useful in young children. Our aim was to determine whether there was a relationship between QRS duration and the risk of seizures or arrhythmias in children under the age of five years presenting with TCA poisoning. Methods: Case notes of all children <5 years with TCA poisoning admitted to St Mary's Hospital PICU between 1994 and 1999 and all those reported to the NPIS (L) over the period June 1998 to August 1999 inclusive were examined. The NPIS (L) data was collected prospectively and the subsequent clinical course of the symptomatic cases was obtained by follow up call and/or postal questionnaire. QRS durations were measured on the presentation ECG by clinicians at the corresponding hospitals. Results: During the period 1994–1999, St. Mary's Hospital PICU managed 3 children less than 5 years of age with TCA poisoning. In the fifteen months of prospective data collection, 88 cases of suspected TCA poisoning in children <5 years of age were reported to NPIS (L), of whom 14 developed serious symptoms. Of these 17 cases (combined NPIS and PICU data), 11 had taken dothiepin (dose 10.7–51.9 mg/kg; mean ± SD 25.8 ± 12.0 mg/kg), 4 had taken amitriptyline (dose 9.6–27.3 mg/kg; 20.9 ± 8.3 mg/kg) and in one case each, amoxapine (unknown amount) and imipramine (up to 40 tablets) had been ingested. All required admission to a PICU. 12 (71%) developed seizures, 11 (65%) had a QRS prolongation of >0.10s and 5 (29%) had QRS prolongation >0.16s. Of these 5, all developed arrhythmias; 2 developed VT, 2 VF and 1 an EMD arrest. A QRS duration of >0.16s predicted the risk of developing arrhythmias (chi-squared analysis $p < 0.00005$). However, there was no relationship between QRS duration and the risk of seizures ($p = 0.1$). Conclusions: TCA poisoning in children remains an important cause of PICU admission and fatalities still occur. Severe clinical effects can occur with doses as low as 10 mg/kg. On the basis of this study, QRS prolongation is a determinant of the risk of arrhythmias but not seizures in children under five years of age with TCA poisoning.

73 PEDIATRIC BUPROPION EXPOSURES REPORTED IN TEXAS: 1998–1999

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Background: Currently there is limited information describing the effects of ingestions of bupropion in young children. Bupropion is available in the US under 2 different trade names for separate indications. The purpose of this retrospective case review is to describe the characteristics of bupropion exposures in this age group. Methods: A retrospective review of all cases of bupropion ingestion in children 6 years old and younger that were reported in Texas during 1998–1999. Cases without follow up or cases involving multiple substances were excluded. Evaluation was based on American Association of Poison Control Centers data coding and reviews of the written case record. This review evaluated dose, certainty of ingestion, dosage form, management site, clinical effects, therapies, outcome and duration of observation. Results: Of 142 cases 114 met the criteria for evaluation. Reasons for exposures were either therapeutic errors (n = 1) or unintentional general (n = 113). Most cases (78%) involved Wellbutrin®. 85% of the cases involved SR dosage forms. Most cases (79%) were evaluated in health care facilities. No differences in poison center triage patterns or outcome could be detected with respect to dosage form or brand name. Self-referral was 50% more frequent for Wellbutrin® than for Zyban®. Decontamination was performed in 57% of the cases. Follow-up/observation times ranged from 1 to 24 hours. Over half of the cases were observed for over 6 hours. Most ingestions were 2 tablets or less. Ingestion was observed in 61 cases. There were no seizures, deaths, moderate or major outcomes. Most patients (92%) did not develop any symptoms. Conclusions: There were no significant clinical effects reported in this population. Decontamination did not appear to make a difference in outcome. Public perception of the toxicity of a medication may be influenced by the indication for which the drug is prescribed.

74 ACCIDENTS CAUSED BY *BOTHROPS* SPP SNAKES IN CHILDREN

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Objectives: The Brazilian Ministry of Health reports about 20,000 accidents per year caused by venomous snakes, mainly by the genus *Bothrops* (90.5%). Despite this high frequency, few studies have reported the clinical aspects of venomous snakebites in children in Brazil. This study evaluated the clinical aspects and outcome of bites caused by *Bothrops* spp snakes in children less than 14 years old. Case Series: From January 1984 to March 1999, 73 children (ages 1 y–14 y, median 9 y) were admitted after being bitten by *Bothrops* spp. The severity of envenoming was classified according to the recommendations of the Brazilian Ministry of Health. Upon hospital admission, 26.0% patients were classified as mild envenoming, 50.7% as moderate envenoming and 20.6% as severe envenoming. Two patients (2.7%)

showed no signs of envenoming (dry bites). Most of the patients presented local manifestations, mainly indurated edema (95.9%), pain (94.5%), ecchymosis (74.0%) and local bleeding (21.9%). Systemic bleeding was observed in 9 patients, mainly gingival (9.6%). Before antivenom (AV) administration, blood coagulation disorders were observed in 62.5% (incoagulable blood in 39.3%) of the 56 children that received AV only in our hospital (mild accidents, median = 4 vials; moderate accidents, median = 7 vials; severe accidents, median = 10 vials; 1 vial = 10 mL of Fab'_2). AV early reaction was observed in 44.6% of the cases (15/30 patients not pretreated and in 10/26 patients pretreated with hydrocortisone and histamine antagonists H1 and H2), most of which were considered mild. Only one patient presented an acute severe early reaction. More than 76% of these 56 children received AV less than 6 h after the bite. The main clinical complications observed were local infections (15.1%) (including cellulitis, 9.6%, and abscesses, 5.5%), compartment syndrome (4.1%), gangrene (1.4%) and acute renal failure (1.4%). There were no significant differences with regard to severity of envenoming and the frequency of blood coagulation disorders among the three categories of envenoming ($p = 0.75$), or in the frequency of early AV reactions between the groups that were and were not pretreated ($p = 0.55$). The frequency of local infections was significantly greater in severe cases ($p = 0.001$, RR = 6.53, OR = 11.38). Patients admitted more than 6 h after the bite had a higher risk of developing severe envenomation ($p = 0.04$, RR = 1.65, OR = 4.75). No deaths were observed. Conclusion: The prognosis can be very good as long as the children receive prompt medical care, including adequate AV prescription, fluid replacement to avoid renal ischemia, and correct treatment of the main complications.

75 POISONING AFTER LOCAL APPLICATION OF ATROPINE EYE DROPS IN CHILDREN

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Objective: Atropine containing ophthalmic preparations are used for diagnosis and therapy of a variety of eye diseases in children. Between 1994 and 2000 we encountered 14 cases of poisoning with atropine eye drops, 8 of them after prescription by a physician. Here we present three typical cases. Case Reports: *Case 1*: A 9-month-old boy receiving 0.5% atropine ocular solution (one drop in each eye twice daily) was admitted with mydriasis, flushing, somnolence, vomiting, tachycardia, and increased temperature (38.5 °C) three days after beginning an ocular instillation. The dose was estimated at 0.07 mg/kg/d. 24 hours after the last application the atropine level was 42.6 ng/mL (therapeutic range: 3–25 ng/mL). Symptoms disappeared quickly following controlled administration of physostigmine. Case 2: A 1.5-year-old girl was hospitalised with flushed, warm, dry skin, tachycardia, excitatory state, mydriasis and increased temperature of (37.4 °C) 45 minutes after the second application of 0.5% atropine eye drops twice daily at 12 hourly intervals. The dose could not estimated exactly. The parents declined treatment and flushing and tachycardia continued over the next 6 hours. Mydriasis was observed for more than 24 hours. Case 3: A 3-year-old boy was admitted with tachycardia, an excitatory state, mydriasis and respiratory depression three days after beginning therapy with 1% atropine eye drops. This was given as a therapeutic dose of one drop three times a day. The dose was estimated at 0.1 mg/kg/d. The patient was intubated and ventilated. Symptoms resolved after two doses of physostigmine intravenously. Conclusion: Local application of atropine containing eye drops in therapeutic dose may cause systemic poisoning in infants. Inexpert application, short dosing intervals, and administration in both eyes appear to increase the risk of overdose. Following occular instillation atropine may be absorbed from the conjunctival sac directly as well as from the ingested overflow. Physostigmine is an effective antidote but it ought to be given only in severe cases of anticholinergic symptoms and under carefully controlled conditions.

76 MUNCHAUSEN'S SYNDROME BY PROXY IN THE FORM OF CHILD ABUSE BY POISONING: CASE REPORTS AND REVIEW OF THE LITERATURE

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Four case reports will be described representing various presentations of Munchausen's syndrome by proxy (MSBP): (i) Chronic diarrhea and failure to thrive in a 23 month old; (ii) Factitious hypoglycemia in a 2 year old; (iii) Recurrent vomiting, muscle weakness, and rhythm disturbances in a 16 month old; (iv) Unexplained recurrent vaginal bleeding in a 16 year old. The origin of Munchausen's syndrome refers to Baron Munchausen, an 18th century mercenary who went to battle with the Turks. On the basis of his writings, which later proved to be a total fabrication, other authors published "Baron Munchausen's Narrative of His Marvelous Travels and Campaigns in Russia." In 1951, Dr. Richard Asher described a disorder where patients fabricated histories, feigned illnesses, and deceived multiple care takers,

heralding the birth of Munchausen's syndrome. In 1977, Meadow described Munchausen's syndrome by proxy (MSBP), with children as the primary victims. A review of the literature reveals that the perpetrator is often the natural mother of the child, with a high degree of intellect and often some medical training. The perpetrator alternates as unknowing accomplices in the investigation of the complaint. The characteristics of MSBP include either simulated or, in most cases, actual illness which is created by a caretaker with access to the child. Repetitive visits to the medical facilities often prompt complex medical workups. Perpetrator denial is common. Most importantly, symptoms will abate in the absences of the perpetrator. The toxicological literature is filled with case reports of patients with Munchausen's syndrome by proxy, as contained in the table.

Reported Cases from the Literature

Age	Sex	Presentation	Agents	Outcome
36	F	Apnea	Pepper	Death
19	M	Prolonged sleep	Phenothiazines	Survival
½	F	Hypoglycemia	Salicylates	Survival
30	M	Vomiting, hypernatremia	Table salt	Survival
4	M	Seizure, hyponatremia	Diluted formula	Survival
18	M	Coma, hematemesis	Pine Oil	CNS damage
48	F	Fever, irritability	Vitamin A	Survival
56	F	Fever, recurrent infection	Dirty IV fluids	Survival
22	F	Polyuria, glucosuria	Glucose	Survival
84	M	Coma, apnea	Imipramine	Survival

Demographics of MSBP cases demonstrate that 84% of symptoms will occur within the hospital proper, and many patients will suffer from other forms of child abuse as well. In addition, sibling deaths have been reported. Of interest, Munchausen's syndrome is present in nearly 10% of the perpetrators. Warning signs of MSBP include unexplained persistent or recurring illness, signs and symptoms that do not make sense, symptoms that do not make sense, symptoms that magically disappear in the mother's absence, variable levels of maternal concern, and the presence of a fascinoma in general. Disposition of the MSBP includes first and foremost separation of the child from the perpetrator. The clinician must perform an educated toxicological workup. In many cases occult surveillance may be necessary to unearth the diagnosis. Most importantly, the clinician is cautioned to keep an open mind; nothing is too far fetched. References: Meadow R. Munchausen syndrome by proxy: The hinterland of child abuse. *The Lancet* 1977;**2**:343–345. Morris M. Munchausen syndrome by proxy and factitious illness. *Curr Opin Psychiatry* 1994;**4**:225–230. Sigal M, Gelkopf M, Meadow RS. Munchausen by proxy syndrome: the triad of abuse, self-abuse, and deception. *Compr Psychiatry* 1989; **30**:527–533. McClure RJ, Davis PM, Meadow SR, Sibert JR. Epidemiology of Munchausen syndrome by proxy, nonaccidental poisoning and nonaccidental suffocation. *Arch Dis Child* 1996;**75**;75:57–61. Alexander R, Smith W, Stevenson R. Serial Munchausen syndrome by proxy. *Pediatrics* 1990;**86**:581–585. Morley CJ. Practical concerns about the diagnosis of Munchausen syndrome by proxy. *Arch Dis Child* 1995;**72**:528–530. Asher R. Munchausen syndrome. *Lancet* 1951;**1**:339–341. Meadow R. Nonaccidental salt poisoning. *Arch Dis Child* 1993;**68**:448–452. Berger D. Child abuse simulating apparent "near miss" sudden infant death syndrome. *J Pediatr* 1979;**95**:554–556. Boros SJ, Ophoven JP, Andersen R, Brubaker LC. Munchausen syndrome by proxy: a profile for medical child abuse. *Aust Fam Physician* 1995;**24**:768–769. Rosenberg DA. Web of deceit: a literature review of Munchausen syndrome by proxy. *Child Abuse Negl* 1987;**11**:547–563. Meadow R. Management of Munchausen syndrome by proxy. *Arch Dis Child* 1985;**60**:385–393. Bools C, Neale B, Meadow R. Munchausen syndrome by proxy: a study of psychopathology. *Child Abuse Negl* 1994;**18**:773–788. Mitchell I, Brummitt J, DeForest J, Fisher G. Apnea and factitious illness (Munchausen syndrome) by proxy. *Pediatrics* 1993;**92**:810–814. Johnson JE, Carpenter BL, Benton J, et al. Hemorrhagic colitis and pseudomelanosis coli in ipecac ingestion by proxy. *J Pediatr Gastroenterol Nutr* 1991;**12**:501–506. Orenstein DM, Wasseman AL. Munchausen syndrome by proxy simulating cystic fibrosis. *Pediatrics* 1986;**78**:621–624. Sutphen JL, Saulsbury FT. Intentional ipecac poisoning: Munchausen syndrome by proxy. *Pediatrics* 1988;**82**:453–455. Nichols GR, Davis GJ, Corey TS. In the shadow of the Baron: sudden death due to Munchausen syndrome by proxy. *Am J Emerg Med* 1990; **8**:216–219. Senocak ME, Turken A, Buyukpamukcu N. Urinary obstruction caused by factitious urethral stones: An

amazing manifestation of Munchausen syndrome by proxy. *J Pediatr Surg* 1995;**30**:1732–1734. Babcock J, Hartman K, Pedersen A, et al. Rodenticide-induced coagulopathy in a young child. A case of Munchausen syndrome by proxy. *Am J Pediatr Hematol Oncol* 1993;**15**:126–130.

77 CARBON MONOXIDE POISONING: CHARACTERISTICS OF CHILD INTOXICATION

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Carbon monoxide (CO) poisoning is a frequent casualty and occurs in children who may experience different clinical manifestations than adults. Objective: To compare clinical manifestations and outcome of CO poisoned children to those observed in adult patients. Method: During 2 years, all children (age under 15 years old) admitted to the pediatric emergency department for a CO poisoning were enrolled. Case report included poisoning history, clinical manifestations, in-hospital course and outcome. Comparison was made with a series of CO poisoned adult patients. Statistical analysis was made by chi-square test or variance analysis according to the case, taking a p value of less than 0.05 as significant. Results: During a 2-year period, 140 children were admitted for CO poisoning. 29 were under 2 years of age (21 per cent), 70 were between 2–10 years (50 per cent), 41 between 10–15 years (29 per cent). CO sources were gas water heater in 50 cases (36 per cent), charcoal stoves in 31 (22 per cent), gas heater in 21 (15 per cent) and fire in 15 (11 per cent). Clinical presentation included loss of consciousness in 52 (37 per cent), headache in 37 (26 per cent), vomiting in 32 (22 per cent), lethargy and coma in 27 (19 per cent), convulsion in 7 (5 per cent). Clinical examination revealed exaggerated reflexes in 59 cases (42 per cent), flaccidity in 24 (17 per cent), abnormal extensor plantar responses in 11 (8 per cent), cerebellar impairment in 11 (8 per cent). Mean carboxyhemoglobin level at hospital admission was 17 ± 13 per cent with no close relation with clinical manifestations. 95 children underwent hyperbaric oxygenation while 45 received only normobaric pure oxygen. 1 child died from brain death after an on site resuscitated cardiac arrest, 1 other experimented secondary neurological manifestations. All other were cured and free of symptoms at 1 year after poisoning. Compared to adult patients (774 cases), gas water heater as CO source, lethargy and flaccidity, especially in the youngest children, are more frequent. Late neurological manifestations are less frequent. Conclusion: Due to differences in clinical expression of CO poisoning in children, special attention has to be given to clinical manifestations to evaluate poisoning severity and to decide treatment.

78 ACUTE SMOKING INHIBITS CYTOCHROME *C* OXIDASE (COMPLEX IV) OF PERIPHERAL LYMPHOCYTE MITOCHONDRIA

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Background: Smoking is associated with a decrease of cytochrome *c* oxidase activity (the fourth and last complex of mitochondrial respiratory chain, MRC) in chronic smokers, but whether this toxic effect is due to the acute action of cigarette smoke itself or to other smoking-related factors coexisting in chronic smokers is unknown. Objective: To assess the acute effect(s) of smoking in MRC function from non-smoking healthy individuals. Individuals and Methods: 15 non-smoking individuals (25 ± 4 years; 9 men) were enrolled to smoke 5 cigarettes in 45 minutes. Blood samples were taken before (t_0), and after 5 minutes (t_1) and 24 hours (t_2) of smoking. Carboxyhemoglobin was determined in all blood samples by CO-oxymetry, and carbon monoxide concentration in exhaled breath of participants at t_0, t_1 and t_2 by an electrochemical transducer. Lymphocytes were isolated by Ficoll's gradient, protein content was determined by Bradford's methodology, and MRC function was studied through a double way. First, individual enzyme activities of complex II, III and IV of MRC were spectrophotometrically assessed. Second, oxygen consumption was polarographically measured in permeabilized lymphocytes using pyruvate, succinate and glycerol-3-phosphate as complex I, II and III substrates, respectively. This later methodology allows us to ascertain if eventual dysfunction of any MRC complex has some effect in the whole mitochondrial respiratory capacity. Paired t test was used to compare measurements at t_1 and t_2 relative to those at t_0. Results: Cigarette smoke inhalation by participants was considered to be effective as judged by carboxyhemoglobin levels of 0.5 ± 0.3% at t_0, 3.3 ± 1.5% at t_1 ($p < 0.001$), and 0.6 ± 0.3% at t_2 (p = NS), and by exhaled carbon monoxide concentrations of 2.9 ± 2.5 ppm at t_0, 26.1 ± 9.9 ppm at t_1 ($p < 0.001$), and 3.9 ± 2.3 ppm at t_2 (p = NS). We found that while enzyme activity for complexes II and III remained unchanged along the study, complex IV activity fell from 49.4 ± 17.1 nmol/min/mg protein at t_0 to 38.1 ± 11.7 nmol/min/mg protein at t_1 ($p < 0.05$); further, it returned to basal levels at t_2 (50.0 ± 19.3 nmol/min/mg protein; p = NS). Despite this 23% (95% CI:

from 1% to 44%) acute inhibition of complex IV activity, no statistical differences were found in oxygen consumption at t_1 and t_2 relative to t_0 for either of substrates employed. Conclusions: Acute smoking causes a reversible mitochondrial complex IV inhibition of blood lymphocytes, with no apparent repercussion in MRC oxidative capacity. These results support that smoke itself is one of the causes of the decrease of complex IV activity observed in chronic smokers. (Supported by FIPSE 3102/00, FIS 00/0927, DGCYT PM99-0038, SGR 1999/00279 and Premi Fi de Residència 1998 of HCP).

79 CLINICAL SAFETY OF HIGH DOSES OF HYDROXOCOBALAMIN IN FIRE VICTIMS

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Background: Cyanide (CN) poisoning in fire victims is frequent and rapidly fatal. Several antidotes to cyanide are available. However, their safety has not been assessed in the complex setting of smoke inhalation. In a prospective study we tried to assess the clinical tolerance of a high dose of hydroxocobalamin (HOCo) administered at the scene of the fire in fire victims suspected of CN poisoning. Methods: Inclusion criteria: fire victims were included according to the presence of the following criteria: soot deposits in mouth or sputum plus any degree of neurological impairment. Exclusion criteria: children, pregnant women, burn of total surface body area >20%, multiple trauma were excluded. Protocol design: following examination and the collection of a blood sample in dry heparin, a 5 g dose of HOCo (10 g in case of cardiovascular collapse) was administered intravenously over 15 min. Blood cyanide concentrations were measured using the method of Rieders. The heart rate and systolic blood pressure were monitored before and after the administration of HOCo, and one hour later. A particular attention was paid to the occurrence of allergic reactions: Quincke edema, cutaneous rash, and bronchospasm. Results: There were 28 females and 22 males. The mean blood CN concentration was 83 ± 73 μmol/L. The mean blood carbon monoxide was 3.2 ± 2.1 mmol/L. Nineteen fire victims eventually died. In the 27 non-CN-intoxicated patients (blood CN < 40 μmol/L), there was no significant change in arterial blood pressure. In the 33 CN-intoxicated patients (blood CN > 40 μmol/L) a significant increase in blood pressure was observed both immediately ($p < 0.001$) and 1 hour later ($p < 0.001$) after the administration of HOCo. No allergic reactions were observed. The lone side effects were a deep red coloration of the urine that disappeared within 7 days after injection. Conclusions: In fire victims with cyanide poisoning, the administration of a high dose of hydroxocobalamin was associated with an improvement in systolic blood pressure. Hydroxocobalamin is well tolerated in fire victims without CN poisoning. No allergic reactions were observed both in cyanide poisoned and non-cyanide poisoned fire victims.

80 COMMON METHODS OF ILLICIT PREPARATION OF KETAMINE: GAS CHROMATOGRAPHY/MASS SPECTROMETRY (GC/MS) ANALYSIS OF PRODUCTS

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Objective: Ketamine hydrochloride is a dissociative anesthetic commercially produced as a liquid preparation with benzethonium chloride preservative intended for parenteral administration. Ketamine has become a major drug of abuse in dance parties and dance clubs in urban areas of Europe, North America, and Asia. Illicit use of ketamine is typically by intranasal insufflation of ketamine powder. Evaporating the liquid diluent in which ketamine hydrochloride is dissolved produces ketamine powder. Evaporation is typically performed by one of three methods: 1) allowing the solution to evaporate under ambient conditions, 2) heating the solution with a double-boiling apparatus, or 3) heating the solution in a microwave oven. The medical and forensic management of patients using such powder is based on the assumption that the substance patients are using is ketamine hydrochloride. Illicit preparation involving heating may cause the ketamine hydrochloride to form ketamine free base, react with itself, or with the preservative benzethonium chloride to produce products other than ketamine hydrochloride. Methods: Equal aliquots of ketamine hydrochloride (100mg/mL) in benzethonium chloride preservative were processed by: 1) evaporation at ambient conditions with room temperature of 23.8°C, 766 mmHg, 2) use of a double-boiler with water temperature at 99.4°C; 3) and use of a microwave oven producing 2450 MHz for 60 seconds. The products of each of these processes were analyzed by GC/MS and compared to each other as well as a standard of ketamine hydrochloride. Results: Evaporation required approximately 30 hours at ambient conditions, 23 minutes when double-boiled, and less than 60 seconds when microwave was used. GC/MS

analysis of products demonstrated three peaks at 16.59 minutes 18.25 minutes, and 19.59 minutes, corresponding to two unidentified ketamine-like products, and ketamine free base, respectively. Trace amounts of dibenzofuran were also identified in an earlier peak of the same GC/MS analysis. Conclusions: These methods are precisely those used to convert ketamine hydrochloride solutions to solid form for illicit intranasal use. The products of such preparation aren't pure ketamine hydrochloride: All contained free base of ketamine, and as well, all illicitly prepared forms contained two unidentified ketamine-like products and trace amounts of dibenzofuran. The ketamine hydrochloride standard was converted to free base as a result of heating in the GC/MS analyzer. It is unknown whether the products of illicit ketamine preparation contain benzethonium chloride because this substance may not elute from the chromatography column. Illicit preparation of commercially available ketamine hydrochloride solution by evaporation at ambient conditions, double boiling, and microwaving produces ketamine free base as well as other currently unidentified products.

81 COCAETHYLENE AND ECGONINE ETHYL ESTER IN HOSPITALISED PATIENTS WITH A MIXED COCAINE-ETHANOL INTOXICATION

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Objective: Cocaine acute poisoning and toxicity is an increasing cause of concern in most developed countries. Since cocaine abuse is frequently associated with alcohol consumption a relatively high number of hospitalizations and fatalities result from a combined cocaine-ethanol overdose. The formation of cocaethylene, a psychoactive derivative, in tissues may account for some toxic effects of this specific combination. The aim of this study was to determine the rate of combined ethanol poisoning among hospitalized patients with acute cocaine intoxication and the proportion of patients with detectable levels of cocaethylene and other specific metabolites of the combination ethanol-cocaine. Methods: We collected biological samples (blood and urine) of cocaine-poisoned patients from ''Hospital Clínic'' and other nearby hospitals (Barcelona, Catalonia), during 1998-2000 and we studied the levels of ethanol, cocaine, cocaine metabolites (benzoylecgonine, ecgonine methyl ester, norcocaine) and ethanol-cocaine metabolites (cocaethylene, ecgonine ethyl ester, norcocaethylene). All metabolites were analyzed by gas chromatography-mass spectrometry (GC-MS, HP-5971A) after solid-phase extraction (Bond Elut, Varian®), BSTFA derivatization column separation (Tracsil®, TRB-5MS; 0.20 mm, id.) and quantification with deuterated standards. Ethanol was analyzed by FID, head-space gas-chromatography (HP-5890). Results: 57 cases (29,5%) out of total N = 193 cocaine poisoned patients showed detectable levels of ethanol in blood (limit of detection: 0.05 g/L). The analysis of cocaine metabolites by GC-MS showed that all patients (N = 21) with ethanol in blood > 0.75 g/L presented detectable levels of cocaethylene and ecgonine ethyl ester in urine and/or serum (limits of detection: 25 ng/mL) in addition to cocaine specific metabolites (benzoylecgonine, ecgonine methyl ester). In 19 cases cocaethylene concentration in urine was > 250 ng/mL (range 250–2200 ng/mL). Conclusion: The results strengthen the view that the formation of specific metabolites is a very frequent outcome of the combined cocaine-ethanol poisoning. In our series 100% of patients with alcohol in blood > 0.75 g/L showed detectable concentration of these metabolites. Moreover, the selective quantification in urine shows that both cocaethylene and ecgonine ethyl ester appear as major metabolites when cocaine intake is associated with medium/high ethanol blood levels, thus suggesting an important role of this metabolic pathway and a probable important role of cocaethylene in the overall toxic effects of the combination cocaine-alcohol.

82 UNACCEPTABLE TIME DELAY IN USE OF ACTIVATED CHARCOAL

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Objective: Over the past two centuries, attempts to salvage orally poisoned patients have usually involved some process of ''decontamination''–e.g. emptying the stomach or curtailing absorption of poisons. Today, use of the nasogastric tube is at the bottom of the list of effective remedies. Induction of emesis with Syrup of Ipecac boomed in popularity in the early 1960s, but then declined with the ''re-discovery'' of activated charcoal by Holt in 1964. It burgeoned as promoted by members of the emerging specialty of Emergency Medicine in the 1970s and 80s and appears to have been cast in concrete by the release of ''expert-based guidelines'' in 1997. Particularly over the last decade, more and more data support the contention that both liquid as well as many solid medications are rapidly absorbed so that time lapse till decontamination rather than its technique has emerged as the most significant contributor to the variance of treatment effectiveness. Often, charcoal fails to be administered for more than an hour after victims have arrived at hospital. We sought data to support or refute the validity of this concern. Method: Two studies were undertaken; the first (Plan A) was a community wide effort seeking data explicitly related to time lapses occurring until: A) recognition

of a poisoning, B) response of 911 team, C) completion of trip to hospital, and D) successful administration of activated charcoal. The second (Plan B) involved a survey of Washington's 110+ hospitals with ED requesting data on delay times between arrival in the ED until completion of charcoal administration. Both studies were classified as ''Quality Assurance Investigations'' thus necessitating neither consents nor Institutional Review Boards' attention. Results: Under Plan A, data from some 50 cases were collected and analyzed. In no instance had charcoal been administered in the field or during the ambulance transport. The anticipated 30–90 minute delay ensued after arrival. In contrast, Plan B's more than 60 responses revealed a mean of 46 minutes delay between arrival in hospital till completion of charcoal administration. Discussion: Recent publications starting with Wax & Cobaugh (1998) have also recorded significant delay times–e.g. 88, 91, 131 and 93 minutes respectively–before charcoal had been successfully administered after ED arrival. In contrast with patients suspected of myocardial infarctions or strokes, time lapse till treatment averages less than 5 minutes in many ED's. Conclusion: While proponents of activated charcoal are adamant in their advocacy position, evidence from existing literature and from our studies raise doubts about using charcoal in the real world. We feel the problem could be rapidly remedied with but a single multi-million dollar negligence award.

83 HAZARDS OF METALLIC MERCURY FROM AN ABANDONED JEWELRY FACTORY

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Objective: Metallic mercury is commonly used in jewelry manufacturing. We describe two related clusters of mercury exposure cases that resulted from the improper removal of metallic mercury from an abandoned jewelry factory. Methods: Following the report that metallic mercury was found on the ground outside an apartment complex, an epidemiological investigation was initiated to trace the source of the mercury and to perform 24-hour urine mercury testing on residents who may have been exposed to the mercury. Results: Two teenage brothers (Brother A lived with mother, and Brother B lived with father 10 miles apart from Brother A) divided a several kilogram cache of metallic mercury that they had taken from an abandoned jewelry factory. Both brothers brought their mercury allotment home and stored it in their respective residences, intermittently showing it to friends. After several months, Brother A decided to throw the mercury into the trash outside his apartment. Unfortunately, the mercury container broke as he was discarding it, resulting in ground contamination. After the mercury spill was identified and confirmed by positive air sampling, Brother A and residents from his apartment complex were brought to the hospital for mercury testing. Brother A's 24 h urine mercury was 46.6 μg/L. Repeat urine mercury 8 days later was 22.8 μg/dL. Of 11 neighbors tested, 3 had urine mercury levels of 15–20 μg/dL with a mean neighbor mercury level of 9.5 μg/L $\pm$ 5.7. Further investigation revealed that Brother B lived next to the jewelry factory separated by a 50-meter alleyway. The ground adjoining his residence was grossly contaminated with mercury. Brother B's urine mercury was 157.5 μg/dL. His car was secondarily contaminated by his mercury contaminated shoes. Brother B's repeat urine mercury level 15 days later without chelation was 88.8 μg/dL. Brother B's father had a urine mercury of 57.7 μg/L. Two other residents from the second residence had a urine mercury of 37.9 and 15 μg/L respectively. The remaining 5 neighbors of Brother B all had urine mercury < 7 μg/dL. Neither brother nor any of the other tested individuals had any symptoms of mercury toxicity, and no one received chelation. Cost of the environmental cleanup of the mercury at the two residences and adjacent grounds was approximately $500,000 USD. Conclusions: The improper disposal of mercury from a jewelry plant has the potential to become a public health problem and requires a costly cleanup. Careful tracing of the origin of the spilled mercury is important in order to evaluate all potentially exposed individuals and to make sure all the mercury can be accounted for.

84 ACUTE ELEMENTAL MERCURY EXPOSURE IN AN INNER CITY COMMUNITY

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Background: Most residential cases of mercury exposure result from chronic exposures. In April, 2000, an acute exposure to elemental mercury occurred within an inner city neighborhood in Allentown, Pennsylvania. Case Series: 73 individuals, the majority of whom were children, were exposed to elemental mercury when a large quantity was found in a garage. This material was divided into small aliquots and sold to children within the community by several individuals. These children applied the material to their skin and to jewelry in play. A large quantity of the material was ignited and burned in a closed space by children. In addition, the material was spilled in several apartments and accumulated on carpets, rugs, clothing, and furniture. Six dwellings were deemed uninhabitable by environmental testing and several families were relocated to hotels. Environmental clean up was organized by local public health officials. Blood mercury

levels and 24-hour urine mercury levels were obtained for most (but not all) of the exposed population. 30% of exposed individuals had elevated mercury levels however no acute illness was reported. Follow up was complicated by inconsistent community cooperation. Conclusion: Mass acute elemental mercury exposure can present a public health challenge of enormous proportions with regard to risk assessment and risk communication as well as remediation and treatment. The lack of legal mandate requiring testing of exposed individuals and medical treatment when indicated further complicates the public health issues in such incidents.

85 MERCURY POISONING OF A NEONATE WITH MAJOR OMPHALOCELE

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

86 PERCUTANEOUS MERCURY INTOXICATION IN YOUNG DIABETIC MAN

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Objective: Intoxication due to cream containing mercuric ammonium chloride occurs very rarely. Itching, flushing, swelling and desquamation of the skin, generalized muscle stiffness, muscular tremor, anxiety, depression and paranoid delusions, sleeplessness and irritability, sometimes connected with impaired renal functions or nephrotic syndrome have been reported. Case Report: A 21-year-old man with a history of diabetes type I, used cream containing 10% mercuric ammonium chloride for eczema on his face, neck, and thorax for about 3 weeks. After the end of this treatment he felt very tired and sleepy, and he was sweating and had mild fasciculations of his extremities. His stable diabetes began to became less well controlled, and during one month his total daily insulin dose had to be doubled. Nephrotic syndrome was diagnosed and the patient was treated with ACE inhibitors and beta blockers. He was admitted to our hospital 2 months after the cream application. At this time he was very weak with an unsteady gait, almost unable to walk, tremors of the hands, and had lost almost 20 kg of body weight. He was depressed, anxious, sleeping during the day, but unable to sleep at night. His behaviour and speech suggested an acute psychosis. The patient had proteinuria 4.5 g/day, and his urine mercury level on admission was 0.252 mg/L. He was treated with Dimaval (sodium-2,3-dimercaptopropane (-1) sulfonate) capsules. for 12 days. The highest urine mercury level during antidote treatment was 2.100 mg/L, and the last one 0.052 mg/L. His clinical condition improved rapidly during the first two weeks, particularly his general demeanor, tremor, mood, and sleeping pattern. However, signs of nephrotic syndrome, peripheral polyneuropathy and the requirement for increased insulin doses were still present two weeks after the end of antidote treatment. Renal biopsy revealed signs of membranous glomerulonephritis and no apparent diabetic nephropathy. Similarly, the peripheral neuropathy did not have typical signs of diabetic neuropathy. Conclusion: We describe an unusual case of percutaneous mercury intoxication in a patient with eczema and type I diabetes mellitus. The patient had pronounced symptoms of mercury intoxication and significant deterioration in his diabetes. At the end of the antidote treatment, when only low quantities of mercury were being excreted in urine, signs of neurological and renal damage persisted, these were not

typical for diabetes. Further follow-up is necessary in this case, who may have been more susceptible to mercury because of his pre-existing eczema and diabetes. Acknowledgement: The case report was supported by MSMJ13/98 111100005.

87 INTRABRONCHIAL ASPIRATION OF ELEMENTAL MERCURY: A 14-MONTH FOLLOW-UP

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Objectives: We report an unusual case of aspiration of elemental mercury. Only 7 cases have been reported over the past 40 years, predominantly following aspiration of mercury during removal of Miller-Abbott tubes. Toxicity seems uncommon although one patient died (Zimmerman, 1969). Inhaled as a vapor, elemental mercury is almost completely absorbed and diffuses rapidly across the cell membranes. The central nervous system, kidney and lungs are key targets of mercury vapor toxicity. Case Report: A 21-year-old man aspirated a large amount of elemental mercury from an unidentified apparatus by accident (precise circumstances are unclear). He remained asymptomatic for two days. He was admitted to hospital complaining of vomiting and faintness. The only objective pathological finding on admission was widespread metallic deposits on X-ray. Six weeks after aspiration only mild EMG signs of motor and sensory nerve conduction delay in the lower limbs were detected. Emotional lability and mild short-term memory loss were observed at the end of 14-months follow-up. The kidney function was normal with only intermittent proteinuria up to 0.5 g/24 hr. Spirometry was normal during the whole period of follow-up. A decrease of density of radiopaque mercury particles in lungs on X-ray was only seen in month 14. Despite intermittent chelation therapy with DMPS, Dimaval (2,3-dimercaptopropane sulfonic acid sodium salt) and Metalcaptase (Penicillamine) the levels of Hg in plasma and urine 10 months after aspiration remained markedly elevated, 124 μg/L and 382 μg/L, respectively (maximum Hg plasma and urine concentration during the follow-up were 252 μg/L and 6,130 μg/L, respectively). Except for the symptoms on admission and later intermittent mild arthralgia the patient did well. Conclusion: There was no evidence of typical features of chronic mercurialism in this patient after intrabronchial aspiration of liquid elemental mercury even though large amounts of mercury were present in the lungs after 14-month follow-up. Plasma and urine levels remain elevated and the long term prognosis is uncertain. Reference: Zimmerman, JE. Fatality following metallic mercury aspiration during removal of long intestinal tube. *JAMA* 1969;**11:** 2158–2160.

88 DO POISON CENTER TRIAGE GUIDELINES ADVERSELY AFFECT MEDICAL OUTCOMES AS HEALTHCARE REFERRAL VALUES INCREASE?

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Objective: Many US poison centers use internal triage guidelines to help Specialists decide when to refer patients to hospitals for treatment. Such guidelines often tie healthcare referral to a specific dose called a healthcare referral value. When the value is met or exceeded the patient is referred to a healthcare facility for evaluation and treatment. This study examined whether patient outcomes were adversely affected as healthcare referral values increased for two common poisonings: acute, unintentional acetaminophen (APAP) poisonings and acute, unintentional iron (Fe) poisonings. Methods: An adverse effect was defined as the patient becoming symptomatic while being treated at home. Qualifying 1997 exposures were separated by substance (APAP or Fe) and then further stratified into three healthcare referral value ranges. Symptomatic and asymptomatic patients were totaled for each stratum. The distribution of symptomatic to asymptomatic patients was compared in each referral value stratum, by using chi-squared test for independence with Bonferroni inequality to correct for multiple comparisons. Results: Study data are summarized below:

Table 1

APAP Referral Values and Outcomes

	HCF Referral Value (mg/kg)	# of Centers	Asymptomatic	Symptomatic (%)
High	>179	17	2869	90 (3.0)
Medium	145–179	25	3858	121 (3.0)
Low	<145	15	1384	53 (3.7)

Table 2

FE Referral Values and Outcomes

	HCF Referral Value (mg/kg)	# of Centers	Asymptomatic	Symptomatic (%)
High	>50	26	1047	81 (7.7)
Medium	30–50	21	833	76 (9.1)
Low	<29	9	232	19 (8.2)

There were no statistically significant differences in the distribution of symptomatic patients within referral value strata for APAP (P = .44) and for Fe (P = .61). Conclusion: There was no evidence of adverse medical outcomes associated with referral values as high as 200 mg/kg for APAP and as high as 60 mg/kg for Fe.

89 POISON CONTROL CENTER SURVEILLANCE OF ADOLESCENT TOXIC EXPOSURES IN THE WORKPLACE: TRENDS IN THE UNITED STATES FROM 1993–97

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Objectives: We compared the toxic agents involved in and severity of work-place poisonings involving adolescents in different regions of the US. We also studied the monthly trends in the proportionate incidence of adolescent vs. adult workplace poisonings in the United States over a five year period. Methods: An analysis of occupational toxic exposures occurring in the US between 1993-97 was performed using the American Association of Poison Control Centers's (AAPCC) Toxic Exposure Surveillance System (TESS) database. A validation study was also performed comparing entries in the TESS database with redacted paper records from a sample of poison control centers. States in the country were clustered into 4 groups for comparative purposes. Contingency tables with the X^2 statistic were used to test bivariate associations. Logistic regression was performed to investigate frequency trends. Results: Between 65–75 poison control centers, with a combined catchment covering more than 95% of the United States population, contributed data to TESS during the study period. Of 301,228 US workplace toxic exposures occurring over the 5 years, 3.1% (n = 8,779) involved adolescents <18 years old. More exposures overall were recorded in Southern states (32.5% vs. 22.5% elsewhere), although a greater proportion (17.6% vs. 14.5% elsewhere) of the severe exposures were reported from the Midwest. Over 37% of these toxic exposures were triaged to a health care facility; 2.2% were hospitalized; 2 children died from their injuries. As the Table shows, a disproportionate number of toxic exposures in the workplace involved males ($p < 0.001$ across regions); toxins involved varied considerably between regions (all except acids $p < 0.05$):

	N	Male	Alkali	Fume	Cleaner	Bleach	Acid	Hydrocarbon
West	2230	61.5%	16.7%	6.4%	14.2%	9.7%	7.6%	5.8%
South	2850	62.1%	8.5%	18.8%	6.1%	7.4%	6.5%	6.7%
Midwest	2587	67.0%	15.1%	11.0%	10.2%	8.3%	7.7%	7.2%
East	1112	66.1%	13.8%	9.0%	8.7%	7.6%	6.6%	9.2%

The proportionate frequency of exposures occurring among adolescents vs. adults increased from 1993–97 ($p < 0.001$) in the United States. Trends in each region varied, but generally reflected an increasing frequency. Conclusion: Occupational toxic exposures involving adolescents are an under-recognized but important hazard in the US, increasing in frequency in all regions. This trend may reflect an increasing risk of exposures, an increasing number of adolescents working in a tight labor market, and/or an increased utilization of poison control centers. Many of these exposures seem potentially preventable with adequate training and precautions; proper eye and skin protection could have averted almost 50% of these exposures.

90 REVIEW OF CHEMOLUMINESCENT GLOW STICK EXPOSURES

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Objective: Ingestion of dibutyl phthalate, the active toxic ingredient in chemoluminescent products, has been reported to cause significant symptoms including gastrointestinal hemorrhage, metabolic acidosis, and even death in massive overdose. Exposures involving chemoluminescent products containing dibutyl phthalate, particularly glowing plastic rods commonly called "glow sticks" or "light sticks," are increasingly common. This is a review of human exposures involving chemoluminescent products reported to the New York City Poison Control Center, an urban poison control center serving a catchment of 7.3 million people, in the past year. Methodology: Computerized records of all telephone reports made to our poison center were retrospectively reviewed and exposures to chemoluminescent products were evaluated with regards to demographic group exposed, type of product responsible for exposure, circumstances of exposure, symptomatology at time of initial report and symptomatology at time of follow-up. Results: Forty human exposures were reported, involving 4 adults (≥18 years), 8 teenage children (13–17 years) and 28 younger children (0–12 years). The preponderance of exposures involved glow sticks (n = 38) and other exposures involved a glowing necklace (n = 1) and a glowing bracelet (n = 1). Regarding symptomatology, the following were noted: Only patients exposed to an opened container reported symptoms (n = 18). These symptomatic patients complained exclusively of irritation at the site of exposure, 16 involving the mouth or throat, and two involving ocular exposure. No patient who reported ingesting an intact glow stick had symptoms at the time of reporting or during follow-up. All adults reporting exposure had inadvertently swallowed an intact light stick while dancing at a dance club or dance party and none developed symptoms. The preponderance of exposures (n = 35) occurred on the weekend, defined as after 5 p.m. on Friday and before 8 a.m. the following Monday. Conclusions: Exposure to chemoluminescent products is increasingly common. To some degree, particularly in adults, this is likely the result of their popularity and use at dance clubs and parties. The clustering of exposures on weekends also suggests that recreational use of these products may be a contributory factor. Most exposures involve asymptomatic ingestion of an intact glow stick. In this series, the only symptoms that resulted from exposure occurred with a broken product that leaked fluid. The only symptoms reported were transient, self-limited irritation at the site of exposure, typically the mouth or throat. The reviewed exposures to chemoluminescent products infrequently resulted in symptoms and the symptoms reported were minor. Exposures to chemoluminescent products may be unlikely to cause significant morbidity or mortality, but such a conclusion cannot be drawn from these results at this time.

91 ASSESSMENT OF ACUTE POISONINGS BY POISONING SEVERITY SCORE

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Objective: The poisoning severity score (PSS) is a standardized and generally applicable scheme for grading the severity of poisoning. It was developed by the International Programme on Chemical Safety, the Commission of the European Union, and the European Association of Poison Centres and Clinical Toxicologists. This scheme allows a qualitative evaluation of morbidity and facilitates comparability of data. It grades the severity of poisoning at three levels: minor (1), moderate (2), and severe (3). On either side of these grades there are the extremes: asymptomatic cases with no symptoms at all (0) and cases with fatal outcome (4). Methods: The poisoning severity score was determined for all patients on admission over a three month period at the National Poison Control Centre in Belgrade. Results: The 588 patients were initially graded in this way: 210 (35.7%) as PSS 0, 229 (38.9%) as PSS 1, 100 (17.0%) as PSS 2, and 49 (8.3%) as PSS 3. 464 patients (78.9%) were managed as outpatients in the Emergency Room only and the rest were hospitalized. The initial severity grades are detailed in the following table.

PSS	Outpatients	Admitted to Hospital	Total
0	197 (93.8%)	13 (6.2%)	210 (100%)
1	190 (83%)	39 (17%)	229 (100%)
2	51 (51%)	49 (49%)	100 (100%)
3	25 (51%)	24 (49%)	49 (100%)

Most of the patients initially graded as 2 and 3 managed in the emergency room were poisoned by ethanol or heroin. 124 patients were admitted to hospital. The reason of admission for patients with PSS 0 and 1 was for clinical observation. Only one patient initially graded as PSS 1 deteriorated and developed moderate (PSS 2) symptoms. 13 patients died–(PSS 4); two of them were initially graded as PSS 2, and eleven as PSS 3. Conclusion: PSS is helpful in assessing accurately the clinical severity of poisoning, and the likelihood of further deterioration.

92 IMPACT OF CALL DIVERSION ON ENQUIRIES TO A NATIONAL POISONS INFORMATION CENTRE

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Objectives: The National Poisons Information Centre, Dublin, Ireland contracted the National Poisons Information Service (Cardiff Centre), Wales to answer its nighttime enquiries from 1st May 2000, due to staffing problems. Calls received between 10 p.m. and 8 a.m. are diverted automatically to Cardiff. Hospital Accident and Emergency (A&E) departments which regularly use the service were given advance notice of the new arrangements. The aim of this study was to see if these arrangements had any impact on the number and nature of night-time enquiries. Methods: We retrospectively compared night-time enquiries between 1st May and 30th September inclusive, in 1999 and 2000. We also conducted a telephone survey of A&E departments to see if they had changed their protocol for contacting the NPIC. Results: 1848 night-time enquiries were received during the study period in 1999 and 1769 in 2000 ($p > 0.10$, Chi square test). The number of day-time (8 a.m.–10 p.m.) enquiries was similar during both study periods (4557 enquiries in 1999 and 4579 in 2000). In 1999, 1622 (88.0%) night-time enquiries were from hospitals, 77 (4.2%) from general practitioners/primary care and 141 (7.6%) from members of the public. 647 (35.0%) enquiries were received between 10 p.m. and midnight. 972 (52.6%) patients were female, 763 (41.3%) male and the gender of 113 (6.1%) was unknown. 236 (12.8%) enquiries were about children (0–9) years, 331 (17.91%) about adolescents (10–19 years), 1042 (56.4%) about adults (≥20 years) and the age of 239 (12.9%) patients was unknown. In 2000, 1615 (91.3%) night-time enquiries were from hospitals, 48 (2.7%) from general practitioners/primary care, 97 (5.5%) from members of the public and 9 (0.5%) from other types of caller. 587 (33.9%) enquiries were received between 10 p.m. and midnight. 1010 (57.1%) patients were female, 699 (39.5%) male and the gender of 60 (3.4%) was unknown. 208 (11.8%) enquiries were about children (0–9) years, 364 (20.6%) about adolescents (10–19 years), 1092 (61.7%) about adults (≥20 years) and the age of 105 (5.9%) patients was unknown. None of the 34 A&E departments surveyed had altered their protocols for contacting the NPIC since we contracted the night-time service to Cardiff. Conclusions: Diverting night-time enquiries to a poisons information centre in another country had no significant impact on calls to a national poisons information centre. Medical and nursing staff in hospital A&E departments, who had been informed about the new arrangements, continued to use the service.

93 NATIONAL MULTICENTER STUDY OF ACUTE INTOXICATIONS IN SPANISH EMERGENGY DEPARTMENTS

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Objective: There are no studies about the kind of acute intoxications observed in Spanish Emergency Departments (ED). We designed a multicenter study to identify the epidemiology and management of intoxicated patients in Spain. Methods: Prospectively, we recorded cases from 14 EDs during 14 randomly selected days between February and April 2000. We included all age patients and all acute intoxication cases (including alcohol), except alimentary cases, bites, inert foreign bodies and deaths before arrival to the ED. Results: 419 cases were recorded, 0.66% of ED visits. There were more cases on the weekends and on Mondays ($p < 0.001$). The mean age was 33 years (ST 18.10); 56% of cases were males. 54% of patients arrived at the ED within 4 hours, and 35% later than 8 hours. 77% were deliberate intoxications (26% alcohol), 6% accidental and 6% illicit drug abuse. 30% of cases had a previous intoxication history (20% alcohol intoxication) and 40% had a psychiatric history. Pharmacological drugs were involved in 42.7% of cases, domestic products in 9.5%, illicit drugs in 10%, agricultural products in 1.7%, and alcohol in 64% (alone or associated with other substances). The most common drugs involved were benzodiazepines (57%, $p < 0.01$), as well as NaOH, CO and Cl_2 vapours. 65% of patients went directly to the ED. Only 5% were comatose and 15% asymptomatic. Treatment was clinical observation alone or symptomatic in 71.6%, antidotes in 20.76%, and forced renal elimination in 2.14%.

Blood samples were obtained in 35% and Urine Triage test in 2%. 80% were outpatients (50% within first 12 hours), 3% were admitted to the ICU, 6% to the hospital, 5% left ED without medical permission, and 0.2% died. Discussion: We have recorded a slightly lower incidence of acute intoxications than other European countries, but with the same epidemiological profile. There is a lower incidence of acetaminophen cases. Most patients were treated in the ED, without hospital admission.

94 THE IMPACT OF A NATIONAL INTERNET POISONS INFORMATION SERVICE. 2. PATTERN OF ENQUIRIES

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Objective: To investigate agents accessed on a national poisons internet database by different categories of users. Methods: All hits on our website for the first 10 months of 2000 were entered on an Access database and analyzed by country of origin, agent and user type. Results: The number of products accessed on TOXBASE® by each of the four UK countries was England (103,010), Northern Ireland (14,994), Scotland (22,427) and Wales (9,609). The 10 most common agents involved in product monographs accessed on TOXBASE® for the four countries are compared in Table 1.

Table 1

ENGLAND	N IRELAND	SCOTLAND	WALES
Paracetamol (11372; 11.0%)	Paracetamol (2647; 17.7%)	Paracetamol (2397; 10.7%)	Paracetamol (847; 8.8%)
Ibuprofen (2880; 2.8%)	Codeine (638; 4.3%)	Aspirin (591; 2.6%)	Aspirin (266; 2.8%)
Aspirin (2637; 2.6%)	Diazepam (580; 3.9%)	Ibuprofen (574; 2.6%)	Ibuprofen (194; 2.0%)
Codeine (2436; 2.4%)	Aspirin (485; 3.2%)	Codeine (527; 2.3%)	Codeine (187; 1.9%)
Zopiclone (2235; 2.2%)	Ibuprofen (385; 2.6%)	Diazepam (417; 1.9%)	Caffeine* (149; 1.6%)
Diazepam (1750; 1.7%)	Thioridazine (320; 2.1%)	Fluoxetine (405; 1.7%)	Diazepam (129; 1.3%)
Fluoxetine (1738; 1.7%)	Fluoxetine (302; 2.0%)	Paroxetine (380; 1.7%)	Ethanol (119; 1.2%)
Paroxetine (1722; 1.7%)	Temazepam (302; 2.0%)	Caffeine* (367; 1.6%)	Ecstasy (1191.2%)
Caffeine* (1647; 1.6%)	Codeine (285; 1.9%)	Thioridazine (343; 1.5%)	NaClO (117; 1.2%)
Thioridazine (1621; 1.6%)	Zopiclone (281; 1.9%)	Zopiclone (339; 1.5%)	Zopiclone (104; 1.1%)

* Caffeine from compound analgesics

Paracetamol was top in all countries, accessed approximately four times more frequently than the next most common agent. The overall pattern of frequent enquiries is similar in the four geographical regions. Three categories of users were analyzed–hospitals (139,258 product accesses), NHS Direct centres (which provide direct access to NHS advice from specially trained nurses using computer triage systems, 8504 accesses) and general practitioners (1502) and results are shown in Table 2.

Table 2

HOSPITALS	NHS Direct	GPs
Paracetamol (16454; 11.8%)	Paracetamol (696; 8.2%)	Paracetamol (95; 6.3%)
Aspirin (3852; 2.8%)	Sodium hypochlorite (257; 3.0%)	Sodium hypochlorite (56; 3.7%)
Ibuprofen (3789; 2.7%)	Ibuprofen (202; 2.4%)	Ecstasy (38; 2.5%)
Codeine (3627; 2.6%)	Ethanol (185; 2.2%)	Sodium hydroxide (34; 2.3%)
Zopiclone (2928; 2.1%)	Sodium hydroxide (179; 2.1%)	Paraquat (23; 1.5%)
Diazepam (2795; 2.0%)	Codeine (162; 1.9%)	Ethanol (21; 1.4%)
Fluoxetine (2458; 1.8%)	Solvents (158; 1.9%)	Detergent (17; 1.1%)
Caffeine (2356; 1.7%)	Calcium carbonate (147; 1.7%)	Surfactants (16; 1.1%)
Paroxetine (2336; 1.7%)	Hydrocarbons (122; 1.4%)	White spirit (15; 1.0%)
Thioridazine (2335; 1.7%)	Terpineol (114; 1.3%)	Calcium carbonate (15; 1.0%)

Top agents accessed by hospitals are pharmaceuticals while for NHS Direct centres and GPs many of the top agents are common ingredients of household products. Conclusions: The type of enquiry to this Internet database varies with the type of enquirer. The pattern of database enquiries is similar in the 4 countries of the UK.

95 FORTY YEARS POISON INFORMATION SERVICE IN FINLAND—WHAT HAS CHANGED AND WHAT HAS NOT

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Objective: The Finnish Poison Information Centre was founded in 1961. The call statistics through 1999 were analyzed to better understand how the operations have changed. Methods: The Centre is the only one in Finland and serves the whole country. During 1961–1999 it received more than 538,000 calls. It has always accepted calls from both the public and health-care providers. Data collection and classification have changed in many ways during the years, so only comparable variables were used for the analysis. Results: The total number of calls increased almost yearly, and was 37,216 in 1999 (720 calls/100,000 inhabitants). The percentage of general inquiries grew from 3.8% in 1972 to 22.4% in 1999, while the calls related to actual poisonings slightly decreased from 95.1% in 1972 to 73.0% in 1999. Since 1994 more than 50% of the calls came from the public, and over 80% after 1997. The number of calls received from the health-care providers (mainly physicians) was 3000–4000 between 1992–1996 but increased to 4700–5000 thereafter. Until 1997 >60% of calls concerned children less than 6 yrs old. The age group >16 yrs represented >20% of calls until 1996 and since then exceeded 30%. The major groups of substances involved remained remarkably unchanged throughout the years. Medicinal products were always the largest group and were involved in 30–40% of poisonings. The largest changes were observed in the recommendations for treatment. After 1980 >50% of the calls could be managed at home and in the 1990s 75–85%. In 1975, when the first numbers were available, induced emesis was recommended in 24.6% and activated charcoal in only 10.9%. The recommendation to induce emesis decreased to less than 1% in 1996. Between 1981–1992 activated charcoal was recommended in >30% of the cases, in 1998–1999 in 15–16%. The number of no recommended interventions mirrors the recommendations to use activated charcoal and has increased from a minimum of 6.8% in 1991 to 15% in 1999. Conclusions: The demand for the services of the Finnish Poison Information Centre has increased continuously. The increased number of calls has come more from the public than from the health-care providers. The number of general inquires has increased more than the number of calls related to actual poisonings. Medicinal products have always been the largest group of substances involved. Most of the poisonings have been managed at home. Treatment recommendations have varied with the medical trends. Induced emesis is currently rarely recommended. Activated charcoal is the most often recommended treatment, and it may have been over enthusiastically used in the 1980s.

96 SUICIDE ATTEMPTS IN CHILDREN AND TEENAGERS: EXPERIENCE OF THE MARSEILLE POISON CENTRE DURING 1997 AND 1998

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Objective: The number of suicide attempts in France is estimated at about 150,000 cases a year. Epidemiological studies usually collect data about suicidal adults. The Marseilles Poison Centre examined the data on suicide attempts by patients less than 15 years of age for the period 1997–1998. Cases series: 706 cases were studied (88% girls, 12% boys). The average age of the patients was 13 years (range 6–15 years). 57% of them had no previous history, 24% had depression or psychiatric disorder, 10% of the toxic ingestions were recurrences. The reasons for the ingestion are not clearly defined: 42% had no particular reasons, 17% for depression, 12% family conflict, 8% severe illness, 6% family psychiatric disorder, 5% obesity, 4% low socio-economic status and 3% school problems. Symptoms were absent in 30% of the cases. The most common clinical signs were drowsiness 55%, abdominal pain 20%, vomiting 13%, behavior disturbances 6%. In 95% of cases medicines were ingested (benzodiazepines 44%, acetaminophen 19%), 4% ingested household products and 1% industrial or agriculture products (10% of the boys and 4% of the girls ingested products other than pills). The toxic ingestion was associated with alcohol in 3% of the cases. 19% of the patients stayed at home, 5% consulted an emergency unit and went back home, 76% were treated in hospital (4.4% in intensive care units, including 3.8% of the girls and 8.4% of the boys). 18% only of the 536 hospitalized patients had a psychiatric consultation. The average duration of hospitalization was 1.17 days (range 1–5 days). In hospital, 80% had enhanced diuresis, 20% activated charcoal, 18% gastric lavage and 13% antidotes (acetylcysteine, flumazenil, naloxone). 79% of cases recovered and 1% had complications (seizures, rhabdomyolysis). There were no fatalities. Conclusion: The

data on parasuicide by children and teenagers for the period 1997–1998 were analyzed. Attempted suicide by ingesting toxins is seen mainly in girls and rarely in boys.

97 ACUTE POISONING EPIDEMIOLOGY TRENDS TO IMPROVE THE HEALTH SERVICES FOR POISONING TREATMENT

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Objectives: In the Middle Ural (population 4.5 million) the number of poisoning cases is rising each year. The substances involved have also changed. Methods: The epidemiology of acute poisonings in the last thirty years have been recorded by the Middle Urals Regional treatment center for acute poisonings. Statistics from general hospitals, specialized treatment centers for poisonings and telephone-call consultations were used. Results: In the 1970s the number of patients admitted to hospitals because of acute poisonings ranged from 4,984 in 1971 to 6,322 in 1979, 11.5–16.5% of them being children. An average mortality rate was 4.6 ± 0.14%. During that decade the substances most frequently involved in human exposure were corrosive poisons (30%), medicines (25.5%), carbohydrates (17%), alcohols and glycols (15.9%), pesticides (5.2%). By 1989 the number of patients reached 7,780 (25% children), the death rate rose to 5.36 ± 0.26%. The poisoning substances altered greatly in the 1980s: poisonings with medicines ranked first (37.3%), corrosive poisons (21.4%), pesticides (19.5%), carbohydrates (14.3%), alcohols and glycols (13.2%). In 1999 12,237 acute poisonings (10.1% children) were registered, the mortality rate fell to 3.2%. In addition to a marked rise in poisonings the substances involved also changed in the 1990s: alcohols and glycols 33.4%, medicines 27.9%, opiates 19.6%, corrosive poisons 10.5%. We assume the decrease in mortality over the years can be ascribed to the foundation of specialized treatment centres for poisonings in the region. The first centre of that type appeared in 1973, and the second in 1988. Since 1998 six centres have been active in five large cities in the region. At present about half the patients with acute poisonings are treated in specialized centres. Specialists from the general hospitals are able to have telephone-call consultation with the 24 hour information service of the Regional treating centre for acute poisonings. The centre also teaches and certifies specialists in clinical toxicology. If the need arises a toxicologist from the centre goes to hospitals up to 300 km away by ambulance or by helicopter to provide necessary medical aid and to transport the patient to the Regional centre. Conclusion: Toxicology information and treatment service need further development and improvement as the incidence of poisoning rises and the substances change.

98 A TOXIC EVENT SURVEILLANCE SYSTEM IN THE EMERGENCY DEPARTMENT OF SPANISH HOSPITALS

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Objective: The importance of knowing the health effects of toxic exposures by chemical products has been long recognized; the US Toxic Exposure Surveillance System includes around 25 million human poison exposures reported to US poison centres. Discrepancies in epidemiological estimates have been attributed to incomplete reporting by member hospitals. In 1999 the Health Ministry in collaboration with the Clinical Toxicology Section of the Spanish Association of Toxicology decided to develop a program looking at the toxic cases from chemical products (excluding drug overdoses and plant and animal poisonings) that reach the Emergency Departments of Public Hospitals. Methods: Data was submitted by members of the emergency department staff of the participant hospitals. The clinical data for each patient include: sex, age, symptoms, treatment and outcome and product identification, exposure cause, exposure place and exposure route. We present here the results of the first 18 months of the program. Results: There are 14 participant hospitals which reported a total of 847 cases. Admission was required in 251 cases (29.6%). Mean age was 37.7 years. Males represent 52.7% and females 47.3%. Reason for the exposure was accidental in 718 cases (82.2%), suicidal in 94 cases (11.1%) and unknown in 35 cases (4.1%). Accidental exposures occurred at home in 527 cases. The chemical compounds were toxic gases 268 cases (31.6%), caustics 272 cases (32.1%), solvents 59 cases (7%) and detergents 51 cases (6%). The most frequent agent was domestic bleach (205 cases) followed by CO (144 cases). The route of exposure was oral in 324 cases, respiratory in 332 cases, cutaneous in 61 cases and ocular in 167 cases. 749 cases had some symptoms: neurologic 175, respiratory 177, digestive 265, cutaneous 42 and ocular 157. Treatment was given in 713 cases: gastric decontamination in 71, cutaneous or ocular decontamination in 93, antidotes in 152, enhanced elimination in 8 and symptomatic measures in 547 cases. Mean time in hospital was around 24 hours. There were 14 deaths (methanol,

paraquat, HCl, pesticides and CO). Most of the non-lethal cases had a good outcome with a few minor sequelae. Conclusion: This program analyzed the types of poisoning by chemical products and will help to develop a Poisons Prevention Program.

99 THE ROLE OF A POISON CONTROL CENTRE IN THE PREVENTION OF BLEACHING CLEANERS INTOXICATIONS

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Objective: Household, industrial and hospital products containing sodium hypochlorite as a bleaching cleaner and disinfectant are widely used in this country. Household bleaches contain a concentration of sodium hypochlorite less than 6%. However when it is mixed with an acid or ammonia, subsequent chlorine or chloramine gas forms which is quite irritant to the respiratory tract. Twenty six percent of the total number of intoxications received in our Centre were due to household cleaners. Sodium hypochlorite was implicated in 17% of this type of intoxication. Therefore, the calls were investigated in order to develop appropriate control measures. Methods: 1142 human inhalation exposures related to products containing sodium hypochlorite alone or combined with an acid (i.e. hydrochloric acid, sulphuric acid or phosphoric acid), or with ammonia were recorded during the first six months of the present year. Data including age, gender, type of product, evolution, and clinical signs and symptoms, as well as the circumstances of exposure and aetiology were analyzed. Results: Eighty-one of the studied cases were female and 19% male. Ninety seven percent were adults. Aetiology was "accidental" in 100% of the cases, ninety-one of them were household accidents and 7% were occupational. The vast majority of the cases were caused by the mixture of sodium hypochlorite plus ammonia (42.5%), followed by sodium hypochlorite plus acid (33.8%). Only 23.6% of the exposures were caused by sodium hypochlorite alone. Clinical manifestations after exposure to mixtures were more significant and severe than those occurring with sodium hypochlorite alone. Thirty-five percent of these patients were asymptomatic. Clinical features recorded were: dyspnoea due to sodium hypochlorite plus acid (55% of the exposures) and due to sodium hypochlorite plus ammonia (33.3%). Cough due to sodium hypochlorite vapour inhalation was observed in 29.3% of these accidents. Conclusions: Due to the common use of sodium hypochlorite combined with other bleaching cleaners such as acids or ammonia, exposures to chlorine and chloramine gas are very frequent. Mixing of bleaching cleaners is often associated with significant respiratory symptoms. Our centre proposed measures to prevent these exposures in the future to the manufacturers. Regulations developed by the authorities for the same reason are being implemented.

100 CLOSTRIDIUM DIFFICILE INFECTION AND PSEUDOMEMBRANOUS COLITIS RESULTING FROM LONG TERM OCCUPATIONAL INHALATIONAL EXPOSURE TO CEPHALOSPORINS

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Objectives: Inhalation exposure to antibiotics has been reported in the pharmaceutical manufacturing industry as a result of worker exposure to airborne particle loads of antibiotic dusts and aerosols during the manufacturing and container filling processes. Clostridium difficile is an important pathogen and is seen almost exclusively as a complication of antibiotic therapy. It is particularly associated with exposure to third-generation cephalosporins. Depletion of the indigenous gut microflora by antibiotic therapy has long been established as a major factor in the development of this disease. We are reporting the first case of C. difficile infection and pseudomembranous colitis secondary to long term inhalational exposure to and presumed pulmonary absorption of airborne cephalosporins in the workplace. Case Report: A previously healthy 26-year-old microbiologist with no significant family history or past medical history developed moderately severe pseudomembranous colitis. The patient had not taken antibiotics prior to the onset of this illness. This individual was employed by a pharmaceutical company and was exposed to airborne dusts and aerosols from at least six different third generation cephalosporins during container filling operations over a three month period. Environmental testing revealed the presence of cephalosporins in the ambient air and cephalosporin residuals throughout the workplace including the surrounding grounds and the parking lot adjoining the worksite. Random surface swipe samples revealed the presence of cephalosporin residuals in the patient's private motor vehicle which she drove to and from work as well as on her hair, skin, and clothing. After three months of daily exposures to this environment, the patient developed severe crampy abdominal pain associated with bloody diarrhea with onset typically on the weekends and resolution

before the start of the next work week. Clinical investigations revealed colonoscopy proven and culture proven C. difficile related infection and pseudomembranous colitis. Removal from exposure was recommended and the patient was treated with oral metronidazole. With this therapy, her symptoms resolved. Conclusion: This is the first reported case of the development of C. difficile infection with pseudomembranous colitis resulting from occupational exposure to airborne cephalosporin antibiotics.

101 AN INCIDENT INVOLVING A COMMERCIAL FOX REPELLENT

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Objective: We report a case of human exposure to a bone-oil pest repellent. Renardine is advertised as a non-toxic repellent of cats, dogs, rabbits, foxes, moles and badgers. The manufacturer describes it as a dark brown flammable liquid that can cause discomfort on contact with eyes and skin, but the possibility of symptoms after inhalation is not recorded. It contains 30% bone oil and its strong smell confuses animals into believing rivals are present so that treated areas are avoided. Renardine is reported to have been used in the UK during the 1990s to repel badgers and has been tested in Canada to protect sheep from coyotes. Case Report: The Vale Resource Centre, Hen Goleg, in Barry, Wales provides services for people with physical disability or chronic illness and their caretakers. In early June 1998 a fox had become established below the ground floor. A subsequent flea infestation caused part of the building to be closed. To repel the fox, renardine was selected since it was thought to be non-toxic and humane. The treatment was unfortunately applied while the center was occupied. Within hours, the staff was complaining of the smell. Complaints continued throughout the following day. The centre closed, remains of the renardine were removed and ventilation of the building was increased. The centre re-opened the following week although the smell continued to linger, despite efforts to increase ventilation. Due to continuing concerns the centre closed for a second time thirteen days after the initial application. Camera survey of the ventilation shaft revealed a large void under the main hall. Forced air ventilation was used to attempt to remove any remaining chemical from dead air pockets. Air testing failed to detect toxic constituents of bone oil (particularly pyridine and aniline). Within the first two weeks twenty-nine members of staff had visited their general practitioner. The most commonly experienced symptoms were sore eyes, dry throat, nausea and headache. Symptoms also included a bad taste in the mouth, chest pain and cough. A meeting of social services management, expert panel and staff decided that it was safe for the centre to open, nearly three months after the Renardine was applied. Conclusion: We conclude that inhalation of this type of repellent may be associated with symptoms, and appropriate preventive procedures should be employed when it is used.

102 BARKING BACLOFEN!

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Introduction: Since 1992 the London centre of the National Poisons Information Service (NPISLn) has provided a subscription-based service to veterinarians seeking poisons information for animal cases. Many of these enquiries involve animal exposures to human medications that prove rather more toxic to the animals than expected from knowledge of their effect on man. Objective: A retrospective review of all consecutive veterinary cases from 1984–2000 involving canine ingestion of baclofen. Method: All the NPISLn call record forms were searched for those concerning baclofen exposure in dogs. Since 1992 all call records have been augmented by specific follow-up questionnaires sent to the veterinarian within 14 days of the initial telephone consultation. These returned data allow verification of case information recorded by NPISLn at the time of enquiry, with additional information about the occurrence, clinical course, treatments and investigations instituted, and case outcome. Case Series/Results: From 1984 to date 65 cases of baclofen ingestion by animals have been reported. Of these, 54 (83%) cases involved dogs and the remainder cats. Follow up reports were returned for 35 of these 54 (64.8%) canine cases. Remarkably, only 2/35 dogs remained asymptomatic; one consuming a calculated dose of 1.2 mg kg^{-1}, while the other, an adult Springer Spaniel, ate 60 mg. For average adult Springers, with a body weight of 20 kg, this estimates at 3 mg kg^{-1}. 5 Cases were fatal (3 died and 2 euthanased). For two of these the fatal dose could be calculated at 24 mg kg^{-1} and 30 mg kg^{-1}. Dogs that survived ingested doses between 0.5–16.7 mg kg^{-1}. Where information was available the clinical effects developed rapidly, usually between 30 minutes to 3 hours after ingestion. The predominant effects reported were excess salivation, vomiting, vocalising, ataxia and incoordination. Some animals showed excitability with twitching or shaking, which in some progressed to

convulsions. Other reported effects included hypothermia, bradycardia, shock, collapse and coma. Treatments instituted by veterinarians included gastric decontamination by use of emetics, gastric lavage and adsorbents. Other management was supportive i.e. warming measures, intravenous fluids and use of atropine and anticonvulsants. Most animals had fully recovered by 72 hours post-ingestion. Conclusion: a) Baclofen is potentially very toxic to dogs; b) Doses in excess of 3 mg kg^{-1} should be managed aggressively; c) Absence of clinical effects within 3 hours is a good prognostic sign; d) Patients with spastic conditions such as multiple sclerosis should be warned of the potential hazards of baclofen to both themselves and their pets.

103 LIVER DAMAGE AFTER EXPERIMENTAL SIMAZINE ADMINISTRATION IN AN EXPERIMENT

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Objective: Triazine herbicides are widely used for extensive agricultural production. However, there are ecological and health hazards associated with the use of these herbicides due to water and food contamination. The aim of this study was to evaluate the effects of long-term simazine feeding on the development of hepatic steatosis and these changes of liver bioenergetics in experimental animals. Methods: B6C3F1 mice were fed simazine (2g or 4g/kg/day, respectively) for 35 weeks. The concentration of cholesterol and triacylglycerols were then measured in liver tissue. Liver mitochondria were isolated and parameters of oxidative phosphorylation were assessed polarographically using a Clark oxygen electrode with the NAD substrate glutamate and/or the FAD substrate succinate. Results: Significant changes ($p < 0.001$) expressed as medians (with confidence intervals [CI]) versus control animals were found in both experimental groups after simazine feeding. The concentration of triacylglycerols increased from 10.3 (CI: 8.8–10.9) mmol/kg to 20.1 (CI: 18.0–20.9) mmol/kg and 47.7 (CI: 23.8–56.0) mmol/kg, respectively. The parameters of oxidative phosphorylation with the NAD substrate glutamate decreased as follows. The index of respiratory control fell from 7.7 (CI: 6.4–9.0) to 4.8 (CI: 4.0–6.3) and 4.4 (CI: 3.9–4.6), respectively. The rate of oxygen consumption by mitochondria stimulated with ADP (in state 3) fell from 84.2 (CI: 82.0–92.3) nAtO/mg prot/min to 65.4 (CI: 50.8–70.7) nAtO/mg prot/min and 69.9 (CI: 65.0–78.4) nAtO/mg prot/min, respectively. Oxidative phosphorylation rate fell from 215.3 (CI: 204.4–232.2) nmol ATP/mg prot/min to 166.3 (CI: 120.4–193.6) and 169.6 (CI: 155.3–176.9) nmol ATP/mg prot/min, respectively. Similar trends were observed with the FAD substrate succinate. Conclusion: After long-term simazine feeding in mice liver steatosis developed and hepatic mitochondrial energy production was depressed. However, energy production was sufficient for the maintenance of liver function.

104 WWW INFORMATION QUALITY FOR THE GENERAL PUBLIC: FOCUS ON MUSHROOM POISONING

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Background: The worldwide web (WWW) is an increasingly used source for health information, both by physicians and laypersons. However, many authors have expressed concern about the quality of e-health information accessible by the general public. Objective: To evaluate the quality of WWW information in the Italian language about mushroom poisoning accessible by laypersons. Methods: Sites were identified through Altavista and Yahoo, using ''mushrooms poisoning'' as the search term. Evaluation was limited to the first 100 items for each search engine, and non-pertinent sites were excluded. Criteria to evaluate the reliability of the site were defined using WHO recommendations[1], the Hiti guide[2], and med-PICS labels[3]. Additionally, we searched for the HON logo[4], the most popular ethical code for health-related web publishing. The reliability score (SR, range 0–9) was defined as the number of positive answers to the first 9 criteria reported in Table 1. The information content was evaluated through accuracy (0 = incorrect, 1 = some incorrect, 2 = correct, 3 = referenced statements), comprehensiveness (0 = isolated, 1 = several, 2 = most, 3 = all issues), and objectiveness (0 = disagreement with accepted medical practice, 1 = partial agreement, 2 = full agreement, 3 = full agreement including alternatives) by three independent toxicologists. The content score (SC, range 0–9) was defined as the sum of the answer values for each item. Results: Among 286 sites, 9 had some medical content. Table 1 shows the percentages of occurrences for each criterion. Table 2 shows SR and SC distribution. Two sites reported wrong and potentially dangerous medical advice.

Table 1

Reliability Criteria Compliance

Presence of	% Yes
A stated responsible person or organization for the content	44
E-mail addresses of the responsible person or organization	11
Credentials verifiable by lay persons	33
Stated aim of the site	22
Stated intended reader	11
Currency of the site	22
Clear disclaimer	11
HON symbol	0
Funding policy	22
Medical trained content provider	11
Interactive personal communication facility	66
Privacy policy	0

Table 2

Score Distribution

	0	1	2	3	4	5	over
SR	33%	22%	11%	11%	11%	11%	0%
SC*	11%	33%	33%	11%	11%	0%	0%

* Mean value between toxicologists.

Conclusions: Available internet resources are very poor in term of contents. Compliance with basic rules for health-related information web publishing is low. Medical authors are almost absent, while interactive communication is frequent. We propose that health professionals and scientific associations take an active role in www publishing, thus avoiding low quality information dissemination, and exploiting this technology as an effective educational tool. References: [1]www.who.int. [2]www.hitiweb.mitretek.org. [3]Eysenbach G. Towards quality management of medical information on the Internet. *BMJ* 1998;**317**:1496–1500. [4]www.hon.be.

105 CHEMICAL INCIDENT RESPONSE SERVICE (CIRS) CHRONIC CHEMICAL INCIDENTS SURVEILLANCE IN SIX HEALTH REGIONS IN THE UK, 1999–2000

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Background: Recent environmental legislation (UK and Europe) covering issues relating to land contamination (including Part IIa of the Environment Act 1995), the Integrated Pollution Prevention and Control (IPPC) Directive (effective from 31st October 1999 covering new industrial processes such as incinerators), the New Water Drinking Directive (98/83/EC, December 2003) and the UK Air National Quality Strategy (Autumn 2000) are all aimed directly at low dose environmental exposures. Public Health Authorities are increasingly requesting information and advice from CIRS about these issues. Significantly, the UK Environment Agency anticipates that Health Authorities will, in relation to IPPC processes: A) Advise on local health problems that they consider relevant; B) Assess the likely impact of releases on human health (acute & chronic); C) Identify priority substances for control, from both routine and potential accidental releases[1]. The study objectives were to analyse data of chronic chemical incidents reported to CIRS between January 1999 and June 2000, to determine frequency of chronic chemical incidents, incident type, incident location and health region, and specific chemicals involved. Results: Eighty-nine (10% of annual total) chronic chemical incidents were reported to CIRS in 1999 and in 2000 76 (15% of total) had been reported by June. The increased frequency of Public Health Authorities reporting chronic chemical incidents may be attributable to awareness of the new legislation and

better understanding of chemical incident health impact. Chronic air, land and water chemical incidents are the most common incidents reported to CIRS.

Type	Air	Land	Water	Other
1999	(14) 18%	(13) 16%	(15) 18%	15 (12%)
January–June 2000	(21) 29%	(14) 18%	(6) 8%	13 (17%)

The majority of CIRS chronic incidents in 1999 originated in the South East Health Region; by June 2000 most enquiries were from the North West Health Region. Heavy metals, hydrocarbons and solvents being the most common chemicals involved 1999 and 2000. It is concerning that the specific chemicals were initially unknown in 11% (1999) and 15% (2000) of reported chronic chemical incidents. Significantly most chronic chemical incidents reported occurred in residential areas followed by open space and industrial areas. Conclusions/Recommendations: Chronic chemical incidents are becoming increasingly important public health issues and often prove to be very difficult to investigate and manage. In our experience they may use considerable public health resources. Public Health authorities notify chemical incidents more frequently, which suggests greater awareness of legislation and better implementation of local networks and feedback mechanisms. References: [1]Weston P. IPPC and Health Authorities. *Chemical Incident Report* 2000;**15**:18.

106 STRIKING EXTRAPYRAMIDAL MOVEMENTS SEEN IN LARGE OLANZAPINE OVERDOSES

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Objective: Newer antipsychotic medications such as olanzapine have the advantage of fewer side effects. However, in large overdoses, we have observed extrapyramidal appearing spastic movements. We report 2 cases and review the topic. Case 1: A 29-year-old man had "jerky movements of the arms and legs." He had taken an intentional overdose of his olanzapine, valproic acid, and simvastatin 1–2 hours earlier. Drowsy but oriented, his vitals were normal. Pupils were 3 mm and decreased to 2 mm with light. With extraoccular movements he would have uncomfortable, dramatic, involuntary nystagmus/occulogyric crisis. His eyes would forcefully bound up or to the side for a few seconds, causing him to push the examiner's finger away and close his eyes. Involuntary twitches of his arms and legs were also seen. One observer noted that "it appeared that someone was bumping into the stretcher." He was given diazepam 10 mg IV that dramatically decreased the movements. Serum olanzepine was 120 ng/mL (proposed therapeutic 5–75 ng/mL). Valproic acid was 52 mg/L (therapeutic 50–100 mg/L). He had an uneventful recovery. Case 2: A 33-year-old woman had lethargy and disorientation. She had told her brother that she had taken 30 of her 10 mg olanzepine tablets in an effort to kill herself. Vital signs were normal. She was somnolent but arousable with a simple nudge or shake. She would then open her eyes and shake all over in a one-to-two second total body spasm. Pupils were 2 mm and minimally reactive. A CT scan of the head and a lumbar puncture were normal. Urine olanzapine was >500 ng/mL (upper limit of normal). No serum olanzapine level was available. She was admitted to the intensive care unit and had an uneventful recovery. The Delaware Valley Poison Center in Philadelphia, Pennsylvania, USA, has had 140 calls involving olanzapine in year 2000, 34 of these have been overdoses of olanzapine alone. Four had lethargy, ataxia, and small pupils. One reported trouble moving his tongue, another rigid muscles, and another "jerking" of his extremities. Olanzapine affects many receptor types and subtypes including serotonergic ($5HT_{2A}$, $5HT_{2C}$), dopaminergic (D_1, D_4), adrenergic ($alpha_1$, $alpha_2$), histaminic (H_1), and muscarinic receptors. The extrapyramidal movements seen with large overdoses suggests that with high levels there may be receptors stimulated that are not at therapeutic levels. The small pupils and somnolence is characteristic of olanzapine. Conclusion: If "spastic" movements are seen in a lethargic patient, consideration must be given to a large olanzapine overdose. As with routine olanzapine overdose, the treatment is still supportive care, with the addition of a benzodiazepine if tolerated.

107 HYPOTENSION IN AN UNINTENTIONAL OVERDOSE WITH TIZANIDINE

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Background: Tizanidine, an imidazoline with central α_2 adrenergic agonist properties, is used to treat spasticity from spinal cord injuries. Although hypotension and bradycardia are adverse effects occasionally described in therapeutic doses, overdose data are limited. We report a case of severe hypotension following an unintentional overdosage of tizanidine. Case Report: A 47-year-old woman with spastic paraplegia secondary to sarcoidosis mistakenly took four (4 mg) tablets of tizanidine rather than her usual dose of one (4 mg) tablet and presented to the hospital within 15 minutes of her ingestion. On arrival, she was drowsy with the following vital signs: blood pressure, 107/69 mmHg; pulse, 86/min; respirations, 16/min; temperature, 97.4°F; O_2 saturation 100%. Her physical examination was remarkable only for pinpoint pupils. An electrocardiogram showed a normal sinus rhythm and was otherwise unremarkable. Activated charcoal with sorbitol was administered and a crystalloid infusion was started at 150 mL/hour. About 3 hours post ingestion; she became hypotensive to a blood pressure of 74/48 mmHg. There were no mental status changes or other clinical symptoms associated with the hypotension. The crystalloid infusion was increased, and her blood pressure normalized after 4 L of normal saline. She was observed in an intensive care unit overnight with no further episodes of hypotension. Her vital signs the next day were: pulse, 70/min; blood pressure, 130/80 mmHg; respirations 12/min; temperature, 98.8°F. She was restarted on her normal doses of antispasticity medications including tizanidine and discharged home. Conclusions: Centrally acting α_2 adrenergic agonists are known to cause lethargy, hypotension and bradycardia. Interestingly, this patient was also mildly hypothermic, which is described with centrally acting α adrenergic agonists. Clinical trials of tizanidine reported that the most common side effects were fatigue, dizziness, muscular weakness, and dry mouth. Hypotension occurred only occasionally and was altogether absent in some trials. There has only been one previously reported overdose case, and the patient developed hypotension with sino-atrial and atrioventricular node dysfunction. Although tizanidine may be well tolerated in therapeutic dosages, this case illustrates the potentially severe side effects from a small overdosage of tizanidine.

108 TWO DECADES OF PARAQUAT SURVEILLANCE IN THE UK

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Objectives: The introduction of the herbicide, paraquat, in the UK in 1962 was followed by an increasing number of fatalities, mainly from intentional but also accidental ingestion. The NPIS (London) has maintained a database of reported human exposures from 1980 onwards with the potential to discern any trends over time. Method: Information was obtained from enquiries received by NPIS (London) with the addition of a few cases obtained via the manufacturer. Follow-up data was obtained where possible. Clinical effects were classified using EAPCCT severity scoring guidelines, relationship of effects to reported exposure was also assessed. Results: Over the 20 year period, 2886 cases were documented. There was a striking decline in the number of paraquat enquiries per annum compared to an over 7-fold increase in enquiries overall. Paraquat represented 0.6% of the NPIS (London) call load in 1980 but only around 0.05% from the mid 1990s onwards. The number of adult cases peaked in 1982 (196 reports) declining to about 75 per year after 1995. Cases involving children were relatively constant throughout the study period; 68% showing no clinical effects and 49% probably not actually exposed to paraquat. Occupationally-related cases were constant throughout with the majority showing no or only minor clinical effects. Accidental exposures decreased gradually, but intentional cases fell substantially from 109 in 1982 to approximately 30 per year from the mid 1990s. Cases of ingestion peaked at 158 in 1982 falling to 50 per year from the mid 1990s as a result of the decline in intentional but also accidental ingestions by adults. There was little variation in other routes of exposure but there were occasional bizarre ones e.g. injection, vaginal. There were 348 deaths reported of which 22 were not paraquat-related, 284 were recorded as 'intentional', 30 as 'circumstances unknown' and 12 as 'accidental', (two of which had insufficient information to confirm death as paraquat-related). The other 10 accidental deaths were due variously to: decanting into unlabelled bottles (3), inebriation (2), spillage onto skin (1), accidental occupational ingestion of concentrate (1) and unknown (3). Most of these cases occurred in the early 1980s with the last one recorded in 1992, confirming the virtual disappearance of accidental fatalities since their peak in the early 1970s. Conclusion: Over the study period there has been a dramatic decrease in

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paraquat enquiries to NPIS (London). This decrease is mainly due to a substantial decline in cases of suicidal ingestion; non-ingestion cases have stayed relatively constant and tend to show either no or minor effects. Deaths were almost entirely due to intentional exposure.

109 TOXICOLOGICAL RISKS AND STRATEGIES TO CONTROL PEDICULOSIS CAPITIS

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Background: Pediculosis capitis (*Pediculus humanus capitis* infestation) is an ectoparasite infestation that occurs worldwide. It affects mainly school children ranging from 3–11 years old, more girls than boys, all socioeconomic classes, and shows increased prevalence after seventies. It is not a vector of any human disease. It usually causes a characteristic pruritus due to allergic reactions to an anticoagulant present in its saliva, and heavy infestation may be complicated by pyodermatitis of the scalp. The general prevalence of infestation is difficult to establish because the outbreaks occur in schools and spread rapidly. When controlled, usually some oligosymptomatic children remain who act as reservoirs. In a 1992 survey among 1696 school children in Paulínia, Brazil, we found a prevalence of 35.7%. The control of infestation includes individual chemical treatment using solutions, shampoos or cream rinses with variable percentages of insecticides like pyrethroids, organophosphates or carbamates. Organochloride insecticides are forbidden in several countries due to neurologic toxicity and environmental persistence. In France, more than 4 million bottles of pediculicides are sold every year. In USA around 12 million, and in Brazil around 7.5 million bottles, showing the high prevalence of infestation all over the world. All these products are over the counter, and treatments are usually made by parents without medical advice due to its social stigma. Adverse effects of lice treatment can occur and are frequently reported in the medical literature. However, the risks of these treatments are underappreciated because physicians see only children who show severe side effects. Conclusion: For effective and safe control of pediculosis capitis we need a comprehensive approach that includes mass treatment of affected and exposed individuals on the same day to avoid reinfestation, and educational measures with incentives to use mechanical removal methods to decrease the extensive use of insecticides by alarmed parents. Head lice are a benign inconvenience with frequent occurrence that require a rational approach for control. Extensive and repeated chemical treatments can be more risky than the infestation itself.

110 CLINICAL OUTCOME IN GLYCOL ETHER INGESTIONS

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Objective: Experience with glycol ether poisoning in humans is limited. The goal of this study was to characterize all glycol ether ingestions reported to a regional poison center over a 5-year period. Methods: A retrospective review of all records from a regional poison center (annual volume: 47,000 human exposure calls/year) of glycol ether ingestions was performed for the period 1994–1998, inclusive. Data recorded included: patient age, ingested product, estimated concentration and amount of glycol ether, symptoms, evaluation and outcome. Results: 279 cases of glycol ether ingestion were identified. Median patient age was 2 years (range: 7 weeks to 63 years). 173 (62%) of patients were less than 3 years of age. Ingested agents included 5 chemical formulations containing glycol ether in various concentrations, and 37 products reported only as "glycol ether." Medical evaluation at a health care facility was sought in 18 cases. In only 13 cases (5%) did the victim ingest more than 15 mL of the agent. The greatest amount ingested was 200 mL of a low-concentration ethylene glycol butyl ether in a suicide attempt. This patient was the only one in the series hospitalized for observation secondary to abdominal pain. The majority of patients 108 (71%) were asymptomatic. Vomiting/gagging, oropharyngeal irritation, and bad taste were the most commonly reported symptoms. Conclusions: From our series of 279 glycol ether ingestions, no serious clinical manifestations were observed. The majority of patients were under 3 years of age and experienced ingestions of less than 15 mL of glycol ether compounds.

111 AN AUDIT OF PARACETAMOL ASSAYS USED IN SCOTTISH LABORATORIES

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Objective: To survey the assays used and detection thresholds quoted at laboratories in Scotland which report paracetamol levels. Introduction: The Scottish Poisons Information Bureau takes calls from throughout the UK and beyond, although most of our enquiries originate in Scotland. Recent calls to the Bureau, along with comments from enquirers, led to us to believe that doctors were not always aware of the change in thresholds of detection (TOD) from laboratory

to laboratory. Method: A telephone survey was conducted of the relevant laboratories. As no central record of laboratories and assays they conduct currently exists, a list of 'likely suspects' was drawn up, which we were later able to compare with a list provided by the Association of Clinical Biochemists. In addition to identifying the respondent, the survey asked 6 questions; which assay is used, reporting detection threshold of laboratory, how a level below that threshold is reported, the claimed sensitivity of the assay kit, the units the kit works in and the units the laboratory reports in. Results: A total of 32 sites were identified. Of these replies have been elicited from 21 laboratories so far. Most of these conduct a formal assay, but 1 peripheral unit used bedside ''Ascetite'' testing. 15 of the remaining 20 laboratories use the same assay kit, with a stated sensitivity of 0.01 mmol/L. However none of the laboratories quotes a threshold of detection as low as this, the lowest actual TOD was 0.03 mmol/L; 0.1 mmol/L was by far the most common TOD used (6 of 20). The highest threshold encountered was 0.13 mmol/L (19.7 mg/L), giving a range of 0.03–0.13 mmol/L. Conclusion: Although laboratories in Scotland are working to harmonise their reporting systems wherever possible, there are clear differences in thresholds of detection. Such differences may have implications for the management of patients who present ''late'' after poisoning. Doctors must be encouraged to establish the local TOD, and to be aware of the potential for change when moving to a different place of work. Acknowledgement: We are grateful to Mrs M Rae of Yorkhill Hospital NHS Trust, Glasgow for assistance with data acquisition.

112 IONIZING RADIATION CONTAMINATION: ARE WE READY?

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Background: Radiation poisoning is a rare but challenging entity. The preparedness of health care facilities to deal with such emergencies will directly influence the outcome of victims and caregivers. Most hospitals do not prioritize elaborate radiation emergency plans. For the rare radiation accident, it would be far more prudent to have a simplified procedure in place. Case Report: On April 13th, 2000, during an extremely brief conversation, a caller identified himself as a physician on an Armed Forces base who was caring for 2 children with an unspecified type of plutonium exposure. The patients were already en route to the hospital. The Poison Control Centre, the Atomic Energy Control Board, and Canada's Radiation Health Protection Branch were called. The Radiation Protection Branch provided guidelines on protection, decontamination, and treatment. During the course of preparation, it became clear that the call was a hoax. However, the hospital disaster plan was initiated, as an exercise of readiness. The full preparations required 3 hours, and were plagued by staff anxiety regarding personal safety. Identified strengths included the rapid mobilization of resources, available expertise in radiation, identification of a room and pathway to be used, a prepared ventilation system, maintenance of normal ER flow, and cooperation. Identified weaknesses were lack of a clear plan for radiation accidents, poor staff knowledge of the risks of radiation, poor communication, no decontamination facility in the city, no equipment for collecting draining water, the loss of the room to be used due to contamination, no funding, and an inability to trace outside calls. Conclusion: While ionizing radiation contamination is rare, hospitals will benefit from developing an ionizing radiation disaster plan. Community and intra-facility resources should be identified. This protocol should be reviewed, disseminated, and discussed at regular intervals. Education and interdisciplinary cooperation are of paramount importance in assuring success.

113 ACUTE POISONINGS WITH DIFFERENT MERCURY COMPOUNDS

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Objectives: Evaluation of acute poisonings with different mercury compounds. Methods: The clinical, anatomic, and histological results of 32 autopsied patients who died by poisoning with different mercury compounds in the period 1920 to 1999 were evaluated. The cause of death was poisoning with mercury dichloride in 24 patients, mercury oxycyanide in 3 patients, gray mercury ointment in 3 patients, yellow mercury oxide in 1, and metallic mercury in 1 patient. Skeletal muscle biopsy material and the resected lower lobe of the right lung from two patients, 23 and 19 years old, respectively, who injected themselves with metallic mercury in muscles and in the cubital vein have also been studied. Results: Analysis of the material from 1920 to 1956 revealed that the main cause of death was acute renal failure due to coagulation necrosis of the nephrocytes in the proximal and distal tubuli which resulted in uremia. Since 1960 hemodialysis and antidotal treatment substantially diminished the number of lethal outcomes. In subsequent years morphological investigation of the kidneys has shown striking differences which consist of rapid clearing of the tubuli lumen from necrotic masses, absence of lime deposits and differentiation of the nephrocytes of convoluted tubuli. Effects of uremia were not present at autopsy. In patients deceased from acute gray mercury ointment poisoning as a result of self treatment of pediculosis, acute renal failure in 2 cases and toxic epidermal necrolysis (Lyell syndrome)

in one case were observed. Two patients injected themselves with metallic mercury; the investigation of muscle and fat tissue biopsies of these patients revealed a high number of metallic mercury drops in the investigated tissues, and histological investigation showed foreign body granulomas with mercury drops, muscle and fat tissue necrosis. Many mercury drops were observed in the resected lower lobe of the right lung. Histological investigation revealed thrombovasculitis and hemorrhagic lung infarcts. Conclusion: Evaluation of autopsy material of mercury intoxicated patients from 1920–1999 showed specific altered morphology especially in the kidney.

114 AMITRAZ INTOXICATION IN A BABY OF 4 MONTHS OLD

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Background: Amitraz poisonings in adults and children have been published in the literature, but as far as we know not in a baby. Amitraz is an alpha-2-adrenergic agonist used as an insecticide and acaricide. Symptoms reported in poisonings resemble those caused by alpha-2-adrenergic agonist drugs such as clonidine. Most relevant symptoms observed after amitraz exposure are CNS depression, miosis, bradycardia, hypotension and hyperglycaemia. Vomiting may occur and is probably due to the solvent, usually a petroleum distillate, in which the amitraz is dissolved. In several pediatric patients hypothermia has been reported. The onset of symptoms is usually within 0.5–2 hours. Duration of the symptoms has ranged from a few hours to more than 24 hours. Treatment is symptomatic. Case Report: According to the father, he erroneously gave his 4-month-old baby amitraz instead of an antitussive. This antitussive was stored next to the amitraz. The amitraz having been removed from the original packaging closely resembled the antitussive bottle and this presumably caused the mistake. As he realised his mistake while giving the amitraz, the child received only 0.3 mL of a 5% amitraz solution. Consequently, with a body weight of 6 to 7 kg the calculated dose was approximately 2.5 mg amitraz/kg bdw. At that time, 9.30 p.m., the baby was presented to an Emergency Department. The baby was sleepy although it could be woken easily. As there were no other symptoms and it was already two hours after ingestion, the parents received instruction (regular wake-up advice), and were sent home. At home, the child developed hypothermia (temperature taken by the parents) and drowsiness and was difficult to wake. The parents presented the child to a different children's hospital. On admission, the baby was drowsy, hypotonic, and had bradycardia of 80 beats/min. After consultation with the National Poisons Control Centre, the baby was admitted to the Intensive Care department. An intravenous catheter was introduced for parenteral fluid administration. 18 hours after ingestion the baby was awake and drank again. 1.5 days after the exposure the baby was discharged in good health. Conclusion: In babies, a small oral dose of amitraz can result in severe clinical effects, therefore, intensive care observation is advocated. The symptoms may develop even 3–4 hours after ingestion. Proper storage is essential in order to prevent erroneous exposure to this pesticide.

115 ACUTE POTASSIUM DICHROMATE POISONING TREATED WITH EXTRACORPOREAL ELIMINATION PROCEDURES AND LIVER TRANSPLANTATION

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Objective: Acute oral poisonings with potassium dichromate are infrequent and have a high fatality rate. This report describes the clinical course and the analytical data of a severe acute potassium dichromate poisoning treated by hemofiltration, high-flux hemodialysis, albumin dialysis and liver transplantation. Case Report: A 49-year-old woman drank an aqueous solution containing approximately 16.8 g of potassium dichromate in a suicide attempt. The patient initially developed abdominal pain, nausea, vomiting and bloody diarrhea, followed by a bleeding disorder, renal and hepatic failure. On day 7 hepatic function deteriorated to such an extent that orthotopic liver transplantation had to be performed. The post-operative course was complicated by the development of a critical illness polyneuropathy. Four months after the ingestion the patient was discharged well with only a mild residual polyneuropathy. On admission the patient had a chromium level in whole blood of 13,000 µg/L, in serum 7000 µg/L and in urine 60,000 µg/L. Before liver transplantation chromium concentration in whole blood and serum had decreased to 1900 µg/L and 600 µg/L, respectively. The majority of identified chromium elimination (183 mg) was due to extracorporeal elimination procedures including hemofiltration and albumin dialysis. Faecal excretion accounted for 21 mg chromium, exchange transfusion for 15 mg chromium and renal excretion for 6 mg chromium. In the first seven days chromium half-life in serum, whole blood and

red blood cells was 38.9 hours, 66.6 hours and 92.4 hours, respectively. The hemofiltration clearance without DMPS ranged between 31.2 and 56.6 mL/min, the hemofiltration clearance with DMPS was between 21.3 and 46.0 mL/min. The albumin dialysis clearance amounted to 21.6 mL/min and hemodialysis clearance came to 22.4 mL/min. During renal failure the renal clearance for chromium was 1.17 mL/min but subsequently increased to 15.7 mL/min. Conclusion: In acute potassium dichromate poisoning acute hepatic failure may be the crucial life-threatening complication and liver transplantation may be the only life-saving treatment procedure. Of the extracorporeal elimination procedures used hemofiltration, albumin dialysis and high-flux hemodialysis appeared most effective. Chromium blood concentration fell to a level which did not harm the transplanted liver.

116 UNRECOGNIZED SEVERE THALLIUM POISONING

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Objective: In former times thallium poisoning was frequent in many countries as a result of ingestion of rodenticides. Therefore WHO spoke against further use in 1973. Contemporary thallium poisoning has been rare in Germany. We describe a case which was diagnosed late. Case Report: A 14-year-old female was admitted with headache, abdominal and dorsal pain. Her general condition deteriorated and a loss of body-weight about 4 kg was noted. The patient complained of dizziness and muscular weakness of the lower limbs. In addition she reported numbness in the anogenital region. Proteinuria and ketonuria were discovered. All other laboratory and analytical findings were in the normal range. The first tentative diagnosis was anorexia nervosa. Increased blood pressure was treated first with atenolol, later in combination with nifedipine. Numbness, weakness, and piercing pain in the lower limbs as well as genital dysaesthesia intensified during the first period of hospital stay. Nuclear magnetic resonance imaging of head and lumbar spine was unrevealing. Cerebrospinal fluid composition was normal. Finally alopecia areata appeared within one week. The patient's disorder was now diagnosed as a psychosyndrome. The patient rejected further diagnostic and therapeutic measures and left the hospital. Five days later she was readmitted because of dramatic weight loss and almost complete paresis of lower limbs, which further progressed to tetraparesis. Acute axonal polyneuropathy with complete degeneration was diagnosed electrophysiologically. The EEG indicated degenerative and chronic inflammatory processes. At this time analysis of thallium in serum and urine was performed and high levels were found (serum: 300 µg/L, normal range <0.6 µg/L; urine: 2236 µg/L, normal range <0.7 µg/L). Immediately treatment with Prussian blue (potassium ferric ferrocyanide; Anidotum Thallii Heyl) was started (dosage initial 3000 mg, followed by 250 mg/kg/d divided in 2 to 4 doses orally). The thallium excretion was increased effectively. One month later the thallium level was still slightly elevated (serum: <5 µg/L; urine: 22 µg/mL). The clinical state was substantially improved in the same time. The hypertension disappeared with decreasing thallium level. The patient's hair began to grow again. The tetraparesis receded gradually. The origin of this thallium poisoning is still unclear. Conclusion: The patient developed signs of subacute thallium poisoning. Initial symptoms were misinterpreted because the complete toxic differential diagnosis was not taken into account. The initial toxicological analysis was incomplete. When Prussian blue is administered by mouth it forms a non-absorbable complex with thallium during the enterohepatic circulation of the metal which is excreted in the faeces. This measure enhances elimination.

117 A CASE OF CHRONIC COPPER POISONING

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Objective: To report on the case of a child with elevated levels of serum copper. Case Study: A 15-month-old child was admitted to hospital with persistent vomiting, respiratory tract infection and poor weight gain. 3 weeks later he was seen by the GP who reported purplish/bluish discoloration to his hands and feet. 1.5 weeks later he was admitted to hospital with bruising and fractures of varying ages. On further investigation blood count was normal, liver and renal function tests were normal, but serum copper level was raised at 38.8 µmol/L (normal range 11–22 µmol/L[1]). Serum caeruloplasmin was normal. A month later the serum copper level had dropped to 29.4 µmol/L and the following month was 23.4 µmolL with urinary copper levels normal. His weight increased from 8 kg on initial admission to 9.2 kg 3 months later. Copper levels were measured in the cold and hot water supplies at his home and found to be <200 µg/L and 915.4 µg/L respectively (maximum acceptable concentration = 3000 µg/L[2]). Discussion: The pattern of symptoms are very similar to those of acrodynia (pink disease), usually associated with mercury poisoning. There are striking

similarities between this case and one described by Salmon and Wright in 1971[3]. In that case a child, also aged 15 months, presented with symptoms of acrodynia, liver function abnormalities and a serum copper level of 28.6 μg/L. The child's parents had used water from the hot tap for all food and beverage preparation. The level of copper in the hot water system was found to be 790 μg/L. Conclusion: It is possible that some of the symptoms in this child were due to the elevated serum levels of copper. The normal caeruloplasmin level shows that this child was not suffering from Wilson's disease. There is no evidence to suggest that copper toxicity would cause bone abnormalities and therefore is unlikely to account for his fractures. The source of copper could possibly have been due to the hot water used to fill the kettle for food and beverage preparation. It is possible that some children may suffer from copper poisoning when exposed to water containing levels well within acceptable limits. References: [1]Dirckx JH Ed. *Steadman's Concise Medical and Allied Health Dictionary*. 26th Ed, Appendices 987. [2]Murley L, ed. *Pollution Handbook*, NSCA, 1997: 460. [3]Salmon MA, Wright T. Chronic Copper Poisoning Presenting as Pink Disease. *Archives Dis Child*1971;**46 (245)**: 108–110.

118 METHANOL POISONINGS TREATED IN SWEDISH HOSPITALS 1995 TO 1999

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Objective: Intoxications with methanol have been a long-term problem in Sweden. The aim of this study was to evaluate the epidemiology in recent times, the circumstances, chemical products involved and severity of poisoning. Case Series: The Swedish Poisons Information Centre received 73 discharge summaries concerning intoxications with methanol in Swedish hospitals 1995 to 1999. In this material, 78% were males. With the exception of 3 children ≤5 years and 7 teenagers, all patients were adults. 54 patients (74%) had a history of alcohol abuse. 13 cases (18%) were classified as pure accidents as there was no indication of heavy alcohol abuse or suicidal intent. 6 cases (8%) were classified as suicide attempts. A household fuel for spirit stoves containing about 20% methanol and 80% ethanol was the probable product used by 34 patients, most of whom were alcoholics. In 3 cases the product involved was fuel for model vehicles and in 11 cases pure methanol. The chemical product was unknown in 25 cases, but in 4 of these, long-term use of ethanol-based fuel with a small amount of methanol was suspected. The poisonings were graded according to the Poisoning Severity Score as follows: Asymptomatic: 4 cases (5%); Minor: 25 cases (34%); Moderate: 15 cases (21%); Severe: 29 cases (40%), among which 10 (14%) were fatal. At least 4 survivors had sequelae, i.e. blindness and/or cerebral damage. All children were asymptomatic. Four of the accidents resulted in severe poisoning, one of which was fatal. Most of the poisonings with the household fuel containing 20% methanol were graded as moderate or less, in spite of high or very high serum methanol levels. Severe symptoms were avoided due to the ethanol content of the product and, subsequently, treatment with ethanol and dialysis. The symptomatology were in all patients entirely typical. Ethanol therapy and dialysis were used in 86% and 68% of the cases respectively. Fomepizole® was used in one severe poisoning, that finally proved fatal. Conclusion: Methanol poisoning continues to be a problem in Sweden, particularly among heavy alcohol abusers who drink ethanol-based fuels for spirit stoves. One specific product, the most commonly involved in this case series, contained about 20% methanol up to 1999. Due to new regulations in the EU, household fuels now contain no more than 10% methanol. This might reduce the morbidity of methanol poisonings, in particular among alcohol abusers. The overall hospital mortality of 14% in this material must be regarded as considerable. It is also notable that 4 accidents resulted in severe poisoning or death.

119 INHALATIONAL METHANOL POISONING DURING PREGNANCY TREATED TWICE WITH FOMEPIZOLE

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Objective: We describe a patient with a long history of inhalant abuse who came to our hospital twice during her pregnancy. Both of these times she had elevated methanol levels and acidosis, for which she was treated with 4-MP. The new alcohol dehydrogenase antagonist, fomepizole (4-methylpyrazole or Antizol®) has been available in the United States since 1996 for the treatment of ethylene glycol poisoning. Most of the initial studies on the use of 4-MP excluded pregnant patients. Case Report: A 21-year-old white female is brought to the hospital after being bingeing on carburetor cleaner (Carb cleaner® from AutoZone, which contains methanol (MeOH), toluene, methylene chloride, and carbon dioxide) for a day. She had a long history of toluene abuse and of depression. She was not taking any prescription medicines. The patient was currently in the first trimester of her second pregnancy (11 weeks of gestational age by dates). On evaluation, she acted intoxicated, smelled like hydrocarbons, and was uncooperative. Her vital signs were: blood pressure 124/76, heart rate 84, respiratory rate 18, and the temperature 36.3° C. Her physical exam was otherwise

unremarkable except for optic nerve hyperemia. The electrocardiogram showed normal sinus rhythm. Her serum bicarbonate was 12 mmol/L, potassium of 2.9 mmol/L and an anion gap of 14. Her initial MeOH level was 24 mg/dL. She received one dose of 4-MP (15 mg/kg) and vitamins (thiamine, folate, and pyridoxine). The repeat MeOH level was non-detectable, so the antidotal treatment was discontinued. The acidosis resolved. The patient was discharged to an in-patient psychiatric facility. Her second visit was at a gestational age of 16–17 weeks. She had been huffing constantly again with the same product. Her laboratories showed an acidosis, sodium 134 mmol/L, potassium 4.2 mmol/L, chloride 105 mmol/L, bicarbonate 8 mmol/L, MeOH level of 42 mg/dL. She received again one dose of 4-MP and underwent hemodialysis. After dialysis, her acid-base disturbances corrected and her methanol was non-detectable. An obstetric ultrasound revealed a normal intrauterine pregnancy without gross fetal anomalies. The outcome of the pregnancy is unknown since the patient never returned for follow up. Conclusions: This case illustrates a case of methanol poisoning from inhalant abuse. This patient developed acidosis without visual problems. She was treated with 4-MP, without any immediate evidence of adverse effects. The long-term effects of our intervention are unknown, since we lost this patient to follow up.

120 RECORD HIGH ETHYLENE GLYCOL INGESTION

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Objective: Ethylene glycol (EG) is a known toxic alcohol that is abused by alcoholics or in attempts to commit suicide. Toxicity can occur with a 100 mL ingestion or at levels of 20 mg/dL and higher. We report a case of suicidal ethylene glycol ingestion with a record high level. Case Report: A 47-year-old male with 3 previous suicidal attempts was found in a motel room drinking antifreeze and vodka. He reported drinking almost 3 liters of Peak antifreeze (100% EG) and a one-liter bottle of vodka. He was awake, alert and oriented, still suicidal but in no acute distress. EMS took him to a small community hospital and was subsequently transferred to our institution. Physical exam was unremarkable. Initial lab results in the ED were remarkable only for ethanol of 119 mg/dL, and serum osmolarity elevated to 444 mosm/L. Initial EG level was pending, but pt was admitted to the hospital and empirically given a loading dose of fomepizole (4-MP) based on history and large osmal gap. The initial (12 hours post ingestion) EG concentration was found to be 2361 mg/dL by gas chromatography with internal standards. He was transferred to the ICU and dialysis was initiated. Another EG concentration 4 hours later was 1210 mg/dL. Levels were followed at frequent intervals until EG was undetectable. Serum osmolarity levels were also followed and corrected to normal. He received a total of 880 mg of 4-MP over 5 doses. Hospital course was uncomplicated and he was discharged on hospital day 7 to an inpatient psychiatry facility. Conclusion: This case reports the highest concentration of ethylene glycol reported to date. Our patient never developed the hallmark signs of the EG toxicity. By co-ingesting vodka the patient essentially drank his own antidote. Ethanol has an affinity for alcohol dehydrogenase that is about 100 times greater than ethylene glycol. This explains his lack of clinical signs and lab results that did not demonstrate a large anion gap acidosis on arrival at our institution despite late presentation. There may be a longer than normal delay in the onset of symptoms when ethanol is co-ingested. Prompt treatment should result in a good outcome.

121 FATAL CEREBRAL EDEMA DUE TO EXCESSIVE WATER INTAKE AFTER RECREATIONAL USE OF BENZYLPIPERAZINE ("A2") AND ECSTASY

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Objective: MDMA- (methylenedioxymethamphetamine, "ecstasy") induced hyponatremia has been described in rare cases. Excessive water intake by ravers is invoked as a possible cause as well as hormonal influences since >90% of the victims were young females. The role of interactions with other illicit drugs has not been clarified to date. 1-Benzyl-1-piperazine (street name: "A2"), a substance with amphetamine-like effects, is known since the 1970s, but there are no data on toxicity in humans. Benzylpiperazine has begun to be abused in Switzerland during the year 2000. We report a case of fatal use of MDMA and benzylpiperazine in a young woman. Case Report: A 23-year-old female consumed hard drinks and two capsules of benzylpiperazine during a rave party. In the following 15 hours she drank 10 liters of water and took one tablet of ecstasy. Seven hours after the ingestion of benzylpiperazine and three hours after MDMA intake she experienced minor malaise including frontal headache. Another three hours later she developed dizziness

and somnolence (GCS = 8). In the Emergency Department she was found to be disoriented, deeply somnolent (GCS = 6), bradycardic (36 bpm), and hypothermic (core temp. 34.1 °C). Her pupils were wide and non-reactive to light, and she did not react to painful stimuli. She had two convulsions and was intubated. Brain CT scan revealed massive brain edema with tonsillar herniation. The laboratory investigation revealed marked hyponatremia (115 mmol/L). Plasma osmolality was 246 mosm/L, and urine sodium concentration and osmolality were 23 mmol/L and 108 mosm/L, respectively. Blood ethanol was non-detectable. Plasma MDMA concentration was 68 μg/L. Urine drug screen was positive for benzodiazepines, caffeine, nicotine, benzoylecgonine, MDMA and benzylpiperazine (5.7 mg/L) and negative for amphetamines, barbiturates, cannabinoids, cocaine, opiates and phencyclidine. She was admitted to the MICU. After correction of serum sodium urine production increased to 1300 mL/h. The patient remained completely unresponsive (GCS = 3). Circulatory and neurologic condition further deteriorated, and the patient was declared brain dead 51 hours after admission. An autopsy was refused by her relatives. Conclusion: This is the first report of fatal outcome after concomitant benzylpiperazine and MDMA abuse. Hyponatremic hypotonic hyperhydration with cerebral edema is a rare but well-known complication of MDMA abuse. Young women are at particular risk. Therefore unrestricted water intake after MDMA ingestion (i.e. at rave parties) should be vigorously discouraged. The role of coingested drugs (benzylpiperazine in this case) in the etiology of MDMA-induced cerebral edema is not known.

122 HYPONATREMIA AND ALTERED MENTAL STATUS FROM WATER INTOXICATION AND ECSTASY-INDUCED SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE RESULTING IN A MOTOR VEHICLE ACCIDENT

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Objective: 3,4-methylenedioxymethamphetamine (MDMA), or "ecstasy," is a resurging drug of abuse at nightclubs and "raves" in the US. Toxicity is due to adrenergic excess (agitation, hyperthermia, seizures, cerebral hemorrhage, tachyarrythmias, hypertension, mydriasis, myoclonus, rhabdomyolysis) and psychedelic effects (euphoria, hallucinations). Unique features of toxicity include hepatotoxicity and hyponatremia from water intoxication and/or the syndrome of inappropriate antidiuretic hormone (SIADH). Although ecstasy-related hyponatremia has been reported in 6 cases in Europe, only 1 case has been reported in the US literature. We publish the second US case of ecstasy-related hyponatremia from water intoxication and SIADH, which resulted in a motor vehicle accident. Case Report: A previously healthy 16-year-old female was witnessed to slump over the steering wheel of her car and suffer a low speed, broadside collision with trees. On arrival to the ED, the patient was confused, nonverbal, unable to follow commands, and vomiting. Initial physical exam findings were a HR of 130/minute, BP of 158/70 mm Hg, fine hand tremors, rotary nystagmus, and GCS of 8. Cervical spine film, head CT, and abdominal/pelvic CT were normal. Upon return from radiology, the patient became unresponsive and was endotracheally intubated. Abnormal lab results included a serum sodium of 122 mmol/L, potassium 3.2 mmol/L, chloride 86 mmol/L, and urine toxicology screen positive for amphetamines. All other toxicology screens, ECG, and blood chemistries were unremarkable. After receiving 2 L of normal saline and 50 g of AC, the patient was transferred to our institution. The first repeat serum sodium was 120 mmol/L and associated with a serum osmolality of 252 mOsm/L, urine osmolality of 298 mOsm/L, urine sodium of 112 mmol/L, and urine specific gravity of 1.009. Hypertonic saline (3%) was given, and the serum sodium was corrected over the subsequent 2 days of ICU hospitalization. A creatine phosphokinase was initially 776 U/L and peaked at 5000 U/L. After recovery, the patient confirmed that she took "2 hits" of ecstasy about 10 hours prior to the motor vehicle accident. She also confirmed being a "water addict," drinking more than a gallon of free water daily. Conclusion: The diagnosis of ecstasy-induced SIADH is supported by a decreasing serum sodium and a urine osmolality > serum osmolality despite 2 L of NS. The history of free water intake as well as the low serum sodium and urine specific gravity is consistent with dilutional hyponatremia. Both conditions are associated with ecstasy intoxication, resulting in this patient's altered mental status and subsequent motor vehicle accident.

123 SEVERE HYPONATREMIA WITH RESULTANT NONCARDIOGENIC PULMONARY EDEMA AND CEREBRAL EDEMA SECONDARY TO ECSTASY INTOXICATION

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Objective: Hyponatremia with secondary cerebral edema and noncardiogenic pulmonary edema (NCPE) has been reported in marathon runners but has not been reported in the primary literature with ecstasy use. We report a fatal case

of ecstasy use with severe hyponatremia, NCPE and cerebral edema similar to that found in marathon runners. Case Report: a 17-year-old male returned home with friends after an evening at homecoming. After vigorous dancing to loud music for an unknown period and ingesting large amounts of water, the adolescent stated he did not feel well and began to vomit for multiple hours. After increased lethargy, he eventually became unresponsive. His adolescent friends vehemently denied use of alcohol or drugs. EMS was activated and paramedics found the patient unresponsive, combative, with sonorous respirations. Nasal and oral airways were attempted but were unsuccessful. Fifteen liters O_2 was administered via NRB mask. Respirations were 20/min, O_2 saturation was 84%, pupils were fixed and dilated, and temperature was 35° C. While in transport to hospital, BP was noted to be 200/120 mm Hg. After arrival to the ED, the patient received naloxone 4 mg IV without effect, 10 mg of haloperidol for agitation, 25 grams activated charcoal via NG tube, and 2700 cc of fluid (Normal saline and Lactated Ringers). Initial sodium was 124 mmol/L and ABG was 7.16/43.7/62.3/13.7. Pulmonary edema developed rapidly and 80 mg furosemide was administered intravenously. The patient was transferred to our institution and was noted to have fixed pupils dilated to 10 mm. His ocular vestibular reflex was absent; lung examination revealed diffuse rales and rhonchi. He withdrew to painful stimuli only. Laboratory results were significant for sodium of 118, and potassium 2.8 mmol/L. Chest radiograph revealed marked fluffy infiltrates consistent with NCPE. The head CT revealed moderate cerebral edema with loss of cerebral sulci. Significant hypotension developed requiring normal saline infusion, dopamine, norepinephrine and FFP. Mannitol was administered. Repeat head CT scan after mannitol administration revealed complete effacement of the ventricles. Family agreed to discontinue support and approved organ donation. Toxicology panel confirmed MDMA (ecstasy) and chlorxylenol. Conclusion: Extreme exertion and vomiting with large volume ingestions of water resulting in severe hyponatremia, noncardiogenic pulmonary edema and cerebral edema may occur after ecstasy ingestion.

124 HYPOCALCEMIA AND HYPOMAGNESEMIA DUE TO OCCUPATIONAL DERMAL CONTACT WITH HYDROFLUORIC ACID

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Introduction: Hydrofluoric acid readily penetrates the skin and mucous membranes, causing deep tissue layer destruction. Severity and rapidity of onset of signs and symptoms depends on the concentration, duration of exposure, and penetrability of the exposed tissue. Dermal exposure to hydrofluoric acid can produce hypocalcemia, hypomagnesemia, hyperkalemia, cardiac dysrhythmias, and death. Case Report: A 52-year-old patient, without relevant preceding medical history, was soldering a pipe on the deck of a ship when a hose ruptured above him. The hose held a 3% hydrofluoric acid/ 10% nitric acid solution, used to clean and remove encrustation from the ship's tanks and pipes. The worker's body was almost completely sprayed by the solution and his clothes saturated. A colleague removed the man's clothes, showered him for 15 minutes and applied a 2.5% calcium gluconate/ 1% magnesium chlorate gel over the affected parts of his body before sending him to the Emergency Service of our hospital where he arrived some 30 minutes later. Examination revealed the presence of an erythema which affected some 13% of the body surface (face, neck and pelvic region) and a 3rd degree burn of approx. 2 cm^2 on the right knee which was then infiltrated with 5mL of calcium gluconate 10%. In addition, the patient had a severe bilateral keratoconjunctivitis which was treated topically by frequent washing and anti-inflammatory and cycloplegic eye-drops. Continuous electrocardiographic monitoring was begun, and the plasma concentrations of calcium and magnesium were controlled. Initial ionized calcium level was normal but a hypomagnesemia was detected (1.5 mg/dL) which was treated with 1.5 g magnesium sulphate IV. At three hours, magnesium levels had normalized (2.3 mg/dL) but calcium levels had decreased to 0.75 mmol/L. Five mL of 5% calcium gluconate IV were administered. Subsequent observation revealed normal levels of calcium and magnesium, without any other clinical manifestations, electrocardiographic alterations or changes in vital signs. The patient was discharged from hospital 36 hours after admission. Follow-up showed a complete recovery without sequelae. Discussion: Although the patient suffered burns from a low concentration of hydrofluoric acid, and was treated in situ with a calcium gluconate/magnesium chlorate gel, he developed hypocalcemia and hypomagnesemia. These were potentially severe systemic consequences which were corrected by IV administration of calcium gluconate and magnesium sulphate. The most important steps that should be taken in this type of intoxication are preventive, and it is important that workers know the toxicity of the substances they are handling. The workers should use adequate personal protective equipment and know the first-aid measures to use in the case of an accident.

125 HEMOLYTIC ANEMIA AFTER METHYLENE BLUE THERAPY IN ANILINE INDUCED METHEMOGLOBINEMIA

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Background: Methylene blue is conventionally utilized as the main treatment of methemoglobinemia, but it may be ineffective in individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency. We report a case of acute substance-induced methemoglobinemia with initial good response to methylene blue despite G6PD-deficiency, but hemolytic anemia appeared later. Case Reports: A male worker suffered from acute methemoglobinemia caused by accidental dermal exposure to aniline dye. Methylene blue was given with good response initially. However, mild methemoglobinemia recurred and severe Heinz body hemolytic anemia followed 3 days after treatment. G6PD deficiency was identified, then adequate hydration and packed blood transfusion were performed with adjuvant Juvela-N (dl-α-tocopheryl nicotinate), and he recovered uneventfully. Conclusion: We suggest that caution should be taken in using methylene blue as the antidote for acute methemoglobinemia, especially when the history of G6PD deficiency is obscure. Titrating the dose of methylene blue and starting with 0.3–0.5 mg/kg initially are recommended. Some other cellular antioxidants could be considered as a supplement treatment.

126 CARBON MONOXIDE INTOXICATIONS ATTENDED IN A HOSPITAL CASUALTY SERVICE

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Aims: To analyze the characteristics of acute intoxication (AI) caused by carbon monoxide (CO) and its treatment in a hospital casualty service over the last 12 months. Methodology: Descriptive and prospective study of patients diagnosed with AI caused by CO in a hospital casualty service. A data collection sheet was designed in which epidemiological, clinical and toxicological variables were set out. Results: In this period, 961 cases of AI were attended. Of these, 34 were CO intoxication (3.53%). A progressive increase of this diagnosis has been verified over the last 5 years (in 1996, 11 cases presented). The distribution was: 44.15% men and 55.85% women. Mean age was 37.05 years. The causes were: boilers (64.70%), fires (17.64%); vehicle exhaust fumes (8.82%); other mechanisms (8.82%). Twenty eight cases (82.35%) were domestic, 4 (11.76%) at work and 2 (5.88%) were suicide attempts. The carboxyhemoglobin (COHb) blood levels were measured, obtaining a mean of 14.76% (range 1.1%–34%). In 38.23% of the cases a control was performed at 12 hours, showing a decrease of the figures (mean: 3.13%). Admission was required for 73.52% of the patients (96% in the Short Stay Unit and 4% in Internal Medicine). The distribution by month revealed a greater frequency in November, December and January. 76.47% of the cases occurred in family groups. The symptomatology, (present in 93%) was neurological (21 cases), respiratory (6 cases), cardiovascular (7 cases) and digestive (9 cases). Oxygen therapy was applied in 61.76% of the cases, always at atmospheric pressure. Evolution was positive in all cases, the patients being discharged in less than 24 hours without any symptomatology. Conclusions: 1. In our casualty service, an increase of CO AI cases has been observed over recent years. 2. The majority of the cases are accidental, domestic and caused by boilers, with a clear prevalence in the winter months, and grouped in family nuclei. 3. It presents clinically as a moderate intoxication, treated with oxygen at atmospheric pressure (despite the high levels of COHb found in some patients). On the other hand, oxygen therapy protocol was not applied to all patients and a hyperbaric chamber was never used. This, however, did not influence the good short-term evolution of all cases.

127 BIOLOGICAL TOLERANCE OF HYDROXOCOBALAMIN IN FIRE VICTIMS INTOXICATED BY CYANIDE

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Cyanide (CN) poisoning in fire victims is frequent and rapidly fatal. Several antidotes to cyanide are available. However, their safety has not been assessed in the complex setting of smoke inhalation. In a prospective study we tried to assess the biological tolerance of a high dose of hydroxocobalamin (HOCo) administered at the scene of the fire in fire victims suspected of CN poisoning. Methods: Inclusion criteria: fire victims were included according to the presence of the following criteria: soot deposits in mouth or sputum plus any degree of neurological impairment. Exclusion criteria: children, pregnant women, burns of total surface body area $> 20\%$, and multiple trauma. Protocol design: following

examination and the collection of a blood sample in dry heparin, a 5 g dose of HOCo (10 g in case of cardiovascular collapse) was administered intravenously over 15 min. Blood cyanide concentrations were measured using the method of Rieders. The following parameters were collected on admission and then daily during three days: plasma lactate concentration, blood glucose, serum creatinine, liver functions tests, prothrombin time, serum CPK, blood cell counts. Results: 42 (22 females, 20 males) of 63 patients enrolled were found to be intoxicated by cyanide, with a median blood cyanide concentration of 96.1 μmol/L (range: 52–149). The median carbon monoxide concentration was 2.86 mmol/L (range: 2.06–4.10). Fourteen deaths were observed, 9 of these by decerebration, 4 due to septic shock, and one by hypoxemic pneumopathy. The median dose of hydroxocobalamin was 5 g, with a range of 5 to 15 g. Excluding the patients with initial cardiorespiratory arrest, there were no obvious side effects on blood glucose and serum creatinine concentrations and liver function tests. There was only a trend towards a decrease in hemoglobin level and platelet count over the 3-day period which, however, remained within the normal range. The lone side effects were a deep red coloration of both plasma and urine that disappeared within 7 days after injection. The deep red coloration of plasma may induce some interference with clinical chemistry. Conclusion: Hydroxocobalamin is well tolerated and is not associated with a significant effect on renal or hepatic function in fire victims. These results may be viewed as an indicator of the safety of use of hydroxocobalamin at the fire scene in smoke inhalation victims.

128 COURSE OF SKIN NECROSIS WITH IV PHENYTOIN EXTRAVASATION

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Objective: IV Phenytoin extravasation can cause severe damage to the skin. We report a patient followed until recovery with conservative treatment. Case Report: A 44-year-old man with epilepsy presented with pain, swelling, and purplish-black discoloration on the dorsum of the left hand. On the previous day he had received an intravenous load of phenytoin through a 20-gauge catheter placed in the dorsum of his left hand. He was unable to make a fist (skin too tight), but could move the fingers against resistance without pain. The area was hypesthetic with small blisters. The surrounding skin was warm, tender, and erythematous. Sulfadiazine cream, a non-adherent dressing, and a wrist splint were applied. The patient returned the next day for worsening swelling and blistering. One of the blisters was unroofed and drained serosanguinous fluid. He did not keep his follow-up appointments with the Hand Service. In 4 weeks a thick, hard, black eschar had formed. He was able to continue painting houses. We removed the eschar with blunt dissection. Yellow-white exudate and a black, thrombosed vein (into which the phenytoin had been infused) were visible. Wet-to-dry dressings were prescribed. Six weeks later the wound had healed. Discussion: In one review the incidence of phenytoin extravasation was 6%. Some authors have referred to the pain, swelling and discoloration as the "purple glove syndrome." There is most likely a spectrum of pathology from minor discoloration to severe necrosis requiring amputation. Our patient would fit into the moderate to severe category. Possible substances responsible for the necrosis are the high pH, the phenytoin (crystallizes at physiological pH), or propylene glycol (noxious diluent). The flame shaped lesion and the rapid time course suggests a directly noxious rather than an immunological etiology. Some authors recommend irrigating the subcutaneous space, if caught early, first by injecting hyaluronidase, producing an irrigatable space, then placing stab peripheral stab wounds for irrigant exit. Conclusion: Patients complaining of pain at the site of intravenous infusion of phenytoin must be taken seriously. The infusion should be continued at an alternate site. The solution should be checked for crystals, and the rate of infusion should be slowed. Consider giving hyaluronidase and irrigating. When the extent of necrosis was moderately large, but still limited, as in this reported case, conservative treatment produced good results.

129 VERAPAMIL OVERDOSAGE UNRESPONSIVE TO DEXTROSE/INSULIN THERAPY

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Objective: Insulin/dextrose has been advocated in recent reports to rapidly improve haemodynamic parameters in calcium channel blocker poisoning. We report a case of verapamil overdose unresponsive to this and conventional therapies. Case Report: A 58-year-old male with a history of myocardial infarction, hypertension and an aortic lesion presented to the Accident and Emergency Department with severe chest and abdominal pain with guarding. Although the patient was conscious blood pressure was initially unrecordable and was then recorded at 70/50 mmHg after administration of intravenous fluids. Peripheral pulses were unpalpable. His medication on admission consisted of frusemide, verapamil,

warfarin and amlodipine. Arterial blood gas analysis revealed metabolic acidosis, hypercarbia and hypoxia (pH 7.28, PCO_2 7.49 kPa, HCO_3 11.8 mmol/L, SBE–12.2 mmol/L, O_2 82.8, SBC 13.4 mmol/L). ECG showed sinus bradycardia (rate 62) and left-bundle-branch block. Other biochemical abnormalities included hyperglycaemia (13.5) slightly elevated liver enzymes (ALT 84 IU/L, AST 63 IU/L) creatinine 197 μmol/L, creatinine kinase 295 IU/L (troponin level negative) and an INR of 4.17. The patient denied self-poisoning and blood toxicology (including paracetamol) was negative. A chest x-ray revealed cardiomegaly and possible mediastinal widening. Echocardiography was consistent with findings at a previous examination. Abdominal ultrasound and CT scans demonstrated no abnormalities. The patient developed respiratory failure and was intubated and ventilated. Calcium channel blocker poisoning was suspected because of the bradyarrhythmias, hyperglycaemia and hypotension unresponsive to treatment. Management at this point was supportive with large doses of inotropes. Intravenous calcium chloride, glucagon, sodium bicarbonate, dextrose/insulin were given and a temporary pacemaker was inserted to treat severe bradycardia. Renal replacement therapy was started because of deteriorating renal function. Verapamil level was taken on the second day and retrospectively confirmed the self-poisoning (Verapamil 1740 μg/L and norverapamil 850 μg/L, target range for both is 100–200). Continuous veno-veno haemodialysis (CVVHD) was again attempted owing to rapidly elevating liver transaminases (ALT 1252 IU/L, AST 1377 IU/L) but this was tolerated poorly and discontinued. The patient died two days post admission to the Intensive Care Unit despite these interventions. Conclusion: This report demonstrates calcium channel blocker poisoning unresponsive to standard interventions of intravenous fluids and inotropes, calcium chloride salts, glucagon and pacing. The patient was also treated with dextrose/insulin (insulin euglycaemia), a novel therapy suggested in literature, but this did not show any benefit.

130 CYANIDE POISONING MANAGED WITH HYDROXOCOBALAMIN IN THE UK

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Objectives: Serious or fatal cyanide poisonings are a very rare occurrence in England and Wales. In the UK the licensed antidotes for cyanide poisoning are dicobalt edetate or sodium nitrite, both of which may have serious adverse effects if used when cyanide exposure has not actually occurred. We report a recent cyanide case with a fatal outcome where prompt analytical confirmation of exposure proved difficult and where the antidote hydroxocobalamin, together with sodium thiosulphate, was used in the management of the victim with benefit. Case Report: A 30-year-old male was admitted to Accident & Emergency (A&E) at 3 am on a Saturday morning following ingestion of potassium cyanide salt as suicide in the hospital car park. Initially he was noted to be pale with dilated pupils. Within one hour he had collapsed with cardiac arrest. Investigations revealed an arterial pH of 7.16, an elevated lactate of 11 mmol/L, a blood glucose of 8 mmol/L and a carboxyhaemoglobin level less than 5%. He was intubated, hyperventilated, given IV sodium bicarbonate, and adrenaline as an inotrope. He was also paced by means of a trans-jugular wire, which overcame the EMD and raised the BP to 80/60 mmHg. On consultation with the poison centre use of either sodium nitrite or dicobalt edetate was advocated. Despite strong circumstantial evidence and the clinical presentation dicobalt edetate was not used, although available in A&E, because of the precarious cardiovascular condition of the patient and the fact that laboratory confirmation of cyanide exposure could not be undertaken. Sodium nitrite was located and administered. By chance, NPIS (London) had one sample Cyanokit® pack (Hydroxocobalamin) which was dispatched by police courier, reaching the hospital within 25 minutes. On administration the patient's cardiovascular status improved markedly, but there was unfortunately no CNS improvement, probably because the prolonged arrest had resulted in brain hypoxia. Subsequent analysis of a sample taken on admission revealed a cyanide level in excess of 15 μmol/L (> 4 mg/L). 5 days post-ingestion, despite cardiovascular stability, the man was pronounced brain dead. Conclusion: The case illustrates the importance of having cyanide antidotes readily accessible, and the facility for cyanide assay to be performed outside office hours, particularly if dicobalt edetate is the only antidote available. Hydroxocobalamin, which has less inherent toxicity than other antidotes, proved effective in restoration and support of cardiovascular function in this case. In the absence of laboratory confirmation it can be seen to be a safer antidote than either dicobalt edetate or sodium nitrite.

131 RESOURCES AND QUALITY OF TOXICOLOGICAL CARE IN SPANISH HOSPITALS

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Objective: Previous studies have analyzed the availability of antidotes in Emergency Departments, however, they have not evaluated the existence and organization of other necessary toxicological resources. The aim of this study was

toevaluate the available resources and the quality of toxicological care in the Emergency Departments of Spanish hospitals. Method: A specific survey was designed, which contained 6 sections: the availability of emergency toxicological screening; the presence and composition of an emergency toxicological kit (antidotes and other substances useful in toxicology); post-graduate toxicological training among Emergency Department physicians; the protocols and clinical-toxicological procedures carried out; the access to toxicological information; and the criteria used for gastrointestinal decontamination. In March 2000, the survey was sent to the Emergency Departments of 176 Spanish hospitals chosen at random, but with an equal representation of primary, secondary and tertiary hospitals. The results were analyzed descriptively. Results: Seventy-seven hospitals (44%) responded to the survey, consisting of 24 primary, 27 secondary and 26 tertiary hospitals. 1. The availability of emergency toxicological analyses was inconsistent. 2. All the hospitals had an emergency toxicological kit. In the toxicological kits of the secondary and tertiary hospitals, 81.3% of the 18 most-common substances, components specifically for toxicological use, were present. 3. Some form of post-graduate toxicological training had been undertaken by 73% of Emergency Department physicians. 4. Some toxicological protocols existed in 87% of the Emergency Departments, but in only 57% were these protocols designed by the Department itself. In 26% of the hospitals, the medical staff had published in the area of clinical toxicology, while in 60% the staff had made toxicology-related presentations to a meeting. 5. Toxicological information was available in all Emergency Departments. The quality of this information was scored at an average of 7.3/10. 6. Quality of care was evaluated according to the gastrointestinal decontamination protocols. Eighty-seven percent of hospitals suggested gastric lavage as the preferred method (54% with nasogastric tube). In 35%, patients with a normal consciousness were felt to have incorrect policies regarding gastrointestinal decontamination. Thirty-nine percent of these policies were judged to be incorrect regarding comatose poisoned patients. Conclusions: The availability of emergency toxicological screening and the contents of the emergency toxicological kit should be standardized. Post-graduate toxicological training, clinical research, and the number of clinical protocols should be increased.

132 ACUTE RENAL FAILURE FOLLOWING LATRODECTISM

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Objective: Latrodectism is a syndrome that has been considered very dangerous for human beings. Although acute renal failure after envenomation is reported to be uncommon and usually results from prerenal failure, we describe a patient with acute oliguric renal failure due to combination of prerenal and renal causes following latrodectism. Case Report: A 59-year-old man presented to the emergency room after being bitten by a shiny black spider. He complained of pain ensuing around the wound and radiating proximally and incessant diaphoresis. He also reported palpitations, shaking chills and vomiting. He had a characteristic history of latrodectism with anxiety, severe hypertension, tremor, facial edema and generalized diaphoresis. Additionally, acute renal failure developed following envenomation. Physical examination revealed an ill-appearing, anxious, shivering and flushed patient who was minimally cooperative with the examiner. Facial edema with flushing and generalized diaphoresis were noted. Blood chemistry revealed blood urea nitrogen (BUN) and creatinine levels as 21 and 2.1 mg/dL, respectively with normal electrolytes. Urinary sodium concentration was 120 mEq/L and calculated fractional excretion of sodium (FENa) was above 2 percent. Serum creatinine phosphokinase (CPK) was found as 216 IU/mL. The urinary output diminished to less than 100 mL within the following 6 hours' observation despite aggressive fluid resuscitation. Creatinine levels showed a rapid upward trend over 5 hours (2.1, 2.5 and 2.7 mg/dL, respectively). Ultrasonographic examination did not reveal any kind of abnormality. BUN and creatinine levels rose to 28 and 2.9 mg/dL, respectively, after 8 hours following envenomation. Laboratory finding of the patient such as BUN/creatinine ratio, urinary sodium concentration, FENa and urine sediment support the diagnosis of acute tubular necrosis. After proper monitoring, the patient was administered copious amounts of intravenous saline. The urinary output gradually increased to more than 100 mL per hour until the sixth hospital day while BUN and creatinine levels were 13 and 1.3 mg/dL, respectively. After stabilization and proper treatment, the patient was discharged at the seventh day of admission without any sequelae. Conclusion: It is the first case report on acute renal failure (ARF) resulting from both renal and prerenal causes after a black widow spider envenomation in human species. Clinicians should not overlook the possibility of acute renal failure resulting from renal factors in latrodectism.

133 HYDROFLUORIC ACID AS AN AGENT FOR SELF-MUTILATION

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Background: Estimates indicate that 1% of the US population may manifest self-injury behavior syndromes ranging from self inflicted lacerations to ocular enucleation. Individuals who self-mutilate usually do so with the intent to injure,

but not to kill. Hydrofluoric acid (HF) exposure usually involves industrial accidents, HAZMAT incidents or other accidental exposures. We present the first reported use of HF for self-mutilation. Case Report: A 34-year-old female presented to the emergency department with the complaint of intense pain, redness, and swelling of the right hand for the previous seven (7) days. Physical examination revealed a female patient in moderate distress, with multiple excoriations of the dorsal right hand. The hand was grossly swollen and red but was not infected. The patient was afebrile with a normal white blood cell count. The patient indicated that she used HF in her hobby of etching glass items. On close questioning, she admitted to intentionally applying a dilute (7%) HF solution to her hand, intermittently, for one week in an inexplicable gesture of self-mutilation. She was admitted to the hospital for care of her injury and psychiatric evaluation. Investigation of her past medical history revealed multiple previous episodes of self-mutilation. Conclusion: While the vast majority of HF exposures result from accidental skin exposures, it must be borne in mind that HF may, in rare instances, be used as an agent for self mutilation. The medical toxicologist may be the physician in the best position to diagnose this form of the self-mutilation syndrome.

134 TREATMENT OF ESOPHAGEAL STRICTURE DUE TO CORROSIVE SUBSTANCE INGESTION

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Objective: Corrosive substance ingestion produces chemical burns on contact with tissues. Depending on the depth and the extent of injuries, progressive narrowing of the esophagus or stomach may occur during wound healing. These strictures may become a chronic problem, requiring repeated dilatations or surgical repair. There is little information on endoscopic balloon-dilatation efficacy and safety in the treatment of corrosive esophageal stricture. We report the results of our experience with the technique. Case Series: Esophageal stricture developed in 9 patients (19%) of 47 cases hospitalized due to grade II-III corrosive burns. In 8 patents the stricture developed following suicidal acid ingestion, and one was secondary to alkali ingestion. The stricture appeared clinically 3 weeks to 3 months post-ingestion. At the time of diagnosis, the stricture was long and dense in 2 patients, so esophagocoloplasty was necessary. Seven patients were treated by the endoscopic balloon-dilatation. The diameter of the initial balloon was 8–12 mm. After the successful first dilatation, the interval between dilatations was 1–4 weeks. Progressive widening of the esophageal lumen to 15 mm, was achieved by 3–7 repeated dilatations. Treatment was successful in 5 patients with annular or short tubular stricture, and they currently eat regular diets. Stricture was undilatable in 2 patients. The reason for the poor result in one patient was probably late initiation of the treatment (2 months post-ingestion). In the second case, after the initial successful dilatation, the stricture developed progressively, and despite the repeated dilations, we could not achieve satisfactory widening of the esophageal lumen. We performed a total of 27 dilatations in 7 patients without any complication. Conclusion: Endoscopic balloon-dilatation is safe and efficient, and could be the treatment of choice in the case of esophageal stricture caused by corrosive injury. We point out that it should be started expeditiously, sometimes a few weeks post-ingestion, before the stricture becomes clinically manifested.

135 LACK OF EFFECT OF SINGLE HIGH DOSES OF BUPRENORPHINE ON ARTERIAL BLOOD GASES IN RAT

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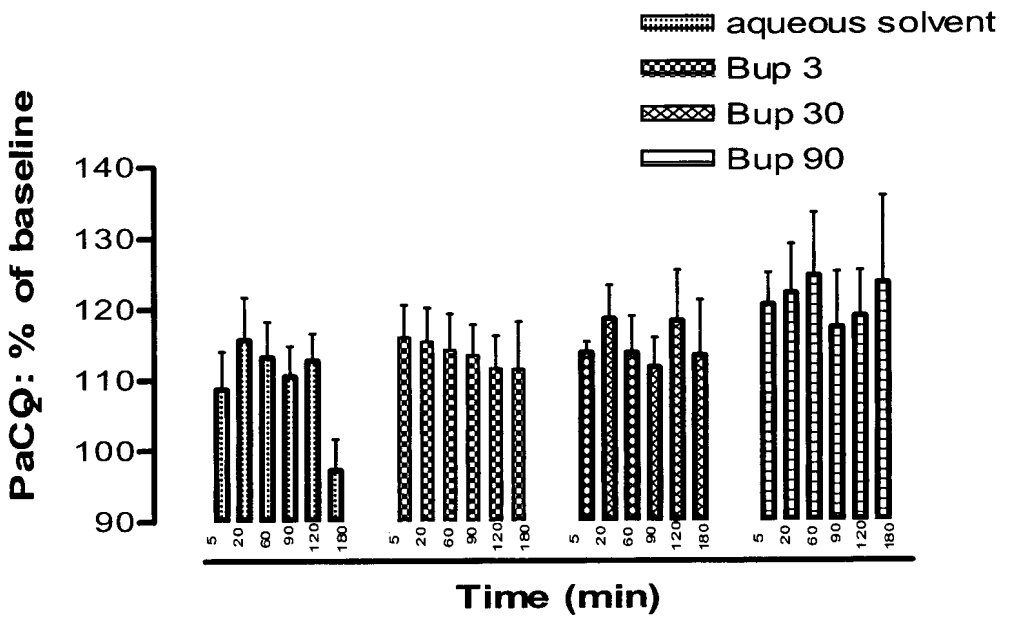
Objectives: High dose buprenorphine, a potent semisynthetic agonist-antagonist for opiate receptors, is now used in substitution treatment of human heroin addiction. Deaths have been reported in addicts (mis)using buprenorphine. We determined the median lethal dose and studied the effects of high doses of intravenous buprenorphine on arterial blood gases in rats. Methods: Male Sprague-Dawley rats were administered buprenorphine intravenously to determine the LD50 using the up-and-down method. Catheterized groups of 10 rats received either no-drug, saline, acid-alcohol aqueous solvent (required to dissolve buprenorphine at a high concentration), or 3, 30 or 90 mg/kg of buprenorphine intravenously. Serial arterial blood gases were obtained over 3 h. Results: The LD50 determined in triplicate was 146.5 mg/kg (range: 142.6–176.5). The mean dose received by surviving animals was 96.9 ± 46.7 mg/kg. There was a significant effect of the acid-alcohol aqueous solvent on arterial blood gases. Excluding the solvent effect, 3, 30 and 90 mg/kg buprenorphine doses had no significant effects. No animal in the arterial blood gas study died, even at intravenous



doses up to 90 mg/kg. Conclusion: The toxicity of intravenous buprenorphine in adult rats, assessed by the LD50, is low. Buprenorphine in doses ranging from 3 to 90 mg/kg had no significant effects on arterial blood gases in adult rats compared with animals receiving the aqueous solvent. These data are consistent with the low toxicity of intravenous buprenorphine in rats. The mechanism of death after the intravenous administration of a lethal dose of buprenorphine remains to be determined.

136 SEVERE AMLODIPINE OVERDOSE TREATED WITH HYPERINSULINEMIA

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Background: Recent recommendations for calcium channel blocker (CCB) overdose include the use of hyperinsulinemia/euglycemia as adjunctive therapy. We report a case of a CCB overdose treated with hyperinsulinemia alone. Case Report: A 34-year-old female with previous medical history of renal failure and hemodialysis attempted suicide by ingesting 12 × 2.5 mg amlodipine tablets. When found one hour after ingestion, she was intubated and transported to the Emergency Department where vital signs were P 60, BP 40/P (on norepinephrine), afebrile; her serum glucose was 325 mg/dL. Because of the history of renal failure, treating physicians did not administer intravenous calcium. Poison Center recommendations were that an insulin infusion of 0.5 units/kg/hr be administered, with serum glucose measurements every fifteen to thirty minutes. Thirty minutes after the insulin infusion (35 IU/kg/hr) was begun, blood pressure improved to 140/60; the norepinephrine infusion was therefore stopped. Approximately six hours after ingestion her vital signs were P 100, BP 140/55 with serum potassium 4.7 mg/dL and glucose of 210 mg/dL. The insulin infusion was stopped. The patient never required supplement glucose. Discussion: CCBs antagonize L-type calcium channels to produce hypodynamic shock, hyperglycemia, and acidosis. During CCB-induced shock, myocytes switch from oxidation of free fatty acids to glucose as primary source of metabolic energy.[1] CCBs, however, prevent adequate utilization of glucose via 1) inhibition of Ca-dependent insulin secretion in the pancreas; 2) resistance to insulin at the receptor level; and 3) development of shock which impairs glucose circulation and delivery.[1] HIE has been documented as an effective adjunctive therapy in verapamil overdose[2]. Hyperinsulinemia without supplemental glucose has been utilized in the treatment of nifedipine overdose; in that case report, hypoglycemia never developed.[3] At infusion rates of 1 IU/kg/hr, the primary effect of insulin is positive inotropic action, with peak effects reached in 15 minutes.[2] Conclusions: Hyperinsulinemia may be effective in reversing hypodynamic shock secondary to amlodipine overdose, and supplemental glucose infusions may not be required in CCB overdose. Further studies are necessary to delineate the role of hyperinsulinemia as primary therapy for CCB overdose, as well as dosing recommendations for concomitant glucose administration. References: [1]Kline J, Leonova E, Raymond R. Beneficial myocardial metabolic

effects of insulin during verapamil toxicity in the anesthetized canine. *Crit Care Med* 1995;**23**:1251–1263. [2]Yuan T, Kerns W, Tomaszewski C, Ford M, Kline J. Insulin-glucose as adjunctive therapy for severe calcium channel antagonist poisoning. *J Toxicol Clin Toxicol* 1999;**37**:463–474. [3]Place R, Carlson A, Leiken J, Hanashiro P. Hyperinsulin therapy in the treatment of verapamil overdose. *J Toxicol Clin Toxicol* 2000;**38**:576–577 (abstract).

137 ACCIDENTAL PARENTERAL EXPOSURE TO DETOMIDINE, AN ALPHA2-ADRENERGIC AGONIST

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Objective: Detomidine is a selective alpha$_2$-adrenoceptor agonist with anxiolytic, analgesic, and sedative properties, which is used as a sedative/hypnotic agent in large animals like horses and cattle. Detomidine is short acting with a plasma half-life of about 1–2 h. One case of intentional poisoning with detomidine and butorphanol has been reported.[1] Accidental, symptomatic human injections of the drug also happen, but data on how to manage them are not easily available. We present a case of accidental exposure causing symptoms and provide additional data that is useful in the management of such poisonings. Case Report: A 23-year-old female injected accidentally 0.3 mL of Domosedan® (3 mg detomidine) IM. The dose was intended for a 200 kg horse. On admission to hospital she was sleepy. No other clinical signs were observed. Oxygen saturation was 97%, BP 122/85, pulse regular, 56 bpm and ECG normal. During the follow-up in the emergency department she developed no further symptoms. After 7 hours she felt normal and did not want to stay in the hospital any longer. No abnormalities were noted in the chest x-ray, or in any of the laboratory tests performed. The marketing authorisation holder has received reports of 4 cases of human detomidine exposures during the last 5-years, none of them fatal. Two were accidental and two intentional exposures. Doses ranged from a few mg to 200 mg. In all poisonings the major symptom has been sedation. Conclusion: In accidental nonsymptomatic human detomidine exposures observation with monitoring of vital signs is a reasonable treatment strategy. If other symptoms than mild sedation develop treatment is symptomatic and analogic to clonidine poisoning. Atipamezole, a specific α_2-adrenoceptor antagonist is used in animals as a detomidine and medetomidine antidote. Although it has not been necessary to use atipamezole in the known cases of human detomidine poisoning, it is reassuring to know that atipamezole has been shown to reverse the cardiovascular and sedative effects of dexmedetomidine in humans.[2,3] References: [1]Reid FM, Tracey JA. Parenteral exposure to detomidine and butorphanol. *J Toxicol Clin Toxicol* 1994;**32**:465–9. [2]Karhuvaara S, Kallio A, Salonen M, Tuominen J, Scheinin M. Rapid reversal of alpha$_2$-adrenoceptor agonist effects by atipamezole in human volunteers. *Br J Clin Pharmac* 1991;**31**:160–5. [3]Scheinin H, Aantaa R, Anttila M, Hakola P, Helminen A, Karhuvaara S. Reversal of the sedative and sympatholytic effects of dexmedetomidine with a specific alpha$_2$-adrenoceptor antagonist atipamezole. *Anesthesiology* 1998;**89**:574–84.

138 TOXIDROMES ASSOCIATED WITH THE MOST COMMON PLANT EXPOSURES

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Objective: Toxicology and botanical references describe a myriad of symptoms that are associated with the ingestion of plants. The symptoms listed are based largely on a limited number of case reports or the personal experience of the authors; there is no consistency between and among the references. The objective of this project is to compile the symptoms that are associated with common plant ingestions Methods: In 1993 the AAPCC TESS began to collect symptom data based on an organ system approach. To profile the most frequent symptoms (toxidromes) associated with commonly ingested plants, AAPCC TESS was queried electronically for the years 1993–1999. The analysis of plant exposures was limited to ingestions involving only one plant to insure that the symptoms were profiled accurately. Exposures to unknown plants and those ingestions involving symptoms coded as unrelated or unknown if related to the exposure were excluded from analysis. Results: The total number of plant ingestions analyzed was 768,284. Symptoms occurred in 53,081 (6.9%) of the patients. Twenty plants (Capsicum, Dieffenbachia, Philodendron, Caladium, Datura, Spathiphyllum, Ficus, Zantedeschia, Narcissus, Epipremnum, Iris, Phytolacca, Eucalytpus, Ilex, Euphorbia, Arisaema, Hedera, Piper, Ephedra, Begonia) accounted for the symptoms in 54.2% of patients. The most common symptoms were: gastrointestinal 64%, dermal 17%, neurological 5%, ocular 4%, respiratory 4%, other 4%, cardiovascular 2%. The toxidrome for each plant has been compiled. Conclusions: Most plant ingestions were not associated with

the development of symptoms. The use of toxidromes based on the symptoms reported with plant exposures will assist in the recognition and assessment of plant ingestion patients.

139 ACUTE CEREBRAL GAS EMBOLISM FROM HYDROGEN PEROXYDE INGESTION SUCCESSFULLY TREATED BY CONTINUOUS INHALATION OF 100% OXYGEN THROUGH THE DAN RESPIRATORY MASK

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Objective: Hydrogen peroxide is an oxidizing agent, but it is very unstable and readily breaks down to oxygen and water. The generation of oxygen in closed body cavities can potentially cause mechanical distension as well as venous or arterial gas embolism. Case Report: A 50-year-old male was brought to our emergency department after the ingestion of alcohol and undiluted hydrogen peroxide (35%), which he assumed to have been water in the refrigerator. On the initial presentation he complained about nausea, vomiting and headache. The acute endoscopy showed II/B oesophageal and III/A stomach injuries in the absence of oral burns or obvious dysphagia. X-rays of the chest and abdomen did not demonstrate air in the mediastinum or free abdominal air from oesophageal or gastric perforation. Toxicological data showed the presence of plasma ethanol level of 2.4%. A few hours later he had increasing symptoms including clumsiness of his right arm, difficulty in speaking an amaurosis totalis. The neurological examination revealed ataxia, dysmetria of the right hand and inability to touch his right hand to his nose. These focal findings with the history of hydrogen peroxide ingestion led to the diagnosis of cerebral gas embolism. A non-contrast computed tomography failed to show a deficiency. The patient was treated by continuous inhalation of 100% oxygen through DAN respiratory mask for 6 hours. He had rapid and complete resolution of his symptoms including neurological deficits. The magnetic resonance imaging (MRI) showed substantial damage as a consequence of two-sided gas emboli. A month later the control MRI demonstrated considerable regression, the control endoscopy revealed subacute gastro-duodenitis. Conclusion: Our patient suffered of cerebral gas embolism due to the ingestion of concentrated hydrogen peroxide. An appropriate but never published therapeutic protocol was introduced with a success.

140 COLLECTIVE INTOXICATION CAUSED BY DATURA STRAMONIUM

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Objectives: Clinical evaluation and outcome of a group of six teenage patients with acute *Datura stramonium* intoxication. This plant has hallucinogenic and therapeutic properties, is widely distributed and rich in alkaloid substances such as hyoscyamine, atropine and scopolamine. The patients presented with an anticholinergic syndrome with variable involvement. Case Series: In October 1999, six teenagers were treated in our emergency department with signs and symptoms of anticholionergic intoxication. (See table).

No	1	2	3	4	5	6
Age (years)	15	18	16	17	21	18
Gender	M	M	M	F	M	M
Seeds	60	30	60–70	20–30	40	20
Toxic substances	ALC + CANN	CANN +AMP	—	—	—	CANN
Pulse	130	120	80	100	120	72
Blood pressure	150/90	130/70	145/80	120/80	157/83	130/60
Respiratory rate	24	26	16	16	20	22
Temperature	40	36	36	36,5	37,4	36
Mydriasis	+	+	+	++	+	++
Hallucinations	+	+	−	+	+	+
Consciousness	COMA	CONFUSE	C & O	AGITATED	AGITATED	C & O
Symptomatic treatment	*	*	*/BZD	*/BZD	*/BZD	*/BZD
Specific therapy	−	−	−	−	−	−
Observation (hours)	1	4	8	17	20	15
Destination	ICU	ADMISSION	HOME	HOME	HOME	HOME
Outcome	FAVOUR	FAVOUR	FAVOUR	FAVOUR	FAVOUR	FAVOUR

ALC: alcohol. CANN: cannabis, AMP: amphetamines, BZD: benzodiazepines. *unspecific support measures for intoxication. M: male, F: female, Favour: Favourable

Results: Anticholinergic symptoms were prominent and early in onset. Treatment of *Datura stramonium* poisoning includes general management measures, (i.e., gastric lavage) and symptomatic therapy for hallucinations and the use of benzodiazepines for agitation. Specific treatment with physostigmine is controversial, owing to the dangers associated with its use. Clinical evolution was favourable in all cases, even in those with multiple substance abuse. In concordance with other reports, *Datura stramonium* intoxication with recreational purposes is mild. In the case of a patient with symptoms and effects of an anticholinergic intoxication, *Datura stramonium* intoxication should be considered.

141 SEIZURES AND CARDIOPULMONARY ARREST AFTER UNINTENTIONAL ACETOMINOPHEN AND DIPHENHYDRAMINE INGESTION IN A PEDIATRIC PATIENT

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Objective: Diphenhydramine is known to have cardiovascular effects including QRS prolongation but these effects are rarely reported in children. We report a case that involves an unintentional ingestion of Tylenol PM (acetaminophen and diphenhydramine) complicated by a prolonged cardiopulmonary arrest with successful resuscitation. Case report: A 2 year old female without any past medical history had an episode of emesis and gagging at home with no apparent cause. It was later discovered that she had access to Tylenol PM (acetaminophen and diphenhydramine). She subsequently had a seizure and was rushed to the pediatrician's office where she received 0.2 mg/kg of diazepam for persistent seizure activity. From there she was transported by helicopter to our institution. During transportation she was intubated and given 0.35 mg/kg of lorazepam for seizures. She then developed wide complex rhythm and two self-limiting episodes of ventricular tachycardia. On arrival to our institution she remained in status epilepticus requiring additional lorazepam. Initial vital signs were: Blood pressure, 86/45 mmHg; Heart rate, 150 beats per minute; Temperature, 39.2 °C. Physical findings included pupils fixed and dilated at 8 mm bilaterally. She developed ventricular tachycardia and torsade de pointes. A normal sinus rhythm was restored by pediatric advanced life support protocol. The patient was lavaged and given activated charcoal. Initial laboratory analysis was significant for acetaminophen, 452 μg/ml. The patient was admitted to an intensive care unit and N-acetylcysteine therapy was started. Her hospital course remained uncomplicated and she was discharged with outpatient Cardiology and Neurology follow up. Conclusion: This case demonstrates serious but rare neurologic and cardiovascular complications that can occur with pediatric diphenhydramine overdoses. Particularly, the various types of arrhythmia that can occur following large ingestions. Pediatric ingestions of adult strength products are more likely to produce life threatening clinical effects. Meticulous history taking and searching of the area give important clues that aid in the management of suspected toxic ingestion.

142 SEA-URCHIN ENVENOMATION ASSOCIATED WITH HEPATITIS

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Objective: Sea urchin stings may produce an injurious and venomous wound. Although numerous writers refer to the danger of pedicellarial stings, there is little worthwhile clinical data. We report a case that developed an infected wound and hepatotoxicity. Case Report: A 47-year-old Taiwanese woman accidentally stepped on a sea urchin with both feet during scuba diving on a beach in Thailand. She had hundreds of stings, faintness, immediate and intense pain. Only three spines were removed on the spot; most spines remained embedded. Treatment with betadine immersion, antibiotics and analgesics were given at a local hospital. When she returned to Taiwan on the next day, she had fever, chills, nausea, and persistent serous discharge and tenderness at the site of the stings. In the following days she felt weakness, and the urine color changed to a tea-color. She received out-patient-department treatment but in vain. Consequently, she was admitted to a hospital on the seventh day post envenomation. Physical examination showed multiple punctate lesions over both soles, painful swelling and redness over the right sole and right lower leg, tender lymphadenopathy over the right inguinal area. Laboratory tests were remarkable with alanine transaminase 810 U/L, aspartate transaminase 320 U/L, WBC 10,200 mm^3 (segmented 63%). The abdomen sonogram was normal. She then received intravenous antibiotics, wound debridement and removal of residual spines. She recovered gradually and was discharged two weeks later. Conclusions: Travel related marine animal injury has an increasing presence in Taiwan. Awareness of such injury could lower the severity in the case of such incidents. This case had an unusual presentation of severe infection and hepatitis; immediate and more aggressive spine removal might lessen the degree of injury.

143 HAVE THE NEW PACK SIZE REGULATIONS IMPACTED ON UK PARACETAMOL OVERDOSE?

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Objective: To ascertain whether recent UK legislation is impacting on calls to a poisons centre involving paracetamol. Introduction: Paracetamol is consistently the most common drug taken in overdose in the UK. Steps taken by the UK government to reduce the number of overdoses included limiting the pack size available to 16 (8 g) on GSL (i.e. available outwith pharmacies), and 32 (16 g) in pharmacies. Method: SPIB telephone enquiry statistics were analyzed for the two years before and after the new legislation was introduced (16th September 1998).
Results:

Year	96–97	97–98	98–99	99–00
Total no. of patient cases	7175	6781	6704	6121
Total no. of paracetamol cases	694	620	592	546
Paracetamol: Patients > 12 years	379 (54.61%)	422 (68.06%)	379 (64.02%)	359 (63.92%)
Paracetamol: Patients ≤ 12 years	267 (38.47%)	162 (26.13%)	163 (27.53%)	137 (25.09%)

In patients of all ages, where amount of paracetamol ingested known:

Year	96–97	97–98	98–99	99–00
>8 g paracetamol ingested	29 (43.94%)	67 (34.90%)	66 (32.67%)	49 (31.01%)
≤ 8 g paracetamol ingested	37 (56.06%)	125 (65.10%)	136 (67.33%)	109 (68.99%)
>16 g paracetamol ingested	17 (25.76%)	25 (13.02%)	35 (17.33%)	25 (15.82%)
≤ 16 g paracetamol ingested	49 (74.24%)	167 (86.98%)	167 (82.67%)	133 (84.18%)

In patients > 12 years:

Year	96–97	97–98	98–99	99–00
>8 g paracetamol ingested	26 (65%)	62 (52.99%)	60 (51.28%)	44 (50.57%)
≤ 8 g paracetamol ingested	14 (35%)	55 (47.01%)	57 (48.72%)	43 (49.43%)
> 16 g paracetamol ingested	17 (42.50%)	24 (20.51%)	31 (26.50%)	21 (24.14%)
≤ 16 g paracetamol ingested	23 (57.50%)	93 (79.49%)	86 (73.50%)	66 (75.86%)

In patients ≤ 12 years:

Year	96–97	97–98	98–99	99–00
> 8 g paracetamol ingested	2 (10%)	0 (0%)	0 (0%)	0 (0%)
≤ 8 g paracetamol ingested	18 (90%)	65 (100%)	74 (100%)	56 (100%)
> 16 g paracetamol ingested	0 (0%)	0 (0%)	0 (0%)	0 (0%)
≤ 16 g paracetamol ingested	20 (100%)	65 (100%)	74 (100%)	56 (100%)

Conclusion: Paracetamol enquiries (as a percentage of total enquiries) have fallen slightly. However the proportion of calls regarding patients who have taken more than 16 g appears to have risen. Conversely, there has been little difference in the number of patients who have taken more than 8 g. These early data suggest that reduction in pack size has not made an impact on the scale of overdose commonly encountered, and has not deterred a small proportion of patients from consuming much higher doses of paracetamol.

144 ETHYLENE GLYCOL TOXICITY DESPITE THERAPEUTIC ETHANOL LEVEL

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Objective: Ethanol competitively inhibits alcohol dehydrogenase and is used therapeutically to treat ethylene glycol (EG) ingestions to prevent EG metabolism to toxic organic acids. We report an apparently previously unpublished phenomenon: Classic EG toxicity including high anion-gap metabolic acidosis, hypocalcemia, and renal insufficiency in a patient with a "therapeutic" ethanol level. This patient ingested ethanol subsequent to ingesting and metabolizing EG, thus developed EG toxicity despite presenting to medical care with a therapeutic ethanol level and a non-toxic EG level. Case Report: A 60-year-old man found unresponsive was brought to the emergency department. Hypoglycemia with glucose 1.33 mmol/L (24 mg/dL) was treated with IV infusion of 50 mL of 50% dextrose. Laboratory investigations revealed: initial serum ethanol 46.2 mmol/L (213 mg/dL), ethylene glycol 2.2 mmol/L (14 mg/dL), bicarbonate 3 mmol/L, blood urea nitrogen (BUN) 12.1 mmol/L (34 mg/dL), creatinine 274 μmol/L (3.1 mg/dL), calcium 2.2 mmol/L (9.1 mg/dL), anion gap 39 mmol/L; and 45 minutes later calcium 2 mmol/L (8.3 mg/dL), anion gap 35 mmol/L. The patient received one liter of intravenous normal saline and sodium bicarbonate (132 mEq, IV). Serial laboratory investigations were: at three hours; ethanol 27.7 mmol/L (128 mg/dL), pH 6.92; PCO_2 14 mmHg, serum electrolytes unchanged; at seven hours, calcium 1.7 mmol/L (7.1 mg/dL), anion gap 28 mmol/L, osmolal gap 67 mOsm/L, pH 7.01, and PCO_2 39 mmHg; at 10 hours ethanol 2.1 mmol/L (10 mg/dL), bicarbonate 7 mmol/L, BUN 13.5 mmol/L (38 mg/dL), creatinine 291.7 μmol/L (3.3 mg/dL), anion gap 24 mmol/L, osmolal gap 44 mOsm/kg; at 20 hours, bicarbonate 7 mmol/L, BUN 14.2 mmol/L (40 mg/dL), creatinine 282.8 μmol/L (3.2 mg/dL), calcium 1.9 mmol/L (7.7 mg/dL), anion gap 29, pH 7.29, PCO_2 32 mmHg; and at 28 hours, BUN 14.9 mmol/L (42 mg/dL), creatinine 194.4 μmol/L (2.2 mg/dL), calcium 1.3 mmol/L (5.5 mg/dL). After four-hour hemodialysis, the bicarbonate was 28 mmol/L, BUN 3.9 mmol/L (11 mg/dL), creatinine 79.5 μmol/L (0.9 mg/dL), and calcium 1.9 mmol/L (7.7 mg/dL). Subsequent serum chemistry remained normal or rapidly normalized. Conclusion: A serum ethanol of 21.7 mmol/L (100 mg/dL) prevents EG metabolism up to 128 mmol/L (800 mg/dL) and is considered adequate empiric treatment of EG ingestions. This patient's presenting levels of ethanol 46.2 mmol/L (213 mg/dL) and ethylene glycol 2.2 mmol/L (14 mg/dL) might mislead clinicians to inappropriately exclude the diagnosis of EG toxicity. This patient developed classic EG toxicity including high anion-gap metabolic acidosis, hypocalcemia, and renal insufficiency that corrected after hemodialysis. The patient later admitted to ingesting EG and subsequently ingesting ethanol. As a result, he metabolized EG in the absence of a protective serum ethanol level, resulting in severe EG toxicity despite his initial misleading ethanol and EG levels. This unusual case demonstrates that EG toxicity cannot be excluded based on a therapeutic ethanol level or a non-toxic EG level.

145 ACUTE ACONITE POISONINGS: A REVIEW OF 219 CASES

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Objective: Aconite (common name for *Aconitum napellus*) is one of the well-known toxic plants since antiquity. There are 14 species of aconite growing in Kazakstan where it is still popular as a part of traditional herbal medicine. Therefore, it often becomes a reason for severe accidental and suicidal poisonings. During the last 5 years (1995–1999) 278 patients were hospitalized with acute aconite poisoning and 36 of them died (12.9%). The objective of our study was to characterize the clinical presentation of acute aconite poisonings in our region. Methods: A retrospective case review of 219 hospitalized patients (including 8 fatalities) was performed. Results: There were 53 (24.2%) suicidal and 166 (75.8%) accidental poisonings. In 101 (46.1%) cases symptoms appeared within the first 15–20 minutes after ingestion, in 39 (17.8%) cases–within 20–60 minutes, in 29 (13.2%) cases–after 1 hour. Major symptoms of poisoning included: nausea (80.8%), vomiting (62%), sensory disturbances (84.0%), agitation (51.1%), coma (14.1%), respiratory arrest (16.9%), cardiac disturbances (85.6%). Cardiac disturbances included: bradycardia (6.8%), tachycardia (11.9%), extrasystole (73.1%), fibrillation (7.8%) and arterial hypotension (51.1%). In 199 (90.9%) cases aconite alkaloids were detected in the urine of the patients. Treatment included gastric decontamination, forced diuresis and supportive treatment. Lidocaine was used routinely to reverse cardiac arrhythmias. In 26 (11.9%) cases cardiopulmonary resuscitation was performed. Conclusion: Acute aconite poisoning has potentially dangerous clinical features. Further investigations are necessary to define effective treatment protocols including a search for specific antidotes for major aconite alkaloids.

146 SEVERE BRADYCARDIA IN A PEDIATRIC LITHIUM TOXICITY

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Background: Although CNS, renal, and GI effects are commonly encountered in lithium toxicity, cardiovascular effects are less well understood and infrequently described. Case Report: An 11-year-old, nonverbal, autistic female with no history of cardiac abnormalities presented with 1 week of fever, anorexia, and vomiting and subsequently developed ataxia, lethargy, and respiratory distress. In the PICU, she had several general tonic-clonic seizures that were managed with benzodiazepines and phenobarbital. She had no independent access to her medications which were continuously dispensed by her parents even throughout her GI illness despite her anorexia. They included sustained release lithium, methylphenidate, haloperidol, benztropine, and guanfacine. The initial vital signs were: BP, 80/40 mmHg; P, 30–40 bpm; RR, 24–44 min; T, 102°F rectal; O_2Sat, 80% on room air. The physical examination was significant for bradycardia without murmurs, clonus, and hyperreflexia. Her ECG showed a junctional bradycardia with a QRS 64 ms and QT_c 507 ms. Transesophageal esophogram showed an ejection fraction of 21% and global hypokinesis of the left ventricle. The laboratory results were normal except for her BUN/Cr (24/0.9) and a lithium level of 6.54 mEq/L for which she received emergent hemodialysis. During dialysis, the patient's cardiac rhythm spontaneously reverted to normal sinus rhythm. A repeat echocardiogram following her second dialysis showed an ejection fraction of 31% and a normal LV shortening fraction. Conclusion: Although lithium poisoning is infrequently associated with reversible cardiac conduction abnormalities, this has not been previously reported in children. This case also demonstrates lithium-induced myocardial depression and its reversal by hemodialysis.

147 GRADE I-II ATRIOVENTRICULAR BLOCK FOLLOWING LILY-OF-THE-VALLEY (CONVALLARIA MAJALIS) INTAKE: A REPORT OF THREE CASES

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Objective: Although it is well known that "Lily-of-the-valley" contains cardiac glycosides, the risk of poisoning following ingestion is considered small. We report three cases of ingestion of Lily-of-the-valley by children. Case Report 1: A 6-year-old boy was admitted to hospital following ingestion of 8–10 flowers from Lily-of-the-valley. The patient is otherwise healthy. On admission physical examination was normal, ECG showed sinus rhythm with a PQ-time of 0.17 seconds and incomplete right branch block. He was treated with gastric lavage and active charcoal and was observed. His PQ-time increased to 0.20 seconds without any arrhythmias. The further course was uneventful. Case Report 2: A 6-year-old boy was admitted following ingestion of 1–2 flowers from Lily-of-the-valley. Upon admission his physical examination was normal. ECG showed sinus rhythm, with 110 beats per minute and a PQ-time of 0.18 seconds. He was observed in the intensive care with increasing PQ-time to 0.40 seconds without arrhythmias. He had normal lab values. He was treated with gastric lavage, but not with active charcoal. The further course was uneventful. Case Report 3: A 4-year-old boy was admitted following chewing on a leaf from Lily-of-the-valley. He was given active charcoal in the day care center and admitted to hospital. He was observed by telemetry and had during the evening and night four episodes of AV-block grade II. The further course was uneventful. The concentrations of cardiac glycosides are presently being analyzed. Conclusion: Lily-of-the-valley contains digitalis glycosides but is considered to be rather harmless following ingestion. The cases reported indicates that significant conduction disturbances may occur following ingestion of small amounts of Lily-of-the-valley.

148 ANALYSIS OF PEDIATRIC CLONIDINE EXPOSURES REPORTED TO POISON CENTERS

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Objective: The prevalence of pediatric clonidine use is increasing, especially in young children. The purpose of this study was to evaluation the demographics, toxicity and trends in pediatric clonidine exposures reported to the American Association of Poison Control Centers (AAPCC) Toxic Exposure Surveillance System (TESS) over a 7-year period. Methods: The AAPCC TESS database was queried for all pediatric clonidine exposures between 1993 and 1999. Cases involving more than one substance or not followed to known outcome were excluded. Clonidine was assumed to be the child's medication in acute-on-chronic and chronic exposures. Using a relational database, summary statistics were

compiled. Results: Of 17,374 pediatric clonidine exposures, 10,060 cases involved clonidine only and were followed to known outcome. The age distribution was: 5,696 (57%) children under 6 years, 3,470 (34%) 6–12 years and 894 (9%) 13–18 years of age. Controlling for the overall increase in pediatric exposures in the database, the number of exposures increased annually, with 2.5 times as many clonidine exposures in 1999 compared with 1993. Clonidine was the child's own medication in 2,011 (20%) of exposures. In 6–12 year olds clonidine was the child's medication in 35% of exposures, compared with 10% in children < 6 years old and 26% in adolescents. The proportion of cases in which clonidine was the child's medication increased during the study period with a five fold increase in children under 6 years of age, a two fold increase in 6–12 year olds and a 50% increase in adolescents. While accidental overdose was the most frequent reason for exposure in children under 6 years of age, therapeutic error and suspected suicide attempts were the most frequent reasons in 6–12 year olds and adolescents, respectively. In 40% of children, no symptoms occurred. In symptomatic children the most common symptoms were lethargy (80%), bradycardia (17%), hypotension (15%) and respiratory depression (5%), of which 23 children experienced respiratory arrest. The majority of exposures resulted in no effect (40%) or minor effects (39%). Moderate effects occurred in 1,907 (19%) children, major effects in 230 (2%) children and there was one fatality in a 23-month-old girl. Of the moderate and major outcomes, 20% and 12%, respectively, involved the child's own medication. Conclusions: Poison center data demonstrate similar trends as therapeutic use data. While the majority of clonidine exposures resulted in minimal toxicity, serious toxicity and death can occur. The trend toward increasing number of exposures in children, especially with evidence of toxicity in children on clonidine therapeutically, is cause for concern.

149 APNEA IN AN INFANT AFTER INTRAOCULAR ADMINISTRATION OF ADULT STRENGTH OPHTHALMIC SOLUTIONS FOR GLAUCOMA

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Background: Medical therapy of glaucoma involves multiple topical medications to decrease aqueous humor production and increase aqueous outflow. Brimonidine is a relatively selective α_2 adrenergic agonist with effects similar to the antihypertensive medication clonidine. Cosopt® is a combination of a carbonic anhydrase inhibitor (dorzolamide) and a β adrenergic antagonist (timolol). We report a case of apnea associated with ophthalmic brimonidine and Cosopt® use in an infant. Case Report: A 6-month-old infant born prematurely at 27 weeks gestation with no prior history of respiratory difficulties was treated for glaucoma with 2 ophthalmic eye drops, brimonidine 0.2% and dorzolamide hydrochloride/timolol maleate/20mg/5mg/mL. One drop of each medication was instilled in each eye, and about 30 minutes later, the patient developed several episodes of apnea, each lasting 10–15 seconds with no associated cyanosis, witnessed by her parents. In the hospital, her vital signs were: pulse, 120/min; BP, 101/49 mmHg; respirations, 42/min; O_2 sat, 100%; temperature, afebrile. She was easily arousable with normal pupils and an unremarkable physical examination. Interventions were not needed. She was observed in the PICU overnight and did not have further apneic episodes. Her vital signs the next day were: pulse, 135; BP, 87/41; respirations, 35; temperature, 96.9°F. The brimonidine and Cosopt® were discontinued, and her apnea did not recur. Conclusions: Apnea has been previously reported for both the alpha$_2$ adrenergic agonists especially clonidine and beta adrenergic antagonists. Both medications have only one formulation for both adults and children. This case illustrates the potentially severe side effects of using adult strength ophthalmic solutions in infants.

150 A CASE OF SEVERE LEVOTHYROXINE POISONING IN A CHILD

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Objective: Accidental ingestion of levothyroxine in children has been frequently reported, but symptoms are in general mild or lacking, even though in some cases quite high doses have been ingested. Only a few cases of massive overdose followed by severe symptoms have been described. Therefore this case of massive exposure in a child will add to the experience as concerns symptomatology and treatment. Case Report: A 3.5-year-old and previously healthy boy ingested around 10 mg of levothyroxine. He was brought to the emergency department 3 to 4 hours postingestion and was admitted for observation. Emesis was not induced and no activated charcoal was given. On admission he was asymptomatic apart from a slight increase in heart rate (125 beats/min). 10 to 12 hours after ingestion the patient became increas-

ingly agitated, restless, hyperactive and unable to concentrate. His mental status did not return to normal until day 7 after exposure. During the first 3 days after admission, his heart rate increased to 195 beats/min when awake (120 beats/min during sleep). ECG showed sinus rhythm with isolated ventricular extrasystoles. Body temperature was raised to 38 °C and remained so until 7–8 days after ingestion. No other symptoms occurred. Treatment with propranolol (2 mg/kg/24h divided into three doses) was started 24 hours after exposure. The patient's heart rate gradually decreased, and propranolol was discontinued 10 days after admission with a remaining slight increase in heart rate. He was discharged on day 12 with no medication. Serum free thyroxine (T_4) and free triiodothyronine (T_3) levels 24 hours postingestion were >80 pmol/L (normal range 10–25 pmol/L) and >60 pmol/L (normal range 4–9 pmol/L), respectively. 10 days after ingestion free T_4 was 34 pmol/L and free T_3 was 11 pmol/L. Conclusion: 10 mg of levothyroxine caused severe poisoning (PSS grade 3) in a 3.5-year-old boy, requiring beta blocker treatment for 10 days and a hospital stay of 12 days. Symptomatic treatment was sufficient to alleviate symptoms, and this confirms the assumption that enhanced elimination–discussed in cases of repeated overdose–is not necessary after acute exposure, even if massive. In consistency with earlier reports, T_3 and T_4 levels in this case were still elevated 10 days after the exposure although the patient by this time was asymptomatic.

151 INTERMEDIATE SYNDROME AFTER EXPOSURE TO CHLORPYRIFOS IN A 16-MONTH-OLD FEMALE

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Objective: We describe a case of intermediate syndrome (IMS) after chlorpyrifos ingestion in a toddler despite a continuous pralidoxime infusion. Case Report: A 16-month-old, 10 kg female with a history of pica ingested and spilled on her skin a termite pesticide containing chlorpyrifos. All clothes were removed immediately and the child was bathed. She was brought to an emergency department where she initially displayed no signs of cholinergic excess. Over the next three hours she developed pulmonary edema, became increasingly lethargic with tachycardia (HR 160 bpm) and required mechanical ventilation. She received 25 gm activated charcoal by mouth and two doses of 0.02 mg/kg IV atropine. Her neurological status deteriorated and the patient became unresponsive. Pralidoxime was administered in two 150 mg boluses at 4.5 and 5 hours post ingestion. At 8 hours post-ingestion she experienced a generalized tonic clonic seizure which resolved after 0.5 mg IV lorazepam. At 9 hours post-ingestion a pralidoxime infusion was begun at 15 mg/kg/hr and she was transported to a tertiary care facility. Over the next 48 hours she remained on a continuous infusion of pralidoxime at 15 mg/kg/hr without interruption. Twice throughout the night the child was given lorazepam 0.05 mg/kg for agitation. Her vital signs remained normal and lung sounds were clear bilaterally. At 24 hours post-ingestion the child had a normal neurologic exam, showed no signs of cholinergic excess and was extubated without difficulty. She was noted to be playful and physically active in the Pediatric ICU. 26.5 hours post-ingestion the child suddenly became flaccid and bradycardic (HR 44) with no respiratory effort. She received 0.02 mg/kg IV atropine for bradycardia and was emergently re-intubated. Muscarinic symptoms of excessive secretions did not return and she did not receive further atropine. The pralidoxime infusion continued at 15 mg/kg/hr. She required mechanical ventilation for the next 17 hours. She was subsequently extubated and had a normal neurological exam. She remained on pralidoxime for another 12 hours post extubation. She was discharged home four days post ingestion with no residual neurological deficits. Conclusion: The child's delayed onset of respiratory arrest and flaccid paralysis after an asymptomatic period is consistent with intermediate syndrome. We believe this case is unusual because it is the first reported case associated with the organophosphate chlorpyrifos and the first reported case in a young infant. Additionally, this event occurred while the child received continuous and adequate oxime therapy.

152 CHRONIC CARBON MONOXIDE POISONING IN CHILDREN

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Introduction: Acute carbon monoxide poisoning in children has been well described and the possibility of chronic neurological effects is recognised. However, the effects of long term, low level exposure have not been discussed. Methods: We have retrospectively reviewed the cases of children seen in our out-patient clinic where the referral indicated a history of possible chronic exposure to carbon monoxide at levels which did not cause severe acute poisoning.

Data analysis included patient demographics, history and technical evidence for exposure, clinical and educational findings, and outcome. Results: We identified ten children from five families. Their ages at the time when exposure ceased ranged from 22 months to 16 years. The symptoms reported included headaches, nausea, vomiting, dizziness, tiredness and lethargy, emotional liability, upper respiratory tract symptoms and chest tightness. School reports indicated poor performance and behavioural problems. The family backgrounds were not limited to the socially disadvantaged. After exposure ceased, there was evidence both of improved health and educational performance. Conclusions: Chronic low level exposure to carbon monoxide in children can cause both ill health and impaired school performance. Diagnosis in such cases often lacks evidence of raised carboxyhaemoglobin concentrations and must be made on the medical history, technical reports on the source of carbon monoxide and school records.

153 POISONING IN CHILDREN UNDER ONE YEAR

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Objective: To evaluate enquiries made to a poison centre concerning children under the age of 1 year. Method: All telephone enquiries involving children under the age of 1 year were extracted from our databases for the years 1990 to 1999 and analyzed for age, sex and agents involved. For the years 1997–1999 data for incident type and location was also available for analysis. Statistical comparisons were by Chi-squared analysis. Results: In the 10 years 1990–1999 NPIS, Edinburgh centre received a total of 69293 telephone enquires, of which 1801 (2.6%) involved children <1 year and 24113 (34.8%) 1–<5 y. More than 87% of telephone enquiries come from Scotland, which has a population of 5.5 million. Of the calls about those under 1 y: 168 children were aged 0–<3 months, 176 3–<6 months, 457 6–<9 months and 1005 9–<12 months, reflecting increasing access to agents with increasing age. Gender was known in 86% of cases: 812 boys and 739 girls. Enquiries involved pharmaceuticals (37%), household products (24%), plants (15%), toiletries (7%), others (17%). The pattern of agents ingested varied with age reflecting mobility and access. The pattern of enquiries in those aged 1-4y was pharmaceuticals (52%), household products (19%), plants (5%) and toiletries (8%), others (16%). The distribution of products was significantly different between the age groups <1 and 1 to <5 years ($p < 0.01$). Where data was available (1997–1999) therapeutic errors were found to be more common in those <3 months (21%) and 3–<6 months (27%). For those 6 months or over therapeutic errors were rarer (4% and 1.2% for each group). All incidents in those < 1 year were accidental, and the majority took place at home. However, in the < 3 month group 9 incidents took place in hospital or at a GP surgery, for 3–<6 months 4 were in a medical facility and for those 6 months or over only one. Only 5 cases in the under ones were considered serious or potentially serious. These involved aminophylline by injection, baby wipes, extravasation with an infant's parenteral nutrition, chloroform and cannabis. Conclusions: In the period of this study only 3% of enquiries to this National Poisons Information Service centre involved children under 1 year and these incidents were seldom serious. Therapeutic enquiries are more common in those under 6 months. The pattern of enquiries varies with age and is different to that in children aged 1–5 y, e.g. household plants were common in the 6–<9 month group and garden plants in the 1–<5 group. Preventative approaches need to target the needs of those <1 y as well as those of older children.

154 TOXIC EXPOSURES IN CHILDREN UNDER 6 MONTHS: CHARACTERISTICS AND PLEA FOR PREVENTION

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In contrast to older, active exploring children, toxic exposure in babies under 6 months of age has received little attention in the medical literature. Objectives: To describe patterns of toxic exposures in children under 6 months. Method: Descriptive analysis of all toxic exposures recorded on the on-line toxicovigilance database "CIGUE" from 1995 to the end of 1999. Data collected included age, sex, type of incident, route of entry, toxins involved, location of incident, form of presentation, symptoms, treatment, outcome. For children at home, outcome was obtained by phone follow up. Severity of clinical course was evaluated using Poison Severity Score calculated retrospectively after follow up. Results: From 1995 to the end of 1999 we collected 960 toxic exposures involving children under 6 months, compared to the 46,700 toxic exposures in children under 15 years recorded during the study period. This was 2% of inquiries in children and 56% of these were in males. Most exposures were unintentional (99%), and resulted from therapeutic mistakes (504; 52%), therapeutic accident (20; 2%), product misuse (120; 13%), or indoor air pollution (85; 9%). The incident

was in the home in 97%. Only 2% (18) of exposures occurred in hospital. Therapeutic mistakes involved liquids in 81%, and were related to overdosage (280; 56%), errors in the route of administration (121; 24%) and confusion between drugs (103; 20%). The commonest drugs involved in mistakes were synthetic anticholinergic and anticholinergic agents (15%), antiseptics and disinfectants (15%), expectorants (13%), caries prophylactic agents (10%), and paracetamol (10%). This pattern differs from that in older children. Product misuse almost always involved liquids (92%) and was related to confusion and accidental drug administration by oral (97/120; 81%), nasal (7; 6%) or ocular (4; 3%) routes. The commonest products involved were glass sterilizers (44; 37%), disinfectants (8; 7%), and mercury thermometer fluid (5; 4%). Complete follow up was obtained for 824 (86%). No deaths occurred. 196 (20%) children were symptomatic. Only 30 cases (3.6%) resulted in moderate (23) or severe poisoning (7). The most severe were carbon monoxide (10), glass sterilizers containing quaternary ammonium compounds (6), antiemetics and antinauseants (5). Conclusion: Toxic exposures in children under 6 months result from adult mistakes and are far less frequent than in older children. Therefore, preventative measures must be focused on baby caretakers. There is a strong case for educating mothers in liquid drug administration and, if required, dilution techniques both in hospital and at home. To avoid confusion with pharmaceuticals, sterilization products should never be marketed in the same types of package.

155 THE BgVV STUDY: DANGEROUS LAMP OILS. DOCUMENTATION OF LAMP OIL ACCIDENTS IN GERMAN CHILDREN'S HOSPITALS

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Background: The toxic risks of aspiration of a lamp oil are increasingly recognised. These formulations include kerosene, petroleum distillates and, in more recently introduced products, paraffin fractions with chain lengths between C8 and C16. Small aspirations can lead to chemical pneumonia and several children have died because there is no specific therapy for the lung damage caused. Because of the high incidence of episodes involving lamp oils this is an increasing public health concern. A number of European countries have supported German proposals for the restriction of coloured and perfumed lamp oils based on paraffin and petroleum distillates. Such oils were prohibited in the European Community after the 1st of July 2000. Germany introduced restrictions from 1st of January 1999 and substitute preparations, based primarily on "Biodiesel"–fractions in the form of fatty acid methylesters were developed for use in ornamental lamps. Thus in 1999 a number of substitutes for lamp oils were introduced to the German market, but there was a lack of human toxicology data on these new preparation. In order to obtain clinical information on lamp oil accidents and to develop preventative strategies the BgVV established a "Dangerous Lamp Oils" study in collaboration with the Research Centre for Paediatric Epidemiology ESPED (*Documentation Unit for Rare Paediatric Diseases in Germany*) and German children's hospitals. Methods: Over 400 hospitals were informed of the potential problems of lamp oil ingestion using informal study material. If children were admitted after lamp oil ingestion, data was collected by questionnaire and telephone follow up was made to the responsible physician and to the parents of the child. The main focus of the study was on product identification, to be achieved by obtaining photocopies of the labels of the products involved, or by a validation process from information provided by physicians or parents. Cases were to be assessed on the basis of the precise product ingested and the symptoms, signs and clinical course. Data was analyzed using the SAS® package after the first year of the study in March 2001. Preliminary results: from 1st March to 15th November 2001, 107 lamp oil ingestions requiring clinical care were reported. In the 65 questionnaires examined physicians reported 30 chemical pneumonias, in one case requiring ventilation in an intensive care unit. Lamp oil was often drunk straight from an ornamental lamp and the amounts swallowed were small, often only a sip. A major problem identified was the exact identification of the lamp oil involved, especially in hospital cases. Although parents often brought the container to the hospital for identification these were thrown away to avoid further accident. Product labels were therefore often unavailable, and product validation lacking. We have attempted to improve product identification using a poster pointing out the importance of this aspect. In the data set analyzed to date all chemical pneumonias were caused by lamp oils containing kerosene, petroleum distillate, and paraffin formulas. Three cases of Biodiesel ingestion developed no toxicity but this number is too small for a proper risk assessment. These data indicate that households still have available older preparations of lamp oil and that substitution of these with potentially safer products will take more time. We think it is necessary to collect data over a period of three years in view of these findings. Fuller details of this study will be presented.

156 RETROSPECTIVE STUDY OF PATIENTS EXPOSED TO ESSENTIAL OILS IDENTIFIED BY ENQUIRES TO THE NPIS (CARDIFF CENTRE)

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Objectives: Essential oils are potentially toxic substances. This study examined the outcome of cases of essential oil poisoning reported to the NPIS (Cardiff Centre) over a six-month period. Methods: Follow-up questionnaires were sent to consultants of patients suspected of essential oil poisoning identified by enquiry to the NPIS. The questionnaire requested details of agent, quantity, time between exposure and presentation, clinical features and outcome. Results: Fifty-five cases were identified (0.4% of total enquiries for 6-month period). Forty-five percent of the patients were male, 87% of patients were under five years and 75% of cases remained asymptomatic. There were no cases of serious toxicity and the majority (86%) of the symptoms recorded were gastrointestinal (vomiting, nausea, abdominal discomfort or oropharyngeal discomfort). There was one report of ataxia, two of flushing, three with dermal irritation and one case of slightly raised ALT and eosinophilia. Of the patients with symptoms, 93% developed these within 1 hour of exposure or presentation at hospital. Conclusion: There were no serious cases of essential oil poisoning reported during the six months of this study and no serious cases have been reported to the NPIS (Cardiff Centre) since the study closed. It appears that serious poisoning due to essential oils is rare. The majority of symptoms were of a mild gastrointestinal nature and appeared either within one hour of exposure or by the time of presentation. Literature reports of cases of serious essential oil poisoning symptoms also report rapid onset.[1–5] Therefore, hospital admission may only be necessary for patients who present with symptoms or who develop them within one-hour of exposure.

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157 CHILD ABUSE BY POISONING: AN EMERGING THREAT TO THE HEALTH OF CHILDREN

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Background: Child abuse and neglect is an important issue. Abuse by poisoning (chemical abuse) is the most difficult to identify because of the high number of unintentional toxic exposures and the lack of external signs of violence. Literature reports are rare. In recent years we have observed an increase of such cases in Trieste. Objective: To emphasize the need for considering intentional poisoning of children and adolescents by parents or other persons. Methods: At Trieste children under 16 years presenting to the Emergency Room (ER) because of toxic exposure are recorded. Information stored includes epidemiological and clinical characteristics. Exposure is classified as accidental (unintentional self-exposure), deliberate (intentional self-exposure), passive (unintentional from environment or other persons) or due to abuse (intentional by other persons). Severity is assessed according to MSPC score (*J Toxicol Clin Toxicol* 1995;**33**:223–231). For the present study the database for 1990 to 1999 has been reviewed with respect to Trieste residents. Results: Out of 816 subjects registered in 1990–99 under the age of 16 abuse by poisoning has been documented in 8 cases, reported in the table. In other cases abuse was also suspected but not demonstrated.

Age	Gender	Substance	Offender	Score	Place
0*	M	Benzodiazepine	Mother	2	home + hospital
1	F	Carbamazepine	Mother	2	home
3*	M	Benzodiazepine	Mother	2	home + hospital
5*	F	Benzodiazepine	Mother	2	home + hospital
9	M	Benzodiazepine	Mother	2	home
10	F	Not identified	Unknown	1	outside home
14	M	Whisky, benzodiazepine	Friends	1	other house
15	F	Cannabis	Adult	2	disco

* From one family.

Abuse was observed both in boys and girls, with higher frequency in older children (3/173 aged 10–15, 2/70 aged 5–9) than in the youngest children (3/544 under 5). In most of the cases benzodiazepines were used and the offender was the mother, with psychiatric problems. In most instances severity was moderate. In subjects older than 10 the abuse was explicitly declared, in younger children it was diagnosed on the basis of the unlikeliness of the history, or of repeated poisoning (4 cases). In the three children from one family the abuse carried out at home was repeated in hospital. Conclusions: Our study demonstrates an increasing recognition of cases of abuse by poisoning in children and adolescents at Trieste, in addition to other types of abuse and neglect. We cannot determine if this is a true increase of these events, or simply our higher diagnostic suspicion. The possibility of intentional poisoning of children, particularly by parents, must be carefully considered in childhood poisoning.

158 QUATERNARY AMMONIUM FOR BABY BOTTLE GLASS STERILIZERS: ISN'T IT TOO RISKY?

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Background: The potential toxicity of glass sterilizer misuse in children under 6 months is overlooked and may expose babies to potential risk. Objectives: to evaluate toxicity of different glass sterilizers in children under 6 months. Method: descriptive analysis of all toxic exposures collected on the online regional toxicovigilance database "CIGUE" from 1995 to the end of 1999 admitted in 6 different participating departments. Data collected included age, sex, type of incident, location of incident, route of entry, products involved, product presentation, symptoms, treatment, and outcome. The severity of the clinical course was evaluated using the Poison Severity Score calculated retrospectively after follow up. For comparison glass sterilizers were classified in 2 groups, according to composition and form of presentation. *Group 1*: Glass sterilizers containing sodium hypochlorite; *group 2*: Glass sterilizers containing quaternary ammonium. Results: From 1995 to the end of 1999, 960 toxic exposures concerning children under 6 months were collected. Forty-four (4%) resulted from glass sterilizer unintentional misuse (group 1: 34, 77%; group 2: 10, 23%) and 26 (59%) were in males. Incidents occurred in the home in 43 babies (98%) and hospital in 1 (2%). In 39 cases (89%) the route of entry was oral and was related to preparation of a baby bottle with water containing diluted glass sterilizer or administration of pure glass sterilizer confused with pharmaceuticals. In 5 cases (11%), route of entry was nasal and was related to administration of pure glass sterilizer confused with saline sodium solution for nasal administration. 13 children (30%) were symptomatic. Severity was moderate in 1 and severe in 5. All the 5 severe cases belong to group 2 (50%) and should be compared to the overall low severity of toxic exposures in children under 6 months (less than 1%). At 1 month there were residual complications in 2 children, both in group 2. In comparison, group 1 toxic exposures were less harmful, required less hospital admission, less ICU intervention and resulted in fewer sequelae. Conclusion: Toxic exposure to baby bottle glass sterilizers containing quaternary ammonium has a high incidence of severe complications in children under 6 months when compared with those induced by sterilizers containing sodium hypochlorite. To reduce the consequences of misuse of baby bottle glass sterilizing solution, quaternary ammonium should be prohibited, and to avoid confusion with pharmaceuticals, these products should never be packaged in the same way as drugs.

159 SEVERITY OF CLINICAL MANIFESTATIONS AFTER EXPOSURE TO QUATERNARY AMMONIUM BY NASAL ROUTE: 9 CASES IN CHILDREN

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Background: The caustic effects of quaternary ammonium on digestive mucous membranes are well known when toxic exposure occurs by the oral route. Little has been published on toxic manifestations of quaternary ammonium by the nasal route. Objective: To describe the clinical manifestations of toxic exposure by the nasal route. Method: Descriptive analysis of all nasal toxic exposures with products containing quaternary ammoniums collected on the online regional toxicovigilance data base ⟨⟨CIGUE⟩⟩ from 1997 through 1999. Data collected included age, sex, type of incident, location of incident, route of entry, products involved, product presentation, signs, symptoms, paraclinic investigations, treatment, and outcome. Severity of clinical course was evaluated using Poison Severity Score calculated retrospectively after follow-up. Results: From 1997 to the end of 1999, nine cases concerning quaternary ammonium toxic exposure by the nasal route were collected out of 74,410 cases on the database. Eight were in males; all 9 cases were in very young children, aged from 15 days to 5 months. All were symptomatic and hospitalized. Symptoms were immediate in 7 cases

and delayed in 2 cases (<12 hours). Severity was low in 3, moderate in 1 and severe in 5. Symptomatology included: digestive pain (9 cases; all), hypersalivation (9 cases; all) and bradypnea (9 cases; all), cough (6 cases), vomiting (5 cases), skin pallor (2 cases), bradycardia (1 case), keratitis (1 case). Six infants needed endotracheal intubation for acute respiratory failure complicated in 4 cases by acute pulmonary edema. In all children, inflammation and edema were observed in the mucous membrane of the nasal cavity. Erosive pharyngitis and esophageal caustic burns proven by endoscopy was observed in all cases. No fever was noted. Treatment was symptomatic. All were hospitalized (duration from 2 days to 23 days). Outcome was favorable in 7 cases, two infants developed sequelae: nasal membrane damage and pulmonary segmental atelectasis. No esophageal stenosis was observed. Conclusions: These toxic exposures were very rare, but serious when they occurred. Their potential severity may be under-estimated. Nasal exposure to quaternary ammoniums requires intensive management and special care.

160 SEVERE VITAMIN D OVERDOSE IN TWO INFANTS

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Objective: This is a report of vitamin D overdose in two infants. Such overdoses are quite rare. Case Report: Two previously healthy 6-month-old twins presented to the Emergency Department because of sluggishness, drowsiness, hypotonia, constipation and decreasing weight. Their mother reported that she gave them 1 mL of a multivitamin product to drink twice daily. Later she gave to them 1 mL of a vitamin D solution for 17 days (the total dose was approximately 1.5 million units for each infant). After this period, symptoms developed. Most laboratory tests (K, Na, Mg, creatinine, pH, etc.) were normal except calcium and phosphate (see table; Ca is total calcium in mmol/L; Ca^{++} is ionized calcium in mmol/L; P is phosphate in mmol/L) and parathyroid hormone (<0.1 pmol/L).

	On the 1st day		After 10 days		After 20 days		After 27 days		After 2 months	
	1st infant	2nd infant	1st infant	2nd infant	1st infant	2nd infant	1st infant	2nd infant	1st infant	2nd infant
Ca	3.8	3.9	3.4	3.3	3.2	3.4	2.8	2.7	2.6	2.6
Ca^{++}	1.7	1.8	1.6	1.6	1.5	1.7	1.2	1.2	1.1	1.2
P	1.5	0.8	1.2	1.2	1.2	1.0	1.7	1.9	1.7	1.9

Polyuria (6–11 mL/kg/h) and calciuria (33.8 and 23 mg/kg/d) were observed in both patients during the period of treatment. Ultrasonography was performed and nephrocalcinosis was found in both cases. The treatment instituted was a diet with a decreased amount of calcium, infusion with calcitonin 4 units/kg, and prednisolone was administered to one child. The twins have been observed for six months, psychomotor development is normal, only a moderate polyuria and nephrocalcinosis (demonstrated by ultrasonography) remains. Conclusions: Inappropriate use of vitamin D caused severe complications including nephrocalcinosis in these two children. No benefit was noted from the prednisolone treatment.

161 A PROSPECTIVE EVALUATION OF HOUSEHOLD DETERGENT EXPOSURES IN CHILDREN

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Objective: Household detergent exposures occur mainly in the paediatric populations (61.3% according to our experience). Hand dishwashing solutions, laundry detergents and softeners are among the most common substances responsible for poisoning in the domestic environment (34% of consults to our centre). The aim of this work was to study the characteristic of childhood poisoning by these compounds. Methods: A prospective study over 12 months of paediatric exposures to hand dishwashing solutions, laundry detergents and softeners was designed. Information was recorded in a semistructured telephone questionnaire. Five hundred and twenty nine of exposures to hand dishwashing solutions, 60 to laundry detergents and 33 to softeners were analyzed. Patient age, symptoms, route, and circumstances of the exposures were recorded. Results: In 90.6% of the occasions our Poison Control Centre was consulted by private

persons. Ninety-six percent of the exposures were ingestions. Transfer from the original container occurred in 51.6% of the cases. However this was seen mainly in older children (72.8% of children older than 2 years ingested hand dishwashing liquid from a different container and that was only the case in 40.5% of younger children). 67.3% of the children who ingested these detergents remained asymptomatic at the time of the consult. The most frequent symptoms observed were: vomiting (18.4%), cough (6.3%), and diarrhoea (5.2%). One hour after the ingestion 98.3% of the children were asymptomatic. Six patients were vomiting and had abdominal pain, local burns or nausea. Diarrhoea was the only sign that lasted for more than 12 hour in one patient. No important respiratory consequences were reported. 98% of callers complied with the advice given. A contraindicated or unnecessary treatment was given in 12 occasions before or after the call to the PCC (10 emesis induction, 1 gastric lavage and 1 patient to whom laxatives were given). Conclusions: Transfer from the original dispenser was detected in an large number of cases probably due to the fact that this practice is common and containers are accessible to children. Although over 500 paediatric exposures occurred, morbidity was low and all the exposures had a good outcome. Considering the good compliance with the PCC therapy advice combined with the number of private persons who consulted the PCC, many unnecessary visits to an emergency department were be avoided.

162 EPIDEMIOLOGY OF ACUTE SEVERE PAEDIATRIC POISONING REPORTED TO A POISON CONTROL CENTRE

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Objective: Paediatric exposures to toxic substances are usually mild, although the Poisons Control Centre is frequently consulted for these exposures. The aim of this study was to determine the circumstances surrounding severe cases of poisoning in children in order to implement prevention efforts. Methods: All paediatric exposures, between January 1995 and June 2000 referred to our Poison Control Centre were recorded. Data including type of product, sex, age, route of exposure, aetiology, place of intoxication and clinical symptoms, were recorded for toxic exposures with a severe outcome. The possible outcome was estimated by the medical doctor based on data collected at the time of the call. A total of 1659 cases (55.5% male, 40.7% females and rest unknown) were analyzed. Self-referral calls were 41.9% and calls from a health care professional, 57.9%. Results: Main routes of exposure were as follows: oral 94.5%, respiratory 1.7%, and parenteral 0.8%. Intoxications occurred at home in 92.2% of the cases. Substances involved were mainly drugs (63.5%): acetaminophen (44.2%), antihistaminic agents (9.1%), bronchodilators (4%), antitussives (3.8%), salicylates (2.8%), fluorides (2.4%) and other drugs including neuropsychiatric and cardiovascular agents. We noted a significant number of methadone ingestions (12 cases). A significant group of patient was exposed to industrial products: professional cleaners (5.7%), pesticides and rodenticides (2.6%), and others (fuels, solvents, methanol, antifreeze agents, etc., 7.1%). Other toxic products were household detergents (5.7%), bleaches (2.2%) and ammonia (1.6%), plants and drugs of abuse (1.7% each). Seven percent were children less than 2 years of age whereas 64% were between 2–3 years. Three per cent were suicide attempts: 84% female. Eighty nine per cent of them ingested a drug. Iatrogenic exposures were described at hospitals mainly by parenteral administration of drugs. Inhalation of insecticides, solvents, bleaches or industrial products occurred at home in 72.4% of cases. Conclusions: The majority of severe poisonings occurred at home with medications normally found at home. On the other hand, industrial products still represented an important number of cases occurring in the household environment in spite of our recommendations. Particular attention is needed for suicide attempts in pediatric patients.

163 ACUTE POISONING WITH ACKEE FRUIT IN JAMAICA

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Objectives: Ackee Fruit (*Blighia Sapida*) is served in Jamaican traditional breakfasts. The ingestion of unripe ackee fruit has been identified as a source of severe disturbances known as "Jamaican vomiting sickness." It is mainly characterized by hypoglycaemia that has been attributed to hypoglycin A, a natural toxin present in unripe ackee fruit and its major metabolite methylenocyclopropyl acetyl-CoA, by inhibiting fatty acid oxidation. Incidence of ackee fruit poisoning in the Caribbean has been estimated to be 1:1000 persons per year, with a peak in the winter months. The aim of this study is to describe the epidemiology of ackee fruit poisoning in Jamaica. Methods: Retrospective collection

of clinical and biological data for all patients admitted from 1/98–12/99 to the emergency department of a hospital in an urban area of Jamaica, for disturbances associated with the ingestion of ackee fruit. Results: During 2 years, 12/220 patients admitted for an acute overdose were identified as cases of ackee fruit poisoning and included 7M/5F, median age 20 years [range 3–55]. Four of 12 cases were observed in the same family. Consumption of unripe ackee fruit was reported either at breakfast (7/12) or lunch (5/12). All cases occurred in the winter months. Patients were admitted 12 hours [12–48] after ingestion. On admission, the most common symptoms were severe vomiting (11/12), abdominal pain (9/12), weakness and paresthesia (8/12), tachycardia (4/12), headache (4/12), drowsiness (2/12), hypotonia (2/12), diarrhoea (2/12) and seizures (1/12). All patients had hypoglycaemia (3.9 mmol/L [1.1–4.4]). Biological findings showed acute renal failure due to severe dehydration in 4/12 patients and cholestatic liver disease in 1/12 patient. One patient died in the emergency department on arrival. Patients were managed with supportive treatment including oral (2/12) or intravenous (10/12) glucose administration, anti-ulcer (6/12), anti-spasmodic (4/12), anti-emetic (3/12) and corticosteroid therapy (2/12). The median length of stay was 1 day [0–9]. Conclusion: Ingestion of ackee fruit may be responsible for severe epidemic intoxication in Jamaica, with a large proportion of children. The management in the emergency department is primarily symptomatic.

164 SEVERE ADVERSE EFFECT ASSOCIATED WITH KAVA-KAVA

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Objectives: Abuse of herbal preparations may be in part due to the perception of its safety. Herbal preparations of kava kava (*Piper methysticum*), a remedy for insomnia, nervousness and depression are easily obtained through health food stores and herbal shops in our country. A case of severe adverse effects due to kava kava is described here. Case Report: In April 2000, a 45-year-old female with a clinical history of essential trembling in her family was seen by several psychiatrists because she complained of depression, insomnia, and anxiety; she was treated with anxiolytic and antidepressants agents. Neurological manifestations did not improve and the patient was admitted to a hospital neurology service. Bradykinesia, bilateral rigidity, and resting and postural tremor were observed. She was treated with levodopa and bromocriptine. Five months later she was transferred to another hospital where a severe rigid akinesia syndrome, resting and intentional tremor, akathisia and swallowing difficulty with significant weight loss were observed. The differential diagnosis was Creutzfeldt-Jackob syndrome, drug induced familiar parkinsonism or Wilson' disease. Pramipaxol was added to the previous treatment. The patient admitted to having taken *Hypericum perforatum* as a diet adjuvant for weight loss i.e. *Fucus vesiculosus* as well as minerals i.e. iron, zinc, magnesium, potassium, chloride, chromate and vitamin B complex. She had taken a cap of kava-kava for 10 days before the start of the symptoms. Our Centre received a call from the neurologist asking about kava-kava toxicity. This herb has antidopaminergic and anti-gabanergic actions, and causes extrapyramidal effects. As a result of this information, 5 mg IV of biperiden was administered to the patient and a second dose 1 hour later. This treatment was continued orally and clinical improvement on the third day was noted. A mild resting tremor still was present at the fourth day after admission in both arms and she was discharged with amantadine, levodopa and biperiden. High levels of homovanillic acid (150 ng/mL normal values: 30–40 ng/mL) were detected in cerebrospinal fluid analysis. Conclusions: Outstanding features of this case are the severe presentation and the long period of time before a correct diagnosis was made. When anticholinergic agents were administered, she improved dramatically. Adverse effects associated with these kind of herbal remedies are largely unknown in western medicine.

165 *RUTA GRAVEOLENS* AS AN ETHNIC ABORTIFACIENT

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Objective: For centuries, herbal remedies have been used as abortifacients. The use of specific agents tends to follow cultural lines. An abortifacient tea made from the leaves of common rue (*Ruta graveolens*) is a well-established remedy in Mexican culture. We report a case of toxicity in a healthy woman after consumption of such a tea. Case Report: A 26-year-old female of Mexican descent presented to the hospital complaining of abdominal pain and a sensation of ants crawling over her body. The previous day the patient had been informed that she was pregnant and received counseling regarding an elective termination of pregnancy. A relative cautioned her that the surgical procedure involved would be very painful and advised her to make a tea from chocolate, cinnamon and the leaves of a plant growing in her garden. After consuming the beverage, the patient developed abdominal cramps and yellow vaginal discharge. She

drank a second dose 5 hours later, vomited several times and developed formication. At this point she sought medical attention. Her physical examination was unremarkable and all standard laboratory tests were within normal limits, except for large ketonuria and a positive urine pregnancy test. A family member was sent home to retrieve the plant used, which was identified as common rue (*Ruta graveolens*). The treating physicians were also concerned about quinine and sent serum for a quinine assay, which was negative. After two days, her symptoms resolved without any complications. The patient was discharged and sent for pregnancy counseling. Conclusion: The most common adverse affect of rue is a furanocoumarin-induced photodermatitis. Psoralens present in the plant can act as a vesicant and may induce vomiting. However, animal studies have suggested that certain chemicals present in the plant (chalepensin and quinoline) can prevent implantation or increase muscle activity (i.e. abdominal cramps). Either of these mechanisms can induce an abortion. The list of herbal abortifacients and emmenagogues is too extensive for clinicians to remember. Awareness of specific ethnic remedies can assist in the management of patients poisoned by such remedies.

166 LIFE-THREATENING VAGINAL HEMORRHAGE CAUSED BY THERAPEUTIC CHINESE GINSENG DOUCHE

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Objective: Ginseng (*Panax spp.*) has been used for centuries as a natural remedy for numerous varied ailments. We report a life-threatening vaginal hemorrhage that resulted from the use of therapeutic Chinese ginseng douche. Case Report: A 38-year-old nulliparous, virginal, Asian woman presented to the emergency department after vaginal bleeding for 30 minutes. Her past medical history was significant only for a vaginal yeast infection 6 months earlier for which her gynecologist prescribed a douche of Chinese ginseng extract. She used this douche every three days for the previous six months, despite the fact that her yeast infection had resolved. She denied any sexual activity ever, and denied vaginal instrumentation other than use of the douche, which was inserted with a standard soft, flexible tapered plastic tip on a bottle. She reported her last menstrual period 5 weeks earlier. Her vital signs were: heart rate 100/minute, blood pressure 150/100 mmHg, respirations 16/minute, and she was afebrile. Her physical examination was normal excepting for skin pallor and an actively hemorrhaging erosion of the vaginal mucosa. The cervical os was closed and was not bleeding. Initial laboratory investigations were: white blood count 7.8 $\times$ 10^9/L, hemoglobin (Hb) 13.3 g/dL, hematocrit (Hct) 39.6%, platelet count 288 $\times$ 10^9/L, prothrombin time (PT) 12 seconds, partial thromboplastin time (PTT) 30.1 seconds, INR 1.07, and a negative serum pregnancy test. Her blood pressure decreased to 80/40 mmHg and a repeat Hb was 7.5 g/dL. The patient received 2 liters of crystalloid and 500 mL of packed red blood cells. After the blood transfusion, her Hb increased to Hb 11 g/dL. The vaginal hemorrhage ceased after conservative treatment with intravaginal packing, and the patient was discharged the following day with instructions to avoid further use of ginseng douche. The patient suffered no further gynecologic problem after discontinuation of the douche. Conclusions: For centuries, Eastern and Asian cultures have used herbal remedies such as ginseng for numerous ailments. Traditional Western medicine is increasingly accepting and engaging in the use of ginseng and other natural therapies. Ginseng douche has been advocated as a treatment for vaginal yeast infections and cervical dysplasia. Unfortunately, the safety, efficacy, and side-effects of ginseng and many other potentially effective herbal therapies has not been well-studied. This patient suffered a life-threatening vaginal hemorrhage as a consequence of using therapeutic Chinese ginseng extract douche to treat and prevent vaginal yeast infection.

167 INTRAVAGINAL ALUM RESULTING IN MUCOSAL EROSIONS

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Background: Alum is any group of chemical compounds made up of water molecules and two kinds of salts, one of which is typically aluminum sulfate ($Al_2(SO_4)_3$). Therapeutically, alum is typically used for its astringent properties. We describe a pediatric patient who performed an ethnic healing practice improperly and subsequently sustained severe vaginal injury. Case Report: A 17-year-old nulliparous Haitian female presented to the emergency department complaining of severe vaginal pain. Two days prior, the patient had inserted alum "crystals" to increase her vaginal tone. Her motivation for using intravaginal alum stemmed from a cultural practice of Haitian women, who use alum postpartum as a means to increase vaginal tone for enhanced sexual pleasure. The patient did not understand that this practice typically involves use of a dilute douche and instead inserted solid crystals. On presentation, the patient had normal

vital signs. Intravenous conscious sedation was necessary in order to conduct an adequate pelvic examination, which was significant for vulvar edema and gray-yellow charring of the vaginal mucosa with areas of ulceration. There were no signs of vaginal perforation and there was no active bleeding. The remainder of the physical examination was normal. Laboratory investigations revealed a normal PT/PTT, normal CBC and a negative pregnancy test. Initial treatment consisted of vaginal irrigation with normal saline and administration of analgesics. The patient was observed for 24 hours and discharged. The crystals were identified using atomic absorption spectroscopy for aluminum sodium and barium sulfate precipitation and turbidometric analysis for the sulfate ion. Percentage composition determined after analysis was consistent with the theoretical composition of $AlK(SO_4)_2 \cdot 12\ H_20$ or aluminum potassium sulfate, otherwise known as alum. Conclusion: Intravesicular alum instillation is a common treatment for hemorrhagic cystitis. Aluminum sulfate is also used as an antiperspirant for its astringent properties. Rare complications associated with various different uses for alum include fistulas, encephalopathy and death. In certain ethnic groups, the alum douche is used in conjunction with sitz baths after postpartum to enhance healing in the perineal area. This case describes an ethnic practice that can cause severe injury. This practice should be recognized by physicians living in the multi-cultural urban environment because it may aid in diagnosis and management for an unusual cause of vaginal pain.

168 PAROXYSMAL SYMPATHETIC STORM (DIENCEPHALIC SEIZURES) FOLLOWING METHADONE OVERDOSE

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Objective: We report a case unique to the literature involving development of diencephalic seizures or sudden diencephalic crisis following a methadone overdose. Case Report: A 17-year-old male ingested unknown amounts of his mother's methadone. The patient was last seen at 0100 hours in his normal state of health. He was found unresponsive in his bed approximately 12 hours later. EMS was activated and upon arrival the patient was found to have pinpoint pupils and a GCS of 3. Two milligrams of Narcan was administered without effect. On arrival to the hospital, he was noted to be hypotensive, tachycardic, and tachypneic. Oxygen saturation was 84% on 100% oxygen by non-rebreather mask and temperature was 37.5 °C rectally. The patient was endotracheally intubated and given activated charcoal by nasogastric tube. Following initial stabilization, the patient again developed hypotension with a systolic blood pressure of 50 mm Hg. He required intravenous fluids and dopamine to maintain a systolic blood pressure $>$ 100 mm Hg. Serum potassium was 7.3 mEq/L, rapid urine toxicology screen was positive only for THC. Electrocardiogram revealed peaked T-waves diffusely. A right lower lobe infiltrate consistent with aspiration pneumonia was on chest x-ray. The head CT scan was unremarkable. The patient was transferred to the medical intensive care unit where he was gradually tapered off the dopamine. The following day, the patient was noted to be profusely diaphoretic and exhibited pupillary dilation, decerebrate posturing, and transient hypertension upon painful stimulation. Emergent EEG showed diffuse theta and delta wave slowing but no focal findings or epileptic activity. The comprehensive toxicology screen reported a large quantity of methadone in the serum. Repeat head CT scan revealed diffuse cerebral edema secondary to anoxic injury. Intravenous mannitol was administered. Neurology Service was consulted and diagnosed diencephalic crisis. Morphine and bromocriptine treatment was recommended. The patient was gradually weaned off the morphine, but bromocriptine taper was unsuccessful. With continued improvement, the patient was admitted to the Traumatic Brain Injury Program. After approximately 3 months post-ingestion, the patient is able to walk, talk, and independently manage activities of daily living. Conclusion: Diencephalic seizures have been reported with tumors, hydrocephalus, and closed head injuries. The syndrome may incorporate symptoms of diaphoresis, vomiting, increased salivation, and extensor posturing. Following methadone overdose with resultant anoxic brain injury, our patient exhibited symptoms consistent with diencephalic seizures reported in the literature. Of note, the patient did not demonstrate any clinical or diagnostic findings of epileptiform activity. Symptoms of sweating, tachycardia, and posturing were well controlled with use of morphine and bromocriptine.

169 EPIDEMIOLOGICAL PROFILE OF EMERGENCY CASES OF DRUG ABUSE

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Objective: Although the consumption of abusive substances is almost as old as humanity itself, in the last 25 years new drugs of abuse have appeared. This has modified the type of overdose patient treated. We present the results of

a study carried out in our hospital with the aim of profiling this type of patient. Methods: Records of patients attended in the Emergency Department of our hospital for a period of three months (March–May 2000) were reviewed and patients presenting because of drug overdose, including alcohol, were identified. The variables studied were age, sex, drug type, target organ, treatment given and evolution. The results were analyzed with the SPSS statistical program. Results: During the period studied, 36.712 patients attended the Emergency Department of which 151 (0.4%) were for overdose of drugs of abuse. Two-thirds (98 cases) were alcohol-only intoxications, and the remaining third (53 cases) overdoses of other drugs: cocaine (49%), amphetamines or ecstasy (21%), cannabis (19%) and heroin (17%); cases of LSD, liquid ecstasy, ketamine and trichloroethylene were also detected. The patients who presented after taking drugs of abuse were younger than those presenting after alcohol overdose (27 years vs 37 years). These patients (drug of abuse group) also needed greater therapeutic intervention (use of antidotes, pharmacological sedation or physical restraints), a longer stay in the Emergency observation unit and, on occasion (4 cases), admission to Reanimation or Intensive Care Units. The presence of patients who had ingested liquid ecstasy (gamma-hydroxybutyrate) or "Special-K" (ketamine) was also notable, confirming the constant changes in abusive substances in our society. Mortality was 0% for both groups. Conclusion: Overdoses of drugs of abuse continue to be a daily problem in the Emergency Department. Patients seen as a result of drugs such as cocaine, amphetamines or heroin are younger and more often male than those seen as a result of alcohol abuse. Morbidity associated to alcohol abuse is less than with other drugs of abuse, requiring the use of less resources and with a shorter stay in the Emergency Department. In both groups the prognosis is good.

170 A CASE OF ATRIAL FIBRILLATION ASSOCIATED WITH GHB INGESTION

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Background: GHB use has been on the rise with the number of ED visits more than quadrupling between 1994 and 1998. Although many of its CNS effects have been well characterized, it is generally accepted that there is minimal cardiotoxicity. In a study using human volunteers, GHB administered at anesthetic doses demonstrated a mild reduction in heart rate and blood pressure but no significant cardiac events. A pediatric patient that accidentally ingested GHB presented with a right bundle branch pattern with ST elevation in leads V2 through V5 that resolved approximately 6 hours after ingestion. Animal studies have demonstrated that GHB effects repolarization through its action on potassium channels. In a clinical setting this could lead to ECG disturbances and/or dysrhythmias. Case Report: A 25-year-old white male was noted to be ataxic and confused at a dance club. He subsequently became unresponsive and was transported by EMS. Upon arrival to the ED, he was more arousable and demonstrated a mild akathisia. The patient was slightly tachycardic (heart rate 100–120) and demonstrated atrial fibrillation on ECG. He admitted to taking GHB but no other drugs or alcohol. The atrial fibrillation resolved spontaneously after approximately 30 minutes to a normal sinus rhythm. The patient did have a sporadic history of anabolic steroid abuse but had not used any in over 3 months. Drug screen and alcohol level were negative. Urinalysis, electrolytes, CBC, and LFTs were all normal except for a creatinine of 1.6 mg/dL and a slightly elevated TSH (13.1 mIU/L) but a normal free T4 (1.10 ng/dL). He had negative serial cardiac enzymes and an echocardiogram that was normal. He remained in a normal sinus rhythm until his discharge 24 hours later. Conclusions: With the increasing use of GHB, physicians should be aware of the potential cardiotoxicity associated with its abuse.

171 IMPAIRED IMMUNITY CAUSED BY DRUG ABUSE AND ACUTE POISONING BY NARCOTICS AND PSYCHOACTIVE DRUGS

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Objectives: To identify the features of impaired immunity associated with drug abuse and acute poisonings by narcotic and psychoactive drugs. Methods: 20 drug abusers, 57 patients with narcotic drug poisoning, and 45 patients with psychoactive drug poisoning were studied. We determined the blood leukocyte and lymphocyte, and absolute and relative T- and B-lymphocytes count in blood. The levels of immunoglobulins A, M, and G were determined as was phagocytosis status using latex and NST-tests, and the level of circulating immune complexes. Results: Drug abusers had 1.3 times higher leukocytes and 40% lower total lymphocytes compared to normal values. The relative T-lymphocyte count was moderately increased in combination with a relative B-lymphocytopenia. The levels of A, M, and G immunoglobulins were increased by 15.9% to 35.8%. Oxygen-dependent metabolism of phagocytic neutrophils was increased. As a nearly threefold increase of circulating immune complexes was observed. Acute poisoning by narcotics was accompanied by

an increase of leukocytes and severe lymphocytopenia. The relative and absolute T-lymphocytes were reduced in combination with severe B-lymphocytopenia. The levels of A, M and G immunoglobulins were decreased. The metabolic activity of neutrophils was considerably lowered. The patients with acute psychoactive drug poisoning also had leukocytosis and severe lymphocytopenia. The absolute and relative number of T- and B-lymphocytes were lowered in combination and there was a decrease of the A, M, and G immunoglobulins. Although the activation of oxygen-dependent metabolism of phagocytic neutrophils was 23% above normal, this was considerably lower than observed in patients with acute and chronic narcotic drugs intoxication. Total circulating immune complexes was 78.6% above normal. Conclusion: Impaired cell and humoral immunity, and phagocytic activity of neutrophils associated with chronic drug abuse appears to be due to effects of the narcotic drugs on various immune response parameters. Immune status change during acute poisoning caused by narcotic and psychoactive drugs resemble those seen with physiological stress.

172 A STUDY OF THE PREVALENCE OF ILLICIT DRUG USE IN PATIENTS PRESENTING WITH DELIBERATE SELF-POISONING IN A BELFAST HOSPITAL

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Objective: Illicit drug use is common in our society. Recent surveys suggest a lifetime prevalence of drug use for 16–59 year olds in the UK at 24–30%. Deliberate self-poisoning is also a major public health problem. Socio-economic deprivation is associated with both behaviours. This study aims to determine the prevalence of substance abuse in patients presenting with deliberate self-poisoning in the Belfast area. Methods: The study consisted of an anonymous questionnaire. A sample of patients in the age range 15–45 years presenting with deliberate self-poisoning was identified and informed consent was obtained. Details recorded included age, sex, drugs taken in overdose and recreational drug use. If illicit drugs were used the types of drugs were recorded as was the recency of use in the past week, month, year or longer ago. In addition, a 20 mL urine specimen was taken as soon as possible after admission to screen for drugs of abuse (amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine metabolites, opiates, LSD and methadone). Results: Forty-nine patients admitted to hospital following deliberate self-poisoning were interviewed. The pattern of drugs taken in overdose was similar to other recent studies with paracetamol being the most commonly ingested drug (40.8%). Alcohol was involved in 30.6% of overdoses. Twenty-five out of the forty-nine patients (55.1%) had used drugs at some time in their lives. 14.3% of individuals had used drugs within the week prior to admission and a further 6.1% within the preceding month. Cannabis was the most commonly used drug having been tried by 51% of subjects. Ecstasy had been used by 26.5% of subjects, 10.2% having used the drug within the week or month or month preceding admission. Only 34 urine samples were received by the laboratory. Positive results for benzodiazepines, alcohol and codeine were in keeping with drugs taken in overdose Conclusions: Illicit drug use is more common in patients presenting with deliberate self-poisoning than in the general population. Patients were happy to volunteer their drug history and urine screening did not yield any useful additional information.

173 BRADYCARDIA, HYPOTENSION, AND TINNITUS AFTER ACCIDENTAL OXCARBAZEPINE OVERDOSE

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Objective: Oxcarbazepine (Trileptal) is a new antiepileptic drug derived from carbamazepine. It is approved for use in the United States for treating partial seizures and trigeminal neuralgia. It is reported to be better tolerated and have fewer adverse effects than carbamazepine. Adverse cardiac effects and tinnitus have not previously been reported with ingestion of oxcarbazepine. We report the first case of bradycardia, hypotension, and tinnitus after overdose of oxcarbazepine. Case Report: A 38-year-old female with a history of simple partial seizures presented to the emergency department four hours after accidental oxcarbazepine overdose. The patient's normal dose was 2100 mg/day. Due to a prescription refill error, the patient consumed 3300 mg of oxcarbazepine that day (900 mg that morning and 2400 mg that evening). The recommended maximum daily dose is 2400 mg. Two hours after the evening ingestion, the patient developed diplopia, somnolence and fell asleep. She awoke 90 minutes later, with burning of her throat, vertigo, tinnitus, and was noted to be lethargic. In the waiting room of the hospital, she had a brief witnessed partial seizure and was brought into the treatment area of the emergency department. Her initial vital signs were: blood pressure 142/84 mm Hg, heart rate 60 beats per minute (bpm), respiratory rate 15 breaths per minute, temperature 37 degrees C and pulse oximetry 95% on room air. Glasgow Coma Scale was 13 and she was oriented. Ten minutes after her initial assessment,

she was described as more lethargic, with a systolic blood pressure of 60 mm Hg by palpation and a heart rate of 27 bpm. Administration of IV fluids and atropine 1 mg increased her heart rate to 108 bpm and her blood pressure to 130/78 mm Hg. The patient was more alert and had no residual ECG abnormalities. A 6 hour post-ingestion serum oxcarbazepine level (10-hydroxy metabolite) was 45.6 μg/mL (therapeutic range 10–35 μg/mL). A comprehensive urine toxicologic screen was negative except for "carbamazepine and metabolites," however her serum carbamazepine level was non-detectable. The patient was taking no other seizure medications, had no previous cardiac history or adverse events with oxcarbazepine. The patient was discharged symptom free from the hospital within 24 hours without further episodes of bradycardia or hypotension. Conclusion: This is the first report of adverse cardiac effects and tinnitus with oxcarbazepine. The manufacturer notes oxcarbazepine has a narrow therapeutic index in regards to adverse CNS effects. This case demonstrates oxcarbazepine doses greater than 2400 mg/day, may cause such adverse effects as bradycardia, hypotension and tinnitus.

174 TRANSESOPHAGEAL CARDIAC PACING IN PATIENTS POISONED WITH CARDIOTOXIC AGENTS

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Objectives: Cardiotoxic agents cause about 10% of poisonings. If normal electrocardiography (ECG) doesn't give a complete insight in the cardiac conduction disturbance, it is relevant to evaluate new diagnostic procedures. Since 1989 transesophageal ECG and electrocardiostimulation were carried out at the Regional Toxicology Centre in the patients with acute cardiotoxic agents poisonings, on admission and during their treatment. Non-invasive transesophageal pacing causes fewer iatrogenic accidents than the transvenous one. Transesophageal electrocardiostimulation is possible in cases of severe bradycardias unresponsive to atropine. Moreover, it may be a method of choice for the treatment of atrial and AV nodal paroxysmal tachycardia. Methods: We examined 173 patients with acute poisoning, 70 of them poisoned with clonidine, 36 veratrum alkaloids, 42 amitriptyline, 8 digitalis, 9 propranolol and 8 verapamil. When admitted to the centre all patients underwent an ECG, transesophageal ECG, and electrical stimulation. The sinus node recovery time, corrected sinus node recovery time, sinoatrial conduction time, interval between stimulus and ventricular response, the Wenckebach threshold, and the effective refractory period were determined. Results: A toxin-induced depression of the sinus node function was observed in 42.7% of patients with clonidine poisoning, 19.4% of patients with veratrum alkaloids poisoning, and 50% of patients with digitalis poisoning. 2.2% of patients with amitriptyline poisoning had a marked depression of sinus node activity with periods of sinus arrest. A sinoatrial entry block was observed in 41.3% of patients with clonidine poisoning, and in 50% with digitalis and verapamil poisonings. A decrease in AV conduction was observed in 70.7% of patients with clonidine poisoning, 20.5% of patients with veratrum alkaloids poisoning, and 50% of patients with digitalis poisoning and in every patient with propranolol poisoning. Most patients with amitriptyline poisoning had an increase in the AV conduction and a reduction of the effective refractory period. Urgent transesophageal cardiac pacing was carried out in 1.7% of patients. Conclusion: Application of transesophageal cardiac pacing allows for a quick and safe determination of cardiac pacemaker and conduction abnormalities, which are not seen on surface ECG. Prophylactic placement of a transesophageal electrode allows for urgent cardiac pacing, if necessary.

175 LITHIUM POISONING CAUSING A SEVERE BRADYCARDIA

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Background: Although lithium salts are commonly used and gastrointestinal and CNS toxicity well recognized, cardiac toxicity is rarely reported. Case Report: A 72-year-old female with bipolar disorder presented with increasing lethargy, ataxia, fever, anorexia, and cough. She had no history of cardiac problems. The patient was only taking lithium which was administered by family. There was no change in her lithium dose for the past 2 years. Her vital signs included a rectal temperature 101°F, RR 25/min, HR 42 bpm, BP 117/40 mmHg, and O_2Sat 80%. Her physical examination revealed coarse breath sounds bilaterally, bradycardia without murmurs or rubs. Her neurologic examination was significant for lethargy, hyperactive deep tendon reflexes, hypertonic muscle tone, and clonus. Her electrocardiogram showed a junctional bradycardia at a rate of 39 bpm, QRS duration of 100ms, and no acute ST-T changes. The patient received mechanical ventilation and a transvenous pacemaker was placed. She did not respond to atropine or transcutane-

ous pacing. The laboratory data was unremarkable except for a lithium level of 4.1 mEq/L. Serum digoxin and drugs of abuse urine screen were also negative. Shortly after dialysis, she developed tachycardia. Blood and cerebral spinal cultures ultimately were negative. During a second dialysis for rebound to 2.28 mEq/L, the patient became hypotensive, developed ventricular fibrillation, and could not be resuscitated. Conclusion: Lithium toxicity typically occurs in the setting of dehydration and chronic use. This patient's heart rate was blunted with the elevated lithium concentration and responded appropriately after dialysis. Although neurologic signs predominate, cardiotoxicity may occur and should be routinely assessed in all patients taking lithium.

176 TOXICITY OF PARATHION, CHOLINESTERASE STATUS AND NEUROMUSCULAR FUNCTION DURING ANTIDOTAL THERAPY IN A FATAL CASE OF PARATHION POISONING

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Objective: Lethality in severe parathion poisoning is still high. Due to the persistence of this lipophilic poison, there may be a prolonged cholinergic syndrome requiring oxime treatment for a longer period of time than that usually recommended. To evaluate the effectiveness of oxime therapy, we monitored the cholinesterase status, neuromuscular function, and parathion toxicokinetics in a fatal case of intoxication with Folidol-Öl®. Methods: Erythrocyte acetylcholinesterase (Ery-AChE) and plasma cholinesterase were measured with a modified Ellman procedure. Potential reactivatability of Ery-AChE was assessed *ex vivo* by incubating diluted blood with obidoxime. Parathion, paraoxon and obidoxime in plasma were determined by HPLC. Atropine was assayed by a radioreceptor assay. Neuromuscular function was estimated by stimulation of the ulnar nerve and recording the compound action potential of the abductor dig. min. muscle. Obidoxime was given as an IV bolus (250 mg) followed by continuous infusion at 750 mg/24 h. Atropine was administered as required. Case Report and Results: A 49-year-old male having ingested 250 mL of Folidol-Öl® (25 g parathion), received emergent care and was treated in the ICU according to above regimen. Blood concentration of parathion and paraoxon peaked at 12 and 1 mg/L, respectively. Ery-AChE and plasma cholinesterase were undetectable and neuromuscular function (NMF) was impaired. After obidoxime administration Ery-AChE increased marginally and NMF remained abnormal. When paraoxon concentration decreased to 0.3 mg/L, Ery-AChE increased to approximately 15% of normal and NMF improved. However, during this period plasma parathion increased from 1 to approximately 3 mg/L, coinciding with severe impairment of cardiovascular function, and reduced P_aO_2, hepatic and renal function. Coincidental with the improvement in his multiorgan failure, parathion levels fell and paraoxon increased, resulting in inhibited Ery-AChE and NMF. Despite maintenance of approximately 20 µM obidoxime in plasma, reactivatability gradually disappeared due to aging of the inhibited fraction of AChE. After 10 days, obidoxime infusion was discontinued because reactivation was no longer possible. The patient's clinical condition gradually worsened due to pulmonary insufficiency and he died of oil pneumonia after 16 days. Post-mortem-analysis of the fat tissue revealed 6.5 mg parathion/kg. Conclusions: In this case of mega-dose-poisoning parathion and paraoxon concentrations remained unusually high until death, with paraoxonase activity remaining quite low. Hence, obidoxime was unable to counteract the rapid re-inhibition of AChE. Ironically, transient reactivation of Ery-AChE with significant improvement of NMF was found when the liver function was dramatically impaired and toxication of parathion to paraoxon was reduced. The patient's fatal outcome, however, was due to the early aspiration of the ingested solvent, containing 50% mineral oil, resulting in irreversible lung damage.

177 HERBAL INFUSIONS USED FOR INDUCED ABORTION

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Summary: The circumstances surrounding an induced abortion involve complex legal, ethical and social aspects. The patient usually denies the ingestion of any abortive substances. Very little scientific development has taken place in the area of toxic plants and most of the studies conducted are experimental. The purpose of this report is to enhance knowledge on this issue and to help achieve the correct diagnosis, treatment and prevention of the toxic effects of herbal infusions. To this end, we are relying on information and experience acquired at the Toxicological Information and Advisory Centre of Montevideo. Methods: A descriptive retrospective survey was conducted on the consultations

received at the Centre between 1986 and 1999 due to ingestion of herbal infusions with abortive intent. Results: A total of 86 consultations, involving 30 different plant species were identified. The species most frequently involved were Ruda (*Ruta chalepensis/graveolens*), Cola de Quirquincho (*Lycopodium saururus*), Parsley (*Petroselinum hortense*) and the four species included in the herbal store product Carachipita®: Póleo (*Mentha pulegium*), Yerba de la Perdiz (*Margiricarpus pinnatus*), Oregano (*Origanum vulgare*) and Guaycurú (*Statice brasiliensis*). Abortion occurred in 23 cases after the ingestion of the following plants: Parsley, Ruda, the herbal product Carachapita®, Celery, Cedron, Francisco Alvarez, Floripon, Espina Colorada. Out of the 23 cases, 11 involved the sole ingestion of plants. The remainder of cases involved instrumental manipulations; 4 cases associated Carachapita®, 4 cases added injected drugs (presumably hormones) and 4 cases included instrumental manipulations. Multi-systemic failure was found in those patients that had taken Ruda only, Ruda together with Parsley or with Fennel. Other plants causing multi-systemic failure were Carachapita®, Arnica and Bardana. Death took place in one case of ingestion of Carachapita® and four cases of ingestion of Ruda (in two cases Ruda alone and in the other two Ruda in association with Parsley and Fennel). Instrumental manipulations were confirmed in only 4 of the patients with multisystemic failure and in only one of those who died. Conclusions: The results of this report are not conclusive, but can guide further research on each individual plant species. Nevertheless, it can be concluded that the ingestion of plants to induce abortion involves the risk of severe intoxication that could result in death of the patient or result in future reproductive complications.