose, over 14-year old, visited ED in 15 hours, the dose of 100 mg/kg can be safely suggested as a toxic exposure for NAC antidote therapy following the UK guideline.

KEYWORDS Acetaminophen, Drug Overdose, Acetylcysteine

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128. Relationship Between Health Insurance Type and Choice of Non-poison Center Alternative Care

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Background: In a preliminary study, we identified that visiting emergency department (ED) and calling physician were substitute options if the poison control center (PCC) were not available. The objective of this study was to review multiple years of data to determine what callers to PCCs would do in a poison-related emergency if a PCC was not available, and whether or not health insurance is a factor that influences that decision.

Methods: This was a retrospective analysis of a PCCs customer satisfaction survey (CSS) responses over a 4-year period to determine the relationship between 2 survey questions. 1) "If the poison center was not available, what would you do first in solving poison-related emergencies?" and 2) "Does the person you called about have health insurance? If yes, which kind is it?" The alternative actions are: call 911, call/visit ED, call/visit urgent care, call/visit physician, search online, and other. The insurance types are separated into four categories: government (state/federal), commercial, self-pay, and other/unknown/refused. Data was obtained from surveys between 2015 to 2018 cases, and the data were cross-tabulated for analysis.
Results: A total of 3852 surveys were completed between 2015 to 2018. The majority of responder had commercial insurance (2500; 64.9%), followed by government (959; 24.9%). The top three most common alternative actions were call/visit physician (1030; 26.74%) followed by call/visit ED (963; 25%) and call 911 (788; 20.26%). For callers with government insurance, the most common alternative action response was call/visit ED (307; 32.01%); the most common response for callers with commercial insurance was call/visit physician (752; 30.08%); for self-pay the most common response was visit ED (57; 35.19%); and for other/unknown/refused callers, the most common response was visit ED (55; 23.81%). The trends in percentages for each category of alternative actions remained relatively the same between 2015 to 2018. Some alternative action had some notable differences. Between 2015 and 2016, call 911 increased from 16.87% to 24.3% and call/visit physician office decreased from 26.91% to 20.32% for those with government insurance. Those with commercial insurance saw a decrease in 911 call from 22.5% to 16.8% between 2016 to 2018 and an increase of call/visit ED from 18.49% to 23.27% between 2015 and 2016. For self-pay, call 911 increased from 12.9% to 22.9% from 2015 to 2016 and remained about the same since, and call/visit ED decreased from 48.4% to 24.5% from 2015 to 2017, but increased up to 38.3% in 2018. Lastly, for other/unknown/refused, call/visit ED saw an overall decrease from 28% to 18.8% from 2015 to 2018, call/visit physician had an increase from 11.5% to 26.1% from 2017 to 2018, and other had an increase from 18% to 30% from 2015 to 2016 and back down to 19.2% in 2017.
Conclusion: If the PCC is not available, most callers will seek care at a healthcare facility with the two most common action call/visit ED and call/visit physician depending on the insurance type. PCCs should target outreach efforts to insurance providers to enhance awareness and utilization of PCC services.

KEYWORDS Survey, poison center, insurance

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129. Extracorporeal Membrane Oxygenation (ECMO) for Poisonings Reported to U.S. Poison Centers: 2000-2018

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Background/Objective: The use of ECMO in critically ill patients is increasing worldwide, however there is little literature describing its use in poisoned patients, particularly in the U.S. This study sought to describe ECMO use for cases reported to the U.S. Poison Centers this century.
Methods: We identified all cases reported to National Poison Data System (NPDS) from 2000-June 2018 with ECMO coded as either a Recommended or Performed therapy. All cases were reviewed by two board-certified medical toxicologists and excluded if the case details were consistent with miscoding (e.g. ECMO performed for dermal exposure to benign substance with "no effect" as the outcome). Ingested substances were reviewed and grouped based on similar mechanisms/classes. Initial 3-digit zip codes were re-coded to the lowest corresponding 5-digit zip code and geospatially mapped using ArcGIS Online (esriTM) to identify geographic trends. Descriptive statistics were used to analyze data (Stata, version 15.1). Patients were dichotomized by age: "adults" were defined as age >12 years; "children" as age ≤12 years.
Results: We identified 517 unique cases; 67 were excluded as likely coding errors leaving 450 for analysis. Of these, 67 were coded as "Recommended only," 63 were coded as "Recommended and Performed," 299 were coded as "Performed only," and 21 were coded as "Recommended but not Performed." Median age was 24 years (IQR 15-39); 54% were male. Median number of ingested substances was three (IQR:2-4); 52% were single-substance ingestions. Additional clinical data are displayed in the Table. Since 2008, the incidence of patients receiving ECMO has increased (Figure 1), increasing by 9-100% per year (test for trend, z = 3.79, p
Conclusions: The use of ECMO to treat poisonings in the U.S. is increasing, driven by increased utilization of ECMO in patients >12 years of age. We observed no trends in survival over time, though survival has tended to increase over time in adults. Large regions of the U.S., primarily rural areas, have reported no cases of ECMO for poisoning this century. Further research should determine if this derives from lack of access to ECMO as a therapy, or if poisonings necessitating the use of ECMO are uncommon in rural areas.

Table Patients Dichotomized by Mortality.

	Survived	Died
Number of cases receiving ECMO	253 (69.9%)	109 (30.1%)
Age	25.8 +/- 17.8	28.5 +/- 19.6
Male gender (%)	131 (51.8%)	64 (58.7%)
Single ingestion cases (%)	132 (52.2%)	57 (52.3%)
Multiple ingestion cases (%)	121 (47.8%)	52 (47.7%)
Substances ingested, median (IQR, range))	3 (2-4, 2-16)	3 (2-4, 2-12)
Selected clinical effects (related)		
Asystole	18 (7.1%)	31 (28.4%)
Cardiac arrest	47 (18.6%)	58 (53.2%)
Conduction disturbance	51 (20.2%)	22 (20.2%)
Dysrhythmias (vtach/vfib)	27 (10.7%)	15 (13.8%)
Respiratory arrest	24 (9.4%)	38 (34.9%)
Selected therapies (performed)		
Antiarrhythmic	23 (9.1%)	17 (15.6%)
CPR	59 (23.3%)	44 (40.4%)
Pacemaker	18 (7.1%)	11 (10.1%)
Vasopressor	177 (70%)	89 (81.7%)
Single-ingestion case substances (total)*, #		
Hydrocarbon (28)	20	8
Calcium channel blocker (18)	13	5
Unknown (16)	10	6
Antiarrhythmic (14)	10	4
Antidepressant (11)	6	5
Mitochondrial poisons (11)	7	4
CNS Depressants (10)	6	4
Opioid (10)	10	0
Other chemical (9)	5	4
Corrosive (7)	6	1
Antihistamine (6)	5	1
Stimulant (6)	5	1
APAP (4)	2	2
Other OTC (4)	2	2
Other drugs of abuse (4)	4	0
Antimicrobial (3)	2	1
Beta-adrenergic blocker (3)	3	0
NSAID (3)	3	0
Psychotropic (3)	3	0

*2 cases each: insects, plants, salicylates, unknown non drug, anticonvulsants, opioid with APAP.
#1 case each: Antineoplastic, asphyxiant, diabetic agent, heavy metal, toxic alcohol, foreign body, paralytic.

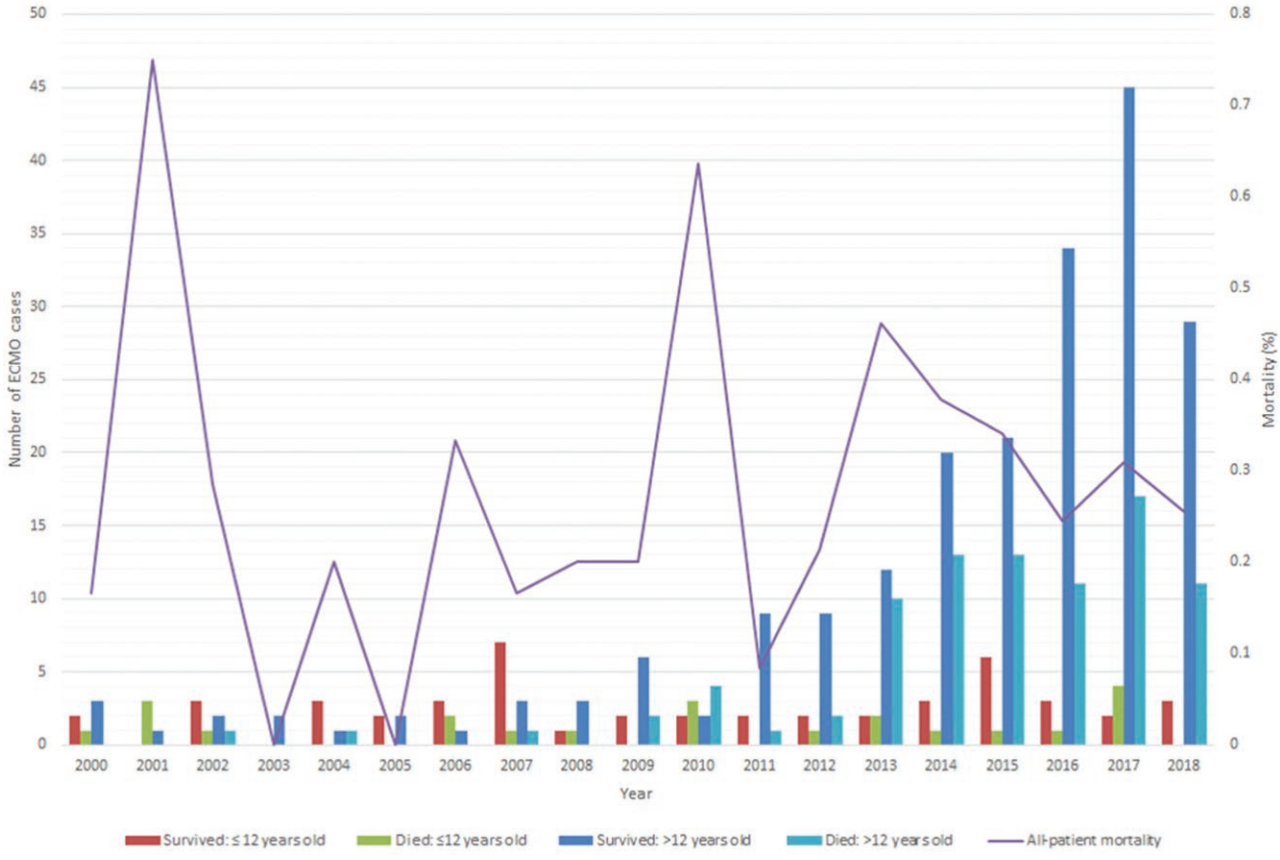

Figure 1 Trends in ECMO utilization and mortality: 2000-mid 2018.

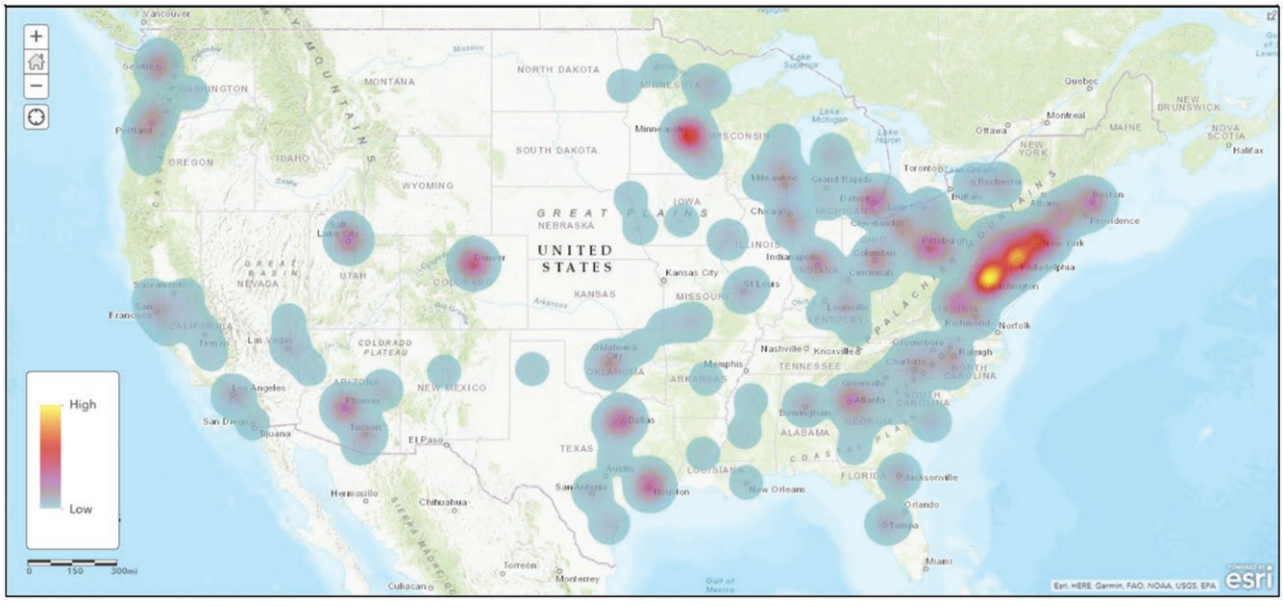

Figure 2 ECMO cases by site of initial Poison Center call: 2000 - 2018.

KEYWORDS ECMO, Poison Centers, Critical Care

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130. Adult Exposure to Single-Use Liquid Laundry Detergent Packs in the U.S. (2013-2018)

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Background: Following the introduction of Liquid Laundry Detergent Packs (LLDPs) in the U.S. (early 2012), a prospective observational study was initiated among 12 regional poison centers serving 24% of the total population. Several case series have analyzed LLDP exposure trends in children, however data on adult exposures is lacking. This is an analysis of adult LLDP exposures using data from the ongoing prospective study.
Method: LLDP exposures involving an adult patient (age >19 years) reported from 2013-2018 were extracted from the study database. Patients of known age (years) were classified as a 'Young Adult' (20-64 years) or 'Elderly Adult' (65+ years). The case narrative was reviewed to verify coding accuracy and to extract information relevant to the patient's exposure history and clinical course. Patients with a history of cognitive impairment (e.g. dementia, developmental delay, etc.) were designated as 'mentally impaired'. Multi-route exposures were assigned a 'primary route' based on the patient's clinical presentation and exposure history. Trend and comparative analyses were performed on absolute case counts, relative proportions, and exposure reporting rates, which were normalized using Nielsen consumption data as a surrogate for household availability and expressed as 'exposures per million units purchased' (EMU). To more accurately reflect reporting rates for the entire US population, rates were adjusted by a factor of 4.
Results: During the period of analysis (2013-2018), 19,499 LLDP exposures were reported to the PC study sites and 4.9% (N = 955) involved an adult patient. Among patients with known age (N = 825), Young Adults (aged 20-64 years) accounted for 81.0% of exposures and most (66.0%) were under age 40. Exposure reporting rates were similar from 2013-2016 (overall mean 0.10 EMU), however increased 40% in 2017 (0.14 EMU) and 2018 (0.20 EMU). This increase was primarily driven by the Young Adult cohort (see Figure 1). The primary route of exposure was ingestion (48.7%), followed by ocular (29.4%), and dermal (21.3%); however, the proportion of ingestion cases was higher for Elderly Adults (70.6%) vs Young Adults (44.6%). Most exposures (86.7%) were unintentional in nature and mental impairment was a contributing factor in 33.7% of Elderly cases vs 4.7% of Young Adults. Intentional exposures were more common among Young Adults (14.7% vs 3.1%) and roughly half involved self-harm/suicide. Intentional cases that did not involve self-harm often involved deliberate "playing" or "tasting" of the product. Among patients who were followed to a known medical outcome, the proportion who experienced a serious outcome (moderate, major, death) was higher for Elderly Adults (23.1%, n = 27) vs Young Adults (16.6%, n = 62) and this difference was most pronounced for ingestions (17.1% vs 7.6%). Two death cases were reported, both of which involved an Elderly Adult with cognitive impairment.
Conclusion: Although adults represent a small proportion of LLDP exposures overall, they provide distinct challenges. Current exposure reduction efforts may impede access by children, mentally impaired adults and/or the elderly. However, in this analysis, the majority of exposed adults were not seniors, but rather, younger adults. Therefore, further instruction regarding proper use and handling may be needed.

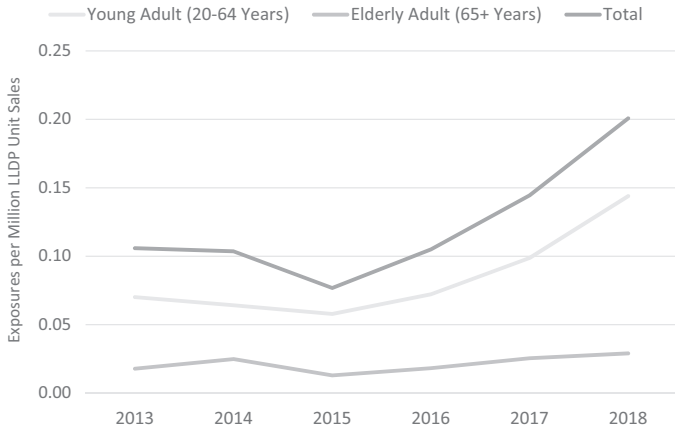

Figure 1 LLDP Exposure Reporting Rate by Adult Age Cohort.

KEYWORDS Liquid Laundry Detergent Packs, Adult Exposures, Prevention

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131. Massive iron overdose in an adolescent: consider additions to standard therapy

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Background: Iron is prescribed as a supplement during pregnancy and to treat iron-deficient states. Overdose deaths are rare: between 2013–2018,
Case report: A 70 kg, 16-year-old female presented 9.5h after ingestion of 100 tablets of 325mg ferrous sulfate (approximately 93mg/kg elemental iron). She presented pale and lethargic. Vital signs were: HR64, BP125/102, Sp02 96%, and RR26. Given history and exam, DFO was empirically initiated at 10mg/kg/hr. Initial iron concentration was 4,206mcg/dL, and metabolic acidosis (bicarbonate 14mmol/L) was noted. Whole bowel irrigation was initiated when abdominal radiography revealed persistent intestinal pills; DFO was increased to 20mg/kg/hr, and the patient was transferred to a tertiary referral center. The regional Poison Center recommended exchange transfusion to the receiving center; this was never performed. Subsequent iron concentrations remained approximately 4,000mcg/dL until 18h post-presentation, when the concentration plummeted to 347mcg/ml (Figure 1). The patient was intubated for decreasing mentation; 24h post-ingestion hypoglycemia developed, as did vasodilatory shock requiring multiple vasopressors and inotropes. Thrombocytopenia, anemia, and coagulopathy ensued. DFO was stopped 32 hours post-ingestion; serum iron was 125mcg/dL. N-acetylcysteine was initiated, as was continuous renal replacement therapy. Due to hepatic failure, the patient was transferred to a transplant center on hospital day (HD) 3 and listed for liver transplantation. Transaminases peaked from HD3-4 (ALT 4585U/L, AST 5136U/L). Coagulopathy peaked on HD3, with INR of 8.01. Hepatic synthetic function improved on HD8, she was removed from the transplant list, and was extubated on HD9. On HD11 a bowel perforation forced emergent resection, complicated by delayed fungal and bacterial peritonitis and recrudescent hepatic failure. The patient died on HD50 from sepsis and multisystem organ failure.
Case discussion: Deferoxamine binds about 8mg elemental iron per 100mg of DFO, based on 1:1 molar binding.[8] Intravenous DFO is associated with rate-related hypotension.[9] Despite adverse events with DFO, animal models demonstrate reduced mortality in iron

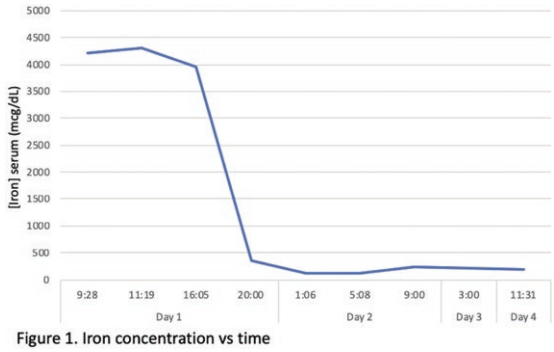

Figure 1. Iron concentration vs time

Figure 1 Iron concentration vs time.

overdose.[10,11] We recommended increasing DFO to 30mg/kg/hr given prior case reporting of similar dosing.[12] Nonetheless, even high-dose DFO was unlikely to adequately chelate this patient's massive iron burden. Given DFO's limitations, other options were pursued. Whole bowel irrigation was started to limit absorption, and exchange transfusion was recommended. Although seldom reported in the literature, reports of human exchange transfusion for iron toxicity exist; animal evidence suggests that exchange transfusion eliminates significantly more iron than DFO alone.[6,13]

Conclusion: All therapies considered in this severely iron-poisoned patient were warranted. High-dose DFO failed to adequately chelate the patient; exchange transfusion, though recommended, was not performed. Classically described profound iron toxicity followed, ending with fulminant hepatotoxicity, corrosive gastrointestinal injury, and sepsis. Active PC management remains vital to optimize care of the critically ill patient with massive iron exposure.

KEYWORDS Iron, Deferoxamine, Exchange

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132. Prolonged serotonergic toxicity following multiple serotonergic pharmaceutical ingestion

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Background: Serotonergic toxicity is described following ingestion of medications that increase synaptic serotonin concentration.[1] Signs include neuromuscular excitability, dysautonomia, and altered mentation.[1] Cases of serotonin toxicity frequently involve multiple pro-serotonergic medications.[2, 3] While most cases of serotonergic toxicity resolve by 24 hours, cases of prolonged toxicity have been reported.[4, 5] Here we report a case of a 59 year old male who ingested multiple serotonergic medications and had evidence of serotonin toxicity for 15 days.

Case report: A 59 year old male presented to the emergency department (ED) following an overdose on an unknown amount of home medications. Medications and maximum possible doses based on pharmacy count of remaining pills are listed in Table 1. On arrival to the ED, he was comatose and thus intubated. Activated charcoal was administered. On hospital day (HD) 2, the toxicology service was consulted and noted sustained ankle clonus. He remained intubated and sedated until HD15 on various sedatives, including propofol, dexmedetomidine, and midazolam infusions. Marked lower extremity rigidity, clonus, hyperreflexia, and hyperthermia persisted. Due to persistent serotonin syndrome, cyproheptadine was initiated at 4mg every six hours on HD8 and was discontinued on HD13. On HD15 he was extubated, and by HD16 clonus resolved. He was transferred to inpatient psychiatry on HD 24.

Table 1 List of medication bottles brought in with patient and maximum available tablets.

Medication	Count	Dose	Total
Colchicine	101	0.6 mg	60.6 mg
Amitriptyline	49	10 mg	490 mg
Quetiapine	90	50 mg	4500 mg
Trazodone	60	50 mg	3000 mg
Fluoxetine	23	40 mg	920 mg
Aripiprazole	7	5 mg	35 mg
Alprazolam	7	1 mg	7 mg
Vilazodone	23	20 mg	460 mg
Ibuprofen	33	200 mg	6600 mg
Naproxen	14	500 mg	7000 mg
Allopurinol	3	300 mg	900 mg
Pravastatin	61	80 mg	4880 mg

Discussion: This case highlights prolonged serotonergic toxicity secondary to multiple serotonergic medications; several were CYP450 inhibitors or competing substrates. Fluoxetine, an SSRI with a half life of 1-3 days after acute use and 4-6 days in chronic use, played a major role in the duration of toxicity.[6] Further prolonging toxicity, fluoxetine is metabolized primarily by CYP2D6 to norfluoxetine, an active and long-lived metabolite.6 Fluoxetine potently inhibits CYP2D6 and thus its own metabolism.[7] Coingestion of aripiprazole, vilazodone and amitriptyline supplied competing CYP2D6 substrates. The unique features of this exposure led to prolonged serotonergic toxicity. Previous literature reports serotonergic toxicity of up to 12 days following fluoxetine ingestion.[5] While uncommon, combined exposure to irreversible monoamine oxidase inhibitors or delayed release preparations may prolong toxicity.[5] Our case suggests another potential risk factor: active or long lived metabolites in the presence of cytochrome inhibitors or competing enzyme substrates. Despite the prolonged course in our patient, treatment was similar to other cases of serotonergic toxicity: sedation with GABA-A potentiating agents and avoidance of pro-serotonergic agents, in addition to adjunctive ICU sedation, hemodynamic and ventilatory support, and cyproheptadine.

Conclusion: Polypharmacy is common, and increasingly so. Providers caring for the acutely intoxicated patient must be alert to a pharmacological milieu replete with competing substrates and enzymatic inhibitors may predict extended courses of serotonin toxicity that might otherwise be unanticipated.

KEYWORDS Serotonin, Toxicity, Prolonged

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133. Prazosin Exposures reported to US Poison Centers

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Introduction: Prazosin is an alpha-1 adrenergic antagonist approved for the treatment of hypertension in 1976. While its use as an antihypertensive agent has declined significantly, it is currently being used off-label for the treatment of post-traumatic stress disorder. The purpose of this study was to describe trends in exposures to prazosin reported to US poison centers.

Methods: This is a retrospective review of human exposures reported to the American Association of Poison Control Center's National Poison Data System (NPDS) between 1 January 2006 and 31 December 2016. Exposures involving prazosin were included in the analysis. The IRB determined this project did not meet the definition of Human Subjects Research. Frequencies and cross tabulations were used to describe the

data. Analysis was conducted on all exposures involving prazosin as well as only those cases that involved prazosin alone.
Results: A total of 4565 exposures involving prazosin were reported to NPDS between 1/1/2016 and 12/31/2016, 1312 involved prazosin only. Prazosin exposures increased steadily over the 11 year period from 99 cases documented in 2006 to 905 cases in 2016, a 918% increase when adjusting for total human exposures. A similar increase was seen with prazosin only exposures from 33 in 2006 to 245 in 2016. The majority of exposures involved females (2879; 63.1%) and adults >20 years (3431; 75.2%). The mean age was 32 years with a range of 7 months to 93 years. Less than 10% (368) of exposures involved children less than 6 years of age. The reason for exposure was suspected suicide (3042;66.6%) followed by therapeutic error (627;13.7%), unintentional general (455;10.0%), intentional other/unknown (264; 5.8%), adverse reaction (119; 2.6%), unintentional other/unknown (7; 0.2%), other (3; 0.1%), unknown 48 (1.1%). When more than one substance was involved, the number of substances ranged from 2 to 36 and a mean of 3.24 substances. Reason for exposure for prazosin only was suicide (617;47.0%) followed by unintentional general (274;20.9%), therapeutic error (264;20.2%), intentional other/unknown (93;7.1%), adverse reaction (51;3.9%), unknown (13;1.0%). The majority of prazosin only exposures were already in or referred to a health care facility (HCF) (897; 68.4%), 380 (29.0%) were managed onsite, 25 (1.9%) other and 10 (0.8%) unknown. Level of care for prazosin only was as follows: ED only (383;29.2%); admitted medical (171; 13.0%), admitted psychiatry (205;15.6%); lost to follow-up (100;7.6%) and refused referral (38;2.9%). The most common clinical effects noted with prazosin alone were tachycardia (203; 15.5%); drowsiness (200; 15.2%), hypotension (157;12.0%). The outcome when known was no effect (386;29.4%), minor (270;20.6%), moderate (211;16.1%) and major (10; 0.8%). There were no deaths in cases involving prazosin only.
Conclusion: Prazosin exposures reported to NPDS have increased over the last 10 years. A disproportionate share of the cases involved adults and were suspected suicides. Although the majority of cases involving only prazosin were treated in a HCF facility, most had no or minor effect. Clinicians should be aware of adverse and unintended consequences of increasing availability of medications in the home.

KEYWORDS Prazosin, NPDS, poisoning

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134. A fatal case of massive acetaminophen overdose despite timely initiation of n-acetylcysteine

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Background: N-acetylcysteine (NAC) is a widely available, cost-effective antidote for acetaminophen (APAP) overdose that ameliorates hepatotoxicity when initiated early in the course of poisoning. We report a case of an intentional massive APAP overdose resulting in fulminant hepatic failure and ultimately death, despite timely initiation of NAC.
Case report: A 32-year-old female with history of anxiety and depression was brought to the emergency department (ED) by her family after she admitted to an ingestion of 187.5 g of APAP. The patient was asymptomatic at the time of initial evaluation in the ED. Her transaminases and renal function were normal, and a 5-hour serum APAP level was 301.9 mcg/mL. Time of ingestion was determined by patient report and corroborated by the patient's family, as well as a time-stamped pharmacy receipt for the purchase of the APAP. The patient was treated with 50 g of activated charcoal and started on NAC within 5 hours of ingestion, per Poison Control Center (PCC) consultation. She received a bolus of 150 mg/kg over 1 hour, followed by 50 mg/kg over 4 hours, and 6.25 mg/kg/hr thereafter. Transfer was arranged to the nearest tertiary care center. During transport, the patient was noted to become increasingly lethargic, and she was intubated upon arrival to the ED at the tertiary care center, approximately 10 hours after ingestion. At that time, APAP level was 777 mcg/mL and lactate 9.5 mmol/L. PCC was updated and recommended hemodialysis, however the on-call nephrologist declined. NAC infusion was continued, and approximately 24 hours after ingestion, her transaminases began to rise. APAP levels remained significantly elevated at this time (386 mcg/mL). Her mental status improved, and she was extubated on hospital day 2. The following morning, the patient was noted to have signs of liver failure as evidenced by coagulopathy (INR 2.2) and new encephalopathy. Transfer to a transplant center was arranged. Upon arrival to the transplant center (approximately 48 hours post-ingestion), the patient became increasingly acidotic, with an arterial pH 7.16 and lactate 10.2 mmol/L. Her transaminases were both >1,000 U/L. The NAC infusion rate was increased, first to 15 mg/kg/hr, then to 30 mg/kg/hr after initiation of hemodialysis for persistent acidemia. She was listed by the transplant team as Status 1A. On the evening of hospital day 3 and throughout hospital day 4, the patient became increasingly ill, as evidenced by hemodynamic instability (hypotension, unstable supraventricular dysrhythmias, asystolic arrest), persistent acidosis (arterial pH 6.75, lactate >30 mmol/L), worsening coagulopathy (INR 7.0), and severe hepatic encephalopathy requiring re-intubation. Following the patient's second asystolic cardiac arrest, her parents changed her code status to Do Not Resuscitate. She was declared dead on the morning of hospital day 5, approximately 93 hours after ingestion.
Discussion: Severe acetaminophen-induced hepatotoxicity necessitating transplant is uncommon in the age of n-acetylcysteine, and death following acetaminophen overdose even rarer. Massive acetaminophen overdoses represent a distinct challenge in toxicology and require a multidisciplinary approach to management. Fulminant hepatic failure and death may result despite timely aggressive medical therapy.

KEYWORDS Acetaminophen, n-acetylcysteine, fatal

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135. Adverse effects of ingested buprenorphine reported to National Poison Database System (NPDS) 2010-2019

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Background: Buprenorphine is increasingly recommended as a primary therapy for Medication Assisted Therapy (MAT) given reported favorable pharmacologic and clinical features as compared to other treatment options such as methadone. One of these benefits includes a reported "ceiling effect" on respiratory depression. However, there is a paucity of published literature describing risk of respiratory depression or other potential adverse effects of MAT approved buprenorphine products in large population sampling. We sought to evaluate the characteristics of single substance ingestions of MAT approved buprenorphine products reported to the National Poison Database System (NPDS).
Methods: The NPDS was queried for all single-agent buprenorphine exposures reported between Jan 1,2010 and Feb 28,2019. Only cases coded as ingested were selected. Data was analyzed using IBM SPSS Statistics for Mac, Version 25.0 (Armonk, NY).
Results: A total of 16,956 cases were identified. Of these, 15551 were reported to be ingestions. 10,264 were followed to known outcome.

Table 1 describes characteristics of these cases. A 293% increase in the number of buprenorphine ingestions was observed over the study interval. The majority of exposures were unintentional in nature (62.2%). Despite a reported "ceiling effect" on respiratory depression, 10% of reported single-substance ingestions experienced respiratory depression and 1.6% of cases were intubated. Fourteen deaths were observed with 10 experiencing respiratory arrest or respiratory depression and intubation.

Conclusion: Exposures to MAT approved buprenorphine products by ingestion are increasing and in almost a third of cases are associated with moderate/major/fatal outcomes. Respiratory depression or arrest does appear to occur and is associated with a majority of deaths. Further studies are warranted to better characterize the risk of respiratory depression with MAT approved buprenorphine products.

Number of cases	15551
% increase over study period	293%
Age (years) [SD]	19.64 (16.2)
% female (n)	47% (7344)
Reason	
Unintentional	
General	8251 (53.1%)
Therapeutic Error	1409 (9.1%)
Intentional	
Abuse	1613 (10.4%)
Misuse	1154 (7.4%)
Suspected Suicide	670(4.3%)
Clinical effect	
drowsiness/lethargy n (%)	4649 (45%)
vomiting n (%)	1911 (19%)
respiratory depression n (%)	1065 (10%)
Therapies performed	
IV fluid n (%)	2369 (23%)
Naloxone n (%)	2302 (22%)
Oxygen n (%)	908 (9%)
Intubation n (%)	165 (1.6%)
Moderate/Major Outcomes (n)	2884/380
Deaths (n)	14
cardiac arrest	9
respiratory arrest	6
respiratory depression	4
Intubation	10
CPR	6
Naloxone	6

KEYWORDS Buprenorphine, Medication Assisted Therapy, respiratory depression

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136. Double Whammy: A Fatal Case of Sodium Channel and Beta Blocker Overdose Unresponsive to VA-ECMO

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Background: Flecainide is a potent class 1C antidysrhythmic that interferes with sodium channel cardiac conduction. In overdose, overwhelming sodium channel (NaCh) blockade results in QRS widening, ventricular dysrhythmias, and cardiogenic shock. Coingestion of other cardiodepressants such as beta or calcium channel blockers, can markedly worsen outcomes. Pharmacological management of these complicated cardiotoxic overdoses is often inadequate and may warrant mechanical hemodynamic support.

Case Report: A 32 year old male with history of hypertension, atrial fibrillation, and depression, presented to the ED after ingestion of 30 tablets of flecainide 100 mg, an unknown amount of metoprolol succinate 100mg, and ethanol in a suicide attempt. He was alert with BP 127/73, HR 102, and presenting ECG showed sinus rhythm with heart rate (HR) 93, QRS 142ms, R in aVR 7 mm, and QTC 517 ms. Patient received bolus sodium bicarbonate (NaHCO3) and NaHCO3 infusion was started at 150 mg/hr. A repeat ECG two hours later demonstrated HR 103 and QRS 160 ms, so additional NaHCO3 was administered and the infusion was increased. In addition, he was given calcium gluconate, potassium and magnesium. Patient became hypotensive and norepinephrine infusion was started with further escalation of NaHCO3 therapy. Eight hours after arrival to ED, ECG demonstrated worsening QRS widening to 180 ms with HR remaining in 80's. Shortly thereafter, the patient was found unresponsive in cardiac arrest with ventricular fibrillation followed by pulseless electrical activity (PEA). ACLS was started and CPR continued uninterrupted until initiation of veno-arterial extracorporeal membrane oxygenation (VA-ECMO), approximately 12 hours after arrival. During the arrest period, the patient was also given more NaHCO3 boluses, glucagon, lipid emulsion, and a transvenous pacer was placed. Hypertonic saline was recommended. After initiation of VA-ECMO, high-dose insulin euglycemic therapy (HIET) was started. Despite continuous mechanical support, patient remained in refractory shock on multiple vasopressors, and died 36 hrs into his hospital course.

Case Discussion: Aggressive administration of NaHCO3 remains the mainstay of initial management of potent NaCh blockade. Hypertonic saline has been suggested as adjunctive treatment once NaHCO3 therapy is maximized. Antidysrhythmics are contraindicated, as they can worsen the NaCh blockade. Delayed beta-blocking effects of the metoprolol succinate may have further complicated the management of cardiogenic shock in this case. HIET and lipid emulsion infusion should be considered for refractory cardiogenic shock from both flecainide and metoprolol. Even optimal medical management may have limited efficacy in refractory cases and swift escalation of care may be required if resuscitation is to be successful. VA ECMO has been used successfully in previous reports, however it is prudent that support be initiated as early as possible in patients who are at risk of clinical deterioration from refractory ventricular dysrhythmia and cardiogenic shock.

Conclusion: Coingestion of multiple cardiodepressant medications, as demonstrated here with NaCh and beta-blockers, compounds their morbidity and mortality. Pharmacological management is often inadequate and should warrant early consultation for cardiovascular mechanical support.

KEYWORDS Flecainide, ECMO, overdose

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137. Poison Center Workload: Complexity and Quality Surpassing Simplicity and Quantity

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Background: According to the latest published National Poison Data System annual report, exposures to US Regional Poison Centers (RPCs) have decreased by 15% over the previous 10 years. Historically, exposure volume alone has been the key RPC workload metric, and a continued downward trend could imply that RPCs are becoming less relevant or effective. Data from a large US RPC was reviewed to explore changes in the type and complexity of exposures managed.

Methods: RPC data were queried from 2009 and 2018 for total number of exposures, exposures originating from a Healthcare Facility (HCF),

Table 1.

	2009	2018	% change
Total exposures	84,628	71,951	-15%
Exposures originating from home/general public	66,971	47,784	-29%
Exposures originating from HCF	17,657	24,167	37%
Moderate outcome	4,161	7,277	75%
Major outcome	294	824	183%
Death	50	97	94%
Follow-up contacts	61,477	88,510	43%
Total contacts (initial exposures + follow-up)	146,105	160,461	10%

Table 2.

	2009	2018	% change
Top 5 substance categories with largest decrease			
Topical preparations	4,156	2,198	-47%
Cough and cold preparations	2,846	1,778	-37%
Cosmetics/Personal care products	8,054	5,132	-36%
Foreign bodies/toys/ Miscellaneous	4,305	3,008	-30%
Vitamins	2,655	2,124	-20%
Top 5 substance categories with largest increase			
Stimulants and street drugs	1,622	2,809	73%
Anticonvulsants	1,620	2,455	52%
Antidepressants	3,686	5,253	43%
Antihistamines	3,089	3,672	19%
Cardiovascular drugs	3,025	3,530	17%

all follow-up contacts, outcome, and substances involved in exposure. A percent change over time was calculated for each.
Results: In the 10 years between 2009 and 2018, total exposure volume decreased by 15%. However, the complexity of the exposures managed increased considerably—there were more exposures originating from HCF, more serious outcomes, and more overall contacts in 2018 than 2009. See Table 1. All of the exposures lost were those originating from the general public calling from home and many of those were simple nontoxic exposures. See Table 2. Additionally, the number of exposures involving 3 or more substances increased 32% from 2009 to 2018.
Conclusion: Despite a decrease in total exposure volume of 15% over the past 10 years, total contacts on RPC exposures—one measure of workload—increased 10%. This RPC is managing more HCF exposures, more exposures involving multiple substances, exposures with more serious outcomes, and is overall performing far more follow-up on cases managed. This increasing complexity of cases that RPCs manage demonstrates their importance in healthcare and contributions to public health. When evaluating RPC impact, it is important to consider more than just number of exposures.

KEYWORDS Poison Center, Epidemiology, Public Health

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138. Pediatric Fatality Risks: An Examination of the National Poison Data System Annual Reports, 2009-2017.

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Background: Drug and poisoning-related pediatric deaths in the United States are a serious public health challenge. Despite significant prevention efforts, greater than 70,000 children are seen in emergency departments, annually, for poisoning exposures. Poison control centers (PCCs) provide vital information, exposure, and prevention services; and handle over 2 million cases annually across the United States (US). The purpose of this study was to conduct a comparative review of the pediatric subset of nonpharmaceutical and pharmaceutical poison exposures and resulting fatalities as reported by the US PCCs to the National Poison Data System (NPDS).
Methods: The research team conducted a retrospective review of nine annual NPDS reports (2009-2017), with a focus on exposures in the population, aged 19 years and under. The data represent calls to 55 regional members of the American Association of Poison Control Centers (AAPCC). The subset of poison exposure-related fatalities was organized into three groups: children age
Results: During the nine-year period of annual NPDS reports, every year the proportion of PCC calls for potential poison exposures for children dropped dramatically (88%) after the age of 6 years. The most common route of non-pharmaceutical exposure-related deaths for children aged
Conclusions: National trends in fatalities in age groups 19 years and under demonstrate a difference in exposures and risk, across various age groups. The analysis underscores the need to expand age-related prevention measures. The results present an opportunity for inter-professional collaborative practice to establish sustainable public health education efforts, with targeted poisoning and suicide prevention strategies that address fatality risk and relevance for age-specific categories.

KEYWORDS Pediatric fatalities, Poisonings, Public Health

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139. Somatostatin for Persistent Hypoglycemia following High Dose Insulin Therapy for Beta Blocker Overdose

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Christiana Care

Background: Beta-blocker overdose is a frequently encountered toxicologic emergency both in the emergency department and in the medical Intensive Care Unit (ICU). It is associated with significant shock and often presents with profound hypoglycemia as well as reduced cardiac output. A mainstay of therapy is the use of High Dose Insulin (HDI) therapy. While this treatment is often effective in reversing the negative inotropy and shock associated with beta-blocker overdose, it can precipitate several metabolic derangements including hypoglycemia and hypokalemia.
Case Report: In this case report, we discuss the case of an individual who intentionally ingested large doses of propranolol and quetiapine with subsequent ICU admission and treatment with HDI. This individual, a 37-year-old male, presented to the emergency department in cardiogenic shock. He was quickly transferred to the ICU and started on HDI, with hourly blood glucose checks, supplemental potassium, and a simultaneous infusion of dextrose, at first 10% (D10) and later 20% (D20). The patient was on HDI for a total of 24 hours, with rapid improvement in blood pressure and heart rate. However, following the cessation of HDI therapy the patient continued to require supplemental dextrose in the form of D20 administered via a central line for approximately 36 hours. His persistent carbohydrate requirement prompted further investigation, the patient was found to have a C-peptide level of 11.3 ng/mL, well above the upper reference limit of

4.4 ng/mL, 30 hours after HDI therapy had been stopped. We proposed that his recalcitrant hypoglycemia was due to endogenous overproduction of insulin, and administered a single dose of somatostatin (60 micrograms) subcutaneously. This prompted rapid resolution of hypoglycemia and cessation of the supplemental dextrose requirement.

Case Discussion: We discuss the previously described sequelae of HDI, and reported prolonged, recalcitrant hypoglycemia requiring large quantities of dextrose replacement persisting well beyond 24 hours after the cessation of HDI, a potential sequela that has not previously been described. We discuss the potential mechanism of this persistent supplemental dextrose requirement; namely that it was likely due to excessive endogenous insulin production. We also discuss the novel and effective use of single dose subcutaneous somatostatin for the reversal of post-HDI hypoglycemia that resulted from an apparent over-production of endogenous insulin following cessation of HDI.

Conclusion: High-dose insulin therapy can be an effective way to treat the shock associated with beta-blocker overdose. The potential for hypoglycemia following the cessation of HDI is a very real and dangerous consideration for all providers embarking on implementation of HDI therapy. Single-dose somatostatin may offer clinicians a means of effectively treating this prolonged hypoglycemia.

Time (h)	Dextrose (g/h)	Insulin (U/h)	Serum Glucose (mg/dL)	Somatostatin
1	15	30	148	
2	20	30	124	
3	20	30	86	
4	20	45	212	
5	30	45	138	
6	30	45	178	
7	30	45	170	
8	30	45	195	
9	30	60	196	
10	30	80	190	
11	30	80	192	
12	30	80	206	
13	30	80	181	
14	30	80	189	
15	30	80	179	
16	30	80	148	
17	30	80	132	
18	30	70	120	
19	30	60	126	
20	30	60	113	
21	30	40	88	
22	40	20	119	
23	40	0	136	
24	20		93	
25	0		159	
26	0		43	
27	20		253	
28	20		183	
29	20		147	
30	20		119	
31	20		76	
32	20		146	
33	20		142	
34	20		121	
35	20		135	
36	20		158	
37	20		136	
38	20		152	
39	15		127	
40	15		117	
41	15		133	
42	15		121	
43	15		117	
44	15		131	
45	15		118	
46	15		107	
47	15		117	
48	15		117	
49	15		102	
50	15		107	
51	15		107	
52	15		112	
53	15		114	
54	15		113	C peptide measured
55	15		127	
56	15		125	
57	15		138	
58	15		115	60 mcg/subQ
59	15		167	
60	15		244	
61	0		100	
62			117	
63			106	
64			126	
65			110	

Continued.

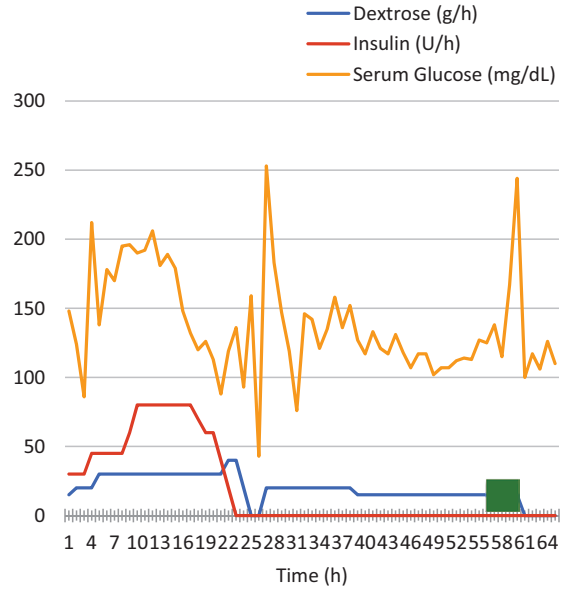

Figure 1 Rates of insulin infusion, supplementary dextrose infusion, and serum glucose concentration versus time. The patient required dextrose infusion of 15-20 g/h to maintain euglycemia for more than 30 hrs after cessation of the insulin infusion. The star above indicates where, after measuring an elevated c-peptide level, 60 micrograms of somatostatin was given subcutaneously leading to rapid off-titration of dextrose and maintenance of euglycemia.

KEYWORDS Beta blocker, Somatostatin, Hypoglycemia

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140. Severe hypokalemia following barium acetate ingestion resulting in early ventricular dysrhythmias

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Background: Barium toxicity is manifested by gastrointestinal distress with severe toxicity including flaccid quadriplegia and life-threatening hypokalemia. Hypokalemia results from inactivation

of passive potassium efflux without impeding Na+/K + ATP-ase activity, producing intracellular potassium sequestration. Muscle weakness and paralysis are thought secondary to the degree of hypokalemia, though barium concentrations may also correlate. Barium, an element, is more commonly found in salt form and toxicity results from exposure to soluble salts. We report a case of barium toxicity resulting in cardiopulmonary arrest less than 3 hours following ingestion.

Case Report: A 33-year old female presented to an Emergency Department (ED) following a reported suicide attempt. She reportedly ingested 'a couple' scoops of barium acetate one hour prior to ED arrival. She initially was alert and oriented for EMS, in no apparent respiratory distress, with primary complaint of emesis. Initial 12 lead ECG demonstrated a wide complex tachycardia. She denied ingestion of other drugs or alcohol. Past medical history included hypertension, depression, PTSD, borderline personality disorder, and dissociative disorder. Upon ED arrival, she remained alert with intermittent somnolence and poor concentration, normal respiratory effort, and moved all extremities. Initial vital signs included: blood pressure 151/83mmHg, pulse 151bpm, and respiratory rate 18 breaths/min. Repeat ECG demonstrated sinus rhythm with a rate of 67bpm and first degree AV block and the following intervals: PR 232ms, QRS 96ms, QTc 526ms. With protective airway reflexes intact and adequate oxygenation obtained, endotracheal intubation was not initially pursued. However, over the following thirty minutes, intermittent periods of tachyarrhythmias progressed to wide complex tachycardias. Arterial blood gas revealed a pH 7.34, pCO2 29mmHg, pO2 77mmHg, HCO3 15.6mmol/L, potassium 1.6mmol/L and lactic acid 3.6mmol/L. Other notable labs included serum potassium and magnesium of 1.4mmol/L and 2.1mg/dL respectively. Intravenous potassium and magnesium replacement were immediately initiated at rates of 20mEq/hour potassium chloride and 2 grams magnesium sulfate over 20 minutes. The patient received an initial loading dose of lidocaine, and synchronized cardioversion was attempted twice for a perfusing ventricular tachycardia. Sodium bicarbonate bolus was also administered; soon after the patient decompensated into ventricular fibrillation. The patient underwent defibrillation and standard ACLS treatment, though remained in asystole following defibrillation. Additional therapies included 3mg epinephrine IV, 2g magnesium sulfate IV, and 1g calcium chloride IV. The patient was intubated soon after decompensating. Resuscitative efforts were discontinued and time of death called 1 hour and 36 minutes following ED arrival. Autopsy findings include postmortem toxicologic analysis demonstrating a serum barium concentration of 13mg/L.

Case Discussion: This case displays early conduction changes, likely related to intracellular sequestration of potassium. A wide complex tachyarrhythmia was evident on ECG less than one hour following ingestion. Our patient did not show evidence of flaccid paralysis or respiratory compromise prior to cardiopulmonary arrest.

Conclusion: Early conduction abnormalities with severe hypokalemia may occur with barium ingestion, highlighting the need to provide early, aggressive electrolyte replacement. Early airway management should also be considered with expectant clinical deterioration.

KEYWORDS Barium, Overdose, Hypokalemia

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141. Diagnosing toxic alcohol poisoning: The cost of using the right tool for the job

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Objective: To determine the financial benefits of real-time toxic alcohol (TA) serum concentrations in an urban public health system in the United States. We hypothesized that increased availability of TA testing decreases overall healthcare expenses by reducing unnecessary medical interventions.

Methods: We performed a 20-year retrospective review of all calls to our poison control center (PCC) to identify all TA exposures. We including only patients from one large public healthcare system. Unnecessary treatments were defined as meeting all of the following criteria: (1) suspected TA (ethylene glycol or methanol) poisoning, (2) absence of metabolic acidosis, (3) one or more doses of fomepizole administered, (4) hospital admission for one day or more, and (5) a documented initial TA serum concentration of <20mg/dL. We extracted the total number of hospital admission days and doses of fomepizole administered. Costs were defined as the fomepizole wholesale price and reported averages for regular or critical care (CC) beds. We compared these costs to the costs of obtaining diagnostic equipment, maintenance, and transportation of samples.

Results: 1.7 million PCC calls were screened; 290 were possible TA exposures. Ten met final inclusion criteria to build the model. Thirteen inappropriate doses of fomepizole were given over 15 inappropriate hospital days. These hospital days included 9 regular floor admission days, 4 pediatric CC admission days and 2 adult CC admission days. Daily hospital costs were $2,775 for floor beds, $4,500 for pediatric CC, and $6,700 for adults CC days. Costs for fomepizole were $1290/1-gram dose, for a total of $73,245 in unnecessary expenses. Purchasing a gas chromatograph (GC) and flame ionized detector (FID) would cost $25,000. Annual estimates were based on 60 specimens per year. Maintenance and supply costs were estimated at $1500/year. All other equipment already exist in a hospital laboratory. Total initial costs were estimated at $25,000. Annual operation costs of $2500 would be expected. No charge was attached to staffing as existing technicians could be trained to operate the GC. Thus total estimated costs over the 20 year period for running TA serum concentrations would have been $75,000, without accounting for inflation or return of investment. The difference in costs in the two groups were similar with an annual difference of $88. An additional 14 patients were identified with no confirmed TA serum concentrations who received at least 1 fomepizole dose, some of which were dialyzed empirically, likely adding to unnecessary costs.

Conclusion: The ability to obtain rapid TA serum concentrations can reduce healthcare expenses over time. Based on our model, the cost of only 3 instances of inappropriate therapy per year would exceed operating costs by $9,195 annually. We did not factor in potential costs of the adverse effects to unnecessary drugs, hospital acquired infections and medication errors on the unnecessary cost side. Additionally, we did not factor in recovery of investment which would include charges for running these tests. Further benefits can be extrapolated from this data including reducing hospital admissions, limiting hospital length of stay, improved hospital throughput, and likely overall improved care

KEYWORDS Toxic alcohol, gas chromatography, cost analysis

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142. Midodrine Overdose Presenting With Junctional Bradycardia

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Background: Midodrine is synthetic oral alpha1-agonist that is approved for treating symptomatic orthostatic hypotension. It is also commonly used off-label to treat dialysis-associated hypotension and recurrent reflex syncope. Here we present a case of midodrine

ingestion overdose that resulted in hypertension and junctional bradycardia.
Case Report: A 46-year-old female with past medical history of chronic kidney disease (CKD) stage III, HIV, migraines, depression, and obsessive-compulsive disorder presents to the emergency department two hours after ingesting approximately 40-50 midodrine 5 mg tablets in a self-harm attempt. She complained of chest pain and headache. Initial vital signs reflect blood pressure (BP) 154/80 mmHg and heart rate (HR) 37 bpm. On physical exam, she had normal mentation. An electrocardiogram (ECG) is done which showed a narrow complex bradycardia with a rate of 37 bpm, a QRS of 88 msec, and QTc of 397 msec. Lab results were significant for a creatinine 1.35 mg/dL (Ref 0.51-0.95 mg/dL), glomerular filtration rate 42 mL/min, and troponin T 25 ng/L (Ref
Discussion: Midodrine acts on alpha1-receptors to cause vasoconstriction and increase peripheral venous resistance. Few case reports exist in the literature of midodrine overdose though all documented cases presented with potent vasoconstriction that resulted in hypertension and a reflex bradycardia. In one case, a 22-year-old female ingested 350 mg of midodrine that resulted in a BP 210/100 mmHg and HR 43-60 bpm and was treated with nitroglycerin patches. To our knowledge, this is the first case of midodrine overdose that resulted in significant bradydysrhythmias. It is possible that her history of CKD could have contributed to her presentation as the active metabolite of midodrine, desglymidodrine, is predominately renally cleared.
Conclusion: In overdose, midodrine can cause vasoconstriction leading to chest pain, headache, hypertension and bradydysrhythmias such as junctional bradycardia.

KEYWORDS Midodrine, Junctional, Bradycardia

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143. Trends of Intentional Drug Overdose Among Youth: A Population-based Cohort Study

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Background: Intentional overdose is the commonest form of self-harm in adolescents globally. We explored temporal trends in intentional overdose among youth.
Methods: Using multiple linked healthcare databases, we conducted a population-based cohort study in Ontario, Canada, from 2002 to 2015. We included all patients aged 8 to 20 years who presented to an emergency department (ED) or were hospitalized for intentional overdose, stratifying by age and agent(s) consumed. We determined the annual incidence of intentional overdose over time. For context, we contrasted these against the annual incidence of select unintentional injuries in the same group over the same period.
Results: Intentional overdose in youth displayed a U-shaped trend over the study period. From 2002 – 2010 there was a decline in hospital visits for intentional overdose, however, between 2010 to 2015 ED visits increased by 75% and hospital admissions doubled. The sharpest increases were observed in adolescents aged 14 to 17 years, and the most commonly implicated substances were acetaminophen, antidepressants, and NSAIDs. Over the study period intentional overdose involving antidepressants nearly doubled and acetaminophen increased by 50%. In contrast, we observed steady and sustained declines in rates of hospital care for accidental injuries in the same population during the study period.

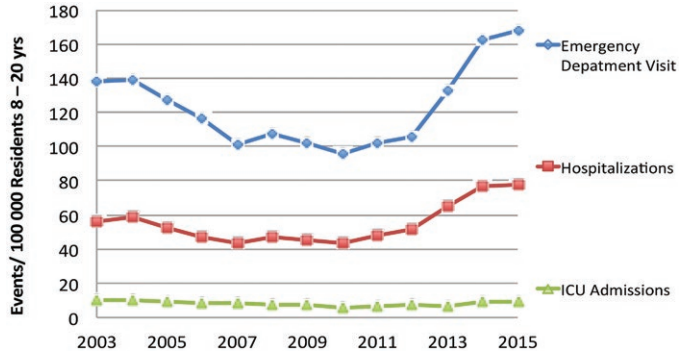

Figure 1 Incidence of Emergency Department visits, Hospitalizations and critical care admissions for intentional overdose in Ontario youth (8 to 20 years old), 2003-2015.

Conclusions: We observed a marked departure in the secular trend of injuries in youth 2002 to 2015 when comparing intentional poisoning to unintentional injuries, with a particularly striking increase from 2010 onward. Specific strategies to mitigate the sharp increase in intentional overdose should be particularly targeted to youth aged 14 to17 years and should focus on drugs commonly found in the home, with special attention to antidepressants.

KEYWORDS Overdose, Adolescent, Intentional

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144. Sickeningly sweet: a genetic disorder to include in a toxic alcohol differential

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Background: Maple syrup urine disease is a rare inherited autosomal recessive disorder, with an estimated incidence of 1 in 380 newborns among those in the Mennonite population. There is an inability to breakdown certain amino acids, leading to bioaccumulation and subsequent toxicity. When the individual is under metabolic stress, the urine imparts a sweet odor, similar to maple syrup. Characterized by vomiting, ketoacidemia, and lethargy, the condition is often evident at birth, but variants of the gene mutation may present later in childhood. We present a pediatric patient from the Amish community who presented to the emergency department (ED) with a high osmol gap, anion gap, vomiting and decreased urine output, leading to a differential that included possible toxic alcohol ingestion.
Case report: A previously healthy, well-nourished, non-diabetic 3 year old presented to the ED earlier in the day for nausea and vomiting, and was brought back for ongoing complaints. Labs drawn during the second presentation resulted in the following abnormal findings: osmolar gap of 20, anion gap of 36, pH of 6.96, and a beta-hydroxybutyrate of 10.75. She was acidotic with kussmaul respirations. The urinalysis revealed the presence of protein and ketones, while the blood glucose was 100 mg/dL. Her creatinine was 0.59 and co-ingestant suspicion of acetaminophen, aspirin and ethanol were negative. The family had taken the child to a naturopathic specialist due to the vomiting, and was given bottles of vital blend, adrenal/endocrine support, and parasite clear to treat the child. The family denied access to a toxic alcohol, but the hospital's treating team considered treating her from a toxic alcohol standpoint, and consulted the poison control center. Eight hours post arrival her acidosis improved spontaneously. The methanol

and isopropyl alcohol levels were negative, but the acetone level was 47.3 and the glucose rose to 270 mg/dL. She received insulin/ glucose to keep her euglycemic, but did not receive either fomepizole or hemodialysis. The ethylene glycol level was still pending when a final diagnosis of maple syrup urine disease was confirmed by the genetic testing team nearly 18 hours post presentation to the ED. The ethylene glycol concentration eventually resulted as negative and she was cleared from a toxicology standpoint. She was started on an individualized tube feed of total parenteral nutrition and later transitioned to a strict lifelong new restrictive diet.
Case discussion: This case report highlights the importance of including genetic disorders in the differential of patients who present with a history inconsistent with the suspected toxicological agent. Many of the clinical manifestations in this child were consistent with toxic alcohol ingestion, and the recommendation to treat with fomepizole was made based on clinical and lab findings. Ultimately the child was not treated with fomepizole as the results from genetic testing provided clarity on the cause of her symptoms.
Conclusion: We report an unusual case of maple syrup urine disease, a rare genetic disorder, that presented similar to a toxic alcohol ingestion in a young Amish child.

KEYWORDS Maple syrup urine disease, toxic alcohol, genetic testing

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145. Characteristics and trends of pediatric suicide attempts reported to poison centers in a single US state

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Background: In recent years, suicide attempts in the pediatric population has been noted to be rising at an alarming rate in nearly every state and is considered one of the 3 leading causes of death. In this study, we aimed to review and describe pediatric suicide attempts reported to poison control centers in the state of Ohio.
Methods: Using Toxicall database repositories, we queried all human cases coded as intentional suicide in ages 6 -19 from 2009 to 2018. All routes of exposure were included for review. We excluded cases coded as confirmed non exposures.
Results: Of the 28,102 cases included in this review 76% were female. The case count steadily increased over the study period from 1941 in 2009 to 3805 in 2018. Ninety five percent of all cases were in the 13-19 year group with the highest number of suicide attempts reported in the 16 year old group (n = 4960). Of the 1,274 cases in the 6-12 year group, the number of suicide attempts increased with age ranging from 8 attempts in the 6 year age group, to 834 in the 12 year group. The majority of cases (71%) were acute exposures, 26% acute on chronic and 1% were chronic exposures. Analgesics accounted for 30% of substances reported. Eighty nine percent of the analgesics were over the counter products (OTC). After analgesics, antidepressants accounted for the second highest substance coded (18%) followed by sedatives/antipsychotics (11%). Among patients admitted to a healthcare facility (HCF), 20% were admitted to critical care, 19% were admitted to non-critical care, and 32% were admitted to a psychiatric facility. There were 711 cases with a major effect, 7035 cases of moderate effect and 9913 cases with a minor effect. There were 12 deaths in the 13-19 year age group. There were no deaths in the 6-12 age group. The case count average was highest across all age groups on Mondays and lowest on Saturdays. Months with the lowest number of attempts for the 13-19 year old age group were June, July and August. The month with the most attempts in the 13-19 age group was January. In the 6-12 age group, the months with the least number of attempts was June and July and April was the month with the most attempts.
Discussion: Intentional self-harm attempts with overdoses in adolescents and pre-adolescents continues to rise and is concentrated in females. In this data set the majority of children and teens attempted suicide using medications they were not taking regularly. Keeping all medications including OTC products locked and inaccessible to this vulnerable population may be an important anticipatory guidance measure to consider discussing with families and physicians. Demographic and temporal patterns are similar to previous reports.

KEYWORDS Pediatric, suicide, poison center

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146. Intoxication By Methyl Parathion: A Report of Two Cases

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Methyl parathion (MP) is an organic phosphorus insecticide that acts by irreversible inhibition of acetylcholinesterase, triggering a cholinergic syndrome. As with other organophosphate, intoxication by MP is a clinical diagnosis, although confirmation requires the measurement of cholinesterase activity. Treatment begins with decontamination and measures to prevent hypoxia. Pharmacological therapy include atropine and oximes. Due to its high toxicity (rat oral LD50 13 mg/kg) MP is banned by many countries.
Case 1: A 22-year-old female, in her 12th week of gestation was found in her room with sialorrhea, vomiting and agitation. On admission to the emergency department, her Glasgow Coma Scale score was 8 (E2, V2, M4), presenting miotic pupils, sialorrhea, bradycardia (36 bpm), bronchorrhea, bronchospasm, diarrhea and urinary incontinence. Cholinergic syndrome was considered, so HAZMAT protocol was activated and patient was intubated and decontaminated. Few hours later, her boyfriend found an empty bottle of methyl parathion in her room. In the course of her treatment she required a total of 121 mg of atropine, administered in both bolus and continuous infusion, as well as norepinephrine infusion, and obidoxime initially in bolus and subsequently in continuous infusion for 24 hours. After stabilization, an obstetric ultrasound was performed which reported a 12.3 week pregnancy. She was extubated 48 hours later and was transferred to a psychiatric ward 5 days after admission. The patient was closely monitored, continued with prenatal control until delivery resolution and had a healthy newborn without complications.
Case 2: A 56-year-old female with a history of depression and suicide attempt, was presented to the emergency department referring to having ingested about 250 ml of methyl parathion one hour before her admission in a suicide attempt. Her Glasgow Coma Scale score was 12 (E3, V4, M5). She had nausea, vomiting, diaphoresis, abdominal cramps and mild bradycardia (52 bpm). Prior to notification to the toxicology team, some members of the emergency department had direct contact with the diaphoretic skin of the patient, which caused general discomfort, nausea and abdominal cramps in one of them. After that, HAZMAT protocol was activated and patient was decontaminated. Atropinization was performed and obidoxime was administered at 3 hours after exposure. She required only 4 mg of atropine and no complications were observed. Three days later she was transferred to a psyquiatric unit.

Methyl parathion is a highly toxic organophosphate, although the rapid recognition of intoxication and early treatment can counteract the effects and prevent complications. In the first case, the evolution time is unknown, however, 12 to 18 hours elapsed before the patient received obidoxime, which was probably related to her response being delayed, unlike the second case, in which the rapid identification of the xenobiotic allowed the early onset of obidoxime, showing rapid improvement with a low amount of atropine. Organophosphates are liposoluble xenobiotics with great absorption potential, so that Health care providers who have contact with the patient must have adequate protection. The lack of knowledge of these measures by the first contact physicians and nurses could generates more victims.

KEYWORDS Methyl Parathion, Organophosphorates, oximes

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147. Tetramethylammonium Hydroxide (TMAH): In vitro water and amphoteric solution decontaminaton, Ex vivo human skin explants studies, and TMAH RAMAN spectroscopy

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Introduction: TMAH is a strong corrosive base widely used in industry. It dissociates into TMA + and OH- ions, causing chemical skin/eye injuries and reported fatal systemic toxicity. Dermal injury from OH- may increase TMA + tissue penetration.
Materials and Methods: In vitro experiments used a semipermeable cellophane membrane to mimic TMAH skin diffusion. TMAH decontamination was performed with tap water or an amphoteric solution. pH measurements were taken every 10-30 seconds until pH stabilized. Mean decontaminant volumes were recorded. Ex vivo human skin explants: 25% TMAH was applied to human skin explants for 1, 5, 10 or 20 minutes. Explants were sampled on Day 1 (after exposure and 10 min running water rinsing) and Day 1 (24 hrs post-exposure) for general morphology. RAMAN spectroscopy: TMAH peaks were obtained not found in human skin.
Results: 25% TMAH rapidly diffused through the semipermeable membrane. pH was >12 after 4 minutes. During tap water decontamination, a precipitate occurred and persisted. With amphoteric solution, a precipitate occurred after 18 ml and then disappeared after 600 ml was added. For a physiologically tolerable pH of ~9, it was necessary to add 100 ml of amphoteric solution. With tap water, added volumes needed to be ~10 times greater (1,100 ml). Ex vivo explants: General morphology revealed the best concentration (25%) and contact time (20 min) for future studies. TMAH RAMAN Spectroscopy found 4 useful peaks at 317, 456, 1457, and 2824 cm-1.
Conclusion: In vitro studies highlight the corrosive base nature of TMAH and better amphoteric solution decontamination for returning pH to physiologically tolerable. Ex vivo human skin explant studies defined the best TMAH concentration (25%) and contact time (10 min) for future studies. TMAH RAMAN Spectroscopy found 4 spectra useful in further penetration and comparative decontamination studies.

KEYWORDS TMAH, Tetramethylammonium Hydroxide, In vitro Ex vivo studies

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148. Cyanide Toxicity After Mistaken Identity of Apricot Seeds

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Background: Apricot seeds contain amygdalin, a cyanogenic substance. Despite this toxic content and a lack of supporting evidence, apricot seeds are marketed as a health food with potential protective effects against cancer. Prior cases of cyanide toxicity after ingestion of apricot seeds as a health food have been reported. Almonds contain vitamin E, magnesium, potassium, calcium, protein, and other nutrients. We report a case of cyanide toxicity resulting from ingestion of apricot seeds which were mistaken for almonds and bended in a smoothie.
Case Report: A 78-year-old female with cardiovascular issues including coronary artery disease, atrial fibrillation, high cholesterol, hypertension, and valvular heart disease drank a "healthy smoothie" that she had made by blending banana, avocado and a handful of what she thought were almonds. Less than an hour after drinking the smoothie she felt unwell and began vomiting. She then realized that instead of almonds she had added her son's bitter raw apricot seeds. The package cautioned against consumption of more than 3 per day; she estimated she drank nearly 60 (1200 mg amygdalin) blended seeds. She induced further emesis prior to presenting to the emergency department with symptoms including headache, chest pain, diffuse abdominal pain, and weakness. At presentation (3.5 h post ingestion) her blood pressure was 120/55 mmHg, pulse 90 bpm, respirations 20/minute, temperature 97.4oF, and oxygen saturation 100%. She was awake, alert, and conversational. Her hemoglobin was 11.8 g/dL, anion gap 16, mixed venous blood gas pH 7.40 and pCO2 41 mmHg, and lactate 2.4 mm/L. She was given 12.5 g sodium thiosulfate at 4.25 h post ingestion. Her antihypertensive medications were held. Lactate at 7.25 h post ingestion was 1.3 mm/L and patient reported resolution of her symptoms. The patient was monitored for 24 h including during restart of her home medications. Serial troponin results and EKGs were not suggestive of acute ischemic changes. A subsequently resulted cyanide concentration, drawn at 3.5 h post ingestion, was 1.4 mg/L (reference).
Discussion: Due to complicated underlying cardiovascular disease, this patient was attempting to improve her nutritional status. A mistake confusing apricot seeds for almonds led to cyanide toxicity. Her clinical presentation and response to antidote therapy as well as the documented cyanide concentration, support the diagnosis.
Conclusions: Since 1979, reports of cyanide toxicity have resulted in calls to prevent further poisonings through regulatory action regarding the sale of apricot seeds for purported health benefits. Despite these calls, the marketing and sale of such products continue. This case highlights a new need if those calls remain unanswered. In addition to education about apricot seeds containing a cyanogenic substance, there needs to be education about their similar appearance to almonds, a food item with true nutritional benefits, and the potential for unintended ingestion leading to toxicity.

KEYWORDS Cyanide, apricot seeds, poison prevention education

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149. Use of intravenous lipid emulsion in the management of a citalopram overdose

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Background: Citalopram is a selective serotonin reuptake inhibitor which is highly lipid soluble and extensively metabolized in the liver by CYP3A4 and CYP2C19 to its active metabolites. The main metabolite among these is demethylcitalopram, which has been shown to be more cardiotoxic than the parent compound by blocking inwardly rectifying potassium channels. Intravenous lipid emulsion (ILE) has been used successfully in the treatment of local anesthetic toxicity and is thought to act as a lipid sink, binding lipid-soluble drugs. There have not been any reported successful cases of using ILE in a single-drug ingestion of citalopram. We describe here the use of ILE in the management of a single-drug ingestion of citalopram with refractory hypotension to conventional treatment.
Case Report: A 53 year old female ingested roughly 180 tablets of citalopram 40mg at approximately 1700. An hour and a half later, she became hypotensive, developed seizures and pulseless electrical activity with successful resuscitation. Upon presentation to the emergency department, her heart rate and blood pressure were within normal limits. She was transferred to the intensive care unit and despite aggressive management with vasopressors, fluids and inotropes she remained hypotensive with a blood pressure of 55/21 mmHg and required a transvenous pacer for pulses in the low 40's. On the basis that citalopram is a lipid-soluble drug, 20% ILE was administered. Blood pressure gradually improved during the infusion and 4 hours after lipid administration remained steady at 120/50 mmHg. By the end of hospital day 4 her seizures had resolved, blood pressure had remained stable and electrocardiography showed normal sinus rhythm. On hospital day 12 she was extubated, after which she remained alert, awake and oriented until her discharge home on hospital day 40.
Discussion: In overdose, it has been shown that the half-life of citalopram can be prolonged up to 80 hours due to the saturation of liver enzymes. The refractory hypotension and bradycardia, along with the increased cardiotoxic risk of demethylcitalopram required the use adjunctive therapy. Due to the lipid-soluble nature of citalopram and the proposed lipid sink mechanism, our patient was given ILE at 1.5 mL/kg over 1 minute, followed by 0.25 mL/kg/min for 10 minutes with a positive response. There have been large variations in the outcomes of using ILE as an "antidote". These wide variations are likely due to the variable proposed mechanisms and should be considered prior to administration.
Conclusions: In this case, we believe that the steady increase in blood pressure and heart rate and the improvement in neurological function following the administration of ILE was the result of the reduced plasma levels of citalopram and possibly its primary metabolite demethylcitalopram. We also believe that the effects of ILE as a metabolic substrate for the myocardium proved valuable in the positive outcome. Intravenous lipid emulsion appears to be a potentially useful adjunctive therapy in the management of severe overdoses with lipid-soluble drugs that are refractory to conventional treatment.

KEYWORDS Citalopram, Intravenous lipid emulsion, Overdose

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150. A Tylenol® by Any Other Name

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Background: Poison center research suggests that nearly half of the products in poison charts are incorrectly coded. Many factors contribute to the accurate identification of each substance involved in a poisoning. In this study, we seek to identify where the discrepancy occurs between the stated product involved in a poisoning and the product documented in the electronic medical record.
Methods: Charts of single-ingredient acetaminophen pediatric cases from our regional poison center (RPC) previously included in the survey "Monitoring the Impact of Interventions Involving Pediatric Formulations of Acetaminophen: Poison Center Surveillance Program" (MAPS) were retrospectively reviewed from March 2018 to March 2019. Variables evaluated to establish product identification accuracy included the survey response about the product name, substance code, written product description, and recording of the initial call. Cases in which the call recording was not found or the survey was not performed were excluded.
Results: Inclusion criteria were met by 156 cases, of which 65 (42%) matched all 4 variables (call recording, substance code, written description, survey response) to product name. In 70 cases (45%), the substance code and/or the written description of the product did not match the name of the product on recording. Forty-nine (31%) of these cases reported brand Tylenol products on recording, but the chart described a generic product in either substance code or written description. In 21 cases (14%), a generic acetaminophen product was on the recording, but the case was coded as either brand Tylenol or another generic manufacturer-specific product. There were 38 cases in which the survey response about the name of the product did not match the name initially heard on recording. When the product name was clarified during the initial call, the product name did not match on recording and the survey in only 5 cases, compared to 32 mismatched entries otherwise (3% vs 21%, p5000 results.
Discussion: Proper substance identification is critical to proper medical management, product research, and third-party data use. Clarification and verbal confirmation of the exact product during the initial call seem to improve accuracy. "Real-time tracking" of poison outbreaks depends on accurate identification of the substance involved.
Conclusions: Variables affecting proper identification and documentation of a product include packaging availability, caller interpretation of label information, clarification of the product name, and presence of the product in PDX. The use of brand names like "Tylenol", "Vaseline", or "Airwick" for other manufacturers' products can impact the reliability of the initial history. With time being a limited resource in handling poison emergency calls, fine-tuning the search function of PDX or utilization of product-specific Universal Product Codes or an International Article Number (EAN) may positively impact the accuracy of manufacturer-specific products coded by poison centers.

KEYWORDS Poison center, documentation, product identification

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151. No more red eyes: Toxic tetrahydrozoline ingestion in an adult. A case report and literature review.

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Background: Tetrahydrozoline, the active chemical found in many over-the-counter ocular decongestants, is a well-documented toxin in numerous pediatric ingestions. With systemic effects on the imidazoline receptors and central alpha-2 adrenoceptors, tetrahydrozoline can cause life-threatening toxicity, including central nervous system depression, hypotension, and bradycardia, in very small amounts. Physiologic derangements may be subtle on initial presentation but can worsen for days post-ingestion. With increasing numbers of adult tetrahydrozoline toxic ingestions, it is important for toxicologists and emergency medicine physicians to understand the presentation, progression, and management of tetrahydrozoline toxicity.
Case Report: A 40 year-old African American male with a past medical history of hypertension, diabetes, asthma, and alcohol abuse ingested a 15mL bottle of over-the-counter Visine® (Tetrahydrozoline) in a suicide attempt. He presented to the emergency department 2 hours post-ingestion with delirium and normal vital signs. His vital signs

worsened over the next 36 hours, requiring admission for prolonged supportive treatment of severe hypotension and bradycardia. Six days following ingestion, his vital signs normalized and he returned to his baseline state of health.

Case Discussion: This case demonstrates the effects of a relatively large tetrahydrozoline ingestion in an adult. Hemodynamic effects may not manifest for hours to days and may persist for up to 6 days following ingestion. Care is mainly supportive, with fluids and cardiac monitoring. For patients who remain asymptomatic, an observation period of 6 hours is adequate; symptomatic patients, however, should be admitted for at least 24 hours. Rarely, intubation and/or vasopressors will be required. Gastrointestinal decontamination is rarely indicated, and although not studied to date, hemodialysis is likely ineffective due to a large volume of distribution.

Conclusions: While ingestion of tetrahydrozoline products in pediatric patients and sexual assault victims is well described, few reports highlight the presentation of acute, purposeful – and possibly larger - ingestions in adult patients. Systemic, life-threatening effects of tetrahydrozoline ingestion may not manifest immediately and, therefore, are important for emergency physicians to be aware of. Discharging a patient who has ingested tetrahydrozoline before an adequate observation period may lead to deleterious consequences outside the hospital. Alternatively, patients may be admitted to the emergency department already unresponsive or unreliable, with profound CNS depression and severe hemodynamic compromise

Table 1 Receptors implicated in tetrahydrozoline toxicity.

Receptor	Receptor Subtype	Location	Physiologic Role
Alpha-Adrenergic Receptor	Alpha 1	Smooth muscle cells of blood vessels in the eye, bladder neck, prostate, urethra, liver, and salivary glands	Vasoconstriction (↑PVR and afterload leading to ↑BP), mydriasis, contraction of bladder trigone
	Alpha 2A	Central nervous system and locus coeruleus	Decreases sympathetic outflow (↓BP, ↓HR); anesthetic properties
	Alpha 2B	Vascular smooth muscle cells	Mediates vasopressor effect (↑BP, ↑heart rate) in the acute phase; net effect: ↓HR, ↓BP
	Alpha 2C	Central nervous system and basal ganglia	Analgesic (via release of substance P), sedative and anxiolytic effects
Imidazoline Receptor	I_1	Plasma membrane, lateral reticular nucleus, locus coeruleus, kidney, pancreas, and platelets	Modulates sympatholytic response (↓BP, ↓HR)
	I_2	Mitochondrial membrane, interpeduncular nucleus, arcuate nucleus, pineal gland, liver, heart, kidney, white adipocytes, and striated muscle	Monoamine oxidase (MAO-A and MAO-B) turnover; allosteric binding site of MAO; ↓ core body temperature
	I_3	Pancreatic beta islet cells	Stimulates insulin secretion in a glucose-dependent manner

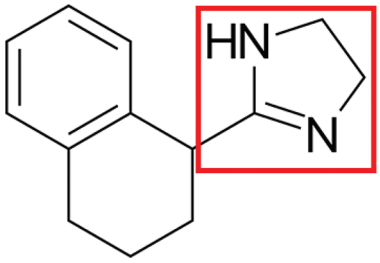

Figure 1 Tetrahydrozoline structure.

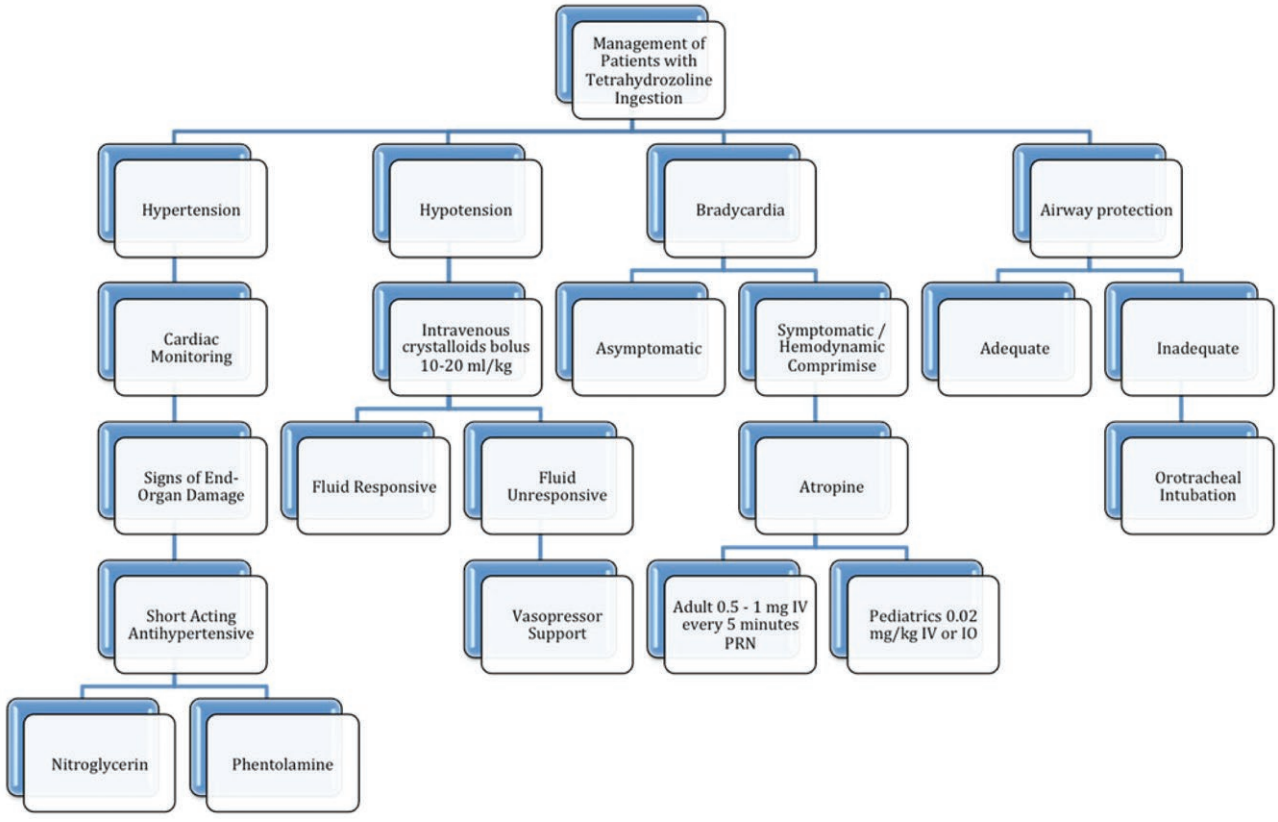

Figure 2 Medical management following tetrahydrozoline ingestion.

mimicking other toxic ingestions. A high index of suspicion is needed to identify tetrahydrozoline toxicity in these cases, and an understanding of the pharmacokinetics and pharmacodynamics of tetrahydrozoline products is vital to guide treatment of these patients.

KEYWORDS Tetrahydrozoline, Imidazoline Receptor, Alpha Adrenergic Receptor

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152. Death due to intentional colchicine overdose after clozapine-induced myocarditis

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Background: Significant morbidity and mortality are associated with colchicine overdoses. We describe the clinical course of a fatal intentional colchicine overdose in a schizophrenic patient who was prescribed this medication to treat his clozapine-induced myocarditis and pericarditis.
Case Description: An 18-year-old male with a past medical history of schizophrenia presented to the emergency department (ED) 12 hours after a presumed overdose. He was found somnolent on the floor by his mother lying next to empty pill bottles containing an estimated 60 tablets 0.6mg colchicine, 28 tablets 1mg lorazepam, and 20 tablets 20mg aripiprazole. His mother reported that he had several episodes of vomiting and diarrhea the night prior. Of note, he had been hospitalized two weeks previously for clozapine-induced myocarditis & pericarditis. Upon ED arrival his vitals were: heart rate 140 breaths per minute, blood pressure 103/83 mmHg, respirations 18 breaths per minute, SaO2 100% on 2L O2, and temperature 36.8 degrees celsius. He was obtunded and subsequently intubated for airway protection. Greenish-white fragments were noted in the post-intubation emesis. Given the morbidity associated with the overdose, gastric lavage, activated charcoal, and whole bowel irrigation (WBI) were utilized. Initial labs were significant for: WBC 20.1 K/uL, creatinine 3.27, lactate 7.4, pH 7.1 and bicarbonate 10mmol/L. The patient was aggressively resuscitated with IVF, vasopressors, bicarbonate infusions, and admitted to the ICU. Continuous veno-venous filtration was used in lieu of hemodialysis on account of hemodynamic instability. His vasopressor requirement escalated through day 2, at which time his echocardiogram showed severely reduced cardiac function, and the decision was made to perform VA-ECMO. The latter being complicated by a left femoral artery injury that was repaired in the OR; he received 22 units of blood products, tranexamic acid, and prothrombin complex concentrate in the process. On hospital day 3, plasma exchange was performed twice, but the patient ultimately expired after a bradycardic arrest. His peak colchicine level was 11 ng/mL (therapeutic:
Discussion: The peak colchicine level we observed in this case is consistent with the estimated 0.5mg/kg quantity ingested, as well as the clinical course and outcome. While colchicine may represent a valid treatment option for many instances of myocarditis or pericarditis, this case illustrates the critical importance of preventing this overdose in the first place. Interdisciplinary communication during the management of complex vulnerable patients is of utmost importance. Clozapine use is on the rise by psychiatrists, and we should expect to see more patients with clozapine-induced myocarditis admitted to cardiology services in the coming years. As such, extreme vigilance should be exercised whenever colchicine is prescribed to schizophrenic patients whose last-resort neuroleptic has been discontinued.
Conclusion: Prevention may be the most practical approach to curtail colchicine toxicity, and providers across all specialties should be cognizant of its potential to cause harm, particularly amongst our most vulnerable patient populations. Even the most aggressive therapeutic interventions appear unable to rescue patients from severe colchicine toxicity.

KEYWORDS Colchicine, ECMO, Plasmapharesis

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153. Wood Ash and Water: Cause of Superficial Alkaline Burns in a Toddler

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Background: Chemical burns are common injuries, but some potentially caustic agents are not obvious. The ash residue which remains after wood is burned contains variable amounts of potassium hydroxide and potassium carbonate. The solution of lye that results from wood ash soaked or boiled in water is a strong alkali used to make soap from rendered fat. Those unfamiliar with this process may view ashes as harmless. We report a skin exposure to wood ash and water that rapidly produced superficial burns in a toddler.
Case Report: A 2-year-old boy was playing with his water gun in cold wood ash left in an outdoor wood-burning stove. Afterwards, he complained of pain but no itching; his mother noted redness over his hands, arms, and thighs. She administered diphenhydramine and called the pediatrician, who advised bathing with soap and water. She was then referred to the poison center. Photographs of the injured sites sent electronically to the poison center showed multiple, confluent, poorly-defined erythematous lesions interspersed with punctate lesions over the dorsal aspect of the hands, the posterior forearm, and the anterior thighs, bilaterally. Some of the larger lesions appeared to be edematous but no blisters were noted. The working diagnosis was acute contact dermatitis due to some component of the ash. Over-the-counter hydrocortisone cream was recommended to minimize inflammation, and an antibiotic ointment for soothing and protection. The lesions improved significantly overnight. By the fourth day post-exposure, the skin on the thighs had returned to normal; there were scattered small eschars on the hands and fingers. One site over the base of the thumb showed a healing ulceration from a deeper lesion.
Discussion: While wood ashes are considered nontoxic, they contain alkaline material in the form of potassium carbonate and potassium hydroxide, often referred to as pearl potash and potash, respectively. These compounds are highly soluble in water and react with water to release hydroxide ions which can produce a pH as high as 12. This alkaline solution is capable of causing clinically significant burns. There are only a few cases of such burns in the literature and these were quite severe. Most resulted in full thickness burns and necrosis which required surgical debridement and skin grafting. The alkaline potential of wood ashes diminishes after the first encounter with rain since it readily leeches out the caustic compounds. This may be one reason why burns that come to medical attention are an unusual occurrence. Despite our patient having only brief skin contact with wood ash splashed with water, the exposure caused unexpected superficial burns.
Conclusions: While chemical exposures causing acute irritant contact dermatitis are relatively frequent cases in poison centers, those from wet wood ash are distinctly uncommon and may not be recognized. This case should alert poison centers and primary care practitioners of the association of chemical burns with wood ashes and water.

KEYWORDS Wood ash, Dermal burns, Alkali substances

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154. Adolescent Suicidal Ingestions: Increases Reported to New Jersey Poison Information and Education System from 2000-2017

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Background: Suicide is the second leading cause of death in adolescents in the United States. According to the Centers for Disease Control and Prevention, the number of suicides in girls ages 10 to 14 has nearly tripled since 1999. The primary objective of this study is to characterize the cases of overdose with suicidal intent in the adolescent population reported to the New Jersey Poison Information and Education System (NJPIES) in both males and females in the 10-14 and 15-19 age categories.
Methods: This was a retrospective review of all cases of self-poisoning with suicidal intent reported to NJPIES from 2000-2017. Data was collected on patients ages 10-19 years old in whom reason for ingestion was coded as intentional-suspected suicide. Descriptive statistics were computed, Spearman's correlation was used to report on the strength of any monotonic trends, and Mann-Whitney U tests were used to compare differences between groups.
Results: A total of 18,050 cases were reviewed; the absolute number and relative proportion of cases as related to total yearly calls to NJPIES increased throughout the study period. The proportion of total NJPIES calls related to self-poisoning in adolescents with suicidal intent increased by 110% between 2000 and 2017 (1.1% vs 2.3%; Spearman's rho =0.80, $p < 0.0001$). There was a female predominance of average cases per year in both age groups (10-14: 167/198 $p < 0.00001$, 15-19: 591/804 $p < 0.00001$). The average cases per year in the 15-19 age group was larger than the 10-14 age group (794 cases vs 199 cases $p < 0.00001$). Females in the 10-14 age group had the largest increase between 2000 and 2017 with an overall rise of 170% between 2000 and 2017 (0.18% vs 0.49%; Spearman's rho =0.55, $p = 0.018$). This was followed by a rise of 112% between 2000 and 2017 in females ages 15-19 (0.66% vs 1.4%;Spearman's rho =0.75 $p < 0.0001$). Males in the 10-14 age group had an increase of 100% between 2000 and 2017 (0.03% vs 0.06%; Spearman's rho =0.58; $p = 0.011$) while males ages 15-19 had an increase of 71% (0.21% vs 0.36% Spearman's rho =0.75; $p < .00001$). Between 2000 and 2017 there were a total of 13 adolescent deaths reported; 8 females vs 5 males. There was a minimum of 1 death reported per year and a maximum of 3 deaths per year reported between 2001 and 2015.
Conclusion: There was an increase in suicide attempts by self-poisoning in both males and females ages 10-14 and 15-19 reported to NJPIES from 2000 to 2017. There were more cases of self-poisoning in females than males. The greatest increases were seen in females in both the 10-14 and 15-19 age group. This information can help focus provider and public health efforts in suicide prevention in this at-risk population.

KEYWORDS Suicide, adolescent, poison control center

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155. Death by medicine cabinet - Tragic adolescent hydroxychloroquine fatality

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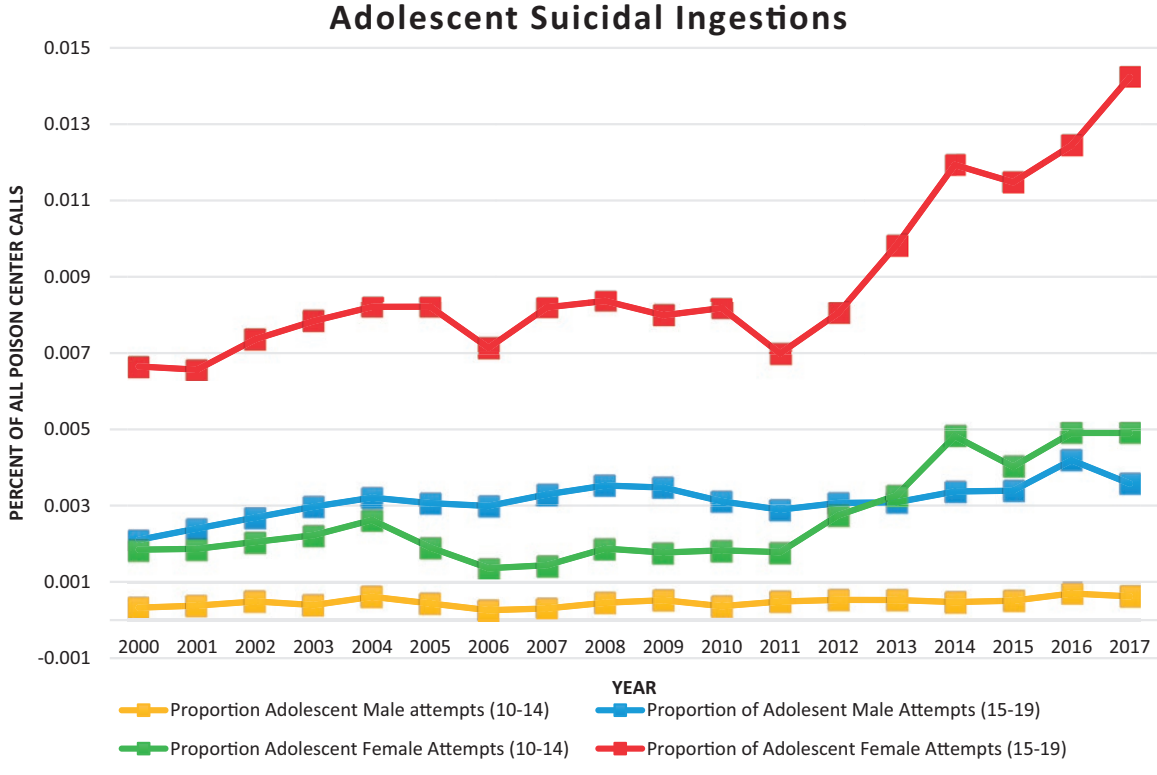
Background: Failure to dispose of unused medications results in accumulation of potentially toxic substances that may serve as a source for accidental or intentional poisoning. Hydroxychloroquine is an



anti-malarial and anti-rheumatic medication that may be viewed as innocuous by the lay public, particularly when so much focus regarding medication safety and disposal is focused on opioids. However, large overdoses of hydroxychloroquine can be rapidly fatal.
Case report: A 14-year-old female was transported to the Emergency Department (ED) by ambulance following ingestion of ~60 tablets of 200 mg hydroxychloroquine, several 750 mg naproxen, and an unknown quantity of 200 mg ibuprofen. Time of ingestion was estimated as 1-2 hours prior to presentation. The patient reportedly had an argument with classmates at school and was upset, thus resulting in the overdose. Shortly after arrival she suffered multiple episodes of cardiac arrest requiring intubation and resuscitative measures. Severe pulmonary edema and renal failure were noted. Vasopressors and intravenous fluids were required due to repeated hypotensive episodes. Potassium measured 1.5mmol/L ~ 30 minutes post-arrival. Bicarbonate, activated charcoal, and potassium were administered. At this time, the toxicologist recommended use of an Impella Heart Pump for cardio-circulatory support. The patient was transported to the cardiac lab and the device was inserted. Repeated epinephrine boluses were required throughout the procedure. Oxygen saturation was in the upper 70s to lower 80s despite 100% oxygen via manual ventilation. The patient was transported to the ICU, but her blood pressure remained tenuous even with continuous epinephrine and additional pressors. Calcium administration slightly reduced the rate of BP decline. HR was 62 at this time with marked metabolic acidosis. Pupils were fixed and dilated, there was no response to painful stimulus, and there was no spontaneous movement. Due to poor prognosis, a do not resuscitate order was obtained, care was withdrawn, and the patient died approximately five hours following arrival. Autopsy was performed and acute hydroxychloroquine intoxication was deemed the cause of death with a hospital serum/plasma level of 26,000 ng/mL.
Case discussion: Very few reports of significant, acute hydroxychloroquine overdose have been reported in the literature. At least three deaths have been documented including death following a 12 gram ingestion in a 2-year-old, a 12 gram ingestion in a 16-year-old, and a 14 gram ingestion in a 29-year-old. Our case includes the findings of hypokalemia, pulmonary edema, and cardiotoxicity that have previously been identified with significant overdose. This patient ingested approximately 230mg/kg. Cardiotoxicity is attributed to the quinidine-like action of chloroquine-like drugs. In some cases, cardiac arrest has been reported as the first manifestation following overdose. Unique to our case was discovery that the medication had been stored in the medicine cabinet for three to four years – well past its intended period of use.
Conclusion: Severe, acute hydroxychloroquine overdose may lead to rapid, life-threatening symptoms and must be treated aggressively. However, even with aggressive treatment, death may occur. In this case, appropriate disposal of a prescription medication, may have prevented the tragic loss of life of an adolescent.

KEYWORDS Hydroxychloroquine, fatality, medication disposal

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156. Pharmacy Students as a Natural Pipeline to Specialist in Poison Information

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Background: At our poison control center, Poison Information Provider-IIs (PIP-IIs) are doctor of pharmacy students that manage incoming calls under a limited scope of practice on the 24-hour hotline during peak call times (weekdays 1700-2200, weekends 1700-2300). One PIP-II FTE is needed to provide peak coverage and is shared across 4 PIP-IIs annually. Our center does not hire part-time Specialists in Poison Information (SPIs) and would need 2 SPI FTEs to provide coverage in the evening 7-days a week. As PIP-IIs reach specific levels of experience and years of didactic schooling, their scope of practice expands. The purpose of this study is to evaluate PIP-II workload, costs, and employment beyond graduation.
Methods: Inbound call, scheduling, and electronic medical record data were analyzed for PIP-IIs across all levels of scope (beginner, intermediate, and advanced) for a 2 year period. A cost benefit analysis was performed on yearly PIP-II costs and SPI costs (excluding training).
Results: From January 1, 2017 thru December 31, 2018, our center employed 6 PIP IIs who received 16,626 inbound calls, managed 10,347 exposures, and 728 information calls. Beginner PIP-IIs received an average of 4.7 inbound calls per hour (range 4.5-5) and managed an average 2.8 exposure and information calls per hour (range 2.5-2.9). Intermediate PIP-IIs received an average of 4.3 calls per hour (range 4.1-4.6) and managed an average 3 exposure and information calls per hour (range 2.7-4.2). Advanced PIP-IIs received an average of 4.3 calls per hour (range 4.2-4.4) and managed 3.1 exposure and information calls per hour (range 2.8-3.3). The average number of hand-offs per hour for out-of-scope calls decreased per advancement level – beginner 0.8, intermediate 0.6, and advanced 0.5. Cost benefit analysis showed that 1 PIP-II FTE costs 75.8% less than 2 SPI FTEs. Of PIP-IIs within the study that have graduated, 2 work as SPIs, 1 is seeking employment as a SPI, and 1 begins a hospital residency in July.
Conclusion: Implementation of a PIP-II program in our center was a cost effective way of adding third person coverage during peak evening hours with an added bonus of a potential pipeline of future SPIs.

KEYWORDS Poison Information Provider, Specialist in Poison Information, Cost

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157. Real time data reporting IS the REAL DEAL

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Background: Progressive organizations are now turning machine data into business solutions. Insights found within machine data are helping organizations better understand customer behavior and measure agent and departmental performance. While business leaders have long used data to drive decision-making on a day-to-day basis, data accessed in real time is now what is required. Leaders want data visualization, reporting tools and real-time analytics that provide immediate guidance. With real time data in hand, decision makers could develop more focused strategies and with confidence, take steps to move their organization forward.
Methods: At our poison center, we use proprietary analytic software to help shape business decisions in real time. This software solution directly interfaces with our contact center platform providing us with instantaneous data that allows not only upper level management to transform insight into action but the department as a whole. The innovative use of this type of information via real time dash boards, visual data trends and call center performance metrics has created value in our organization by generating results that can be readily understood and acted upon.
Results: Before the deployment of this software solution, creating reports and providing real-time data for interested staff was daunting. Preparing reports that displayed service metrics like employee performance or customer satisfaction was time-consuming and still did not deliver the impact necessary for staff to understand its

importance. The need for visual reports of real-time data was critical. After implementation, customized dashboards were developed with the end user in mind. These dashboards leveraged real-time data by allowing us to develop service level goals. For example, we reduced abandoned call rates by 62.4%. Average Wait time to Answer decreased by nearly 50% because staff and management could now view who was logged in and see how many people were waiting in our call queues. Presently, we are seeing additional reductions in other key performance indicators that have brought about great improvements in operating performance for our center. It was apparent that once we presented staff with instantaneous access to real-time data streams, it had the obvious potential to greatly improve work flow and enhance the customer call-in experience. Staff were more motivated, informed and empowered to improve their own performance because of the new software capabilities. The use of this analytical solution is not limited to just our environment. Other contact center applications utilize this program to improve workflow and efficiency. We look forward to setting new goals and expectations that center personnel can track and pursue.
Conclusion: Call center data is a mountain of gold waiting to be further mined and transformed into new capabilities. Our ability to collect and analyze this data is evolving at an exponential rate. Our PC is only beginning to understand the true impact it can have on the way we run our center. Discovering valuable insights from machine data can only help forward thinking organizations deliver a better customer experience and enhance business processes.

KEYWORDS Data trends, Performance metrics, Analytics

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158. Same Day Transportation for Opioid Use Disorder: Impact of the Crisis Addiction Recovery Transportation (CART) Program

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Background: Patients with opioid use disorder (OUD) frequently present to the emergency department. Delays or barriers in referral to recovery programs from the ED may decrease successful linkage to ongoing care. Thus, our institution partnered with a community program, Team Wellness (TW), to offer direct transportation 18 hours per day to the TW facility. TW provides medical, addiction, housing, and other psychosocial services. The referral process started 11/26/2018.
Methods: This is a retrospective chart review of patients referred to TW from three university-affiliated hospitals to assess for percent of successful handoff and program uptake from 11/26/2018 through 4/23/2019. A log of patients picked up by TW was cross-referenced with their ED encounter. Demographics, presenting ED complaint (i.e. opioid withdrawal, opioid overdose, seeking addiction treatment, other drug overdose, other drug withdrawal, and mental health complaint), substance(s) used, successful transport, ongoing treatment, and buprenorphine initiation were tabulated. Furthermore, all opioid-related complaints were compiled from an automatic report that collects ED visits with the following ICD-10 codes: 965.00; T40.0X1A; T40.0X2A; T40.0X3A; T40.0X4A; T40.1X1A; T40.1X2A; T40.1X3A; T40.1X4A; T40.2X1A; T40.2X2A; T40.2X3A; T40.2X4A; T40.4X1A; T40.4X2A; T40.4X3A; T40.4X4A; T40.601A; T40.602A; T40.603A; T40.604A; T40.691A; T40.692A; T40.693A; T40.694A
Results: From 11/26/2018 to 4/22/2019 there were a total of 409 ED encounters with the above ICD-10 codes. A total of 79 patients were referred to Team Wellness. 44 of the 79 referred patients presented with documented opioid use or an opioid-related complaint: 17 (39%) with opioid withdrawal, 11 (25%) with opioid overdose, 6 (14%) seeking addiction treatment referral, and 10 (23%) with other complaints but a history of opioid use. 37 (84%) were successfully transported to the TW facility. Of those, 24 were initiated on buprenorphine (10/17 with opioid withdrawal, 7/11 after opioid overdose, 3/6 seeking addiction treatment, and 4/10 with a non-opioid-related complaint). 22/24 patients initiated on buprenorphine have continued treatment as of 4/23/2019.
Discussion: Patients with OUD present to the ED frequently and at least a portion are interested in entering a recovery program. In this cohort of patients, 44 patients enrolled with TW and almost all patients that were initiated on buprenorphine have continued treatment. Healthcare literacy and social barriers prevent successful linkage to care. By removing the barriers of transportation and time to placement in an addiction treatment facility, ED patients can be successfully linked to care at a high rate. However, only a small percentage (44/409 or 10.8%) of all patients with opioid-related ICD-10 codes are being referred for treatment.

KEYWORDS Opioid Use Disorder, Addiction, Intervention

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159. Point of Care Ultrasound for the Toxic Patient (TOX POCUS)

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Introduction: Calcium channel blockers (CCB) or beta-blocker (BB) overdose can cause impaired contractility of the left ventricle (LV). Serial examinations of the LV and other circulatory parameters have yet to undergo rigorous examination in poisoned patients and remain an understudied phenomenon in clinical toxicology. Speckle-tracking echocardiography is an innovation that has potential utility in patients with toxic CCB or BB ingestions because of its ability to quantitatively assess the degree of LV myocardial deformation using a proprietary semi-automated algorithm. Our study objective was to describe the LV longitudinal strain in patients with toxic CCB or BB ingestions prior to and during initiation of treatment.
Methods: We conducted an analysis of patients who presented to our institution between October 2018 and March 2019 with suspected toxic ingestions of a CCB or BB. Patients were eligible for inclusion if they presented to the ED with a toxic ingestion of a CCB or BB and a member of the research echocardiography study team was available to perform an echocardiogram with speckle-tracking prior to, or concurrent with the initiation of any treatment. A limited echocardiogram was performed by a member of the study team using a Vivid q (GE Healthcare, Chicago, IL) portable ultrasound machine. Images from the apical window (4 chamber, 2 chamber and long axis) were obtained and saved on the ultrasound system for offline analysis using the speckle-tracking algorithm Automated Feature Imaging (GE Healthcare, Chicago, IL). A trained study team member recorded information regarding the clinical course of study subjects. Patients provided informed written consent and our Institutional Review Board approved the study.
Results: Three subjects were enrolled and all three had baseline "limited" echocardiograms prior to treatment. Two of the subjects had no substantial hemodynamic abnormalities (no episodes of sustained hypotension) and both of these subjects had normal LV global

longitudinal strain (GLS), -20.3% & -19.6% respectively (normal GLS range: -18 to -22%). One subject did have sustained hypotension ultimately requiring treatment with an epinephrine and a high-dose insulin infusion. This subject did demonstrate mild impairment in GLS on the baseline pre-treatment echocardiogram as LV GLS was -17.2% which resolved sometime between the 3 and 24 hour US examination. The visualized LV ejection fraction (EF) for all three study subjects on the baseline echocardiogram was greater than 50%. All three of the subjects were discharged alive from the hospital within 4 days of hospitalization.
Discussion: Point-of-care echocardiography to assess impairments in LV contractility in patients that have ingested toxic quantities of CCB or BB drugs is an understudied phenomenon in clinical toxicology.
Conclusions: Speckle-tracking echocardiography is a promising tool that could be utilized to determine both the dysfunction caused by various BB and CCBs as well as response to the various treatment options (e.g. vasopressors, high dose insulin, calcium, etc.). Results of these types of studies could lead to more specific and tailored therapy for patients that overdose on cardiotoxic xenobiotics.

KEYWORDS Ultrasound, Overdose, Cardiotoxicity

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160. Prospective Study of Acetaminophen Multiplication Product as a Risk Assessment Tool at a Regional Poison Center

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Background: Acetaminophen (APAP), the most common pharmaceutical overdose reported to poison centers (PCs), can lead to delayed hepatotoxicity (HT) despite minimal early symptoms. The Rumack-Matthew nomogram predicts patients at risk of HT who would benefit from acetylcysteine treatment, but is only applicable in cases involving single ingestions at a known time. The multiplication product (MP), serum APAP concentration multiplied by simultaneously obtained alanine aminotransferase (ALT), has been shown to correlate with development of HT regardless of ingestion time. An MP 10,000 predicts high risk. This study aims to validate use of the MP as a risk assessment guide at a PC managing APAP overdose patients in collaboration with hospitals of various sizes and capabilities.
Methods: This ongoing institutional review board-approved study includes patients age ≥12 years admitted to a hospital and reported to the PC for APAP ingestion. Data extracted from the PC record and documented in a spreadsheet includes demographics, exposure timing and type, initial and repeat APAP and ALT, acetylcysteine treatment, and medical outcome. MP is calculated for both initial and repeat labs. Cases without repeat lab values are excluded, except for fatalities. The primary goal is to assess the association between initial MP and development of HT (ALT >1000 IU/L). A secondary goal is to determine the proportion of APAP cases not amenable to use of the nomogram. For patients with undetectable APAP, MP is calculated using a value of half that hospital lab's limit of detection (5 if reported as <10, 1 if reported as <2 mcg/mL).
Results: From 4/29/18-3/8/19, 386 cases were entered into the study and 28 were excluded for incomplete data leaving 358 to be considered for analysis. Age range was 12-82 and 91 (25.4%) were male. 175/358 cases (48.9%) involved an acute ingestion at a known time. 107/358 (29.9%) had detectable APAP with unclear ingestion time. HT developed in 46 cases (12.8%). There were six fatalities (two likely unrelated to APAP), and one referral to an out-of-state transplant center. MP range in patients with HT was 712-887,695. Two patients with HT had an MP <1500 (712 and 1180); both had undetectable APAP and delayed presentation following acute overdose. In the four fatalities clearly related to APAP, MP range was 53,641-496,926.
Discussion: The APAP nomogram is applicable in only about half of patients reported to PCs. The MP appears to have the greatest potential as a risk assessment tool in the large proportion of patients with detectable APAP but poorly defined ingestion time. For those with undetectable APAP and suspected overdose or repeated supratherapeutic ingestion, the optimal approach to calculation and interpretation of the MP remains unclear. For such patients, other assessments of liver injury and function may be preferred.
Conclusions: The APAP MP is most useful as a risk assessment tool in patients with measurable APAP and uncertain ingestion time, but has limited utility when APAP is undetectable.

KEYWORDS Acetaminophen, Multiplication product, Hepatotoxicity

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161. Assessment of the familiarity by retail pharmacists regarding the availability and use of intranasal naloxone within a State.

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Background: In response to an increase in opioid-related deaths throughout a state, the Governor signed an emergency declaration to coordinate public health efforts between state, local, and private-sector partners. This allowed the state to utilize all its public health resources, including the distribution of naloxone, throughout the community to help prevent opioid-related deaths. Additionally, the Director of the Department of Health Services (DHS) issued a standing order for naloxone, making naloxone available at retail pharmacies for purchase. DHS, in conjunction with the State's two Poison Centers, created the Opioid Assistance and Referral (OAR) Line. The purpose of this line is to provide clinical guidance to prescribers, as well as management and referrals to the public. Through this line, the poison centers identified a lack of availability of naloxone at retail pharmacies. A survey to determine the availability of naloxone, understand the familiarity by retail pharmacists of the State's standing order for naloxone, their knowledge with its use, and their compliance with completing a free, 1-hour continuing education (CE) on pharmacist directed opioid antagonist dispensing is described.
Methods: A list of all currently registered pharmacies was obtained from the State Board of Pharmacy. Only retail pharmacies that were publicly available and used for the filling of legend and controlled substance prescriptions were included in our analysis. A phone call was placed to the pharmacy whereupon a brief survey with the pharmacist on duty was conducted. This survey (see Fig 1) consisted of 7 dichotomously answered questions regarding the availability and instructions for use of naloxone familiarity of the States' standing order, and compliance with the CE requirement. Pharmacists answers were documented in an Excel spreadsheet for analysis.
Results: 1121 retail pharmacies (chain or independent) throughout the State were identified. Phone calls to 139 pharmacies were attempted with 97 (8.7%) surveys completed. 87 pharmacies (89.7%) reported they currently had naloxone in stock and 92 (94.8%) reporting having recently dispensing it. 88 pharmacists (90.7%) reported being familiar with their ability to dispense naloxone pursuant to the State's standing order with 83 (85.6%) pharmacists reporting completion of the CE requirement. All pharmacists reported having direct knowledge

of and counseled their patients on the use of naloxone. 89 pharmacists (91.8%) reported having no previous knowledge of the Opioid Assistance and Referral Line.
Conclusions: Despite our initial concerns of a lack of availability of naloxone throughout our state's retail pharmacies, most surveyed pharmacies did have naloxone for immediate dispensing and were familiar with its use and availability pursuant to the State's standing order. Additionally, many pharmacists reported they identified patients at risk for opioid overdose and would offer to dispense a naloxone kit along with their opioid prescription. An opportunity to have pharmacists promote other opiate resources, such as the OAR Line, was identified.
Script for Naloxone/OARL Retail Pharmacy Survey
Step 1: Upon calling the retail pharmacy, ensure you're speaking with a pharmacist and not a pharmacy representative or pharmacy technician.

Step 2: "Hello, my name is… and I'm with Arizona/Banner Poison Control and I have a few questions regarding naloxone?

1. Do you have naloxone currently in stock?
2. If a patient presents to your pharmacy without a prescription, can you still dispense it?
   a. AZ DHS Standing Orders for Naloxone *(can be provided as requested)*
3. Did you complete the CE training to be able to dispense naloxone pursuant to the states' standing order?
   a. Pharmacist Directed Opioid Antagonist Dispensing (Naloxone CE Training) *(can be provided as requested)*
4. Have you, or another pharmacist dispensed naloxone previously?
5. Are you familiar with how to use naloxone?
6. Do you have an instruction sheet for use to provide patients?
   a. Naloxone Instructions for Use *(can be provided as requested)*
7. Are you familiar with the Opioid Assistance and Referral Line? *It's a toll-free number provided the Arizona Poison Centers for medical providers and the public that provides information regarding new opioid prescribing laws, practice guidelines, and connecting patients with treatment programs for substance use disorders. If you'd like, I can fax or email you more information.*
   a. OAR Line Information (888) 688-4222 *(can be provided as requested)*

KEYWORDS Naloxone, opioid, retail pharmacy

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162. Text Communications at a US Poison Center

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Background: Text messaging is immensely popular and is gradually replacing direct telephone calling as the preferred method of communication via mobile electronic devices. As poison centers continue to lose calls from the general public, institution of text communications may be a way to re-engage the patients of the digital age. The experience of one poison center in text communications with its clients may be instructive for other centers which are considering using it.
Methods: Two years of monthly data obtained from the texting service, covering January 1, 2016 through December 31, 2017, were analyzed. Separate Excel spreadsheets for the incoming and outgoing texts for each one month period were combined so that the messages (one or more) comprising a single conversation could be delineated. Each conversation was designated as an Exposure, Information Query, Spam or Other (intended for the poison center, but not about exposure or information). Exposure conversations were further identified as either originating from the poison center as a follow-up to a case in progress, or as originating from a member of the general public. The state which mapped to the area code of the incoming mobile device was recorded for each message. The total number of texts related to each Exposure conversation was tallied.
Results: This poison center does not advertise the availability of text messaging for communication. Never-the-less, in a two year period, the poison center sent or received 52,768 text messages, averaging 40 per day. In addition, 10,334 spam texts were received (most originating out-of-state) but were ignored. Some spam was considered inappropriate or harassing. Over 9,000 Exposure conversations were identified, of which 608 were new incoming exposure cases from the public, and 8,416 were existing poison center cases for which text messaging was used by the SPIs for at least part of the follow-up. Home state Exposure conversations averaged 4 ± 2.9 texts each (median 3, range 1 to 80). Overall texts to and from out-of-state mobile devices represented 45% of the total text traffic, but only 12.5% of the Exposure conversations. Of the poison center's home state Exposure conversations, only 0.5% were originated by text to the poison center; 99.5% were originated by the poison center following-up on existing cases.
Conclusions: There appears to be a substantial utility to text messaging in a poison center. In this case, since the service is not advertised, the majority of traffic was initiated by the poison center itself as a tool to get follow-up responses on telephone cases. Out-of-state text traffic to a poison center will occur, just as it does for telephone calls. Our data shows that exposure cases can be handled entirely by text, but the logistics and time implications of handling both phone and text communications at the same time need further study. Since modes of communication continue to shift towards computer and mobile messaging services, it may be beneficial for more centers to adopt this service.

Results over a 2-year period	Poison Center's Home State	Out-of-State
Texts In	12,695	16,073
Average per day	17	44
Texts Out	16,387	7,613
Average per day	22	21
Total Texts	29,082	23,686
Average per day	40	65
Spam	255	10,079
Average per day	<1	28
Exposure Conversations		
General public initiated	24	584
PC follow-up	7,855	561
Total	7,897	1,127
Texts per Exposure Conversation Mean (median, range)	4 ± 2.9 (3, 1-80)	9 ± 10.3 (4, 1-88)

KEYWORDS Texting, communication, poison center

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163. Geographic mapping of crotalid bites can lead to better antivenom allocation for health systems.

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Background: Our state has two endemic venomous snake species: the timber rattlesnake, Crotalus horridus, and the Northern copperhead, Agkistrodon contortrix. Bites are infrequent and many smaller hospitals choose not to carry crotalidae polyvalent immune fab (Crofab) due to its short shelf life and high cost. When a crotalid bite does occur, the poison center is often tasked with locating the nearest hospital with a sufficient antivenom supply. Our affiliated health network requested guidance in allocating their antivenom stock. To assist, we performed a retrospective review of crotaline snake bites in the state.
Methods: A local poison center database search was performed for all calls related to snake bites between 1/1/2012-4/15/2019. Human cases with a bite from a known or suspected crotaline snake that required Crofab were included. Information calls, out-of-state exposures, animal exposures, bites from a non-snake species, and bites from non-venomous snakes were excluded. Data collected were date of exposure, location of the caller, and number of vials of Crofab administered. Exposures were then geographically mapped and compared with hospital locations and estimated transport times.
Results: A total of 195 calls met search criteria. 178 were excluded (17 out of state, 12 information calls, 8 animal exposures, 15 non-snake exposures, and 126 non-venomous snake bites). Of 17 known or suspected crotaline bites, 13 received crotalidae polyvalent immune fab. With two case exceptions, all cases were clustered in two regions of the state.
Discussion/Conclusions: When independent hospitals partner to become larger health networks, resources can be consolidated. In this case, the information gained from a poison center search allowed a health system to place antivenom stock in those facilities most likely to treat snakebites based on the geographic distribution of bites in state.

KEYWORDS Snake, Crotalidae, Antivenin

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164. Life-Threatening Multi-Drug Overdose Successfully Treated with Extracorporeal Membrane Oxygenation, Intravenous Lipid Emulsion and Glucagon

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Background: Poisoning of either antiarrhythmics, antidepressants, antipsychotics or sedatives can result in severe outcome. A combination overdose of medications from all the above pharmacological groups is potentially life-threatening and greatly challenging. The presented case is a multi-drug overdose that resulted in a rapid cardiac arrest and successfully treated.
Case report: A 35-year-old woman with a history of multiple suicide attempts presented to the emergency department about one hour after ingesting flecainide 2,500mg, quetiapine 3,000mg, venlafaxine 1,000mg, escitalopram 250mg, bisoprolol 25mg, and clonazepam 5mg. Shortly after admission, the patient collapsed and became unresponsive with agonal breathing and no peripheral pulse. Pulseless electrical activity (PEA) was observed. Resuscitation was immediately initiated with intubation, ventilation, oxygenation, chest compressions, intravenous (IV) fluids and vasopressors. On-call clinical toxicologist recommended administering IV lipid emulsion 140mg (2mg/kg) and IV glucagon 5mg, and urgently connecting the patient to extracorporeal membrane oxygenation (ECMO). The patient was transferred to the intensive care unit (ICU) while resuscitated and the recommended interventions were performed. The patient gradually improved. Repeated echocardiogram demonstrated improved cardiac output. She was disconnected from the ECMO 4 days after the overdose and extubated 2 days later. The ICU hospitalization was complicated with anemia, thrombocytopenia and infection that were resolved following treatment. The patient was discharged after 12 days with no apparent residual impairment.
Case discussion: While the successful use of ECMO and IV lipid emulsion was previously reported with flecainide poisoning, the use of these interventions in multi-drug overdoses including flecainide was not. This report further supports the intensive approach of early use of ECMO for cardiac arrests secondary to medication overdose with no direct antidotes and limited potential for enhanced elimination (e.g., hemodialysis). IV lipid emulsion is seemingly a safe intervention with potential benefit. Additional treatments should be tailored depending on the drug combination overdose, such as glucagon for b-blockers.
Conclusion: ECMO, IV lipid emulsion and glucagon supplementing traditional resuscitation and supportive therapy led to the successful treatment of a life-threatening multi-drug overdose including flecainide, quetiapine, venlafaxine, escitalopram, bisoprolol, and clonazepam. ECMO and IV lipid emulsion should be considered early in multi-drug overdoses that may result in severe cardiac toxicity.

KEYWORDS ECMO, IV LIPID EMULSION, OVERDOSE

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165. Piloting Exposure Category Call Volume Algorithms to Improve National Public Health Surveillance Using Poison Control Center Call Data

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Background: The Centers for Disease Control and Prevention, National Center for Environmental Health (CDC-NCEH) collaborates with regional poison control centers (PCCs) and the American Association of Poison Control Centers (AAPCC) to conduct routine national surveillance of public health significant (PHS) incidents using PCC data uploaded to the National Poison Data System (NPDS). Algorithms in NPDS automatically detect anomalies in total call volumes to regional PCCs. Toxicologists and epidemiologists from CDC-NCEH and AAPCC review these anomalies and notify the state health department and regional PCC when an anomaly is deemed to be of PHS. Current NPDS call volume algorithms identify anomalous increases in total hourly call volumes to PCCs compared to historical baselines. In order to improve national surveillance for PHS incidents, CDC-NCEH proposed leveraging new NPDS surveillance capabilities to pilot test call volume anomalies using pre-specified categories of exposures (e.g. chemicals, pesticides, drugs of abuse). The objective of this project was to pilot exposure category call volume algorithms and determine if the new approach identified PHS incidents not otherwise captured through total call volume surveillance.
Methods: Based on public health and toxicological importance, we derived 20 exposure categories using existing product coding categorizations in NPDS. Exposure categories were broadly grouped as chemicals (n = 4), drugs of abuse (n = 6), pesticides (n = 3), gas/fume/vapors (n = 2), contaminated food/water (n = 1), and others (n = 4). Nine PCCs and six states were chosen to deploy pilot algorithms for the 20 exposure categories. We analyzed all anomalies generated by the pilot algorithms to determine whether the captured groups of calls were potentially PHS. We compared anomalies deemed to be potentially

PHS with anomalies generated from total call volume surveillance to determine whether the pilot algorithms were capturing different or additional incidents.
Results: From August 1 2017 through April 1 2019, the pilot algorithms generated 419 anomalies. Most anomalies were related to drugs of abuse (n = 144, 34.3%) and gas, fumes, and vapors (n = 124, 29.6%). The number of exposure calls per anomaly ranged from three to 28, with a median of six exposure calls per anomaly. After review, 136 (32.5%) anomalies were determined to be potentially PHS. Of these, 107 (78.7%) were not identified through total call volume surveillance.
Conclusions: The exposure category pilot project shows promising results for identifying new PHS incidents that would not be captured through the current total call volume surveillance. The lack of overlap between PHS incidents identified using exposure category surveillance and total call volume surveillance suggests complementary capabilities. Conducting exposure category call volume surveillance could improve national public health surveillance efforts using PCC data.

KEYWORDS Public Health, Surveillance, Exposure

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166. Treatment of pediatric baclofen overdose by elimination hemodialysis

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Background: Severe baclofen toxicity (BT) can result in respiratory failure, hypotension or hypertension, bradycardia, hypothermia, seizures, coma and death. While hemodialysis (HD) is well-described in the treatment of BT in patients with end-stage renal disease (ESRD), the utility of HD in the treatment of acute BT in patients with normal renal function is less clear. Implementing HD to speed recovery after a large acute baclofen exposure is tempting, considering: a) the potential for prolonged coma and ventilator-associated morbidity, and b) baclofen's low protein binding (31%), small size (213 Da), and moderate volume of distribution (Vd =2.4 L/kg). We report the lack of efficacy of HD in a patient with an intentional baclofen overdose and normal kidney function.
Case report: A 51-kg 14-year-old girl with no past medical history presented to the emergency department with hypotension, obtundation, and status epilepticus after an intentional ingestion of 1200 mg of baclofen. Her post-intubation neurologic exam was concerning for coma without any need for sedation. A 14-hour post-ingestion baclofen level was 882 ng/ml (therapeutic range 80-400 ng/ml). Urgent hemodialysis was performed due to her concerning neurologic status, with the goal of reducing her time on the ventilator. A Revaclear 300 dialyzer was used with an ultimate blood flow rate (Qb) of 300 ml/min and a dialysis fluid flow rate (Qd) of 400 ml/min. A total of three HD sessions were performed, with serum concentrations collected accordingly (Figure 1), and slow but progressive improvement in mental status. Baclofen concentrations were also systematically obtained in urine and dialysate during her hospital course. The total baclofen removed in the first three-hour HD session was 3.05 mg. The total amount of baclofen removed from urine over 24 hours on hospital day one was 42 mg, hospital day 2 was 9.2 mg, and hospital day 3 was 27.8 mg. A follow-up magnetic resonance imaging of the brain showed no evidence of anoxic brain injury. She was discharged without neurologic deficits to inpatient psychiatry on day 14.
Case Discussion: There are few reports on the use of HD for acute BT in patients with normal renal function, and limited data on HD efficacy. Given the 12-fold increased quantity of baclofen recovered in urine during hospital day one in comparison to HD, the use of HD appears largely ineffective in enhancing elimination for patients with normal kidney function. Furthermore, baclofen was below the limit of detection in the dialysate on days 2 and 3. Post-HD rebound was consistent with redistribution given baclofen's larger volume of distribution. It is possible that the drug had already been distributed to tissue at the time HD was started, about 30 hours post-presentation, rendering elimination by HD ineffective.
Conclusion: The amount of baclofen recovered during hemodialysis is negligible in comparison to normal renal elimination in this patient with normal renal function.

KEYWORDS Baclofen, Hemodialysis, overdose

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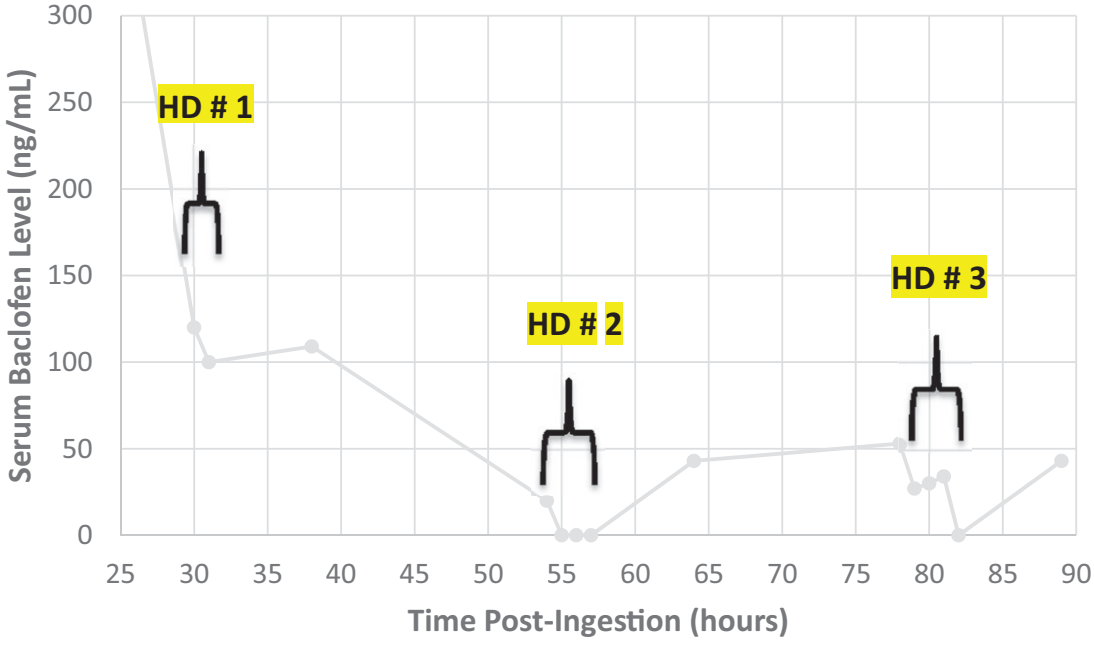

Figure 1 Venous blood baclofen levels throughout hospital course. HD = Hemodialysis.

167. The penetration of literature describing bupropion related harm after overdose in the non-toxicology related literature: a scoping review

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Introduction: Bupropion is an aminoketone antidepressant used for multiple indications including major depressive disorder and smoking cessation. The medication has been available in the United States since 1986 and the effects after overdose are well described in the literature that targets practitioners who treat overdoses (e.g, toxicology, emergency medicine, critical care [TEMCC]). Despite serious toxicity after overdose, bupropion is the fourth most commonly prescribed antidepressant in the United States. Multiple factors contribute to prescribing. If the literature that prescribers frequently read portrays limited risks of bupropion, this could impact medication prescribing patterns. This study sought to identify if articles describing the risks associated with bupropion overdose are published in the non-TEMCC literature.

Methods: A scoping review of all human overdose or suspected overdose of bupropion was conducted to address two questions. First, what quantity of literature directly describes bupropion overdoses or harm after overdose in non-TEMCC journals and conferences? Second, do other fields, specifically psychiatry and general practice, reference bupropion overdose articles published in TEMCC journals? A medical librarian searched for reports of human bupropion overdose in PubMed, Embase, Cochrane Library, and Scopus. Two independent authors screened each article for inclusion and a third author settled any discrepancies. Inclusion criteria were case reports, case series, studies, and systematic reviews describing primarily bupropion-related harm after overdose. Studies or cases of complex multi-agent co-ingestions, articles that did not primarily focus on bupropion, narrative reviews, and editorials were excluded. The Scopus database was used to determine the subject focus of each journal and to identify any literature that referenced the

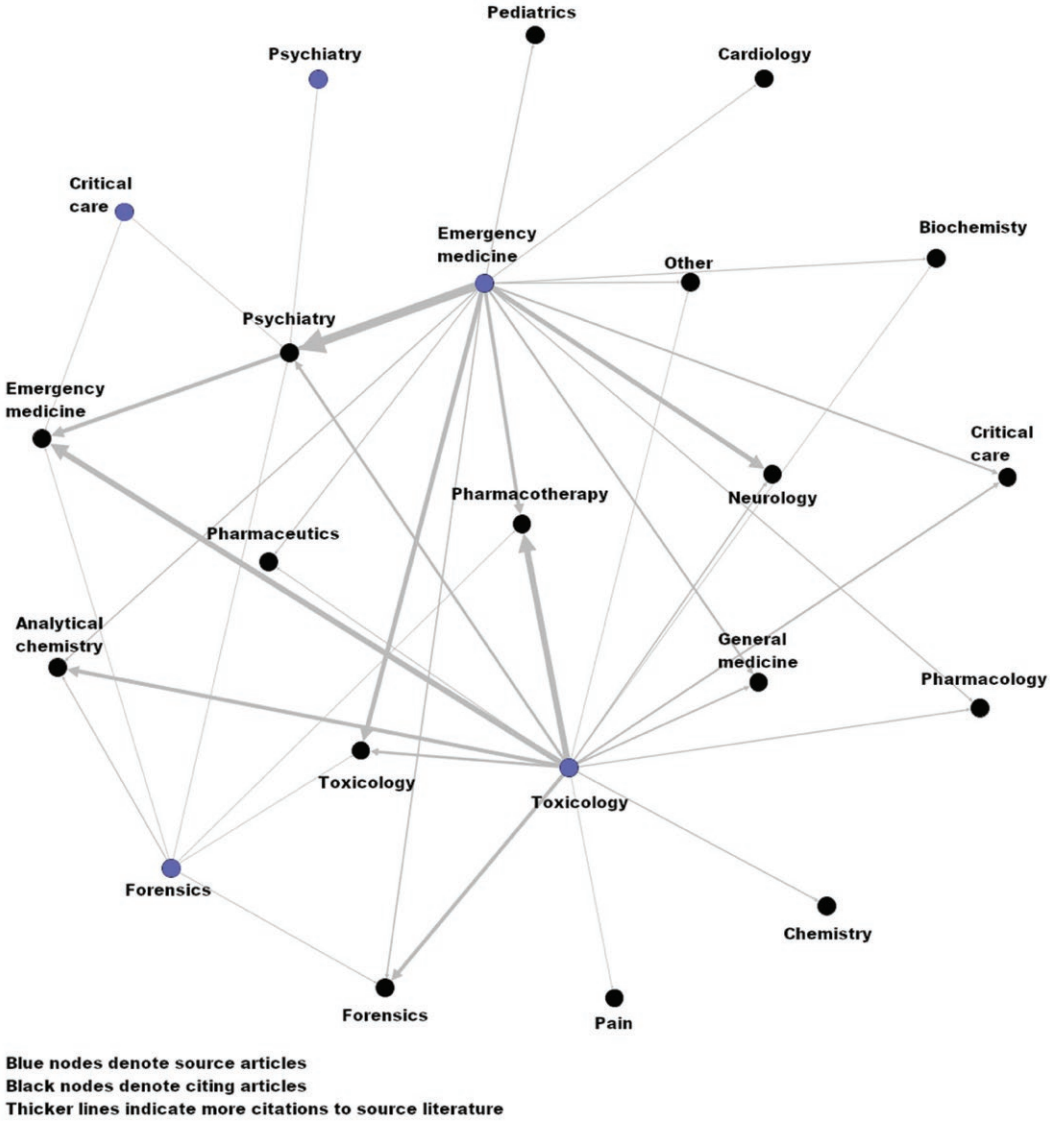

Figure 1 Network graph displaying connections between source journal topics (blue) and target journal topics (black) for any bupropion overdose article that included at least one death; thicker lines denote more citations to the source journal topic.

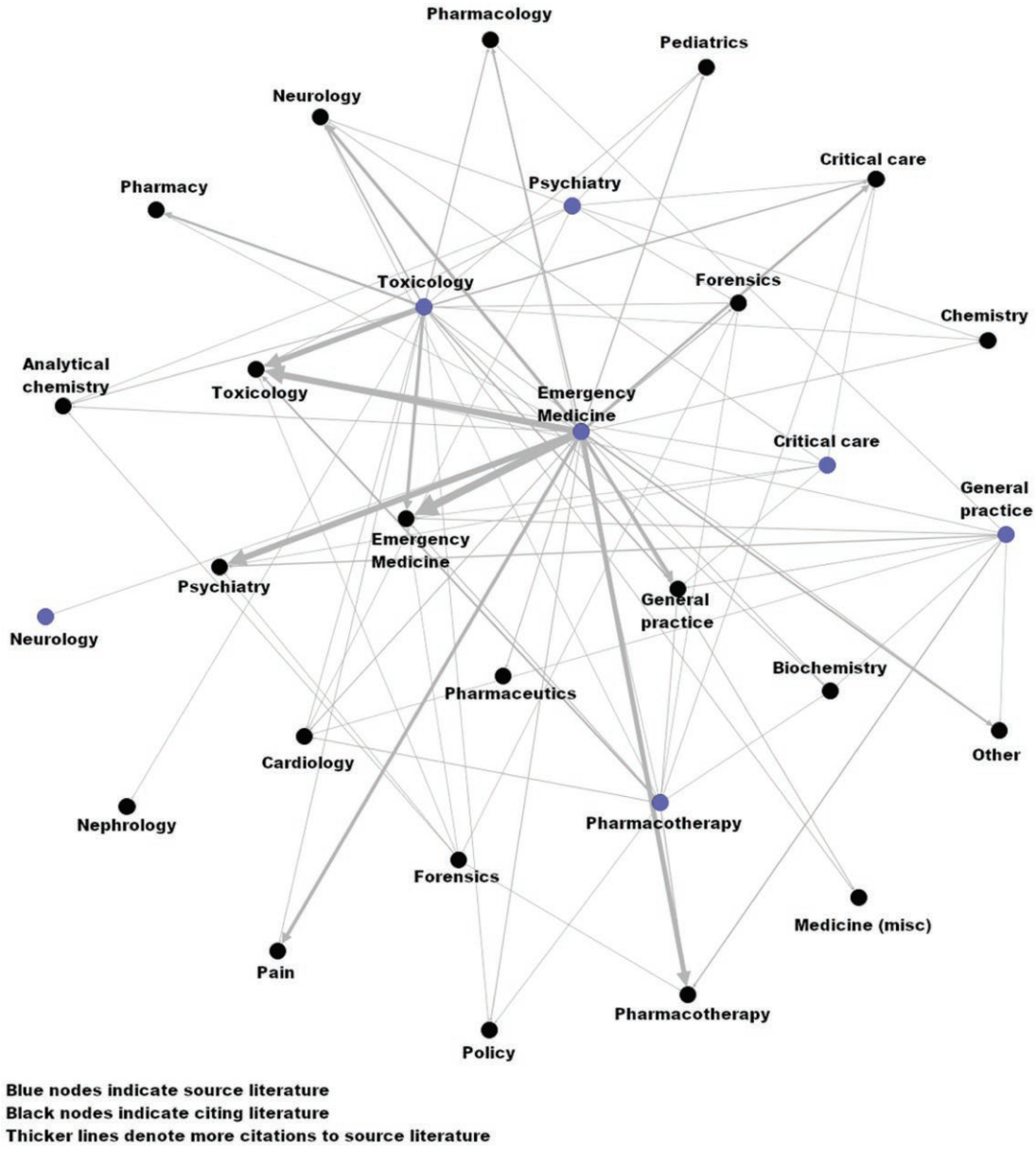

Figure 2 Network graph displaying connections between source journal topics (blue) and target journal topics (black) for any bupropion overdose article that included cardiotoxicity as an outcome; thicker lines denote more citations to the source journal topic.

included articles. If a journal had multiple topics, the one most relevant to the primary question was used. If the journal had multiple topics of focus relevant to the review questions (e.g., psychiatry and pharmacology), preference was given to the topic that was closer to psychiatry or general practice. Gephi social network analysis software was used to identify connections between the articles.

Results: The search identified 2622 articles, 159 were included for full text review and 117 were ultimately included in the analysis. Seventy-six were published in clearly TEMCC journals, 22 were published in clearly psychiatry/general medicine journals, and the remaining 19 were published in other fields (Table). The full-text articles were cited 1,458 times by 791 unique articles with the multiple focuses. Psychiatry literature represented the largest number of unique articles (n = 127), followed by toxicology (n = 107), then emergency medicine (n = 101), and other topics (n = 457). Figures 1 and 2 show networks of citations limiting the articles that describe only bupropion related deaths and bupropion related cardiotoxicity, respectively. The psychiatry literature cited primarily emergency medicine literature for both deaths and cardiotoxicity.

Conclusion: Despite most literature describing bupropion-related harm being published in the TEMCC literature, the psychiatry literature heavily cites the TEMCC literature regarding serious morbidity and mortality associated with bupropion overdose.

Journal topics for articles included in full text review and each of the referencing articles

	Included articles	Referencing articles
Journal focus	N = 117	N = 791
Toxicology	43	107
Emergency medicine	24	101
Psychiatry	12	127
General practice	10	66
Critical care	9	30
Neurology	6	53
Pharmacotherapy	4	77
Forensic medicine	4	21
Anesthesia/pain medicine	1	52
Other*	4	157

*Topics include: biology, nephrology, pediatrics, pharmacology, COPD, cardiology, analytical chemistry, epidemiology, dermatology, nursing, health policy, surgery, chemical engineering, public health, urology, biochemistry, and pharmaceutical science.

KEYWORDS Bupropion, overdose, scoping review

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168. Demonstrating the Value of a Poison Center Through a Visual Dashboard

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Background: The Washington Poison Center (WAPC) is a 501(c)(3) nonprofit organization that provides a 24/7/365 emergency telephone helpline for the public and healthcare providers. There is no established benchmark funding for this vital public health service for which no other public agency in the state can provide. Using objective metrics to demonstrate value of critical services is important in funding requests to legislators and other stakeholders.
Objective: To create a Value Dashboard using selected metrics.
Methods: We identified 5 statewide and 3 federal poison center (PC) value metrics. Federal metrics were published in the Lewin Reports, 2012 and 2017, using national PC data.
Results: Statewide value metrics: Availability: provide 24/7/365 telephone services including a national toll free poison helpline– in 2018 we answered 113,586 calls ad managed 64,140 cases. Accessibility: telephone consultative services are accessible to anyone in Washington State (WA)—all citizens including vulnerable and underserved populations, and healthcare providers; 26.3% of calls were from healthcare providers. Wraparound Healthcare: follow patients from initial call to resolution; over 39,000 follow-up communications were done from 2016-18 after initial healthcare facility call. Healthcare Savings (state): From 2016-2018, WAPC saved WA state an estimated $77 million by managing people with selected toxic exposures at home. EMS Savings: In 2018, when 911 called the WAPC first and the patient was kept at home, ~$3.3million was saved. Medical Toxicology Physician Consultations (MTPC): In 2017 and 2018, provided 1389 MTPC with an unreimbursed value of $420K. Federal metrics: Poison Centers decrease hospital length of stay by 0.5 days, saving $201 million annually; reduce probability of hospital admissions by 13.9%; total annual medical cost savings of almost $2 billion.
Conclusions: WAPC and PCs provide a value-based public healthcare model through their specialized telephone consultations. Developing a visual Dashboard demonstrating objective metrics is an important tool in demonstrating value. WAPC has saved WA state millions in healthcare costs. Investing in poison centers is a cost-effective way to invest in public health. As a vital public health service, it is important to ensure uninterrupted funding.

KEYWORDS Poison center, value, healthcare savings

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169. The Vast and Varied Unknown: A Retrospective Review of "Unknown Pill" Ingestions in the Pediatric Population.

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Background: Pediatric ingestions of unknown pills are reported daily to poison centers. These "unknowns" in children less than 6 years of age may range from worst-case scenarios of highly toxic medications ("one pill could kill") to nontoxic substances. To standardize and efficiently treat each "unknown" case, a chart review was instituted to better understand the regional statistics and the treatment required involving pediatric ingestions of the unknown.
Methods: This IRB approved retrospective chart review of a regional poison center (RPC) involved children 6 years with acute exposures to "unknown" drug substances from January 2000 to December 2017. Basic descriptive statistics were performed to analyze the data.
Results: Over an 18-year period, 1453 "unknown" drug substance exposures were reported to our RPC involving children 6 years. Boys were involved in 51%, similar to overall trends for ingestions in this age. The most common exposure site was the patient's own residence (79%), followed by other residences (11%) with the remaining noted to be in public areas, hospitals, schools and worksites (6%). The top reason for the exposure was unintentional (92%). Only 649 (45%) cases were successfully followed to a known outcome. Of those with known outcome, no effect cases totaled 356, minor 144, moderate 118, major 30. There was only one reported death. Management site was HCF in 983 cases (67%); 328 (23%) were managed at home. Out of the 983 patients managed in a hospital, our RPC recommended specific observation times in 492 cases (50%). The most common obs time was 24 hrs (300 cases), followed by
Discussion: Unknown medications continue to pose difficult decisions for poison center triage. Guidelines need to be better defined and tailored to individual circumstances, however, recommending a standard observation time >12 hours may reflect safer PC practices and may improve outcome data. Factors likely to discriminate between the likely minimal toxicity circumstances to those posing higher risk need to be better understood. The inconsistency of coding and follow-up practices of the reporting poison center should also be taken into consideration.

KEYWORDS Unknown substance, pediatric toxicity, poison center

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170. Death Averted: A Case of Polyurethane Aspiration

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Background: Spray foam insulation is formed from the polymerization of polyurethane (PU) compounds, and is commonly used to fill, seal, and repair cracks around the home. On average, US poison centers (PC) are notified about 500 cases per year involving PU foam products, mostly accidental pediatric exposures. Common routes of exposure include dermal, ingestion, and inhalation. The only case report describing an intentional oral exposure to PU foam describes a fatal outcome due to aspiration. We report an intentional exposure to PU foam with survival.
Case Report: A 57-year-old female presented to the emergency department after an intentional effort at self-harm by spraying a PU foam insulation product into her mouth. She presented tachycardic, hypertensive, tachypneic, with decreasing oxygen saturation. She was complaining of mild difficulty swallowing, drooling, and shortness of breath. Per exam, the foam was noted to be "hard as a rock" and visible in the nostrils and the right posterior oral pharynx. Respirations were labored, with moderately decreased breath sounds on the left lung. An initial chest radiograph showed a clear right lung with evidence of possible decreased lung volume on the left. The patient was eventually intubated before a nasal endoscopy was performed. Insulation material that was solidified from the nasal cavity, as well as some pieces from oral cavity, were removed. Bronchoscopy showed white colored insulation material in the left main bronchus obstructing the left mainstem completely at the level of primary carina. Insulation material was present in the right upper lobe completely obstructing this area as well.

The patient was admitted to ICU for recovery and was subsequently extubated 48-hours later. A repeat bronchoscopy performed 4 days later resulted in further fragment removal. Due to an acute onset of abdominal pain, the patient had an abdominal CT, which showed pieces of foam product still in her esophagus and stomach 4-days post exposure. Following unsuccessful use of olive oil in an effort to facilitate elimination of the hardened foam, a gastrostomy was required to facilitate removal of the spray insulation from the stomach. Lungs subsequently remained clear until discharge. There was no evidence of systemic effects from the chemicals in the insulation.
Discussion: Most PU foam products contain diisocyanates and polymerizing agents. On contact, these products can be irritating to the skin, and harden quickly on exposure to air. Inhalation of fumes can produce respiratory irritation. Obstruction can result from lodged polymerized foam.
Conclusion: This case of an intentional PU foam aspiration presented with unique challenges not often experienced by emergency medicine and poison center personnel. According to national poison center data and cases reported in medical literature, it is uncommon to attempt to self-harm with a spray foam insulator. There is little evidence describing systemic toxicity of these chemicals; however, the sheer amount of occlusive material in the lungs, nose, and stomach proved challenging but ultimately resulted in her survival.

KEYWORDS Polyurethane, aspiration, poison center

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171. Poison Center identification of point-of-sale naloxone availability in a seven county metro area

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Background: Expanding naloxone availability remains an important component of harm reduction as opioid exposures and overdoses continue to increase. A recent study has demonstrated low, variable naloxone availability in urban areas of a single state (Lozo et al 2019). We sought to characterize naloxone availability at the point of sale across a large midwestern metropolitan and surrounding area.
Methods: All pharmacies within a 7-county metropolitan area were identified via the state department of administration. From this a random sample (150/569) of pharmacies was generated using Microsoft Excel (2013). The sample was geospatially mapped using ArcGIS 10.7 (esri 2019) and distributed evenly among seven investigators (5 female, 2 male; 1 emergency medicine resident, 4 specialists in poison information, 1 medical toxicology fellow, 1 medical toxicologist) who approached pharmacy counters in person to inquire, "May I purchase naloxone here without a prescription from my doctor?" Following an answer of "no," the response to a follow-up question, "Are you aware of the state statute that allows you to dispense naloxone to the public under a standing order?" was recorded. Responses were recorded and uploaded in real time to a RedCAPTM database using RedCAPTM Mobile App. The governing human subjects review board identified this study as qualifying for exempt status.
Results: Of 150 pharmacies, 15 were excluded a priori as specialty pharmacies serving limited roles (infusion, mail-order, veterinary, other). Of the remaining 135 pharmacies, we identified three over-represented chain pharmacies (>10 survey sites each), and replaced 1/3 of each with independent pharmacies from the randomly generated list, without reference to name or geospatial distribution. We approached 134 pharmacies, of which 36 were not surveyed: 22 (61.1%) were closed; 8 (22.2%) were not retail pharmacies; 2 each (5.6%) required membership to gain entry, were no longer at the identified location, or were not approached for another reason. Pharmacies were widely distributed, with more rural areas served by independent pharmacies than chain pharmacies. Of 98 pharmacies successfully approached, 71 (72.5%) were capable of dispensing naloxone without a patient-specific prescription (Figure 1). Chain pharmacies were more likely than independent pharmacies to report naloxone availability (84.7% v. 38.5%, Chi2 20.48, p = 0.000; Figure 2). Ten pharmacies (37%) were unaware of the state statute allowing for dispensation of naloxone under a collaborative agreement or physician's standing order; these were divided evenly among chain and independent pharmacies. No significant difference in awareness of statutory support was evident between independent and chain pharmacies (Chi2 0.564, p = 0.453). Reported rationale for availability of naloxone included no physician collaborator, no consumer demand, no product stocking plan, and refusal to provide the medication.
Conclusion: Point of sale naloxone availability is widespread in this metropolitan area. Significant variability in point of sale naloxone availability exists between chain and independent pharmacies, and among pharmacies of the same chain, although awareness of statutory guidance does not. An opportunity exists for Poison Centers to define the true availability of point of sale naloxone within their region through a boots-on-the-ground approach, while educating pharmacy providers and effecting change throughout their communities.

KEYWORDS Naloxone, availability, opioid

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172. Incidence and Epidemiological Profile of Pediatric Chemical Burn Injuries in a Tertiary Pediatric Hospital

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Introduction: according to data from the American Burn Association (ABA) in United Estates 486,000 patients with burn injuries receive medical treatment annually, 100,000 being children. Chemical burns represent 3% of pediatric burns. Chemical burns are a cause of morbidity and serious health outcomes by association with systemic toxicity. The majority of cases occur in patients younger than five years old. Common causes include acids, Bases, Oxidants, Vesicants, organic compounds and Miscellaneous etc. Chemical burns can cause damage by different chemical reactions, depending on the chemical agent to which the patient is exposed. The most common injuries are caustic ingestions, eye injuries, and cutaneous chemical burns. There is limited information of the incidence of chemical burns in our pediatric population. The aim of this study was to evaluate the incidence and characteristics of chemical burns in pediatric patients evaluated in a tertiary pediatric hospital.
Methods: A cross – sectional, descriptive record review was done on patients presenting chemical burns who were evaluated and/or admitted to a pediatric tertiary hospital over an 8 – year period, from 2010 – 2017. Data including patient demographics, affected body region, depth of burns, etiological agent, percent of total body surface area, and management were collected and analyzed.
Results: During the study period, a total of 1237 patients were evaluated due to burns, including 41 cases of chemical burns for an incidence of 3.3% of total pediatric burns. They were most common in

male patients (58.5%) with a mean standard deviation of 3 ± 4 years old. Skin lesions were mostly second degree (85.37%) with a total body surface area of 4 ± 6 %, affecting multiple body regions (45%). The most common etiological agent was nail primer (39.02%) followed by intravenous fluid infiltration (21.95%), and bleach (9.76%) other agents include sulphuric acid and sodium hydroxide. Injuries most commonly occurred in domestic settings (68.29%). 34 patients (82.93%) required hospital admission with a mean length of stay of 6 ± 5 days. Regarding management, in most cases patients were treated with closed advanced dressings (29.27%), other patients were managed with multiple modalities or ophthalmological management.

Conclusion: Pediatric chemical burns have a high incidence in our population when compared to prior studies elsewhere. These lesions most commonly occurred in the domestic setting due to accidental exposure to chemicals. Most patients required hospital admission for specialized care with an associated risk for other complications, including potential disability. Even though this is not the most common cause of burn injuries, it is important to be aware of the incidence and mechanisms of injury in our population to ensure adequate management protocols and to deliver proper prevention strategies to parents and caregivers to minimize these uncommon, but preventable lesions.

KEYWORDS Chemical burns, epidemiological characteristics, pediatric burns

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173. Extended release acetaminophen overdose: a case series

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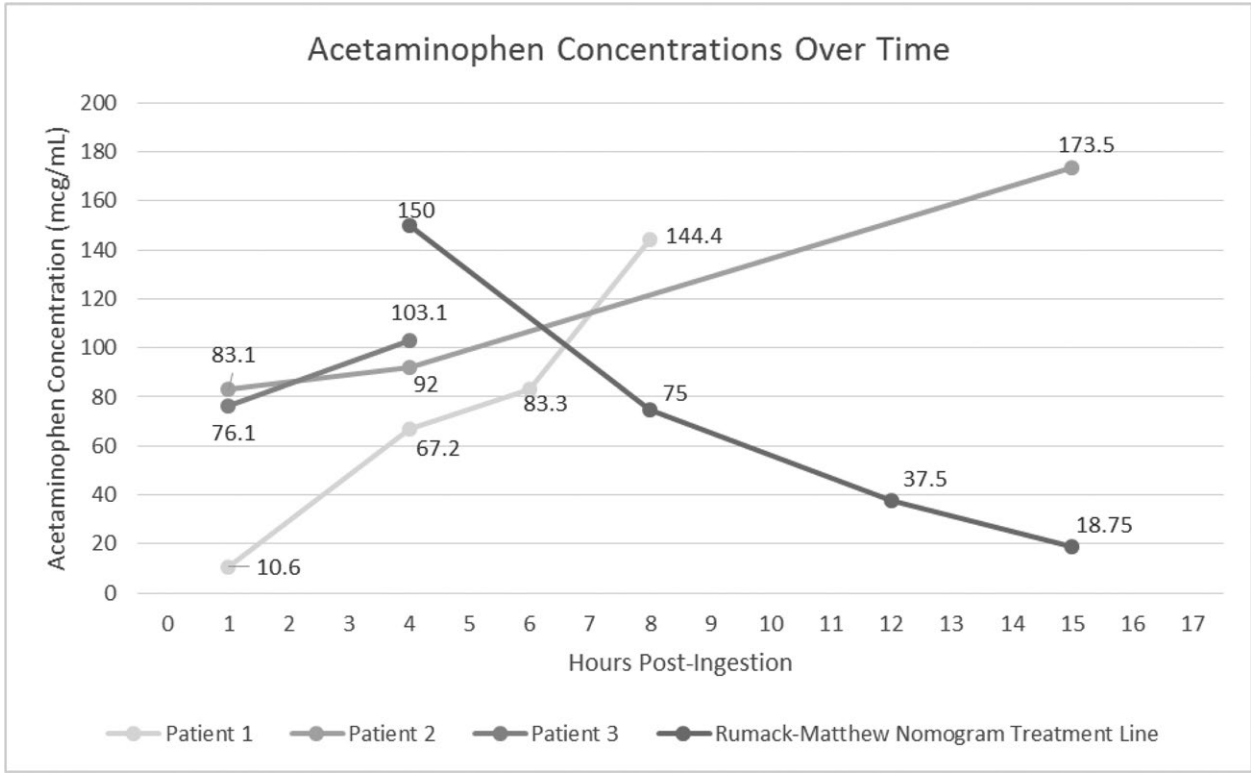
Background: Treatment of short acting acetaminophen overdose is standardized with the Rumack-Matthew nomogram. Overdose of extended release acetaminophen (APAP) is less well understood as the formulation has variable rates of absorption and unpredictable pharmacokinetics.

Case reports: We present three patients who overdosed on extended release acetaminophen. Patient one was a 26-year-old female who overdosed on extended release APAP with rising APAP concentrations at four, six, and eight hours (67.2, 83.3, and 144.4 mcg/mL, respectively). With this, n-acetylcysteine (NAC) was initiated when the 8-hour concentration returned. Patient two was a 19-year-old male who overdosed on extended release APAP 65 grams. He was given activated charcoal every two to three hours for three doses, and NAC was initiated at two hours post-ingestion. This patient an increase from the one-hour to four-hour APAP concentrations (83.1 mcg/mL at one hour, then 92 mcg/mL at four hours), and the APAP concentration at 15 hours was 173.5 mcg/mL. With this, NAC 100mg/kg was administered for an additional 16 hours. With the increased concentration at 15 hours after time of ingestion, an additional dose of charcoal was also administered for presumed continued absorption from the massive OD. Patient three was a 20-year-old female who overdosed on extended release APAP. Her one-hour APAP concentration was 76.1 mcg/mL and the four-hour APAP concentration was 103 mcg/mL. She was given charcoal at two hours after ingestion. In all cases, NAC was administered intravenously 150 mg/kg over one hour, then 50 mg/kg over four hours, then 100mg/kg over 16 hours. Figure 1 shows APAP trends with time with each patient.

Case discussion: All three patients were below the treatment line initially, but later crossed the treatment line on the Rumack-Matthew nomogram. Based on previous case series as well as the cases presented here, it is possible for patients with four-hour APAP concentrations to have rising APAP concentrations crossing the treatment line on the Rumack-Matthew nomogram and require NAC.

Conclusions: It is likely not enough to stop with a four-hour APAP concentration if it is below the treatment line on the nomogram and if the concentrations are rising in a patient who ingested extended release acetaminophen. It is our recommendation that patients who overdose on extended release APAP get repeat APAP concentrations at least at four and eight hours after ingestion, or at two-hour intervals until concentrations are decreasing. We would recommend this regardless if it if falls above or below that treatment line on the Rumack-Matthew nomogram as this nomogram was created using the



immediate release APAP, and APAP ER appears to follow different pharmacokinetics and toxicokinetics.

	Patient 1	Patient 2	Patient 3	Rumack-Matthew Nomogram Treatment Line
0				
1	10.6	83.1	76.1	
2				
3				
4	67.2	92	103.1	150
5				
6	83.3			
7				
8	144.4			75
9				
10				
11				
12				37.5
13				
14				
15		173.5		18.75
16				
17				
18				
19				
20				
21				
22				
23				

KEYWORDS Acetaminophen, extended release, n-acetylcysteine

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174. Misinterpretation of carbon monoxide reference range leading to mass public concern

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Background: Misinformation can spread rapidly through social media outlets leading to misguided public response to potential exposures. This may lead to unnecessary testing, significant distress to the public, and burden to the healthcare system. Poison control centers (PCCs) are often the initial contact point for environmental exposures, making them uniquely suited to properly interpret and communicate risk. Despite being a free public resource, familiarity with and utilization of PCCs is inconsistent. We describe a case of mass public concern for carbon monoxide (CO) poisoning in a small town, reinforcing the need for improved public awareness of PCCs as a community resource.
Case: During a school day, several children and staff at an elementary school noted noxious odors from a malfunctioning boiler and developed symptoms of fatigue, headache, nausea, and vomiting. No CO detector alarmed and all were found to be functioning properly. The fire department detected no elevated CO with independent equipment. Children returned to class. The odor recurred four hours later. During this second event, several parents utilized social media to rapidly disperse information and organize removal of children from school to an emergency department. All tested children had detectable CO concentrations ranging from 2-4%. The lab analyzer listed. This perceived CO exposure caused mass distress among parents. Treatment variability created significant confusion within the community. Parents reported their concerns to local news organizations. Subsequently, >25 separate online news articles were published in 24 hours, reporting 14 children with CO poisoning. The following day, the public health department contacted the PCC for clarification and education regarding CO exposure. We generated a plan for evaluation of the odors and to issue a statement to regional medical providers. Only one news agency contacted the PCC for clarification of CO concentration interpretation. A review of the poison center database revealed no reported cases of CO toxicity related to this event.
Discussion: This case highlights the importance of the PCC in risk communication to the public regarding potential toxic exposures. Unfortunately, not all medical providers, government agencies, and news resources understand the role of PCCs in the healthcare system. While many healthcare providers and civilians utilize PCCs appropriately, this case demonstrates that further education and outreach are needed on behalf of PCCs to avoid similar detrimental events in the future.
Conclusion: PCCs should be utilized in potential mass poisonings and can help guide appropriate use of the health care system and public health resources. Further outreach and educational campaigns are needed on behalf of PCCs to attenuate similar events in the future.

KEYWORDS Public Health Outreach, Risk management, Carbon monoxide

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175. A Case of Inorganic Mercury Toxicity with Good Short-term Clinical Outcomes

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Background: Inorganic mercury poisoning is rare but can have a potentially severe and life-threatening course. While aggressive resuscitation and chelation are considered the treatments of choice, the role of decontamination is unclear. We report the case of a significant voluntary ingestion in the context of a suicide attempt, which presented with relatively low toxicity following early interventions.
Case report: A 27 year old male reported having ingested 2 grams of mercury sulfate in an attempt to self-harm. He obtained the substance from his workplace and drank it after dilution in a soda beverage. He arrived in the emergency department lethargic, but hemodynamically stable about 2 hours after the ingestion. He had vomited during transport with the emergency medical services and passed one large loose watery stool on presentation. He complained of his "insides burning" and had copious oral secretions. The patient received aggressive rehydration. Whole bowel irrigation was started after intubation. Dimercaprol (British anti-Lewisite) was the chelator available at the time and the first dose was given 6 hours after ingestion. The following gastrointestinal toxicity was remarkable only for very small amounts of blood in stools on the third day. He remained hemodynamically stable and was also extubated on day 3. Renal function remained good. Other clinical effects observed included one seizure, fever, mild hypoglycemia, mild hypokalemia and a transient small elevation of transaminases. Dimercaprol was discontinued on the sixth day as a good clinical course was noted. Patient was asymptomatic without significant laboratory anomalies. There was a significant delay in obtaining mercury levels. A whole blood mercury level of 3392.8 nmol/L (680.6 ug/L) drawn 2 hours after ingestion was reported on the ninth day of hospitalization. A repeat level from day 3 of hospitalization showed a significant decrease to 683.9 nmol/L (137.20 ug/L). Again, this value was delayed. Our patient was lost to follow up as he left the hospital and the country.
Case discussion: We describe the case of a patient who presented with relatively low toxicity despite the significant amount of inorganic mercury reportedly ingested. For mercuric chloride, doses reported to

cause fatality generally range from 1 to 4 g, although death has occurred following ingestion of 0.5 g. It is unclear if decontamination through vomiting and whole bowel irrigation may have limited gastrointestinal toxicity and absorption. Importantly, the patient did not develop significant hemodynamic instability, nor renal failure. Empiric chelation with dimercaprol was necessary because of the delayed laboratory reporting. It significantly decreased the mercury levels and was well tolerated. The observed short-term outcomes were good but our patient was lost to follow up which limits our analysis of the long-term clinical outcomes.
Conclusion: Inorganic mercury toxicity can be associated with a significant morbidity and mortality. A rapid and aggressive management may limit the toxicity and lead to good outcomes. Note : A more detailed description of mercury levels and laboratory anomalies will be available on poster.

KEYWORDS Mercury poisoning, Mercury sulfate, Inorganic mercury

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176. Assessing knowledge of zoanthid coral-related palytoxin risk amongst commercial aquarium shops and enthusiasts

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Background: Zoanthid corals, a source of palytoxin, are commonly traded amongst aquarium enthusiasts. There has been an increase in cases of palytoxin exposure reported to poison centers. Guidelines from the Ornamental Aquatic Trade Association (OATA) provide information on safe handling of these corals. The extent to which the public understands risks and safe handling practices related to zoanthids is unknown. We aimed to assess the knowledge of palytoxin risk from zoanthids amongst commercial aquarium shop managers and enthusiasts.
Methods: An internet search identified aquarium stores in cities with populations greater than 60,000 in our poison center's catchment area. Three pet chain stores were excluded from our study following confirmation that they did not sell live corals. Aquarium enthusiast groups were identified through an internet search and access to online forums was requested. A five-question survey was presented to two groups: stores that sold live corals and aquarium enthusiasts.
The following questions were asked:

1. Do you sell zoanthid corals?
2. Are you aware that zoanthids can secrete palytoxin?
3. Do you provide information to staff or customers about risks from zoanthids, and require the use of personal protective equipment (PPE) when handling?
4. Do you provide cleaning instructions to employees and customers for aquariums containing zoanthids?
5. Are you aware that OATA has guidelines for safe handling of zoanthids?

Store managers were presented the survey via telephone. Aquarium enthusiast groups within the same cities were identified and a survey link was posted to their online communications board. Free text options for each question allowed for elaborated responses. The survey was open for a 2-month period, with a reminder sent one week after posting the initial survey.
Results: Aquarium shop managers:Nineteen stores from eight cities were invited to participate in the survey, with a 79% response rate (15/19). Eight of fifteen (53%) stores that responded sold zoanthids and all claimed awareness of palytoxin risk. Six of 8 stores (75%) reported sharing information with staff and customers about risks and four stores (50%) suggested PPE when handling. Four stores (50%) provided cleaning instructions to customers. Six shops (75%) claimed palytoxin is common knowledge and expect customers to do research prior to purchase. Two stores claimed awareness of OATA guidelines. Aquarium enthusiasts: Fifty-five enthusiasts responded to the survey. All bought or traded zoanthids and were aware of palytoxin. Twenty of 55 (36%) received information from retailers about risks associated with zoanthids. Five respondents (9%) received cleaning instructions for tank maintenance. Free text responses suggested many did their own research prior to purchasing. Others stated they learned of risk post- exposure, or from fellow enthusiasts.
Conclusion: A majority of consumers purchasing zoanthids are not receiving information about appropriate PPE or clean-up procedures to avoid palytoxin exposure. Awareness of palytoxin amongst aquarium shops exists, however, information is not consistently communicated to customers. Learning of risk post-exposure is problematic and aquarium stores should consider distributing OATA guidelines at the point of sale to reduce palytoxin exposure.

KEYWORDS Palytoxin, Natural Toxins, Public Health

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177. Managing Methemoglobinemia in the Resource-Limited Setting: a Case Report from Nepal

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Background: Methemoglobinemia is a relatively uncommon but potentially significant disease characterized by increased production of methemoglobin.The workup and treatment of methemoglobinemia in well-resourced clinical settings typically includes co-oximetry for methemoglobin percentage and methylene blue as an antidote. Unfortunately, exposure to oxidizing agents that cause methemoglobinemia occurs in resource-limited clinical settings where the work up and treatment is not straightforward. We present a case of severe methemoglobinemia induced by nitrobenzene exposure which was successfully treated with adjunctive therapies in a resource-limited hospital in Nepal.
Case Report: A seventeen-year-old male presented to a semirural emergency department (ED) in Nepal after reportedly ingesting a 500 mL of "Ki-flower," a nitrobenzene-containing fertilizer. Upon arrival to the ED, the patient was anxious and confused, complaining of mild dyspnea; his vital signs included a systolic blood pressure of 120mmHg, a heart rate of 100 beats per minute, a respiratory rate of 24, and pulse oximetry of 84% on room air. Approximately one hour into his clinical course, he developed worsening confusion, cyanosis, and respiratory distress. His respiratory rate increased to 55 and his blood pressure decreased to 80s/50s. The only laboratory analysis available was an arterial blood gas (ABG) drawn upon arrival. Two hours into the patient's course the ABG demonstrated a pH of 7.46, PaCO2 of 25 mmHg, and PaO2 of 28 mmHg. The blood drawn appeared "chocolate brown" in color. Neither methemoglobin percentage nor methylene blue were available. The patient was intubated, received intravenous fluids, and underwent gastric decontamination via nasogastric tube. Additionally, he received oral riboflavin and ascorbic acid (intravenous formulations were unavailable) for seven days. Simple single volume exchange transfusions were performed by sequential removal of patient blood and transfusion of whole blood (packed red blood cells were unavailable). A total of five units were transfused. After a week in the hospital the patient was discharged home with baseline neurologic function.

Case Discussion: Despite the lack of a methemoglobin percentage, a clinical diagnosis of methemoglobinemia was made. A known, large ingestion of nitrobenzene, a persistently mid-eighties oxygen saturation despite supplemental oxygen administration, blood with a chocolate brown discoloration, and the clinical course were consistent with severe methemoglobin toxicity. Although its efficacy in the setting of methemoglobinemia is unknown, riboflavin works similar to methylene blue in the NADH-dependent methemoglobin reductase enzyme pathway by transferring electrons to ferric ions. Ascorbate is sometimes used in the treatment of congenital methemoglobinemia. Typically, it is not used in the treatment of acquired methemoglobinemia due to its slower reduction rate compared to other intrinsic enzymatic systems. Given the patient's degree of instability and our limited options, we opted to use these therapies in addition to exchange transfusion and decontamination.

Conclusion: A hemodynamically unstable patient with apparent nitrobenzene-induced methemoglobinemia was successfully treated with adjunctive therapies including riboflavin, ascorbic acid, and simple exchange transfusion in a resource limited hospital in Nepal. It is important for providers practicing in resource limited clinical settings to be aware of adjunct therapeutic options if required to treat a patient with methemoglobinemia.

KEYWORDS Methemoglobinemia, Resource-limited, Nepal

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178. Successful use of ECMO and lipid emulsion for massive bupropion overdose

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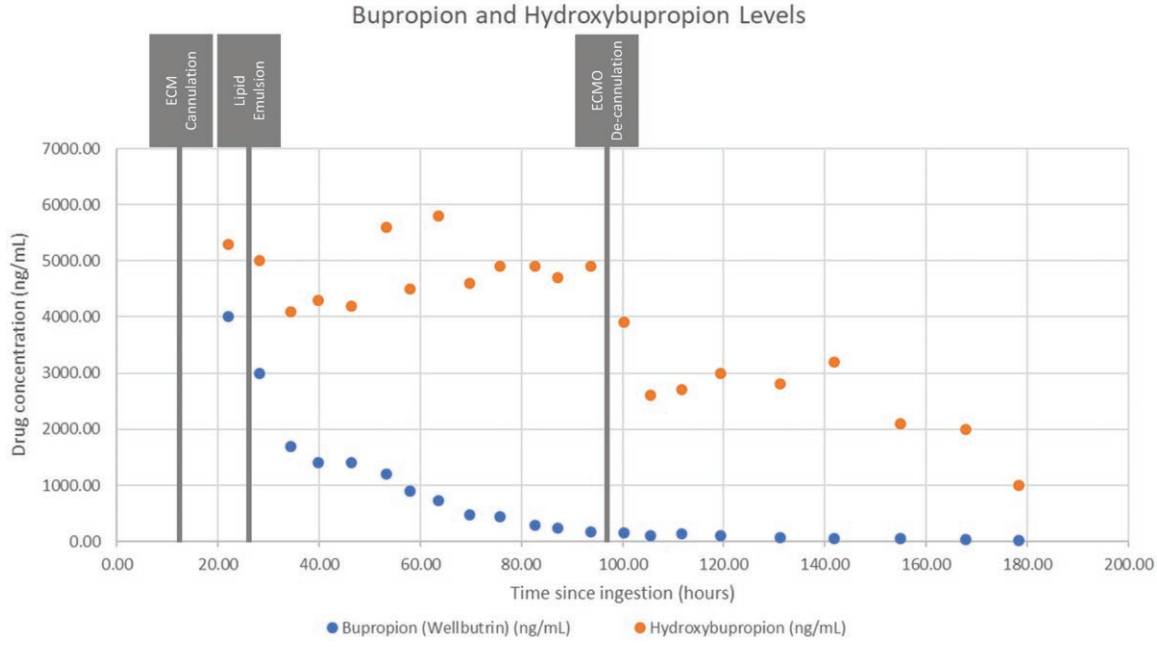
Background: Bupropion is a commonly ingested medication, with 14,204 single substance exposures reported to poison centers in 2017. Ingestions of bupropion may cause hemodynamic collapse or generalized tonic-clonic seizures. Lipid emulsion and extracorporeal membrane oxygenation (ECMO) have been used to treat symptomatic bupropion ingestions, but there are conflicting reports as to whether lipid emulsion disrupts the ECMO circuit. We report a case of a patient who was successfully treated with lipid emulsion and ECMO following an overdose of 644 mg/kg of bupropion. In this case, intralipid did not interfere with the ECMO circuit. Our report also describes the treatment of the highest reported nonlethal bupropion level.

Case Report: A 22-year-old female presented to an outside emergency department hemodynamically stable and neurologically intact 1 hour after ingesting a 90-day supply of 450 mg bupropion extended release tablets (estimated total bupropion dose 40.5g). She experienced 4 generalized tonic-clonic seizures, received 12 mg IV lorazepam, started on norepinephrine due to hypotension, and was intubated and transferred to our facility. On arrival to our hospital her EKG showed normal sinus rhythm with a QTC of 597 msec and a QRS of 162 msec with frequent PVCs. The patient's cardiac rhythm devolved into torsades de pointes, then pulseless electrical activity. Return of spontaneous circulation (ROSC) was achieved following chest compressions, epinephrine, magnesium, and sodium bicarbonate. She continued to be hypotensive despite norepinephrine, dopamine, phenylephrine, and methylene blue. The patient was cannulated for venous-arterial (VA) ECMO. In tandem, the patient experienced recurrent ventricular tachycardia despite 13 defibrillations, electrolyte repletion, amiodarone, and lidocaine. Given the refractory ventricular tachycardia, a bolus of 1.6 g/kg of 20% lipid emulsion was given. Subsequently, the patient needed only one more defibrillation. Bupropion and hydroxybupropion serum levels were obtained every 6 hours starting 20 hours after ingestion. Her initial bupropion level was 4000 ng/mL and hydroxybupropion level was 5300 ng/mL. The patient's neurologic status improved over the next week such that she was extubated and found to be verbally interactive, and fully oriented. This patient was safely and effectively supported with VA ECMO for 84 hours. There were no adverse effects noted with the ECMO machine or tubing related to the lipid emulsion. There were not clots in the ECMO circuit or oxygenator and the circuit did not need to be exchanged for any reason, nor were cracked stopcocks reported.

Case Discussion: Despite multiple reports of problems associated with the combination of ECMO and lipid emulsion, this case demonstrates the safety and efficacy of these therapies for cardiovascular collapse secondary to bupropion toxicity. The occurrence of tachydysrhythmias requiring electrical defibrillation decreased following the



administration of 20% lipid emulsion. We also report the highest nonlethal serum bupropion level at 4000 ng/mL, as well as the most extensive course of drug metabolism and excretion with 22 bupropion and hydroxybupropuion levels each.
Conclusions: This bupropion overdose demonstrated the highest nonlethal serum bupropion level (4000 ng/mL) that required the concomitant use of ECMO with 20% lipid emulsion therapy, without complications to the patient or ECMO circuit.

KEYWORDS Bupropion, ECMO, Lipid emulsion

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179. Naloxone rescue kits in the setting of syringe exchange services: Guess who is saving lives with 0.4 mg?

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Background: Naloxone rescue kits used by non-medical laypersons have been documented to save thousands of lives nationwide since equipping community members and laypersons began over 2 decades ago. One of the most successful access points for placement of layperson naloxone kits has been in the setting of syringe exchange. Partnerships that lead to naloxone being placed directly in the hands of people who use drugs have demonstrated important strides in preventing preventable deaths, and gives us frontline information about appropriate naloxone dosing for those using the substances we often know the least about.
Objective: To describe the reported use of intramuscular (IM) injectable naloxone rescue kits (containing 0.4 mg/ml doses of naloxone) within a population of layperson participants in one syringe exchange services (SES) program.
Design/Methods: Anonymous self-reporting of the use of a naloxone rescue kit included: the number of doses/vials of 0.4 mg naloxone used to achieve a reversal of an opiate overdose, who a kit was used on, if EMS was called, and if the individual survived. Kits provided to participants each contain 2 doses of 0.4 mg naloxone vials and 2 syringes. Participants in SES are able to obtain multiple kits if desired. Reversal data was collected anonymously and reported to a staff member of the SES.
Results: 300 individual reports of naloxone rescue kit use were documented over 26-mos (02/17-04/19). Kits had been furnished during SES outreach services by one community-based entity. 97% (291) of the reports describe that an individual had a successful reversal and survived. The reported use was on a friend/acquaintance (76%), self (10%), family member (7%), stranger (6%), spouse (1.3%), or other (0.05%). One dose of naloxone (0.4 mg IM) was used to reverse an overdose in 26% (77) of the reports, two doses 56% (168), 3 doses 11% (32), 4 doses 3% (10), 5 doses 1% (2), 6 doses 1% (3), unknown doses 3% (8). There were 7 unsuccessful reversal reports during this time period using 2,1,2,2,4,2,3 vials of naloxone. EMS was called 53% of the time a layperson kit was used in this setting.
Conclusions: Individuals participating in a syringe exchange services program self-reported use of naloxone rescue kits that had been furnished to them. Most of the kits were reported used on a friend, family member, or on the participant themselves. 97% of those receiving naloxone in this setting survived. Over 82% of the reversals were reported successful with 1 or 2 doses of 0.4 mg IM injectable naloxone. There is no indication from these results that an increased dose of naloxone is required or needed for equipping laypersons. These results do suggest that individuals in this setting should have access to multiple kits or kits with at least 3-4 doses given that over half of the reports did not include a call to EMS. Increased education about the role of EMS as well as ensuring individuals in this setting have access to multiple kits/doses is recommended. People who use drugs are saving the lives of those around them.

KEYWORDS Naloxone, opioid overdose, overdose death prevention

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180. When Melatonin is No Longer Considered the "New GHB."

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Background: Melatonin is a hormone produced by the pineal gland. Secretion in a constant pattern is essential in maintaining the sleep-wake cycle. Melatonin as a sleep aid is available in a variety of forms for sale over-the-counter. Pediatric unintentional ingestions of melatonin have become more frequently reported to poison centers (PC). Literature described ingestions of 3 to 80 mg have resulted in minimal toxicity in children. Our aim is to report the incidence and outcomes of exposures involving melatonin in children as reported by our PC.
Methods: This is an IRB approved retrospective chart review from January 2004 to December 2018, analyzing children 6 years with a single acute ingestion of melatonin. Other inclusion criteria required a historical amount of melatonin ingested and a documented outcome.
Results: Over a 15-year period, 4638 cases of pediatric melatonin cases were reported to our RPC. On average, there was a 30% rise in pediatric melatonin cases each year during the study period, from 27 cases in 2004 to 992 in 2018. Only 208 cases met all inclusion criteria (4.4% of all cases). For all included exposures, the average age was 2.5 years with 53% females. There were 21 cases describing exact known amounts ingested (10%), 75 estimated amounts (35.9%), and 113 were described as maximum possible (54.1%). The known exact amounts ingested ranged from 0.5 mg to 300 mg, with a possible maximum amount of 680. The most common symptoms reported were mild CNS depression (43.8%, ranging from max 2.1 mg to max 680 mg), and agitation (4%, ranging from 1 mg to max 680 mg). No effects were reported in 112 (53.8%) of the cases. There were 46 cases in which mild CNS depression was reported when the ingested dose 80 mg (22.1% vs 21.6%, $p = 0.91$). Out of 208 cases, 108 (51.7%) were treated at home and 95 (45.5%) were evaluated in a hospital. Of those treated in a hospital, 6 were given activated charcoal for ingestions averaging 117 mg. The remaining 89 were observed with no further therapeutic intervention necessary. No hospital admissions were reported as all were discharged after a few hours of observation. The average dose ingested managed onsite was 147 mg compared to the average dose ingested in which hospital triage was recommended was 146 mg.
Conclusions: Fifteen years of data from our RPC suggests that with an unintentional ingestion of melatonin in children less than 6 years of age, even for possible maximum ingestions up to 680 mg, no serious toxicity is expected and referral to a healthcare facility is not likely necessary. Until further studies are concluded, any cases with more than minor symptoms should continue to warrant HCF referral and evaluation. The inconsistency of coding and follow-up practices of the reporting poison center should also be taken into consideration.

KEYWORDS Melatonin, pediatric, poison center

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181. Unusual Mechanism of Carbon Monoxide Poisoning

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Background: Inhalation of carbon monoxide continues to be a common method used in attempted suicide. As a by-product of combustion, carbon monoxide is found commonly among household products including car exhaust, gas burning furnaces, and portable generators. Less commonly, and scarcely reported in the literature, is the formation of carbon monoxide from the mixture of formic acid and sulfuric acid. Chemical suicide attempt, carried out by combining chemicals in a container in a closed environment to create a toxic gas, are an emerging toxicologic problem. Typically these events involve a combination of chemicals, including sulfuric acid that release hydrogen sulfide. Sulfuric acid, however, may also be combined with formic acid, to produce carbon monoxide. Here we report such a case.
Methods (case report): A 29 year old male presented to the emergency department with nausea, vomiting, and headache. Initial vitals were reported as blood pressure 134/74, heart rate 86, and respiratory rate 20 breaths per minute. His neurologic exam was unremarkable. The patient's significant other found him minimally responsive in a pile of vomit; next to the patient was a bottle of formic acid and another bubbling liquid, confirmed to be sulfuric acid. For unclear reasons, the patient's significant other was concerned for carbon monoxide poisoning so the physician checked a carboxyhemoglobin (COHb) level which returned at 28%. He was placed on 100% oxygen by non-rebreather mask. The patient also reported that he ingested this combination of liquids, however he had no evidence of oral irritation or burns. Initial troponin was negative; EKG revealed normal sinus rhythm. The hyperbaric medicine service was consulted who elected to medically observe patient since his exam has been normal and patient was doing well clinically. The repeat COHb level ~5 hours later was 4.2%. The patient's symptoms improved within that time as well. Vitals continued to be stable throughout hospitalization and labs remained unremarkable including cardiac workup.
Results (case discussion): Carbon monoxide (CO) poisoning due to combustion of formic acid and sulfuric acid has been rarely reported in the literature. The chemical reaction by which this occurs is below.
CH_2O_2 (formic acid)+H_2O_4S (sulfuric acid)→CO (carbon monoxide)+H_2SO_4•H_2O
Both of these acids with ingestion are known to cause immediate severe oral and gastrointestinal burns making the ingestion of this product unlikely for our patient. The patient's carbon monoxide level decreased as expected due to the decrease in half-life from 5-6 hours to 1-2 hours with 100% oxygen by tight fitting mask. If the patient had a higher COHb level or more significant symptoms, he likely would have been treated with hyperbaric oxygen therapy.
Conclusion: The mixture of formic acid and sulfuric acid forms carbon monoxide which can lead to severe toxicity. The treatment includes 100% oxygen and could potentially involve hyperbaric oxygen therapy depending on severity. This case highlights the potential risks to patients and first responders of carbon monoxide due to a unique mechanism.

KEYWORDS Carbon monoxide, carboxyhemoglobin, chemical suicide attempt

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182. Adherence to Poison Center Hemodialysis Recommendations for Lithium Poisoning

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Background: When to perform hemodialysis in lithium poisoning remains a subject of debate. Whether hemodialysis changes outcomes in lithium toxic patients is also a matter of controversy. The goal of this study is to identify the rate of adherence to poison center recommendations with regard to dialyzing lithium poisoned patients.
Methods: We performed a retrospective review of hospitalized lithium cases reported to our poison center from January 1, 2016 through December 31, 2018. Cases were abstracted for poison center recommendations regarding hemodialysis (yes/no) and if hemodialysis was performed (yes/no). Cases were included if hemodialysis was either performed or recommended. Cases were excluded if the patient received hemodialysis prior to poison center involvement or dialysis was performed for either a co-ingestant or non-toxicological indication. Rates of intravenous crystalloid administration and fatalities were also abstracted. Proportions were used to analyze the data.
Results: A total of 505 healthcare facility lithium poisoned patients were managed by our poison center over the 3 year study period. Eighty-four cases met inclusion criteria. Sixty-nine patients had specific recommendations for hemodialysis but only 51 (74%) actually received hemodialysis. A total of 66 patients received dialysis for lithium poisoning. Of the patients who received hemodialysis, 15 (23%) were not recommended for hemodialysis by the poison center. Of the 84 cases which met inclusion criteria, 33 (39%) were not managed according to poison center recommendations. All patients were managed with supportive measures including intravenous crystalloid administration. No patient in this cohort died.
Conclusion: A large number of clinicians either did not perform hemodialysis when it was recommended by our poison center, or performed the procedure when the poison center deemed it unnecessary. Further study is required to evaluate clinician non-adherence to poison center recommendations for hemodialysis in lithium poisoning. The reasons for non-adherence, the effect on patient outcomes, and the interplay between toxicology and nephrology recommendations are all areas for future study. Hemodialysis in lithium poisoning remains a controversial therapy.

KEYWORDS Lithium, Hemodialysis, Poison Center

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183. Tramadol Exposures Reported to the U.S. Poison Centers

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Background: There were more than 72,000 overdose-related deaths in the United States in 2017, with 68% of these fatalities involved opioids. Tramadol prescriptions increased by 88% between 2008 and 2013. Tramadol-related emergency department visits involving misuse or abuse of tramadol increasing by 250% between 2005 and 2011. This study aims to examine the national trends in tramadol exposures reported to U.S. poison centers (PCs).
Methods: The National Poison Data System (NPDS) was queried for all closed, human exposures to tramadol from 2012 to 2018 using the American Association of Poison Control Center (AAPCC) generic code identifiers. We identified and descriptively assessed the relevant demographic and clinical characteristics. Tramadol reports from acute care hospitals and EDs were analyzed as a sub-group. Trends in tramadol frequencies and rates (per 100,000 human exposures) were analyzed using Poisson regression methods. Percent changes from the first year of the study (2012) were reported with the corresponding 95% confidence intervals (95% CI).
Results: There were 84,800 tramadol exposures reported to the PCs from 2012 to 2018, with the calls decreasing from 13,113 to 9,599 during the study period. Among the overall tramadol calls, the proportion of calls from acute care hospitals and EDs increased from 53.5%

to 60.9% from 2012 to 2018. Multiple substance exposures accounted for 52.1% of the overall tramadol calls and 62.4% of the calls from acute care hospitals and EDs. The most frequent co-occurring substances reported were benzodiazepines (13.9%) and alcohol (8.9%). Residence was the most common site of exposure (95.7%) and 64.4% cases were enroute to the hospital when the PC was notified. Tachycardia and hypertension were the most frequently demonstrated clinical effects. Naloxone was a reported therapy for 7.9% cases, with this therapy being performed prior to PC contact in most cases. Demographically, 61.2% of cases were females, and the most frequent age groups were 20-39 years (33.1%) and 40-59 years (25.8%). Suspected suicides (45.3%) and intentional misuse (7.8%) were commonly observed reasons for exposure, with the proportion of suicides being higher in cases reported by acute care hospitals and EDs (66.2%). Approximately 18% of the patients reporting tramadol exposures were admitted to the critical care unit (CCU), with 11% of patients being admitted to non-CCU. Major effects were seen in 5.1% cases and the case fatality rate for tramadol was 0.5%, with 416 deaths reported. There were 208 deaths reported within acute care hospitals and EDs during the study period. The frequency of tramadol exposures decreased by 26.8% (95% CI: -28.8%, -24.8%; $p < 0.001$), and the rate of tramadol exposures decreased by 20.7% (95% CI: -29.9%, -1.4%; $p = 0.002$).
Conclusions: PC data demonstrated a decreasing trend of tramadol exposures, which may in part be attributed to the rescheduling of this medication by Drug Enforcement Administration to Schedule IV in 2014. Our study demonstrated a significant proportion of tramadol exposures associated with suicide. Despite an overall decreasing trend in tramadol exposures, there was an increase in tramadol exposure reports from acute care hospitals and EDs during the same time period.

KEYWORDS Tramadol, National Poison Data System, Toxic Exposure Trends

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184. Decreasing Opioid Information Calls to the National Poison Data System

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Background: According to the National Institute on Drug Abuse (NIDA), approximately 130 people die as a result of an opioid overdose every day in the United States (U.S.). Opioid overdoses increased 30 percent from between 2016 and 2017. Poison control centers (PCs) are key in providing clinical management and improving patient outcomes in cases of toxic exposures. PCs have also demonstrated utility in decreasing the healthcare costs by reducing the use of emergency medical services. Apart from the clinical expertise, PCs also provide valuable information regarding drugs including medication identification, dosage, interactions, storage and disposal. This study analyzed the trends in information calls received by the PCs.
Methods: The National Poison Data System (NPDS) was queried for drug information and drug identification calls regarding opioids from 2012 to 2018. Opioid related calls were identified using the American Association of Poison Control Center (AAPCC) generic code identifiers. We descriptively assessed the relevant characteristics. Trends in information call frequencies and rates (per 100,000) were analyzed using Poisson regression methods. Percent changes from the first year of the study (2012) were reported with the corresponding 95% confidence intervals (95% CI).
Results: There were 566,983 drug identification calls reported to the PCs from 2012 to 2018, with the number of calls decreasing from 188,291 to 16,836 during the study period. The frequency of drug identification calls decreased significantly by 91.1% (95% CI: -91.2%, -90.9%; $p < 0.001$), and the rate decreased by 90.3% (95% CI: -90.9%, -89.6%; $p < 0.001$). During the study period, there were 57,434 drug information calls received by the PCs. The number of drug information calls decreased from 16,465 to 4,499, a decrease of 72.9% (95% CI: -73.8%, -72.1%; $p < 0.001$). The rate of drug information calls decreased by 70.7% (95% CI: -74.8%, -65.9%; $p < 0.001$). Among the calls from confirmed sources, drug information calls from emergency medical services providers including rescue squad/dispatcher, EMT, paramedic, hazardous materials team, and police were the most common (87.7%). The most common caller site was the residence (55.4%). Most calls (82.1%) required information or identification about a single substance. Drug identification and information calls regarding oxycodone (39.1%) and hydrocodone (34.1%) were the most common.
Conclusions: Opioid drug information and identification calls drastically decreased during the study period. The PCs, a reliable source of information, are being utilized less for such services. The majority of the confirmed callers for opioid drug information and identification were EMS providers. The proportion of unconfirmed call sources was high with these potentially being from the general public. This marked decreased utilization limits the ability to follow epidemiological trends in various regions through poison center databases.

KEYWORDS Drug Information, Drug Identification, Opioids

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185. National Estimates of Marijuana-related Poison Center Calls

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Background: Marijuana is one of the most frequently used illicit drugs in the United States (U.S.) with 7.3% of the population >12 years reporting marijuana use in the past month. Several U.S. states have legalized and regulated the use of marijuana for recreational purposes. The objective of our study was to evaluate the trends in marijuana calls to the U.S. poison centers (PCs) since these regulatory changes were undertaken.
Methods: The National Poison Data System (NPDS) was queried for all closed, human exposures to marijuana from 01/01/12 through 12/31/18 using the American Association of Poison Control Center (AAPCC) generic code identifiers. We identified and descriptively assessed the relevant demographic and clinical characteristics. Marijuana reports from acute care hospitals (ACHs), emergency departments (EDs), and overall calls including the public were evaluated as a subset. Trends in marijuana frequencies and rates (per 100,000 human exposures) were analyzed using Poisson regression methods. Percent changes from the first year of the study (2012) were reported with the corresponding 95% confidence intervals (95% CI).
Results: There were 49,268 marijuana exposures reported to the PCs during the study period. The frequency of exposures increased by 109.4% (95% CI: 102.5%, 116.5%; $p < 0.001$), and the rate increased by 127.1% (95% CI: 93.8%, 165.9%; $p < 0.001$). Of the total marijuana calls, the proportion of calls from ACHs and EDs decreased from 66.1% to 60.9%, with the percentage of calls from the general public increasing. Multi-substance exposures accounted for 59.2% of the overall marijuana calls and 70% of calls from ACHs and EDs. Approximately 15% of the patients were admitted to the critical care unit (CCU), with 9% of patients being admitted to a psychiatric facility. Residence was the most common site of exposure (83.4%), and 73.5% of these cases were enroute to the hospital via EMS. Cases were predominantly male (58.7%), with the most common age group being 13-19 years (27.1%). The reports for young children under 12 years of age (6.2% to 19%) and older adults above 60 years (3.5% to 7.6%) increased. Intentional abuse (48.1%) was the common reason for exposure, with the proportions of suspected suicides being higher in cases reported by ACH

(20.3% vs 26.9%). The proportion of reported marijuana abuse exposures decreased (50.3% to 35.3%), while unintentional exposures increased (11.4% to 22.4%). Major effects were seen in 5.6% cases and there were 223 deaths reported, with 10 fatalities reported for single substance marijuana exposures. The most frequently co-occurring substances associated with the cases were alcohol (16%) and benzodiazepines (15.7%). Tachycardia (27%) and agitation (17.5%) were commonly observed clinical effects.
Conclusions: Our study results demonstrate a significant increase in the reports of marijuana exposures made to the PCs. The timeline of this study coincides with changes in federal and state laws regarding medical or recreational marijuana use in many states. The exposures in the adolescent age group increased which might be attributed to the unsafe storage practices of adults. Continued surveillance and public health prevention efforts are key to track the population effects of marijuana legalization.

KEYWORDS Marijuana, Intentional Drug Abuse, Epidemiology

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186. What's in a handful? Interpreting a commonly used unit of measurement.

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Background: Patients often quantify ingestions in number of handfuls. However, limited data exists on the number of pills a handful contains. We sought to determine the number of pills in a handful of various common medications.
Methods: Fourty healthy adult volunteers were given bottles of readily available over the counter medications: aspirin (ASA) 81mg tabs, diphenhydramine (DPH) 25mg tabs, acetaminophen (APAP) 500mg tabs, and Vitamin C 500mg tabs. Volunteers were read the following prompt: "You are attempting to overdose on pills, please pour out a handful." Individuals were then asked to place the medications in a container and estimate the number of pills dispensed.
Results: Of the 40 participants, 25 were males (62.5%), 15 were females (37.5%). On average, pill count was underestimated for all medications and the average margin of underestimation was higher in smaller pill sizes. Vitamin C and APAP are larger pills and were estimated more accurately.
The average estimated versus actual number of pills was:
27.1 vs 32.75 pills for Vitamin C (17% underestimate).
40.1 vs 51.75 pills for APAP (23% underestimate).
68.6 vs 106.7 pills for DPH (36% underestimate).
97.7 vs 151.7 pills for ASA (36% underestimate).
An average handful contained 25.9g APAP, 7.9g ASA, 2.7g DPH, or 16.4g vitamin C.
To better assess the variability in estimation accuracy between pill size, we evaluated the standard deviation between actual and estimated number of pills. Paired t-tests were performed comparing ASA, the smallest pill, to all other medications. Overall, individuals were more accurate at predicting the actual number of pills in a handful of larger medications. The size of the pills were as follows: Vitamin C > APAP > DPH > ASA. Comparing ASA with other medications we found all mean differences to be statistically significant.
Mean difference in estimated number of pills between:
ASA and DPH was 54, SD 62.3 versus 38, SD 37.2 (p
ASA and APAP was 54, SD 62.3 versus 11.7, SD 21.8 (p
ASA and Vitamin C was 54, SD 62.3 versus 5.65, SD 11.7 (p
Notably, sample variance represented by standard deviation, was inversely proportional to pill size.
Conclusion: One handful, as estimated by healthy volunteers, can contain toxic doses of common medications. On average, the number of pills in a handful was underestimated, and this margin of underestimation increased with smaller pill size. Pill size correlates to accuracy of estimation, with smaller pills being more difficult to estimate.

KEYWORDS Handful, Overdose, Suicidal

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187. Fatal Overdose of Iron Tablets: A Case Report

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Introduction: Intentional iron tablets overdose in adults is uncommon. Most acute iron toxicity cases occur in children who present with accidental ingestion of iron supplements. We report here a case of severe iron toxicity following intentional overdose of ferrous sulfate that resulted in multiorgan failure and death in an adult female.
Case Report: The patient was a 41-year-old obese female with a history of depression, and gastric bypass surgery that presented to the emergency department (ED) of an outside hospital following a suicide attempt, she ingested an unknown quantity of ferrous sulfate, ibuprofen, and diphenhydramine. On arrival to the ED, her vital signs became irregular with tachycardia, hypotension, and lowered oxygen saturation. She became increasingly lethargic and was intubated. Norepinephrine infusion was started with increasing dose requirements and she was transferred to a tertiary care center. Arterial blood gases showed pH 6.79/ CO2 29, with a lactate of 6 mmol/L and a serum iron concentration (SIC) greater than 2100 mcg/dL. Aggressive resuscitation was continued with IV crystalloid fluid, lung protective ventilation, escalation of vasopressors, and deferoxamine. Despite treatment, the patient proved to be in a state of refractory shock and her clinical condition continued to deteriorate over the next 36 hours. Approximately 24 hours after the estimated time of ingestion, SIC remained elevated at 1441 mcg/dL. She began to exhibit frankly bloody orogastric output as well as oozing from multiple puncture sites. Liver function tests revealed progression to liver failure which resulted in disseminated intravascular coagulation. Around this time, the patient also developed acute respiratory distress syndrome with increasing oxygen requirements and refractory hypoxia so prone positioning was performed. Despite all measures, the patient was pronounced dead approximately 38 hours after the polypharmacy ingestion.
Discussion: The treatment for acute iron poisoning is determined by the type of iron preparation, time of intake, and the onset of symptoms. The clinical features of iron toxicity are typically described in five phases. Acute liver failure and cardiovascular collapse (phase 4) are the main causes of death. An SIC greater than 1000 mcg/dL is associated with severe toxicity and patients should be in a facility that can provide age-appropriate intensive care. In patients with significant clinical manifestations of toxicity, chelation therapy should not be delayed while one awaits serum iron levels, with escalation to higher doses within the first 24 hours. Whole bowel irrigation is an effective method of GI decontamination for serious iron overdoses.
Conclusions: Although an intentional overdose with iron tablets is uncommon in adults, a delay in diagnosis and administration of chelation therapy may rapidly allow progression of toxicity to multi-organ failure and even death.

KEYWORDS Iron, fatality, overdose

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188. Clinical Manifestations of Acute Selenium Toxicity

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Introduction: Although selenium is an essential trace element, it is toxic if taken in excess. Case reports in literature describing acute toxicity are extremely rare. We present a case of acute selenium toxicity as a result of ingestion of gun-bluing agent.
Case Report: A 17-year-old female with a history of bipolar depression, and anxiety, had been in an altercation with her boyfriend. The altercation escalated to the point where the boyfriend took a gun-bluing agent and, in an attempt at self harm, poured it into soda, which the girlfriend promptly took from him and drank in an attempt to save the boyfriend. She immediately became nauseated and began vomiting, with multiple episodes of blue diarrhea, and severe abdominal pain. On arrival to the emergency department, the patient was hemodynamically stable. The initial examination was normal except for a blue discoloration of the oropharynx and a mildly tender abdomen. No laboratory abnormalities were noted, including CBC, electrolytes and liver and renal function. The patient was admitted to intensive care for cardiac monitoring. She continued to have abdominal pain, vomiting, followed by diarrhea. Supportive care included intravenous fluids, pantoprazole, and anti-emetics. Within hours of admission, the patient became bradycardic with heart rate dropping into the 50s. This was felt to be an effect of the selenium toxicity and no immediate action was taken. The patient was transferred to a general floor bed the following day, observed for one more night, and was discharged to home on hospital day three.
Discussion: Gun bluing solution, used in the care of firearms to restore the natural color to the gun barrel, is composed of 2-9% selenious acid which can cause caustic injury to mucus membranes followed by toxicity from selenium. Some authors have proposed a "triphasic" course of acute inorganic selenium toxicity, with GI, myopathic, and circulatory symptoms as the overdose progresses. In reality, acute selenium poisoning is often rapid and fulminant, with onset of symptoms within minutes and, in some cases, death within 1 hour of ingestion. The underlying mechanism for this fulminant clinical syndrome is not well understood but may stem from a multifocal disruption of cellular oxidative processes and antioxidant defense mechanisms. Although it is possible to obtain blood, urine, and hair selenium concentrations to confirm exposure, there is no clear relationship between levels and clinical outcome.
Conclusion: This case report presents an overview of acute selenium poisoning. It underscores the value of close monitoring of patients who present with toxicity, as well as illustrating the potential severity of outcome, which is often but not always fatal. Supportive therapy remains the standard of care.

KEYWORDS Selenium, ingestion, gun-bluing

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189. Utilizing thromboelastography (TEG) to monitor coagulopathy for suspected rivaroxaban toxicity

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Background: Standard laboratory assays used to measure coagulation such as activated-partial thromboplastin time (aPTT), prothrombin time (PT), and international normalized ratio (INR) do not account for all phases of clot formation and degradation. Thromboelastography (TEG) provides more comprehensive evaluation of coagulation factors measured by reaction time (R), fibrinogen function by angle, platelet activity by maximum amplitude citrated-kaolin (MA-CK), and fibrinolysis as lysis at 30 minutes (LY30). Platelet-mapping™ (TEG-PM) may detect antiplatelet agents providing inhibition percentages of arachidonic acid (MA-AA) and adenosine-diphosphate (MA-ADP). TEG is used in cardiac surgery and trauma to guide use of blood products.
Case Report: A 53 year old male with a past medical history of coronary artery disease, atrial fibrillation, hypertension, stroke, and depression with a previous suicide attempt was brought to the emergency department (ED) in cardiac arrest. After 15 minutes of cardiopulmonary resuscitation the patient was found in a narrow complex bradycardia. Due to recent prescriptions for metoprolol tartrate and nifedipine extended release the patient was managed for beta blocker and calcium channel blocker toxicity. The patient was started on high-dose insulin therapy, glucagon, epinephrine, norepinephrine and vasopressin. His initial labs were as follows: ALT 1512 U/l, AST 1243 U/l, ionized calcium 1.07 mmol/l, pH 7.26, WBC 23,600/mm3, hemoglobin 10.6 g/dl, hematocrit 21.4%, platelets 296 K/ul, serum creatinine 1.79 mg/dl, fibrinogen 166 mg/dl, PT 31.4 s, INR 3.2, aPTT 41.3 s. Post-intubation the patient began to bleed from the orogastric tube and intravenous puncture sites. A pantoprazole infusion was started for suspected gastrointestinal (GI) bleeding. This prompted nursing to recommend rapid-TEG® (r-TEG) and TEG-PM assays; the ED pharmacist was consulted for TEG interpretation and recommendations. Medication reconciliation identified the patient was on aspirin, clopidogrel, and rivaroxaban. Initial TEG results reveal the following: ACT 296 s (80-120), R 32.4 min (5-10), 41.6 degrees (53-72), MA-CK 62 mm (50-70). Severe inhibition of MA-AA and MA-ADP (97.4% and 82.9% respectively) was also noted. The patient then received 4 units of fresh frozen plasma, prothrombin complex concentrate (human), and desmopressin. The post-intervention ACT was 235 s. On day 4, GI bleed was ruled out by endoscopy and 8 undigested tablets were removed during procedure. On day 5, repeat TEG results were R 6.5 min, angle 73.9 degrees, and MA-CK 79.4 mm. The MA-AA and MA-ADP inhibition continued to resolve (42.7% and 58.8% respectively). Hemoglobin >7 g/dl was maintained and psychiatry was consulted for management pending discharge from the intensive care unit.
Case Discussion: The patient's TEGs demonstrate a multifactorial coagulopathy complicated by hepatic failure, hemodilution and co-ingestion of rivaroxaban. Limited studies suggest patients on rivaroxaban may have R >7 min and ACT values >120 s at non-toxic concentrations.

Table. Daily trending of thromboelastography and standard laboratory values.

Laboratory Test	Day 1	Day 2	Day 3	Day 4	Day 5
Rapid TEG					
ACT (s)	269				
Angle (degrees)	75.8				
MA-CK (mm)	72.2				
LY30 (%)	0				
Standard TEG					
R (min)	32.2		14.6	8.9	6.5
Angle (degrees)	41.6		57.7	71.4	73.9
MA-CK (mm)	62		72	80	79
MA-AA (mm)	9.1		33.6	44	62
MA-AA inhibition (%)	97		98	94	43
MA-ADP (mm)	17		36	75	59
MA-ADP inhibition (%)	83		92	75	59
LY30 (%)	5		0	0	0
Standard Values					
aPTT (s)	41.3	38.8			
POCT ACT (s)	235				
INR	3.2	2.2	2.1	1.2	1.1
PT (s)	31.4	22.2	20.9	12.4	11
Fibrinogen (mg/dl)	166	226		710	
Platelets (K/ul)	296	159	121	101	110

Additionally, the half-life of rivaroxaban may exceed 9 hours in moderate renal and hepatic impairment. Prolonged MA-AA and MA-ADP inhibition also suggests co-ingestion of aspirin and clopidogrel.
Conclusion: Further research should be conducted for TEG guided management of coagulopathy. To our knowledge this is the first case report for TEG monitoring in rivaroxaban toxicity.

KEYWORDS Thromboelastography, Coagulopathy, Bleeding

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190. Dragon Medical One®: A Novel Way to Decrease Poison Specialist Stress

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Background: Specialists in Poison Information (SPIs) manage poisoned patients and document information in electronic health records (EHRs) in real time. High quality, timely documentation is important in delivery of medical care and increasingly, SPIs are documenting more detailed information due to increased exposure complexity. This has been attributed to an increase in both hospital-based calls and intentional exposures. Documentation recorded by typing on a computer keyboard has been associated with neck pain and repetitive motion injuries to the wrist and hand. An alternative is use of conversational artificial intelligence tools such as Dragon Medical One® (Dragon). Widely employed, it is designed for speed, accuracy, and flexibility. A survey on the use of this dictation tool by SPIs and its effect on workplace wellness was conducted.
Methods: An anonymous survey was administered to all SPIs using Dragon at a poison center two months post-implementation. It had three sections: (1) Satisfaction with training, impact on day-to-day workflow and how likely SPIs were to recommend Dragon were measured using a five-point Likert scale; (2) Documentation time and workplace stress were assessed using yes or no questions; (3) Strengths and weaknesses of Dragon were recorded using open-ended questions and free text fields.
Results: The survey was administered to 6 SPIs (n = 6). Response rate was 100%. All of the respondents were either satisfied or very satisfied with their training. All respondents felt that Dragon had a "huge positive impact" on their daily workflow and were "extremely likely" to recommend its use. The majority (5/6; 83%) reported that they saved at least 5 minutes per case on core documentation. No SPI reported an increase in documentation time. All respondents reported a decrease in workplace stress. Multiple strengths and weaknesses were identified. Strengths: Two SPIs listed improved documentation accuracy; one SPI stated that it appreciably decreased long-standing arthritic wrist pain, reporting her pain went from 7/10 to 0-1/10, although she also started using an ergonomic computer mouse around the same time. Weaknesses: 83% cited spelling errors/lack of word recognition (affectionately dubbed "dragonisms"); one SPI with arthritis developed de novo thumb pain from pressing the activation button on the Dragon microphone (though this improved on use of keyboard activation of the microphone).
Limitations: Study sample was small. No pre-implementation survey was performed. System optimization had not yet been performed. Many variables can influence a SPI's workload.
Conclusions: The use of Dragon conversational dictation tool was associated with a reduction in workplace stress. SPIs reported saving time on documentation and greater job satisfaction. Further research on the effects of dictation tools on SPI workplace wellness is needed.

KEYWORDS Poison specialist, conversational dictation system, stress reduction

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191. How A Dragon Helped Increase Productivity at a Poison Center

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Background: Exposures managed by poison centers have increased in complexity over the last decade due to an increase in hospitalized cases. Additionally, after years of steady decline, call volume to poison centers increased in 2018. These changes place increased demands on poison center staff. Specialists in Poison Information (SPIs) manage poisoned patients and document patient information in an electronic health record (EHR) in real time. High quality documentation is important to delivery of medical care; it improves communication, supports diagnostic and therapeutic decision-making, enables evidence-based decisions, provides safeguards, and enhances research and monitoring of quality improvement measures. Higher quality documentation can be achieved with conversational artificial intelligence tools. Dragon Medical One® (Dragon) is a widely used conversational dictation system that is designed for speed, accuracy, and flexibility. To date, no report on the implementation or the use of a conversational dictation tool in a poison center environment exists.
Objective: Determine whether use of a conversational dictation system increases the productivity of SPIs.
Method: A single arm pre-post intervention study was conducted following implementation of Dragon conversational dictation system. The pre-period was February and March 2018 and the post-period was February and March 2019. Productivity was measured in new cases/hour. Cases were the total number of new cases entered in Toxicall® per month and hours were the total hours worked per month as recorded in payroll records. Five SPIs were included in this study; they were blinded to the study outcomes. A p value of 0.05 or less, calculated using the paired student t-test, was considered statistically significant.
Results: All five SPIs increased their number of cases/hour. The mean increase was 44% (range 12.3-158.9%). Four were certified-SPIs. The fifth SPI was in-training and experienced a 158.9% increase in cases/hour; this was considered an outlier so was not included in the statistical analysis. Mean cases/hour were 1.33 vs. 1.55 in the pre- and post-intervention periods, respectively. The difference of 0.22 cases/hour represents a 15% increase and is statistically significant, $p < 0.05$.
Discussion: This is the first report of implementation of a conversational dictation system within a poison center data collection system. We observed a statistically significant increase in the productivity of all SPIs. More importantly, the increased productivity allowed this poison center to better position itself to meet the shifting demands of callers and continue to improve delivery of medical care.
Limitations: Sample size is small. Study period was only 4 months. Changes in cases/hour during study period cannot be fully attributed to the specific intervention due to changes in call volume over time. Follow up calls were not included. Optimization of Dragon had not been performed. Cases entered using Dragon were not reviewed for quality.
Conclusion: The use of a conversational dictation tool (Dragon) increased the number of new cases/hour entered in an EHR by a certified-SPI by 15.0%. We anticipate further improvement as the system is optimized to meet the needs of the poison center.

KEYWORDS Conversational artificial intelligence tool, productivity of poison specialists, documentation

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192. Methadone Storage and Education: A Survey Conducted with Clinic Directors

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Background: Unintentional exposures to opioids including methadone and buprenorphine have been rising. A previous study analyzed calls to the Poison Control Center (PCC) involving pediatric exposures to opioids and developed safety information. This study aims to assess the medication safety education given to methadone clinic patients, the use and distribution of medication lock boxes/bags for storage in the home, and medication disposal information provided to patients.
Methods: A list of outpatient substance abuse clinics was downloaded from the state substance abuse services website. The list was limited to methadone clinics serving the PCC catchment area. A survey was developed to gather information on medication safety education and practices from methadone clinic directors. The survey was sent via email and follow-up calls were made to clinics who did not complete the survey after two weeks. Two email reminders were sent.
Results: The survey was sent to a list of methadone clinics (N = 60) in the PCC catchment area in July 2018. A total of 35 surveys were analyzed; 8 partial and 27 with completed information. All clinics responded that they distribute take home doses of methadone. Less than half (46%) prescribe buprenorphine. Medication safety education is provided through various methods (answers reflect more than one): individual instruction (100%), printed information (52%), and group instruction (18%). Those who provide the education are counselors (93%), nurses (89%), and physicians (79%). Educational sessions are delivered at intake (89%), immediately prior to take home methadone prescription (78%), and during counseling sessions (78%). Almost all (89%) reinforce medication safety: with every take home dose (32%), quarterly (32%) or monthly (9%). Most sites (67%) responded that there is a method of identifying and targeting medication safety education to those who are homeless; often reporting that they do not provide take home doses to those who are homeless. Almost all programs (89%) responded that there is a method of targeting medicine safety education to patients living with or caring for young children. When asked about medication safety messages, the majority include the following: a small amount is potentially fatal (89%), safe storage in a locked location can prevent injury (96%), and unused medicine should be disposed of safely (81%). The most common recommendation for disposal of methadone was returning it to the clinic for disposal (n = 18/26). The majority of clinics reported that they do not distribute medication lock boxes (89%) or bags (96%). Of the three sites who do distribute lock boxes/bags, all include safe storage information with these boxes/bags in the form of verbal instruction and written information. None of the sites charge patients for the medication lock boxes/bags.
Conclusion: In our study, methadone clinics reported that safe storage education is provided to patients, particularly those living with young children. Although medicine lock boxes are not provided, safe storage messages are discussed with patients periodically. Medication disposal information is also provided. Our next step is to ensure that the medicine safety information being conveyed to patients is standardized statewide and addresses essential key messages.

KEYWORDS Methadone, buprenorphine, education

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193. Amitriptyline Overdose with Refractory Ventricular Dysrhythmias Rescued by Intralipid Emulsion Therapy

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Background: Tricyclic antidepressants (TCAs) have a narrow therapeutic index with risk of serious toxicity. Severe poisoning is characterized by seizures, coma and ventricular dysrhythmias. We present a case of severe amitriptyline toxicity complicated by refractory ventricular dysrhythmias rescued by intralipid emulsion (ILE).
Case: A 30-year-old woman ingested amitriptyline in a suicide attempt. She was initially alert but rapidly became unresponsive and developed generalized seizure activity with tachycardia and hypotension. She was intubated, started on sodium bicarbonate therapy, and transferred to a tertiary care center. Upon arrival, the patient was in status epilepticus with relative bradycardia (70 bpm). Electrocardiogram (EKG) showed QRS duration of 188 ms. She was given 400 mEq of sodium bicarbonate and 1950 mg of intravenous (IV) phenobarbital with subsequent narrowing of her QRS to 136 milliseconds. She had improvement of her hemodynamics and resolution of her seizures. Her sodium bicarbonate drip was increased and she was started on a norepinephrine infusion. Despite these interventions, her QRS widened to 160 ms, her blood pressure decreased to 61/34 mmHg and she developed breakthrough seizures. She was given an additional 520 mg of phenobarbital, 200 mEq of sodium bicarbonate and lidocaine bolus and infusion. She developed ventricular tachycardia, and was given 2 mg of IV epinephrine and 1500 mL 20% ILEtherapy over 25 minutes. After completion of the ILE infusion, her hemodynamics improved and we were able to wean her norepinephrine infusion. An hour after her ILE infusion, she had narrowing of her QRS to 134 ms. She was admitted to the ICU and had no further seizures or QRS widening. Lidocaine and sodium bicarbonate infusions were discontinued on hospital day one and norepinephrine was discontinued on hospital day three. Comprehensive urine drug screen confirmed the presence of amitriptyline and cyclobenzaprine only. She was transferred to a psychiatric facility on hospital day 13 without neurologic sequelae from her overdose.
Discussion: Tricyclic antidepressants like amitriptyline account for 50% of the mortality from antidepressant overdoses. Sodium channel blockade prolongs QRS leading to unstable cardiac dysrhythmias. Sodium bicarbonate therapy is the mainstay of treatment by competitively overcoming sodium channel blockade and plasma alkalinization which theoretically displaces the TCA from the sodium channel itself. ILE therapy has been described in animal studies and human case reports of TCA toxicity. Our patient had transient improvement with aggressive sodium bicarbonate loading and infusion however developed worsening toxicity. Intralipid emulsion therapy was successfully used with improved clinical, hemodynamic and electrocardiographic findings.
Conclusion: TCA overdose can result in cardiotoxicity and neurotoxicity that is refractory to sodium bicarbonate and supportive therapy. ILE should be considered in severe cardiotoxicity from amitriptyline overdose.

KEYWORDS Amitriptyline, Intralipid, Ventricular dysrhythmia

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194. Patiromer as a Novel Adjunctive Therapy for Refractory Hyperkalemia in Intentional Potassium Chloride Overdose

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Background: Hyperkalemia can cause ventricular dysrhythmias and cardiovascular collapse. We describe a patient who ingested extended release potassium chloride, was refractory to renal replacement therapy, and improved with patiromer, a potassium binding polymer.
Case: A 60-year-old man with a history of atrial fibrillation, hypothyroidism, ischemic cardiomyopathy and chronic ethanol use presented

after massive intentional potassium chloride ingestion. He was initially responsive and complaining of shortness of breath. He was noted to have a wide complex bradycardia and subsequently had a witnessed cardiac arrest and was given epinephrine, furosemide, calcium chloride, sodium bicarbonate and insulin with subsequent return of spontaneous circulation. He was intubated and transferred to a tertiary care center. He was further stabilized with calcium chloride and sodium bicarbonate. His initial potassium was 8.9 mMol/L, sodium was 188 mMol/L and creatinine was 1.6 mg/dL. He was placed on norepinephrine and epinephrine. He received approximately 7.5 hours of hemodialysis with improvement of his potassium to 3.7 mMol/L. His potassium rebounded to 5.1 mMol/L approximately four hours after discontinuation of hemodialysis and he was started on continuous venovenous hemodiafiltration (CVVHDF). Despite being on CVVHDF for 20 hours, potassium remained greater than 5 mMol/L. He developed ileus, precluding the use of kayexalate. He was given patiromer on the evening of hospital day two for refractory hyperkalemia. His potassium subsequently declined to 4.6 mMol/L and remained less than 5 mMol/L. CVVHDF was discontinued the next morning and his pressor requirements improved. He was transferred to a psychiatric facility on hospital day 12 without sequelae from his overdose.

Discussion: Although multiple therapies temporarily treat hyperkalemia by shifting potassium intracellularly and stabilizing cardiac membranes, definitive therapy removes excess potassium from the body. Kayexalate was not given as it contains sorbitol, which has been associated with intestinal necrosis in ileus. Patiromer is a sorbitol-free exchange polymer. The patient was treated successfully with patiromer as a novel adjunctive therapy.

Conclusion: In severe refractory hyperkalemia, patiromer is a safe adjunctive therapy, with utility in cases where kayexalate is contraindicated.

KEYWORDS Patiromer, Hyperkalemia, Potassium chloride

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195. Toxicity Resulting from Acute Methotrexate and Ibuprofen Ingestion

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Background: Methotrexate (MTX) is an anti-folate drug used in treatment of malignancy, chronic inflammatory conditions and ectopic pregnancy. Despite its frequent clinical use, reports of acute oral overdose are limited. A recent meta-analysis suggested that because of low MTX oral bioavailability, treatment is not necessary for acute ingestions. We report a case of MTX toxicity following acute ingestion of MTX and ibuprofen.

Case report: A 61 kg 17-year-old transgendered male presented to the Emergency Department approximately three hours after intentionally ingesting 30-40 tablets of unknown dose MTX that he took from a neighbor's garage. He also co-ingested approximately 40 tablets of 200mg ibuprofen, after reading that the combined ingestion would worsen toxicity. He denied other ingestions. On presentation, he was mildly lethargic and nauseous, with an otherwise unremarkable exam. Initial vital signs were: HR 103, BP 126/65, RR 20, T 98.2, SpO2 100%. A 1 mg/kg dose of activated charcoal was administered in consultation with toxicology, while awaiting laboratory results. He then developed intractable vomiting and decision was made to begin leucovorin therapy. His serum methotrexate concentration later resulted at 2.17 mcgmol/L. He received leucovorin 100 mg/m2 IV initial dose and then 10 mg/m2 IV every six hours until his serum MTX concentration was less than 0.1 mcgmol/L. He also received urinary alkalinization with sodium bicarbonate during this period. His vomiting and lethargy resolved 12 hours after starting leucovorin and he has remained asymptomatic since. He never developed renal insufficiency (peak creatinine 0.67 mg/dL) or metabolic acidosis consistent with severe ibuprofen toxicity (pH, 7.39; pCO2, 37; HCO3 23 mmol/L). He showed mild transaminitis on day two post-ingestion with peak AST 40 U/L and ALT 41 U/L. Initial complete blood count was unremarkable, though WBC dropped from 9.36 K/mcgL to 5.05 K/mcgL one week later. Hematocrit and platelet counts remained stable. Complete blood count from day 14 post-ingestion is still pending.

Case discussion: The oral bioavailability of MTX is saturable, reducing bioavailability to a reported 10-20% for doses greater than 80 mg/m2; this lends credence to the idea that a single acute ingestion should not produce toxic concentrations. Nevertheless, our patient's MTX concentration was more than twice the accepted potentially toxic concentration (1 mcgmol/L) and he showed symptoms of toxicity. A recent meta-analysis suggested that such levels are not concerning as they are below the leucovorin rescue nomogram for high-dose chemotherapeutic MTX therapy. However, such high-dose chemotherapeutic therapy leads to bone marrow suppression in up to 25% of patients, so the nomogram would not be an appropriate guide for avoiding toxicity in MTX-naïve patients. Additionally, while there was no evidence of renal insufficiency caused by ibuprofen overdose, it is possible that the co-exposure altered clearance, allowing for increased toxicity.

Conclusion: Acute overdoses of MTX can result in toxicity. Co-ingestion of non-steroidal anti-inflammatory drugs (NSAIDs) may increase susceptibility, even without apparent renal impairment.

KEYWORDS Methotrexate, acute, toxicity

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196. Overdose on First FDA Approved Deuterated Pharmaceutical

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Introduction: In 2017, the FDA approved the first deuterated pharmaceutical, deutetrabenazine for Huntington's disease. Similar to its parent compound, tetrabenazine, it blocks VMAT 2, but with the added theoretical benefit of an extended half-life and lower peak drug levels, which allows for less frequent dosing and reduces adverse effects. Experience in overdose is extremely limited and we report the first documented case of overdose.

Case Study: A 21-year old man with an idiopathic movement disorder presented after overdosing on deutetrabenazine (120 six mg tablets). He was encephalopathic with increased writhing movements different than his baseline chorea. He had persistent psychomotor agitation and required 15 mg of diazepam and 10 mg of midazolam initially. After his first six hours, he was given 1mg benztropine and administered lorazepam as needed (8 mg over 18 hours). No co-ingestions were identified on urine gas chromatography/mass spectroscopy. His EKG showed a normal sinus rhythm with a normal QRS and QT interval. Twenty-four hours after his overdose, his toxic encephalopathy cleared and his baseline ataxia returned.

Discussion: Although documented cases of overdose are rare, confusion, tremors, and acute dystonia have been reported. In this case, the patient had similar toxic effects, which may be secondary to acute synaptic dopamine release or, alternatively, a depletion of dopamine leading to Parkinsonian features. In addition to high doses of benzodiazepines we administered benztropine which may have been efficacious as only two milligrams of lorazepam were required afterwards. Deutetrabenazine contains deuterium substitutions at six hydrogen sites in the tetrabenazine molecule. The molecular bonds between

deuterium and carbon are more difficult to break therefore leading to slower metabolism of deutetrabenazine. In overdose, the kinetics of either xenobiotic is not yet elucidated and the duration of toxicity in this case may be attributable to inherent properties of the deuterated compound or to saturation of metabolic and elimination pathways.
Conclusion: We describe an overdose on a deuterated pharmaceutical with toxicity mirroring that of the parent compound but with a longer duration of toxicity.

KEYWORDS Deutetrabenazine, Overdose, Huntington's

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197. Analysis of Food Grade Hydrogen Peroxide Ingestions over 10 years

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Background: Hydrogen peroxide (H2O2) is an oxidizing agent that exerts its primary toxicity through corrosive injury and oxygen gas formation. A specific 35% (food grade) concentration is available for "hyperoxygenation therapy" and is promoted for ingestion for various disease states, allowing for potentially significant morbidity and mortality. Recently, our regional poison center (RPC) has received an increase in call volume from patients who have ingested concentrated H2O2 products. This study aims to report the incidence of poisonings involving varying H2O2 concentrations, with an emphasis on ≥35%, over 10 years.
Methods: This is an IRB approved retrospective chart review from January 1, 2009 to December 31, 2018, analyzing all patients with a single-agent ingestion of H2O2 using our RPC electronic records database, ToxSentry®. Patients were excluded if the substance was not H2O2 or an ingestion did not occur.
Results: A total of 4525 patients met criteria for inclusion; with 72 patients documented as ingesting ≥35% H_2O_2. For all exposures, the average age was 25 years with 55% females compared to exposures ≥35% that had an average age of 50 years with 62% females. Pediatric patients ≤6 years accounted for 42.5% of all ingestions. The total number of H_2O_2 exposures decreased from 648 in 2009 to 302 in 2018 (rate ratio =0.91, 95% CI: 0.89-0.92), paralleled with a decrease in overall exposure calls to the poison center, 91,582 in 2009 versus 72,472 in 2018. However, the number of H_2O_2 exposures ≥35% increased from two in 2009 (0.3%) to 19 in 2018 (6.3%) ($p < 0.001$). There were also 973 (21.5%) exposures that were classified as unknown percentage. Of the ≥35% exposures, 10.1% were classified as intentional/misuse whereas only 0.6% of exposures <35% were classified as intentional/misuse ($p < 0.001$). Callers with exposures ≥35% were more likely to have major (3.8% vs 0.3%), moderate (9.1% vs 0.3%), and minor (13.4% vs 3.9%) clinical effects ($p < 0.001$). The most common effects seen in ≥35% exposures were gastrointestinal in nature, such as nausea, vomiting, throat irritation, or hematemesis. Hyperbaric oxygen was performed on one patient with a confirmed portal venous air embolism. Total H_2O_2 exposures showed that 66% of ingestions were ≤1 mouthful, 17.4% were >1 mouthful, and the remainder were unknown. Finally, 30.5% of exposures ≥35% were managed on site in a non-healthcare facility compared to 91% of H_2O_2 exposures <35%. The ≥35% exposures that were managed on site were primarily <1 mouthful or a mouthful or more of a diluted solution.
Conclusion: At our RPC, H2O2 ingestions ≥35% have significantly increased in 2018 compared to prior years despite the steady decline in the total number of H2O2 calls received over 10 years. Exposures ≥35% were more likely to be intentional, managed in a healthcare facility, and associated with increased minor, moderate, and major outcomes. There were also a large number of H2O2 exposures that had an unknown concentration each year. As such, increased awareness, education, and a thorough history are needed if a food grade H2O2 ingestion is suspected.

KEYWORDS Hydrogen peroxide, Ingestion, Toxicology

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198. Does Therapeutic Hypothermia Decrease Efficacy of Hyperinsulinemia Therapy in Calcium-Channel Blocker Toxicity?

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Background: High-dose insulinemia with euglycemia (HIE) has been proposed as a treatment for calcium-channel blocker (CCB) toxicity, however HIE use has never been reported in a CCB-toxic patient concurrently undergoing therapeutic hypothermia. Current AHA guidelines suggest using the standard ACLS algorithm, including therapeutic hypothermia, for patients experiencing return of spontaneous circulation (ROSC) following cardiac arrest in the setting of CCB toxicity. Metabolic derangements are expected as body temperature decreases; specifically, insulin resistance and decreased pancreatic insulin release are theorized to be the underlying cause of hyperglycemia in hypothermic patients. We report a case of a patient undergoing therapeutic hypothermia following ROSC, who was later found to have CCB toxicity and did not respond to HIE.
Case Report: A 66-year-old female with reported history of acute myocardial infarction three weeks prior presented to the ED complaining of chest pain. She was sent emergently to the catheterization laboratory for possible STEMI. While undergoing percutaneous coronary intervention, she experienced a cardiac arrest with ventricular fibrillation. ROSC was obtained after 15 minutes, and a transvenous pacemaker and Impella® device were placed for hemodynamic support. Therapeutic hypothermia protocol was initiated with goal temperature 34 °C. The patient was noted to be in persistent distributive shock despite mechanical interventions and "max" doses of epinephrine, norepinephrine, and vasopressin. About 30 hours after presentation, case management found a suicide note with empty bottles of diltiazem XR and amiodarone. As the planned rewarming process began, the patient developed cardiogenic shock. Toxicology was consulted and recommended HIE as well as increased epinephrine. After eight hours of HIE at 10 units/kg/hr, the infusion was stopped due to lack of cardiovascular improvement and concerns for fluid overload. Due to dismal prognosis, family agreed to comfort measures, and the patient expired seven days after ED presentation.
Discussion: CCB toxicity is theorized to cause hyperglycemia via blockade of pancreatic calcium channels leading to impaired insulin release. HIE is thought to mitigate the effects of CCB poisoning by increasing cardiomyocyte uptake of glucose, allowing for increased utilization of carbohydrates during stress. Our patient did not exhibit improved hemodynamics despite 8 hours of HIE. Hypothermia has been shown to decrease the expression of insulin receptors in animal models, thus it is plausible that decreased expression of insulin receptors could limit the efficacy of HIE in a hypothermic patient. More research is necessary to assess the possibility that predictable metabolic changes seen with therapeutic hypothermia contribute to the lack of expected response from usual dosing of HIE.

KEYWORDS Calcium channel blocker, therapeutic hypothermia, high dose insulinemia euglycemia therapy

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199. Observational study on the use of methylene blue for shock in fatal overdoses.

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Background: A recent systematic review of methylene blue (MB) for drug-induced shock reported a total of 17 published cases, with calcium channel blockers representing ten of those. Nine had hemodynamic improvement. Adverse reactions noted in these cases was blue discoloration of urine, skin, and eyes. Evidence was considered inconclusive to be able to recommend MB for treatment of refractory shock in poisonings. Other published adverse effects reported with the use of MB for shock include serotonin toxicity. This study explored as primary aim to quantify the number of fatalities in the National Poison Data System (NPDS) in which intravenous MB was used to treat drug-included shock. Secondary aims included the nature of substances involved, the rate of reporting of adverse effects of MB therapy, prior or concomitant administered therapies, the circumstances of administration (first line, last resort, in combination with other agents), and association of MB therapy with de-identified patient characteristics such as age, sex, or comorbidities.
Methods: Published fatality abstracts from NPDS for the years 2010–2017 were searched for cases in which MB was coded as a therapy performed. Fatalities for which the poisoning was attributed as "undoubtedly"or "probably responsible" as the cause of death were abstracted for predefined variables on a standardized data collection tool. Cases were excluded if: MB was to treat methemoglobinemia; MB was not explicitly mentioned in the fatality abstract; or if MB was mentioned but clearly not given.
Results: A total of 106 cases were reviewed. Eighteen were excluded due to: MB use in methemoglobinemia (12 cases); no explicit mention of MB use (5 cases); or MB clearly not given (1 case). MB was used in 28 cases as a last resort, but never used as first-line therapy; in 15 cases MB was given alone. Substances were dominated by calcium antagonists (57/88 cases), primarily amlodipine (42 cases). Other substances included metformin (7 cases), beta blockers (9 cases), and carbon monoxide (2 cases),. MB resulted in a transient increase in blood pressure in 12 cases (4 of those amlodipine), minimal effect in 3, and no effect in 23 cases in which a response was documented. Adverse effects included hyperreflexia in one case and interference with hemodialysis in another.
Conclusion: This study adds to the literature regarding use of MB in distributive drug induced shock in patients with ultimately fatal outcomes from poisoning exposures. A transient response is possible, particularly with amlodipine overdose. Further research is needed to define the role of methylene blue in treatment of poisoning-related shock.

KEYWORDS Methylene blue, shock, calcium channel blockers

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200. Intentional suspected Suicide cases in ages 10 to 25 reported to NPDS from 2000 to 2018

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Objective: evaluate the substances used and temporal association involved in the increase in suicide attempts in adolescents and young adults ages 10 to 25.
Methods: Review of intentional suspected-suicide cases reported to NPDS from 2000 to 2018 for patients 10 to 25 years old. We evaluated age, gender, monthly and annual incidence, substances involved (single substance and polysubstance), and medical outcome. Serious outcome was moderate, major outcome and death.
Results: There were 1,677,435 cases reported to NPDS with the reason of Intentional suspected suicide and with an age of 10 years old to 25 years old from 1/1/2000 to 12/31/2018. The majority (n = 1,184,691, 71%) were female and involved a single substance (n = 1,074423, 64%) and there were 410,940 cases (24.5%) with a serious outcome.
Temporal trends: For the age groups of 10-12, 13-15 and 16-18 there was an inflexion point occurring after 2011 with a dramatic increase of >200% ($p < 0.05$), which was influenced primarily by females. In the age groups of 10-12, 13-15 and 16-18, there was a significant decrease of (27%, 27% and 18% respectively) ($p < 0.01$) in cases during non-school months of June, July and August. There was no change in the age group 19-21 and an increase in 22-25 group (Figure 3). Substances involved in cases with a serious Outcome: The majority of substances used in Intentional suspected suicide were pharmaceutical (n = 2,482,014, 92%). The leading substances involved in cases with a serious outcome changed with time and by age group, but the top

Substance group	number of cases (% of total cases)	Number of cases with serious outcome	Percent of substance group with serious outcome
Other Analgesics	743,091 (27.5%)	153,361	20.6
Antidepressants	412,736 (15.5%)	135,314	32.8
Sedatives/Hypnotics	208,938 (7.7%)	61,420	29.4
Antihistamines	169,787 (6.3)	59,859	35.3
Antipsychotics	166,515 (6.2%)	62,984	37.8
Alcohols	120,420 (4.5%)	39,759	33.0
Anticonvulsants	116,091 (4.3%)	40,051	34.5
Opioid analgesics	107,105 (4%)	31,858	29.7
Cold and Cough Preparations	106,106 (3.9%)	30,277	28.5
ADHD Drugs	67,852 (2.5%)	33,618	49.6
Antimicrobials	56,591 (2.1%)	11,579	20.5
Muscle Relaxants	55,411 (2.1%)	20,781	37.5
Cleaning Substances (Household)	48,087 (1.8%)	6,281	13.1
Cardiovascular Drugs	46,915 (1.7%)	19,269	41.1
Stimulants and Street Drugs	37,639 (1.4%)	17,149	45.6
Unknown Drug	36,386 (1.4%)	11,591	31.9
Dietary Supplements/ Herbals/ Homeopathic	31,988 (1.2%)	6,651	20.8
Hormones and Hormone Antagonists	30,920 (1.1%)	10,305	33.3
Gastrointestinal Preparations	25,922 (1.3%)	7,123	27.5
Cosmetics/Personal Care Products	17,654 (0.7%)	1,372	7.8

two substance groups in all age groups were antidepressants and the over-the-counter analgesics (Figure 3). Antipsychotics were noted in the top 5 leading substance in all age groups and antihistamines were a leading substance in all age groups except the 22-25. The ADHD medications were common in the younger age groups of 10-12 and 13-15, while the sedative/hypnotics (primarily benzodiazepines) occurred more commonly in the older age groups (16-18, 19-21 and 22-25). Prescription opiates were less commonly involved (4%) and decreased in occurrence in all age groups after 2013. Over time there was an increase in percentage with serious outcomes (within substance groups) in the substance groups of: OTC analgesics, antihistamines, antidepressants, opiates and ADHD medications, and no change in percentage of serious outcome in antipsychotic or anticonvulsant groups. The substance groups with the greatest increase in percentage of serious outcomes (within group) were ADHD drugs, cardiovascular drugs and stimulants/street drugs. The groups with the greatest number of serious outcomes were OTC analgesics, antidepressants, antipsychotics and sedative/hypnotics.
Discussion: There was a significant increase in 10 to 18 age groups and in females after 2011 that coincided with the expansion and influence of social media in adolescence. Both the number and percentage of cases with a serious outcome increased over time and by age. Substances involved were primarily pharmaceutical and appeared to be those available to each age group. Opioids were not an influence on these trends.

KEYWORDS Suicide, Adolescents, social media

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201. Hispanic Ethnicity and County-Level Poison Control Center Utilization in Florida, 2010-2015

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Background/Objectives: Increasing utilization of poison control centers is a priority as the nation continues to grapple with a worsening opioid crisis, yet little is known about how demographic and other contributory factors are associated with calls to poison centers. This study aimed to identify the role of the concentration of Hispanic residents in poison center utilization while adjusting for confounders.
Methods: This study was a longitudinal, ecological analysis examining the association of county-level Hispanic ethnicity with poison control center utilization in Florida, as measured by human exposure call volume. Data were assembled regarding all 67 counties in Florida: demographic and fixed covariate data from baseline and poison center call data for each year from 2010-2015. Analysis was conducted using a generalized linear mixed model with a Poisson distribution which included an offset variable to account for county population. The final model estimated fixed effects and coefficients for year, Hispanic concentration, opioid sales, age-adjusted hospitalization rate, and percentage of population under 5. The model also included a quadratic term and random factors for slope, intercept and year2.
Results: Poison center use generally declined over the time period across counties. After for adjusting for factors that significantly varied between high Hispanic and low Hispanic counties, including percentage of residents under 5, opioid sales and hospitalization rate for poisoning, there was no discernable difference in the negative call trends based on ethnicity at the county level. The incidence rate ratios for variables in the final model were as follows: High Hispanic ethnicity, .96 (95% C.I. 0.84-1.1), percentage residents under 5, 1.12 (C.I. 1.06-1.18), opioid sales, 1.33 (C.I. 1.13-1.57) and hospitalization rate, 1.002 (C.I. 1.001-1.003). Factors found not to be significantly associated with poison center utilization (and thus left out of the final model) included poverty rate, urban/rural status, and poison center service region (south, central, north).
Conclusions: Contrary to the original hypothesis that counties with higher than average concentration of Hispanic residents would underutilize Florida poison centers, we found no significant difference after adjusting for important confounding variables. These findings indicate that, while outreach to Hispanic communities is important as a matter of health equity, factors other than ethnicity play a larger role in driving poison center utilization for human exposures. The results also indicate a disappointingly low rate of co-management of hospitalizations for poisoning, given the well-documented improvement in outcomes for co-managed cases. These results indicated the need to attack underutilization broadly by better publicizing the poison center across populations.

KEYWORDS Poison center utilization, Hispanic, epidemiology

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202. Where We've Been....Where We're Going: Poison Information Provider (PIP) Progression

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Background: The inception of Poison Information Providers (PIPs) for our poison center was 2004. Primary tasks focused upon drug identification of medications and management of bites and stings exposures, specifically helpful due to the large number of scorpion envenomations received. Additionally, PIPs managed lower acuity exposures, under the direct supervision of a SPI/CSPI. We employed one PIP at that time. Initial experiences of the role of the PIP in our center is described. Method: By 2006, our center employed six PIPs of various work schedules and by 2008, we employed nine PIPs. Managing Director, Clinical Nurse Manager, and Educator sought ways to increase the duties of the PIP role by active engagement in our host facility and review of current contracts.
Results: In 2009, our host facility was in need of a triage line to assist their Occupational Health department with Post-Exposure Prophylactic (PEP) calls; due to 24/7 availability, the poison center was given the PEP line to manage. Initially, SPI/CSPIs obtained all information, with PIPs only assisting with specific questions . By 2013, PIPs were fully managing the intake of all of PEP calls. As poison control centers experienced a decrease in call volume and the number of drug identification requests, our center reached out to partnering agencies to encourage them to take advantage of our 24/7 availability. Our poison center had maintained a dedicated after-hours phone line since 2004 for the local county health department. 2015 saw another dedicated health department phone line providing information regarding local county immunization clinics (i.e., hours, location, etc.) fully maintained by PIPs. Our PIPs participate in our center's patient satisfaction surveys and maintain the current records regarding rattlesnake and scorpion anti-venom availability at various hospital pharmacies throughout the community. Utilizing our PIPs in the best interest of the department, new substances/items have been added to the work flow of our PIPs in regards to exposures. Senior staff, along with our educator and medical director, reviewed various suggested substances after surveying other poison centers for input and approved seven new substances for PIPs to manage. PIPs received additional training specific to these new substances. The addition of these substances/exposures to a PIP's competency aids in the work-flow of our department and allows our SPI/CSPIs to be available for higher acuity exposures. Orientation for our PIPs has also evolved over the years. Initially, SPI/CSPIs provided the lectures and directly precepted the new hires, but in our center the educator

created a three-week orientation process thus providing a more specific and detailed educational experience and allowing current, senior PIPs to provide direct, hands-on phone orientation.
Conclusion: Our poison center continues to benefit from PIPs. Expanding their knowledge base makes it beneficial to the SPI/CSPI's availability for higher acuity calls, and also time management and budgetary needs of the department. In order to enhance staff engagement, poison centers can continue to benefit from what PIPs can provide to partnering agencies, as well as the community at large; PIPs are an integral part of our workplace.

KEYWORDS PIP, Progression, Years

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203. Impact of a Prescription Medication Education Program on Adolescent Knowledge and Behavior

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Background: Analgesics are the leading substance category nationwide that prompt people to contact poison control centers. Opioid prescribing rates remain high across the country. Furthermore, the myth that street drugs endanger more lives than prescription drugs is still prevalent, especially among teens, according to Monitoring the Future survey data. As a result, a regional poison control center created a teen educational curriculum focusing on healthy prescription drug habits with the intent to measure teens' knowledge and behavior before and after education delivery.
Methods: The curriculum was mailed to 1,118 middle and high school health teachers across the center's territory. Teachers agreeing to administer the curriculum, evaluate it, and give students both a pre and post-test were supplied a $50 gift card. Exemption was obtained under the Electronic Code of Federal Regulations for protection of human subjects. Data was extracted over a five-month period. Twenty-nine teachers agreed to deliver the education and complete the companion steps; however, only 15 teachers completed all steps. The student pre-test was completed by 556 respondents, and the student post-test was completed by 433 respondents. The education was delivered in 14 counties, 12 of which have higher rates for opioid prescriptions per 100 persons than the national rate reported by the CDC (58.7 per 100). Teachers estimated that 2,343 students would be educated by the curriculum in a calendar year.
Results: Curriculum was delivered almost evenly between middle and high school students (47% middle school) to a majority rural population (67%). Evaluations revealed a high level of teacher satisfaction: 86% stated they would deliver the curriculum again. Student post-tests showed an increase in knowledge as well. Of note, 80% of teens identified friends/relatives as the major source of receiving prescription drugs for abuse, up from 51% in the pre-test; 74% identified prescription drugs as a more likely cause of death than street drugs, up from 52% in the pre-test. Recognition of how a poison control center can help guide teens through medication questions or overdoses also rose. Seventy-seven percent of students responded that poison control is a first place to contact for a medication concern, up from 48%; also, 77% reported knowing the number to poison control, up from 20%. Post-test responses showed that nearly half of students (47%) programmed the poison control number into their phones after receiving the education.
Conclusion: Adolescents 13-19 make up only 8% of the patient base for poison control centers nationwide, yet unintentional poisoning is the second leading cause of injury death among teens and young adults. After administration of the prescription drug education, teens demonstrated a greater understanding of why contacting poison control for medication related issues is an appropriate step. Likewise, evaluation of the student pre and post-tests showed improved knowledge about the harms of taking prescription drugs inappropriately. The education was delivered in at-risk counties where opioid prescribing rates outpace the national rate; however, more research is needed to determine whether the curriculum helped to decrease teen medication poisonings or increase teen interactions with poison control.

KEYWORDS Prescription drugs, adolescents, education

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204. An Analysis of Isolated Melatonin Exposures – Why Hospital Evaluation May Not Be Necessary

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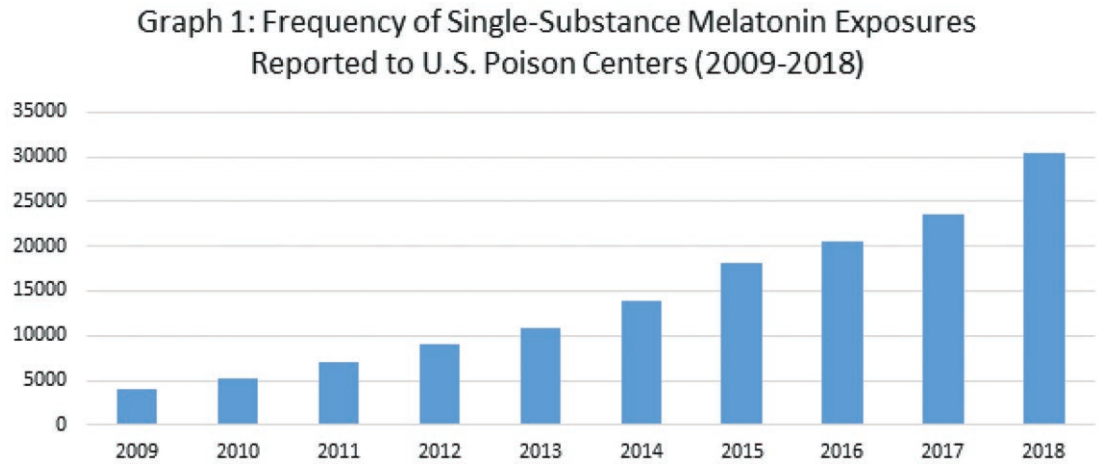
Background: Melatonin, a natural hormone that aids in the regulation of sleep cycles, is becoming an increasingly common treatment for sleep disorders and other conditions that impact an individual's sleep and wake times. Between 2007 and 2012, melatonin use in adults doubled in the U.S.. Increased use leads to increased availability and risk of symptomatic exposures, whether intentional or unintentional. In a healthy adult, a maximum response has been observed 2 hours after ingestion, with an elimination half-life of 4 hours. Current resources commonly used by U.S. Poison Control Centers (PCC) generally recommend home management, based on data from less than 800 cases from 1998-2003 and two case series of small sample size. However, ingestions of greater than 1 gram/day for 25-30 days in adults or greater than 80mg in pediatric patients do not have clear recommendations for non-healthcare facility (HCF) observation. To the authors' knowledge, no large analyses have assessed outcomes associated with known, single-substance melatonin exposures recently reported to U.S. Poison Centers.
Methods: Data from U.S. PCCs was collected through the National Poison Data System. Inclusion criteria for analysis: human, single-substance melatonin exposures that occurred between 2008-2019. Other data reviewed included: patient demographics, dose and route of exposure, symptoms, and treatment. Descriptive statistics were used to analyze pertinent outcomes.
Results: 142,493 single-substance melatonin exposures reported to U.S. PCC were included. Exposures increased 7.5-fold from 2009 to 2018 (Graph 1). 76.7% of cases occurred in patients less than 6 years of age, and 53% of patients were male. Overall, nearly all (99.8%) cases were oral ingestion and categorized as acute exposures (92.1%). Three-fourths of cases documented either an exact, estimate, or maximum possible amount ingested. The average number of tablets/capsules/caplets ingested was 12.7 (range: 0.1-1000; n = 64,083). The average dose ingested was 38.4mg (range: 75mcg-6.5gm; n = 43,284). Of known scenarios, the most common reason for ingestion was inadevertently taking or receiving a double dose (17.6%). 15% of all cases were either already in/enroute to a HCF or referred to a HCF by the PCC. The most common symptom reported after ingestion was drowsiness (11.62%). The most common therapy given was dilution (20%), followed by food (7.9%). Two-thirds of patients did not require any type of treatment. Overall, 96.5% of cases had minor or non-toxic effects documented.
Conclusion: From the analyzed data, there was evidence that melatonin exposure has low order toxicity and ingestions may be monitored at home. While clinical judgement should always be used, HCF referral can generally be avoided. Limitations to this analysis include incomplete or inaccurate documentation in PCC electronic records and reliance on contact with a U.S. PCC.



KEYWORDS Melatonin, poison center, patient management

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205. Development and Validation of Poison Control Center Trigger Reports for Quality Assurance

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Background: Quality assurance is an important component of Poison Control Centers' operations. Many poison control centers are plagued with limited funding and personnel resources, thus creativity in operational efficiency is necessary. Quality of service, including coding, is of the utmost importance. Our Poison Control Center is seeking to enhance our quality assurance program by adding Trigger Reports to help us efficiently identify and correct coding errors on a larger scale. It is prudent that these reports show a high rate of error detection, thus the rate of error detection will be used as a factor in the determination of the reports as an addition to our existing Quality Assurance Program.
Objective: To develop and validate trigger reports as a means to create meaningful and efficient Quality Assurance measures to supplement our existing Quality Assurance initiatives and improve coding and documentation.
Methods: We created several Trigger Reports using various coding combinations that would not be expected to be seen together. The reports we started with were:a. Ocular effects, null ocular routeb. Dermal symptoms, null dermal routec. Blood per rectum, null rectal routed. Call site residence, outcome patient in/in route to HCFe. Call site HCF, outcome patient referred by PCCf. Substance heroin, route ingestiong. Rectal route, reason unintentional generalh. Caller site HCF, patient not followed/minimal clinical effectsi. Respiratory bronchospasm, null inhalational routej. Dermal irritation/pain, null dermal routek. Generic code with no Poisindex codel. Unintentional general ages 13-19We ran these reports for the previous month and manually reviewed each case to determine the effectiveness of the report.
Results: Our threshold for selection was reports with a 25% or greater accuracy of error detection. The results in the selected reports ranged from 0-80% of charts identified as potential errors with actual errors that were subsequently corrected. During this process, we were able to eliminate reports that yielded no results or had limited accuracy in detecting errors (c., g., i. from above list). We then determined that the reports that had high accuracy of error detection would be added to our standing Quality Assurance measures. After our initial trial, reports were run and reports returned to staff to correct their coding errors.
Conclusion: Running monthly Trigger Reports will help our Poison Control Center improve coding accuracy by identifying cases with possible errors and correction of these errors as well as increasing awareness of common coding errors by the CSPI/SPI for overall improvement of errors being made.

KEYWORDS Trigger Reports, Quality Assurance, Coding

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206. Utilizing Multiple Data Sources to Understand the Burden of Opioid-Related Overdose

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Background/Objectives: Opioid-related overdose deaths rose steadily and more than doubled in our county between 2012 and 2017. Our objective was to first, define the scope of both fatal and non-fatal opioid-related overdose using multiple local data sources to gain a more comprehensive understanding of the interactions and gaps with health systems prior to death. We were then able to identify evidence-based strategies that could be implemented at primary, secondary, and tertiary levels of prevention.
Methods: Opioid-related data were extracted from multiple sources, including: medical examiner autopsy and toxicology reports; emergency medical services (EMS) suspected opioid-related overdose responses; a community needle exchange/naloxone distribution program regarding layperson overdose reversal attempts; regional poison center (RPC) opioid exposures; and behavioral health community recovery services regarding individuals seeking substance abuse treatment and recovery services. Demographic data were analyzed using frequencies. Trends were established for year over year data when applicable.
Results: Opioid-related overdose deaths increased steadily from 2012 to 2017 (144 to 337) with a small decrease from 2017 to 2018; 337 to 302, respectively. The majority (73%) of those who die are found to have taken more than one drug that contributed to their death. Fentanyl remains a key contributor, found to be present in 62% of overdose deaths (n = 187) in 2018. Similarly, data collected from our RPC between 2014 and 2017 showed that the majority (56%) of calls related to an opioid exposure included exposure to 2 or more substances. EMS responses where naloxone was administered and RPC calls regarding opioid exposure were highest among 20-29 year olds. ME data skewed

older, with the majority of deaths occurring between 30-59. While 88% of EMS cases were transported to a local emergency department following naloxone administration, needle exchange data revealed that only 15% of program participants called 9-1-1 following an opioid overdose reversal attempt, despite being trained to do so. Analysis of unstructured death investigation narratives demonstrated that 11% had some mention of suicidal ideation, yet 80% of the time it was undocumented. RPC case data revealed that 53% of HCF calls and 28% of all calls were suicide attempts.
Conclusions: The comprehensive approach to data collection and analysis in this project shows individual data sources often incompletely describe the opioid crisis and the data sets differentially represent certain demographics. In our county, the RPC saw high rates of exposures due to suicide attempts, which is not reflected in the ME or EMS data sets. Furthermore, the needle exchange data highlights the importance of including data sets outside of traditional healthcare units.

KEYWORDS Opioid, suicide, overdose

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207. Intentional Overdoses of First Generation Antihistamines: Not Created Equal?

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Background: First-generation antihistamines such as diphenhydramine, doxylamine, and hydroxyzine are commonly involved in intentional overdoses. Diphenhydramine is well known to cause seizures and arrhythmias in overdoses and this toxicity is frequently ascribed to the other first-generation antihistamines though such generalizations may not be accurate. We sought to examine the characteristics of single substance suicide attempts with diphenhydramine, doxylamine, and hydroxyzine reported to the National Poison Data System (NPDS)
Methods: The NPDS was queried for all single agent diphenhydramine, doxylamine and hydroxyzine exposures reported during the time period of 1/1/2007 to 12/31/2017. Only cases coded as intentional-suspected suicide were selected. All data in the NPDS was analyzed using IBM SPSS Statistics for Mac, Version 25.0 (Armonk, NY)
Results: A total of 86,429 cases were identified. Of these 25,836 were reported to be intentional – suspected suicide cases. Table 1 lists and compares key characteristics of these cases by substance and includes 95% CI where applicable. All three first generation antihistamines showed dramatic increases in exposures over the study period led by diphenhydramine which increased by 790%. Diphenhydramine had the highest % of reported seizures, reported arrhythmias and moderate/major medical outcomes. Hydroxyzine had the lowest % of reported seizures, arrythmias and moderate/major medical outcomes. Sodium bicarbonate and physostigmine were used in a higher % of diphenhydramine cases compared to the other two antihistamines. Diphenhydramine was associated with the most deaths, including a high of 8 deaths in 2017.
Conclusions: Single agent intentional suspected suicide exposures to diphenhydramine, doxylamine and hydroxyzine have increased dramatically over the last 10 years. Diphenhydramine exposures were associated with the highest rates of complications while hydroxyzine was associated with less toxicity.

KEYWORDS Diphenhydramine, Doxylamine, Hydroxyzine

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	Diphenhydramine	Doxylamine	Hydroxyzine
Number of Cases	15719	1438	8679
% Increase Over Study Period	790%	210%	429%
Age (years)[SD]	34.8 [13.9]	35.9 [14.7]	35.6 [14.0]
95% CI	34.6 - 35.0	35.1 - 36.7	35.3 - 35.9
% Female (n)	61.3% (8866)	63.6% (805)	67.9% (4990)
95% CI	60.5% - 62.1%	61.1%- 66.1%	66.9% - 68.9%
% Cases with Drowsiness Reported (n)	31.4% (4933)	33.8% (486)	28.8% (2497)
95% CI	30.7% - 32.1%	31.4% - 36.2%	27.8% - 29.8%
% Cases with Seizures Reported (n)	3.9% (617)	2.5% (36)	0.98% (85)
95% CI	3.6% - 4.2%	1.7% - 3.3%	0.94% - 1.2%
% Cases with Arrythmias Reported (n)	1.70% (151)	1.85% (9)	0.66% (33)
95% CI	1.5% - 1.9%	1.15% – 2.55%	0.46% – 0.86%
% Bicarbonate Given (n)	2.37% (372)	1.46% (21)	0.47% (41)
95% CI	2.17% - 2.57%	0.86% - 2.06%	0.37% - 0.57%
% Physostigmine Given (n)	0.82% (129)	0.07% (1)	0.06% (5)
95% CI	0.64% - 1.0%	0.0% - 0.08%	0.0% - 0.07%
% Intubation (n)	3.80% (600)	1.67% (24)	0.71% (62)
95% CI	3.5%-4.1%	1.04%-2.3%	0.5%-0.9%
% Moderate/Major Outcome Cases (n)	38.6% (6062)	29.2% (420)	16.6% (1438)
95% CI	37.8% - 39.4%	26.8% - 31.6%	15.8% – 17.4%
Deaths	32	1	4

208. Improving Blood Flow Using PC Work Flow?

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Background: Poison Centers (PC) are adapting resources and expanding skillsets toward improving partnership with public health to better serve communities. Our PC has literally and figuratively taken this to heart and brain. Utilizing the framework/technology of our contact center and building upon the strengths of our SPI's, we partnered with vascular neurologists (VN) at our host institution to create a telestroke consultative service. Our PC provides televideo medicine consults bridging VN to patient rooms over two hundred miles away.
Methods: To gain a sense of the number of anticipated stroke patients per day, we were provided historical data for several remote area hospitals. To provide twenty-four-hour coverage for the expected increase of one stroke call per day to workflow, our directors requested funding for five additional FTE SPI's. A SPI was chosen to lead the initiative acting as liaison between the director of the telestroke program, PC directors, and SPI staff. The lead SPI created a stroke manual, trouble-shooting guide, training videos, and spent over two hundred man-hours with one-on-one and group training of SPI's. SPI's already possess a repertoire of history taking, documentation, triage, and communicating vital information between nurses, physicians, and toxicologist in life and death situations. To this skillset, we educated our SPI's with an array of knowledge from neuroanatomy to emergency stroke assessment and treatment. Our SPI's were also trained to become experts in utilizing and troubleshooting the web-based application for televideo conferencing and documentation. The telestroke director coordinated training sessions with nursing staff, physicians, and radiologists at our designated hospitals to learn the web interface adapting hospital procedure to stroke

management. The PC liaison and the director of the telestroke service have worked together to troubleshoot issues generated by technology, staffing, and procedure. The role of our SPI has broadened to include "stroke specialist," coordinating stroke care as efficiently and effectively as they manage a poisoned patient.
Results/Conclusions: With the addition of five FTE SPI's, our PC launched the telestroke consultative service in October of 2018 starting with one hospital. Over the next two months, our PC increased coverage to a total of six hospitals. Stroke patients previously diverted to hospitals forty-five minutes to over an hour away can be assessed by a VN at a hospital within their community. Our SPI's have managed most stroke calls from onset to completion of charting in under one hour. Expectations of an average less than one call a day quickly increased to an average of three calls a day. In just under six months, our PC added over four hundred unique stroke alerts to our operation allowing patients to have quicker access to assessment and treatment. When every second counts to restoring blood flow, addition to our work flow helps save lives and reduce disability.

KEYWORDS Workflow, Stroke, telemedicine

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209. Public Health Services, a Case Specific Model of Shared Responsibility between a Poison Center and a Local Health Jurisdiction

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Background: Due to the highly contagious nature of measles and delay between exposure and onset of symptoms, the two investigators in this LHJ were overwhelmed with an increase in call volume regarding measles from the public and healthcare professionals. These calls absorbed the investigators time and left them unable to conduct needed public health inquiries and enact communication strategies necessary to protect the public during a measles outbreak. A pre-existing Emergency Preparedness contract between the state Department of Health (DOH) and the PC enabled the PC to function as a Public Information Call Center (PICC) established by the LHJ.
Objective: To describe the collaborative process between one Poison Center (PC) and Local Health Jurisdiction (LHJ) during a public health emergency that ensured the public had access to services needed.
Methods: Thirteen days after the first confirmed measles case, the DOH alerted the PC of a LHJ requiring PICC activation for a measles hotline. Measles activation included a dedicated measles hotline number, Just-in-time training of PC staff and contracted students to answer hotline calls. The DOH and LHJ provided the PC with measles and immunization information, scripts to follow, and triage guidelines; these were updated as needed. By 5 pm the following day, the Measles Hotline went live and the first call was answered at the PC. The PC continued taking calls for the next 54 days.
Results: The PC answered 1604 phone calls and created 1182 case records in the 54 days of activation. There were over 450 case records with one or more follow up call. The majority of cases were from the public (971), followed by healthcare professionals (151), and schools (60). The first 21 days of the activation resulted in 82.5% (n = 975) of the case records. Telephone call volume was highest on Mondays and lowest on the weekends; the busiest time of day was 8-9 am. While the majority of calls were from the same state as the LHJ (1058), there were calls received from 20 other states as well. Daily meetings between the Public Information Officers (PIO) from the DOH, LHJ, and PC ensured timely communication between the groups, and supplemented on-going email and phone conversations.
Conclusions: Poison Centers can stand up to assist Local Health Jurisdictions, or state Departments of Health, in their response efforts to link people to needed health services and assure the provision of health care. These efforts enabled the LHJ to continue to provide the community with reliable and efficient public health services, information, and response. The PC continued its mission to prevent harm through expertise, collaboration, and education and we demonstrated that public health services are a shared responsibility. It is important to note, the PC took these calls under the guise of the LHJ, not as a poison center, thus ensuring that the public did not call the PC directly and obstruct providing timely poison treatment advice.

KEYWORDS Emergency preparedness, surge coverage, public health

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210. Using Student-Contractors to assist with a Measles Outbreak Emergency Response – A Win-Win for a Poison Center, Students, and Public Health

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Background: A pre-existing Emergency Preparedness contract between the state Department of Health (DOH) and the Poison Center (PC) ensures activation of the PC for emergency response situations. A recent activation, for a telephone hotline during a measles outbreak in a local health jurisdiction (LHJ), occurred at a time when PC staff were already working overtime to cover a staffing shortage. Three recently hired pharmacists had not started their training and joined PC staff Just-in-Time instruction for measles calls. They were placed first in the queue, along with PC management, to take calls before distribution to online staff. Measles call volume was such that PC leadership determined additional staff was needed to ensure uninterrupted service with the poison helpline. Rather than utilizing student volunteers as the operational plan delineated, the decision was made to hire students as contractors to increase accountability and participation.
Objective: To demonstrate how one PC solved a staffing dilemma to meet contract deliverables without compromising poison calls.
Methods: Emails with the program requirements outlined were sent to local university pharmacy, nursing, public health, and naturopathic medicine instructors requesting student-contractors. The PC leadership team developed a contract and policy, wrote a contractor specific toxiCALL® training manual, and ensured the talking points and script provided by the LHJ were consistent with toxiCALL® templates and key cuts created for this activation to ensure simple, accurate, and consistent charting. PC staff were trained on working with and supervising the student-contractors, including reviewing charting for quality assurance. Two training sessions were scheduled; for both sessions, students were turned away due to space. SignUpGenius, a free online volunteer management tool, was used to allow self-scheduling.
Results: 24 hours after the first training session, and 36 hours after PC leadership conceived the student-contractor program, the first student-contractors were answering calls. Student-contractors worked in two hour blocks from 8am to 7pm, 29 of the 49 student-contractors trained worked at least one 2 hour shift; ten worked 10 or more hours. Two student-contractors were asked not to return due to the quality of their work. The cost of the program to the PC was 11.6 % of the total amount invoiced to the DOH per the Emergency Preparedness contract; 3% in leadership hours (65) to create the program, 2.2% in staff hours (36) to supervise the program, and 6.4% to pay the student-contractors (200.5 hours). During the 10 days they worked,

student-contractors took 42.9% (606) of all phone calls received during the 54 day activation.
Conclusions: Utilizing the student-contractors ensured the PC was able to meet required deliverables and enabled the LHJ to continue to provide the community with public health services and response. The DOH requested the PC continue with funded annual training to ensure student-contractors for future events. Students expressed this was a positive opportunity to hone their communication skills and gain experience in healthcare and public health. The PC demonstrated that public health services are a shared responsibility. This experience can be applied to other emergency response efforts.

KEYWORDS Surge, program, staffing

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211. Systematic variations in the anion gap complicate the management of poisoned patients

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Background: The measured anion gap (AG) is a historically important tool for evaluating patients with acid-base disorders. Currently, the Food and Drug Administration does not require that manufacturers of electrolyte analyzers provide reference ranges for calculated values

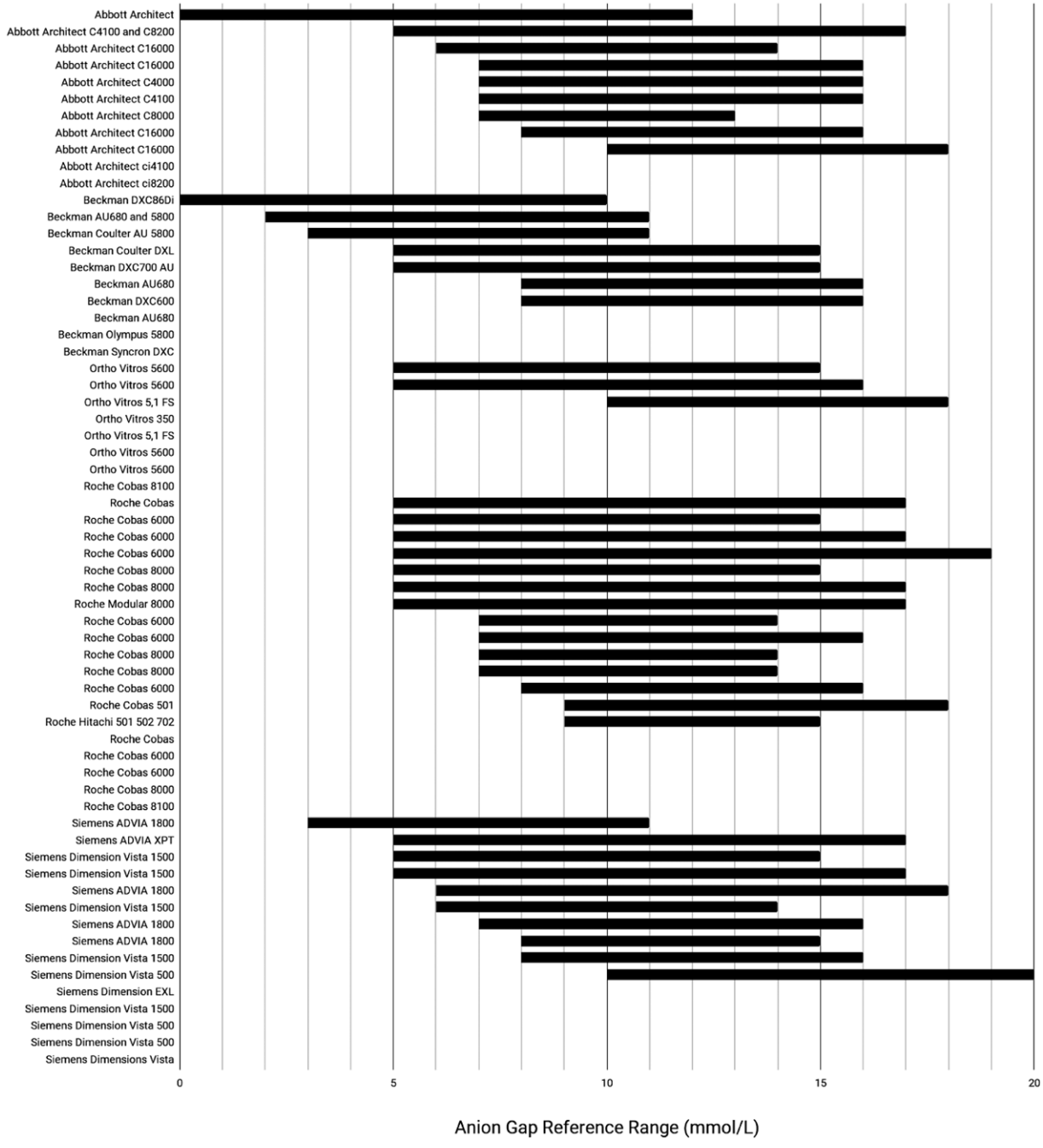

Figure 1 Anion gap reference ranges of lower New York hospitals arranged by brand of electrolyte. An absent range indicates that none is reported at that hospital.

such as the AG. Institutional variations in the AG reference range may therefore confound poison center recommendations.

Methods: We conducted a phone survey of hospitals within the catchment area of the [study territory] poison control center to determine the anion gap reference range and electrolyte analyzer(s) being used by the central laboratory of each site. Hospitals without on-site electrolyte analyzers were excluded. We conducted a follow-up survey of a convenience sample of medical laboratory directors to determine how each institution determined their local reference range. For additional context, we analyzed the reference ranges recommended by 13 medical reference texts from internal medicine, emergency medicine, nephrology, critical care, and toxicology along with their supporting references.

Results: We identified 74 hospitals in [study territory] and excluded 9 for not having an on-site electrolyte analyzer. All 63 remaining hospitals responded to the survey. There was considerable variation in the AG reference range across institutions (Figure 1). Many of the ranges had little overlap and two were mutually exclusive. Twenty-one hospitals (33%) did not report a reference range. Seven out of 12 laboratory directors responded to our follow-up survey. All respondents derived their reference range using different methodologies (Figure 2). AG reference ranges also differed between medical reference texts (Figure 3). Both nephrology textbooks and UpToDate provided guideline values but recommended that clinicians refer to their local reference ranges. Eight of the reference texts had no citation to support their recommended range.

Conclusions: We observed large variations in the AG reference range across geographically proximate hospitals in [study territory], many of which used the same electrolyte analyzer and shared patient populations. This is a quality control issue for all poison control centers because misinterpreting the AG may lead to inappropriate management recommendations. Even when the local AG reference range is known, additional information is required to define its clinical role: each institution is subject to unique constraints in determining their range with regards to time, resources, and laboratorian bias. The upper limits of the AG reference range were also lower in general internal medicine and nephrology reference texts compared to the other specialties we examined. These systematic errors amplify the considerable random error in the AG derived from propagating the statistical uncertainty of serum Na, Cl, and HCO3 measurements. Greater communication and coordination between clinicians and laboratorians about the AG will enhance the future utility of this oft used, oft misunderstood value.

Institution type	Method
Private, academic	120 healthy volunteer data
Private, academic	Normal patient values, compared to other labs using same instrument, literature review
Private, academic	120 healthy volunteer data, literature review, consultation with nephrology service
Private, academic	Historical hospital reference range, validated using normal patient data
Private, community	Single academic paper
Public, academic	Normal patient values
Public, academic	Reference text, normal patient values
Public, community	Suggested by manufacturer of electrolyte analyzer

Figure 2 Method of determining the anion gap reference range in seven New York hospitals.

KEYWORDS Anion Gap, Clinical Chemistry, Quality Control

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212. Is there a Correlation Between Increasing Insurance Coverage in Medically Vulnerable Populations and Changes in Case Volume to Regional Poison Control Centers?

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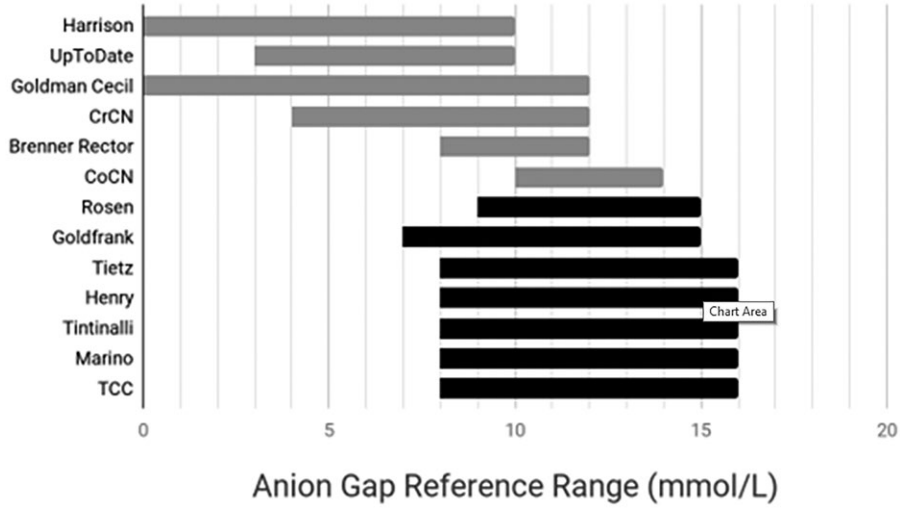

Figure 3 Anion gap reference ranges suggested by medical reference texts. Internal medicine and nephrology references are gray. Emergency medicine, critical care, and clinical chemistry references are black. Abbreviations: Harrison = Harrison's Principles of Internal Medicine 20e; UpToDate = UpToDate "Approach to the Adult with Metabolic Acidosis" accessed April 1, 2019; Goldman Cecil = Goldman-Cecil's Medicine 25e; CrCN = Critical Care Nephrology 3e; Brenner Rector = Brenner and Rector's The Kidney 10e; CoCN = Comprehensive Clinical Nephrology 6e; Rosen = Rosen's Emergency Medicine 9e; Goldfrank = Goldfrank's Toxicologic Emergencies 10e; Tietz = Tietz Textbook of Clinical Chemistry 6e; Henry = Henry's Clinical Diagnosis and Management by Laboratory Methods 23e; Tintinalli = Tintinalli's Comprehensive Study Guide 8e; Marino = Marino's The ICU Book 4e; TCC = Textbook of Critical Care 7e.

Background: The Affordable Care Act (ACA) was enacted in March, 2010 to reduce the amount of uncompensated medical care in the US by increasing access to government subsidized health insurance and requiring all residents to obtain medical coverage. Kentucky was an early adopter of Medicaid expansion allowing families making 138% of the national poverty level to receive state sponsored insurance through managed care organizations (MCOs). The ACA has decreased the overall uninsured rate in Kentucky by 62% to a low of 5.1% since its enactment. During that same time period, the regional poison control center (PCC) has seen a steady decline in case volume, both from cases originating from health care facilities (HCF) and non-healthcare facilities (non-HCF). It is unclear what contribution, if any, increasing insurance access and thereby reducing the cost of medical care, has affected utilization of the PCC.
Methods: Kentucky is divided into 15 area development districts (ADD) - counties grouped together in distinct geographical regions for services planning purposes. The change in PCC case volume from HCF and non-HCF users in each ADD was evaluated from 2012-2013 and from 2012-2016 to assess the initial change in case volume prior to Medicaid expansion as compared to the overall trend including increased coverage of vulnerable populations. An r value was determined for the decrease in uninsured persons (UP) compared to the change in non-HCF cases and the change in HCF cases via Pearson's Correlation.
Results: The mean decrease of UP in KY ADDs was 15.62% from 2012-2013 vs. a mean of 71.76% from 2012-2016. From 2012-2013 there was no correlation between mean change in non-HCF calls, -0.39%, r= 0.17) and mean change in HCF cases (-15%, r = 0.17). However, when including the time period covering Medicaid expansion, 2012-2016, there was a modest positive correlation between decreasing UP and decreasing non-HCF cases (-19.1%, r = 0.59) as well as to increasing HCF cases (24.2%, r = -0.58).
Conclusions: Although declining PCC case volume is multi-factorial, reducing the burden of cost of care may lead individuals to choose evaluation at an HCF rather than contact their regional PCC for assessment of potential poisonings. While an unintended consequence of Medicaid expansion, this correlation reveals an opportunity for PCCs to better coordinate outreach with MCOs to encourage utilization of the regional PCC to further reduce unnecessary medical cost.

KEYWORDS Poison Control, Insurance, Affordable Care Act

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213. Analysis of 10 Years of Intentional Lurasidone Overdoses Using National Poison Center Data

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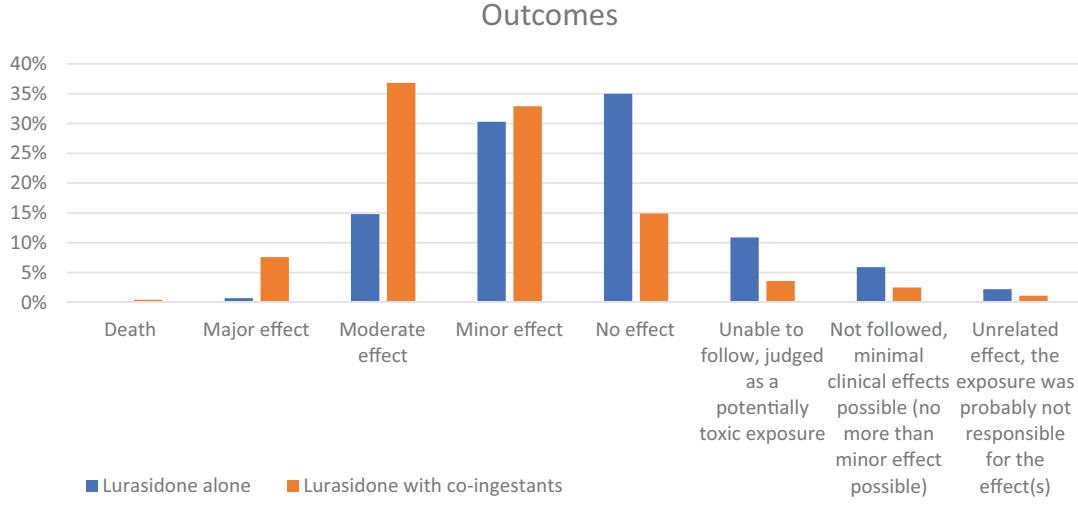
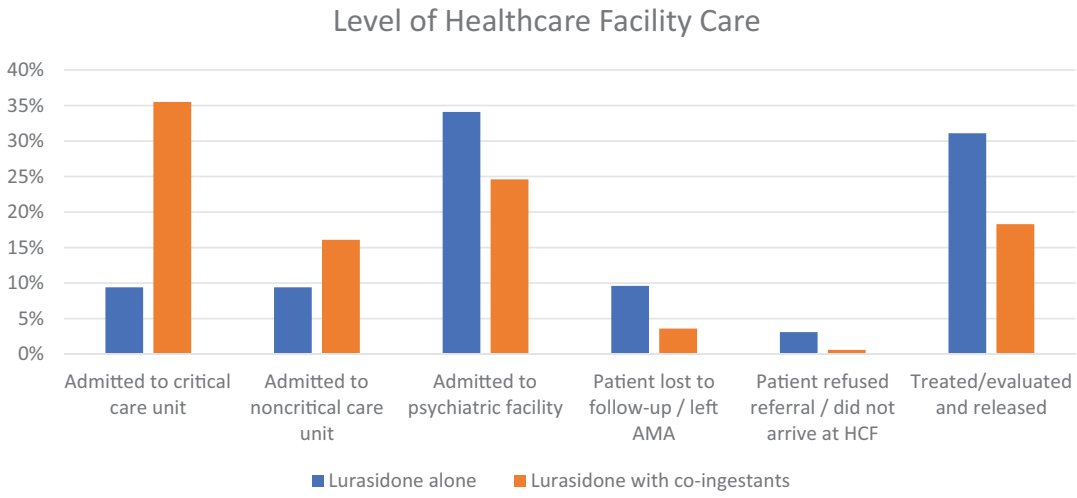
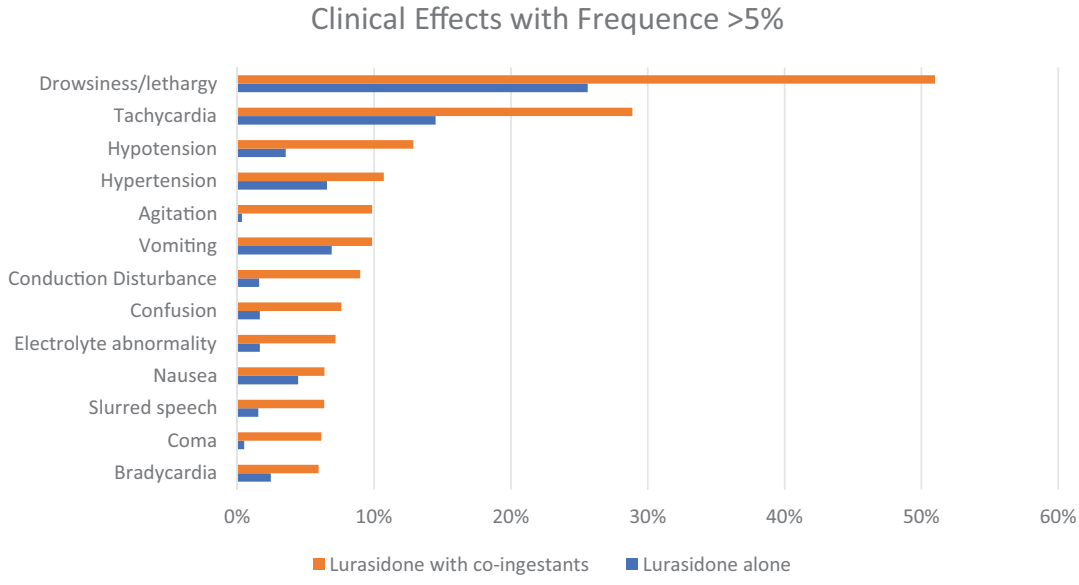
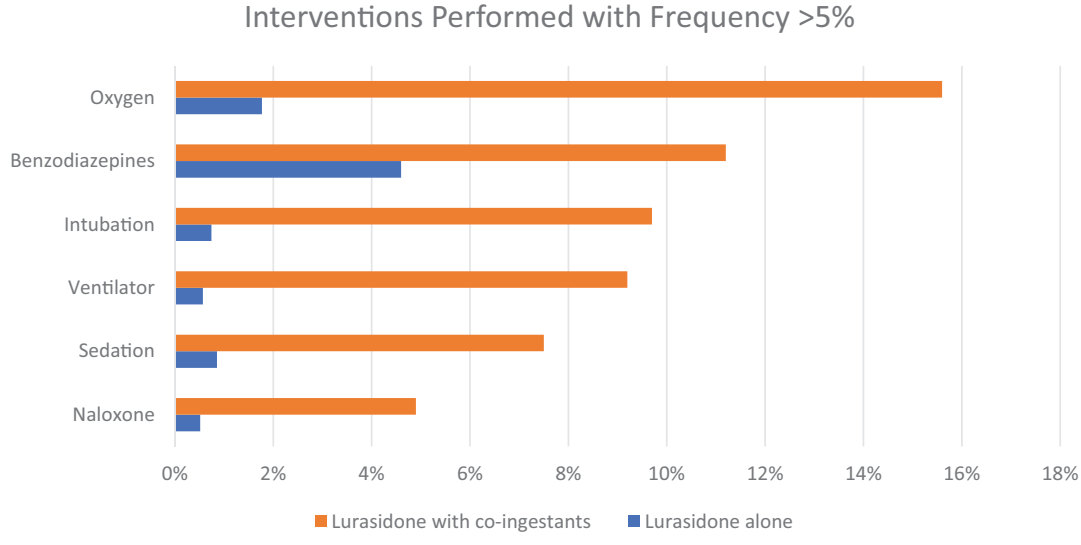
Background: The ziprasidone analogue lurasidone was FDA approved for the treatment of schizophrenia in 2010 and bipolar disorder in 2013. One overdose report has been published, describing ingestion of 1360 mg with no adverse effects by a 31-year-old man. Small sample studies suggest a low rate of major adverse effects.
Objective: To describe intentional lurasidone ingestions reported to poison centers.
Methods: Cross-sectional analysis of intentional lurasidone ingestions from the National Poison Data System (NPDS) from 1/1/2008 to 6/30/2018.
Results: There were 6852 intentional lurasidone overdoses, of which 1753 (25.6%) were single-substance. Average age was 30.9 years (range 6-86 years) and 4729 (69%) of patients were female. There were 22 deaths (0.32%), all in patients with polysubstance ingestions. Overdoses per year increased over time. In all overdose cases, major effect or death was documented in only 423 patients (6.2%), moderate effect in 2134 (31%), minor effect in 2209 (32%), and no effect in 1373 (20%). Among patients who ingested lurasidone alone, major effect was documented in 12 (0.68%), moderate effect in 259 (14.8%), minor effect in 531 (30%), and no effect in 614 (35%). Of all overdoses, 3333 (48.6%) were discharged either home or to psychiatric facilities, 2958 (43%) were admitted, 1973 (66.7%) to critical care units (ICUs). Among single-substance cases, 1143 (65%) were discharged; 328 (18.8%) were admitted, half of whom admitted to ICUs. Of all the patients admitted to ICUs, 1101 (55.8%) received either no therapy or intravenous fluids alone. The most common adverse effects were drowsiness (3049, 44.5%), tachycardia (1726, 25.19%), hypotension (718, 10.48%), hypertension (661, 9.65%), and vomiting (624, 9.11%). Rates of each of these adverse effects were approximately half in the group that ingested lurasidone alone, and hypotension occurred in only 62 (3.54%) of patients. Conduction disturbance was noted in 486 patients (7.09%), and in only 28 patients (1.6%) who ingested lurasidone alone.
Discussion: These data suggest that major effects are uncommon from lurasidone ingestions alone. Despite a high rate of admission to critical care units, a substantial proportion received either no therapy or only intravenous fluids. However, the indications for admission




Level of Healthcare Facility Care
40%
35%
30%
25%
20%
15%
10%
5%
0%
Admitted to critical care unit
Admitted to noncritical care unit
Admitted to psychiatric facility
Patient lost to follow-up / left AMA
Patient refused referral / did not arrive at HCF
Treated/evaluated and released
Lurasidone alone
Lurasidone with co-ingestants


Clinical Effects with Frequence >5%
Drowsiness/lethargy
Tachycardia
Hypotension
Hypertension
Agitation
Vomiting
Conduction Disturbance
Confusion
Electrolyte abnormality
Nausea
Slurred speech
Coma
Bradycardia
0%
10%
20%
30%
40%
50%
60%
Lurasidone with co-ingestants
Lurasidone alone


Interventions Performed with Frequency >5%
Oxygen
Benzodiazepines
Intubation
Ventilator
Sedation
Naloxone
0%
2%
4%
6%
8%
10%
12%
14%
16%
18%
Lurasidone with co-ingestants
Lurasidone alone

to critical care settings remain unknown and may not be included in NPDS data, e.g. risk of loss of airway protection or need for monitoring that is not available in other units. Compared to studies of ziprasidone, rates of drowsiness and tachycardia were comparable, but conduction disturbances were far less frequent. Compared to all single-substance atypical antipsychotic ingestions, regardless of intent, intentional lurasidone ingestions had fewer major effects, moderate effects, or death (22.62% vs. 15.46%).

Conclusion: Lurasidone ingestions alone were not associated with a high rate of major adverse effects, and there have been no documented deaths. Admissions to critical care units were common, but critical interventions were not. Conduction disturbances were rare. Ingestion of lurasidone with other substances, however, increased the rate of complications.

	Lurasidone alone	Lurasidone with co-ingestants		Lurasidone alone	Lurasidone with co-ingestants
Death	0.00	0.00	Admitted to critical care unit	0.094	0.355
Major effect	0.01	0.08	Admitted to noncritical care unit	0.094	0.161
Moderate effect	0.15	0.37	Admitted to psychiatric facility	0.341	0.246
Minor effect	0.30	0.33	Patient lost to follow-up / left AMA	0.096	0.036
No effect	0.35	0.15	Patient refused referral / did not arrive at HCF	0.031	0.006
Unable to follow, judged as a potentially toxic exposure	0.11	0.04	Treated/ evaluated and released	0.311	0.183
Not followed, minimal clinical effects possible (no more than minor effect possible)	0.06	0.03			
Unrelated effect, the exposure was probably not responsible for the effect(s)	0.02	0.01			

	Lurasidone alone	Lurasidone with co-ingestants		Lurasidone alone	Lurasidone with co-ingestants
Bradycardia	0.02	0.06	Naloxone	0.005	0.049
Coma	0.01	0.06	Sedation	0.009	0.075
Slurred speech	0.02	0.06	Ventilator	0.006	0.092
Nausea	0.04	0.06	Intubation	0.007	0.097
Electrolyte abnormality	0.02	0.07	Benzodiazepines	0.046	0.112
Confusion	0.02	0.08	Oxygen	0.018	0.156
Conduction Disturbance	0.02	0.09	Charcoal, single dose	0.144	0.159
Vomiting	0.07	0.10	IV fluids	0.246	0.557
Agitation	0.00	0.10			
Hypertension	0.07	0.11			
Hypotension	0.04	0.13			
Tachycardia	0.14	0.29			
Drowsiness/ lethargy	0.26	0.51			

KEYWORDS Lurasidone, atypical antipsychotic, intentional overdose

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214. High Grade Gastric Injury After Household Bleach Ingestion

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Case: A 16-year-old male with a history of depression presented to the Emergency Department after ingesting household bleach approximately 45 minutes prior to arrival. Medics who transported the patient to the hospital confirmed the presence of an empty bottle of bleach next to the patient, who stated he drank "less than 275 mL" of the bleach in an attempt at self-harm. On arrival, he was complaining of odynophagia, chest pain, nausea and vomiting. His vital signs and physical exam were unremarkable, including no evidence of oropharyngeal lesions. However, he felt he was unable to tolerate his own secretions and was spitting clear white sputum. A chest radiograph revealed no abnormalities, and his laboratory evaluation was normal with the exception of a mildly elevated anion gap of 19, a potassium of 3.2 mmol/L and a urine toxicology screen that was positive for tetrahydrocannabinol. Because he was unable to tolerate his secretions, he was placed on IV fluids and admitted to the pediatric floor. Gastroenterology was consulted, who subsequently performed esophagogastroduodenoscopy (EGD). EGD performed 26 hours post-ingestion revealed esophageal hyperemia, as well as diffuse gastric ulceration and necrosis (images available). The duodenum was normal. He was started on sulcrafate and, after having his diet slowly advanced, was subsequently transferred to the psychiatric floor.

Discussion: Household bleach is a dilute sodium hypochlorite solution, with a concentration of 4-6% and a pH of 11. It is generally considered a non-harmful ingestion. Two previous case series of patients who had ingested household bleach reported no incidents of high-grade upper gastrointestinal tract injury on EGD. One case does describe severe esophageal injury, with perforation and mediastinitis in an adolescent who had reportedly ingested a 4.5% sodium hypochlorite solution, but, to our knowledge, severe gastric injury has never been reported. The patient presented here had no evidence of oropharyngeal injury, but on endoscopy was found to have mild esophageal injury (grade 1) and severe gastric injury (grade 3a).

Conclusion: Historically, household bleach is considered a non-harmful ingestion. Treatment is generally supportive and EGD is rarely recommended. However, we report the case of a patient who had reportedly ingested a large amount of household bleach and was clearly symptomatic. Despite a lack of concerning findings on physical exam, but because of symptomatology, EGD was performed and revealed a grade 3a gastric injury, with diffuse ulceration and necrosis. While severe injury is rare after household bleach ingestion, this case demonstrates that it remains a possibility. Symptomatic patients, as well as older patients who may have ingested larger quantities of bleach in an attempt at self harm may require prolonged observation as well as EGD to determine the severity of upper gastrointestinal tract injury.

KEYWORDS Bleach, gastric, injury

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215. Strange Mayo: Unintentional ingestion of unknown pain cream mistaken for mayonnaise

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Background: Toxicity associated with oral ingestion of topical analgesic creams is not well documented in the literature. The components of compounded creams are not standardized and may include different combinations of drugs. Treatment is targeted to the implicated agent or agents and may include administration of an antidote and supportive care. Here we present a case of accidental ingestion of an unknown analgesic cream.
Case Report: A 47-year-old female health aide and her 86-year-old female client were brought into the Emergency Department, both lethargic, bradycardic, and bradypnic, with pinpoint pupils and dry mucous membranes. All other vital signs including blood pressures were normal. One hour prior, both patients had eaten sandwiches prepared by the aide. An unlabeled "pain cream" from Honduras was kept in the fridge. The aide had mistaken it for mayonnaise. Initial Emergency Department management included fluids and cardiac monitoring. Naloxone was given, with transient increase in heart rate but no effect on mental status. Both patients required MICU admission for hemodynamic support with atropine and dopamine for bradycardia in the 30s-50s and mean arterial pressures in the low 50s. The 86-year-old patient became acutely obtunded in respiratory distress overnight and required intubation.Poison Control was contacted. Given presentations, baclofen, clonidine, and lidocaine levels were sent. Empiric lipid emulsion was administered for presumed lidocaine toxicity with no effect. Lidocaine levels were negative, however serum clonidine and baclofen levels were high. Baclofen levels resulted at 112ng/ml for the 47-year-old patient and 162ng/ml for the 86-year-old patient. Only the 86-year-old patient had clonidine levels drawn, which resulted at 6.94ng/ml. Both patients were transferred from the MICU to the general medical floor and discharged after vitals normalized and mental status returned to baseline.
Case Discussion: Pharmacokinetics and pharmacodynamics of oral and topical preparations are well documented; however, gastrointestinal absorption of topically prepared creams is not well understood with limited case reports available in the literature. Clonidine is a presynaptic α2-agonist which decreases sympathetic outflow. Analgesic effects of topical clonidine are thought to be caused by modulation of the α2 receptors on nociceptors in the epidermis. The immediate release formulation of clonidine reaches peak concentrations in 3 to 5 hours and the plasma half-life ranges from 12 to 16 hours; however, this can be prolonged up to 41 hours in patients with severe renal impairment. Adverse clinical effects include hypotension and hypertension, bradycardia, CNS depression, respiratory depression and miosis.
Conclusion: With increasing prescriptions for compounded analgesic creams, emergency personnel must be cognizant of possible toxicities associated with accidental ingestion. In the setting of unknown ingestion, in this case an unlabeled foreign cream, all possibilities must be considered. Supportive care must be started early and if contents of the cream are unknown, drug levels for the most common ingredients including clonidine, baclofen, lidocaine, gabapentin, ketamine, and tricyclic antidepressants should be obtained for analysis.

KEYWORDS Overdose, Clonidine, Toxicology

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216. Hold the Mayo: Unintentional ingestion of a topical compounded analgesic

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Background: Compounded topical analgesics are increasingly used for pain management but reported toxicity is rare. These medications vary in composition of substances and respective concentrations making their presentations unpredictable. Here we describe two unintentional ingestions of a topical analgesic compound mistaken for a condiment.
Case Report: A 47-year-old female, home health aide, and her client, an 86-year-old female, were brought into an emergency department, lethargic, hypotensive, bradycardic, bradypneic with pinpoint pupils and dry mucous membranes. The aide was relatively healthy with only hyperlipidemia. The client had chronic obstructive pulmonary disorder, hypertension, and cardiovascular disease. The aide had prepared sandwiches using generous amounts of a white substance in an unlabeled container in the refrigerator resembling mayonnaise. One hour prior to presentation the two had ingested the sandwiches noting a strange taste with tingling sensation. The client's family reported that this cream was obtained in Honduras and was used topically for pain. Initially, both patients were given intravenous fluids. Naloxone 0.4 mg was given to both patients with improvements in bradypnea but with a continued depressed mental status. Blood pressures (BP) began down-trending to 92/78 mmHg in the aide and 115/65 mmHg in the client. They were given atropine with improvements in heart rate. Both had improvements in BP and were admitted to an intensive care unit. Both received intravenous fat emulsion due to concern for lidocaine toxicity with no improvement. Both patients were started on dopamine infusions for recurring bradycardia. The client was intubated for respiratory failure. Poison control was consulted on day 2. An imidazoline and baclofen were suspected components of this topical preparation, given the presenting symptoms, longevity and response to naloxone. The patients were maintained with supportive care. On hospital day 2, serum concentrations of lidocaine, clonidine and baclofen were obtained for confirmation. Lidocaine was undetectable. Baclofen was elevated at 162 ng/mL for the client and 112 ng/mL for the aide [therapeutic range: 80-400 ng/mL]. The client's serum clonidine concentration was 6.94 ng/mL [therapeutic: <4.5 ng/mL]; the sample sent for the aide was lost. Both patients made full recoveries.
Case Discussion: Topical analgesic preparations are not standardized and setups for severe medication errors. Many components with varying concentrations can be found in compounded topical analgesics including but not limited to: clonidine (0.1-0.2%), local anesthetics, cyclic antidepressants, ketamine (1-20%), NSAIDs, baclofen (1-5%), salicylates, gabapentin, anticonvulsants, menthol, capsaicin, and camphor. Toxic doses of clonidine, baclofen, and ketamine are easily achieved with minimal ingestion based on reported concentrations in the literature. Concentrations demonstrated here were mildly elevated, but certainly much higher on presentation and confirm this exposure.
Conclusion: Medication errors occur often with improper storage or labeling. Even small ingestions of these compounded topical analgesics lead to severe toxicity. Serum concentrations offer little utility in management with the exception of salicylates. Providers should be cognizant of components found in these preparations and treat accordingly with appropriate decontamination, antidote therapy where applicable, and adequate supportive care. Prevention should also be a high priority with efforts focused on improving labeling, handling, and storage.

KEYWORDS Topical analgesics, clonidine toxicity, compounded medicines

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217. A Single Poison Control Center's Characterization of Abuse and Misuse of Gabapentin and Pregabalin Exposures 2012-2017

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Background: Gabapentin and pregabalin abuse and misuse have been an increasing concern as an evolving drug of abuse. This study seeks to characterize exposures of gabapentin and pregabalin reported to a single poison control center from 2012-2017.
Methods: This study was a retrospective chart review of human exposures (n = 190) to gabapentin and pregabalin by a single poison control center from 2012 to 2017. Primary outcomes of gender, age, and medical outcome were analyzed using two-tailed z-scores with calculated p-values to determine significant changes during the study period. This study is IRB-approved.
Results: Gabapentin abuse and misuse increased significantly ($p < 0.00001$) while pregabalin did not ($p = 0.17068$) from 2012-2017. The increase in gabapentin exposures was noted in both sexes and in all individuals ages 20-59 years old. The most likely individual to abuse or misuse gabapentinoids in our study population was a female aged 30-50. Although pregabalin exposures did not increase as a whole, there was a noted significant increase in the 40 to 49-year-old age group. Medical outcomes were not noted to change with either xenobiotic during the study period. Limitations of this study include that it is a single-center study of cases voluntarily reported to a poison control center.

Table 2 Incidences of scorpion stings across regions.

Regions	Number of studies	Total Number of stings	Total Study durations	incident per year
Central	8	9,794	48 years	1,958 per year
Northern	5	4,553	4 years	4,287 per year
Southern	6	2,378	22.3 years	336 per year
Western	3	147	7.8 years	319 per year

Table 1 Methodological characteristics of eligible studies.

	(Family name year)	Article title	Study region	Study design	Study period
1	(Neale 1989)	Scorpion sting in eastern Riyadh	Riyadh (Central)	Retrospective analysis	5 years
2	(El amin et al 1990)	Hematological and Biochemical Findings in Scorpion Stung Children	Medina (Western)	Prospective analysis	4 months
3	(El-Amin et al 1991)	Scorpion sting: a management problem	Medina (Western)	Retrospective analysis	3 years
4	(Annobil et al 1991)	Intracranial hemorrhages after Nebo hierochonticus scorpion sting.	Abha (Southern)	Case report	-
5	(Kumar et al 1992)	Scorpion venom cardiomyopathy	Baha (Southern)	Retrospective analysis	4 months
6	(Annobil 1993)	Scorpion stings in children in the Asir Province of Saudi Arabia	Asir (Southern)	Prospective analysis	5 years
7	(Groshong 1993)	Scorpion envenomation in eastern Saudi Arabia	Open desert (Northern)	Prospective analysis	4 months
8	(Ismail 1994)	The treatment of the scorpion envenoming syndrome:the saudi experience with serotherapy	12 multiple regions	Retrospective analysis	8 months
9	(Dittrich et al 1995)	Scorpion sting syndrome: A ten year experience	Riyadh (Central)	Retrospective analysis	10 years
10	(Mahaba et al 1996)	Scorpion sting, is it a health problem in Saudi Arabia? Evaluation of management of 820 cases	Hail (Northern)	Prospective analysis	6 months
11	(Mahaba 1997)	Scorpion sting syndrome: epidemiology, clinical presentation and management of 2240 cases	Hail (Northern)	Retrospective analysis	15 months
12	(Al-Rashed et al 1999)	Scorpion sting in children from northwestern area of Riyadh, Saudi Arabi	Riyadh (Central)	Retrospective analysis	15 years
13	(Gajre et al 1999)	Scorpion Envenomation in Children: Should All Stings be Given Antivenom?	Baha (Southern)	Retrospective analysis	5 years
14	(Soormo et al 2001)	A clinical evaluation of the effectiveness of antivenom in scorpion envenomation	Baha (Southern)	Retrospective analysis	12 years
15	(Dittrich et al 2002)	Cardiac arrest following scorpion envenomation	Riyadh (Central)	Case report	-
16	(Al-Sadoon et al 2003)	Epidemiological study of scorpion stings in Saudi Arabia between 1993 and 1997	11 multiple regions	Retrospective analysis	5 years
17	(Hamed 2003)	Treatment of the scorpion envenoming syndrome: 12-years experience with serotherapy	Riyadh (Central)	Retrospective analysis	6 years
18	(Al-Asmari et al 2004)	Scorpion sting syndrome in a general hospital in Saudi Arabia	Riyadh (Central)	Retrospective analysis	5 years
19	(Jahan et al 2007)	Scorpion stings in Qassim, Saudi Arabia—A 5-year surveillance report	Qassim (Central)	Retrospective analysis	5 years
20	(Jarrar et al 2008)	Epidemiological aspects of scorpion stings in	6 Northern regions	Retrospective analysis	2 years
21	(Al-Asmari et al 2008)	Role of prazosin on cardiovascular manifestations and pulmonary edema following severe scorpion stin	Asir (Southern)	Case report	-
22	(Al-Asmari et al 2012)	Clinical aspects and frequency of scorpion stings in the Riyadh Region of Saudi Arabia	Riyadh (Central)	Retrospective analysis	2 years
23	(Al-Hemairi et al 2013)	Scorpion envenomation :an experience with children at Rabigh General Hospital, ksa	Rabegh (Western)	Prospective analysis	4.5 years
24	(Mallick et al 2018)	Priapism in Scorpion Stings within the Kingdom of Saudi Arabia: A Case Report	Tabuk (Northern)	Case report	-

Table 2 Distribution of age, gender and scorpion species characteristics across various regions.

	Central region	Northern region	Southern region	Western region
Age category				
Pediatrics	488 per year	977 per year	176 per year	321 per year
Adults	1,139 per year	3,310 per year	-	-
Missing references	(17)	-	(14)	-
Gender				
Male	1,069 per year	2,610 per year	99 per year	191 per year
Female	558 per year	1,677 per year	77 per year	127 per year
Missing references	(17)	-	(14)	-
Scorpion type				
Leiurus Quinquestriatus	27.2 per 100 cases	38.6 per 100 cases	17 per 100 cases	52 per 100 cases
Androctonus crassiccauda	27 per 100 cases	34.7 per 100 cases	1 per 100 cases	22 per 100 cases
Unknown	10.4 per 100 cases	13.0 per 100 cases	3 per 100 cases	18 per 100 cases
Missing references	(3,4,5,7,12,13,14,15,18,23,24)	(3,4,5,7,12,13,14,15,18,23,24)	(3,4,5,7,12,13,14,15,18,23,24)	(3,4,5,7,12,13,14,15,18,23,24)

*Exclude multiple region studies. Missing: figures not reported or restricted articles.

Table 3 Classification of clinical manifestations as reported by the studies.

Classification	Manifestations
Asymptomatic victims	No signs or symptoms Suspected scorpion stings
Class I envenomation (Minor local manifestations at sting site)	Pain, Redness (Hyperemia), Scarification, Itching/burning sensation, Swelling
Class II envenomation (Minor to moderate systemic manifestations involving other body systems)	Gastrointestinal symptoms *(vomiting, Hypersialorrhea, Abdominal pain, Diarrhea)* Rhinorrhea, Lacrymation, Restlessness, Fever, Sweating, Hypothermia, Shivering, Confusion, Myosis, Hallucination, Priapism
Class III envenomation (Life threatening potentially lethal manifestations)	Major hyperthermia, Bronchial congestion, Acute pulmonary edema, Tachypnea, Bradypnea, Tachycardia, Bradycardia, Myocardial toxicity, Collapse, Convulsion, Coma, death

Table 4 Pattern and outcomes of scorpion anti-venom administration.

	References	Number of cases
Anti-venom administration	1,2,3,6,8,9,10,11,12,13,14,15,17, 18,20,21,22,23,24	5,771
Performance of sensitivity tests prior anti-venom administration	10,20,8,11,13	2,521
Time of administering anti-venom	20	1372(15-120 minutes)
	8	602(<1hour);65(2-6hours)
	11	1,832(<3 hours)
	2	81(<120min)
Dose of anti-venom administered.	2	1ml-2ml
	24	2 amps
	10	1 amp and 5 amps
	8,15,17,20,21	5 amps
	13	5;10;15-20 amps
	11	1-4;≥5 amps
	6	5-20 ml
	23	3;5;6;10;>10amps
	22	<5;5amp
Allergic reactions to scorpion anti-venom.	1,8,10,11,13,17,20	658

Table 5 An overview of the revised studies.

(Family name year)		
(Neale 1989)	Study objectives	An emergency department log reviewed for a five-year period and 205 cases of "scorpion sting syndrome" were found.
	Main conclusion	Although frequently benign, cases of scorpion sting as observed may not be innocuous. Severity is likely to be greater in children and in adults with preexisting hypertension.
(El amin et al 1990)	Study objectives	The biochemical and hematological profiles of 96 children admitted to the hospital during one year with scorpion stings were analyzed.
	Main conclusion	No hematological problem was encountered. Hypocalcemia occurred in some patients but did not pose a clinical management problem. Cardiotoxicity was an important complication and ECG examination showed features of myocarditis but did not reveal signs of any electrolyte disturbances.
(El-Amin et al 1991)	Study objectives	Admissions and deaths resulting from scorpion sting over 3 years were analysed. Features that indicated the severity of the clinical condition were identified.
	Main conclusion	Poor management of fluid therapy was responsible for the frequently unsatisfactory resolution of envenoming. The role of antivenom is questioned and controversy regarding the most appropriate sedative to use is not resolved. An in-depth study of these management issues is urgently required.
(Annobil et al 1991)	Study objectives	Case study: A 3-year-old boy, who was previously well, developed acute pulmonary oedema, fundal haemorrhages, temporary blindness and deafness following a Nebo hierochonticus scorpion sting.
	Main conclusion	Cranial CT scan 8 days after admission showed bilaterally symmetrical multiple hyperdense areas consistent with multiple haemorrhages. Cranial CT scan 8 months showed resorption of the haemorrhages.
(Kumar et al 1992)	Study objectives	Cardiac function was evaluated by serial echocardiography in 30 children affected by scorpion stings.
	Main conclusion	Myocardial toxicity is a common and serious complication of scorpion stings in children. Systolic function appears to be affected predominantly. Serial echocardiography is useful to follow changes in left ventricular function. Patients who fail to improve within 24 to 48 hours require particularly close observation.
(Annobil 1993)	Study objectives	There appear to be regional variations in the clinical effects of scorpion stings, due to the different species of scorpions found in the various regions of the Arabian Peninsula.
	Main conclusion	Neurological manifestations were the most prominent. One patient had disseminated intravascular coagulopathy and intracranial hemorrhages. One death due to severe pulmonary edema and congestive heart failure. Antivenom was given in all cases with systemic manifestations of envenomation.

(Continued)

Table 5. Continued.

(Groshong 1993)	Study objectives	Collect data on the initial presentation of victims of scorpion envenomation during the deployment of US forces to Saudi Arabia in support of Operation Desert Shield.
	Main conclusion	In adult patients without serious prior adverse medical conditions, intervention is best limited to supportive measures.
(Ismail 1994)	Study objectives	A protocol for treatment of scorpion sting based mainly on antivenom therapy was applied nation-wide in Saudi Arabia. At least 5 x 1 ml ampoules of antivenom diluted in 20-50 ml saline were injected slowly i.v. in all patients confirmed to have scorpion stings or suspected stings with systemic manifestations.
	Main conclusion	Incidence of pulmonary edema, hypertension, hypotension, cardiac dysrhythmias and neurological symptoms following antivenom administration was very slight. Hospital stay was reduced; most patients were symptom-free within 1-2 days. Early reaction to antivenom was lower than expected and low in severity.
(Dittrich et al 1995)	Study objectives	Assess the risk of morbidity and mortality following scorpion envenomation, define patient demographics of the study group and identify high risk groups for systemic toxicity.
	Main conclusion	The great majority of patients can be treated with analgesia, local ice application, and observation dictated by clinical findings. Usage of antivenin should be restricted to patients with signs of serious systemic toxicity.
(Mahaba et al 1996)	Study objectives	To study the incidence of scorpion stings at Hail region and to evaluate the prognosis in relation to the dose of antivenom received.
	Main conclusion	Treatment with 5 ampoules was not shown to be superior to one ampoule antivenin. High incidence of scorpion stings sets a need to start preventive community programs to decrease the incidence of stings.
(Mahaba 1997)	Study objectives	All cases of scorpion stings (2240)†that attended all primary health care centres and hospitals were recorded and analyzed.
	Main conclusion	Severity of symptoms/signs were marked among infants. Guidelines for the management of scorpion stings are suggested. Local treatment of stings affecting infants and preschool children is IMPORTANT..
(Al-Rashed et al 1999)	Study objectives	To assess the clinical severity of envenomation in children by scorpion species and the potential benefit of antivenin administration.
	Main conclusion	Higher occurrence of stings in children (>3 years) and males. Lower half of the body was significantly affected. Manifestations of illness were not severe. Antivenin therapy didn't affect the outcome of the illness.
(Gajre et al 1999)	Study objectives	To determine whether all scorpion stings need treatment with antivenom, or whether the cases can be categorized so that asymptomatic ones are not given antivenom.
	Main conclusion	Children who were given serum had fewer complications, and shorter hospital stay. No deaths reported. The difference between the two groups with regard to morbidity and hospital stay was highly significant.
(Soormo et al 2001)	Study objectives	Cases of scorpion stings were admitted and treated with 0 to 1 ampule of scorpion antivenom then compared to another group after the aAntivenom policy was changed to 5 or more ampules of antivenom.
	Main conclusion	Mortality rate fell down, and the occurrence of pulmonary edema decreased. The excellent outcome is attributed to scorpion antivenom.
(Dittrich et al 2002)	Study objectives	A case of cardiac arrest, following scorpion envenomation in a 51-year-old male. This complication of envenomation is highly unusual.
	Main conclusion	Excess sympathetic stimulation and direct effect of venom on the myocardium are responsible for the most serious cardiac manifestatioins of toxicity. Because scorpion stings are associated with known instances of serious morbidity and mortality, the place for antivenin should be recognized.
(Al-Sadoon et al 2003)	Study objectives	Evaluated the epidemiological aspects of scorpion stings in different areas of Saudi Arabia.
	Main conclusion	The study showed that there is a low threat to life despite the high number of stings; this is a result of the availability of medical facilities and the multi-center antivenom use in different areas of Saudi Arabia.
(Hamed 2003)	Study objectives	Analysis of the outcome of a protocol for the treatment of scorpion stings based mainly on antivenom therapy was applied nationwide in Saudi Arabia.
	Main conclusion	The incidence of severe venom toxicity following antivenom administration was almost negligible. The length of hospital stay was reduced. The early reaction to antivenom administration was lower than expected.
(Al-Asmari et al 2004)	Study objectives	Evaluate the incidence of scorpion stings and to draw the attention of clinicians, concerning the dilemma of scorpion sting syndrome and its management in the Kingdom of Saudi Arabia (KSA).
	Main conclusion	The beneficial effect of antivenom in protecting victims against scorpion stings is still questionable. Higher risk groups of systemic toxicity were either those with ages less than 10 years or greater than 50 years.
(Jahan et al 2007)	Study objectives	Reviewed and analyzed 5-year (1999–2003) surveillance data of scorpion stings in Qassim, Saudi Arabia.
	Main conclusion	Male-to-female ratio for scorpion stings was 1.9:1. The mean age for cases was 23 ± 17 years. The scorpion stings had a higher incidence in the months of May–October.
(Jarrar et al 2008)	Study objectives	Review and analyses of scorpion sting cases that presented to the emergency department of the hospitals and medical centers in Al-Jouf
	Main conclusion	Findings indicate that scorpion stings are common in Al-Jouf Province, especially during the summer. The overall threat to human health was found to be low.
(Al-Asmari et al 2008)	Study objectives	Report on the ameliorating effects of prazosin on the cardiovascular CV manifestations and pulmonary edema PE after treatment with antivenom failed to improve the conditions of scorpion stung patients.
	Main conclusion	Polyvalent scorpion antivenom may not be beneficial in all cases of envenomation. Prazosin may be an effective alternative to treat scorpion sting cases with cardiovascular manifestations and pulmonary edema..
(Al-Asmari et al 2012)	Study objectives	To study the clinical aspects and frequency of scorpion stings in Riyadh region of Saudi Arabia.
	Main conclusion	There is a predominance of weak venomous scorpion species in central region. The protocol of managing patients with antivenom irrespective of the intensity of manifestations warrants a detailed review.
(Al-Hemairi et al 2013)	Study objectives	To evaluate the epidemiological and clinical characteristics of scorpion envenomation in children.
	Main conclusion	Although most of scorpion envenomations in children have a good prognosis, severe complications and death may occur
(Mallick et al 2018)	Study objectives	To report a 2-year-old Saudi boy who presented with an acute onset of asymmetric pulmonary oedema, moribund state and priapism.
	Main conclusion	The presentation of severe or heavy scorpion intoxication mimics the presentation of sepsis.

Conclusions: Gabapentin, a generic drug, was noted to significantly increase during the study period in all ages. Pregabalin, which becomes available as a generic medication on June 30, 2019, only increased in a single subset of the study population (ages 40-49 years-old). Gabapentin appears to be a broadly evolving drug of abuse and misuse in our study population.

KEYWORDS Gabapentin, Pregabalin, Abuse

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218. Alternative dosing regimens of intravenous N-acetylcysteine for acetaminophen toxicity: A systematic review

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Background: The intravenous N-acetylcysteine (IV N-AC) dosing regimen approved by the FDA for the treatment of acetaminophen toxicity is somewhat complex, requiring three separate infusions administered over 21 hours. This 3-step regimen can potentially contribute to medication errors or suboptimal clinical outcomes. Several recent investigations have proposed modified dosing regimens of IV N-AC for the treatment of acetaminophen toxicity. We conducted a systematic review of the literature describing off-label IV N-AC dosing regimens to evaluate patient-oriented outcomes associated with these alternative methods.
Methods: The Pubmed, Scopus, and Web of Science databases were queried for articles published between January 1900 through November 2018 using combinations of the following search terms: acetaminophen, paracetamol, N-acetylcysteine, acetylcysteine, intravenous, and infusion. For an article to be included in this systematic review, an off-label method of administering IV N-AC must have been utilized for the treatment of acetaminophen toxicity. Only those articles evaluated in humans in the form of case reports, case series, or controlled trials with full text available, and published in the English language were included in this systematic review.
Results: A total of 413 articles were screened for possible inclusion, 13 of which met inclusion criteria. The study types included prospective clinical trials (7 studies), retrospective chart reviews (4 studies), and case reports (2 studies). The most common alternative method for infusion of IV N-AC was through the administration of a two-bag regimen (200 mg/kg infused over four hours, followed by 100 mg/kg infused over 16 hours). Similar rates of hepatotoxicity were observed with modified dosing regimens of IV N-AC with lower rates of both medication errors and adverse drug reactions.
Conclusions: Literature describing the safety and efficacy of modified IV N-AC dosing regimens for the treatment of acetaminophen toxicity is limited. Despite variability in the modified dosing regimens of IV N-AC for the treatment of acetaminophen toxicity, such strategies may be a viable alternative with improved safety profiles and similar efficacy outcomes in comparison to the traditional regimen.

KEYWORDS N-acetylcysteine, acetaminophen toxicity, Modified dosing regimen

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219. Pediatric THC Ingestion Causing Prolonged Sedation and Episodic Hypoxia

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Background: As THC becomes legalized for medicinal and/or recreational use in many states, incidence of unintentional ingestions of THC-containing products by children is increasing. Effects of THC in children include lethargy, respiratory depression, tachycardia or bradycardia, and ataxia. Occasionally, altered mental status prompts extensive workup and interventions including CT scans, CSF analysis, EEG, intubation, anxiolytics, flumazenil, or naloxone. In the vast majority of cases, symptoms last less than 24 hours, though reports of effects lasting up to 48 hours are becoming more common. To our knowledge, symptoms lasting 60 hours are not reported in the literature.
Case Report: A 2-year-old healthy boy ingested several candies totaling 200 mg of THC and presented with sedation and intermittent hypoxic episodes that lasted for 60 hours. The patient had been well prior to the ingestion other than mild rhinorrhea and cough. When visiting a grandparent, he ate 4 chocolate edibles purchased from a local dispensary, each containing 50 mg of THC. Once discovered, he was brought to a local emergency department and became somnolent approximately 45 minutes later. He was transferred to our facility for further management. Urine drug immunoassay was positive for cannabinoids, and THC liquid chromatography-tandem mass spectrometry showed >500 ng/mL of 9-carboxy THC (ARUP labs). Amphetamine, barbiturates, benzodiazepines, cocaine, opiates and TCA screen (immunoassay) were negative, along with serum acetaminophen, salicylate, and alcohol concentrations. On the first night of admission, hospital day (HD) #0, he was somnolent and difficult to arouse, though was maintaining his airway. On the morning of HD #1, he had episodes of low oxygen saturation (as low as 40% on pulse oximetry) which were worsened by crying. Hypoxia prompted chest radiograph, which showed steepling of the subglottic airway, suggestive of croup. He received supplemental oxygen as needed and a dose of dexamethasone, which improved these symptoms. He was very sleepy, requiring tactile stimulation to wake. When awake, he had staring spells and appeared to be responding to internal stimuli, which at times were distressing to him. On HD #2, he was awake, but not alert or responsive to his surroundings. He had slurred speech and was unable to ambulate, though reported a voracious appetite. Later that afternoon, he was able to stand, but could not walk more than 2 steps due to both ataxia and lack of motivation. On HD #3, he returned to his talkative, active neurologic baseline without sequelae.
Case Discussion: In this ingestion of 200 mg of THC, intended for medicinal use, our patient remained symptomatic for 60 hours. We suspect that fearful hallucinations agitated the child, exacerbating symptoms of croup, leading to transient hypoxia. Fortunately, the child did not have a serious adverse event and returned to his baseline.
Conclusions: Though uncommon, large ingestions of THC can lead to prolonged clinical effects, lasting over two days. Children should be observed until symptoms resolve. If history suggests a large ingestion, it may be prudent to admit symptomatic children, as their intoxication may exceed the time frame of a typical ED visit.

KEYWORDS Cannabis, Pediatric, THC

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220. Pediatric clonidine overdose treated with 219 mg cumulative dose of naloxone

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Background: Clonidine is a central α2-adrenergic agonist that exhibits opioid-like effects in overdose. Mixed responses to naloxone have been reported. Although clonidine does not bind to opioid receptors, it may exert opioid-like effects through release of endogenous β-endorphin. High-dose naloxone may be required to elicit a therapeutic response. This case report involves a pediatric patient with clonidine toxicity treated with multiple, high-dose naloxone boluses and a high-dose naloxone infusion.
Case Report: A 23-month-old, 15-kg female was found with an open bottle of clonidine 0.1 mg and 8 tablets missing. She became lethargic and 911 was called. Prior to arrival, EMS contacted the Poison Center and management recommendations included 10 mg naloxone bolus administration. EMS found the child unresponsive with CPR in progress. The child responded to 1 mg of intranasal naloxone (INN) and became alert. Additional 3 doses of 1 mg INN were administered for recurrent episodes of unresponsiveness. Each of these doses resulted in an improved level of consciousness but with residual somnolence. Vital signs following INN included BP 135/85 mmHg, HR 57 bpm, RR 25/min, and SpO2 92% on oxygen via nasal cannula. The next naloxone 1 mg dose was administered intravenously (IVN), again with good response. The patient later became apneic and pulseless. CPR was restarted and 1 mg IVN administered. The child awoke but remained lethargic. Atropine 0.1 mg IV was given for bradycardia. Upon ED arrival, the child was alert but became less responsive. While preparation was underway for endotracheal intubation, IVN administration was continued in 2, 5, and 10 mg boluses for episodes of unresponsiveness with inability to protect her airway. Administration of these additional boluses was followed by improvement in mentation and airway protection avoiding the need for intubation. A naloxone infusion was started at 10 mg/hr, reaching a maximum rate of 15 mg/hr. This was continued in the pediatric ICU except for one 30-minute interval. For decreased level of consciousness during this interval, 10 mg IVN was administered, with resultant clinical improvement. The naloxone infusion was titrated down overnight and discontinued the following morning, approximately 24 hours after clonidine ingestion. She recovered to baseline and was discharged home the following day.
Case discussion: To our knowledge, this is the first case report of a pediatric clonidine overdose requiring a substantially large cumulative naloxone bolus dose (60 mg) plus a high-dose naloxone infusion for a total of 219 mg of naloxone to avoid intubation. Previous literature on high-dose naloxone use in pediatric patients with clonidine toxicity showed mental status improvement for the majority of patients with improvements in bradycardia and hypotension in some patients (Seger et al, 2018). Naloxone boluses up to 18.4 mg and naloxone infusion up to 30 mg/hr were administered in the previous study without adverse effects. Despite a high naloxone blood concentration (Table 1), our patient also experienced no adverse effects.
Conclusions: High-dose naloxone boluses and infusion should be considered in pediatric patients with severe clonidine toxicity as it may prevent the need for endotracheal intubation and associated morbidity.

Table 1.

Sample Type	Collection time post ingestion	Result	Reference Value (NMS Labs)
Clonidine whole blood	2.38 hours	20 ng/mL	0.5-2.0 ng/mL (immediate release, 2 hours after administration)
Clonidine serum	6.75 hours	12.96 ng/mL	1.00-2.00 ng/mL
Naloxone whole blood	9.65 hours	270 ng/mL	10 ± 1 ng/mL (average peak plasma concentration at 2 minutes after IV 0.4 mg dose)
Comprehensive urine drug test (IA; LC/MS-MS)	Positive: clonidine Negative: multiple opioids and/or metabolites No other coingestants identified		

KEYWORDS Clonidine, Naloxone, Pediatrics

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221. Urinary Cannabis Metabolite Concentrations in Cannabinoid Hyperemesis Syndrome

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Background: Cannabis Hyperemesis Syndrome (CHS) is characterized by recurrent episodes of intractable emesis and heavy use of cannabis. Recognition of CHS can be problematic due to the lack of specific biomarkers which can point the clinician to the diagnosis. We present a series of adolescent/young adult patients who presented to a pediatric gastroenterology (peds GI) service with acute on chronic nausea and vomiting, subsequently found to have CHS with associated elevated urinary cannabis metabolite concentrations.
Case Reports: We describe 15 patients referred to our peds GI division for intractable emesis with spot urinary cannabis metabolite carboxy-THC (THC-COOH) concentrations from January 1, 2018 through April 20, 2019. Urinary testing was performed using gas chromatography mass spectrometry (GC-MS) in a manner consistent with CLIA requirements at Mayo Clinic laboratory (Rochester, MN). The laboratory cutoffs were 3.0 ng/mL. The other parameters studied included demographics, symptom development and work-up. There were 7 females and 8 males, with an age range from 16 to 22 years old; the average age was 17.7 years. One patient had a preexisting history of Crohn's Disease, one reported a history of irritable bowel syndrome and one had a history of bulimia; no others had pre-existing gastrointestinal disease. All patients reported frequent marijuana use for at least 1 month; all reported inhalational use and 1 also ingested edibles. All exhibited intractable, non-bilious emesis for at least 2 weeks, and 13/15 reported nausea and abdominal pain. One patient did report intermittent hematemesis. 3 patients were hospitalized.
Case Discussion: GI workups were unremarkable. 9 out of 15 patients underwent endoscopy; 8 were normal and 1 showed mild esophagitis. 12 out of 15 exhibited significant weight loss over a period of 1 week to 6 months, with an average loss of 2.3 kg. 14 out of 15 patients had urinary THC concentrations >100ng/mL, with 7 individuals exhibiting levels >500ng/mL. The 1 patient with a urinary TCH-COOH level under 100ng mL had a urine level of 9ng /L but had not used THC for 2 weeks. Most other patients had used THC within 2 days of providing a sample for testing and their clinic visit. The Binomial test for CHS patients with urinary THC-COOH levels over 100ng/mL was significant with a p-value of <0.0005 (one tail test).
Conclusion: CHS is associated with an elevated urinary THC-COOH levels usually exceeding well over 100ng/ml, which is indicative of significant chronic cannabis exposure. In patients with a history consistent with CHS, urine testing may help guide the diagnostic evaluation of these patients and decrease the need for invasive testing.

KEYWORDS Cannabinoids, Cannabinoid Hyperemesis Syndrome, Pediatric Gastroenterology

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Pt #	Age	Sex	Urine THC COOH Level (ng/mL)	Weight Change (kg)	Marijuana Use With 2 Days of Testing	Reported Frequency of Use	Comments
1	18	F	>500	-1.8	Yes	Daily	
2	17	M	468	-4.5	Yes	Daily	
3	18	M	101	-7.3	Yes	Daily	Synthetic cannabinoid testing negative
4	18	M	>500	-0.9	Yes	"a few times per week"	
5	17	M	403	-1.8	Yes	Daily	
6	16	F	>500	-0.9	Yes	Daily	Delta 9 (blood) 2.4 ng/mL
7	18	M	>500	-4.5	Yes	Daily	Possible co-diagnosis of superior mesenteric artery syndrome
8	19	F	258	-1.8	Unknown	"Occasional"	Uses by vaporizating. Testing for synthetic cannabinoids negative.
9	18	F	218	-1.4	Yes	Daily	Delta 9 (blood): 3.7ng/mL. THC-COOH (blood): 11.6 ng/mL
10	22	M	>500	0	Yes	Daily	Uses by vaporizing, and by consuming edibles
11	16	M	417	6.8	Yes	2-3 times/week	
12	17	F	>500	-4.5	Yes	Daily	Esophagitis on EGD
13	18	M	357	-6.8	Unknown	2-3 times/day	Was using 2-3 times daily, but decreased shortly before urine testing. Unclear when last use was in relation to testing.
14	17	F	9	-4.5	No. Last use 10 days prior to blood testing, 3 weeks prior to urine testing	Unknown	Delta 9 negative. Carboxy THC (blood): 3.6 ng/mL two weeks after last reported use
15	16	F	>500	0	Yes	Daily	History of bulemia

222. Retrospective study of acetaminophen poisoning in children aged up to six years: No liver injury in single-dose poisonings

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Objectives: The aim of the study was to evaluate the use of gastrointestinal decontamination and N-acetylcysteine in small children with acetaminophen (APAP) poisoning aged zero to six years and to identify the patients at risk for liver injury in this group.
Methods: This retrospective cohort study was conducted at Department of Clinical Pharmacology, Copenhagen University Hospital, Denmark. The study was approved by the Danish Data Protection Agency. Children included had been admitted to one of the four paediatric hospital centres in the capital region of Denmark, from 2010-2017. The 4 centres cover approximately 30 % of the pediatric population in Denmark. Inclusion criteria were: i) age between 0 and 6 years, ii) and an electronic patient record including a laboratory analyse of plasma acetaminophen due to suspected APAP poisoning. The following data was collected for each patient: date of birth, gender, and weight, reason for exposure, APAP formulation, amount of APAP ingested (known/unknown), maximum APAP dose possibly ingested (known/unknown), maximum APAP dose ingested (mg), total duration of chronic paracetamol ingestion (days), time form paracetamol ingestion to hospitalization (hours).
Decontamination: vomiting after PCM overdose, charcoal, gastric aspiration, NAC IV treatment completed according to relevant Danish guideline, allergic reaction to NAC IV treatment, advise from the Danish Poisons Information Centre, total NAC IV treatment period. Laboratory results apart from plasma APAP included: plasma alanine aminotransaminase (ALT), plasma aspartate transaminase (AST), coagulation factors II, VII, X (INR), bilirubin, alkaline phosphatase.
Results: We identified 297 children with a mean age og 2.6 years, mean weight of 14.0 kilograms, 60% male. 281 was suspected single dose exposures and the mean time from suspected ingestion to hospitalization was 2.6 hours. In suspected single-dose APAP poisonings the formulation was mainly mixtures (27 %) and tablets (72%) and 97 % exposures were accidental intake during play. Estimated APAP dose (maximum) in this group was 200 milligram/kilogram. In the single-dose group 80% were treated with activated charcoal and 67 % were treated with NAC (98 % before plasma APAP result were available). In 223 of the children the first plasma APAP concentration was below lower limit for detection (100 or 10 micromol/L depending on the laboratory) and only 3 children had at plasma APAP above the 100-line (micrograms/mL) in the treatment nomogram. No patients had plasma ALT >1000 U/L.
Conclusion: The accidental single dose exposure to APAP among children during play gave in no instances rise to liver injury and NAC treatment was used in a large percentage of children even when no APAP was detected in the laboratory analysis.

KEYWORDS Acetaminophen, Pediatric, overdose

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223. Acute Renal Failure from Acute Guaifenesin and Dextromethorphan Abuse

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Background: Chronic guaifenesin abuse was first implicated in causing obstructive renal failure in the late 1990's. There are only two published case reports of obstructive renal failure caused by an acute ingestion of guaifenesin with dextromethorphan (DXM).
Case Report: A 17 year old presented to the ED c/o tremors and "stiff gait," and reported the acute ingestion of two #20 count boxes of Mucinex DM ® (guaifenesin 600mg, DXM 30mg) the night before to get high. HR 113/min, BP 170/98 mmHg, RR 23/min, SpO2 100% on room air, and was reported afebrile. The patient received 1 liter NS bolus and 1 mg lorazepam for the tremors.
Laboratory results: APAP 2 19 mEq/L, glucose 105 mg/dL, BUN 31 mg/dL, Cr 4.18 mg/dL, Ca 9.3 mg/dL, CPK 284 U/L, measured osmolality 299 mOsm/kg (gap 8.1), lactate 1.5 mg/dL, WBC 17K/uL. Urinalysis did not have hematuria, but was "consistent with possible infection" and the patient was started on ceftriaxone for possible urosepsis. Abdominal/pelvic CT scan showed bilateral hydronephrosis with a collection of small stones or sludge within the bilateral ureter-vesicular junctions. Cystoscopy performed on hospital day 2 removed the ureteral sludge and placed bilateral ureteral stents. Post-cystoscopy, labs and vital signs improved with CO2 20 mEq/L, Cr 2.6 mg/dL, temp 36.9oC, HR 96/min, BP 160/89 mmHg, RR 16/min, SpO2 100% RA. Pt was treated with belladonna-opium suppositories and oxybutynin for lower abdominal pain that increased with voiding. On hospital day 3, the patient's creatinine was 1.47 mg/dL and the patient was clinically much improved. The last labs, drawn on hospital day 4, included a CO2 30 mEq/L, and Cr 1.33 mg/dL. Patient was discharged home with resolution of all symptoms. Analysis (LC/MS/MS) of urine collected during the cystoscopy found [d-methorphan] + [l methorphan] ≥ 500 ng/mL. Composition of the kidney stones was not tested.
Discussion: In the two published case reports of ARF caused by acute guaifenesin ingestion (questionably the same patient), both patients acutely ingested 10 unknown type of Mucinex DM ® tablets for a total of either 6 or 12 grams of guaifenesin. Both patients had bilateral flank pain, gross hematuria, and acute renal failure with bilateral ureteral obstruction. A guaifenesin metabolite was identified as the main component of both patient's stones. In the initial report of guaifenesin renal stones in 1999, the patients had been chronically taking 10-20 grams of guaifenesin per day. Our patient's acute ingestion of 24 grams of guaifenesin (and 1,200 mg DXM) is much higher than the amounts of guaifenesin ingested in the case reports / series. The patient's initial presentation was actually that of serotonin toxicity, which responded to IV benzodiazepines. The patient had no symptoms consistent with renal calculi and the elevated creatinine was an unexpected finding.
Conclusion: We report the third case of acute renal failure caused by bilateral ureteral obstruction from the acute ingestion of guaifenesin and dextromethorphan.

KEYWORDS Guaifenesin renal stones, Acute renal failure, Guaifenesin abuse

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224. Characteristics of Exposures from Critical Access Hospitals—One Regional Poison Control Center's Experience.

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Background: Poison control centers (PCC) are receiving a higher percentage of their calls from hospitals. In several rural states, calls from critical access hospitals (CAH) are an important source of calls yet there is a paucity of literature on how calls from these health care facilities differ from larger, more urban hospitals (UH). The aim of this study was to compare call characteristics from critical access hospitals and urban hospitals received by a regional poison control center.
Methods: This was a retrospective chart review of the database of a regional PCC. Records from 2012, 2013, 2016 and 2017 were reviewed for all exposures reported from either a CAH or a UH. CAH were defined using the criteria from the Centers for Medicare & Medicaid Services. In the region covered by this PCC there are 84 CAH. The four largest hospitals by patient volume in the region were selected as the UH comparison. Case information extracted included age, sex, substance(s) involved in exposure, reason for exposure, medical outcome, disposition, whether a medical toxicologist was consulted, whether the patient was transferred, use of activated charcoal, naloxone, and

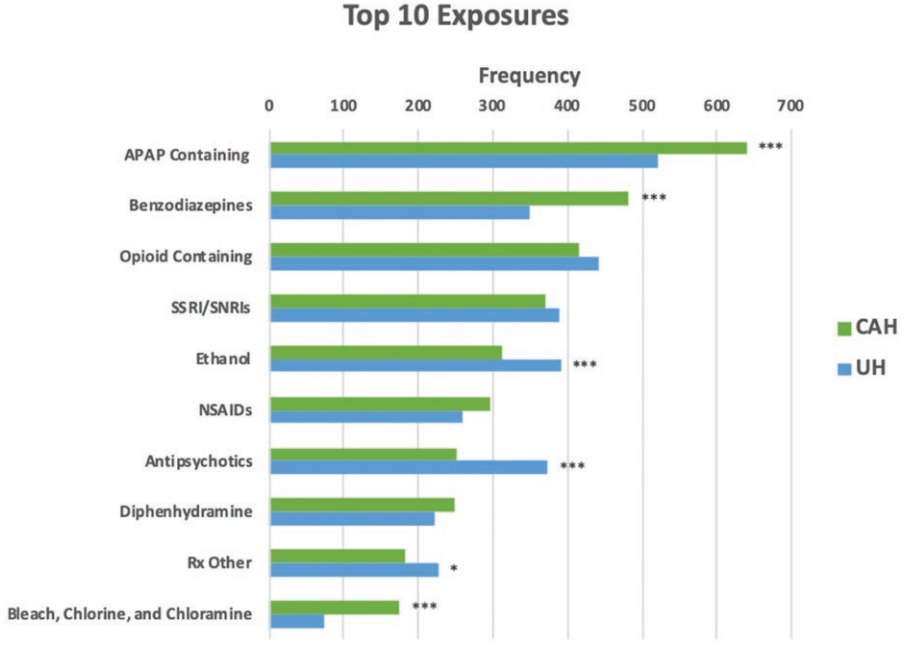

Figure 1 Top 10 Most Frequently Encountered Exposures.

*: p-value <0.05, **: p-value <0.01, ***: p value <0.001.

Table 1 Use of Healthcare resources.

	CAH #	CAH %	UH #	UH %	p value
Referred to Hospital by PCC	655	15.2%	702	16.3%	0.0065
Transferred	581	13.4%	50	3.2%	< 0.0001
Admitted to ICU	640	14.8%	489	28.2%	0.0003
Toxicologist Consult	662	15.3%	302	22.9%	< 0.0001

Table 2 Medical Outcomes.

	CAH #	CAH %	UH #	UH %	p value
Less Than Moderate Effect	2890	67%	2269	56%	< 0.0001
Moderate and Major Effect	1187	27%	1513	38%	< 0.0001

flumazenil. Data imported into Microsoft Excel (Redmond, WA) and analyzed in SPSS (IBM Corp., Armonk, NY).

Results: A total of 6588 exposures from CAH were identified along with 6510 exposures from UH over the study period. Calls from both CAH and UH increased from 2012 to 2017. Between the two health care sites, significant differences in the types and numbers of exposures were found. Figure 1 compares the top 10 most common exposures at CAH with comparison to UH. CAH exposures were more likely to be unintentional, 49% compared to 32% for UH exposures ($p < 0.0001$). Compared to UH, patients at CAH who were exposed to benzodiazepines were statistically more likely to receive flumazenil (9.8% to 4.3%, $p = 0.0047$). However, the use of activated charcoal and naloxone was similar between the two. Table 1 demonstrates that patients at CAH were less likely to have been referred by the PCC, need admission to an ICU, or require a toxicologist consult, but were more likely to be transferred to another healthcare facility. Patients at UH were more likely to have a duration of medical effects greater than 24 hours. Table 2 demonstrates that CAH were more likely to have moderate or major medical effects documented. There were no statistically significant differences in mortality between both groups.

Conclusions: In this study, critical access hospital utilization of a regional poison control center increased and exposures were significantly different in many ways compared to urban hospitals. Future research is warranted to optimize regional poison control center's services for critical access hospitals.

KEYWORDS Critical Access Hospitals, Poison Control Center, Public Health

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225. An Unusual Case of Severe Pediatric Acetaminophen Overdose Treated with Increased Dose N-acetylcysteine

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Background: Acetaminophen overdose is a common cause of liver failure in the United States accounting for 71,193 total cases with 21,776 exposures to acetaminophen in combination with other drugs. Dosing for all patients with acetaminophen overdose at our regional poison center is N-acetylcysteine (NAC) 150mg/kg IV bolus followed by 12.5mg/kg/hr IV until recovery, transplant, or death. We present an unusual case of severe pediatric acetaminophen overdose treated with increased dose NAC.

Case Report: A 2-year-old unimmunized female born at 33 weeks gestation presented with lethargy and vomiting for 2 days to an urgent care center where she had a blood glucose 47 mg/dL. She was given a bolus of D10 and transferred to a tertiary care children's hospital emergency department. On arrival, she developed seizures that were poorly controlled with lorazepam, fosphenytoin, and levetiracetam. She was intubated for airway protection with ketamine and succinylcholine and started on broad-spectrum antibiotics. Initial laboratory work up was remarkable for a pH 7.27, HCO3 14.2mEq/L, AST >4000iU/L, ALT 8852iU/L, ammonia 192umol/L, Hgb 9.6g/dL, WBC 16.3 10e3/uL, PTT 51.3s, INR 11.36, and acetaminophen 101mcg/mL, ASA 1.1mg/dL. Other laboratory values included Cr 0.76mg/dL, undetectable ethanol, undetectable lead level. Her comprehensive urine drug screen collected after intubation detected acetaminophen, lidocaine, monoethylglycylxylidide, ketamine, norketamine, and diphenhydramine. The medical toxicology team was consulted and she was started on NAC 150mg/kg IV bolus followed by 12.5mg/kg/hr IV continuously. Initial ECG showed sinus tachycardia with normal QRS. She was placed on the liver transplant list but removed as her liver function recovered. NAC was stopped on hospital day 3 and liver function continued to improve. She was started on rifaximin, lactulose, sodium phenylacetate, and sodium benzoate with complete resolution of liver failure. Throughout her hospitalization she had episodes of hypoglycemia, treated with D25 boluses, and resolved with her improved liver function. She was discharged on hospital day 36 with deficits in oral skills but otherwise functional. Liver function tests were AST 84iU/L, ALT 128iU/L, ammonia 26umol/L, INR 1.05, and PTT 31.3s at discharge. Genetics evaluation was negative. She was discharged to foster care with PT/OT and continued PO/NG gavage feeds.

Case Discussion: This child had an unusual presentation of acetaminophen overdose. Her seizure is suggestive of a combination of acetaminophen and diphenhydramine, as found on her comprehensive urine drug screen. Acetaminophen overdoses in combination with other drugs have been shown to be more likely to have acute liver failure, although there have been no differences in clinical outcomes. Although NAC has been shown to be safe in children in acetaminophen induced acute liver failure, there has not been a consensus on the appropriate dosing of NAC in these clinical scenarios.

Conclusion: This case highlights that severe acetaminophen-induced acute liver failure in children may benefit from the use of our poison center's increased dose NAC protocol, as demonstrated in this case of critically ill child who had a near complete recovery.

KEYWORDS Acetaminophen, NAC, pediatric

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226. Help for a broken heart: Left ventricular venting and ECMO for severe amphetamine induced Takotsubo Cardiomyopathy

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Background: Takotsubo cardiomyopathy can occur after amphetamine overdose and require significant support including left ventricular venting and extracorporeal membrane oxygenation (ECMO). This is a report of a healthy 26-year-old woman who was evading police and ingested lisdexamfetamine, mixed amphetamine salts, and buprenorphine, none of which were her medications which resulted in severe Takotsubo cardiomyopathy. This is the first case report in the

literature of an amphetamine ingestion which required left ventricular venting and ECMO.
Case Report: A 26-year-old healthy woman on no known medications ingested lisdexamfetamine, mixed amphetamine salts, and buprenorphine and presented to the Emergency Department with the following vital signs: BP 134/60, HR 60s bpm and oxygen saturation 95% on room air. The poison center was contacted two hours after ingestion. Within seven hours the patient had flash pulmonary edema requiring emergent intubation and was transferred to a tertiary level of care. Her echocardiogram showed an ejection fraction (EF) of 10% with global dysfunction concerning for Takotsubo Cardiomyopathy. The initial troponin was 6.37. She received infusions of norepinephrine, vasopressin, and midazolam and had further decompensation on hospital day 2 requiring insertion of a percutaneous ventricular assist device (Impella pump) for left ventricular venting and extracorporeal membrane oxygenation (ECMO). On hospital day 3, follow up revealed patient was completely neurologically intact but her heart was in a severely weakened state (EF was 35-40%). Patient remained with the device for left ventricular venting and ECMO for two days and her conditioned stabilized such that both therapies were discontinued. On hospital day 4, the ECG showed no interval prolongation; EF of 40% and the patient remained intubated but had purposeful movement and followed commands. Her troponins normalized. The following day patient was extubated and on 4L nasal cannula of oxygen. Her echo on hospital day 10 showed an EF of 55%. On hospital day 13, the patient no longer required oxygen and was discharged.
Case Discussion: Takotsubo's cardiomyopathy can result in severely depressed ejection fractions and subsequently life-threatening pulmonary edema and cardiogenic shock. It appears to be induced from severe emotional or physical stress and may be related to increased sympathetic activity. Stimulant drugs as in this case have also been associated with the development of Takotsubo's cardiomyopathy. A multi-pronged approach of life support including mechanical ventilation, venoarterial ECMO, and a percutaneous ventricular assist device for left ventricular venting resulted in survival. Left ventricular venting was necessary to prevent ventricular blood clotting and maintain blood flow with the patient's severely depressed EF.
Conclusion: For patients with severe Takotsubo cardiomyopathy, the use of ECMO can be life- saving and should be considered early in the course of therapy in severely ill patients. In patients with severe left ventricular dysfunction one should entertain a venting strategy either via central cannulation or a percutaneous VAD. This healthy, young woman benefited from these therapies where her outcome was survival with normal neurological status, an EF of 55%, and no persistent organ injury.

KEYWORDS Takotsubo Cardiomyopathy, Left Ventricular Assist Device, ECMO

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227. Getting over the blues from the rush on poppers

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Background: Amyl nitrite was first used in the 1800s for angina and subsequently as an antidote for cyanide poisoning. Now a range of volatile inhalants are used, chemically classed as alkyl nitrites, including amyl, butyl, isobutyl, isopropyl nitrite and colloquially referred to as 'poppers' or 'amyl', 'rush' or 'jungle juice'. Diversification in chemical entities has been prompted by drug legislation and classification as a carcinogen. They have become commonly used recreational drugs, particularly among LGBTIQ+ communities to facilitate receptive anal intercourse (26-46% of gay men). It is also used more generally as a 'party drug', but not as a drug of choice. Risks of methemoglobinemia are well known, but the toxicity profile from misuse is relatively poorly described and emerging hazards of retinal toxicity have only recently been described.
Methods: To evaluate rates of use and harms from alkyl nitrite inhalants, investigate risk factors for adverse events and describe the toxicity profile. Harms will be evaluated from the combined Australian Poisons Information Centres (PIC) alkyl nitrite case consultations 2009-18; the New South Wales Public Health Rapid, Emergency, Disease and Syndromic Surveillance system 2011-18; and the National Coronial Information System 2000-18. Patterns of use will be evaluated through Australian drug use surveys: Ecstasy and Related Drug Reporting System 2009-18 and Big Day Out Study.
Results: Usage appears to have remained relatively constant but significant increases in adverse events were noted with cases to PICs increasing from 28 to 78 over the past decade. Two deaths from exposure to isobutyl nitrite have been described in Australia, occurring in 2007 and 2008 (National Coronial Information System). Almost all cases were in adults, with an increasing trend in females, who now represent one-third. The majority of cases related to accidental ingestion of liquid followed by spilling of liquid into nostrils. Three-quarters were hospitalised but were rapidly discharged. South-Eastern Australia had the highest population-adjusted rates of calls to PICs. The key toxic effect seen was methemoglobinemia, which occurs at low levels when inhaled but life-threatening or lethal effects when ingested, and rarely with excessive inhalation or inadvertent intranasal administration. Severe mucosal irritation was also reported. Toxic effects were rapid in onset and short in duration, subsiding in a matter of hours. Severe effects were in 3%, 15% moderate, 56% minor and 22% none based on Poisoning Severity Score up to the time of the call (4% unknown). Alcohol was commonly described in the context of usage for many cases and appears to be a risk factor for accidental ingestion.
Conclusions: Recent increases in calls to PICs relating to toxicity from alkyl nitrites is concerning, particularly in the context of relatively stable use patterns from illicit drug users. We hypothesise products available more recently are more: pure, toxic nitrites, poorly packaged/labelled; or the users (eg women, 'legal high') are inexperienced and at higher risk of toxicity due to incorrect use. A vigorous debate has been stimulated by this data, led by the Australian Government's Therapeutic Goods Administration considering education, rescheduling, changes in packaging and labelling.

KEYWORDS Amyl, Nitrites, Misuse

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228. Evolution of poison center fatalities

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Background: Our regional poison center (RPC) is managing increasing numbers of patients that are severely ill from potentially non-toxicological etiologies (i.e. sepsis, CNS bleeds, etc.). This has coincided with an increase in healthcare facility (HCF) exposure consults to our RPC over time. The aim of this study is to quantify trends in our RPC exposures, compare these to our yearly fatalities, and further define the number and trajectory of toxicology-related fatalities we manage by contextualizing the trends using each fatality's relative contribution to fatality (RCF) code.
Methods: Yearly exposures (total and originating from HCF only) to our RPC from 2010 through 2018 were computed. The National Poison Data System (NPDS) website (npds.us) was queried for fatalities from our RPC from 2010 through 2018. Number of fatalities for each year was recorded and the RCF codes (per AAPCC standard definitions) were divided into two groups. Group 1 (undoubtedly responsible, probably responsible, and contributory) represent toxicological etiologies

Year	2010	2011	2012	2013	2014	2015	2016	2017	2018
Total Exposures (-9%)	79836	76595	73998	72565	72382	73750	73050	71927	72380
HCF Exposures (+22%)	19096	19659	20333	20606	21310	23183	23938	24831	24337
# Fatalities (+47%)	51	69	49	72	65	71	72	69	97
Group 1 (-11%)	43 (84%)	50 (72%)	36 (73%)	47 (65%)	45 (69%)	43 (61%)	47 (65%)	43 (62%)	73 (75%)
Group 2 (+36%)	8 (16%)	19 (28%)	13 (27%)	25 (35%)	20 (31%)	28 (39%)	25 (35%)	26 (38%)	24 (25%)

associated with the fatality, and Group 2 (probably not responsible, clearly not responsible, and unknown) are considered fatalities that are not toxicologically related. Descriptive statistics were used to compare the two groups.

Results: Total exposures decreased by 9% during the study period. However, there was a 22% increase in HCF exposures over the same time period. A quarter of our reported exposures originated from a HCF in 2010 while one third originated from a HCF in 2018. Total fatalities increased from 51 in 2010 to 97 in 2018 (47% increase). In 2010, 84% of fatalities were Group 1; by 2018 only 75% of fatalities were considered Group 1. Although the proportion of toxicologically-related fatalities has decreased over the study period, the total number of toxicologically-related fatalities increased. See table.

Conclusions: Our RPC has experienced greater numbers of exposures from HCFs in the context of lower overall number of exposures. These patients may be ill from non-toxicological etiologies. Our yearly fatality numbers have surged and these data indicate increasingly more fatalities being managed by our RPC that are not poison related. A multicenter study examining this phenomenon is warranted.

KEYWORDS Poison Center, Fatality, Epidemiology

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229. Do you have the knack for dosing e-NAC?

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Background: Acetaminophen continues to be a common exposure and significant cause of fatalities reported to American Poison Centers. N-acetylcysteine (NAC) has been available as inhalation and intravenous (IV) solutions. In 2016, the Food Drug Administration approved an oral effervescent, lemon mint flavored tablet, to improve palatability. The effervescent 500 mg and 2.5 g tablets are dissolved in water. The package insert provides a table to assist in weight-based dosing and specifies the quantity and strength of tablets to achieve the appropriate dose. Practitioners may use traditional weight-based dosing of NAC instead, leading to improper dosing. There are no published studies demonstrating health care provider ability to dose effervescent NAC (e-NAC) in a timed educational test environment. The objective of this study is to assess healthcare providers ability to dose e-NAC based on the package insert.

Methods: This study was conducted as an educational test distributed electronically to University of Illinois Health's physicians, nurses and pharmacists asking them to anonymously complete three timed scenarios (S) for e-NAC dosing. The primary outcome is the accuracy of e-NAC dosing using the package insert. Secondary outcomes include time to completion of first scenario and e-NAC dosing accuracy using tertiary sources. Both arms received the same three scenarios in random order with the recommended dosing from either the package insert (PI) or a tertiary drug (TS) reference.

Results: Forty-three surveys had at least one scenario completed. There was a statistically significant difference in accuracy of selecting the correct number tablets for loading dose (LD) across all scenarios, the package insert arm being more accurate (2.5g tablets; p = 0.0109 [S1], p = 0.0035 [S3], 500mg tablets; p = 0.0008 [S1]. p = 0.0035 [S3]). There was no statistical significance between the calculation of loading and maintenance doses (MD) across all scenarios. The tertiary arm had the poorest performance on scenario three with 75% accuracy on LD and 70% on MD calculations. There was no statistical significance between pharmacists and non-pharmacist calculations in the package insert group (p = 0.1830, p = 0.5588 [S1 LD,MD], p = 0.4737 [S2 LD,MD],

Accuracy of Package Insert vs Tertiary Source: Maintenance Dose.

	Scenario 1			Scenario 2			Scenario 3		
	PI (n = 18)	TS (n = 22)	P value	PI (n = 19)	TS (n = 22)	P value	PI (n = 18)	TS (n = 20)	P value
Maintenance Dose	83.3	86.3	1	94.7	90.9	1	88.9	85	1
Number of 2.5 g tabs	100	68.2	0.0109	—	—	—	94.4	55	0.0089
Number of 500 mg tabs	94.4	36.4	0.0002	—	—	—	94.4	40	0.0005
Reconstitution mL amount	94.4	72.7	0.1048	84.2	63.6	0.1734	100	90	0.4879

PI = package insert; TS = tertiary source.

Accuracy of Package Insert vs Tertiary Source: Loading Dose.

	Scenario 1 (%)			Scenario 2 (%)			Scenario 3 (%)		
	PI (n = 18)	TS (n = 22)	P value	PI (n = 19)	TS (n = 22)	P value	PI (n = 18)	TS (n = 20)	P value
Loading Dose	88.8	86.3	1	94.7	90.9	1	72.2	85	0.4381
Number of 2.5 g tabs	100	68.2	0.0109	—	—	—	100	60	0.0035
Number of 500 mg tabs	100	54.5	0.0008	—	—	—	100	60	0.0035
Reconstitution mL amount	100	77.3	0.0530	84.2	63.6	0.1734	100	90	0.4879

PI = package insert; TS = tertiary source.

Average time to completion of first scenario.

	Average time in seconds		
	Package Insert (min:sec)	Tertiary source (min:sec)	P value
Scenario 1	340.8 (5:41)	391.4 (6:22)	.829
Scenario 2	211.8 (3:32)	267.2 (4:16)	.47
Scenario 3	310.9 (5:11)	505.4 (8:25)	.44

p = 0.6078,p = 0.1830 [S3 LD,MD]). There was statistical significance between pharmacist and non-pharmacist dose selection in the tertiary arm, pharmacists being more accurate (p = 0.0023, p = 0.0177 [S1 MD;2.5g,500mg], p = 0.194 [S3 LD;2.5g,500mg]. Time to completion of first scenario was not statistically significant between the two groups (p = 0.829 [S1], p = 0.47 [S2], p = 0.44 [S3]). The package insert consistently yielded higher doses, on average 1 g, which results in excess of NaHCO3 compared to using the historical weight based dosing strategy. Of the healthcare providers, nurses most frequently opened the survey but did not complete it. Fifty-seven percent completed the package insert and 50% completed the tertiary arm (4/7 [PI], 5/10 [TS]).
Conclusions: The package insert dosing chart for e-NAC led to a statistically significant difference in tablet selection among all scenarios, a non-significant difference between dose calculation, and non-significant reduction in calculation time. Nurses most frequently didn't complete the survey, anecdotally due to intimidation of calculations. Both groups took over five minutes to complete the first scenario, which may not be practical at the patient bedside.

KEYWORDS e-NAC, acetaminophen, dosing

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230. Pharmacokinetics is toxicokinetics in a massive buproprion ingestion

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Background: Kinetics in massive overdose are thought to approach zero-order, reflecting the saturation of binding sites. Here we report the largest nonlethal bupropion overdose (40.5 g) and note that the kinetics of bupropion were first-order.
Case Report: A 22-year-old female presented to an outside emergency department hemodynamically stable and neurologically intact approximately 1 hour after ingesting 40.5 g of bupropion hydrochloride by mouth. The patient had 4 generalized tonic-clonic seizures, received 12 mg of lorazepam, was intubated and transferred to our hospital. Initial labs demonstrated a bupropion level of 4000 ng/mL (therapeutic range 50-100 ng/mL) and a hydroxybupropion level of 5300 ng/mL (600-2000 ng/mL). Bupropion and hydroxybupropion serum levels were obtained every 6 hours starting ~20 hours post-ingestion. Fifteen hours after ingestion the patient received 20% lipid emulsion bolus (1.5 mL/kg). Ultimately, patient's neurologic status improved such that she was extubated, verbally interactive, and fully oriented by hospital day 7.
Case discussion: The patient's serum bupropion concentrations followed an exponential curve with rate constant 0.0337 (ng/mL/hr), corresponding to a half life of 20.59 hours. The reported half-life of bupropion is 33-37 hours (coefficient of determination 0.97). The active metabolite, hydroxybupropion followed zero-order kinetics with a half-life of 118.5 hours (coefficient of determination 0.709).
Conclusions: Here we describe the largest reported non-lethal bupropion overdose. This is also the first report of the toxicokinetics of bupropion and hydroxybuproprion, its active metabolite, in the context of intralipid administration. The toxicokinetics of bupropion were first order, as its pharmacokinetics are reported to be.

KEYWORDS Pharmacokinetics, toxicokinetics, bupropion

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231. Pediatric Ibuprofen Ingestion: Do we need to be concerned?

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Background: Upon reviewing Regional Poison Control Center (RPCC) data, it was revealed that ibuprofen is involved in 4% of all poisoning exposures and 7.2% of all pharmaceutical poisoning exposures in the pediatric population. Ibuprofen overdose is often felt to be of little clinical significance from a toxicology perspective though in more extreme cases serious toxicity can occur. There is a dearth of recent studies in the literature regarding the effects and outcomes of pediatric patients with these ingestions.
Objectives: To evaluate the epidemiology of pediatric ibuprofen exposures reported to the RPCC by age, intent, amount ingested, clinical effects, and outcome. Our study examines current trends of ibuprofen ingestions in our region as reported to the RPCC.
Methods: A retrospective review of greater than 4000 ingestions over a six-year period involving ibuprofen exposures reported to Regional Poison Control Center was performed. Inclusion criteria were: patients with ibuprofen ingestions, 0-18 years of age, reported to our RPCC from 2012-2017. Cases excluded were those with unknown outcomes or coingestants. Outcome measures were no effect, mild effect, moderate effect, and major effect as defined by The American Association of Poison Control Centers.
Results: In total, 4420 cases were reviewed. Among those cases, outcomes were 2549 no effect, 1 major effect, 68 moderate effect, and 130 minor effect. There were 1415 cases which were lost to follow up. Of the cases lost to follow up, 91% were felt to be of minimal or no toxicity. Of cases with symptoms, the most frequently reported intent was suspected suicide attempts. GI symptoms (129) were the most common symptoms seen, followed by cardiovascular (33) and neurological (29). The single case which resulted in a major effect experienced metabolic acidosis, after intentionally ingesting 20 grams (370 mg/kg) of ibuprofen. Of the categories with clinical effects, approximately 97% of children under 6 years old had no effect whereas only 64% of children in the 13-19 age range had no effect.
Conclusion: Ibuprofen remains a relatively safe over the counter analgesic for use in the pediatric population. In children less than 6 years of age, exposures rarely result in poor outcomes and often have no clinical symptoms. Intentional ingestions are likely to be higher in amount taken as well as have more significant effect.

KEYWORDS Ibuprofen, Ingestion, Effect

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232. Acute on Chronic Lacosamide Monotherapy Overdose: A Case Report

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Introduction: Lacosamide is an anticonvulsant originally approved for the treatment of partial seizures. Its use has expanded to adjuvant therapy for those with intractable seizures already on other antiepileptics and, more recently, as monotherapy for difficult to control epilepsy. It causes augmentation of the slow sodium channels and has been found to have significant neurologic and cardiac symptoms in overdose. All published cases, however, had co-ingestants with other medications, confounding the picture of clinical toxicity. This case report presents a woman with symptomatic acute on chronic overdose of lacosamide monotherapy.
Case Report: A 45-year-old woman with a history of seizures presented to the Emergency Department (ED) after an unintentional overdose of lacosamide. She was prescribed 300 mg twice daily, however, due to a medication dosing error, was taking 450 mg twice a day for approximately 2 weeks. On the day of presentation, she had also taken the already incorrect morning dose a second time (total 900 mg). On presentation, she noted vertigo, diplopia, nausea, and vomiting. Exam showed a woman who was diaphoretic and had spontaneous horizontal nystagmus that was bidirectional on exam. Electrocardiogram showed a prolonged PR interval of 224 ms and a slightly wider QRS, increasing to 108 from 98 ms. The patient was given a liter of fluids as well as 0.5 mg of lorazepam IV for symptom control. She continued to be significantly symptomatic and was admitted for observation. A lacosamide level was elevated at 26.5 mcg/ml (normal 1.0-10.0 mcg/ml). On evaluation the following day, the patient appeared significantly improved. She no longer had any vertigo or nausea and her nystagmus completely resolved. Repeat EKG showed resolution of the PR prolongation and QRS interval. The patient was counseled on correct dosing of her lacosamide and advised to follow up with her neurologist.
Discussion: There have been only nine previously published cases of lacosamide toxicity, but all of these had co-ingestants that likely contributed to the toxic effects seen in patients. Almost all of these cases showed significant cardiac effects, ranging from AV nodal blockade to atrial fibrillation to cardiac arrest. Neurologic effects have been reported and include status epilepticus. This case report elucidates some of the toxic effects specific to lacosamide, since the patient was exclusively on lacosamide at the time of overdose. This patient did show some cardiac effects with PR prolongation and slight widening of the QRS on EKG. She did not, however, develop any cardiac symptoms and her EKG self-resolved after 24 hours. Her more prominent symptoms were neurologic in origin, with severe vertigo and spontaneous nystagmus that was bidirectional on exam. Fluids and lorezapam were given for symptom control and did seem to be effective in this case.
Conclusions: Lacosamide has been recently licensed as an adjunctive treatment for seizures. Cardiac side effects may occur following accidental and suicidal overdose. However, they are mostly limited to ST-segment and T-wave abnormalities and slight lengthening in PR interval and QRS duration. Neurologic side effects following overdose include headache, dizziness, diplopia, and confusion.

KEYWORDS Lacosamide, Monotherapy, Overdose

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233. The Child Lead Astray

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Background: Lead toxicity is a well-recognized health hazard to children, particularly during their developmental years. Lead-based paint and contaminated dust in older buildings are the most common sources of lead poisoning in children; however, other sources include exposure to contaminated water, soil, and foreign body ingestion. We report a pediatric ingestion of the contents of a shotgun shell which resulted in elevated blood lead levels.
Case Report: Our regional poison center was contacted about a 5-year-old boy who reportedly ingested the contents of a shotgun shell. The child was referred to an emergency department, where an abdominal radiograph highlighted 50-60 pellets that were all past the stomach. The contents of the shell were verified by the parents to contain lead. Medical toxicology recommended admission and initiation of whole bowel irrigation with serial imaging. A blood lead level (BLL) sent for analysis roughly 15 hours post-ingestion was 30.5 mcg/dL. Chelation was not started, as the child remained asymptomatic during this time and BLL remained
Discussion: While the shells themselves are typically made of plastic tubing with a brass or metal base, the contents of shotgun shells may contain beanbag rounds, rubber, or lead pellets. Lead poisoning from shotgun shells are uncommon but have been documented in the past. Ingestion of lead contents does not immediately pose a great risk or concern as lead is poorly absorbed through the GI tract. Typically, single foreign body lead ingestions are managed conservatively, with serial imaging to track the progression and expulsion of the object. Levels are obtained if there is prolonged exposure in the GI tract. However, the contents of a shell casing held a large number of lead particles confirmed by imaging which led to a more aggressive management approach in this case. Primary concerns of lead toxicity in the pediatric population are neurodevelopmental, although initial presentation may include GI effects. When progression to severe toxicity occurs in children, anemia is noted, followed at higher levels with signs of encephalopathy. Fortunately, our 5-year-old patient remained asymptomatic throughout the course of management.
Conclusion: Though lead ingestions in children are not a new phenomenon, ingestions of shotgun shells are a unique source of lead not typically encountered. The large ingestion of lead seemed to pose substantial risk, but management to minimize absorption prevented the development of severe toxicity.

KEYWORDS Lead, pediatric, shotgun

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234. Pediatric Guanfacine Toxicity with Severely Elevated Plasma Level

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Background: Central alpha-2 agonists such as clonidine and guanfacine are commonly used in the management of pediatric attention-deficit-hyperactivity disorder (ADHD). These medications are known to produce an opioid-like toxidrome with bradycardia in overdose. We present a case of significant guanfacine toxicity in a child with a confirmatory elevated plasma guanfacine concentration.
Case Report: A six-year-old, 20kg, female with medical history of ADHD, autism spectrum disorder, and oppositional defiant disorder (ODD) presented to our emergency department with 48 hours of lethargy and somnolence. The mother reported that 48 hours prior to presentation, the child was noted to vomit and then become persistently somnolent, but arousable. The child had little oral intake during this time, preferring to sleep. The mother reported this to be entirely unlike the child's usual hyperactive state. Her medications included clonidine 0.1mg daily, and guanfacine 2mg each morning and 1mg each evening. The mother denied any other medications in the home other than children's multivitamins and denied any likelihood of the child unintentionally ingesting extra clonidine or guanfacine. She reported that the child had not received any medications in

48 hours due to running out of all medications. On exam, the child was bradycardic to the 40s. Other vitals were normal. The physical exam was notable for a somnolent young female with 2mm equally reactive pupils that aroused with physical stimuli but promptly returned to sleep when not being stimulated. Naloxone 3mg was given intravenously without improvement. A routine serum toxicologic screen was unremarkable. Extended testing for common synthetic opioids was also unremarkable. Electrocardiogram showed sinus bradycardia at 50 bpm with normal intervals. The patient was admitted for continued monitoring. She was discharged 48 hours later upon return to baseline mental status and vital signs. Admission blood testing revealed a plasma guanfacine concentration of 40ng/mL, which is several fold higher than reported plasma peaks with therapeutic dosing. Clonidine was not detected.
Case Discussion: Central alpha-2 agonist poisoning is a common presentation among pediatric poisonings, typically due to exploratory, unintentional ingestions. Guanfacine and clonidine levels are not usually obtained to confirm the diagnosis but may be useful in the setting of unobserved exposure or unclear presentation. A plasma guanfacine level was helpful in this case to confirm the diagnosis.
Conclusion: Central alpha-2 agonist poisoning can present remarkably like opioid toxicity with concomitant bradycardia. Plasma testing of guanfacine and/or clonidine levels may provide clinical utility to confirm the diagnosis in cases of unclear toxicity.

KEYWORDS Guanfacine, central alpha-2 agonist, clonidine

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235. Complex febrile seizures following pediatric exploratory carbamazepine ingestion

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Background: Febrile seizures are common in pediatric populations with an incidence of 2 to 5 %. Here we report a child who developed complex febrile seizures following accidental carbamazepine ingestion.
Case presentation: A 20-month-old child was brought to the Emergency Department (ED) with acute onset of altered mental status along with focal seizures in the form of flexion and extension of her upper limbs. This was followed by generalized tonic-clonic seizures for a few minutes which subsided after lorazepam was given. On exam, she was febrile with a temperature of 104.5 F, BP - 111/76 mm Hg, HR - 160/min, RR - 25/min and oxygen saturation of 100 % on room air. Neurological exam revealed the child to be sedated following the administration of benzodiazepines; pupils were 2 mm bilaterally and sluggishly reactive to light, midline gaze, symmetric face, flaccid tone all over, absence of neck rigidity and not responding to verbal or noxious stimuli. Deep tendon reflexes were 3+ bilaterally in arms and legs with no clonus and plantar reflex was flexor response bilaterally. After admission into the ICU, the child continued to have recurrent episodes of focal seizures involving the upper limbs along with recorded fever and altered mental status. A presumptive diagnosis of complex febrile seizure was made, considering the focality of the seizures along with recurrent episodes within 24 hours. She was worked up for serious bacterial infection in the blood and cerebrospinal fluid (CSF) along with the starting of empirical antibiotics. Blood, CSF and urine cultures were negative. PCR studies from nasopharyngeal swab were positive for rhinovirus. Imaging of the head and chest were normal. Rest of the labs were significant for hypokalemia (2.6 meq/L), metabolic acidosis (ph - 7.25, HCO3 - 17, CO2 - 33) with high lactate levels (6 mmol/L). During the course of her stay in the hospital, her mother revealed that they have carbamazepine tablets stored at home, belonging to her sister. She reported that a few pills were missing from the bottle. A level of carbamazepine was then ordered which was reported to be 29 mcg/ml. The child was intubated and started on multidose activated charcoal (MDAC). Repeated carbamazepine levels came down to 20, 11 and then less than 2 mcg/ml after 2 days. The child was monitored by Electroencephalogram (EEG) throughout her admission. She was extubated after 2 days with no further seizure activity or neurological deficit.
Discussion: Carbamazepine can cause seizures in overdose. However, in younger children with no background history of ingestion, diagnosis and management may be challenging. Our patient presented with fever, focal seizures with altered mental status and was being worked up and treated as complex febrile seizures. However, a later revelation of the history of carbamazepine ingestion prompted the treating physicians to start MDAC and trend her carbamazepine levels.
Conclusion: Pediatric carbamazepine exploratory ingestions may masquerade clinically as febrile seizures, thereby making it challenging to correctly diagnose and manage such patients.

KEYWORDS Carbamezepine, Febrile seizure, focal seizure

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236. Fatal Flecainide Overdose

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Background: Management of flecainide overdose has only been studied through case reports, with a recorded mortality rate of 10-22.5%. Successful management is challenging due to its complex pharmacology. We present a case of a fatal flecainide overdose.
Case Report: An 18-year-old male with a past medical history including medulloblastoma status post resection and chemotherapy, ventriculoperitoneal shunt, gastrostomy tube, tracheostomy dependence, seizure disorder, diabetes, hypothyroidism, paroxysmal supraventricular tachycardia, and atrial fibrillation presented to the emergency department (ED) with bradycardia, hypotension, and altered mental status. One week prior to presentation he was admitted to a hospital for atrial flutter during which he suffered a brief cardiac arrest following the administration of esmolol and diltiazem; which was successfully treated with 10 minutes of cardiopulmonary resuscitation (CPR). He was administered flecainide 25 mg Q12h for the duration of hospital stay of one week, and was prescribed nadolol and flecainide upon discharge. At home, he became bradycardic and altered after he inadvertently received a second dose of 25 mL (500mg) of flecainide rather than his prescribed 1.3 mL dose.He presented with a blood pressure (BP) of 67/54 mmHg, heart rate (HR) of 72 bpm, respiratory rate of 12/min on a ventilator, and temperature of 97.2 F. EKG showed a broad complex rhythm, a PR interval of 328 ms, and a QRS duration of 328 ms. Treatment in the ED included several boluses of sodium bicarbonate (SB), an epinephrine infusion (EI), hydrocortisone, as well as 1.5 mL/kg of 20% lipid emulsion (LE) with improvement in HR, BP, and QRS interval. He was evaluated by the extracorporeal membrane oxygenation (ECMO) team who deferred the procedure at the time. In the PICU, an infusion of 0.25 ml/kg/min LE was started, and a decrease in HR to 50 bpm was managed with an additional bolus of SB and an isoproterenol infusion. The patient was weaned off the EI with improvement in BP and lactate levels, and ECMO was once again deferred.A few hours later, the patient suffered a sudden, brief wide complex tachyarrhythmia followed by progressive bradycardia and witnessed cardiac arrest. Resuscitation (for 45 minutes) was unsuccessful.
Case Discussion: The pharmacokinetics of flecainide is complex and complicated by stereoselective and polymorphic metabolism, and

active metabolites. Successful management can be extremely challenging given high oral bioavailability (~90%), slow rate of elimination (~23 hours), with dialysis being poorly effective in elimination. SB may be used to narrow the QRS interval but reduces renal elimination prompting authors to suggest hypertonic saline as an alternative that is yet to be proven beneficial. Amiodarone, lidocaine, and magnesium sulfate have also been reported useful in some case reports. Hyperinsulinemic euglycemic therapy may have potential benefits but has not been studied. Specific indications for initiating ECMO have not been established, but it has been reported successful in multiple cases.
Conclusions: Management of flecainide overdose can be extremely challenging. More research is warranted for establishing the best treatment modalities and specific indications for each modality.

KEYWORDS Flecainide, antiarrhythmic, overdose

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237. Implementation of an emergency department-initiated opioid dependence treatment pathway

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Background: Emergency Departments (EDs) are uniquely positioned to identify patients at risk for opioid-related complications, but have not historically offered medication assisted treatment (MAT). Recently, several studies suggest that ED-initiated programs that include brief interventions and buprenorphine/naloxone initiation have increased engagement with addiction treatment programs and reduced self-reported illicit drug use. In August 2018, the Massachusetts legislature introduced an act that requires EDs to offer MAT. In response, a multidisciplinary workgroup was formed to lead a quality improvement project aimed at increasing MAT in the ED at our institution.
Methods: At a 500 bed teaching hospital, a multidisciplinary workgroup comprised of representatives from the departments of addiction, emergency medicine, and pharmacy collaborated to increase ED-initiated MAT. The workgroup developed and implemented a MAT treatment pathway which started on 9/1/2018. Throughout the project, several interventions were implemented in response to newly identified areas for improvement including but not limited to healthcare staff MAT education, stocking MAT in the automated dispensing cabinets, and obtaining DEA X-waivers. Outcome measures included the monthly number of methadone or buprenorphine/naloxone doses administered in the ED, rescue naloxone administration following MAT in the ED, and number of discharge prescriptions for buprenorphine/naloxone. Statistical significance was determined using Shewhart process control charts.
Results: During the 12 months pre-intervention and 6 months post-intervention, 669 doses of methadone or buprenorphine/naloxone were administered in the ED. In the same time period, the median number of doses significantly increased from 24 to 87 per month. Administration of methadone and buprenorphine/naloxone were similar. Few patients received more than one dose of methadone or buprenorphine/naloxone. The median number of buprenorphine/naloxone discharge prescriptions increased from 0 to 2 per month. No patients received rescue naloxone following MAT administration. All patients receiving MAT were offered the opportunity to meet with the licensed substance abuse counselors and/or addiction medicine specialists for assistance with treatment referrals.
Conclusions: Implementation of a multidisciplinary, ED-initiated treatment pathway for opioid dependence increased the number of patients receiving MAT in the ED.

KEYWORDS Opioid addiction, medication assisted treatment, Emergency Department

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238. Iatrogenic Overdose of Ferumoxytol Infusion in a Pediatric Patient

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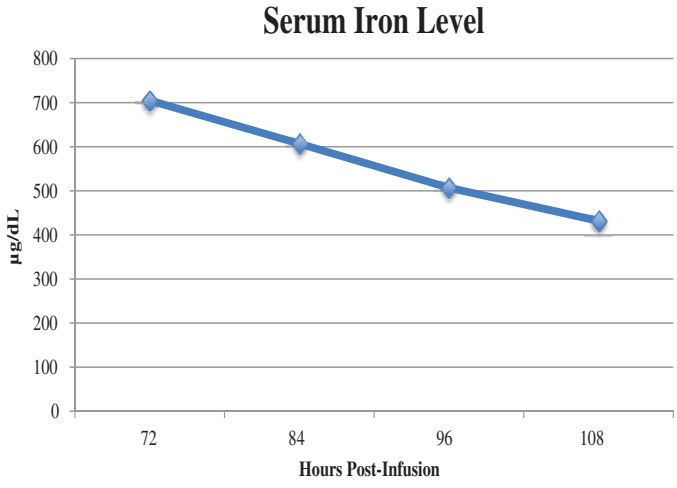
Background: Ferumoxytol is an intravenous (IV) iron preparation used in the treatment of anemia of chronic kidney disease or patients with intolerance to oral iron preparations. The infusion is composed of iron oxide nanoparticles that are coated with a carbohydrate shell. More recently, ferumoxytol is being used off-label as an alternative IV contrast agent in magnetic resonance imaging (MRI). We report a case of ferumoxytol overdose in a patient receiving an IV infusion for MRI.
Case Report: A 14-month-old, 10.4 kg male with a prior history of liver transplantation presents to a hospital for abdominal MRI. An order is placed for a 3 mg/kg ferumoxytol dose as IV contrast media. Though the patient should have been administered 1 mL of ferumoxytol, he instead received the entire 17 mL vial, which contained 510 mg of elemental iron. The medication error was immediately recognized and the patient was monitored overnight. He was asymptomatic and was discharged the next morning. Later that day, a serum iron level drawn the previous night resulted at 825 µg/dL (Ref 50-120 µg/dL) and the family was called to bring the patient back to the hospital. The patient was admitted to the ICU 3 days post-infusion and was started on deferoxamine at 15 mg/kg/hr despite remaining asymptomatic. Serum iron levels were trended (Graph 1). On hospital day 1 of his second admission, patient had serum iron 705 µg/dL, WBC 7,400/mm3, hemoglobin 12.1 gm/dL, and anion gap 8 mmol/L. Deferoxamine was continued at 10 mg/kg/hr on hospital day 2 and then discontinued at 96 hrs post-infusion when serum iron levels reached 507 µg/dL. He was discharged on hospital day 3 without any adverse events.
Discussion: Ferumoxytol contains 30 mg/mL of elemental iron and are supplied in 17 mL vials. Due to medication error, the patient received a 17-fold supratherapeutic dose with a total elemental iron load of 510 mg. The initial serum iron level of 825 µg/dL has been associated with significant systemic toxicity and shock following administration of traditional iron preparations. Despite this, the patient never developed any clinical or laboratory signs of iron toxicity. This is likely



Graph 1. Serum iron level during hospital admission.

due to the fact that the majority of iron is sequestered within a carbohydrate shell and therefore little free iron remains in the serum. The iron-carbohydrate complex circulates in the serum until it enters the reticuloendothelial system where the iron is liberated within the vesicles of macrophages and is stored within ferritin or transported via plasma transferrin.
Conclusion: Despite an inadvertent supratherapeutic administration of a novel iron preparation, which led to markedly elevated total serum iron concentrations, there were no adverse effects reported. A conservative approach to therapy may be warranted with supratherapeutic doses of novel iron preparations.

KEYWORDS Ferumoxytol, Iron, Medication error

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239. A 10-Year Review of Avermectin Exposures Reported to A Poison Control System

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Background: Avermectins are a class of macrocyclic lactone anthelmintic medications. Much of the data on the adverse effects and toxicity of avermectins comes from the treatment of endemic parasitosis, most commonly in Africa and Asia. It is difficult to apply this data to cases of overdose and to populations in the United States.
Methods: This is a retrospective chart review of 10 years of single substance exposures to macrocyclic lactones reported to a poison control system. Substance codes for avermectins were used to identify potential cases. Complete charts were requested for those patients who were referred to a healthcare facility (HCF). Exclusion criteria were polysubstance exposures and informational calls. Case records were abstracted by two reviewers for location of care, age, gender, specific substance, dose, intent, route and treatment. Clinical effects were categorized as cardiac, central nervous system (CNS), gastrointestinal (GI), dermatologic, ocular, respiratory, and other/metabolic.
Results: Four hundred twenty-five exposures were identified over 10 years. Of those, 125 cases were treated in or referred to a healthcare facility. Eighty-seven cases met inclusion criteria and were abstracted. The number of exposures referred to or treated in a HCF were 4, 3, 3, 8, 6, 7, 13, 11, 17, and 15 from the years 2009 to 2018, respectively. Sixty-four (72.7%) were oral exposures, and 11.4%, 5.7% and 10.2% were inhalation, dermal, and other/unknown, respectively. Twenty-five percent of exposures were less than 6 years of age, and 60% of exposures were between 19-59 years of age. Ninety-one percent of exposures under age 12 were unintentional. The commonest intents were unintentional (28.7%), therapeutic misadventure (23%), and delusional parasitosis (17.2%). The commonest symptoms reported were CNS (36.8%), GI (31%), and ocular (11.5%). Thirty (34.5%) cases reported no symptoms. The commonest symptoms were nausea (10), vomiting (10), altered mental status (9), vision changes (7), dizziness (7) and paresthesias (7).
Conclusions: Reported avermectin exposures have increased over the past decade, though the reason is not immediately apparent. Exposures also exhibited a bimodal age distribution, disproportionately affecting individuals less than 6 and greater than 18 years old. Almost all cases under 12 years old were unintentional, which is concordant with what is known about exposures in young children. Increased exposure after 18 years old could be attributed to the peak in development of psychiatric disease in the third decade of life. The commonest reasons for exposure in this group were therapeutic misadventure and delusional parasitosis. As avermectins are available for veterinary use without a prescription, individuals with delusional parasitosis have little difficulty obtaining this medication. The predominance of reported CNS symptoms could be explained by ivermectin's agonist effect at the gamma-aminobutyric acid-A receptor. At antihelminthic doses, the p-glycoprotein transporter is effective at protecting the central nervous system from ivermectin. This protection is lost in overdose. One limitation of this review is the inability to quantify many of the exposures given that most doses were described in number of tubes and not gram amounts.

KEYWORDS Avermectins, Ivermectin, Delusional Parasitosis

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240. A case of low potassium?

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Background: The Tennessee Department of Health's (TDH) Environmental Epidemiology Program (EEP) was contacted by a regional medical director about a patient with a dangerously high barium blood level. The patient was admitted at least 16 times to Veteran's Administration (VA) hospitals around Tennessee over a two-year timespan. The patient thought something in their newly built home was causing their illness and repeated hospitalizations. The patient lead a quiet life, was not working, had a restricted diet, never venturing too far from home. The patient was initially diagnosed with hypokalemic periodic paralysis although the patient lacked some of the characteristic features. Potassium levels swung widely from 1.8 to 8.0 milliequivalents per liter without obvious cause. One of the patient's VA physicians ordered a heavy metal blood screen during a later hospitalization. Results revealed a highly elevated blood barium level over 14,000 micrograms per liter (µg/L), 35 to 70 times higher than the high end of published normal ranges. After another hospitalization, the patient was afraid to return to their home. Patient's Blood Barium ResultBarium, Blood: 14550.0 ng/ml Whole blood barium. Result verified by repeat analysis.
Methods: TDH EEP enlisted help of the U.S. Environmental Protection Agency (EPA), the Agency for Toxic Substances and Disease Registry (ATSDR), the Tennessee Department of Environment and Conservation (TDEC), and the local Poison Center to develop a barium exposure survey. The survey asked 85 questions regarding lifestyle, food habits, occupational and medical history, military deployment history, travel, and hobbies. It took all of a 1.5 hour telephone call to survey the patient. After the survey failed to identify a definitive link between the patient's lifestyle and elevated blood barium, we asked to evaluate the patient's home to look for any item containing high levels of barium. Access to the family's home was granted and a home visit scheduled with EPA, TDEC and EEP. Various materials inside and outside the home were tested using a handheld X-ray fluorescence scanner. Items included fabrics, bedding, carpeting, countertops, dry goods, refrigerated items, garage items, vacuum cleaner dust, interiors of automobiles, etc. Soil around the home, water supplied to the home, and ventilation system return dust were also tested. Other family members' blood barium levels were tested; No elevated blood barium levels were found.
Results: X-ray fluorescence, soil, water, and dust testing did not find anything in the home that could cause barium poisoning. TDH EEP provided a letter report of ours, TDEC's and EPA's findings to the patient.
Conclusion: The patient's wife attempted to murder the patient outside the home where they were living. She shot the patient and left him for dead in the woods and casually drove with their children to a public place where she was apprehended and arrested by authorities.

Our documentation for the case led to a second count of attempted murder by poisoning brought against the patient's wife.

KEYWORDS Poison, barium, investigation

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241. Levocarnitine for the Treatment of Valproic Acid-Induced Hyperammonemic Encephalopathy in Children: The Experience of Large, Tertiary Care Pediatric Hospital and a Poison Center

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Background: Although rare, symptomatic hyperammonemia is sometimes associated with valproic acid (VPA), especially in children. L-carnitine (levocarnitine), sometimes classified as an essential amino acid, is vital to mitochondrial utilization of fatty acids and can be helpful in treating this condition. The data supporting this, however, are limited.
Study Question: The aim of the study was to illustrate the role of L-carnitine in the treatment of patients with VPA-induced hyperammonemic encephalopathy (VPE) at 2 different institutions.
Methods: Medical records of affected patients were reviewed; data collected included exposure history, clinical manifestations, physical examination, and laboratory values.
Results: There were 13 cases of VPE; 12 were associated with therapeutic dosing and 1 with an overdose. The maximum ammonia concentration was 557 µmol/L, and blood concentrations of VPA ranged from 68 to 600 µg/mL (therapeutic range 50-100 µg/mL). In all cases, liver function tests were normal or only mildly increased. In this study, 12 patients received a daily dose of L-carnitine 100 mg/kg, and 1 received 200 mg/kg (intravenous infusion over 30 minutes) divided every 8 hours until clinical improvement. All patients made a full recovery. None developed adverse effects or reactions, and no cases of toxicity were reported.
Conclusion: Our series suggests that intravenous L-carnitine, at a dose of 100 mg·kg·d in 3 divided doses each over 30 minutes until clinical improvement occurs, is a safe and effective treatment in the management of VPE in children.

KEYWORDS Levocarnitine, Valproic acid induced encephalopathy, ammonia

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242. A 10 year retrospective review of otic exposures reported to a single poison control center

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Background: Information on atypical routes of exposures is scant in the toxicology literature. We performed a retrospective case series review of all otic exposures reported to a single poison control center over a 10 year period. The objective of this study was to characterize the epidemiology of otic exposures to one Poison Control Center (PCC).
Methods: A total of 369 exposures with otic coded as the exposure route from Jan 1, 2008 to Dec 31, 2018 were analyzed. After excluding cases with multiple routes of exposure, and therapeutic errors of products meant to be instilled in the ear, we identified 232 cases. Data abstracted included patient demographics, the reason for exposure, management site and outcome of these exposures.
Results: During the study period, 58% were 18 years or older, 22% were 0-5 and 15% were 6-12. The top 3 reasons for exposure for all substances were unintentional therapeutic error (n = 80, 35%), unintentional misuse (n = 68, 29%) and unintentional general (n= 40, 17%). Males accounted for 66% of patients. The top major categories of substances reported were pharmaceuticals (n = 82, 35%), hydrocarbons (n = 64, 28%), and household products (n = 51, 22%). For pharmaceutical products 82% (n = 67) of exposures were due to therapeutic errors. The majority of these (n= 42, 63%) were related to an incorrect route of administration. Gasoline accounted for 60 of the 64 hydrocarbon otic exposures. The most common symptom reported with hydrocarbon exposure was pain / irritation (80%). Half of all gasoline exposures had a minor effect, the other half were either not followed or unable to be followed. Thirty eight percent of all cases (n = 88) were referred to a health care facility (HCF), and 11% (n = 25) were already in a HCF when the PCC was consulted. Hydrocarbons made up 46% of HCF visits (n = 52). Twelve cases which were seen in a HCF and were followed to a known outcome did not receive any treatment. Fifty five percent of patients treated in a HCF and followed to a known outcome received no therapy other than flushing of the affected ear, 6 received antibiotics and 1 received steroids. Over half (n = 124, 53%) were not followed because no more than minor clinical effects were expected. There were 60 patients with minor effects, 9 with no effects and 2 with moderate effects. The moderate outcome cases involved sulfuric acid or ant/roach killer and resulted in bleeding, edema, pain and/or blisters in the ear. There were no major effects in this study.
Discussion: Pharmaceutical products accounted for the highest percentage of cases, the majority related to confusion of the product being used. The most common clinical effects for all substances reported were pain / irritation (28%) with the most common therapy reported as flushing.
Conclusion: Otic exposures reported to the poison center were overall tolerated well. Cases treated in a HCF rarely received treatment beyond flushing. Clear labeling to distinguish between ophthalmic and otic routes may reduce route confusion related to dropper bottles.

KEYWORDS Otic, Misuse, Ear exposures

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243. Phenibut withdrawal syndrome successfully treated using oral baclofen

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Background: Phenibut is a glutamic acid derivative GABA-B receptor agonist used recreationally for its anxiolytic and euphoric effects. It is easily accessible for use, and tolerance is thought to develop within 1 to 2 weeks of use. Abrupt discontinuation of phenibut has been reported to cause withdrawal symptoms; however, there is a paucity of data regarding the treatment of this withdrawal syndrome. We report here a case of phenibut withdrawal presenting as acute agitation and hallucinations, successfully treated with baclofen.

Case Report: A 34-year-old male with past medical history of anxiety, depression, migraines, and prior alcohol use disorder presented to the Emergency Department (ED) with agitation, garbled speech, and visual hallucinations for three days. For the past two months, the patient had been self-treating his anxiety with 10 grams/day of phenibut, after he ran out of the lorazepam he was previously prescribed for this purpose. He stopped using phenibut 5 days prior when he was admitted to a rehabilitation facility. On arrival, vital signs were BP 144/108 mmHg, HR 134, RR 24, T 98.6 degrees Fahrenheit, and SpO2 98% on room air. On exam, patient was tremulous and diaphoretic. Pupils were equal, round, and reactive to light, 3-4mm, no nystagmus. Heart rate was tachycardic with regular rhythm. Lungs were clear to auscultation bilaterally. Abdomen was soft, bowel sounds normoactive, with mild diffuse tenderness. Patient was alert and oriented to person, but not place or time, with active visual hallucinations. Cranial nerves intact, normal muscle tone, with reflexes 2/4 in the bilateral upper and lower extremities, no clonus. Skin was warm and diaphoretic. Electrocardiogram showed normal sinus rhythm with a rate of 85, PR 160, QRS 82, and QTc 435. Serum chemistry and complete blood count were within normal limits. Ethanol, salicylate, and acetaminophen were not detected. Patient was started on intravenous (IV) lorazepam. Toxicology was consulted and recommended starting patient on baclofen 5 mg three times daily. He was also placed on a dexmedetomidine infusion for 24 hours. He was continued on baclofen for 72 hours with decreasing benzodiazepine requirements and improvement of his psychotic symptoms. He was then slowly tapered off baclofen over 5 days.
Case Discussion: As phenibut is primarily a GABAB agonist, we surmised that baclofen, with its GABAB agonism, may have synergistic effects with lorazepam, primarily a GABAA agonist. Baclofen is structurally similar to phenibut and there have been few case reports discussing its use in phenibut withdrawal. Prior to the addition of baclofen, the patient had been requiring large doses of parenteral lorazepam. After the addition of baclofen, patient had decreasing benzodiazepine requirements and overall improvement of the hallucinations and agitation. Administration of dexmedetomidine confounds interpretation of baclofen's effectiveness, but the brief infusion did not result in decreased agitation and likely did not contribute significantly to clinical improvement.
Conclusions: Phenibut is an easily obtainable xenobiotic that is increasingly being used recreationally. As use continues to rise, toxicologists need to be aware of the potential for withdrawal from this agent, as well as methods for treatment.

KEYWORDS Phenibut, Withdrawal, Drugs of abuse

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244. PharmacoXray - The Radiopacity of Sustained Release Pharmaceuticals

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Background: Concerning ingestions presenting to the Emergency Department often involve sustained-release preparations, where life-threatening toxicity may manifest in a delayed fashion. Helpful to the clinician would be a screening tool to assist clinical decision making in ambiguous cases. Abdominal x-ray is a rapid, readily available, non-invasive tool involving minimal risk. Prior studies have shown mixed results as regards the clinical usefulness to identify or confirm ingestions. It has been almost 15 years since it was last evaluated, with subsequent changes in medication formulations and x-ray technology. We describe the radiolucency of current commonly available sustained release medications in a simulated patient model to determine whether new technology and formulations warrant a re-evaluation of this technique in suspected overdose.
Methods: The formulary of an inner-city teaching hospital was reviewed for all extended-release formulations. Controlled substances were excluded. 16 extended release medications were identified. A Hologic model DPA/QDR-1 Anthropomorphic Spine Tissue Phantom, used to calibrate bone densitometers, approximated the average human abdomen in a supine position. Two of each medication were placed underneath in a transparent plastic bag, "2" being the smallest "multiple-ingestion" possible. A U.S. quarter was used to provide contrasting radiopacity.
Results: There was a spectrum of radiopacity found, ranging from phenytoin (most radiolucent) to potassium (most radiopaque). 11 of 16 medications were readily identified, whereas 5 were more difficult to discern. Of the agents that would be most concerning from a toxicologic perspective (i.e. the sustained release calcium channel blockers diltiazem, nifedipine, verapamil), all were easily identified. There were three agents that were not easily identified of potential concern (i.e. venlafaxine, duloxetine, metoprolol).
Conclusions: We were able to create a visual guide of the radiopacity of sustained release medications using abdominal x-ray. The most concerning agents available on our formulary, as regards significant toxicity, were easily identified. This model may be an effective screening adjunct in the evaluation of a patient with a reported acute overdose of concerning sustained release medications.

KEYWORDS Sustained-release medication, diagnostic, x-ray

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245. Prevalence of Illicit Drug and Non-Medical Prescription Drug Use among Pregnant Women in the United States

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Background: Illicit drug use and non-medical prescription drug use during pregnancy have the potential to acutely harm the mother and fetus as well as lead to teratogenic changes that may affect the child for life. Assessing illicit drug use and non-medical prescription drug use in pregnant women is challenging as many patients are hesitant to disclose such use, yet reliable data sources are needed to inform medical providers and public health officials in order to better tailor patient education and clinical care.
Methods: The Survey of Non-Medical Use of Prescription Drugs Program is a cross-sectional, online anonymous survey of the general adult population in United States. Using data from the third quarter 2018 survey launch, prevalence estimates of past month use of any illicit drug or non-medical use (NMU) of any prescription pain reliever,

Table 1 Prevalence of Past Month Illicit Drug Use or Non-Medical Prescription Drug Use Among Pregnant Women Ages 18-49 Years.

	Past Month Use	
	Prevalence % (95% CI)	Estimated No. of Pregnant Women
Any Drug Below	9.35 (5.79, 12.92)	228,291
Rx Pain Reliever NMU	1.55 (0.36, 2.75)	37,896
Rx Sedative NMU	1.52 (0.43, 2.61)	37,104
Rx Stimulant NMU	2.09 (0.65, 3.53)	50,974
Cannabis	7.49 (4.18, 10.80)	182,806
Any Illicit Drug Use	8.86 (5.37, 12.36)	216,296
Any Illicit excluding Cannabis Use	2.90 (1.20, 4.59)	70,691

sedative, or stimulant in respondents were calculated among those self-identified as pregnant and between 18-49 years of age. Data were weighted to provide national prevalence estimates. Illicit drugs included anabolic steroids not prescribed by a healthcare professional, alkyl nitrites, nitrous oxide, cannabis, cocaine powder, crack cocaine, gamma hydroxybutyrate/gamma butyrolactone, heroin, ketamine, kratom, lysergic acid diethylamide, 3,4-methylenedioxymethamphetamine, mephedrone, mescaline, methamphetamine, non-pharmaceutical amphetamine, non-pharmaceutical fentanyl, phencyclidine, psilocybin or mushrooms, salvia, or synthetic cannabinoid receptor agonists. NMU was defined as use in any way not directed by a healthcare provider. The prevalence of any drug use in the past month among respondents with different demographic characteristics were explored; differences were tested using Rao-Scott chi-square tests.

Results: An estimated 9.35% of pregnant women in the US ages 18-49 years have used an illicit drug or non-medically used a prescription drug in the past month; this corresponds to an estimated 228,291 pregnant women nationally (Table 1). Such use was largely driven by illicit drug use, with a past month use prevalence of 8.86%, and specifically cannabis, with a past month use prevalence of 7.49%. Of the prescription drug classes examined, the prevalence of prescription stimulant NMU had the highest prevalence at 2.09%, corresponding to an estimated 50,974 pregnant women nationally. The prevalence of past month use of any illicit drug or NMU of any prescription pain reliever, sedative, or stimulant was significantly higher among Hispanic pregnant women compared to non-Hispanic pregnant women (22.46% vs 6.73%, p = 0.0015, respectively) and among pregnant women who had experienced chronic or acute pain in the past year compared to those who had not (chronic: 24.29% vs 6.71%, p = 0.0004; acute: 21.49% vs 6.61%, p = 0.0013, respectively).

Conclusions: Illicit drug use and non-medical prescription drug use during pregnancy is not uncommon and may disproportionately occur in Hispanic women and women who have experienced pain in the past year. Further studies to better characterize the details of these use patterns and their changes over time are warranted.

KEYWORDS Pregnancy, Non-medical use, Cannabis

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246. Pharmaceutical Exposures in Pregnant Women Reported to the National Poison Data System from 2013 to 2018

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Background: According to the American Association of Poison Control Centers' National Poison Data System (NPDS) annual reports, approximately 6000-8000 calls to poison centers regarding exposures in pregnant women are made each year. Exposures in this population are unique in that they may induce teratogenic changes that could affect the newborn for life, yet pregnant women remain underrepresented in virtually every area of toxicologic research. More robust research on exposures in this population are needed in order to better inform emergency, obstetric, and neonatal care. Therefore, the purpose of this study is to describe pharmaceutical exposures in pregnant women reported to NPDS from 2013 to 2018.

Methods: The 20 most frequently reported pharmaceutical generic codes among exposures in pregnant women for each year between 2013 and 2018 were extracted from NPDS. Pregnancy was defined as any patient whose gender was coded as "Pregnant" as well as any female whose pregnancy status was positive. Demographic information as well as information on the reasons for exposure, clinical effects of exposure, and medical outcomes were collected. Descriptive statistics were performed.

Results: Between 2013 and 2018, there were 9343 total pharmaceutical exposures reported in pregnant women associated with the 20 most frequently reported generic codes for each year. In this cohort, the mean patient age was 26.4 years and the mean duration of pregnancy at the time of the call was 18 weeks. Acetaminophen was the most commonly reported exposure; multivitamins with iron, ibuprofen, benzodiazepines, and antibiotics were other commonly reported exposures (Table 1). The majority of exposures were single product exposures (n = 6827, 73.1%) and intentional (n = 4970, 53.2%), although among single product exposures there were roughly equal numbers of intentional and unintentional exposures whereas among multiple product exposures, over three-quarters were intentional (Table 2). Though many exposures were managed on site, not at a healthcare facility (n = 3557, 38.1%), nearly a quarter required admission to a medical unit (n = 2178, 23.3%) and 1106 (11.8%) required admission to a critical care unit. A moderate or major effect was noted in 1443 (15.4%) and there were 11 deaths (0.1%); nearly 40% were not followed (Table 3).

Conclusions: Common pharmaceutical exposures in pregnant women reported to poison centers in the United States include over-the-counter analgesics and multivitamins with iron as well as certain prescription products such as antibiotics and benzodiazepines. Despite the common myth that pregnancy is protective against suicidal ideation and self-harm attempts, over half of the exposures in our cohort were intentional and over one-tenth required admission to a critical care unit. As such, prioritizing the inclusion of pregnant women in future studies on toxicologic exposures and focusing research attention on this vulnerable population is paramount.

KEYWORDS Pregnancy, Pharmaceutical exposures, National Poison Data System

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Table 2 Characteristics of Pharmaceutical Exposures in Pregnant Women Reported to the National Poison Data System by Year, 2013-2018.

	Single Product Exposures, N (%)	Multiple Product Exposures, N (%)	Total Exposures, N (%)
Intentional	3000 (43.9%)	1970 (78.3%)	4970 (53.2%)
Unintentional	3382 (49.5%)	420 (16.7%)	3802 (40.7%)
Adverse Reaction	318 (4.7%)	75 (3.0%)	393 (4.2%)
Other or Unknown	127 (1.9%)	51 (2.0%)	178 (1.9%)
Total	6827 (100%)	2516 (100%)	9343 (100%)

Table 1 Five Most Common Pharmaceutical Exposures in Pregnant Women Reported to the National Poison Data System by Year, 2013-2018.

	2013	2014	2015	2016	2017	2018
Acetaminophen Alone, Adult	N = 286	N = 291	N = 244	N = 305	N = 244	N = 253
Multivitamins with Iron	164	187	176	166	153	163
Ibuprofen	158	160	160	156	148	145
Benzodiazepines	169	146	134	127	126	111
Systemic antibiotics	149	117	120	142	118	122

Table 3 Medical Outcome of Pharmaceutical Exposures in Pregnant Women Reported to the National Poison Data System by Exposure Reason, 2013-2018.

	Intentional (N = 4970)	Unintentional (N = 3802)	Adverse Reaction (N = 393)	Other/Unknown (N = 178)	Total (N = 9343)
Treated/evaluated and released	1304 (26.2%)	324 (8.5%)	37 (9.4%)	45 (25.3%)	1710 (18.3%)
Admitted to critical care unit	1035 (20.8%)	39 (1.0%)	5 (1.3%)	27 (15.2%)	1106 (11.8%)
Admitted to noncritical care unit	949 (19.1%)	70 (1.8%)	15 (3.8%)	38 (21.3%)	1072 (11.5%)
Admitted to psychiatric care facility	1113 (22.4%)	19 (0.5%)	1 (0.3%)	12 (6.7%)	1145 (12.3%)
Patient refused referral/did not arrive at facility	65 (1.3%)	35 (0.9%)	9 (2.3%)	4 (2.2%)	113 (1.2%)
Patient lost to follow up/left against medical advice	333 (6.7%)	123 (3.2%)	43 (10.9%)	26 (14.6%)	525 (5.6%)
Managed on site (non-healthcare facility)	144 (2.9%)	3123 (82.1%)	268 (68.2%)	22 (12.4%)	3557 (38.1%)
Not followed/unknown	27 (0.5%)	69 (1.8%)	15 (3.8%)	4 (2.2%)	115 (1.2%)

247. A Pregnant Pause: Tizanidine withdrawal with successful conversion to clonidine

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Background: Tizanidine is an imidazoline derivative with central analgesic properties used for musculoskeletal pain. It is a central alpha 2 receptor agonist like clonidine but has a shorter half-life and duration of action. There are limited reports of tizanidine misuse in the literature. We report a case of tizanidine withdrawal in a pregnant woman that was treated with clonidine.

Case report: A 31-year-old pregnant woman at 30-weeks' gestation presented to the obstetric clinic for routine follow up. She was noted to have a systolic blood pressure of 230 mmHg. In the office, she experienced abdominal pain, headache and anxiety. She reported that she missed her dose of tizanidine. An ultrasound done in the clinic revealed a live pregnancy with intrauterine growth retardation and she was admitted to the labor and delivery floor. Her past surgical history was significant for a Roux-en-Y gastric bypass performed 7 years prior to presentation. Her past medical history included untreated hypertension and a prior history of opioid misuse. She is prescribed tizanidine 6 mg orally three times daily as needed for pain however she reported ingesting it as frequently as every two hours and up to 16 mg at bedtime. Her other prescribed medications included clonazepam 2mg orally three times daily, fluoxetine 125 mg orally daily, zolpidem 10 mg orally at night three times a week for sleep as needed, acetaminophen 650 mg for pain and ondansetron 4mg for nausea as needed. All these medications were restarted in the hospital, thiamine and folic acid were added. On physical examination, she was increasingly anxious, agitated and uncomfortable. Vital signs: T 97.7 °F, BP 207/123 mmHg, HR 108/min, RR 16/min, 98% on room air. She was alert and had an otherwise normal physical examination. Laboratory values were unremarkable and pre-eclampsia was ruled out. Her hospital course was complicated by increasing requirements of tizanidine to control her blood pressure, pain and anxiety, and was eventually receiving 48mg daily. The toxicology team was consulted on day three of admission and recommended gradually switching tizanidine to clonidine. Based on healthy

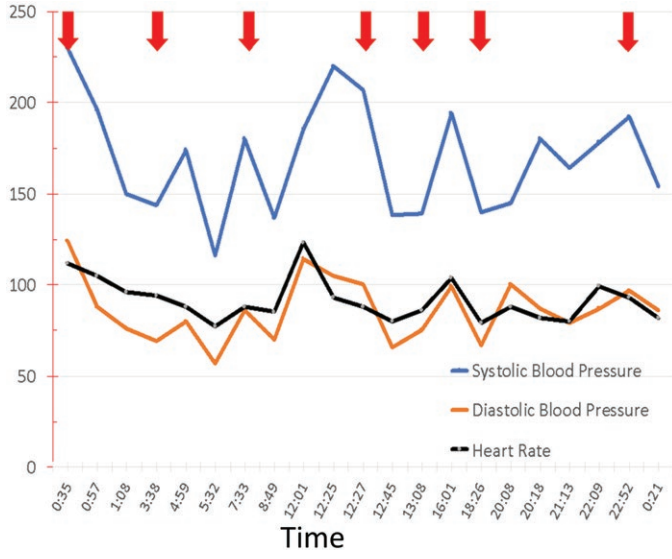

Figure 1a Vital signs on day 3 of admission prior to starting clonidine. Arrows represents time of tizanidine administration.

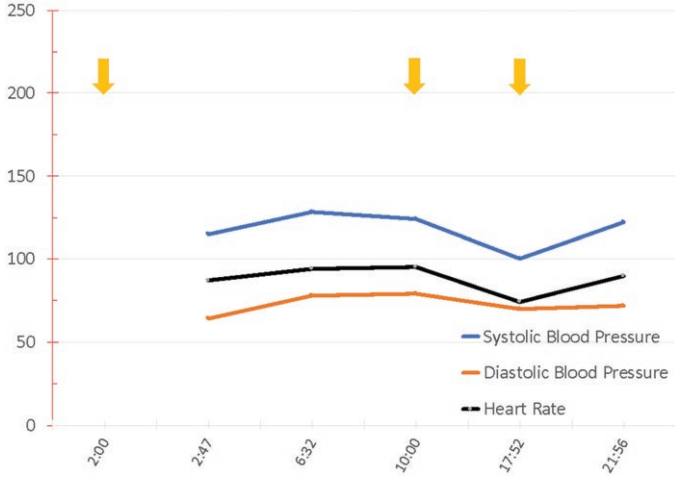

Figure 1b vital signs on day 11 of admission after discontinuation of tizanidine. Arrows represents time of clonidine administration.

Table 1 Oral conversion of tizanidine to clonidine.

	Daily Oral Tizanidine requirements in mg	Oral Clonidine dose in mg
Day 4	36	0
Day 5	48	0
Day 6	16	0.1 mg three times daily
[…]	[…]	[…]
Day 10	3	0.2 mg three times daily
Day 11	0	0.2 mg three times daily

volunteer data, 12mg of tizanidine had similar effects as 0.15mg of clonidine. We converted the daily requirements of tizanidine to clonidine-equivalents and we started at 50% the calculated clonidine dose to avoid hypotension. After 7 days of slow titration, she was placed on clonidine 0.2mg three times daily and tizanidine was weaned off (Table 1). Her blood pressure and heart rate were less labile and anxiety and pain were tolerable (Figure 1a,b).

Discussion: Tizanidine induced tolerance and withdrawal are not well reported in the literature. Similarly, the safety of tizanidine in pregnancy is unknown. Due to the short duration of effects and labile vital signs when she began to withdraw from tizanidine, she was switched to clonidine as it has similar mechanism of action but has a longer half-life.

Conclusion: We report a case of tizanidine dependence in pregnancy that was successfully transitioned to oral clonidine. Tizanidine dependence may become more prevalent as doctors are seeking opioid sparing pain medications.

KEYWORDS Tizanidine, withdrawal, pregnant

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248. Cardiovascular effects of inhaled and ingested cannabis products

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Background: Tetrahydrocannabinol(THC) has been associated with tachycardia in experiments of smoked botanical cannabis. Though high-concentration edibles and concentrates are increasingly available, there is little data on their cardiovascular effects. Research question: Is there a relationship of HR with method of cannabis use, specifically inhalation versus ingestion, in cannabis toxicity?

Methods: Prospective study of subjects where the regional Poison Center(PC) was contacted about a cannabis exposure between 12/4/2015-4/15/2017. A data collection sheet prospectively captured clinical and product data, including method of use and cannabis type. PC charts were searched for cannabis substance codes. Inclusion criteria: cannabis exposure with documented route of exposure and documented HR. Exclusion criteria: co-ingestion of other medications/drugs. Chi Square & Fisher's Exact test were used to compare frequencies of events.

Results: We identified 133 patients in 16 months(35 inhalation; 98 ingestion). Ages ranged from 8mo-76y and 56%(75/133) were male. 35 subjects reported inhalation of cannabis products (22 botanical; 13 concentrated products) and 74%(26/35) had tachycardia. All subjects (100%;22/22) who inhaled botanical cannabis(smoked) had tachycardia, compared to 31%(4/13) who inhaled concentrates (p 98 subjects reported ingestion of cannabis products (76 edibles; 6 botanical; 16 concentrated products) and 50%(49/98) developed tachycardia. Most subjects who ingested edibles(64%; 49/76) and 6/6(100%) who ingested botanicals developed tachycardia, whereas most subjects who ingested concentrated products had normal HR 69%(11/16), none(0%; 0/16) had tachycardia, and 31%(5/16) had bradycardia. Ingestion of edibles were more likely to produce tachycardia than ingestion of concentrates (p Patients who inhaled cannabis were more likely than those who ingested cannabis to develop tachycardia. (p = 0.03)

Conclusion: In patients with cannabis toxicity, tachycardia is more common in those who inhaled cannabis than those who ingested cannabis and more common in ingestions of edibles than of concentrates. Bradycardia was seen most commonly in patients who ingested or inhaled concentrates. Tachycardia was seen in all botanical cannabis smokers.

KEYWORDS Cannabis, Tachycardia, Bradycardia

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249. Baclofen Exposures Reported to US Poison Control Centers from 2000-2017

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Background: Baclofen is a Gamma Aminobutyric Acid (GABA)B receptor agonist. Introduced in 1977 for the treatment of spasticity, baclofen is also used off-label for neuralgia, alcohol use disorder, and other purposes. Large US studies on baclofen exposures are limited. This study describes the demographics and clinical characteristics of baclofen exposures reported to US Poison Control Centers (PCCs) over 17 years.

Methods: A retrospective review of baclofen exposures reported to US PCCs from 2000-2017 was performed. Exposures were excluded if the following medical outcomes were coded: a) confirmed non-exposure and b) unrelated effect- the exposure was probably not responsible for the effects. For information on medical outcomes, clinical effects, and therapies provided, exposures including coingestants were excluded.

Results: A total of 45,355 baclofen exposures were identified after exclusion criteria were applied. The mean patient age was 36.2 years (range 1 day–116 years; median 38.0 years; SD 20.3 years). The sample was 58.7% female. The top three reasons, accounting for 77.1% of all exposures, were intentional suspected suicide (n = 20,764; 45.6%), unintentional therapeutic error (n = 8,364; 18.4%), and unintentional general (n = 5,959; 13.1%). The average percentage increase in exposures per year was 11.8% (SD: 7.5%; range 1.9-37.1%). Nearly half of exposures were to baclofen alone (n = 20,609; 45%), while 24,926 (55%) included 1 or more coingestants. Seventy-nine percent of exposures were managed in a healthcare facility (HCF). Of those managed in a HCF, 46.8% (n = 16,875) were admitted to a critical care unit. In baclofen-only exposures, the most common medical outcome was "moderate effect," which occurred in 5,573 exposures (27%). Major outcomes occurred in 2,033 (9.9%), and there were 50 related deaths. The most common effects were drowsiness/lethargy (n = 8,440; 41%), confusion (n = 2,798; 13.6%) bradycardia (n = 2,348; 11.4%), and coma (n = 2,202; 10.7%). The following effects were observed in 5% to 10% of cases: vomiting (n = 1,791; 8.7%), tachycardia (n = 1,748; 8.5%), respiratory depression (n = 1,613; 7.8%) and hypertension (n = 1,494; 7.3%). The following therapies were provided in more than 10% of single-substance exposures: intravenous fluids (n = 6,247; 30.3%), oxygen (n = 3,935; 19.1%), intubation (n = 2,912; 14.1%), ventilation (n = 2,676; 10.8%), and benzodiazepines (n = 2,156; 10.5%).

Conclusion: Baclofen exposures reported to US PCCs have increased over the past 17 years. Intentional suspected suicide was the most common reason for exposure during the study period. Drowsiness/lethargy, confusion, bradycardia, and coma

were the most common clinical effects reported. Nearly half of those exposures managed in a HCF were admitted to an ICU. Major outcomes, including death, were reported; however airway management and supportive care appear to manage baclofen exposures successfully.

KEYWORDS Baclofen, Poison Control Centers, Critical care

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250. Familiarity and use of alternative substances for opioid use disorder

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Background: Opioid use disorder affects over 2.1 million people in the United States. Loperamide, kratom and tianeptine have been used to treat opioid withdrawal as well as opioid substitutes. Our institution has a bridge clinic serving adult patients motivated to begin (or re-establish) buprenorphine treatment. Patients are managed, anywhere from 1 - 10 weeks, until they are assisted into a long term clinic for ongoing management.
Objectives: To determine the familiarity and previous use of one of these alternate pharmaceuticals in patients being treated at an outpatient dependency clinic for initiation of buprenorphine treatment. We also sought to determine the likelihood that an attendee to this clinic would resume use of one of these drugs if they lost access to buprenorphine.
Methods: All patients age 18 and older at the outpatient bridge clinic were asked to complete a brief questionnaire at the end of their appointment. Participation was voluntary and anonymous. An envelope with the survey was given to all of the patients. The patients were left in the exam room alone and asked to deposit the survey in the envelope (whether completed or not) into a lock box in the waiting room. Surveys were not reviewed from the lock box until 12 weeks after study initiation to ensure anonymity.
Results: Overall participation was poor. 41 surveys were returned over the 12 week duration. 39/41 surveys were completed. Participant ages were 30-39 years (15), 40-49 (8), over 50 (5) and 3 did not report an age. There were 7 females, 17 males and 7 chose not to declare. 7 patients reported previous use of loperamide (up to 80 pills daily). Two of these 7 indicated they would use loperamide again if they lost access to buprenorphine and another 7 patients without prior use loperamide would either somewhat likely or very likely try loperamide. Five patients (12%) reported previous use of kratom. Four of those 5 would use again if they lost access to buprenorphine and another 3 without previous use indicated they would consider use if they lost access to buprenorphine. None of the patients reported previous use of tianeptine and only 2 would consider using it if they lost access to buprenorphine.
Discussion: Alternate medications are being increasingly used in patients with opioid use disorder and 29% of our surveyed patients report having used either loperamide or kratom in the past (and 1 of 12 with prior use of both). More interestingly, 14 of 41 (34%) patients reported that they would be likely to use one of these alternatives if buprenorphine became unavailable. There are several limitations including convenience sample, small sample size and overall poor participation. (It is unclear how many surveys were discarded rather than returned blank so the total denominator is unknown).
Conclusion: Though our population has a low incidence of using these substances, their likelihood of using one of these if they lose their access to buprenorphine is high. The importance of medication assisted therapy should be underscored.

KEYWORDS Loperamide, kratom, tianeptine

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251. Cannabis Use and High Risk Substance Use Behaviors in the United States Non-Medical Use of Prescription Drugs (NMURx) National Survey

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Background: Patterns of cannabis use associated with prescription drug and illict drug misuse and abuse are poorly understood. Conflicting data have been used to conclude that cannabis use is associated with lower risks of opioid use, as well as with higher risks of opioid use. To better understand substance use behaviors of those who use cannabis, we compared the proportion of various substance use behaviors among those use who cannabis, smoke cigarettes, and use alcohol using the United States Non-Medical Use of Prescription Drugs (NMURx) national survey.
Methods: NMURx is an online cross-sectional, anonymous survey of the general adult population in United States. NMURx collects data on nonmedical use (NMU) of prescription drugs, illicit drugs, and over the counter medications as well as demographics and behaviors. Prescription drug NMU was defined as use in a way not directed by your healthcare provider. NMURx data from the third quarter of 2018 are presented. Calibration weights were applied to represent national adult population distributions. National prevalence and proportion of past 12 month behaviors among those who use cannabis, smoke cigarettes, and drink more than 7 alcoholic beverages per week are reported. Percent of those with each associated behavior and 95% confidence intervals are reported.
Results: In the United States, the estimated national prevalence of cannabis use in the past 12 months was 45,109,079 adults or 17.9% (17.4-18.4% 95% CI), estimated prevalence of cigarette smoking was 35,144,420 adults or 13.9% (13.6-14.3%), and estimated prevalence of drinking more than 7 alcoholic beverages per week was 25,857,764 adults or 10.3% (9.9-10.6%). Among those who used cannabis, 14.9% (13.9-15.9%) also nonmedically used prescription opioids compared to 3.8% (3.5-4%) of those who did not use cannabis. Similarly, 19.0% (17.8-20.1%) endorsing cannabis use also reported use of an illicit drug, compared to 3.8% (3.5-4%) of those who did not use cannabis. Overall, cannabis users had higher rates of other substance use behavior than those who did not use cannabis, including injection of prescription opioid pills, and higher Drug Abuse Screening Test (DAST-10) scores (Table 1). However, similar patterns of substance use behaviors were also observed among those who smoke cigarettes and those who drink greater than 7 alcoholic drinks per week. Cannabis use was associated with slightly higher rates of use of illicit drugs and mean DAST-10 scores than cigarette smoking or alcohol use.
Conclusions: The estimated national prevalence of cannabis use in the United States in NMURx data was greater than either cigarette smoking or alcohol consumption of greater than 7 drinks per week. Those who used cannabis had higher rates of opioid nonmedical use, illicit drug use, injection of prescription opioid pills, and DAST-10 scores than those who did not use cannabis. However, both tobacco and alcohol use were also associated with these risky substance use behaviors.

KEYWORDS Cannabis, Ethanol, Substance abuse

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Table 1 Behaviors associated with cannabis use, cigarette smoking, and alcohol use in the past year in the United States Survey of Non-medical Use of Prescription Drugs (NMURx), third quarter of 2018.

Behavior	Cannabis Users % (95% CI)	Cannabis Non-Users % (95% CI)	Cigarette Smokers % (95% CI)	Non-Smokers % (95% CI)	>7 Alcoholic Drinks / Wk % (95% CI)	≤7 Alcoholic Drinks /Wk % (95% CI)
Co-NMU with Opioids	14.9 (13.9, 15.9)	3.8 (3.5, 4.0)	17.0 (16.1, 17.9)	3.9 (3.7, 4.2)	11.4 (10.2, 12.5)	5.1 (4.8, 5.4)
Co-Use with Illicits (excluding cannabis)	19.0 (17.8, 20.1)	3.8 (3.5, 4.0)	16.5 (15.6, 17.4)	3.0 (2.8, 3.3)	12.1 (10.9, 13.3)	4.1 (3.8, 4.3)
Injection of pain reliever pills	1.6 (1.3, 1.9)	0.28 (0.21, 0.34)	2.8 (2.4, 3.1)	0.16 (0.10, 0.22)	1.9 (1.4, 2.3)	0.37 (0.30, 0.44)
DAST-10 Category						
None, 0	32.3 (29.9, 32.7)	70.4 (69.7, 71.0)	43.4 (42.2, 44.6)	66.6 (65.9, 67.3)	52.0 (50.1, 53.8)	64.7 (64.0, 65.3)
Low, 1-2	50.3 (48.8, 51.8)	27.4 (26.8, 28.0)	39.7 (38.5, 40.9)	30.1 (29.5, 30.8)	36.1 (34.3, 37.9)	31.0 (30.3, 31.6)
Moderate, 3-5	13.5 (12.5, 14.5)	1.7 (1.6, 1.9)	11.3 (10.5, 12.1)	2.6 (2.4, 2.9)	8.5 (7.5, 9.5)	3.3 (3.1, 3.5)
Substantial, 6-8	3.9 (3.4, 4.4)	0.41 (0.33, 0.49)	4.2 (3.7, 4.7)	0.52 (0.42, 0.63)	2.3 (1.8, 2.8)	0.90 (0.78, 1.01)
Severe, 9-10	1.1 (0.8, 1.4)	0.11 (0.06, 0.15)	1.4 (1.1, 1.7)	0.11 (0.06, 0.15)	1.1 (0.8, 1.5)	0.19 (0.14, 0.24)
Mean DAST Score	1.51 (1.46, 1.56)	0.40 (0.39, 0.41)	1.34 (1.29, 1.39)	0.48 (0.47, 0.50)	1.03 (0.97, 1.08)	0.55 (0.54, 0.57)

252. Pediatric Mercury Poisoning from Indirect Exposure to Skin Cream

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Background: Sub-acute or chronic non-organic mercury toxicity affects predominantly the gastrointestinal, renal and central nervous systems. Although rare, skin lightening cream remains one of the known sources of mercury exposure, and infants and children appear particularly vulnerable.
Case Report: A previously healthy ten month old male was admitted to hospital with a one month history of a progressive desquamative papular rash, anorexia, weight loss, hypertension and regression in neuro-motor milestones. He initially presented with cough, rhinorrhea and fevers and was treated empirically for acute otitis media. Prior to antibiotics, the patient's mother noticed a peeling rash on her son's hands in the inter-digital zones which progressed. On his third emergency room presentation he was admitted to hospital for advanced workup. Extensive infectious, autoimmune and metabolic workup were unremarkable. Neurological imaging and renal/cardiac examinations were also unremarkable. On day 38 from the rash onset, a whole blood mercury level was found to be elevated at 251 nmol/L. Subsequently, patient urine mercury levels were also elevated confirming the diagnosis on non-organic mercury toxicity. The patient was started on empiric succimer chelation therapy for three weeks with clinical improvement and declining mercury whole blood and urine levels. A detailed history revealed that patient's mother had recently bought a locally made skin cream for sun protection and collagen enhancement in Mexico. She had been using it topically on her face and neck twice daily for 6 weeks prior to onset of the patient's symptoms. The cream was tested to have extremely elevated levels of elemental mercury at 56 000 ppm. In Canada and the USA, the regulatory limit for cosmetic skin products is less than one ppm. A mercury sniffer in the patient's home indicated the washing machine had very high levels of mercury and was treated with a decontamination protocol. Their vacuum cleaner was identified as having high levels of mercury and disposed of.
Case Discussion: Cases of non-organic mercury toxicity have been linked to unregulated skin products marketed and informally sold as skin lighteners. The patient's mother and older brother (toddler) were asymptomatic and had slightly elevated whole blood mercury levels. Infants are vulnerable to inhalation routes drug toxicity due to increased minute ventilation to body area. Skin integrity as well as increased body surface area ratios increase risk of dermal exposures as well. For this patient, ongoing exposure was likely from multiple sources including exposures to vapourized mercury from the washer and vacuum as well as possible dermal and/or oral contact from patient's mother and contaminated household products.
Conclusion: A high degree of suspicion of mercury poisoning is required when a child is presenting with a variety of non-specific symptoms, including skin rash and hypertension. This case highlights a known source and route of sub-acute mercury exposure and toxicity, which resulted from a cosmetic product manufactured and purchased abroad. As mercury-based products are no longer available in households of Canada and USA, clinicians should be aware of this rare form of mercury exposure.

KEYWORDS Mercury, Pediatric, Skin Cream

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253. Methamphetamine Body Stuffing Complicated by Posterior Reversible Encephalopathy Syndrome (PRES) and Delayed Bowel Necrosis

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Background: Methamphetamine body stuffing has been associated with numerous complications, however Posterior Reversible Encephalopathy Syndrome (PRES) and delayed bowel necrosis are not commonly reported.
Case Report: 24 year old male brought to the Emergency Department with altered mental status after 36 hours of incarceration. Initial vital signs were: T 105.9 F (41.1 C), HR 169 bpm, BP 91/59, RR 45, Oxygen saturation 89%. Physical examination revealed an unresponsive patient with dilated pupils and increased lower extremity tone without clonus. Initial labwork showed bicarbonate 19 mmol/L, creatinine 2.02 mg/dL, lactate 13 mmol/L, CPK 6,196 U/L, troponin 3.32 ng/mL, AST 150 U/L, ALT 58 IU/L, INR 1.2 and urine drug screen positive for amphetamine and cannabinoids. EKG showed QRS 138 msec and QTc 520 msec. Abdominal imaging was negative for foreign bodies. Patient was emergently intubated and received fluid resuscitation, sodium bicarbonate, cooling, and midazolam infusion. After 6 hours, repeat vitals were: T 100.2 F, HR 123 bpm, and BP 87/42. Repeat labwork showed CPK 108,200 U/L, Troponin >200 ng/mL, creatinine 3.9, and AST 9000 ALT 3775, INR >11.5, Ptt >200. Norepinephrine was started for persistent

hypotension. Brain MRI on HD 4 revealed T2/FLAIR hyperintensity with restricted diffusion involving bilateral temporal, occipital, and cerebellar lobes, concerning for PRES. Patient was treated for sepsis, coagulopathy, and shock liver. On HD 22 patient passed bloody stool containing an apparently intact baggie. On HD 24 esophagogastroduodenoscopy revealed a baggie in stomach (images available) and colonoscopy showed ischemic bowel. Patient underwent resection of necrotic bowel and removal of intact baggie from stomach. Repeat MRI on HD 27 revealed expected evolution of PRES with significantly improved restricted diffusion foci. Patient was able to follow commands however remained critically ill due to septic shock complicated by abdominal wound dehiscence, intraabdominal abscesses, fulminant hepatitis, DVT, and ongoing coagulopathy. Goals of care were discussed with family and decision was made to not escalate care. Patient expired on HD 62. Confirmatory GC/MS on urine from admission showed amphetamine 3558 ng/ml and methamphetamine >10,000 ng/mL

Case Discussion: We present a patient with many complications of methamphetamine body stuffing, including hyperthermia, shock, DIC, rhabdomyolysis, renal and hepatic failure. Neuroimaging and clinical course supported the diagnosis of PRES which is a condition of reversible vasogenic subcortical edema generally associated with hypertension. Our patient was hypotensive at presentation but may have had undocumented hypertension previously. Additionally patient suffered delayed mesenteric ischemia requiring bowel resection. While this may have been a result of shock and pressor use, methamphetamine abuse may induce sympathomimetic mesenteric vasoconstriction as well as promote vasculitis. it is also possible that leakage of a ruptured packet resulted in local necrosis, as well as contributing to ongoing toxicity. It is unclear whether GI decontamination or earlier operative intervention would have altered the outcome.

Conclusion: We present a case of Posterior Reversible Encephalpathy Syndrome (PRES) and delayed bowel necrosis in a patient with severe methamphetamine toxicity. Clinicians should maintain vigilance for these atypical and delayed complications of methamphetamine body stuffing.

KEYWORDS Methamphetamine, Posterior Reversible Encephalopathy Syndrome (PRES), Bowel Necrosis

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254. Seizure Following Bupropion Insufflation: A National Poison Data System Study

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Background: Bupropion is the drug most frequently associated with seizure following oral overdose. This agent is increasingly abused by nasal insufflation, perhaps due to its amphetamine-like structure, but the risk of seizure following administration by this route is less well documented. A study of cases reported to the four California poison centers (PCs) found that 30% of patients (n = 67) had seizures but all occurred prehospital (Lewis, Clin Toxicol 2014; 52: 969-72). The aim of this study was to estimate the incidence of delayed seizures in a larger sample using the National Poison Data System (NPDS). A secondary goal was to assess the degree variability in PC recommendations following bupropion insufflation.

Methods: We requested NPDS data from the American Association of Poison Control Centers on all single exposure bupropion nasal insufflation cases from 1/1/2000-12/31/2017. There were 878 cases reported during this period, 404 with moderate or major (M/M) effects, and no fatalities. In order to assess timing of seizures and PC recommendations, de-identified case narratives were requested from all PCs that managed cases with M/M effects. Case narratives were then reviewed to determine timing of seizures if they occurred, PC recommendations, and disposition. Because all narratives were de-identified, our institutional review board deemed the study exempt.

Results: De-identified narratives from 243 cases involving 34 PCs were obtained and reviewed. The median age was 33 years (range 9-66) and 84% were male. 78/243 (32.1%) had seizures (66 single, 12 multiple). Most seizures were prehospital with only eight reported in hospital. Of these, five involved ingestion along with insufflation. The three remaining patients seized within two hours of emergency department presentation. PC recommendations included generalized supportive care and admission for altered mentation, but varied for those who were alert post seizure. The most common recommendation was for 23-24 hour observation (90/243 cases, 37.0%), though some recommended much shorter periods (4-6 h), or cleared patients for discharge when significant time had already passed since the seizure (e.g., seized 12 hours earlier but currently asymptomatic). The second most common recommendation was observation for seizures with no specific time frame.

Discussion: This study has a number of limitations. The pharmacokinetics of bupropion following nasal insufflation are not well studied. Furthermore, patients with bupropion insufflation are often involved in illicit substance abuse and may not always offer reliable histories. Providers may have concerns that patients are abusing other psychoactive substances or have also ingested bupropion (intentionally or inadvertently), possibly prompting some to recommend a longer observation period.

Conclusions: The incidence of seizures following bupropion nasal insufflation is comparable to that after ingestion, however onset of seizures is generally more rapid. Delayed seizures following insufflation are uncommon unless there is bupropion coingestion. Prolonged observation of an alert patient with normal vital signs appears to be unnecessary after bupropion insufflation..

KEYWORDS Bupropion, Insufflation abuse, Seizure

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255. Opioid-associated Sensorineural Hearing Loss Associated With Evidence Of Multiple System Organ Dysfunction After Overdose

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Background: Bilateral sensorineural hearing loss (SSNHL) is a rare but significant complication seen in both acute and chronic opioid misuse. Multiple case reports have proposed a variety of mechanisms underlying opioid-associated hearing loss. We present a case series of patients with acute opioid overdose presenting to the emergency department (ED) associated with unresponsiveness and hearing loss. Our hypothesis is that bilateral SSNL following an acute opioid overdose in ED patients is associated with other concomitant end organ dysfunction.

Case Series: This is a retrospective chart review of 6 cases presenting to the emergency department.

Case 1: 36-year-old man with a history of opioid dependence who presented with unresponsiveness after misusing oxycodone.

Case 2: 28-year-old man with a history of substance use disorder who presented with unresponsiveness and cyanosis after misusing alprazolam, marijuana, oxycodone, and buprenorphine.
Case 3: 45-year-old man with a history of opioid abuse who presented with unresponsiveness after IV heroin overdose.
Case 4: 35-year-old man with history of substance use disorder who presented with hearing loss after using cocaine, phencyclidine, alprazolam, and buprenorphine.
Case 5: 48-year-old man with a history of chronic pain who presented with unresponsiveness after ingesting 160 mg oxycodone.
Case 6: 28-year-old man with a history of substance use disorder who presented with unresponsiveness after insufflating one-bag of heroin and two-bags of cocaine with ethanol.
Lab abnormalities included ischemic hepatitis (range AST 981-2428 U/L, range ALT 762-3437 U/L, n = 3, 50%), acute kidney injury (range BUN 18-28 U/L, range creatinine 1.5-3.52 mg/dL, n = 5, 83%), elevated troponin-T (range 0.06-2.15 ng/mL, n = 6, 100%), and rhabdomyolysis (range CK 1397-39398 U/L, n = 5, 83%). All patients had resolution of their multiple-system organ dysfunction and hearing loss within 24-48 hours of initial presentation.
Case Discussion: Bilateral SSNHL following opioid use can develop over minutes, hours, or days. The cochlea is a highly metabolically active structure that is supplied by the labyrinthine artery, a terminal artery for cochlear blood supply. Thus, the cochlear cells are especially sensitive to reductions in blood flow and oxygen supply, such as in an acute opioid overdose with respiratory depression and hypotension. Furthermore, these patients were associated with lab abnormalities indicating ischemic injury. However, all patients recovered from their bilateral SSNHL without any permanent sequalae after resuscitation.
Conclusion: Opioid-associated hearing loss may be associated with concomitant end organ dysfunction following an acute opioid overdose in ED patients. We suspect vestibulocochlear ischemia is the most likely pathophysiology in this subset of patients.

KEYWORDS Opioid-induced hearing loss, opioids, overdose

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256. Assessment of weight-based dose resulting in toxicity after unintentional pediatric ingestions of amphetamines

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Background and Objectives: Pediatric amphetamine exposure can result in sympathetic overstimulation including agitation/inconsolability, hypertension, tachycardia, seizure, and death. There is currently minimal data to provide poison centers with a referral guideline for pediatric unintentional amphetamine ingestions. The objective of this study was to identify the weight-based amphetamine dose associated with symptom development or need for benzodiazepine treatment in order to identify patients requiring referral to a healthcare facility (HCF); secondary outcomes were to assess the effect of activated charcoal and formulation on incidence of symptoms.
Methods: This is a retrospective single poison center study from 1/1/2005 through 11/30/2018. We included single substance ingestions of pharmaceutical amphetamines (dextroamphetamine or mixed amphetamine salts) in children age 0-12 years followed to a known outcome. Poison center text field documentation was searched for signs and symptoms related to amphetamine toxicity and use of benzodiazepines. Case notes were reviewed for a priori designated signs and symptoms of amphetamine toxicity including agitation, age-specific tachycardia, age-specific hypertension, hyperthermia, and seizures along with use of benzodiazepines. Vital signs were only evaluated for patients seen in a healthcare facility. If no weight was documented in the chart, the 50thpercentile by age was used from the gender-specific CDC growth charts. Statistical analysis was performed using GraphPad Prism 8. Data are reported as median and interquartile ranges (IQR). The Institutional Review Board at our university approved the study.
Results: We screened 1,394 cases; 190 met inclusion criteria. The median age of patients was 2 years (IQR: 1.3-2.0) and 53% were male. Median duration of follow-up was 6.0 hours (IQR: 3.9-9.6) and was longer for symptomatic patients. Specific amphetamines involved included mixed amphetamine salts (n = 182), dextroamphetamine (n = 5), and racemic amphetamine (n = 3); 111 (58%) of the products were immediate release formulation with the rest extended release. Symptoms developed in 77/190 patients (40.5%) with the most frequently reported symptoms including agitation/inconsolability (n = 70/190) and tachycardia (n = 42/98). Two patients experienced seizures. The median amphetamine dose in symptomatic patients was 1.5 mg/kg vs 0.9 mg/kg (p
Conclusions: The median dose of amphetamines ingested by patients receiving benzodiazepines was higher than those not receiving benzodiazepines. We recommend referral of any child with an unintentional acute amphetamine ingestion of greater than or equal to 1 mg/kg to a health care facility for monitoring and potential treatment. Further research is needed to prospectively validate these findings in a multicenter study.

KEYWORDS Pediatric, amphetamine, unintentional

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Table 1 Baseline characteristics, monitoring, and decontamination for pediatric amphetamine ingestions.

	All	Symptomatic	Asymptomatic	Median Difference or OR	95% CI
	n = 190	n = 77	n = 113		
Age, years, median (IQR)	2.0 (1.3, 2.0)	2.0 (1.2, 2.0)	2.0 (1.4, 2.0)	0.0	-0.1, 0.3
Weight, kg, median (IQR)[a]	12.0 (10.5, 15.0)	11.9 (10.0, 14.8)	12.2 (10.9, 15.1)	0.25	-0.9, 1.3
Male, n (%)	129 (53.3)	68 (61.8)	61 (46.2)	1.88	1.1, 3.2[c]
Duration of follow-up, hours, median (IQR)	6.0 (3.9, 9.6)	9.6 (6.5, 21.2)	4.5 (3.2, 6.5)	-5.0	-7.6, -4.0[c]
Activated charcoal, n (%)[b]	47 (25.3)	19 (25.0)	28 (25.5)	1.0	0.5, 1.9
Formulation, extended release, n (%)	79 (41.5)	38 (48.1)	39 (35.1)	1.71	0.95, 3.08

CI, confidence interval; IQR, interquartile range; kg, kilograms; OR, odds ratio.
[a]Actual weight available for 151 patients.
[b]Charcoal administration determined in 186 cases.
[c]$P < 0.05$.

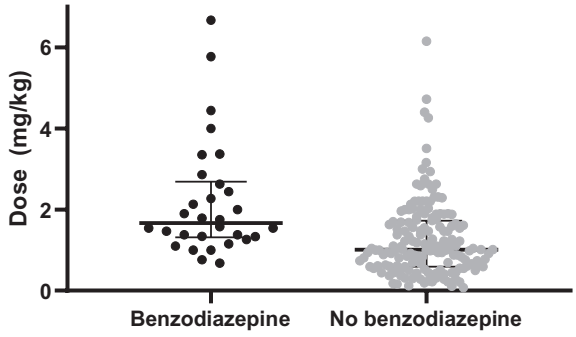

Figure 1 Median and interquartile range (IQR) of dose for patients receiving (1.7 mg/kg; IQR: 1.3, 2.7) or not receiving (1.0 mg/kg; IQR: 0.6, 1.7) benzodiazepines.

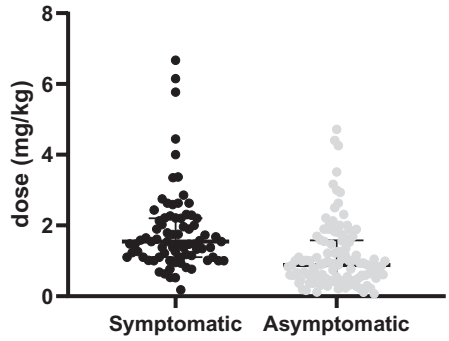

Figure 2 Median and interquartile range (IQR) of dose for symptomatic (1.5 mg/kg; IQR: 1.1, 2.2) or asymptomatic patients (0.88 mg/kg; IQR: 0.5, 1.6).

257. Analysis of initial management of unintentional pediatric sulfonylurea exposures reported to two poison centers

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Background/Objectives: Unintentional ingestion of sulfonylureas by children can cause severe and refractory hypoglycemia, leading to seizures, coma, and death. Food is recommended as the initial management for unintentional pediatric sulfonylurea exposures in asymptomatic patients. Despite this recommendations, initial management varies significantly from liberal administration of food to strict NPO with intravenous dextrose. The objective of this study is to characterize the initial and subsequent management of unintentional pediatric sulfonylurea ingestions reported to two regional poison centers.
Methods: This is a retrospective study of single-substance unintentional pediatric sulfonylurea exposures reported to two regional poison centers from 1/1/2000 through 3/31/2018 for patients
Results: A total of 337 cases were included. Median age was 2 (1.6-2.0) years and 52% of the patients were male. The majority of patients (88%; 295/337) were initially managed appropriately. The most common initial management was food (48%) followed by observation alone (27%), while 54 patients (16%) received IV dextrose alone as initial therapy. The majority of patients (89%) were documented as "awake and alert" at initial presentation and the median initial blood glucose was 86 mg/dL (IQR: 67-100). 130 patients (40%) received activated charcoal. The incidence of hypoglycemia was 30% (102). Both appropriate initial management (OR: 0.426, 95%CI: 0.218-0.832) and activated charcoal (OR: 0.455, 95%CI: 0.272-0.761) were associated with decreased risk of hypoglycemia. Disposition included: 203(60%) were treated in the emergency department, 109 (32%) were admitted to the floor, and 25 (7%) were admitted to the ICU. Subsequent care consisted of primarily observation and food, with over a quarter of patients receiving a dextrose infusion. The median concentration of dextrose infusions was 5% with a median rate of 45 mL/hr.
Conclusion: The majority of patients received appropriate initial management for unintentional sulfonylurea ingestion. Both activated charcoal and appropriate initial management were associated with a reduced risk of hypoglycemia.

KEYWORDS Management, unintentional, sulfonylurea

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258. Treatment of Congenital Lead Poisoning with Succimer

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Background: Pediatric lead poisoning is a well-recognized environmental exposure, known to cause detrimental neurodevelopmental effects. However, congenital lead exposure is rare and little understood. Additionally, the safety and efficacy of neonatal chelation is not well established. We present a unique case of intrauterine lead exposure associated with maternal pica and describe the use of oral succimer (dimercaptosuccinic acid, DMSA) in the neonate.
Case Report: A 26-year-old G6P0141 woman transitioned care to our institution at 27 weeks pregnancy. She reported cravings for dirt consistent with pica, and lab work demonstrated iron deficiency anemia. This anemia proved to be refractory to adequate iron supplementation. The maternal blood lead level was checked at 35 weeks and found to be 38.7 µg/dL. A female infant was delivered at 37 weeks gestation via scheduled induction due to gestational hypertension. Cord blood lead level at delivery was 54 µg/dL. The infant's exam was normal except for mild, brief respiratory distress; no hepatomegaly was observed, and neurologic exam was normal including newborn reflexes, activity, and tone. Her hemoglobin and hematocrit were normal at 16.2 µg/dL and 45.9%, respectively. A capillary lead level, drawn on day of life 2 and pending at time of discharge, resulted at 57.7 µg/dL on her sixth day of life. The infant was admitted for initiation of chelation therapy. She received high dose DMSA at 350 mg/m2 every 8 hours for the first 5 days, followed by 350 mg/m2 every 12 hours for an additional 14 days. She was exclusively formula fed to prevent further lead transmission via breast milk. Her renal and hepatic function remained normal. Skeletal survey revealed no lead lines. Repeat capillary lead level on day 5 of chelation was 23.6 µg/dL. The infant was discharged to a lead-free home to complete chelation therapy with close lead clinic follow up. On day 19 of chelation and on follow up day 59, her lead level stabilized to 28 µg/dL.
Case Discussion: Data is limited on the use of chelation therapy to treat lead poisoning in pregnant women and neonates. Lead crosses the placenta by simple diffusion; neonatal levels often approximate or exceed maternal levels. Chelation during pregnancy is reserved for severe symptomatic maternal lead poisoning because mobilization of maternal lead stores may increase lead distribution to the developing fetus and elimination of essential minerals by chelation may be teratogenic. Chelation therapy is generally reserved for neonates with blood lead levels >45 µg/dL. Although the newborn exam is typically normal at birth, congenital lead exposure continues to be associated with impaired cognitive development in children. While chelation is effective in lowering blood lead levels in infants and toddlers, studies

have unfortunately failed to demonstrate an associated improvement in longterm cognitive function.
Conclusions: We describe a case of neonatal lead poisoning treated with DMSA. Despite initial downtrending blood lead levels, the infant's blood lead level stabilized well above the recommended level. Long term follow up will be crucial to ensure the child reaches her full neurocognitive potential.

KEYWORDS Lead, Succimer, Pediatric

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259. Insurance Reimbursement of Emergency Department Dispensed Naloxone Kits.

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Currently, 130 people in the United States die every day from an opioid overdose (OD). Increased bystander naloxone availability is a tool to decrease OD deaths. Naloxone, a μ-receptor antagonist, reverses respiratory depression caused by opioids, but must be administered quickly making its community availability paramount to its effectiveness. An optimal time to provide at-risk patients with naloxone includes on patient discharge from the emergency department (ED). Historically, grant funds supported ED naloxone distribution; however, these funds are becoming limited. We report a novel pathway is to dispense the naloxone from the ED with retrospective insurance reimbursement submission.
Methods: Included patients received a naloxone kit from 9/1/2018–2/28/2019 on ED discharge at a level I urban, trauma center with around-the-clock ED pharmacist services. Upon receipt of the discharge prescription, the ED pharmacist prepares and delivers the naloxone kit and counsels the patient. The prescriptions were delivered to the affiliated outpatient pharmacy during business hours. Before September 1, kits were grant funded. The primary outcome was the percent of prescriptions covered by insurance. Secondary outcomes include reasons for lack of coverage and ED disposition. Basic demographic information included chief complaint, age, gender, reported agent of abuse, and insurance type. Pre- and inhospital naloxone use and associated withdrawal symptoms were also characterized. Naloxone associated withdrawal was defined as nausea, vomiting, yawning, piloerection, "dope sickness", or antiemetic use proximal to naloxone administration. Descriptive statistics were used.
Results: Naloxone kits were dispensed to 61 patients; 90% were covered by insurance. Patients were primarily male (48 (79%)), 40.6 ± 11.4 years old, and 23% had private insurance. The chief complaint included primarily accidental OD (45 (74%)). Recreational use of an opioid was reported most often (46 (61%)) followed by cocaine (10 (13%)). The intended substance was unknown in 21% of patients. Naloxone administration occurred in 48 patients (79%): 39 prehospital and 9 inhospital. Opioid withdrawal occurred in 22 patients (36%) following naloxone administration. Lack of reimbursement was due to no insurance (5 (83%)) and refill too soon (1 (17%)). Patients were discharged (43(71%)), left against medical advice (4(6%)), or went directly to detox programs (14(24%)).
Conclusions: Reimbursement occurred in most patients, which translated to more than $4000 total savings. Dispensing naloxone kits from the ED may help to prevent gaps in availability. Finally, this pathway allows for the correct appropriation of pharmaceuticals within state and federal regulations. This process does not directly address specific dispensing regulations such as prescription labeling or counseling. This ED has around-the-clock clinical pharmacist presence which allowed for these needs to be met. This process is labor intensive as it requires delivery of the original prescription and entry in the outpatient pharmacy system. Limitations of this study include the small sample size and a minority of included patients had private insurance which, along with state law variability, may impact widespread applicability. Retrospective claim submission may decrease the financial impact of ED dispensing of naloxone kits while facilitating patient needs.

KEYWORDS Naloxone, opioid overdose, emergency department

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260. Cocaethylene Associated with Cardiac Arrest in Emergency Department Patients with Acute Drug Overdose

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Objectives: Cocaine use results in over 500,000 emergency department (ED) visits annually across the U.S. annually and ethanol co-ingestion is reported in 34% of these. Combining cocaine use with ethanol results in the metabolite cocaethylene (CE), which is metabolically active for longer (>120 minutes) compared to cocaine alone (45-90 minutes). However, studies have conflicting results about the cardio- and neuro-toxicity of CE compared to cocaine alone. We aimed to clarify this conflict by observing the total number of cardiovascular events in cocaine versus cocaethylene overdose in two urban EDs. We hypothesized that cocaethylene would be more cardiotoxic and would lead to an increased number of adverse cardiovascular events (ACVE) compared to cocaine alone.
Methods: This was a secondary data analysis of a prospective cohort study at two urban tertiary care hospitals over four years. Patients in the screened cohort were adult (>18) ED patients with suspected acute drug overdose. For the secondary data analysis, cocaine exposure was defined by patients in the cohort with positive cocaine urine drug screens. The CE subgroup was defined by patients in the cohort with confirmed cocaine exposure in addition to confirmed serum ethanol concentrations by quantitative serum measurement. The primary outcome was in-hospital cardiac arrest, defined by loss of pulses requiring CPR. Secondary outcomes included myocardial injury (defined by mean cardiac troponin I levels) and hyperlactatemia (defined by mean lactate levels). Data was collected by trained research assistants and was analyzed using descriptive and bivariate statistics using SPSS v24. Based on a fixed sample size of 200 patients, assuming 3:1 ratio of cocaine-to-CE patients, we had 80% power to demonstrate 5-fold higher odds of the primary outcome in the CE group.
Results: During the study period, there were 199 eligible cocaine patients (72 female, 127 male) ages 18-75 (39 Caucasian, 45 Hispanic, 6 Asian, 109 Other/Unknown) in the cohort (150 cocaine, 49 CE). Myocardial injury was significantly associated with the cocaine group (mean initial troponin 0.16 vs. 0.01 ng/mL, $p = 0.021$), while hyperlactatemia was associated with the CE group (mean initial lactate 4.1 vs. 2.9 mmol/L, $p = 0.038$). Proportion of cardiac arrest was significantly higher in the CE group compared to cocaine patients (4.1% vs. 0.67%, $p = 0.048$).
Conclusions: In ED patients with acute drug overdose, CE was associated with cardiac arrest compared to cocaine alone. CE was also associated with hyperlactatemia, while cocaine alone was associated with myocardial injury. These results may be explained by benzoylecgnonine-induced vasospasm in the cocaine-only group, and CE-induced dysrhythmias. Further studies are needed to clarify the exact mechanism by which CE may increase risk of cardiac arrest and hyperlactatemia.

KEYWORDS Cocaethylene, cardiac, cocaine

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261. The Poisoned Lollipop: Tetrahydrocannabinol Toxicity in a 16-Month-Old Child

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Background: Acute cannabis ingestion in toddlers and infants is a rare but growing problem that can be potentially life threatening. We present a case of a 16-month-old who ingested a lollipop containing tetrahydrocannabinol (THC) which highlights the difficulties inherent in making the diagnosis as well as the potential life-threatening effects.
Case Report: A 16-month-old boy was brought to the emergency department (ED) by his mother with excessive somnolence and apneic spells. The patient had a 3-day history of cough, nasal congestion and tactile fever but had otherwise been well and continued to eat and drink. They were out at a grocery store when quite suddenly he became sleepy, difficult to wake and had 2-3 episodes of apnea with cyanosis around the lips. On arrival to the ED he had oxygen saturations of 67% on pulse oximetry and had poor respiratory effort. With some stimulation he started to cry and was given high flow oxygen via a non-rebreather mask. Physical examination revealed a somnolent, well developed child with no external signs of trauma. He had a tachycardia of 164 beats per minute and was afebrile. His pupils were equal and briskly reactive to light. He did not exhibit any movement concerning for seizure and had normal reflexes. His neck was supple. The rest of his examination was normal. IV access was established, and lab work was sent. A urine drug screen returned positive for cannabinoids. Both parents denied having marijuana or other recreational drugs in the home. On further questioning it came to light that a three-year-old half-sibling was visiting earlier that day. He was licking a lollipop, which he shared with the patient shortly before his change in mental status. A phone call was made which revealed that the half-sibling in question also appeared more sleepy than usual to his caretakers at home. It was deduced that the lollipop in question contained THC. The patient was admitted to the hospital for observation and discharged the next day after making a full recovery.
Discussion: This case illustrates the potential danger associated with recreational edible marijuana use. Systemic THC levels and psychoactive effects after ingestion are highly variable because of differences in bioavailability, rate of gastrointestinal absorption, and metabolic first-pass effect whereby an orally administered drug is partially metabolized (principally in the liver) before reaching systemic distribution. Clinical signs are variable and depend on both the absorbed quantity and duration of exposure. Management of patients intoxicated by cannabinoids is supportive with careful attention to the airway and breathing in children.
Conclusions: This case highlights the growing number of young children exposed to cannabis with the advent of legalization. A high index of suspicion, thorough history taking, and early urine drug testing are essential to identifying the diagnosis and preventing costly and invasive testing such as head CT and lumbar puncture. Once the diagnosis is made symptoms usually resolve within 24 hours with supportive management.

KEYWORDS Tetrahydrocannabinol, Lollipop, cannabis

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262. Vilazodone Induced Status Epilepticus in a Pediatric Patient, Confirmed with Drug Levels

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Background: Cases of vilazodone associated pediatric seizures were first reported in 2012 and there is mounting evidence that suggests that it is more toxic than other serotonin reuptake inhibitors. We report a case of pediatric status epilepticus that developed after vilazodone ingestion, confirmed with drug levels.
Case Report: A 2-year-old girl (11kg) was brought to an emergency department at 11pm by her parents for restlessness, progressive unresponsiveness and shaking movements that developed over the hour prior to presentation. The patient was last seen normal at bedtime, a few hours prior. She had displayed upper respiratory tract infection symptoms in the past week. Triage vital signs were: blood pressure 117/82 mmHg; heart rate 146/minute; respiratory rate 32/minute; Sp02 sat 98% on room air; temperature 36.9 °C. Physical examination was notable for dilated pupils, normal muscle tone, and a Glasgow Coma Scale of 5. Over the first two hours, the patient had two episodes of generalized body shaking without return to mental baseline, persistent disorganized myoclonus, and localization to painful stimulus only. Initial laboratories displayed a salicylate level of 1.8 mg/dl, an undetectable acetaminophen level, a negative urine drug screen, and were otherwise unremarkable. Blood and urine cultures were sent and were ultimately negative. An electrocardiogram displayed sinus tachycardia, a non-specific T wave abnormality, and normal intervals. Due to concern for persistent seizure activity requiring multiple anticonvulsants, the patient was intubated, placed on a continuous midazolam infusion, and transferred to a tertiary care facility pediatric intensive care unit (PICU). There was marijuana in the house, but no other recreational drugs. Prescription medications in the home included trazodone, prednisone, meloxicam, citalopram, vilazodone, acetaminophen/hydrocodone, diphenhydramine, and cetirizine with no known exposure. In the PICU, a lumbar puncture revealed one white blood cell and no organisms from culture. A 4-hour continuous EEG showed no epileptiform activity and a brain MRI was unremarkable. Seventeen hours after presentation she was extubated, displayed purposeful movements and was interactive with her mother, but still disoriented. Thirty six hours after presentation she returned to her mental baseline but remained ataxic. After consultation with the Poison Control Center, the most likely causative agent was felt to be vilazodone. Bloodwork[MK1] was sent from blood obtained 12 hours after presentation and returned with a Vilazodone level of 140 ng/ml.
Case discussion: Pediatric status epilepticus is a life-threatening condition that has both medical and toxicologic etiologies, some requiring specific lifesaving interventions. Determination of changing trends in drug and medication related seizures and status epilepticus is strengthened by reliable case reporting. There is increasing evidence that suggests that small pediatric vilazodone exposures are associated with severe toxicity and seizures. Our case confirms vilazodone ingestion in the context of pediatric status epilepticus and no other identifiable cause.
Conclusion: Our case report suggests that vilazodone ingestion is a potential cause of drug induced seizures and status epilepticus in children. Providers should be aware of this medication as a significant hazard for pediatric patients.

KEYWORDS Vilazodone, pediatric, seizure

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263. Outcomes of unintentional copper sulfate ingestions in children: A report of two cases and review of local poison center data

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Background: Copper sulfate (CuSO4) has been reported to cause significant toxicity in children with accidental ingestion. Case reports detail gastrointestinal symptoms, caustic burns, hemolytic anemia, and other significant organ injuries. However, it's not known how often exploratory ingestions lead to systemic symptoms. We present two cases that illustrated this uncertainty and a review of local poison center data to examine that question.
Case Report: A sibling pair ingested a packet of Magical Flames powder, assuming it was candy. Initial reported estimate was that over ½ the packet was shared between the 3-year-old girl and the 4-year-old boy. Both were referred to the emergency department. The girl had emesis and the boy had abdominal pain. Vital signs were normal. They were intubated to facilitate gastric lavage. The girl's aspirate was blue/green then blood-tinged; his was clear. After transport to a pediatric ICU, the estimated dose ingested was revised to no more than a mouthful each. Endoscopy was completed prior to extubation and both had grade 1 gastric mucosal injury. The girl's serum copper concentration was 1.00 mcg/mL (ref 0.8-1.8). She had a hemoglobin drop from 14.7 mg/dL to 10.4 that stabilized around 11.6 mg/dL and was associated with a low haptoglobin. Her brother had normal serial hemoglobins but elevated creatine phosphokinase at 355 U/L (ref 21-232) which improved during admission. His copper concentration was low at 0.62 mcg/mL. They both received IV N-acetylcysteine, IV fluids, and proton pump inhibitors and discharged to home
Case Discussion: These children had GI injury following low-dose exposure to copper sulfate and clinically insignificant systemic symptoms, raising the question of how often systemic toxicity occurs following accidental exposures in pediatric patients. The local toxicall database was queried for children 6 years of age, with exposure to copper sulfate (generic substance code 0117000 and 0201033), and reason for exposure "unintentional-general" between January 2000 and April 2019. 108 cases were identified; 26 of these were excluded for non-sulfate salt exposures and nine excluded since route did not include ingestion. Case details including age, sex, and outcome were extracted. A total of 73 cases of copper sulfate exposure were analyzed. The average age was 2.6 years (range 10 months - 6 years). 29 patients (39.7%) were not followed after the initial call due to expectation of no or minimal toxicity; there was one patient who was unable to be followed despite recommendation for health care referral. Of the remaining patients, 13 (17.8%) had no effect, and 28 (38.4%) had minor effect. Effects documented were gastrointestinal, with no reports of hemolytic anemia or organ injury. There were two major outcomes (2.7%), which are the cases detailed above. There were no moderate outcomes or deaths noted. Of the 73 ingestions; 8 (11%) had concurrent dermal exposures and 3 (4.1%) concurrent ocular exposures.
Conclusion: Local poison center data suggests that systemic toxicity in unintentional copper sulfate ingestions in children is rare.

KEYWORDS Pediatric, Copper, Exploratory ingestion

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264. Take home naloxone in opioid overdose survivors

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Background: On an average day, 130 Americans die from opioid overdose. Naloxone, an opioid receptor antagonist, is indicated for the emergency treatment of known or suspected opioid overdose. Take home naloxone (THN) programs have reduced mortality in opioid overdose survivors, and the US Centers for Disease Control suggest providers consider offering naloxone to patients with, or with a history of, overdose. The objective of this study was to determine the rate and characteristics of THN prescribing and dispensing following emergency department (ED) or inpatient discharge for opioid overdose.
Methods: We studied opioid overdose patients treated in a Midwestern tertiary care center. THN was ordered as a prescription (Narcan NS®) or provided as a free kit (three vials of naloxone with syringes) from the ED or pharmacy. Exclusion criteria included age 2. Study approval was obtained by the institution's human subjects review board.
Results: In total, 554 patients met inclusion criteria. THN was prescribed to 20.9% (116/554) of patients after opioid overdose. THN was dispensed to approximately half of those patients (62/116, 53.4%). The most commonly documented reason naloxone was not dispensed was lack of prescription pick up at the pharmacy (26/54, 48/1%). Additional reasons for not dispensing naloxone included patient refusal, elopement or discharge against medical advice, and lack of medication delivery to the patient's room. The reasons for lack of naloxone dispensing were not documented in 17 patients (31.5%). Most patients to whom THN was dispensed paid $0 (median $0. IQR $0-$0). ED patients were less likely to be prescribed naloxone than patients admitted to the hospital (63/435 vs. 53/119, χ2 50.99, p2 0.12, p = 0.731).
Conclusion: Prescribing and dispensing of THN to ED and hospitalized opioid overdose patients vary by the origin of the exposure (prescribed vs. illicit opioid) and treatment setting, with differences noted even within a unified healthcare system. Our results suggest improvement opportunities in prescribing and invite further investigation into barriers in retrieving prescribed THN. Identifying differences in ED and inpatient populations may further clarify strategies to optimize naloxone dispensation after hospitalization for opioid overdose. A collaborative practice protocol exists for community and clinic pharmacists to dispense THN and encourages pharmacists to identify patients who may benefit from THN. Expanded efforts in the ED directed at opioid

Table 1.

	Emergency Admissionsn = 435	Inpatient Admissionsn = 119	X^2, p-value	Illicit Drug Overdosen = 476	Prescription Drug Overdosen = 56	X^2, p-value
Naloxone prescribed						
Yes	63 (14.5%)	53 (44.5%)	50.99, < 0.0001	101 (21.2%)	13 (23.2%)	0.12, 0.731
No	372 (85.5%)	66 (55.5%)	-	375 (78.8%)	43 (76.8%)	-
Naloxone dispensed						
Yes	26/63 (41.3%)	36/53 (67.9%)	55.40, <0.0001	50/101 (49.5%)	11/13 (84.6%)	4.12, 0.042
No	37/63 (58.7%)	17/53 (32.1%)	-	51/101 (50.5%)	2/13 (15.4%)	-

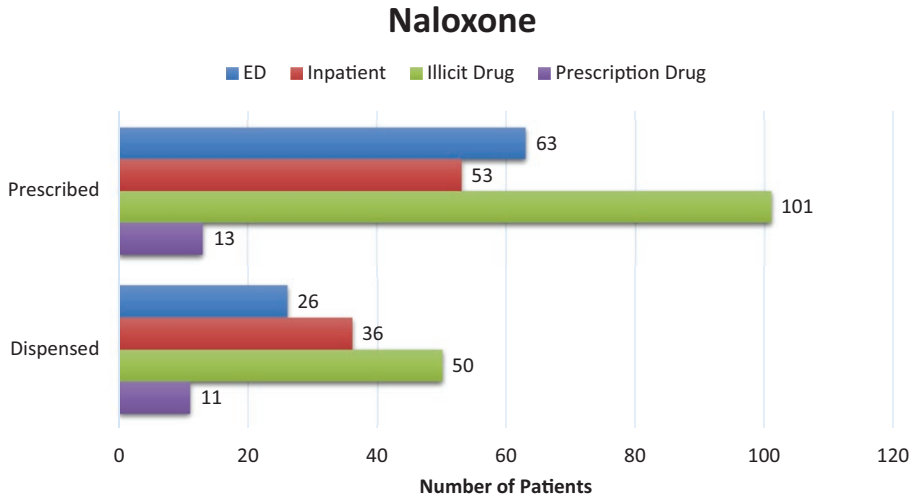

Figure 1.

overdose patients who both present to and are discharged directly from the ED may represent a point to maximize leverage to increase naloxone dispensation following opioid overdose.

KEYWORDS Opioid, Overdose, Naloxone

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265. Pediatric brexpiprazole toxicity with confirmatory testing

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Background: Brexpiprazole is a second generation antipsychotic approved in 2015 in the United States to treat schizophrenia and as an adjunctive therapy for the treatment of major depressive disorder. To our knowledge, there are no reports of pediatric overdose of brexpiprazole. We report a case of a 16-month-old male who became lethargic and hypoglycemic following ingestion of brexpiprazole.
Case report: A healthy 16-month-old male presented to the emergency department with drowsiness and ataxia which had developed over the last 18 hours. Parents reported 10 tablets of brexpiprazole 3 mg were missing, but the patient was not seen ingesting them. Other medications in the home were trazodone and clomipramine but no tablets were missing. Vitals were weight 11 kg, HR 152 bpm, RR 24, and SpO2 99% on room air. EKG was normal. Blood glucose was 33 mg/dL. The patient was treated with dextrose with no further episodes of hypoglycemia. About 24 hours after exposure, the child developed tremor, myoclonic jerks, and agitation which persisted over the next 24 hours. The abnormal movements had ceased by the time of transfer to facility with EEG monitoring. Lorazepam was administered to control agitation. The patient returned to his baseline about 72 hours post-ingestion and was discharged to home. Serum brexpiprazole concentration was 31 ng/mL approximately 48 hours after exposure. An expanded drug screen for >250 common pharmaceuticals, including hypoglycemic agents, was negative except for lorazepam at 12 ng/mL.
Case discussion: Brexpiprazole is an antipsychotic that acts as a partial agonist at 5-HT1a and D2 receptors with antagonist activity at 5-HT2a, alpha1, alpha2, and H1. Peak concentrations are achieved within 4-6 hours with a half-life of 91 hours. Hypoglycemia occurs rarely with antipsychotics but no other hypoglycemic agents were detected in this case. Hypoglycemia has been reported with risperidone, quetiapine, olanzapine, and aripiprazole. The mechanism is not known but may involve increased insulin release via pancreatic alpha2 antagonism. The etiology of the child's abnormal movements is not clear. They did not appear to be sustained, dystonic muscle contractions. No generalized seizure activity was seen. Aripiprazole, with similar properties to brexpiprazole, was more commonly associated with tremor than other agents in a large series of pediatric atypical antipsychotic exposures. Exposure to brexpiprazole was confirmed at 31 ng/mL. Peak brexpiprazole concentrations in adults after a single dose of 1 mg, 4mg, or 6mg were 10.2 ng/mL, 37.0 ng/mL and 69.9 ng/mL respectively at 4-6 hours. Initial adult dosing is 0.5-1 mg daily. Though extrapolation from these data is difficult, this child may have only ingested a single tablet to reach such a concentration at 48 hours.
Conclusion: We report a case of pediatric brexpiprazole toxicity with confirmatory testing and exclusion of other agents. The patient experienced typical symptoms of atypical antipsychotic toxicity with lethargy and ataxia and less common effects of tremor, myoclonic jerks, and hypoglycemia. The prolonged duration of effects may be due to the long half-life of brexpiprazole. Ingestion of a single 3 mg tablet may have been sufficient to cause these effects.

KEYWORDS Brexpiprazole, atypical antipsychotic, pediatric overdose

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266. Tianeptine: A Review of Cases Reported to One US Regional Poison Center

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Background: Tianeptine, an atypical antidepressant, has a tricyclic antidepressant-like structure but effects the mu and delta opioid receptors. At prescribed doses (12.5 mg three times daily), tianeptine improves depressive symptoms and anxiety; however, at higher doses (hundreds of milligrams to grams), it has more opioid like effects. Tianeptine is approved for use in Europe, Asia and Latin America but not approved by the Food and Drug Administration for use in the United States. Often sold as a "research chemical", tianeptine is readily available for purchase as capsules or powder in common online marketplaces, making it easily accessible for consumers self-medicating for depression or opioid dependency. US poison centers have seen an

increased number of calls regarding tianeptine over the last four years. In this study, we evaluated our regional poison center experience with tianeptine exposures between 2008 and 2018.
Methods: In this retrospective study of reported tianeptine exposures between January 1, 2008 and December 31, 2018, the database of one regional U.S. poison center was queried by identifying all reported tianeptine human exposures during this period. Once cases were identified, poison center records were reviewed to confirm accuracy of reported clinical effects, reason for exposure, reason for contacting the poison center, treatments performed, and medical outcomes. Medical outcomes were based on American Association of Poison Control Centers definitions.
Results: In the last 10 years, 13 cases of tianeptine exposures were reported to this poison center. All cases reported to this regional poison center occurred between 2015 and 2018. Most exposures were chronic (n = 10) and related to ingestions (n = 9), one patient reported snorting tianeptine and another reported vaping the drug. Six cases had reason for tianeptine use documented: antidepressant (n = 2), pain (n = 2), weight loss (n = 1), "to feel better" (n = 1). Eight cases involved tianeptine only with most calls being related to withdrawal symptoms (n = 5). Withdrawal onset was reported within 6 hours of drug discontinuation by two patients. Withdrawal symptoms reported by patients included anxiety, insomnia and pain. Providers noted agitation, anxiety, tachycardia, and hypertension in this group. Lorazepam was used most frequently to help with clinical symptoms of withdrawal. Only one case was reported of acute intoxication from tianeptine alone, in which the patient was obtunded and required intubation. Medical outcomes were "moderate" in 53% of cases. All other cases of overdose/intoxication included exposure to multiple drugs including tianeptine.
Conclusions: In early 2018, the CDC described an increased reporting of tianeptine exposures to the US National Poison Database System between 2000 and 2017. They described 218 total calls, 114 cases involving tianeptine only, and 29 tianeptine withdrawal associated calls. We have seen a similar increase in tianeptine exposure related calls in the last three years and were able to better characterize them through our record review. The majority of calls to our regional poison center regarding tianeptine were related to management of withdrawal symptoms. It is important for providers to be aware of the availability and misuse of tianeptine, as well as the clinical effects noted in both withdrawal and overdose.

KEYWORDS Tianeptine, withdrawal, misuse

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267. Loperamide Cardiotoxicity Treated with Isoproterenol

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Background: Loperamide, a common antidiarrheal agent, is frequently taken in excessive doses by individuals using it as an opioid substitute. Several cases of cardiotoxicity and death from loperamide misuse have been reported in the literature. The primary mechanism of action for cardiotoxicity is thought to be related to sodium channel blockade and hERG channel inhibition. We report a case of loperamide cardiotoxicity treated successfully with isoproterenol.
Case Report: A 26-year-old female with a remote history of methadone use for opioid use disorder (OUD) and excessive loperamide consumption (80-120 mg/day) collapsed in front of her family. During EMS transport she developed a wide complex dysrhythmia concerning for torsade de pointes and cardiac arrest. The patient was initially resuscitated with CPR. Her initial potassium was 4.2mmol/L , magnesium was 2.2mg/dL, and ionized calcium was 1.07mmol/L. Due to continued episodes of tachydysrhythmias and cardiac arrest, calcium chloride, magnesium, sodium bicarbonate, and amiodarone were given in the ED. She was intubated, sedated with propofol, and therapeutic hypothermia was initiated. A dopamine infusion was started for bradycardia and hypotension without effect. Due to persistent bradycardia (40-50 bpm) and prolonged QTc of 600-652 msec, both the amiodarone infusion and therapeutic hypothermia were discontinued. An isoproterenol infusion was initiated with improvement in her heart rate and control of the dysrhythmia. On hospital day (HD) 2, she developed recurrent periodic dysrhythmias and a wide complex polymorphic ventricular tachycardia with an undulating appearance after her isoproterenol infusion was decreased from 3 to 1 mcg/min. This dysrhythmia abated after increasing the isoproterenol infusion rate to 4 mcg/min. The isoproterenol was discontinued on HD 3 and the patient remained stable. Blood and plasma loperamide levels obtained on HD 2 were 48ng/mL and 64ng/mL respectively with desmethylloperamide levels of 390ng/mL (blood) and 220ng/mL (plasma). The patient was started on buprenorphine for opioid withdrawal symptoms. On HD7 her QTc interval improved to 483 msec, and she was discharged on HD 9. Eight months following this episode, the patient continued on buprenorphine and her QTc was 435 msec.
Case Discussion: A review of the literature demonstrates no standard recommendations for treating loperamide-associated dysrhythmias. Authors typically recommend electrolyte correction, standard ACLS protocols, use of sodium bicarbonate for QRS prolongation and consideration of lipid emulsion therapy or ECMO for intractable dysrhythmias. However, several case reports document successful use of isoproterenol to treat and prevent bradycardia-induced dysrhythmias. Wu et al reviewed 22 cases reports of loperamide toxicity and 11 of 17 cases that survived received isoproterenol as either first line or rescue therapies after failure of agents like lidocaine and amiodarone. In several of these cases, sodium bicarbonate temporarily decreased the QRS interval but the duration of cardiotoxicity from loperamide lasted several days. Our case adds to this body of literature.
Conclusions: Isoproterenol should be considered a first line therapy along with electrolyte repletion and cardioversion in the treatment of patients with loperamide induced cardiotoxicity. Additionally, providers need to know the consequences of loperamide misuse and educate patients with an OUD about risks of abusing this medication.

KEYWORDS Loperamide, abuse, pulseless idoventricular rhythm

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268. Paradichlorobenzene Toxicity Secondary to Chronic Mothball Ingestion In Pregnancy, A Case Report

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Introduction: Chronic paradichlorobenzene (1,4-dichlorobenzene; DCB) toxicity is associated with neuropsychiatric complications. Although DCB toxicity has been previously reported, to our knowledge, this is the second reported case of human toxicity in pregnancy.
Case: A twenty-eight-year-old female with schizoaffective disorder and chronic ingestion of paradichlorobenzene-containing mothballs presented to the hospital in labor at thirty-six weeks gestation. She exhibited anorexia, tremor, ataxia, dysmetria, ichthyosis-like dermatosis and an aromatic hydrocarbon odor. She reported daily ingestion of approximately one to four mothballs over fourteen years to self-medicate for anxiety and depression and abstaining after discovery of pregnancy at sixteen weeks gestation. Ten months earlier, she experienced cognitive decline and underwent magnetic resonance imaging (MRI) of the brain, which was unremarkable. She delivered a viable, female neonate via cesarean section, weighing 2,325 grams (23rd percentile),

with Apgar scores of 9 and 9 at one and five minutes. Placental weight was 370 grams (less than 3rd percentile for gestational age). The neonate exhibited transient hypoglycemia, periodic lip-smacking and facial twitching, which resolved within forty-eight hours. MRI evaluation and laboratory analysis ensued for both patients after delivery. Mother's MRI demonstrated degenerative leukoencephalopathy while neonate's was unremarkable. Whole blood 1,4-dichlorobenzene and metabolite, urinary 2,5-dichlorophenol, were quantified via gas chromatography (Table 1). Additional laboratory analysis of the mother's serum showed the presence of a chronic, mild, microcytic anemia, but ruled out lead exposure out of concern for Pica. There was no clinical or serologic evidence of naphthalene or camphor exposure.

Discussion: DCB is the most commonly utilized ingredient in moth repellents in the United States. Other purposes include use as a disinfectant, deodorizer, fumigant, insecticide, fungicide, and industrial solvent. DCB is associated with acute and chronic toxicity, primarily following gastrointestinal or cutaneous exposures. Knowledge regarding the mechanism of toxicity is limited. DCB undergoes hepatic metabolism and renal elimination. Its lipophilicity allows wide distribution and persistence in various tissues, and animal studies demonstrate redistribution into breast milk and placenta. Acute DCB toxicity is rare with mild symptomatology. Chronic toxicity includes neuropsychiatric dysfunction, impaired cognition, weight loss, weakness, neuropathy, ichthyosis-like rash, hemolytic anemia, cerebellar dysfunction and leukoencephalopathy. DCB is an IARC 2B agent. Diagnosis is clinical. Exposure can be confirmed by whole blood or urinary measurement of 1,4-dichlorobenzene and its metabolite, 2,5-dichlorophenol. DCB is radiopaque, differentiating it from naphthalene and camphor in the setting of mothball ingestion. Presentations consistent with chronic toxicity warrant brain MRI to evaluate for leukoencephalopathy. Treatment is cessation of exposure and supportive care. Management should include consultation with a medical toxicologist. Despite prior reporting of leukoencephalopathy secondary to chronic DCB toxicity, this appears to be the second reported case of maternal-fetal exposure, and the first case with associated neonatal symptomatology. The neonate's symptoms were nonspecific; however, they were otherwise unaccounted for with no prior documented human neonatal symptomatology for comparison. Limitations include error preventing fetal DCB detection, which begets concern for only placental transmission of metabolite; however, this alone may be of concern.

	1,4-dichlorobenzene (whole blood)	2,5-dichlorophenol (urine; metabolite)
Mother	24 mcg/mL	1,100 mg/L
Neonate	*	540 mg/L
Normal range	<2 mcg/mL	<5.0 mg/L

*unobtained due to collection error.

KEYWORDS Paradichlorobenzene, mothball, pregnancy

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269. Tropicamide intravenous abuse in opioid users: a case report.

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Background: Tropicamide is an antimuscarinic agent used as ophthalmoplegic solution to produce mydriasis and cycloplegia. The misuse of such drug via Intravenous (IV) injection is a growing phenomenon especially between opioid users, due the property of apparently limiting and/or delaying opioids' withdrawal symptoms. Although Tropicamide's mechanism of action is not fully disclosed besides its blocking effect on M4 muscarinic receptors, this may be due to the interaction between cholinergic system and opioids. Its cheapness and widespread availability make it attractive especially to vulnerable, marginalized young people with a history of polydrug use. Nevertheless, reports on this topic in literature are still anecdotal.

Case Report: A 25-years old woman accessed the ER after IV self-administration of Tropicamide and Phenylephrine Hydrochlorine. Approximately 8-10 hours before she injected the whole content of a 10ml vial of eye drops, which contains 50mg of Tropicamide, 1000mg of Phenylephrine, in addiction to various excipients (Sodium Metabisulfite, Sodium Edetate, Sodium Ethylmercurithiosalicylate and purified water). The patient was known for being a heroin user in Methadone maintenance treatment. She later reported that it was not the first time she was using Tropicamide IV in order to alleviate opioids' withdrawal symptoms, or to extend opioids' effects. On the present occasion she was found in critical condition with cardiocirculatory insufficiency and carried to the ER: she was responsive, presenting tachycardia (ranging between 120-160 bpm) with ventricular arrhythmias, anxiety, agitation and sweating. Central Venous Pressure (CVP) was 14mmHg. Due to the severe left ventricular insufficiency with Ejection Fraction (EF) 25%, an Intra-Aortic Balloon Pump (IABP) was inserted. She was then supported with symptomatic therapy and transferred to the Cardiology ward after three days, as she recovered and her vital parameters normalized. She was discharged five days later without any sequelae.

Case Discussion: Anticholinergic agents' abuse is not a new phenomenon for addiction medicine; nevertheless, IV misuse of such drugs by polydrug and opioid users is a growing and alarming trend. The anticholinergic inhibition of dopamine reuptake and storage leading to euphoria may not be sufficient to explain such phenomenon. According to patients' reports, this may be due to its interaction with opioids and to its role in reinforcement and addiction, thanks to cholinergic interneurons in nucleus accumbens and projections to the ventral tegmental area, which mediate the symptoms of opiate withdrawal.

Conclusion: Health professionals should be aware of Tropicamide misuse by opioid users, especially among marginalized and vulnerable people, in order to promptly recognize and treat signs and symptoms of anticholinergic intoxication. Furthermore, associated risks of IV drug intake (such as phlebitis) should be taken into consideration. Since literature data is scarce, further investigation is needed to determine the real extension of this phenomenon.

KEYWORDS Tropicamide, Substance misuse, Anticholinergics

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270. Nephrotoxicity and Acetaminophen: a Rare Occurrence in the Absence of Hepatic Dysfunction

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Background: Acetaminophen (APAP) is a leading cause of liver failure worldwide. Despite having an elusive mechanism, it is reported that acetaminophen is also directly nephrotoxic. Case reports have suggested that acetaminophen overdose may result in renal injury in the absence of hepatotoxicity. The prevalence of this clinical scenario and the characteristics defining patients at risk for its development are currently unknown. Therefore, the purpose of this study is to determine the prevalence of APAP-induced renal injury in the absence of significant hepatic injury in isolated acetaminophen ingestions.

Methods: We extracted data collected over ten years from a large, regional poison center. Only single agent acetaminophen ingestions were considered. Specific data extracted included gender, age, outcomes, disposition, interventions, hepatic dysfunction, and renal dysfunction. Descriptive statistics were performed. Interventions examined included dialysis/hemoperfusion and transplantation. Hepatic injury was defined as an AST or ALT >100 U/L but less than or equal to 1,000 U/L. Hepatotoxicity was defined as an AST or ALT >1,000 U/L. Renal dysfunction included both renal injury, defined as a creatinine >1.5mg/dL or >133 µmol/L., and renal failure. The latter was defined as acute or chronic renal failure associated with clinically significant azotemia and loss of renal function. Descriptive statistics were performed to determine prevalence and proportion of patients with APAP ingestions that developed (1) hepatic dysfunction, (2) renal dysfunction, (3) both hepatic and renal dysfunction, (4) renal dysfunction and hepatic injury (but not hepatotoxicity) and (5) renal dysfunction without hepatic dysfunction.
Results: We extracted and analyzed 38,193 cases of single agent APAP overdose.The mean age was 18.87 years and 55.0% (N = 21,020) were female. Of the overall cohort, 4.0% (N = 1519) developed hepatic dysfunction and of these, 161 (10.6%) developed renal dysfunction. Overall, 0.5% (N = 207) of patients developed renal dysfunction. Additionally, 0.4% (N = 161) of all patients developed both renal and hepatic dysfunction. Furthermore, 0.1% (N = 46) developed renal dysfunction in the absence of any hepatic dysfunction. The mean age of patients who developed renal dysfunction was 49.6 years. The mean age of patients who developed renal dysfunction without any form of hepatic dysfunction was 50.24 years. A greater proportion of patients with renal dysfunction underwent dialysis or hemoperfusion compared to the overall group. All four patients receiving hepatic transplantation had developed renal dysfunction. There were 44 deaths overall (0.12%) and 354 patients experienced major effect (0.93%). Of the deaths, 63% (N = 28) developed renal injury.

Conclusions: Our findings demonstrate that renal dysfunction after acetaminophen ingestion is rare. Renal dysfunction in the absence of any hepatic dysfunction, or in the setting of hepatic injury without hepatotoxicity is exceedingly rare. While it appears that the development of renal dysfunction without hepatic dysfunction in the setting of APAP overdose is an uncommon occurrence, clinicians should be aware that it may occur and that higher ages may be at greater risk.

KEYWORDS Acetaminophen, Nephrotoxicity, Hepatotoxicity

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Table 1 Outcomes.

	Single agent APAP with renal injury		All single agent APAP	
Outcomes	Number of pts	Percentage of pts	Number of pts	Percentage of pts
Death	28	13.53%	44	0.12%
Major effect	78	37.68%	354	0.93%
Moderate effect	86	41.55%	2400	6.28%
Minor effect	1	0.48%	6810	17.83%
No effect	0	0.00%	21447	56.15%
Not followed (judged to have only minor or no effect)	1	0.48%	2920	7.65%
Unable to follow, judged as potentially toxic exposure	2	0.97%	2445	6.40%
Unrelated effect	11	5.31%	1595	4.18%
Not documented	0	0.00%	2	0.01%
Confirmed non-exposure	0	0.00%	176	0.46%
Total	207	100.00%	38193	100.00%

271. Toxicology on the Steppe: Evaluating the Burden of Toxicologic Disease in Mongolia

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Background: Providing healthcare in resource-limited clinical settings is complex. Developing national poison registries in countries with primarily resource-limited clinical settings may reduce this complexity. Mongolia provides public and free medical care to its approximately 3.1 million citizens, nearly half of whom live in the capital city, Ulaanbaatar (UB). All poisoned patients over the age of 16 in UB are

Table 3 Clinical effects.

Clinical effects	Total number of patients	As a percentage of all comers	Mean age
All pts presenting with APAP ingestion	38193	100%	18.87
Ingested APAP and developed renal dysfunction	207	0.54%	49.6
Ingested APAP and developed hepatic dysfunction (injury or hepatotoxicity)	1519	3.98%	39.44
Ingested APAP and developed both hepatic and renal dysfunction	161	0.42%	49.42
Ingested APAP and developed renal dysfunction but not hepatic dysfunction (injury or hepatotoxicity)	46	0.12%	50.24
Ingested APAP and developed renal dysfunction and hepatic injury, but not hepatotoxicity	49	0.13%	49.86

Table 2 Interventions.

	Single agent APAP with renal injury			All single agent APAP		
Interventions	Female	Male	Total	Female	Male	Total
Total no. pts who underwent HD/HP	18	7	25	30	13	43
Mean age at HD/HP	48.11	46.00	47.52	46.60	44.77	46.05
Percentage of total pts	8.70%	3.38%	12.08%	0.08%	0.03%	0.11%
total no. transplanted pts	2	2	4	2	2	4
Mean age at transplantation	40.5	28.5	34.5	40.5	28.5	34.5
Percentage of pts	0.97%	0.97%	1.93%	0.01%	0.01%	0.01%

treated at the National Toxicological Emergency Center, the national center for poisoned patients. Despite the fact that the center sees several hundred patients a year, published data on the burden of toxicologic disease in Mongolia is lacking. Obtaining this data is a necessary first step for developing a national toxic registry. Therefore, the purpose of this study was to characterize poisoning in Mongolia by looking at patients admitted to the National Toxicologic Emergency Center over two years.

Methods: Records of patients admitted to the National Toxicological Emergency Center from 2017 to 2018 were analyzed. We collected age, gender, place of ingestion, intention of ingestion, types of poisoning, and outcomes. Types of poisoning were divided by local physicians into the following groups: carbon monoxide toxicity, alcohol poisoning, chemical poisoning, pharmaceutical poisonings, and other. Outcomes were categorized as: recovery, residual deficits, and death. Descriptive statistics were performed to characterize the nature and severity of commonly seen ingestions.

Results: Of the total 573 patients, 54.45% were female (N = 312). The mean age 35.64 years overall; 34.21 years for females and 37.36 years for males. Approximately 74.04% of all ingestions by a female were intentional (N = 231) and 60.54% of ingestions by a male were intentional (N = 158). Pharmaceutical medicines were the most commonly involved toxins, followed by chemical ingestions and carbon monoxide toxicity. Anticonvulsants, analgesics, and then antipsychotics were the most commonly documented ingested medications. Acetic acid, toxic alcohols, and paint diluents were the most commonly documented ingested chemicals. Of the nine deaths recorded over two years, all but two were from chemical ingestions (one other being a carbon monoxide ingestion, and the other being unspecified).

Conclusions: Data characterizing the burden of toxicologic disease in Mongolia is lacking. Notable findings include a low mortality rate, a tendency towards pharmaceutical ingestion as a means of self-harm, and an overall female predominance. Given that all poisoned patients in the capital are referred to the National Toxicological Emergency Center, this data may be used to characterize the nature of urban poisonings in Mongolia. It also demonstrates that a more robust national registry could help to clarify the current state of toxicologic care throughout the entire country and assist in targeting education campaigns.

Table 1 Presumed place of ingestion.

Location	Number of Patients
Domicile	469
Referred in from another region	29
School	3
Outside of home	30
In vehicle	2
Place of work	37
Unknown	3
total	573

Table 2 Poisoning type characterized by gender and intentionality.

Poisoning type	Intentional	Accidental	Total
Carbon monoxide poisoning	3	80	83
female	1	34	35
male	2	46	48
Alcohol ingestion	16	0	16
female	1	0	1
male	15	0	15
Chemical poisoning	67	88	155
female	20	40	60
male	47	48	95
Pharmaceutical ingestion	302	15	317
female	209	6	215
male	93	9	102
Other	1	1	2
female	1	0	1
male	0	1	1
Total	389	184	573

Table 3 Outcomes characterized by poisoning type and intentionality.

	Recovered	Residual Deficits	Death	Total
Carbon Monoxide Poisoning	81	1	1	83
Intentional	3			3
Accidental	78	1	1	80
Alcohol ingestion	14	2		16
Intentional	14	2		16
Aaccidental				0
Chemical poisoning	141	6	8	155
Intentional	59	2	6	67
Accidental	82	4	2	88
Pharmaceutical ingestion	314	3		317
Intentional	299	3		302
Accidental	15			15
Other	2			2
Intentional	1			1
Accidental	1			1
Total	552	12	9	573

KEYWORDS Resource-limited, Mongolia, Global Health

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272. Inadvertent isobutyl nitrite overdose with co-oximetry assessment

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Background: Alkyl nitrites are inhaled for recreational purposes and frequently cause methemoglobin production with overdose. Pulse CO-oximeters allow for bedside measurement of methemoglobin and carboxyhemoglobin. We report a case of a young male with an inadvertent isobutyl nitrite overdose with a grossly inaccurate methemoglobin estimate by the Masimo Radical-7 Pulse CO-oximeter.

Case Report: Emergency services were called to an apartment complex after a 23-year-old male was found awake but severely cyanotic. Prior to the alert, a friend put a substance in the patient's tea to "help him relax" and encouraged him to drink it. The transport team found the patient on his hands and knees in the bathtub with water running on his head. He was awake, somewhat confused, tachycardic, tachypneic, and cyanotic with an O2 saturation in the 80s on room air. The substance put into his drink was identified as a 30mL bottle of HardWare Original by Never Fake It (isobutyl nitrite). On arrival to the emergency department, he was awake, fully alert and oriented, but diffusely cyanotic with an O2 saturation 90% on nonrebreather. A Masimo Radical-7 Pulse CO-oximeter was placed due to probable dyshemoglobinemia and showed an O2 saturation 87% with an estimated methemoglobin 22.1%. However, laboratory analysis revealed a much higher methemoglobin of 57.9% with a partially compensated respiratory alkalosis (ABG-pH 7.56 / pCO2 20 / pO2 182 / saO2 92 / HCO3 18 / LA 5.7). Methylene blue 2mg/kg (135mg total) was immediately administered. Two hours post-antidote, the methemoglobin level improved to 10.2% and the patient was able to transition to nasal cannula and eventually room air. He was discharged the following day without complications.

Case Discussion: The Masimo Radical-7 Pulse CO-oximeter uses seven or more light-emitting diodes in the sensor to estimate arterial O2 saturation, methemoglobin, and carboxyhemoglobin levels. The brochure states the system has a performance range of 0%-99.9% methemoglobin, but the accuracy of the sensor was based on patients with

methemoglobin levels in the 0%-15% range. The Radical-7 grossly underestimated the methemoglobin level in our patient by 28% to 38%. Because of the relatively normal functional saturation it is doubtful there was significant interference from deoxyhemoglobin which has been previously reported with older Radical-7 sensors. Additionally, our case highlights the potential pitfalls of alkyl nitrite misuse. These products are not sold for human consumption, so there are no readily available instructions for "proper" use. Typical misuse of alkyl nitrites involves the inhalation of fumes from the bottle. However, our patient inadvertently ingested the product due to his inexperience.
Conclusion: The Radical-7's methemoglobin readings may be significantly inaccurate in the setting of severe methemoglobinemia. Healthcare providers should be aware of the potential deficiencies of the pulse CO-oximeter and not use it as the sole basis for making diagnosis or treatment decisions.

KEYWORDS CO-oximetry, Isobutyl Nitrite, Methemoglobinemia

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273. Incidence & Injury due to hydrocarbon abuse: Fluorinated inhalants vs. other hydrocarbons.

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Background: Inhalants are commonly abused substances due to their easy unregulated accessibility. National surveys such as Monitoring the Future track yearly drug use in junior high, high school, and college students as well as young adults. Annual surveys in those age groups typically show higher experimentation rates at a young age that drop off as the respondents mature. Little is known about use in older age groups or about the types of hydrocarbons, such as fluorinated inhalants in computer duster vs propane, butane or other hydrocarbons, which may be abused in each age group.

Table 1 Fluorinated Hydrocarbons vs. Other Hydrocarbons Exposure by Age Group.

	Fluorinated Hydrocarbons	Other Hydrocarbons	
	N (%)	N (%)	Total (%)
Child-Teen 7 to 19	2526 (35.82)	801 (52.66)	3327 (38.81)
20 to 39	3422 (48.53)	456 (29.98)	3878 (45.24)
40 to 59	1072 (15.20)	243 (15.98)	1315 (15.34)
>= 60 years old	31 (0.44)	21 (1.38)	52 (0.61)
Total	7051 (100.00)	1521 (100.00)	8572 (100.00)

Methods: A retrospective observational study using National Poison Data System (NPDS) data from January 1, 2008 to December 31, 2017. Inclusion criteria were single substance intentional abuse exposures to fluorinated inhalants and other hydrocarbons in patients 7 years and older.
Results: A total of 7,501 exposures to fluorinated inhalants and 1,521 exposures to other hydrocarbons were identified. The number of exposures per year to fluorinated inhalants decreased from a peak of 866 exposures in 2010 to a nadir of 584 exposures in 2018, a 33% decrease. The number of exposures per year to other hydrocarbons decreased from a peak of 236 exposures in 2008 to a nadir of 93 exposures in 2018. A total of 4525 (64%) exposures to fluorinated hydrocarbons were in patients 20 years or older. The number of exposures to other hydrocarbons was 801 (53%) in patients 19 years and younger. There were 85 (1.21%) fatalities due to fluorinated inhalants compared to 7 (0.46%) fatalities in the other hydrocarbon group giving a X2 = 6.55; p-value of 0.01. A higher percentage of patients exposed to fluorinated inhalants experienced syncope (15.56% vs 5.06%) and tachycardia (13.1% vs 9.20%) compared to other hydrocarbons. A higher percentage of patients exposed to other hydrocarbons experienced acidosis (3.48% vs 0.72%), ataxia (3.09% vs 1.40%), confusion (10.72% vs 6.92%), electrolyte abnormality (2.63% vs 1.45%), and hallucinations/delusions (1.64% vs 0.85%) compared to fluorinated inhalants.
Conclusions: Overall hydrocarbon abuse is decreasing and is less pronounced in patients who abuse fluorinated inhalants. Patients abusing fluorinated inhalants tend to be older, and are associated with a higher proportion of fatalities compared to other hydrocarbons. Whether these fatalities are due to the nature of fluorinated inhalants themselves, or rather from co-morbidities associated with older age warrant further study. The clinical effects between fluorinated inhalants and other hydrocarbons can be significantly different.

KEYWORDS Fluorinated, Hydrocarbons, Abuse

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274. Pediatric exposures to phentermine

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Background: Phentermine hydrochloride is a sympathomimetic amine and has been FDA approved for weight loss in patients 16 years and older since 1973. Despite phentermine being available for over 45 years, no clear triage threshold for out-of-hospital management of unintentional pediatric ingestions has been established. A toxicosurveillance study performed in children 2 years and younger exposed to phentermine concluded that only minor symptoms developed and minimal therapy if any was required. The aim of this study was to report one regional poison center's (RPC) experience with unintentional phentermine exposures in a pediatric population.
Methods: RPC cases with an unintentional ingestion of phentermine in patients age 5 and under for the period 1/1/2003–12/31/2018 were retrospectively reviewed. Cases were excluded if they involved

Table 2 Fluorinated Hydrocarbons vs. Other Hydrocarbons Clinical Effects.

	Fluorinated Hydrocarbons	Other Hydrocarbons	Chi-square	P-value
Acidosis	51 (0.72%)	53 (3.48%)	79.59	<.0001
Ataxia	99 (1.40%)	47 (3.09%)	21.24	<.0001
Confusion	488 (6.92%)	163 (10.72%)	25.68	<.0001
Electrolyte abnormality	102 (1.45%)	40 (2.63%)	10.75	0.0010
Hallucinations/delusions	60 (0.85%)	25 (1.64%)	8.01	0.0047
Syncope	1097 (15.56%)	77 (5.06%)	116.60	<.0001
Tachycardia	924 (13.10%)	140 (9.20%)	17.50	<.0001
Death	85 (1.20%)	7 (0.46%)	6.55	0.01

Table 1.

Min Exposure	Range (mg)	Mean (mg)	Median (mg)	Standard Deviation	95% CI
No Effect (n = 39)	4.69-150	38.41	37.5	25.06	30.55-46.28
Minor Effect (n = 11)	18.75-37.5	32.84	37.5	7.29	28.53-37.15
Moderate Effect (n = 9)	18.75-56.25	35.62	37.5	10.1	29.02-42.22
Max Exposure	4.69-150	Mean (mg)	Median (mg)	Standard Deviation	95% CI
No Effect (n = 99)	18.75-37.5	51.61	37.5	59.64	39.86-63.36
Minor Effect (n = 23)	7.5-375	64.78	37.5	74.77	34.22-95.33
Moderate Effect n = 14)	15-150	56.47	37.5	41.16	34.91-78.03

Table 2.

Min Exposure	Range (mg/kg)	Mean (mg/kg)	Median (mg/kg)	Standard Deviation	95% CI
No Effect (n = 27)	0.43-9.68	2.91	2.23	2	2.22-3.72
Minor Effect (n = 8)	1.75-3.41	2.38	2.26	0.57	1.98-2.78
Moderate Effect (n = 6)	2.59-3.83	3.11	3.1	0.54	2.68-3.54
Max Exposure	Range (mg/kg)	Mean (mg/kg)	Median (mg/kg)	Standard Deviation	95% CI
No Effect (n = 61)	0.69-30	4.22	2.66	5.24	2.91-5.24
Minor Effect (n = 13)	1.75-18.8	2.59	2.59	4.49	1.44-6.32
Moderate Effect (n = 9)	3.52-8.33	3.52	3.52	3.52	3.03-5.75

co- ingestants, confirmed non-exposure, transferred to an out of state hospital, or were not followed to a known outcome. Based on reported history both a minimum and maximum estimated exposure amount was determined. When a weight was documented both a minimum and maximum exposure in mg/kg was calculated.

Results: A total of 194 cases met inclusion criteria. Patients ranged in age from 6 months to 5 years old with average age of 2. 114 (59%) patients were male compared to 80 (41%) female. 46 (24%) patients were managed at home compared to 148 (76%) in a health care facility. In 91.2% of patients, no effect or minor effects were observed, while 8.8% of patients had moderate or major effects. There no deaths during the study period. The one patient with a major effect was a 2 year old male who ingested an unknown amount of phentermine and developed agitation, tachycardia to 180, hypertension (HTN), ataxia, hallucinations, and hematemesis. Symptoms in patients with a moderate effect included agitation (14), tachycardia (10), HTN (6), tremor (5), hallucinations (4), mydriasis (4), vomiting (3), CPK elevation (3), rhabdomyolysis (2), and other (6). Symptoms in patients with a minor effect included agitation (19), vomiting (11), tachycardia (10), mydriasis (3), HTN (1), and other (9). A total of 36 patients received activated charcoal (1 moderate, 5 minor effect, and 30 no effect). 8 patients with moderate effects and the one major effect patient required treatment with benzodiazepines, 2 moderate effect patients required phenobarbital. A minimum exposure amount was estimated in 51 patients and a maximum amount in 136 patients. A minimum exposure amount in mg/kg was calculated for 41 patients and a maximum amount in 83 patients. There was substantial overlap between no effect, minor effect, and moderate effect for range, mean, median, and calculated 95% confidence intervals (CI).

Conclusion: The majority of pediatric exposures result in no or minor clinical effect, however nearly 10% of exposures can lead to moderate to severe toxicity requiring pharmaceutical intervention. The reported history of an exposure amount to phentermine in pediatric patients does not predict toxic effects.

KEYWORDS Phentermine, Pediatric, Unintentional

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275. Is Mg/Kg a Reliable Triage Strategy for Diphenhydramine Exposure in Children?

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Background: Diphenhydramine (DPH) is ubiquitous in US homes. It is a common call to poison centers, making it is critical to have a reliable triage strategy to assist SPIs in determining if DPH patients can be safely managed at home. Past pediatric DPH exposures were evaluated to assess the appropriateness of the existing DPH triage strategy (>10 mg/kg) as a guide to making recommendations for level of care.

Methods: Archived Toxicall® records were searched for accidental pediatric (0-5 years), single-ingredient diphenhydramine exposures over a 5-year period (January 2014 - December 2018) that were unintentional-general or unintentional-therapeutic error. Patients were divided into 3 groups – managed at home, referred to ED, and self-referred to ED (already in HCF). The amount of DPH ingested in mg/kg (the basis for the triage strategy and level of care) was evaluated for ability to predict symptoms and medical outcome.

Results: There were 1,752 cases that met inclusion criteria: 1,533 managed at home, 87 PC-referred, and 132 self-referred. Ingested amounts for those triaged to home management ranged from 0.36 to 34 mg/kg; for SPI-referred patients, 2.2 to 46.2 mg/kg, and 1.1 to 55 mg/kg for those already in a HCF. Amounts that exceeded 10 mg/kg that were managed at home were often due to significant large spills and calls initially received at or past the peak effect. There was no dose-related correlation between mg/kg amounts and symptoms or outcomes for any of the groups. One explanation is the surprising number (79/87) of referred cases with vague, uncertain or unknown histories of DPH ingestion. These were generally assessed by the SPI as a worst case scenario, resulting in many unnecessary referrals that developed no or only minor symptoms. Of the 1,533 patients managed at home, 6 had moderate effects; of the 87 patients SPI-referred to the ED, 11 had moderate effects; and of the 132 patients already in the ED, 13 had moderate effects. There were no major effects in any of the groups. In home-managed patients, the most common minor effect was drowsiness; moderate effects were limited to drowsiness (4), hallucinations (2), agitation (2), EPS-dystonia (1). In both of the groups of HCF-evaluated patients symptoms most commonly associated with minor outcomes were: drowsiness (46), agitation (15), tachycardia (13), ataxia (5). Symptoms most commonly associated with moderate outcomes were: hallucinations (15), tachycardia (15), agitation (15), other-misc. (7), mydriasis (6), tremor (6), drowsiness (6), EPS-dystonia (5).

Conclusion: Assuming that a vague, uncertain or unknown history in a worst case DPH ingestion likely accounts for the failure of mg/kg amount ingested to predict medical outcome and guide appropriate management site. DPH triage may be better structured to guide SPIs to observe such cases at home with follow-up at 1 and 3 hours, and base ED referral on the development of hallucinations or symptoms intolerable to the child or caretaker. We hypothesize that this may be the case for many childhood exposures to OTC medications accounting for referral of more children to ED than is necessary.

Patients Managed at Home - Medical Outcome	
Major	0
Moderate Effect	6
Minor Effect	517
No Effect	990
Unrelated Effect	20
TOTAL	1,533

Patients Referred by SPI			
Disposition/Level of Care		Medical Outcome	
Admitted to Critical Care	3	Major	0
Admitted to Non-Critical Care	4	Moderate Effect	11
Refused Referral	5	Minor Effect	30
Treated & Released	75	No Effect	46
		Unrelated Effect	0
TOTAL	87	TOTAL	87

Patients Self-Referred / Already in HCF			
Disposition/Level of Care		Medical Outcome	
Admitted to Critical Care	11	Major Effect	0
Admitted to Non-Critical Care	3	Moderate Effect	13
Treated & Released	118	Minor Effect	44
		No Effect	73
		Unrelated Effect	2
TOTAL	132	TOTAL	132

KEYWORDS Diphenhydramine, Triage, Pediatric overdose

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276. Burn 'Baaa'by Burn: A superglue and wool exothermic reaction causing burns

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Background: Cyanoacrylate is commonly found in household glues such as superglue or artificial nail glue. It is an organic monomer that is in the liquid form at room temperature, but through an exothermic reaction with weak alkaline materials it can rapidly polymerize to form a solid substance. This reaction is typically triggered by water; however, some natural materials such as cotton and wool can also serve as triggers to a more rapid exothermic reaction that may lead to burns when in contact with skin. While this rare phenomenon has been reported in wounds and burn literature, this may be the first case reported in toxicology literature.
Case Report: A 52 year-old woman presented to the emergency department with first degree burns to both hands. She reported applying superglue while wearing wool gloves, and that when the glue came in contact with the gloves, they heated to the point of causing the burns. Other causes of cutaneous burns were excluded by history. She was provided topical wound care and discharged home.
Case Discussion: Thermal burns from the combination of wool or cotton in contact with cyanoacrylate glue have been reported. Many reported pediatric burns were reported to have occurred due to glue spillage onto clothing (Eyth et al, 2016; Clarke et al, 2011; Belanger et al, 2013). Full thickness burns have been reported in three cases due to artificial nail glue, all requiring surgical debridement and skin graftin (Kelemen et al, 2016). The contact of cyanoacrylate with cotton or wool fibers produces a violent exothermic reaction due acceleration of the polymerization reaction by the hydroxyl groups contained in natural fibers. Medical grade cyanoacrylate derivatives such as butyl-or octyl-cyanoacrylate have longer chains that lead to slower polymerization, and are thus unlikely to cause thermal injury.
Conclusion: To our knowledge, this is the first report of burns due to the contact between a nonmedical cyanoacrylate adhesive to a natural fabric reported in the toxicology literature. While this is likely a rare occurrence, the wide availability of cyanoacrylate-based glues accessible to consumers portend a significant risk of burns of varying severity among product users unaware of this little-known exothermic reaction.

KEYWORDS Cyanoacrylate, Exothermic, Burns

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277. Ebb Cade and the Ghosts of Oak Ridge

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Background: Oak Ridge, TN was created as part of the Manhattan Project; a city-sized secretive laboratory to develop the atomic bombs that were dropped on Japan in 1945. From 1942 to 1945 the population of Oak Ridge exploded from 3,000 to over 75,000 and a 17-square mile patch of land in the shadows of the Great Smoky Mountain range was daily consuming more electricity than New York City. Secrecy was the single most important element in the day-to-day lives of the citizens of "Atomic City" (also called "The Secret City"). This secrecy included a forgotten chapter of the Oak Ridge story – human radiation experimentation.
Case Report: Ebb Cade was an African-American automobile crash-victim injected with plutonium while an inpatient at the Oak Ridge Hospital. Cade suffered numerous fractures in the car crash, but doctors waited 20 days to attend to his fractures. Biological samples, including fifteen of his teeth, were harvested and sent to the Los Alamos National Laboratory for assay. Cade's ultimate fate is uncertain – whether he was discharged from care or walked away from the hospital is uncertain. He simply disappeared and is thought to be buried in Greensboro, NC.
Case Discussion: Cade was not the only subject injected with plutonium – at least eighteen others (11 in Rochester, NY, 3 at the University of Chicago and 3 at UC San Francisco) were intentionally, and without their knowledge or consent, injected with various doses of plutonium (Pu-239) and their tissue samples collected. President Clinton appointed the Advisory Committee on Human Radiation Experiments (ACHRE) to investigate these and other U.S. Government funded experiments in deliberate exposure of human subjects to plutonium and other radioactive isotopes. The committee's final report was published in 1996.
Conclusion: The story of Oak Ridge TN (located a quick 2 and ½ hour drive from Nashville) and the experimental plutonium people is one that needs to be heard. This presentation is an academic discussion of human radiation experimentation prior to the Nuremberg Trials and the development of modern ethical research practice in the context of the race for national atomic supremacy in the shadow of Oak Ridge, Tennessee.

KEYWORDS Oak Ridge, TN, Plutonium, Human Experimentation

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278. Death by Fungicide

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Background: In the United States, suicide by intentional copper poisoning is rare and results in significant gastrointestinal (GI) symptoms in addition to shock and multi-organ system failure. Commercially, copper is found most commonly in fungicides, algicides, and insecticides. We report a case of suicide by ingestion of copper-containing fungicide with elevated post-mortem copper level.
Case Report: This is a case report of a 77-year-old woman with a history of depression who lived alone and was found dead at home by her relative. Her relative was performing his daily visit to her home to dispense her medications because she had severe depression and suicidal ideation. He found the decedent lying in her daybed, cold to touch, with blue discoloration on her lips. On arrival, paramedics attempted resuscitation prior to pronouncing her deceased. During CPR, blue liquid was noted to be coming from her mouth. There was no suicide note, but a bottle of open blue-colored fungicide labeled "Kop-R-Spray" was found in the kitchen, with a significant amount of fluid missing. Kop-R-Spray contains metallic copper. Her medications included donepezil, duloxetine, lorazepam, mirtazapine, and temazepam. Autopsy was performed and documented dried turquoise-colored material on the lips and around the nostrils, along with on the inside of the thighs, on the legs, and on the dorsal and plantar aspects of the feet. There were CPR-related thoracic injuries and coronary artery and aortic atherosclerosis. Throughout the GI tract extending from the oral cavity to the small intestine was extensive turquoise material consistent with liquid found in the Kop-R-Spray bottle found at the scene. Toxicological testing using inductively coupled plasma/optical emission spectrometry detected copper concentration in the peripheral blood of 5.0 mg/L. Normal blood concentrations of copper are 0.80 to 1.80mg/L. Peripheral blood was also positive for low and sub-therapeutic levels of diphenhydramine, mirtazapine, donepezil, temazepam and oxazepam. Cause of death was determined to be acute copper toxicity.
Case Discussion: Copper salts are highly irritating to mucosal surfaces, and acute oral ingestions often cause characteristic blue-green emesis. Serious copper toxicity is manifested by significant vomiting and diarrhea causing hypotension or shock, gastrointestinal hemorrhage, hemolysis, hepatic and renal injury, and rarely methemoglobinemia, multisystem organ failure, and death. Death occurs soon after ingestion in up to one third of cases. Kop-R-Spray contains 8% copper ammonium complex. It is unknown the quantity the decedent ingested prior to her death. The post mortem peripheral blood copper level was 5.0 mg/L, and this elevated level, in addition to the turquoise material found throughout her GI tract on autopsy, strongly suggest acute copper toxicity as her cause of death.
Conclusion: We report an uncommon case of death due to acute copper toxicity from ingestion of a fungicide containing copper ammonium salts. The decedent developed characteristic turquoise emesis and had turquoise material throughout the GI tract on autopsy, in addition to an elevated peripheral blood postmortem copper level to 5.0 mg/L.

KEYWORDS Copper, fungicide, overdose

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279. High Dose Insulin-Euglycemia Therapy in Pediatric Calcium Channel Blocker Overdose

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Background: There a paucity of data regarding high dose insulin-euglycemia (HIE) therapy for calcium channel blocker (CCB) poisoning in pediatric patients. The use of HIE in adult CCB overdoses remains somewhat controversial, and adult data has been extrapolated to recommendations in pediatric cases. There are some theoretical concerns about safety of this therapy in pediatric patients due to their lower glycogen stores and potentially greater risk of hypoglycemia with HIE. The goal of this review was to describe cases of HIE therapy utilization in pediatric patients.
Methods: This was a retrospective review of CCB exposures in patients less than 18 years old reported to a poison control system between 2003-2017. Cases of CCB overdose in which HIE was implemented were identified and reviewed. This is a descriptive study and no statistical analysis was performed.
Case Reports: A total of 341 cases of CCB overdose were identified. Poison specialists discussed the option of HIE 84 times; however, HIE was only implemented in 3 cases.
Case 1: A 16-year-old female presented after ingesting 3.6 g of extended release verapamil in a suicide attempt. She was initially asymptomatic and was given activated charcoal and whole bowel irrigation was started. Her blood pressure subsequently decreased to 60/40 mmHg and intravenous fluids and calcium gluconate 1 g was administered. She remained hypotensive and HIE at a rate of 1 unit/kg/hr was started along with D10/1/2 NS. Blood pressure improved. She was later noted to have hypoglycemia and HIE was stopped. She recovered and was transferred to psychiatric facility after a 2-day hospitalization.
Case 2: A 16-year-old female presented with polypharmacy overdose, but initial ingestion was unclear. It became apparent late that the patient's polysubstance ingestion included amlodipine. She became obtunded and was intubated. Hypotension that developed was treated with four vasopressors and MAST suit. After toxicology was consulted, she was placed on HIE therapy and calcium was administered. Her dose, duration and response to HIE were not documented. The patient also received plasma exchange and developed complications related to this. The patient had a prolonged course, but ultimately recovered. Details of HIE therapy or complications related to this are unknown.
Case 3: A 14-year-old female presented to the emergency department with an overdose of an unknown amount of metoprolol and extended release nifedipine. She was asymptomatic on presentation, but developed bradycardia and hypotension and was started on epinephrine infusion, glucagon bolus followed by infusion, calcium chloride boluses, and HIE. Her dose, duration and tolerance of HIE are not documented. She survived.
Conclusion: HIE therapy is rarely implemented in cases of pediatric CCB exposures. This may be due to the lack of treatment team familiarity with the therapy, comfort with other modes of hemodynamic support, or hesitancy to implement HIE therapy in children. In the 3 cases in which HIE was implemented, there was 1 case of hypoglycemia, and no other adverse effects documented, although details provided in documentation were limited.

KEYWORDS Calcium channel blocker, high dose insulin, pediatric

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280. Pediatric Lantana camara ingestions reported to poison centers

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Background: Lantana (Lantana camara) is an ornamental shrub found in subtropical-temporate regions around the world, including

at least 12 states in the southern part of the United States. L. camara leaves are rough and produce an aromatic odor when crushed. Its flowers are brightly colored and occur in clusters. The fruit is small and turns purple-black when mature. Ingestion of L. camara by animals has resulted in toxic effects. Information on the adverse effects from human L. camara ingestions is limited, although one large study is reported in the literature. The objective of this study was to describe L. camara ingestions among young children reported to poison centers.

Methods: Cases were all L. camara ingestions among patients age 5 years or less reported to a statewide poison center network during 2000-2018. L. camara ingestions were identified by review of records with PoisIndex codes for Lantana species or "lantana" in the Substance Verbatim field. The distribution of L. camara pediatric ingestions was determined for various factors related to patient demographics, ingestion circumstances, management, and outcome.

Results: A total of 784 L. camara ingestions involving young children were identified. The part(s) of the plant ingested were berry or seed (n= 327, 41.7%), flower (n = 193, 24.6%), leaf (n= 125, 15.9%), stem or branch (n = 13, 1.7%), and unknown (n = 142, 18.1%). October-November accounted for 229 (29.2%) of the cases and January-February for 22 (2.8%). The patient age distribution was 127 (16.2%)

Conclusion: Pediatric L. camara ingestions most often involved the berry or seed followed by the flower and leaf. The ingestions were seasonal, peaking in October-November. The majority (81.8%) involved children age 0-2 years. Most L. camara ingestions were managed outside of a healthcare facility and did not result in a serious outcome.

KEYWORDS Camara lantana, pediatric ingestions, plant ingestion

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281. Predictors of Death in 2,4-Dinitrophenol (2,4-DNP) Overdose

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Background: The frequency of 2,4-Dinitrophenol (DNP) poisoning has increased in the UK and US over the last decade. DNP may promote weight loss through uncoupling oxidative phosphorylation at low doses but can cause rapidly developing hyperpyrexia and profound metabolic disturbances including lactic acidosis, especially after larger acute doses. This research was performed to identify factors that predicting a fatal outcome to allow earlier identification of patients at particular risk.

Methods: The American Association of Poison Controls Centres' National Poison Data System and National Poisons Information Service UK Poison Information Database were searched for calls which included keywords "dinitrophenol" and "DNP" between 1st January 2007 and 31st December 2018. These data were merged and analysed using descriptive, univariate and multivariate statistics.

Results: A total of 204 cases (n = 84, USA; n = 118, UK) were identified, including 30 reported deaths (n = 10, USA; n = 20, UK). There was no difference in proportions of deaths between the US and UK datasets ($X2(1) = 0.96$, $p = 0.33$). The age (OR = 1.00, 95% CI: 0.963 – 1.053; $p = 0.76$) and sex (OR = 0.92, 95% CI: 0.395 – 2.153; $p = 0.85$) of users were not predictors of death. Those reporting an acute or an acute-on-chronic overdose had a greater odds of death than those using DNP chronically (OR = 9.69, 95% CI: 2.21 – 42.45; $p < 0.05$) reflecting ingestion of greater doses in the 24 hours prior to presentation (median [range]: 1.20 grams [0.14 grams – 6.00 grams] vs. 0.68 grams [0.05 grams – 2.80 grams]; $p < 0.05$). The reported ingested-dose was a significant predictor of death (OR = 2.81 per gram increase in reported dose, 95% CI: 1.76 – 4.50; $p < 0.05$). The presence of hypoglycaemia (OR = 15.92, $p < 0.05$), hypertonia (OR = 12.94, p < 0.05), acidosis (OR = 12.54, $p < 0.05$), raised lactate (OR = 8.26, $p < 0.05$), fever (OR = 6.50, $p < 0.05$), tachycardia (OR = 6.39, $p < 0.05$), agitation or confusion (OR = 5.96, $p < 0.05$), hypertension (OR 5.58, $p < 0.05$), tachypnoea/dyspnoea (OR = 2.75, $p < 0.05$) were significant predictors of death in the univariate analysis. Backwards step-wise logistic regression modelling identified the following significant independent predictors of mortality: acidosis (OR = 4.27, 95% CI: 1.32 – 13.93; $p = 0.016$), fever (OR = 2.97, 95% CI: 1.062 – 8.28; $p = 0.038$), tachycardia (OR = 3.63, 95% CI: 1.2 – 11.0; $p = 0.023$) and agitation/confusion (OR = 3.30, 95% CI: 1.2 – 8.9, $p = 0.017$).

Conclusions: DNP toxicity has a high case fatality; significant predictors of a fatal outcome include acute or acute on chronic overdose and high dose ingested. Clinical features associated with mortality as reported during enquiries to poisons centres include acidosis, fever, tachycardia and agitation or confusion. These features should prompt rapid escalation to an intensive care environment for aggressive supportive treatment and monitoring.

KEYWORDS Mortality, 2,4-Dinitrophenol, DNP

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282. Increasing Number of 2,4-Dinitrophenol (2,4-DNP) Cases Reported to Poisons Centers in the USA and UK

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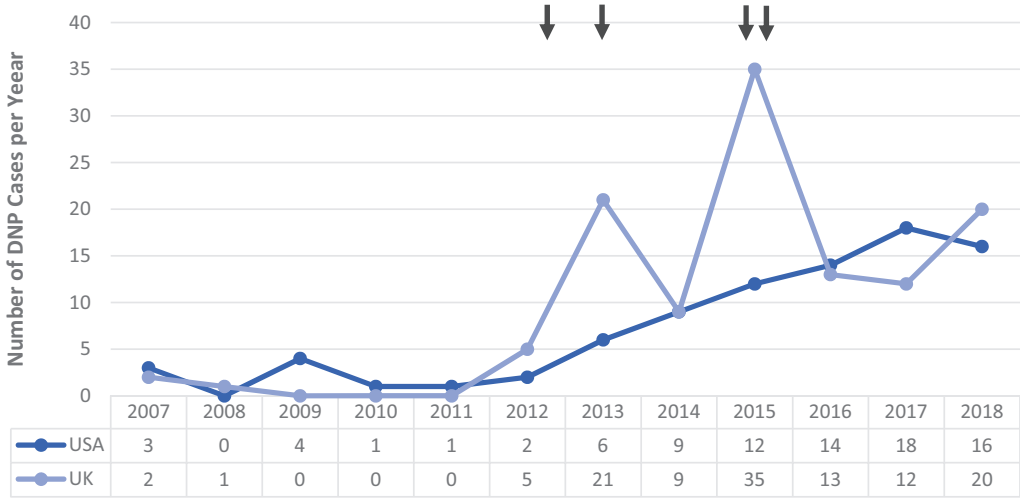
Background: 2,4-Dinitrophenol (DNP) is an industrial chemical misused for weight-loss and body sculpting despite not being a licenced medicine in the USA, Europe or elsewhere. It increases energy consumption by uncoupling mitochondrial oxidative phosphorylation. Although there are many case reports of severe toxicity and death following ingestion, limited information is available on the frequency of current use and toxicity. This research was performed to characterise the toxicoepidemiology of DNP usage and compare trends between the USA and UK using poisons center data.

Methods: The American Association of Poison Controls Centers' National Poison Data System (healthcare professional and public calls) and the UK National Poisons Information Service database (healthcare professional calls only) were searched for enquiries including the keywords "dinitrophenol" or "DNP" between 1st January 2007 and 31st December 2018. The age, sex of the user and details of DNP use were extracted and analysed using descriptive and univariate statistics.

Results and Discussion: Over the 12 years of study there were 86 US cases and 118 UK cases identified. Cumulative incidence of reported DNP toxicity was higher in the UK than the USA during this period (1.78 vs. 0.26 cases per 1,000,000 population over 12 years). There have been recent increases in the number of cases reported over the last decade (Figure 1) with most identified since 2013 (87% of USA and 93% of UK cases). The largest annual numbers of cases were seen in 2017 (USA, n = 18) and 2015 (UK, n = 35). Non-sustained reductions in episodes of toxicity were observed in the UK following public health warnings issued in 2012, 2013 and 2015 (Figure 1). Males were involved more often than females in episodes of toxicity in the USA (80.2% male; 95% CI: 70.6%-87.3%) and UK (64.4% male; 95% CI: 55.4%-72.5%) and accounted for a larger proportion of users in the USA (80.2% vs 64.4%;



	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018
USA	3	0	4	1	1	2	6	9	12	14	18	16
UK	2	1	0	0	0	5	21	9	35	13	12	20

Figure 1 Number of cases reported to healthcare professionals at poison centers in the USA and UK between 2007 and 2018. Grey arrows indicate UK Food Standards Agency (FSA) and Public Health England (PHE) warnings in November 2012, October 2013, July and October 2015.

X2(1) = 6.01, p = 0.014). The median age of USA female users was older than in the UK (median [range]: 29 [19-59] USA vs. 24 [17-34] UK; p = 0.007) however there was no difference in the age of male users (median [range]: 25 [14-61] USA vs. 25 [15-45] UK; p = 0.951). The proportion of use that was acute or acute-on-chronic was 69.0% (95% CI: 58.2%-77.4%) in the US and 44.9% (95% CI: 36.3%-54.0%) in the UK. The proportion of chronic use was 28.0% (95% CI: 20.0%-38.2) in the US and 44.92% (95% CI: 36.3%-54.0%) in the UK. Case fatality proportions are high in the USA (11.63%; 95% CI: 6.44%-20.10%) and UK (16.95%; 95% CI: 11.30%-24.74%; USA vs. UK: X2(1) = 1.12, p = 0.29).
Conclusions: DNP toxicity is uncommonly reported to poisons centres but has been increasing in frequency in the USA and UK since 2013. Compared with the UK, toxicity is less common per population however there is a larger male preponderance, female users are older and there is a higher proportion of acute or acute on chronic episodes in the USA. Case fatality is high.

KEYWORDS 2,4-Dinitrophenol, DNP, Epidemiology

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283. Attitudes toward fentanyl test strips as a harm reduction strategy among emergency department patients who have used opioids in the past year

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Background: Fentanyl fatalities are more common than those from prescription opioids or heroin, and their fatality rates continue to rise. This powerful pharmaceutical is being used as an additive to opioids, which is suspected to contribute to the increase in opioid overdoses in the United States. Fentanyl testing strips are commercially available but poorly studied as a harm-reduction tool. There is little research into drug-user willingness to use such strips or whether knowledge of additives, such as fentanyl, would change the behavior of opioid users. The aims of our study were to: describe a population of non-prescription opioid users; measure attitudes about potential fentanyl exposure; and assess openness to acquiring fentanyl test strips from emergency departments.
Methods: We conducted a prospective, cross-sectional survey of non-prescription opioid users presenting to an urban emergency department between November 2018 and April 2019. We administered a pre-screening survey to a convenience sample of patients age 18 years and older. Eligible patients were those who self-reported using non-prescription opioids in the past year. Non-english speaking and incarcerated patients were excluded. The survey was presented verbally by trained research assistants. Survey questions investigated the feasibility of fentanyl test strips as a harm reduction strategy for opioid users. We surveyed attitudes of participants on barriers to fentanyl test strip cost, access, and use. We used descriptive statistics for analysis.
Results: We screened 197 patients of whom 35 were eligible and 31 consented to participate. One subject was not able to complete the full survey. Participants were between the ages of 20 and 70 years; 17 (57%) were male and 13 (43%) female. The majority of our sample reported comorbid substance use with opioids in the pre-screening survey: 25 (81%) used tobacco products, 23 (74%) used cannabis, and 22 (71%) used amphetamines. Twenty-five (81%) respondents have been in a drug recovery treatment program for their opioid use. Twenty-six (84%) "agreed" or "strongly agreed" that they would like to know if fentanyl is mixed with their drugs. Twenty-six respondents (84%) said if the strips were being offered for free at the emergency department, they would be willing to come get them.
Conclusions: Our survey suggests most patients who use non-prescription opioids are concerned their drugs may be laced with fentanyl and would like the ability to know if fentanyl is mixed with their drugs before they take them. Patients' willingness to obtain fentanyl test strips for free at the emergency department supports the idea that fentanyl test strips may be an effective harm reduction strategy. Many emergency departments are already integrated with treatment referrals and may be logical places to integrate fentanyl test strip distribution and targeted fentanyl risk education into existing efforts to combat opioid related deaths. Future research is needed to determine the impact of behavior and survival changes with fentanyl test strips in the hands of a high risk population.

KEYWORDS Opioid use disorder, Overdose, Nonprescription

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284. END DOPE: Effects of Narcan Distribution and Delivery of Overdose Prevention Education in the Emergency Department

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Background: The overdose crisis is an ongoing public health concern, with death from opioid overdose recently having become the leading cause of preventable death in the United States. Overdose prevention education and naloxone distribution have been ongoing since the 1990s, and have been shown to be effective harm-reduction strategies. However, these programs historically have been implemented in specialty addiction clinics and health centers. There is little data for Emergency Department (ED)-based overdose education and naloxone distribution (OEND) programs.
Objectives: To determine the efficacy and feasibility of an ED based OEND program by targeting patients who present to the ED after an opioid overdose.
Methods: This is an interim analysis of an ongoing prospective study of patients presenting to the ED after an opioid overdose. Patients age 18 and older who presented after an opioid overdose, as well as accompanying family members are eligible for inclusion. Those who agreed to participate were asked to complete a survey and provide contact information for 30 day post-discharge follow up. Patients were given a kit containing 1 naloxone 4 mg nasal spray as well as instructions on how to administer the naloxone and a list of treatment resources prior to discharge from the Emergency Department. Patients are only eligible to enroll in the study once.
Results: Of the 223 patients approached, 98 (44%) declined enrollment, 125 patients (56%) consented to participate, but not all surveys were complete. Data is presented as descriptive. Of those enrolled, 117 kits were given directly to the patient, 5 were given to a family member or friend with the patient. The majority of patients were white (73, 60.3%), male (96, 77.4%) and homeless (45, 36.6%). Most patients reported that this visit was not the first time they were in the ED after an overdose (N = 124, 80, 64.5%). Of those who had been to the ED previously for an overdose, the mean number of times patients reported having been in the ED was 4.74 (SD 4.37). Most respondents (N = 125) reported that they had been treated with naloxone previously (77, 61.6%). Of those, the range of previous naloxone administrations was 1-25 times, Thirty day follow up was completed on 27 respondents. One fatality was reported. Seven patients reported that the naloxone had been used. Three respondents reported that they gave the naloxone to another individual, 2 stated that they used it on themselves and 1 patient said someone other than the owner administered the naloxone to the owner. One patient did not answer. All respondents (28, 100%) stated that they believed that naloxone should be dispensed to patients at the time of discharge from the ED after presenting with an overdose, and 96% of patients stated that they felt safer knowing that they had the kit.
Conclusions: The ED appears to be an effective and feasible setting for overdose education and naloxone distribution. The majority of patients who presented to the ED after an overdose that met inclusion criteria were interested in receiving naloxone and the accompanying educational materials.

KEYWORDS Naloxone, opioid, harm-reduction

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285. Accidental Torsemide and caffeine ingestion leading to significant hypokalemia and cardiac arrest.

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Background: Torsemide being a loop diuretic can cause hypokalemia. Caffeine is also known to cause hypokalemia by multiple mechanisms. Here we report a case of severe hypokalemia leading to cardiac arrest after a mistaken/intentional ingestion of Torsemide and caffeine tablets.
Case presentation: A 24-year-old female with no significant past medical history was found unresponsive by her parents in her room. Her mother started Cardiopulmonary resuscitation (CPR) after confirming cardiac arrest until Emergency medical services(EMS) arrived within 10 minutes. She had multiple episodes of Ventricular fibrillation requiring defibrillation. CPR continued for 16 minutes. During the CPR in the emergency department, venous blood gas sample was taken and Extracorporeal membrane oxygenation (ECMO) team was activated considering her young age and prolonged cardiac arrest. The VBG showed a hypokalemia(1.5 mmol/L), hypocalcemia (0.93 mmol/L) and hyponatremia(128 mmol/L). These values were later supported by the serum analysis. The patient achieved ROSC(Return of spontaneous circulation) after intravenous replacement of potassium and calcium by boluses and infusion. On obtaining a detailed history from the family, it became evident that she was mistakenly taking torsemide 20 mg tablets, belonging to her grandmother, instead of her meloxicam tablets which appear similar to the torsemide pills. She was taking meloxicam for her symptoms of dysmenorrhea. A total of 6 tablets of torsemide were missing from the pack. She was also on caffeine tablets which she was taking for weight loss. A possibility of suicidal intention could not be ruled out from the history available from the family. After resuscitation, she was admitted to the medical intensive care unit (MICU). During the course of her stay in MICU, she was found to have significant anoxic brain injury secondary to the prolonged periods of cardiac arrest. She also developed multi-organ failure. She underwent tracheostomy and is currently undergoing rehabilitation.
Discussion: Torsemide acts at the sodium-potassium-chloride re-absorptive pump located in the ascending loop of Henle interfering with the chloride-binding cotransport system, thus causing increased excretion of water, sodium, chloride, magnesium, and calcium. Caffeine is reported to cause hypokalemia as well, by various mechanisms. When used together, they may synergistically act to augment this electrolyte imbalance, as reported in this case. Torsemide and meloxicam pills look similar in physical appearance, thereby increasing the risk of accidental ingestion of torsemide as in this case.
Conclusion: Torsemide and caffeine can act synergistically to cause significant hypokalemia that can lead to cardiac arrest as happened in our patient. The patient was having resistant cardiac arrest which was reverted only when potassium bolus was given. The importance of looking at reasons for reversible causes of cardiac arrest cannot be overstated and, in this case, discovering hypokalemia and correcting it helped in attaining ROSC. Certain brands of torsemide and meloxicam pills can look similar, thereby increasing the risk of accidental torsemide ingestion.

KEYWORDS Torsemide, Meloxicam, Hypokalemia

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286. Characterization of Oxycodone Misuse using National Survey Data.

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Background: Drug overdoses are a public health crisis with 63,632 fatalities in 2016. Approximately two-thirds of these deaths (66%) involved a prescription or illicit opioid. Prescriptions for oxycodone-containing analgesics exceeded 54 million in 2016 with 182,748 oxycodone related emergency department visits (ED) in 2010. The objective of the study is to characterize the risk markers of oxycodone misuse using the nationally representative National Survey of Drug Use and Health (NSDUH) data.
Methods: The 2017 NSDUH public use data were analyzed. The respondents were classified into two groups, past year oxycodone misusers and non-misusers, based on the screening questions assessing past year misuse of oxycodone. The prevalence of selected demographic, clinical factors and substance use and abuse, including prescription medications, was assessed descriptively using cross tabulated frequencies and chi-square tests. Logistic regression models using a backward selection process were used to identify predictors of oxycodone misuse adjusting for covariates. Adjusted odds ratios (OR) and corresponding 95% Confidence Intervals (CI) were calculated
Results: Overall, the 2017 NSDUH survey comprised of 56,276 respondents, of which 5,212 respondents (9.2%) reported using oxycodone products over the last year. 1,074 respondents reported misuse, accounting for 20.6% of the total oxycodone users or 1.9% of the survey sample. Past year oxycodone misusers were more likely to be males (55.1% vs 42.6%, p
Conclusions: Using data from a nationally representative sample our study indicated a high prevalence of oxycodone misuse. Our study highlighted risk factors associated with misuse of oxycodone products. Several factors such as gender, use and misuse of other substances including other opioids appear to be important predictors of oxycodone misuse. Tailored interventions and risk-screening measures to optimize oxycodone prescribing might be key in limiting the misuse and diversion of tis pain medication.

KEYWORDS Oxycodone Misuse, National Survey of Health Use and Health, Risk Markers

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287. Analysis of Single Substance Heroin Exposures reported to the U.S. Poison Centers from Healthcare Facilities.

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Background: Heroin use has reached a public health crisis in the U.S. Since 2010, the rate for deaths involving heroin has almost tripled, from 1.5 per 100,000 in 2011 to 5.1 in 2016. The number of people using heroin for the first time in the U.S. has increased in the recent years. Hence it is important to track heroin overdoses, especially those reported from the healthcare facilities (HCF) as these may greatly increase resource use. The objective of the current study is to use outline the epidemiology of single substance heroin exposures reported to the National Poison Data System from the HCFs.
Methods: The NPDS was queried for all human single substance exposures to heroin reported to the U.S. Poison Centers (PCs) from HCFs between 2011 and 2017. We descriptively assessed the relevant demographic and clinical characteristics. Trends in heroin frequencies and rates (per 100,000 human exposures from HCF) were analyzed using Poisson regression methods. Percent changes were reported with the corresponding 95% confidence intervals (95% CI).
Results: There were 15,692 single substance heroin exposures reported to the PCs from HCFs. The number of calls increased from 1,142 to 3,865 during the study period. Among these calls, 90.2% were reported from acute care hospitals and emergency departments (EDs), 6.5% were reported from free standing EDs, while 3.3% were reported from physician offices. Acute exposures to heroin were responsible for 67.7% of the calls from HCF. Approximately 19% of the patients reporting such heroin exposures were admitted to the critical care unit (CCU), with 56.6% patients treated and released. Residence was the most common site of exposure (69.7%). Among the patients, m were male, with majority of the individuals between ages 20 and 39 years (70.9%). Pediatric cases accounted for 6.3% of the exposures. Intentional abuse (74.5%) and misuse (10.5%) were commonly observed reasons for exposure. During the study period, the proportion of heroin abuse cases increased (73.3% to 76.1%). Major effects were seen in 19.1% cases and the mortality rate for single substance heroin exposures from HCF was 1.8%. Notably, the number of heroin-related fatalities in this group doubled during the study period. Coma (26.8%) and respiratory depression (27.3%) were frequently observed clinical effects. Naloxone (60.7%) was the most frequently reported therapy. During the study period, the frequency of heroin exposures increased by 238.4% (95% CI: 215.8%, 261.5%; p
Discussion: There was a significant increase in single substance heroin exposures reported to the PCs from HCFs during the study period. This increase may be a result of lower cost of heroin and the tighter regulations on the prescribing of opioids. Changes in the sources of supply and potency of heroin products can result in substantial adverse events seen in the HCFs. Exposures reported to the poison centers further highlight the need for sustained, targeted, and multifactorial responses to the ongoing opioid epidemic, including timely surveillance.

KEYWORDS Heroin, Healthcare Facility, Intentional Abuse

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288. Epidemiology of Gabapentin Exposures using the National Poison Data System

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Background: Gabapentin prescriptions have by 64% between 2012 and 2016, in part due to the off label use for conditions like chronic pain. It was also one of the most commonly reported drug causing overdose deaths in the United States from 2011 through 2016. It has been noted that almost one-fifth of the patients who abuse opioids, also abuse gabapentin. The objective of the study was to describe the epidemiology of gabapentin exposures using a near real-time national poison center (PC) database.
Methods: The National Poison Data System (NPDS) was queried for all human exposures to gabapentin from 2012 to 2018 using the American Association of Poison Control Center (AAPCC) generic code identifiers. We descriptively assessed the relevant demographic and clinical characteristics. Gabapentin reports from acute care hospitals and emergency departments (EDs) were analyzed as a sub-group. Trends in gabapentin frequencies and rates (per 100,000 human exposures) were analyzed using Poisson regression methods. Percent changes from the first year of the study (2012) were reported with the corresponding 95% confidence intervals (95% CI).

Results: There were 122,810 gabapentin human exposure reported to the PCs from 2012 to 2018, with the number of calls increasing from 11,336 to 22,776 during the study period. Polysubstance exposures accounted for 65.2% of gabapentin exposures. Of the total gabapentin calls, the proportion of calls from acute care hospitals and EDs increased from 55.1% to 64.7% during the study period. Multiple substance exposures accounted for 75.4% of the calls from acute care hospitals and EDs. Approximately 21% of the patients reporting gabapentin exposures were admitted to the critical care unit (CCU), while 21.3% patients were treated and released. Residence was the most common site of exposure (94.5%), and 68.2% cases were enroute to the hospital when the PC was notified. Among the patients, 59.1% were females, with the majority of gabapentin exposures occurring between the ages of 40-59 years (33.5%). Suspected suicides (51.6%) was the most commonly reported reason for exposure. The proportion of such cases was higher in reports from acute care hospitals and EDs (71.5%). During the study period, the proportion of suspected suicides increased (46.8% to 54.4%) among gabapentin exposures. Major effects were seen in 5.5% cases and the case fatality rate was 0.4%. Notably, there was an approximately 2-fold increase in the number of deaths during the study period. The most frequently co-occurring substances associated with the cases were benzodiazepines (16.7%) and antipsychotics (10.7%). Tachycardia (16.1%) and hypertension (8.5%) were commonly observed clinical effects. During the study period, the frequency of gabapentin exposures increased by 100.9% (95% CI: 96.4%, 105.5%; p
Conclusions: Gabapentin exposures increased during the study period. Abuse and diversion of gabapentin may be as a result of its low cost and non-schedule status. Gabapentin has also been increasingly associated with suicidal ideation, the most common reason for exposure in our sample. Increasing prescriber awareness and better screening may be key to reduce such overdoses.

KEYWORDS Gabapentin, Overdose, NPDS

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289. Trends and Characteristics of Buprenorphine Sublingual Tablet Toxicities

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Background: The number of patients with an opioid use disorder in the U.S. was estimated to be approximately 2.6 million in 2015. Buprenorphine can be easily dispensed through office-based prescribers and community pharmacies, with 58% opioid treatment programs now offering buprenorphine. Buprenorphine sublingual tablets were discontinued in 2012 due to concerns about the misuse, abuse, and diversion. They were replaced with single dose sublingual films that are considered child-resistant and abuse deterrent. The objective of this study is to evaluate the trends, and characteristics of exposures to buprenorphine tablet formulations.
Methods: We retrospectively queried the National Poison Data System (NPDS) for all confirmed exposures to buprenorphine tablets from 1/1/2011 to 12/31/2016 as specified by the American Association of Poison Control Center Code (AAPCC) generic code and product name. We assessed the relevant characteristics of exposures descriptively. Frequencies and rates of buprenorphine tablet exposures (per 100,000 human exposures) were evaluated using Poisson regression methods, with the percent changes and corresponding 95% Confidence Intervals (95% CI) reported. Predictors of severe outcomes (major effects and death) to tablet exposures were also assessed with adjusted odds ratios (AOR) presented.
Results: Overall, there were 7,406 reports of exposures to buprenorphine sublingual tablets to the PCs during the study period. The reports of buprenorphine tablet exposures decreased from 1,780 to 468 during the study period, a decrease of 73.7% (95% CI: 64.1%, 78.7%; p
Conclusions: Analysis of national data from the NPDS exhibited a significantly decreasing trend in the exposures to buprenorphine tablets, with such exposures being frequent among children under 5 years of age. Considering the discontinuation of the sublingual tablets, it is imperative to explore in greater detail, the reasons for the observed exposures. Possible reasons for these observed exposures might be the continued availability despite discontinuation or potential diversion of the product.

KEYWORDS Bupernorphine Films, Severe Outcomes, Trends

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290. Pediatric Plant Exposures Reported to a Single State Poison Control System over a Fifteen Year Period

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Background: Five to ten percent of all human exposures reported to poison centers involve plants. Approximately 80% of these cases involve individuals younger than 6 years of age. The vast majority of these patients are asymptomatic and less than 7% present to a healthcare setting. We sought to further characterize pediatric exposures with moderate and major effects that were evaluated at a healthcare facility reported to our poison control system.
Methods: We performed a retrospective review of all plant exposures with moderate or major effects including death reported to our state poison control system over a fifteen year period. Cases were limited to patients aged less than 6 years who were managed at a healthcare facility. Data from January 2002 to December 2016 were extracted, yielding a total of 105 exposures. We included all exploratory and intentional exposures of plant material including teas. We excluded exposures to commercial preparations, such as supplements in pill form, pepper spray, and essential oils. Miscategorized exposures, such as plant-derived medications, favism, and food-related anaphylaxis were also excluded. This yielded 69 cases for further analysis.
Results: There were 69 exposures with moderate or major effects reported during this 15-year period. The identity of the plant was unknown in 19 cases. Of the cases with an identified plant, oxalate-containing plants were the most commonly reported with 12 cases. Antimuscarinic plants accounted for 7 cases, and 3 cases involved plants containing cardiac glycosides. Of the remaining cases, there were two or more exposures from the following plants: Euphorbia (3), Illicium anisatum (2), and Wisteria (2). Gastrointestinal (GI) effects were most commonly reported, occurring in 30 cases (43%). The most frequent GI effects were vomiting (73%), abdominal pain (20%), and nausea (13%). Exposure routes included oral (87%), dermal (9%) and ophthalmic (4%). The majority of cases were exploratory exposures (87%). Two deaths possibly related to plant exposure were reported during this time period. A four-year-old male died from fulminant hepatic failure thought to be induced by exposure to a traditional Chinese medicinal tea made from Bupleurum chinense (chai-hu) and Scutellaria baicalensis (Chinese skullcap) and possibly other plants. The other reported death was a one-year-old female who presented in PEA arrest after possible exposure to Euphorbia myrsinites. While the milky sap of Euphorbia myrsinites may cause skin and eye irritation, there are no reported cases of significant toxicity or deaths. It seems unlikely that plant exposure was related to the patient's death.

Conclusions: Most patients had a benign course with GI symptoms predominating. Exposures to oxalate-containing plants resulted in ocular and oral symptoms; however, none of these patients were intubated or experienced long-term sequelae. There were only three exposures to cardiac-glycoside-containing plants, and one of these patients was bradycardic. None had significant cardiac toxicity. This suggests that explorational exposures to these plants likely do not provide a sufficient toxic dose. Overall, pediatric plant exposures, especially explorational exposures, result in a low incidence of significant toxicity.

KEYWORDS Plant, Pediatric, Explorational Exposure

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291. A Little "Dab" Will Do Ya' In: A Case Report of Delirium and Respiratory Compromise Following Use of Cannabis Concentrates

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Background: Cannabis concentrates, also known as "dabs,""oil,""wax", and "budder," are formed by extraction of desired cannabinoids using solvents (frequently butane). Once the solvent evaporates, a "dab" of the concentrate is volatilized and inhaled using a delivery device ("dabbing"), producing a rapidly absorbed dose of highly concentrated THC. This is a novel presentation of "dabbing" which resulted in agitation requiring intubation, hypertension, and tachycardia.
Case Report: A previously healthy 17-year-old female was brought to the emergency department (ED) by her mother for altered mental status. Mother states that the patient was downtown with her boyfriend and his parents for New Year's Eve. Boyfriend's mother states that she thought the patient was having a seizure. Patient was taken home, and both parents agreed the patient seemed altered, so she was brought to the ED. Per mother, patient has a history of cutting and anxiety, and is prescribed olanzapine and escitalopram. Patient had an initial Glasgow Coma Score of 11 but was maintaining her airway. She was found to be tachycardic, hypertensive, have bilateral dilated pupils, and flushed dry skin. She was given a 1-liter bolus of normal saline, at which point her hypertension and tachycardia slowly resolved. She then developed bilateral periorbital, upper lip, and tongue swelling, and the decision was made to intubate. She was given 0.3 mg of epinephrine. Her urine drug screen came back showing positive for her normal meds, as well as marijuana and alprazolam. The patient's tetrahydrocannabinol (THC) was found to be 61 ng/mL and the 11-nor-9-carboxy-Δ9-tetrahydrocannnabinol (THCCOOH) was 366 ng/mL. The patient was transferred to the PICU for continued monitoring. She was extubated the next day, then transferred to the pediatric floor for further management with a safety attendant. She continued to become more alert and oriented, and it was discovered that the substances taken prior to hospitalization were a marijuana dab and one "Xanax bar" (2 mg alprazolam). Patient was discharged on the second day of hospitalization. Instructions included following up with psychiatric clinic for medication management and behavioral therapy.
Discussion: Our patient presented with vital signs suggestive of catecholamine surge and agitation consistent with clinical sequelae of THC-induced toxicity. Dose-dependent relationships between THC exposure and both tachycardia and hypertension have been reported, and several studies describe marijuana-induced vasospasm causing myocardial and cerebrovascular ischemia as well. These effects may be related to alterations in serotonin and catecholamine neurotransmission from high total dose of THC and to an increased THC:CBD ratio in cannabis products. Highly concentrated "dabs" can result in significant toxicity.
Conclusions: Given the increasing use of cannabis concentrates, physicians should be aware of the potential neurological and cardiovascular effects which may result from "dabbing."

KEYWORDS Cannabis, concentrates, dab

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292. Grand GLP-1 Glimpse

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Background: Glucagon-like peptide-1 (GLP-1) agonists are a class of medication with a short history, prolonged half-life, and little is known about their toxicological profile in overdose. Mechanistically, GLP-1 agonists mimic the proglucagon compound incretin produced in the enteroendocrine L cells of the gut that are secreted into the blood stream when food containing fat, protein hydrolysate, and/or glucose enter the duodenum.[1] The presence of incretin in the blood activates GLP-1 receptors that stimulate pathways to induce downstream effects enhancing glucose-induced insulin secretion. Additionally, these medications inhibit dipeptidyl peptidase-4 (DPP-4), the enzyme that breaks down endogenous GLP-1, inhibits glucagon secretion and gastric emptying, and prolongs satiety.[2] GLP-1 agonists are presumed to have a low risk of hypoglycemia since they require the presence of glucose to stimulate insulin release.[3] Recommendations for 24 hours of observation with frequent blood glucose monitoring have been suggested until the toxicokinetic profile is better understood. This is the first published report of a four-fold overdose of dulaglutide, and was not associated with clinically significant hypoglycemia.
Case report: An 81-year-old man with a past medical history of non-insulin dependent diabetes mellitus presented to the emergency department after injecting himself with 6 mg dulaglutide, four times his normal dose. The patient was started on the medication about 1 month prior to this incident and noted a burning sensation with prior injections. He reported repeating the dosing until he felt the usual burning sensation. About 5 minutes after injecting the extra doses, the patient began experiencing abdominal pain, headaches, dizziness, shaking, and nausea. He went to the hospital and was admitted and observed for 33.5 hours; he received a normal diet and no IV glucose. The patient reported resolution of his symptoms prior to presentation to the hospital. During his stay he reached a glucose nadir of 111 mg/dL at 8 hours after his overdose. His maximum glucose during this admission was 137 mg/dL.
Conclusion: Historically, drugs that promote the release of insulin are associated with critical hypoglycemia in overdose; however, currently there is no information concerning the onset or duration of hypoglycemia for GLP-1 overdoses. [4] This single case report found no clinically significant hypoglycemia induced by a four-fold overdose of dulaglutide.

KEYWORDS GLP-1, Dulaglutide, Overdose

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293. Daily Emergency Department Pharmacy Rounds Improves Medication Safety for Behavioral Health Patients Boarding in the Emergency Department

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Background: Our previous research showed ED nurses and physicians perceive behavioral patients with prolonged ED boarding are at risk for medication errors. In response, multi-disciplinary daily rounds led by ED Pharmacy were initiated to determine if our local model is one feasible solution for a growing national ED boarding problem.
Methods: Four months after initiating ED Pharmacy led multi-disciplinary rounds that include physicians, nurses, techs, social work, and psychiatry, a closed-format survey instrument developed by experts in operations, behavioral health, and safety was administered. The instrument was piloted, revised, then sent electronically over a two week period. Eligible subjects were all ED nurses and attending physicians at an urban academic hospital with >130,000 annual visits; participation was voluntary, anonymous, confidential, and approved by our IRB. Data collected included basic patient demographics and staff perceptions about behavioral health patients boarding in the ED.
Results: Participants included 52 nurses and 25 physicians (response rate 81%). Patients boarding in the ED are 77% male with an average age 40.9 years (range: 20-70). Most staff (nurses 98%, physicians 100%) think ED boarding >24hours is "sometimes" or "always" a problem in behavioral health patients, with drug or alcohol use considered a problem by 94% nurses and 100% physicians. After initiating daily ED Pharmacy rounds, 100% physicians and 82% nurses report medication safety has improved. Nurses think physician awareness of patient medication needs improved from 70% to 84%. Previously 90% reported patients did not receive their usual non-psychiatric medications (e.g., diabetes, hypertension) while boarding; now most (74% nurses, 88% physicians) think patients "sometimes" or "always" receive their non-psychiatric medications Although most physicians (64%) reported transitions of care improved, 24% were not sure.
Conclusion: Nurses and physicians perceive improvements in medication awareness, medication safety, and transitions of care with daily rounds led by ED Pharmacy. Since prolonged boarding has reached epidemic proportions, our model can be used in other EDs to enhance medication safety. Our next steps include enhanced clinical pharmacy integration in the ED, assessment of specific medication needs and length of stay, and further study of substance abuse complications in patients boarding in the ED.

KEYWORDS Behavorial health, medication safety, comprehensive medication mangement

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294. Unit-Dose Laundry Pods: And The Research Continues

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Background: Accidental pediatric exposures to laundry detergent pods (LDP) have been associated with a variety of clinical effects, ranging from minor irritation and nausea/vomiting to lethargy, respiratory distress and even rare fatalities. Ocular irritation and corneal abrasions have also occurred. Previous studies have indicated that exposure rates have decreased since implementation of access prevention strategies. Management in the home has become more frequent, but the appropriate length of observation time is still unknown. The number of follow-up calls and their timing vary among poison specialists. This study examines the symptoms experienced by children exposed to LDPs.
Objective: To determine the observation time necessary to identify the development of potentially significant symptoms for children with acute exposures to LDPs.
Methods: A retrospective study of LDP pediatric exposures reported to one poison center over a 6 year period (January 2012 – December 2017) was performed. Inclusion criteria consisted of children (birth to 18 years old) exposed to any brand of laundry detergent pods. Exposures that took place in other states and exposures involving additional substances were excluded. Data were collected by reviewing narrative descriptions of the exposures and included route(s) of exposure, symptoms, time to onset of symptoms, interventions, medical outcomes, time to resolution of symptoms (if known) and patient management sites.
Results: A total of 500 pediatric LDP exposures were reviewed. As previously reported, ingestion and ocular were the major routes of exposure (68% and 7%, respectively). Symptoms developed in 349 cases (70%), but the majority of these (57%) were characterized as minor effects. There were no deaths reported. The time to onset of symptoms varied. Minor symptoms occurred relatively soon post-exposure; these included vomiting, eye irritation or redness, and skin irritation. The majority of the clinically significant symptoms were seen within 2 hours. Lethargy occurred within 2 hours in 92 % of the cases showing central nervous system depression (7 % of the total cases). Respiratory distress occurred in 4 cases, or 0.8 % of total exposures and 75% of those exhibited this finding within 30 minutes (1 unknown). Corneal abrasions (2% of total cases) took the longest time to get diagnosed, but ocular irritation preceded this finding.
Limitations: It was difficult to ascertain onset and resolution of symptoms in some instances due to lack of follow-ups. Some of the cases were not followed for several hours, so timing of resolution of symptoms could not be determined.
Conclusion: Based on this study's findings, it appears that LDP exposures can be safely managed with a 4 hour observation time, as all major symptoms (with the exception of a definitive diagnosis of corneal abrasion) were seen during that time frame.

KEYWORDS Laundry detergent pods, unintentional pediatric exposures, time to symptom onset

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295. Acquired Coagulopathy Secondary to Brodifacoum-laced Synthetic Cannabinoids: A Case Series

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Background: Brodifacoum, a 4-hydroxycoumarin "superwarfarin" pesticide, is a potent inhibitor of Vitamin K epoxide reductase. Exposure induces hemorrhages as Vitamin K-dependent coagulation factors deplete. Being tasteless and odorless, and able to pass through skin and mucous membranes, it is readily implicated in cases of intentional poisoning, such as through recreational drugs. Treatment entails Vitamin K supplementation for prolonged periods due to brodifacoum's extended half-life. Several cases of brodifacoum poisoning have recently been identified by our treatment center secondary to synthetic cannabinoid receptor agonist (SCRA) use.
Case Series: To date, seven cases of life-threatening hemorrhages after using synthetic cannabinoids laced with brodifacoum have been identified in a midwestern state by the regional poison center. Two cases presented to our institution for a full evaluation and treatment. Laboratory assessment for both patients revealed undetectably high PTT and PT/INR values. Mixing studies corrected fully, and coagulation factor testing revealed Vitamin K-dependent factor deficiencies. High-dose Vitamin K administration led to rapid resolution of both the coagulopathy and hemorrhages. On toxicology analysis, high-performance liquid chromatography and tandem mass spectrometry identified brodifacoum in both patients. Untargeted liquid chromatography quadrupole time-of-flight mass spectrometry toxicology analysis identified the urinary metabolite of SCRA AB-FUBINACA (metabolite 3) or (N-[[1-[(4-fluorophenyl)-methyl]-1H-indazol-3-yl] carbonyl]-L-valine), confirming the diagnosis. Both patients admitted to using synthetic cannabinoids prior to presentation. With high-dose oral Vitamin K and frequent outpatient follow-up, both patients were safely discharged from the hospital.
Case Discussion: Our patients demonstrated brodifacoum toxicity with life-threatening hemorrhages acutely after brodifacoum exposure. Factor testing identified a Vitamin K-dependent coagulopathy and an anticoagulant poisoning panel detected brodifacoum, confirming Vitamin K antagonist toxicity. Synthetic cannabinoid metabolites were detected in the urine of both patients. Several brodifacoum coagulopathy cases have arisen in the Missouri area in patients who have used SCRAs. All obtained SCRA products from the Chicago area. Any patient with SCRA use and unexplained hemorrhages with coagulopathy may develop a Vitamin K-dependent coagulopathy. Rapid Vitamin K replenishment reverses the coagulopathy. Given brodifacoum's half-life, long-term oral Vitamin K therapy and coagulation monitoring are needed. Therapy continues until complete normalization of the coagulopathy.
Conclusion: Brodifacoum toxicity causes hemorrhages due to Vitamin K-dependent coagulation factor loss. Treatment entails Vitamin K supplementation and coagulation factor replacement. Providers should maintain a high index of suspicion for brodifacoum toxicity in patients presenting with unusual bleeding and coagulopathy without a clear etiology.

KEYWORDS Synthetic cannabinoids, brodifacoum, AB-FUBINACA M3 metabolite

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296. Evaluation of pediatric nicotine product exposures reported to the Florida Poison Information Center Network

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Background: Nicotine exposures in children are a common encounter traditionally linked to legacy nicotine products (LNPs). Electronic cigarettes (e-cigarettes) and liquid nicotine preparations (e-liquids) are a new source for these exposures. With nicotine concentrations as high as 42 mg/mL and availability in an assortment of flavors, these concentrated nicotine products pose a significant risk for toxicity in both accidental and intentional exposures in pediatric populations. Nicotine toxicity can cause nausea, vomiting, tachycardia, hypertension, paralysis, fasciculations, arrhythmias, seizures and possibly death. Despite FDA regulatory efforts (July 2016), there was a 582.6% increase in e-cigarette use by Florida students ages 11-17 from 2012 to 2018. This study sought to characterize nicotine exposures in pediatric populations within the State of Florida based on exposure data gathered from the Florida Poison Information Center Network (FPICN).
Methodology: This was a retrospective chart review of nicotine exposures in children under 19 years old reported to the FPICN between August 2014 and August 2018; evaluating for exposure, route, reason, clinical effects, medical outcomes, and management site. Exposures secondary to e-cigarettes, e-liquids, and e-cigarette cartridges were defined as e-nicotine related and were compared to reported

Table 1 Most Common Clinical Effects.

Effect	Total – n	E-nicotine Product Exposures – n (%)	Legacy Nicotine Product Exposures – n (%)	p-value
Any Clinical Effect	455	119 (27.23%)	336 (26.65%)	0.8117
Vomiting	351	71 (16.25%)	280 (22.20%)	< 0.008*
Nausea	54	27 (6.18%)	27 (2.14%)	< 0.001*
Dizziness/Vertigo	20	14 (3.20%)	6 (0.48%)	< 0.001*
Cough/Choke	18	4 (0.92%)	14 (1.11%)	0.999**
Agitation	16	3 (0.69%)	13 (1.03%)	0.7743**
Diaphoresis	16	7 (1.60%)	9 (0.71%)	0.0584**
Tachycardia	16	6 (1.37%)	10 (0.79%)	0.2639**
Other/Miscellaneous	15	6 (1.37%)	9 (0.71%)	0.2347**
Pallor	12	4 (0.92%)	8 (0.63%)	0.2015**
CNS Depression (Mild)	9	2 (0.46%)	7 (0.56%)	0.2977**
Drowsiness/Lethargy	9	2 (0.46%)	7 (0.56%)	0.2977**
Irritation/Pain (Ocular)	9	8 (1.83%)	1 (0.08%)	0.0001**

*Chi-Square Tes.t
**Fischer's Exact Test.

Table 2 Medical Outcomes in Patients Followed to a Known Outcome.

	Followed Medical Outcomes (n = 977)			
Product Type	Minor, Moderate, or Major Effect – n (%)	No Effect – n (%)	P-Value	OR (95% CI)
E-Nicotine Products (n = 313)	93 (29.71%)	220 (70.29%)	0.0071*	0.674 (0.505-0.899)
Legacy Nicotine Products (n = 664)	256 (38.55%)	408 (61.45%)		

*Chi-Square Test.

Table 3 Calls for Exposures to Nicotine Products Before and After FDA Mandates on July 26, 2016.

Product Type	Pre	Post	P-Value
E-Nicotine Products (n =437)	234 (53.55%)	203 (46.45%)	0.0479*
Legacy Nicotine Products (n = 1261)	606 (48.06%)	655 (51.94%)	

*Chi-Square Test.

exposures of all other LNPs (cigarettes, chewing tobacco, nicotine gum, etc.).
Results: Of the 1698 nicotine exposures included in this study, 437 were secondary to e-nicotine products. Patients with e-nicotine exposures were significantly older than patients exposed to LNPs (median [IQR], 2 [1.25-2] vs. 1 [0.83-1.5] years, p
Conclusions: Though there was no difference in the incidence of all clinical effects reported, the most frequently reported clinical effects (vomiting, nausea, dizziness/vertigo) were significantly higher in the LNP group. Patients exposed to LNPs were significantly more likely to experience medical outcomes with a minor, moderate, or major effect. There was no difference in the frequency of hospital or critical care admissions between the two groups. Finally, our results suggest that exposures reported to poison centers involving e-nicotine products in Florida did decrease following government regulation of these products and their accessories.

KEYWORDS Electronic cigarettes, nicotine, pediatrics

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297. Temozolomide-Induced Pancytopenia in a Toddler

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Background: Temozolomide is an alkylating chemotherapeutic known to cause hemopoietic toxicity. There are no reports of intentional overdose or accidental ingestion in the literature. We present a case of suspected temozolomide ingestion resulting in severe pancytopenia.
Case Study: This is a single patient case report. A previously healthy 23 month-old girl was brought to the emergency department with pulseless electrical activity. The patient was reportedly in her normal state of health until one day prior to presentation when decreased activity was noted. Cardiopulmonary resuscitation was performed for 1.5 hours until return of spontaneous circulation was obtained. She was intubated, placed on mechanical ventilation, and started on vasopressors for continued hypotension. Neurologic exam was significant for decerebrate posturing. Initial laboratory studies were significant for an undetectable hemoglobin level, leukopenia, and thrombocytopenia. There was no evidence of hemolysis. Chest x-ray revealed cardiomegaly. The patient was transfused with 10 cc/kg of packed red blood cells with improvement of blood counts. Urine gas chromatography-mass spectrometry was positive for levetiracetam and caffeine. Levetiracetam had been administered prior to obtaining the urine sample. Mitochondrial DNA testing, targeted exome sequencing, and chromosome single nucleotide polymorphism (SNP) array testing did not reveal any genetic abnormalities as the cause of hematopoietic failure. Hematology was consulted and could not identify a clear etiology of this patient's pancytopenia. Brain magnetic resonance imaging (MRI) demonstrated evolving restricted diffusion through the caudate, putamen, and thalamus, as well as diffuse supratentorial white matter injury and supratentorial hemorrhagic conversion. Electroencephalogram (EEG) demonstrated severe global attenuation without normal features of sleep or wakefulness. The family had no pharmaceuticals at home except for one bottle of a chemotherapeutic, temozolomide, which the patient's father had been prescribed for an astrocytoma approximately four weeks prior to this patient's presentation. He had only taken a single dose and had not discarded the remainder of the medication. Multiple inquiries to the drug manufacturer were made to find an assay to detect temozolomide and its metabolites, but there were no tests available. The patient did not recover neurologically from her initial hypoxic-anoxic injury. She underwent tracheostomy, gastric tube placement, and was discharged to foster care.
Discussion: The differential for acquired aplastic anemia is broad and requires extensive review of accessible xenobiotics. Hematologic abnormalities secondary to alkylating agents may occur several weeks after exposure. The clinical time course, short half-life of offending agents, and lack of available assays may preclude a definitive diagnosis. Hemopoietic toxicity is a known complication in children treated for central nervous system tumors receiving dosing based on body surface area. This patient was exposed to a potentially much larger dose since it was prescribed for an adult. There are no other reported cases of exploratory ingestion of temozolomide to our knowledge.
Conclusions: Temozolomide exposure in a pediatric patient can be associated with aplastic anemia. Ingestions should be monitored with scheduled laboratory testing due to delayed effects.

KEYWORDS Temozolomide, aplastic, pediatric

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298. Adding a Written Guideline to Verbal Consultation Decreases NAC Medication Administration Errors in APAP Overdose Care

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Background: Acetaminophen (APAP) toxicity is one of the most common causes of poisoning; in 2017 over 108,000 human exposures reported in the National Poison Data System (NPDS) involved an APAP product. Furthermore, 22,793 patients were treated with intravenous (IV) NAC. Previous research has shown that the complex IV 3-bag NAC dosing regimen is associated with medication errors in 33-41% of cases, including some cases with multiple errors. The goal of this study was to determine if the provision of a written guideline in addition to poison center verbal consultation would decrease IV NAC medication administration errors.
Methods: A written guideline on NAC administration was developed by a multi-disciplinary poison center team. From October 1, 2016 to October 1, 2017, the written guideline (in addition to traditional verbal advice) was faxed to the providers of patients who were treated with IV NAC. These cases were reviewed for NAC medication errors, and compared to baseline error rates presented at a previous NACCT. Medication errors were defined as: delayed initiation of therapy, unnecessary treatment, incorrect dose, incorrect rate, therapy inappropriately discontinued, and interruptions of therapy >1 hour. Each chart was reviewed by a single investigator, and actual treatment was compared with poison center NAC guidelines. Faxing of the NAC guideline was captured in the free text and a free area checkbox of the poison center record.
Results: 313 cases were initially reviewed. Of those, 43 were excluded due to coding errors and 29 patients either received oral NAC or NAC was eventually not given, leaving 241 cases for analysis. A total of 78 errors (range 1-3 errors per patient) occurred in 68 (28%) of the 241 cases in which both verbal and written information was provided. Rates for each type of medication error are provided in the attached table, with comparisons to baseline (verbal instructions only) error rates. The total number of errors that occurred in cases coded as major outcome or death also declined compared to baseline data: 21 errors in 55 cases (38%) v 55 errors in 95 cases (58%), respectively.
Conclusions: The provision of a written guideline in addition to verbal consultation resulted in an overall 12.8%, and 32% relative, reduction in the incidence of medication errors associated with IV NAC therapy.

The biggest impact appeared to be in avoiding inappropriate use of NAC and reducing delays in therapy initiation. Challenges remain with getting NAC infusions from pharmacy to the bedside, and with inter-facility transfers. Additional research is needed to evaluate the potential impact of simpler dosing regimens.

Error Type	Error Rates		
	Verbal Only[#]	Verbal & Written	p-value[*]
Delayed initiation	18.9%	9.5%	0.0045
NAC not indicated	10.8%	0.4%	<0.0001
Incorrect dose	1.6%	0.4%	0.1699
Incorrect infusion rate	2.8%	1.7%	0.3702
Interruption in therapy	19.7%	14.1%	0.1058
Premature discontinuation	7.8%	4.6%	0.0631
Overall error rate	40.8%	28.2%	0.0064[‡]

[#]Data from previous study (NACCT 2017).
[*]Using the Pearson's Chi-square test.
[‡]Using a Fisher's Exact test.

KEYWORDS Acetylcysteine, Medication Errors, Written Guideline

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299. Severe Cardiotoxicity and Hypocalcemia from Chronic Inhalation of 1,1-Difuoroethane

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Introduction: Fluorinated hydrocarbons are used recreationally for their intoxicating properties but have significant side effects including "sudden-sniffing death", myocarditis, cardiomyopathy, and renal failure. The keyboard cleaner, Dust-Off®, contains 1,1-difluoroethane and has been associated with these complications. Some halogenated fluorocarbons release hydrofluoric acid when aerosolized and subsequently cause hypocalcemia. 1,2-difluorethane, a related analog, can also cause severe hypocalcemia through a separate mechanism, the creation of fluorocitrate.
Case Study: A 30-year-old man presented with persistent dyspnea after directly inhaling 16 cans of Dust-Off®, which he had been using daily for a few weeks. He was initially treated with lorazepam, methylprednisolone, and racemic epinephrine then transferred to our center where he was tachycardic (130 bpm), tachypneic, and saturating at 99% on room air. A chest x-ray showed no infiltrate, pneumomediastinum, or pneumothorax. He had a grossly abnormal EKG with deep, narrow, inverted T waves and a prolonged QTc, 609 ms. His initial ionized calcium was 0.56 mMol/L and he received 3 grams of IV calcium gluconate. Creatinine was elevated at 2.3 mg/dL. He had a progressive rise in his troponin from 0.15 ng/mL on admission to a peak of 1.04 ng/mL. He received intermittent metoprolol (total of 25 mg in first 24 hours) with improvement in his symptoms. Over four days his QTc gradually improved. An initial echocardiogram showed a dilated left ventricle with diffuse mild hypokinesis and a decrease in his ejection fraction (EF: 45-50%). A cardiac MRI (CMR) was later performed which demonstrated persistent hypokinesis and decreased EF. There was no infiltrative disease, inflammatory process, or infarct identified on CMR. He was discharged on hospital day 5 with improved symptoms, but persistent T wave abnormalities.
Discussion: Chronic exposure to fluorinated hydrocarbons can cause myocarditis and cardiomyopathy. Radiographic and laboratory findings include diffuse hypokinesis on echocardiogram, acute kidney injury, rhabdomyolysis, and troponin elevations. The proposed mechanism of cardiomyopathy is attributed to catecholamine excess in the setting of myocardial sensitization. To prevent the development of malignant dysrhythmias and prevent further myocardial damage, we administered metoprolol prophylactically and therapeutically. Additionally, this patient had severe hypocalcemia, which has not previously been documented in case reports of 1,1-Difluoroethane toxicity and is likely from fluoride disassociation and chelation of calcium in tissues as it is not metabolized to fluorocitrate.
Conclusion: Chronic exposure to 1,1 difluorethane can lead to a cardiomyopathy without any structural changes on CMR as well as acute kidney injury and severe hypocalcemia. Treatment consists of replacement of electrolytes and beta antagonist therapy to prevent further myocardial injury and minimize the risk of dysrhythmias.

KEYWORDS Hydrocarbons, Cardiomyopathy, 1,1-Difluoroethane

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300. Primed for Danger: Dermal Burns from Methacrylic Acid

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Background: Methacrylic acid is a highly corrosive organic carboxylic acid often used commercially as an artificial nail polish primer. Household use is common and products are widely available. We present a case of a pediatric exploratory exposure to methacrylic acid resulting in orofacial burns.
Case Study: A two-year-old girl with eczema presented with facial burns after she was inadvertently splashed with an estimated two tablespoons of her mother's nail primer containing methacrylic acid. The patient's mother immediately irrigated her eyes however the patient developed abdominal pain and vomiting which prompted healthcare evaluation and monitoring. The patient had second degree facial burns and periorbital edema but no respiratory symptoms or oral burns. Ocular pH was 7.0 on ED testing. She never developed airway swelling, had resolution of her abdominal pain, and tolerated advancement of her diet. Ophthalmology evaluation revealed chemical conjunctivitis which was treated with topical erythromycin. She developed impetigo from the superficial burns over eczematous portion of her face and was treated with mupirocin and triamcinolone.
Discussion: While not commonly reported, methacrylic acid can cause significant dermal, mucosal, ocular and pulmonary damage from caustic effects. These injuries are often found in pediatric populations given the commercial availability of methacrylic acid and exploratory exposures. Our patient did well with early copious irrigation of her eyes and supportive therapy, and it is unlikely that she ingested or aspirated methacrylic acid. Monitoring of these patients is important especially if oral ingestion is uncertain as the extent of mucosal injury may not be readily apparent.
Conclusion: We describe a case of dermatitis from methacrylic acid. Although commonly available, this is not a frequently reported chemical cutaneous exposure and it is important to be aware that significant injuries can occur from exposure.

KEYWORDS Methacrylic, Burn, Pediatric

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301. Morbidity and Seizures Associated with eCigarette Exposures

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Objectives: In April 2019 the US Food and Drug Administration (FDA) reported an association between e-cigarette use and seizures (especially for youth or young adults) and encouraged the report of such cases.

Methods: We extracted single substance exposure data from the National Poison Data System (NPDS) for the 15 generic codes (GCs) associated with Tobacco/Nicotine/eCigarette Products including eCigarettes: Nicotine Containing (n=6) and Miscellaneous Tobacco Products (n=9) for 1-Jan-2010 through 8-Apr-2019. We mapped the 15 GCs into 3 categories (eCigarettes, Nicotine eCigarette refills, and Cigarettes (including cigars); Medical Outcome into Total and Serious (Death, Major and Moderate); Clinical Effects of Seizures (single, multi/discrete, or status); and Route (Inhaled=Inhalation/nasal or Any) We defined the Morbidity Index (MI=1000 * Serious / Total Exposures) and calculated the Relative Risk compared to the Cigarette exposures. We examined change over time via linear and quadratic regression. StatsDirect (3.1.22) provided Forest plots and relative risks with 95%

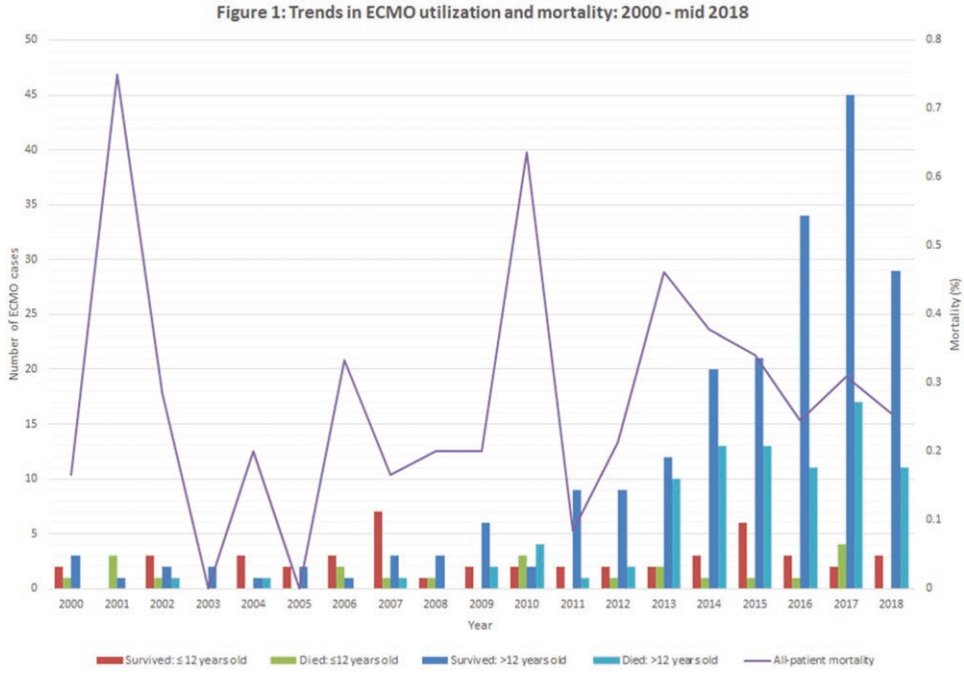

Figure 1 Trends in ECMO utilization and mortality: 2000-mid 2018.

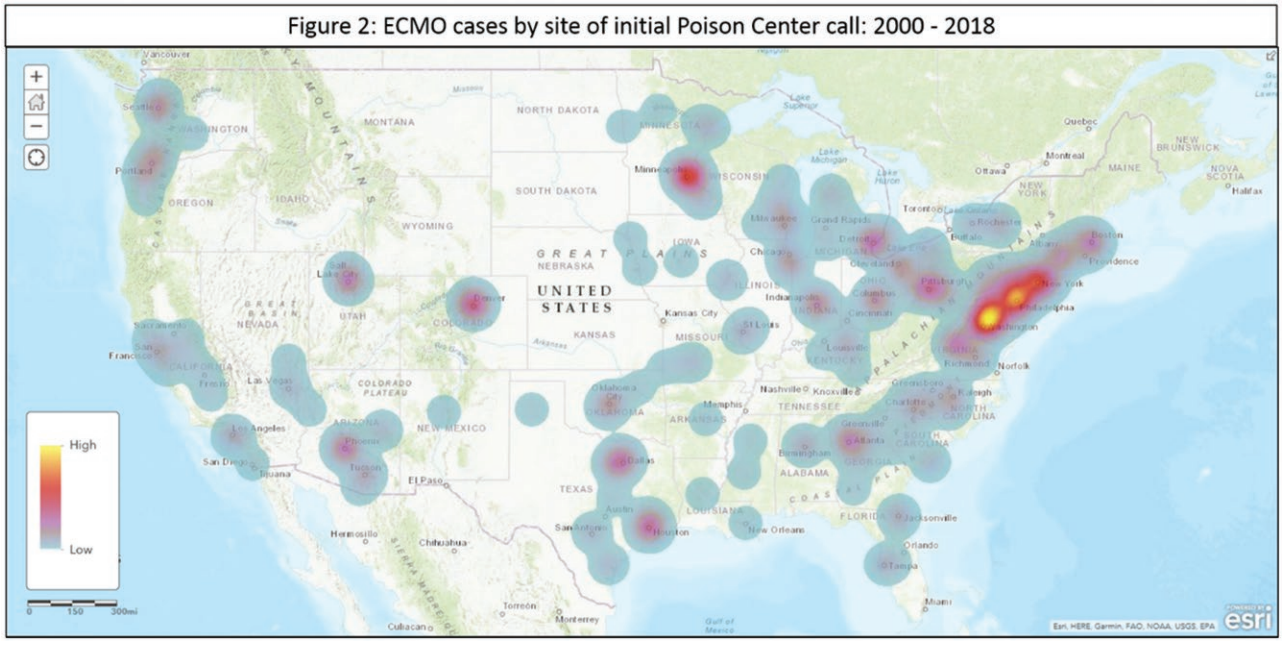

Figure 2 ECMO cases by site of initial Poison Center call: 2000 - 2018.

Table Patients Dichotomized by Mortality.

	Survived	Died
Number of cases receiving ECMO	253 (69.9%)	109 (30.1%)
Age	25.8 +/- 17.8	28.5 +/- 19.6
Male gender (%)	131 (51.8%)	64 (58.7%)
Single ingestion cases (%)	132 (52.2%)	57 (52.3%)
Multiple ingestion cases (%)	121 (47.8%)	52 (47.7%)
Substances ingested, median (IQR, range))	3 (2-4, 2-16)	3 (2-4, 2-12)
Selected clinical effects (related)		
Asystole	18 (7.1%)	31 (28.4%)
Cardiac arrest	47 (18.6%)	58 (53.2%)
Conduction disturbance	51 (20.2%)	22 (20.2%)
Dysrhythmias (vtach/vfib)	27 (10.7%)	15 (13.8%)
Respiratory arrest	24 (9.4%)	38 (34.9%)
Selected therapies (performed)		
Antiarrhythmic	23 (9.1%)	17 (15.6%)
CPR	59 (23.3%)	44 (40.4%)
Pacemaker	18 (7.1%)	11 (10.1%)
Vasopressor	177 (70%)	89 (81.7%)
Single-ingestion case substances (total)*, #		
Hydrocarbon (28)	20	8
Calcium channel blocker (18)	13	5
Unknown (16)	10	6
Antiarrhythmic (14)	10	4
Antidepressant (11)	6	5
Mitochondrial poisons (11)	7	4
CNS Depressants (10)	6	4
Opioid (10)	10	0
Other chemical (9)	5	4
Corrosive (7)	6	1
Antihistamine (6)	5	1
Stimulant (6)	5	1
APAP (4)	2	2
Other OTC (4)	2	2
Other drugs of abuse (4)	4	0
Antimicrobial (3)	2	1
Beta-adrenergic blocker (3)	3	0
NSAID (3)	3	0
Psychotropic (3)	3	0

*2 cases each: insects, plants, salicylates, unknown non drug, anticonvulsants, opioid with APAP.
#1 case each: Antineoplastic, asphyxiant, diabetic agent, heavy metal, toxic alcohol, foreign body, paralytic.

Confidence Intervals (CI); SAS JMP (12.0.1) provided data handling, descriptive statistics, graphs and regressions.

Results: Exposures for 2010-2019 numbered 112,095 including 2,742 Serious with an overall MI of 24.5. MI [95% CI] was 34.9 [31.8, 38.2] for eCigarettes compared to 10.1 [9.30, 11.0] for Cigarettes. A total of 58 seizures were reported including 32 (55%) where the route was Inhaled. Since no 2 seizures occurred on the same day, we examined the 58 cumulative seizures over time which increased at an increasing rate, Cume Seizures = -16703 + 8.30*Year +0.653*(Year-2016.4)^2. Quadratic regression Rsquare =0.983, with a 2nd order coefficient =0. 653 [0.558, 0.748], p

Conclusions: These data demonstrate an increase in the numbers of seizures reported in association with eCigarettes. Our analyses show a much greater hazard for eCigarettes and the liquid Nicotine refills compared to Cigarettes, especially in teens, supporting the FDAs concerns. While the RR for seizures by inhalation 95% CI excluded 1, the RR for seizures by any route was larger. These findings suggest further restrictions in distribution of eCigarettes and Nicotine refills and other measures to reduce the hazard to should be considered.

KEYWORDS eCigarette exposure, Seizures, Morbidity Ratios

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302. Characteristics of Nebulized Naloxone Use in an Academic Emergency Department

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Background. The need for versatility of naloxone administration has been well recognized in opioid toxicity. Intravenous administration is crucial for these clinical scenarios, yet it may increase risk for uncomfortable withdrawal symptoms and refusal of care. Establishing intravenous (IV) access may be challenging in some cases or require time and resources that are limited. For patients not at risk of apnea, nebulization is noninvasive and offers some flexibility for more gradual reversal. There is evidence that nebulized naloxone may decrease the need for supplemental oxygen, improve patient sedation and agitation, and minimize withdrawal symptoms without the need for subsequent naloxone administrations. However, extensive research is lacking and more investigation is needed to help discern its appropriateness in practice. The purpose of this evaluation was to describe the demographic and clinical characteristics of patients administered nebulized naloxone at an academic emergency department.

Methods: This was a retrospective medical chart review at a single, urban academic emergency department of patients aged >17 years who received nebulized naloxone between 06/2012 and 04/2018. Data were collected from an electronic medical record including patient demographics, reported opioid involved, IV access, level of care received after administration, vital signs at the nearest available time pre and post-naloxone, positive or negative response to nebulized naloxone based on staff documentation, additional naloxone doses and routes, Glasgow Coma Scale (GCS), and end-tidal CO2. Descriptive statistics were performed using STATA® (v.15). The study was approved by the university's institutional review board.

Results: There were 133 patients who received nebulized naloxone during the study period. The number of administrations per year increased from 11 in 2013 to 38 in 2017. The most common dose was 2 mg (87%), range 0.1-2 mg. Most patients were male (62%) and aged 46-70 years (48%). The most frequently reported opioid was hydrocodone and the most common reason for exposure where known (n = 101) was use to attain a high (67%). Non-opioid co-exposures were reported in 47% of cases. For 118 patients where IV access was documented, 47% did not have IV access at the time of the nebulization. In 75% of cases, the pre-nebulization oxygen saturation was 95% or higher and respiratory rate was greater than 13. GCS was recorded as 13-15 in 61% of cases (n = 106). Nearly half of patients (47%) were ultimately discharged from the ED while an additional 4 patients left against medical advice. Positive response was noted among 69% of charts where documented (n = 98). After the initial nebulization, 65.4% did not receive any further naloxone, 27.1% received naloxone by other routes, and 7.5% received additional naloxone by nebulization only.

Conclusions: Our findings suggest that nebulized naloxone use at the study site increased over time. Use was frequent in patients with short-acting opioid exposure due to suspected use to attain a high. Most patients presented without significantly impaired oxygenation or mentation. A majority of ED staff noted a positive subjective response to naloxone. Further studies are needed to elucidate effectiveness and safety of this intervention.

KEYWORDS Naloxone, opioid toxicity, nebulization

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303. Identifying Laboratory Factors that Differentiate Alcoholic Ketoacidosis from Toxic Alcohol Ingestion

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Background: Alcoholic ketoacidosis (AKA) is a metabolic derangement caused by poor nutritional status and an altered oxidation-reduction state in patients with alcohol use disorder (AUD). During starvation, fatty acids undergo beta-oxidation, with resulting ketone and ketone-like byproducts causing both an elevated osmolar gap and an elevated anion gap metabolic acidosis. Ingestion of toxic alcohols (TAs), such as methanol or ethylene glycol, also produces an elevated osmolar gap, and subsequently an elevated anion gap metabolic acidosis. It is difficult to distinguish AKA from TA ingestion clinically, many hospitals do not provide timely serum TA concentrations, and the cost of unnecessary fomepizole and/or hemodialysis is significant. The aim of this study is to identify risk factors suggestive of AKA when TA ingestion is the primary alternative differential diagnosis. We hypothesize that a positive ethanol concentration will be predictive of the diagnosis of AKA.
Methods: This is a retrospective analysis of data from a single Poison Control Center (PCC) from 2000 to 2019. A structured query language search (SQL) of Toxicall© records initially identified all cases a) coded as methanol or ethylene glycol or b) coded as alcohol or ethanol with acidosis. Inclusion criteria for AKA were all three of the following: AUD; urine or serum ketones or an elevated beta-hydroxybutyrate; anion gap ≥14 mEq/L. AKA exclusion criteria: patients who received insulin for presumed diabetic ketoacidosis or had hemodialysis before obtaining a clear diagnosis. The only inclusion criterion for TAs was a positive concentration. Logistic regression was performed using univariate and multivariate analysis to obtain odds ratios with SPSS v25.
Results: During the study period, 699 patients were screened for inclusion. Of these, 39 patients were diagnosed with AKA (52.8% female, mean age 51.9 years); 36 patients were diagnosed with TA ingestion (27.8% female, mean age 42.2 years). The most common reasons for exclusion were: undocumented TA concentration; non-oral exposure; failure of AKA criteria despite acidemia. 28 TA patients were confirmed ethylene glycol, and 8 were confirmed methanol ingestions. A positive ethanol concentration was significantly associated with an AKA diagnosis (OR =5.6, 95% CI 1.7-18.2, p = 0.004). The positive predictive value of positive ethanol concentration for diagnosis of AKA was 75.9%. Other readily available laboratory data, including elevated serum creatinine concentration (OR =0.85, 95% CI 0.59-1.24), decreased pH (OR =1.0, 95% CI 0.99-1.01), decreased bicarbonate concentration (OR =1.0, 95% CI 0.96-1.13), increased lactate concentration (OR =0.93, 95% CI 0.82-1.04), and elevated osmolar gap (OR = 1.0, 95% CI 0.98-1.05), were not significantly associated with either diagnosis.
Conclusion: In this retrospective analysis of patients with suspected TA ingestion, a measurable ethanol concentration was significantly associated with a diagnosis of AKA. The limited ability of common clinical risk factors to differentiate these diagnoses highlights the need to obtain quantitative TA concentrations in real time. Limitations of this study include its retrospective nature and small sample size. Prospective validation of this finding is ongoing.

KEYWORDS Alcoholic ketoacidosis, toxic alcohol, ethanol

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304. Pediatric Obtundation due to Phencyclidine Intoxication

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Background: Phencyclidine (PCP) exposure in the pediatric population is not commonly reported. There is a case report from 2008 similar to our experience. They reported an 8-month-old who was obtunded due to PCP exposure.
Case report: 20-month-old 12.4 kg male was brought to the emergency department for concern for altered mental status and reported possible formaldehyde ingestion. The patient's father found him stumbling in the hallway with an open, empty bottle which he though may contain formaldehyde or an alcohol of some sort. When emergency services arrived at the home, they reported many people were there, the mom appeared to be intoxicated, and they were not allowed in the house (the child was brought to them). At arrival to the emergency department, the patient had decreased level of consciousness, but was arousable with intravenous (IV) catheter placement. The patient was given a 20 mL/kg bolus of normal saline. The patient was also given fomepizole 15 mg/kg. Initial vital signs were blood pressure 119/86 mmHg, temperature 36.8C, heart rate 135 beats per minute, respiratory rate 30 breaths per minute, oxygen saturation 100% on room air. An i-stat was obtained, showing pH 7.33, pCO2 44 mmHg, HCO3 23 mmol/L, base excess -3 mmol/L. CBC revealed white blood count 8.9 k/mcL, hemoglobin 11.0 g/dL, hematocrit 32.5%, platelets 380 k/mcL. Complete metabolic panel revealed sodium 141 mmol/L, potassium 3.8 mmol/L, chloride 108 mmol/L, CO2 24 mmol/L, anion gap 9 mmol/L, BUN 5 mg/dL, creatinine 0.23 mg/dL, glucose 78 mg/dL, AST 35 IU/L, ALT 16 IU/L. The ethanol level was undetectable. The serum osmolality was 291 mOsm/kg, and the osmolar gap was calculated to be 3. The urine drug screen was positive for PCP. This was sent for confirmation testing, and the PCP concentration was 480 ng/mL (normal Over the next two hours, the patient became more somnolent and no longer responded to stimuli. With glucose measurement, the patient did not arouse. Naloxone was administered with no response. He would intermittently cry, but did not localize to pain. The patient was then intubated and transferred to another facility for intensive care needs. The patient improved over the next couple of days and was discharged in his normal state of health.
Case discussion: This is a presentation of PCP intoxication which caused obtundation in an otherwise healthy pediatric patient. This child was ill enough to require intubation until the drug metabolized.
Conclusion: The traditional teaching of PCP intoxication is that patients present agitated. In the pediatric population, they may present obtunded instead.

KEYWORDS Phencyclidine, obtunded, PCP

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305. Traumatic Pedestrian and Bicyclist Injuries Associated with Intoxication - Look Both Ways Before Crossing

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Background: Drug and alcohol use are considered risk factors for trauma among operators of motor vehicles and may also contribute to trauma in pedestrians and cyclists. In this investigation, we describe the prevalence of drug and alcohol use and the clinical consequences in a cohort of pedestrians and bicyclists with trauma.
Methods: We analyzed a 13-month data set comprising of 916 trauma team activations from January 2017-January 2018 at an urban, level I trauma center. Serum ethanol levels and urine toxicology screens for amphetamines, benzodiazepines, THC, cocaine metabolite, fentanyl, methadone, opiates and oxycodone were obtained in 56/87 (64%) of pedestrian and bicyclist trauma activations. We compared pedestrians or bicyclists with a positive urine toxicology screen or serum alcohol level (n = 44) to a control group of pedestrian or bicycle trauma activations with negative results in both serum and urine (n = 12). We conducted a retrospective chart review to determine mechanism, injury pattern and disposition from ED.
Results: Overall, 41 (73%) of the injured patients were pedestrians and 15 (27%) were cyclists; the distribution was similar for patients with negative and positive results. The demographics, mechanism, type of injury and patient disposition in each group is shown in Table 1. Pedestrians and bicyclists with positive toxicology screens were significantly more likely to sustain bony fractures ($p = 0.003$) and require an operative procedure during their hospital stay ($p = 0.029$) than patients with negative results. Of the 44 patients with positive results, 32 (73%) screened positive for fentanyl, 17 (39%) for opiates (e.g. morphine, codeine), 13 (30%) for THC, 10 (23%) for benzodiazepines, 5 (11%) for cocaine metabolite, 4 (9%) for oxycodone, 3 (7%) for methadone, and 1 (2%) for amphetamine. Fourteen (32%) patients were positive for ethanol, with an average serum level of 171 g/dL +/- 105. 41 (93%) were positive for multiple drugs and/or ethanol. Four of the positive fentanyl screens were attributable to EMS fentanyl administration.
Conclusion: Pedestrians and bicyclists with positive toxicology screens were significantly more likely to sustain bony fractures injuries and require operative intervention. The majority of patients with positive results were positive for multiple drugs and/or ethanol.

Category	Criteria	Patients Positive for Drugs or Ethanol (n = 44)	Patients Negative for Both Drugs and Ethanol (n = 12)
Demographics	Male	32 (73%)	9 (75%)
	Average Age (+/- SD)	50 (+/- 18)	58 (+/- 15)
Mechanism	Struck by Car	35 (79%)	7 (58%)
	Self-inflicted	9 (21%)	5 (42%)
Injury	Head Injury	15 (34%)	5 (41%)
	Spine Injury	13(30%)	4 (33%)
	Rib Fractures	11 (25%)	6 (50%)
	Bony Fractures*	34 (77%)	4 (33%)
	Intrathoracic or intraabdominal Injury	7 (16%)	3 (25%)
Disposition	Directly to OR	4 (9%)	4 (33%)
	Required Operative Procedure*	30 (68%)	4 (33%)
	ICU	21 (48%)	3 (25%)
	Floor	18 (41%)	4 (33%)
	Discharged	1 (2%)	0 (0%)

*= indicates a statistically significant difference between the groups.

KEYWORDS Trauma, drugs of abuse, pedestrian injuries

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306. Pharmacy Dispensing Error: Ropinirole for Risperidone in a 10-YEAR-OLD Boy Resulting in Hallucinations

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Background: Outpatient pharmacy dispensing errors are rare, but can result in adverse effect and morbidity. Pediatric patients who have developmental delays and concomitant psychiatric disease are a vulnerable population to adverse effects.
Case report: A 10-year-old boy with a history of developmental delay, aggressive behavior, mood disorder, and attention deficit hyperactivity disorder was brought to the Emergency Department (ED) for increased irritability, persistent movements, and itching. He described hallucinations of seeing "men with machine guns". The child had increased appetite/thirst and had an episode of urinary incontinence. The mother stated that the father picked up the prescription refill 1 week earlier for risperidone 1 mg tablet bid with 0.5 mg prn. The father noted that the pills appeared different, but the bottle had the correct prescription on the labeling. The child had been receiving a total of 2.5 tablets each day of the prescription the past 7 days. He had no changes in his other medications which included methylphenidate patch 30 mg, lamotrigine 100 mg q day, and citalopram 10 mg q day. The patient had been evaluated in an outside ED the day prior and given unknown doses of diphenhydramine and benztropine. He slept for a few hours during the night, but was worse again in the morning. The mother called the poison center who identified the pills as ropinirole 1 mg tablets. She brought the bottle to the ED and the pills were confirmed to be ropinirole. He had no rigidity or clonus on examination. The patient was given diphenhydramine and lorazepam intravenously to control the agitation and hallucinations. He was admitted to the hospital and his prescribed medications were restarted the next morning. He had resolution of the majority of his symptoms with the reinstitution of the risperidone. When the outpatient pharmacy was contacted as part of the investigation of the error, the bottles of ropinirole and risperidone were stored next to each other.
Case discussion: Pediatric exposures to ropinirole are rarely reported. Its safety and pharmacologic effects in pediatric populations is not well established. Ropinirole is a nonergoline dopamine agonist with selectivity at the dopamine (D) 2-like receptors. Hallucinations have been a described adverse effect in adults. This child may have been at higher risk for adverse effects given his usual medication being a D2a antagonist. This case demonstrates the importance of considering a dispensing error and checking the actual pharmaceutical to assure it is the correct drug and dose.
Conclusion: We report the adverse effects of a 10-year child whose prescription was incorrectly dispensed with ropinirole instead of risperidone.

KEYWORDS Ropinirole, dispensing error, hallucinations

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307. Spontaneous Resolution of Significant Portal Venous Gas Following Hydrogen Peroxide Ingestion

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Background: The typical 3% hydrogen peroxide found in homes is generally a benign exposure Concentrations above 10% are known to pose hazard and concentrations of 35% have been fatal with minimal ingestions. Hydrogen peroxide causes toxicity both through direct

corrosive effects, lipid peroxidation, and the generation of large volumes of oxygen with 1 mL of 35% hydrogen peroxide producing 100 mL of oxygen. Vascular gas embolism can result in significant morbidity and mortality due to cerebrovascular accidents and cardiopulmonary obstruction. Case: A regional poison center was contacted regarding a healthy 16 year-old girl whom ingested a mouthful of 35% hydrogen peroxide and experienced 2 episodes of vomiting. Family was instructed to present to an emergency department for immediate evaluation. After contacting emergency medical services, they were evaluated by prehospital providers and subsequently not transported. At poison center follow-up with the family, the need for emergent evaluation was re-iterated and the family presented to a nearby emergency department. At presentation, the patient was tachycardic to 126 bpm and complained of nausea and an irritated throat. She was otherwise asymptomatic with a normal physical exam and normal laboratory studies including a complete blood count, comprehensive metabolic panel, acetaminophen level, and salicylate level. A CT with contrast of the chest, abdomen, and pelvis was ordered to evaluate for air embolism and demonstrated extensive air in the portal veins. The patient was subsequently transferred by ground to a regional referral center with hyperbaric capabilities. On arrival, the patient was asymptomatic with an unremarkable exam. A repeat set of labs remained normal. A repeat CT without contrast of the abdomen was performed and demonstrated marked resolution of the patient's portal venous air. The remainder of her hospital course was unremarkable.
Case Discussion: This case describes a patient who ingested a potentially fatal amount of hydrogen peroxide. Despite this, the patient received conflicting information from EMS providers resulting in a delayed presentation which emphasizes the need to foster open communication between poison centers and all members of the healthcare team. Ultimately, the patient was found to have a large burden of portal venous gas with no significant morbidity and spontaneous resolution. This calls into question the value of cross-sectional imaging in the asymptomatic or minimally symptomatic patient. This patient was geographically remote from the nearest hyperbaric center and ground transport is generally preferred in patients with air embolism. The use of cross-sectional imaging confirmed the presence of embolic air and facilitated the early transfer of the patient to allow for observation in a setting with the appropriate services to manage an acute decompensation.
Conclusions: This case demonstrates a patient presenting with minimal symptoms and a benign exam following ingestion of 35% hydrogen peroxide with significant portal venous gas on CT imaging followed by spontaneous resolution. There is a dearth of evidence to guide the work-up and treatment of these patients. Cross-sectional imaging may have a role in determining the need for transfer of asymptomatic or minimally symptomatic patients to a facility with hyperbaric capabilities.

KEYWORDS Hydrogen Peroxide, Ingestion, Embolism

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308. Gadolinium concentrations in blood, serum and urine in individuals not previously exposed to gadolinium based contrast agents

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Background: Gadolinium-based contrast agents (GBCAs) have been used since the 1980s to increase the quality of magnetic resonance imaging (MRI) scans. Approximately 30 million GBCA-enhanced scans are conducted annually worldwide. GBCAs have previously been considered to have an excellent safety profile in patients with normal renal function but recent evidence suggests that gadolinium deposition can occur within the brain and bone in patients with normal renal function, although the clinical significance of this is not known. There is currently no published data to be able to substantiate a normal range for blood/urine gadolinium concentrations which makes assessment of patients concerned about the toxicity of GBCAs and further research to investigate this issue problematic. The aim of this study was to determine whether a cohort of healthy individuals who have not previously received GBCAs had detectable concentrations of gadolinium in their blood and urine.
Methods: Healthy volunteers living in London, UK with normal renal function (estimated glomerular filtration rate values of ≥70 ml/min/1.73m2) and no reported prior exposure to GBCAs were recruited. Venous blood samples were collected to measure both whole blood and serum gadolinium concentrations (detection limits of 0.0786 ng/ml and 0.008 ng/ml respectively). Simultaneous spot 20 ml urine samples were taken from each volunteer to measure urine gadolinium concentrations with a detection limit of 0.0080 ng/ml. Gadolinium concentrations were measured using inductively coupled plasma mass-spectrometry (ICPMS) with rhodium as the internal standard. The measured urine gadolinium concentrations were corrected for measured urine creatinine concentrations to determine the urine gadolinium:creatinine ratio.
Results: 38 individuals were recruited; 20 (52.6%) were female and the median age was 29.5 years old (IQR 25.1 – 34.5). In 36 (94.7%) of the subjects, there was no detectable gadolinium in whole blood, serum and spot urine samples. Two (5.3%) individuals had detectable gadolinium in one or more biological matrix:
i) 29 year old female: whole blood 0.0157 ng/ml, serum 0.016 ng/ml and urine 0.122 mcg/g creatinine; ii) 27 year old female: whole blood 0.0094 ng/ml; not detected in either serum or urine samples.
Conclusion: The results from this pilot study suggest that gadolinium is not present at significant concentrations in the whole blood, serum or urine of healthy individuals who have not been exposed to GBCAs, in contrast to the numerous reports throughout the literature of patients with persistent detectable gadolinium concentrations in their biological fluids following GBCA exposure. Both of the two subjects with detectable gadolinium concentrations in one or more biological matrix reported having had a previous unenhanced MRI scan, it is possible that they were mistaken regarding prior GBCA administration during these scans. Further work is needed to explore the pharmacokinetics of GBCAs and any potential clinical sequelae that may arise from gadolinium retention.

KEYWORDS Gadolinium, Contrast, GBCA

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309. Emergency Medicine provider attitudes toward and experiences with prescribing buprenorphine in the ED of a large academic metropolitan hospital

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Objective: Healthcare providers in the Emergency Department (ED) are on the front lines of the opioid crisis. ED visits present a unique opportunity to approach patients with opioid use disorder (OUD), and to initiate medication-assisted treatment (MAT) therapy. Buprenorphine MAT has been shown to increase treatment success and compliance, as well as to decrease the number of ED visits and hospitalizations for opioid-related complaints. Under the Drug Addiction Treatment Act

of 2000, providers may obtain an "X waiver" to prescribe and dispense buprenorphine after completing an eight-hour training course. In addition, treatment may now be initiated in the ED by any licensed provider with a DEA number. The purpose of this study was to characterize provider knowledge and opinions about buprenorphine initiation in the ED, and to gauge interest for a MAT protocol in the ED of an urban academic tertiary hospital.

Methods: A survey of 21 questions, including 6 demographic questions, was sent to 98 providers, including ED attending and resident physicians, physician assistants and nurse practitioners. The questions focused on MAT therapy induction with buprenorphine in the ED and various provider attitudes toward MAT, as well as perceived gaps in knowledge about the treatment options and process. Descriptive statistics were performed.

Results: Results are based on a participation rate of 39%. Of the responses, 50% were from attending physicians, 34% were from resident physicians, and 16% were physician assistants. Seventy-seven percent of respondents stated that they were interested in obtaining an X waiver, already had a waiver, or stated that they were unsure about a waiver and needed more information. In terms of the perceived importance of MAT, 87% indicated that it was important to the management of the opioid epidemic, and 76% of respondents believe that it is in their scope of practice as emergency medicine providers to induce buprenorphine in the ED. Areas identified for further education include the specifics of buprenorphine dosing and pharmacology, efficacy of treatment, patient outcomes information, and legal issues associated with buprenorphine. Providers also indicated through structured survey answers and free text that downstream resources such as counseling and clinics were a critical piece of the ED buprenorphine induction process.

Conclusion: Among Emergency Medicine providers in a large academic metropolitan ED, there is clear awareness of the opioid crisis as well as near-consensus that MAT is important in the management of the epidemic. While some providers are willing and eager to participate in a program for buprenorphine induction in the ED, others indicate gaps in their knowledge base and understanding of the process. Through this survey, providers have identified topics of critical importance to cover in buprenorphine training and the X waiver process to ensure the consistency and success of MAT therapy initiated in the ED, as well as gaps in existing processes and infrastructure that are critical to the success of such a program.

KEYWORDS Buprenorphine, medication-assisted treatment (MAT), Emergency Department

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310. Characterization of Emergency Department Poisoning Epidemiology in Belize

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Objective: Strengthening Emergency Care in Belize is a collaborative training program to ensure that every citizen and visitor will have timely access to emergency care. Little is known about the epidemiology of poisonings in Belize. The purpose of this study was to characterize poisonings presenting to Karl Heusner Memorial Hospital (KHMH) department of Accident and Emergency (A&E) in Belize, City, Belize.

Methods: KHMH A&E log book was reviewed for patients with diagnoses that could indicate possible toxicologic exposures. Paper and electronic medical chart review was conducted on possible poisoning cases that met the inclusion criteria of: male and female ages 0-100 presenting to KHMH A&E within the specified dates of May 31, 2016 – December 31, 2018. Variables of interest included patient age, type of poisoning, reason for toxic exposure and disposition.

Results: A total of 60,310 patients presented to the hospital during the study period. Of these, 781 patients, or 1.29%, were identified as presenting with toxicologic complaints. The age distribution of toxicology patient varied with the largest proportion being between 19-30 years of age, (29.5%). Forty-four percent of all poisonings involved ethanol as a primary or co-ingestant. Ingestions that were not solely alcohol constituted 21.9% of exposures. Common exposures included cleaning agents ("Clorox" most common, 12.7%), acetaminophen (7.6%), non-steroidal anti-inflammatories (7%), "sleeping pills" and other sedating medications such as gabapentin and benzodiazepines (7%), antibiotics (4.7%), organophosphates (4.7%) and ciguatera poisoning (3.5%). Interestingly, only one case of oral opioid overdose was reported and only one case of inhaled cocaine. Marijuana was the most common drug of abuse. Seven percent of ingestion exposures and 26% of inhalation exposures involved marijuana. Exposures to environmental toxins including snake, insect, scorpion, stingray and plants comprised 23% of identified cases. Forty six percent of all intoxication cases were determined to be the result of intentional abuse, while 7.2% were from suicide attempt. Of identified poisoned patients presenting to KHMH A&E, 84.1% were ultimately discharged from the department, 13.8% were admitted, 1.5% were "self-discharged" and 0.4% died. There was a male predominance of patients, with 59.4% being male and 40.6% being female.

Conclusion: This study illustrates that poisoning is an important cause of visits to the A&E in Belize whose patterns somewhat differ from those in other countries. Epidemiology studies are limited by site-specific documentation and work-flows. Poisonings are likely underreported and skewed toward lower acuity. Understanding the current state of poisonings in Belize can aid in the development of poisoning management protocols as well as direct educational opportunities for poisoning prevention.

KEYWORDS Belize, Epidemiology, Poisoning

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