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Selected Topics: Toxicology

UNINTENTIONAL PEDIATRIC COCAINE EXPOSURES RESULT IN WORSE OUTCOMES THAN OTHER UNINTENTIONAL PEDIATRIC POISONINGS

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☐ **Abstract—Background: Unintentional pediatric cocaine exposures are rare but concerning due to potentially serious complications such as seizures, dysrhythmias, and death. Objectives: The objectives were to assess the demographic and clinical characteristics of pediatric cocaine exposures reported to the California Poison Control System. Methods: This is a retrospective study of all confirmed pediatric (< 6 years of age) cocaine exposures reported to the California Poison Control System from January 1, 1997–September 30, 2010. Case narratives were reviewed for patient demographics, exposure details, clinical effects, therapy, hospitalization, and final outcome. Results: Of the 86 reported pediatric cocaine exposures, 36 had positive urine drug testing and were included in the study cohort. The median age at presentation was 18 months (range: 0–48 months), and 56% were male (n = 20). The most common clinical manifestations were tachycardia and seizures. The most common disposition was admission to an intensive care unit (n = 14; 39%). Eleven cases (31%) were classified as having a major effect as per American Association of Poison Control Centers case coding guidelines. One child presented in asystole with return of spontaneous circulation after cardiopulmonary resuscitation and multiple vasoactive medications. The proportion of cocaine exposures with serious (moderate or major) outcomes (66.7%; 95% confidence interval 50.3–79.8%) was higher than other pediatric poisonings reported to the American Association of Poison Control Centers during the study period (0.88%; 95% confidence interval 0.87–0.88). Conclusions: Although pediatric cocaine exposures are rare, they result in more severe outcomes than most unintentional pediatric poisonings. Practitioners need to be aware of the risk of recurrent seizures and cardiovascular collapse associated with cocaine poisoning.** © 2017 Elsevier Inc. All rights reserved.

☐ **Keywords—toxicology; pediatric poisonings; poison control centers; cocaine**

INTRODUCTION

Unintentional pediatric cocaine exposures are infrequently reported in the medical literature but are very concerning due to the potential for fever, seizure, dysrhythmia, and even death. A naturally occurring alkaloid extracted from the plant *Erythroxylum coca,* cocaine was first used in 1884 as an ocular local anesthetic and continues to have some limited medical usage in otolaryngologic topical anesthesia and vasoconstriction. The first restrictions in its recreational use in the United States were instituted in 1914, but it became popular as a recreational drug in the 1980s as freebase cocaine, and the late 1980s and early 1990s as crack cocaine. Case reports of pediatric cocaine exposures in the medical literature coincide with this time period, most being published in the late 1980s and early 1990s (1–7).

In 2011, cocaine was still the leading cause of illicit drug-related emergency department (ED) visits in the

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United States, accounting for 505,224 ED visits, of which 21,474 were patients under 21 years of age (8). It is widely used worldwide, with one Spanish study from 2014 documenting about one-fifth of hair samples positive for cocaine in children presenting to the ED for various complaints (9). Despite its popularity, there are few reports of accidental pediatric exposures in the medical literature. The purpose of our study was to fill this gap by assessing the demographic and clinical characteristics of accidental pediatric cocaine exposures reported to the California Poison Control System over the 14-year time period from 1997–2010.

MATERIALS AND METHODS

Study Design and Setting

This is a retrospective cohort study of all pediatric (age < 6 years old) cocaine exposures reported to the California Poison Control System from January 1, 1997 to September 30, 2010. This study was approved by the Committee on Human Research at the University of California, San Francisco and the California Poison Control System (CPCS) Research Committee. The California Poison Control System has four centers (San Francisco, Sacramento, Fresno/Madera, and San Diego) receiving over 330,000 telephone calls annually. The service is available 24 h every day and is utilized by the general population, health care professionals, and law enforcement officials. Calls are managed by a Specialist in Poison Information (SPI). SPIs are specially trained pharmacists or nurses, and medical toxicologists are available to assist on complex cases.

Data from the poison center record, including values coded in specific fields of the computerized database and detailed case notes written by poison center staff, were extracted onto a standardized data collection form. SPIs enter each case into a computerized database, Visual Dot Lab (VDL) (WBM Software, Fresno, CA). Once a case is entered into VDL, it is followed at least daily until the final outcome is known. Cases judged by SPIs to be nontoxic exposures are not followed up. Initial information is acquired from the caller, who may be a layperson, law enforcement, prehospital personnel, or health care professional, and subsequent follow-up information is obtained from the treating physician or nurse. Case narratives are entered in a free-text field and clinical symptoms, treatments, and outcomes are coded by SPIs using American Association of Poison Control Centers (AAPCC) guidelines (10). We assigned outcome into one the following categories: no effect, minor effect, moderate effect, major effect, or death. AAPCC guidelines define no effect as "no symptoms as a result of the exposure." Minor effect is defined as "some symptoms as a result of the exposure, but they were minimally bothersome to the patient. The symptoms usually resolve rapidly and usually involve skin or mucous membrane manifestations." Moderate effect symptoms "are more pronounced, more prolonged or more of a systemic nature than minor symptoms." For a major effect, symptoms "are life-threatening or resulted in significant residual disability or disfigurement."

Study Population

The VDL database was queried for all possible cocaine exposures in children < 6 years of age from January 1, 1997 to September 30, 2010. The AAPCC generic code for cocaine (113000) was queried in the search. Cases outside California and cases that did not have a confirmed urine toxicology screening test positive for cocaine were excluded from the study.

Data Collection and Analysis

De-identified VDL case records were examined and data were extracted into a Microsoft Excel spreadsheet by authors PA and MF (Microsoft, Redmond, WA). An initial pilot sample of 10 cases was used to test-run the data extraction form, and a third author (KO) reviewed the two sets of extracted data to calculate a kappa value for agreement between the two reviewers. The kappa values were 0.964–0.985 for Presenting Symptoms, Complications, Treatments, and Outcomes. Areas of disagreement were discussed and resolved. A fourth author (GM) then independently reviewed all de-identified cases to ensure accuracy. There were no discrepancies noted at that time. Data collected included age, gender, location, time elapsed after exposure to CPCS call, if the caller was a member of the general population or a health care provider, route of exposure, drug type, if Child Protective Services was notified, complications, treatments, disposition, and outcome. Complications were defined as any abnormal clinical findings.

Information was largely obtained from the text of the case notes. If data were missing from the text notes, it was supplemented by review of coded values for symptoms and treatments. For example, it was assumed that an anticonvulsant medication was given if this treatment was coded in the treatment field, even if it was not explicitly described in the free text note. The same was true for intravenous fluids given, presence of hypertension or tachycardia, and the estimated time from cocaine exposure to the initial call to the poison control system. For purposes of defining hypertension or tachycardia we used a standard reference table of pediatric vital signs by age group (11).

Descriptive data analysis was performed using Excel, and geographic information systems mapping was performed using Mapsdata (mapsdata.co.uk). Treating

facility zip codes were used to create a heat map to show areas of higher density using a black-and-white color gradient.

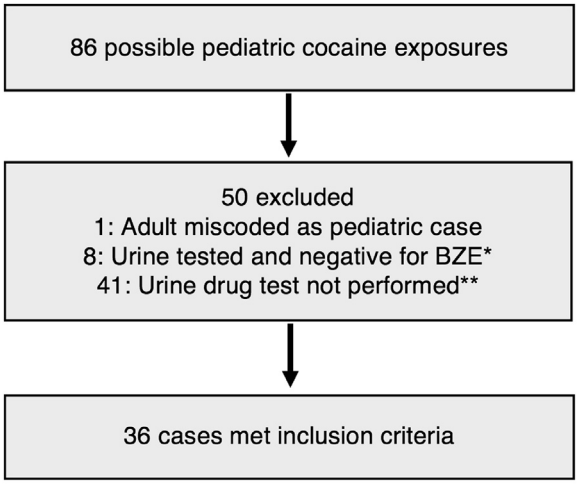
RESULTS

During the study period, 86 pediatric cocaine exposures in patients under 6 years old were identified by the initial VDL query. Of these, 50 cases were excluded, leaving 36 in the final study cohort (Figure 1). Of the excluded cases, 21 were calls from non-health care providers and did not receive any toxicologic testing. General patient and exposure characteristics are presented in Table 1. The median age at presentation was 18 months (range: 0.1–60 months), and 55.6% were male (n = 20). In most cases, the route of exposure was unknown (63.9%). One neonate had in utero exposure (transplacental) and one through breast milk ingestion. In 17 cases with documented exposure times, the median time from exposure to calling the CPCS was 5 h, with a range of 5 min to 2 days.

The most common clinical manifestations associated with cocaine exposure were tachycardia and seizures (Table 2). Agitation was reported in 9 patients, and 8 patients presented with a depressed level of consciousness. A small subset had gastrointestinal symptoms (nausea, vomiting, diarrhea, or abdominal pain). A minority of patients had other clinical manifestations including ataxia, mydriasis, cyanosis, or respiratory depression. There was one case of rhabdomyolysis, one cardiac arrest, and 3 patients that were reported to be asymptomatic.

Medical treatment was required in 24/36 patients exposed to cocaine (67%). Anticonvulsants (lorazepam, midazolam, phenobarbital, phenytoin, and levetiracetam) were used in 13 patients (36%). Twelve children (33%) received decontamination measures, including 11 instances of activated charcoal, two whole bowel irrigations, and one gastric lavage. Whole bowel irrigation was performed in one case of rock cocaine and one cocaine-containing bag ingestion. All 3 of the patients in respiratory distress required endotracheal intubation. The patient who presented in cardiac arrest received multiple medications as detailed below. Twelve patients did not receive any medical interventions and were only observed.

The most common disposition was admission to an intensive care unit (ICU; 14/36 children, 39%), followed by admission to a nonmonitored inpatient bed (13/36, 36%) (Figure 2). There were 10 cases (27.8%) that suffered a major outcome. These included 3 intubated patients, 6 cases of multiple seizures, and a case of rhabdomyolysis and renal failure. One of the patients requiring intubation arrived to the ED in cardiac arrest. Of the major effect cases, most were admitted to an ICU (7/10; 70%). One patient was discharged home after



*Benzoylecgonine (BZE), cocaine metabolite which cross-reacts with standard urine drug of abuse screening tests.
**21 of which were non-health care facility calls.

Figure 1. Flow diagram of cases included in study cohort.

Table 1. Patient Exposure Characteristics

Characteristic	Cases n (%)
Mean age (months, range)	19 (0.1–60)
Male gender	20 (55.6)
Route of exposure	
Unknown	23 (63.9)
Oral	11 (30.6)
Transplacental	1 (2.8)
Breast milk	1 (2.8)
Mean time to PCC call post exposure (hour, range)	13.9 (0.1–48)

PCC = poison control center.
17 cases included in analysis; unknown time for 19 cases.

Table 2. Clinical Findings

Symptom	Cases n (%)
Tachycardia	18 (50)
Seizure	12 (33)
Agitation	9 (25)
Depressed level of consciousness	8 (22)
GI symptoms	6 (17)
Fever	5 (14)
Hypertension	5 (14)
Respiratory depression	4 (11)
Cyanosis	3 (8)
Mydriasis	3 (8)
Ataxia	2 (6)
Cardiac arrest	1 (3)
Rhabdomyolysis	1 (3)

GI = gastrointestinal.

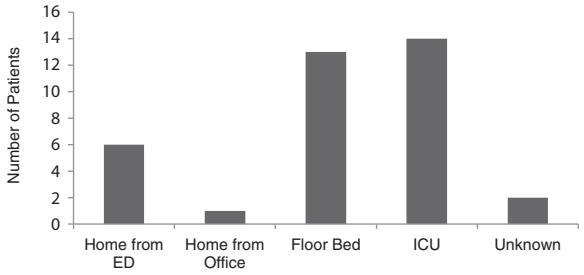


Figure 2. Patient disposition after initial health care provider evaluation. ED = emergency department; ICU = intensive care unit.

observation in the ED after multiple seizures, and 3 were admitted to a noncritical care hospital bed. Patients who presented with agitation, confusion, persistent tachycardia, or a single seizure were categorized as having a moderate effect. This category comprised 14 patients (39%), 6 of which were admitted to an ICU, 6 to a noncritical care unit, one discharged home from the ED, and one was lost to follow-up. The minor effect group (n = 8) consisted mostly of patients with gastrointestinal symptoms or transient tachycardia. Three were discharged home from the ED, 2 were admitted to the ICU, 2 to noncritical care beds, and one was lost to follow-up. In four instances, the patients were asymptomatic during a time when clinical effects would be expected, and it was concluded that the cocaine exposure had no effect. The proportion of cocaine exposures with serious (moderate or major) outcomes and other pediatric poisonings reported to the AAPCC during the study period are presented in Table 3 (12–25).

The most severe case was a 3-year-old girl brought in by a family member 20 min after she was noticed to be "shaking." Upon ED arrival, she was cyanotic, apneic, and in asystolic cardiac arrest. Multiple medications were administered during the resuscitation, including epinephrine, atropine, lidocaine, dopamine, phenytoin, and amiodarone. After return of spontaneous circulation, her systolic blood pressure was 80 mm Hg and temperature was 34.9°C. At first, her cardiac rhythm was ventricular tachycardia at 200 beats/min, followed by junctional tachycardia, and at the time of transfer to a higher level of care it was sinus tachycardia at a rate of 160 beats/min. Her initial head computed tomography scan was negative for mass, shift, bleed, or edema, and initial arterial pH was 6.8. Repeated seizures necessitated fosphenytoin and a vecuronium drip for the first 24 h. On hospital day 3, her pupils became fixed and dilated and a head computed tomography scan showed diffuse cerebral edema. On hospital day 4, she remained unresponsive with fixed pupils and occasional fevers despite being off all sedation. On hospital day 10, her neurological status remained unchanged and she was lost to further follow-up when hospital staff refused to release further information to CPCS staff.

Table 3. Serious Outcomes Associated with Pediatric Cocaine Exposure and All Substance Exposures*

Variable	Cocaine Positive n (%; 95% CI)	All Substances† n (%; 95% CI)
Moderate outcome	14 (38.9; 24.8–55.1)	138,839 (0.81; 0.81–0.82)
Major outcome	10 (27.8; 15.9–44.0)	10,300 (0.06; 0.06–0.06)
All serious outcomes‡	24 (66.7; 50.3–79.8)	149,139 (0.88; 0.87–0.88)

CI = confidence interval.
* Children (age 0–6 years); data from 36 cases reported to the California Poison Control System (1997–2010).
† Includes drugs, herbal products, chemicals and plants; data from Annual Report of the American Association of Poison Control Centers' National Poison Data system 1997–2010; n = 17,042,772 (12–25).
‡ Serious outcomes defined as moderate and severe outcomes, per American Association of Poison Control Centers' criteria.

The geographic locations of all confirmed cocaine exposures during the study period can be seen in Figure 3. The majority of confirmed pediatric cocaine exposures were from the San Francisco Bay Area (n = 21, 58%). Of those cases, 10 were from Oakland and 6 from San Francisco. Seven cases were from the greater Los Angeles and Inland Empire areas, and three were from Sacramento. The remaining five cases were from various locations throughout California.

DISCUSSION

Most of the reported pediatric cocaine exposures in the medical literature are intrauterine exposures from maternal abuse or adolescent recreational abuse cases (26–29). The goal of this study was to examine the presentation and effects of cocaine in pediatric patients under age 6 years reported to a large statewide poison control system. Only 2/36 (5.6%) of our study cohort was exposed in utero or through breast milk. The remainder was due to accidental ingestion and possibly indirect exposure through passive inhalation. It is unclear if any cases were due to intentional administration. We found a high rate of serious complications, with moderate and major outcomes reported in 66.7% of our study cohort. Although this is higher than the rate of serious outcomes from all pediatric exposures reported annually to the AAPCC (0.9% coded as moderate or major effect), our study population was biased toward more severe presentations due to the requirement of a positive cocaine urine drug screen (30). Our observed cases were clustered in the San Francisco Bay area, Los Angeles area, and Sacramento, which are three of the highest-population density

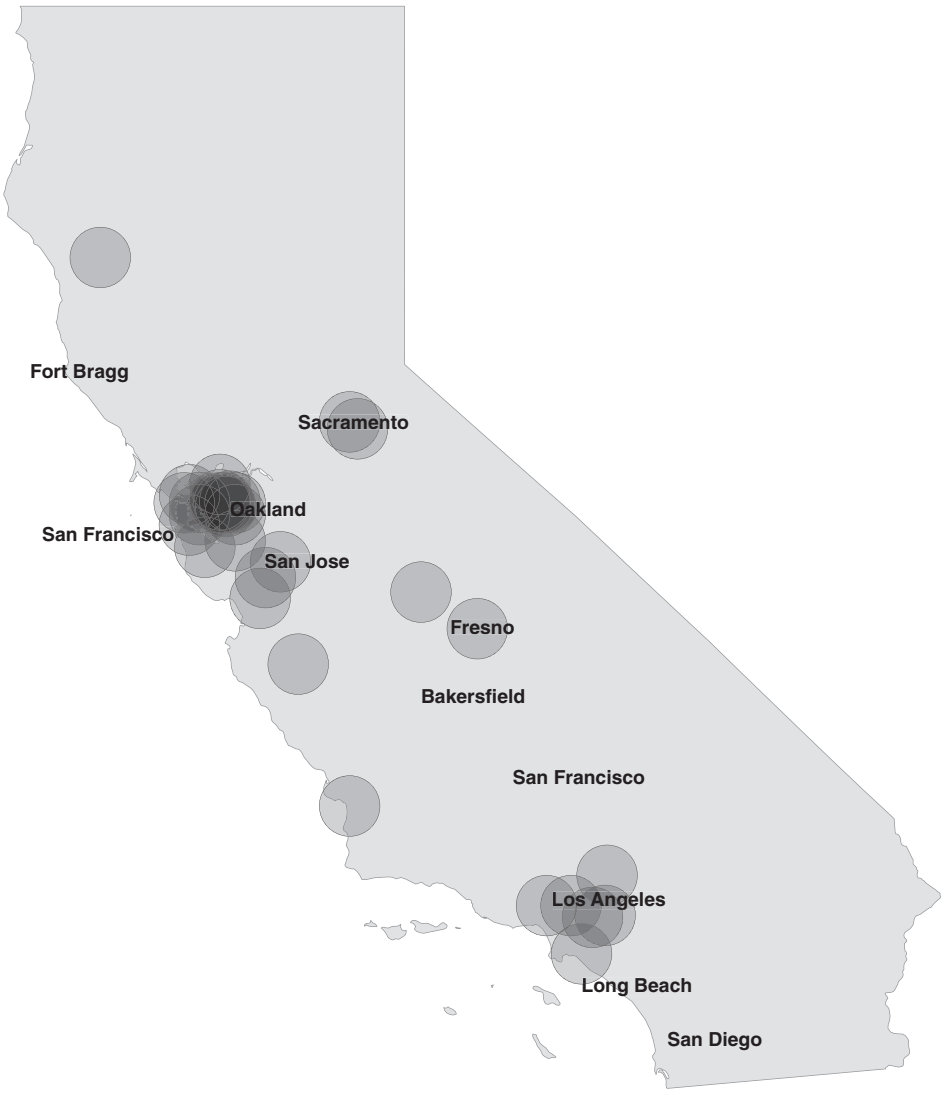


Figure 3. Pediatric cocaine exposure case distribution in California. Darker shading corresponds to more cases of pediatric cocaine exposure reported to the California Poison Control System. Initial health care provider call location zip codes were used to create the heat map.

regions in California. Interestingly, there were very few cases from other high population density regions such as Orange County, San Diego County, and Fresno County, which may reflect different drug abuse habits in the adult population.

Cocaine's cardiovascular and neurologic effects stem mainly from sympathomimetic stimulation by dopamine and norepinephrine and central dopamine and serotonin reuptake inhibition. At high doses, cardiac sodium channel blockade can lead to depressed cardiac conduction and contractility (11). Case reports in infants and toddlers described transient drowsiness, ataxia, dystonic reaction, intussusception, hemorrhagic diarrhea, status epilepticus, apnea, ventricular fibrillation, and death (31–37). In our study, seizures were a common manifestation of pediatric cocaine poisoning, occurring in 12/36 subjects (33%) at some point during their clinical course. Seven cases had multiple or prolonged seizures, with two requiring intubation. Another study examining cocaine-positive cases from 2 months to 18 years of age reported 46% (19/41) having neurologic abnormalities, of which 17.1% (7/41) were seizure(s). In that study cohort, seizures occurred only between ages 1 and 8 years (38).

Cocaine is well absorbed by all routes of exposure, and is extensively and rapidly metabolized in the liver. The metabolite benzoylecgonine is the compound detected by standard hospital urine drug-of-abuse screening immunoassays. Positive urine drug-of-abuse tests for cocaine are not typically confirmed by further testing by hospital laboratories because the assays have very

high sensitivity and specificity (99%) for benzoylecgonine (39). Although the elimination half-life of cocaine is about 1 h, benzoylecgonine can be detected in the urine for up to 3 days after exposure. Therefore, a positive urine test may not indicate acute toxicity, but rather very recent exposure. In four ED prevalence studies based on urine drug-of-abuse screening without clinical suspicion, positive cocaine urine tests were found in 2.4%, 4.4%, 5.4%, and 36.3% of children (5,40–42). Lustbader et al. concluded that their high prevalence rate of 36.3% was likely due to an inner-city population (New Haven, CT), testing only in ill children who already required a urine analysis, and a more sensitive screening test with lower thresholds for positivity. Because the other studies were also done in already-ill patients who had presented to EDs for care and in inner cities (Boston, MA and Detroit, MI), it is likely that selection bias led to the high prevalence of cocaine exposure.

Because cocaine toxicity is a rare event in children, emergency providers may not consider it in the differential diagnosis of a febrile, tachycardic child, perhaps especially in the event of seizure. Febrile seizures are commonly seen in the ED with an incidence of 2–5%, with most cases occurring between 6 months and 5 years of age (43). In the proper clinical and historical setting, a child with a new-onset seizure not easily ascribed to a simple febrile seizure may be considered for urine cocaine screening.

Limitations

The retrospective study design and data source (poison center case reports) are a significant limitation to the completeness of study data. Not all cases of pediatric cocaine exposure are reported to the CPCS, as there is no legal reporting requirement. Poison center staff enter case data at the time of the call that is relevant to immediate case management, but may be incomplete for research purposes. Another limitation with poison center charts is the occasional case lost to follow-up due to patients being discharged prior to the poison center call, or health care providers no longer willing to share information with poison control center staff after the patient is admitted to another unit. In our study cohort, two cases were lost to follow-up: one with no clinical effect and one with a moderate clinical outcome, both of which were not anticipated to have any serious morbidity or mortality.

Because these are retrospective poison center cases, there was no opportunity to evaluate patients in person or to obtain hospital charts that would likely contain more complete information. Coded therapeutic interventions in each poison center chart are included in the data even if those interventions were not mentioned in the free text field of the chart. Another limitation to this study is that we relied on urine drug-of-abuse screening tests, which although very reliable for cocaine, are not confirmatory (such as gas chromatography-mass spectrometry or liquid chromatography-tandem mass spectrometry) and do not represent quantitative levels. Because our study included cases from multiple health care facilities, different brands of immunoassay were used, which may have different thresholds of benzoylecgonine detection. Finally, we did not include the cases that did not have a documented positive cocaine urine drug-of-abuse screen, even though some of these cases may have been cocaine intoxicated but not tested, or were tested positive but not recorded in the poison control center database. This made the number of patients captured very low, but accurate in documenting true cocaine exposure. Because only urine-positive tests were included, this study also has some selection bias, skewing the study population toward more severe presentations.

CONCLUSION

Although accidental pediatric cocaine exposures are rare, they result in more severe outcomes than most unintentional pediatric poisonings. Practitioners need to be aware of the risk of recurrent seizures and cardiovascular collapse after cocaine poisoning. We recommend testing for cocaine exposure for children under age 6 years for new-onset unexplained seizures, agitation, unexplained persistent tachycardia, and altered mental status.

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ARTICLE SUMMARY

1. Why is this topic important?

Accidental pediatric cocaine exposures are rare but may result in serious morbidity and mortality.

2. What does this study attempt to show?

This study attempts to characterize the demographic and clinical characteristics of confirmed pediatric cocaine exposures under age 6 years.

3. What are the key findings?

Tachycardia and seizures were the most common clinical manifestations, with most patients admitted to intensive care units. Compared with other unintentional ingestions in this age group, there were more severe outcomes with cocaine exposure.

4. How is patient care impacted?

We recommend cocaine testing for young pediatric patients presenting with unexplained seizures, tachycardia, or agitation that do not quickly resolve in the emergency department.