Central New York Regional Poison Control Center

The CNYPCC

Toxicology Letter

January, 2002

Vol. VII No.1

A Quarterly Publication

SCHEDULED EVENTS:

Date and times for Grand Rounds
January 9, 2001
February 13, 2001
March 13, 2001

Toxicology Case Conference
CNYPCC, 550 E Genesee Street
Poison Center Conference Room
Every Thursday 1:30 PM – 2:30 PM

PROGRAM ANNOUNCEMENT:

Thanks for participation in our Fifth Annual Toxicology Teaching Day. Our Sixth Annual Toxicology Teaching Day will be held on October 30, 2002 at the University Sheraton. Preliminary information will be available in the April issue of this newsletter.

CNYPCC TIDBITS:

Bio/Chem Weapons – Match

A. Phosgene	1. Can be mistaken for spider bite
B. Sarin	2. Pulmonary edema
C. Cyanide	3. Wet and paralyzed
D. Anthrax	4. Thiosulfate for treatment

TOX TRIVIA:

1. What does “p450” stand for?
2. Why does aspirin cause a fever in overdose?
3. What unintentional toxin exposure is most lethal each year?

Case History

Contributed by: Richard M. Cantor, MD, FACEP

DIFFERENTIATING POISONING VERSUS MEDICAL PROBLEMS

The toxic overdose patient may present with a variety of clinical symptoms. In many cases, during the initial emergency assessment and management, the offending agent is unknown.

How valuable is the history and the physical examination?

Unfortunately, the patient’s recollection may be unreliable. It is important for the clinician to utilize all resources available at the time of presentation, including family and friends, paramedics, a private physician who may be familiar with the patient’s medical condition.

In the emergency setting, a through physical examination is often overshadowed by efforts focused on patient stabilization. However, even a directed examination can yield important diagnostic clues. The following cases illustrate unusual chief complaints involving patients of all ages.

WIDE EYED AND BUSHY TAILED

A 3 year old child is rushed to your emergency department by his parents who claim that he suffered a generalized 2 minute seizure at home. No fever or intercurrent illness is described. Past medical history is negative and there is no family history of seizures. Physical examination includes vital signs: T 39C, HR 140, RR 20, BP 140/95, the patient is combative with dilated pupils. Otherwise the physical examination is significant only for hyperactive bowel sounds and diaphoresis.

What are some of the toxins that cause DILATED PUPILS?

A.A.A.S

A	Antihistamines
A	Antidepressants
A	Anticholinergics, Atropine
S	Sympathomimetics

What are some of the toxins that cause SEIZURES?

O.T.I.S C.A.M.P.B.E.L.L

O	Organophosphates
T	Tricyclic Antidepressants
I	INH, Insulin
S	Sympathomimetics

Continued on Page 2

DIFFERENTIATING POISONING VERSUS MEDICAL PROBLEMS

Continued from page 1

C Camphor, Cocaine
A Amphetamines
M Methylxanthines
P Phencyclidine (PCP)
B Benzodiazepine Withdrawal, Botanicals
E Ethanol Withdrawal
L Lead, Lithium
L Lidocaine, Lindane

It is important here to distinguish the classic features shared by the anticholinergic and sympathomimetic toxidromes. Common to both presentations includes hyperthermia, tachycardia, hypertension and mydriasis. Sympathomimetics are distinguished by the presence of diaphoresis and active bowel sounds.

What are the commons forms of stimulant exposure or abuse?

Pertinent toxins include cocaine, amphetamines, weight loss products, over the counter analeptics, and in some cases, bootleg products.

Cocaine, when utilized by the general public, comes in many forms. The pediatric patient may be exposed to cocaine by placental transfer, breast milk contamination, or passive exposure. The mechanism of action of cocaine involves central nervous system stimulation and inhibition of catecholamine uptake. Presenting symptoms and signs may include severe hypertension, tachycardia, fatal arrhythmias and seizures.

Amphetamines enjoy a long history within our culture. The older formulations included diet pills which have now been replaced by abusers with methamphetamine (speed, ice) and MDMA (ecstasy).

Phenylpropanolamine (PPA), although removed from standard distribution, remains within medicine chests nationwide. It is a noncatecholamine, sympathetic amine which acts as an indirect alpha stimulant.

Caffeine is our most widely used psychoactive substance. It is an anorexiant, co-analgesic, a diuretic, and sleep suppressant.

What was the emergency department outcome of our patient?

Laboratory studies, CT and a spinal tap in this child were normal. Urine toxicology was positive for metabolites of cocaine. After interviewing the parents they admitted to liberal use of cocaine in the home resulting in secondary exposure to cocaine.

YOU'RE NEVER ALONE WHEN YOU'RE SCHIZOPHRENIC

A 15 year old girl arrives with her parents who claim that ever since she woke up this morning she has behaved as if she was "possessed!" She is presently recovering from an active case of chicken pox and had been doing well. She reports no fever, stiff neck, nausea, vomiting, or new rash. Physical examination included vital signs: T 40C, HR 140, RR 20, BP 140/95. The patient was combative, with dilated, reactive pupils. Her pharynx was dry and she exhibited a distended, suprapubic mass, with absent bowel sounds..

What is the problem list concerning this patient?

This adolescent presents with fever and altered mental status and pupillary dilation. In addition, she demonstrates signs of encephalopathy involving posturing and hyperreflexia.

What is some of the toxins that cause hyperthermia?

N.A.S.A

N Neuroleptic Malignant Syndrome, Nicotine
A Antihistamines
S Salicylates, Sympathomimetics
A Anticholinergics, Antidepressants

What investigations should be performed in the emergency department?

Workup of this patient's encephalopathy included a CBC, electrolytes, liver function tests, and a serum pregnancy test . All were normal. CT scanning was unremarkable. A spinal tap failed to reveal any abnormalities.

Could this patient represent a toxidrome?

Careful review of the physical findings in this patient reveals the presence of the classic anticholinergic syndrome: warm, dry, flushed skin, dry mouth, dilated pupils, tachycardia, ileus, and delirium.

What are the most common drugs presenting with anticholinergic effects?

A complete list of anticholinergic medications would be quite lengthy. The most common drugs involved with this toxidrome include antihistamines, antipsychotics, antispasmodics, muscle relaxants, and tricyclic antidepressants.

What was the resolution of this case?

A careful review of the history and physical findings in this child revealed that the parents had been administering topical antihistamine lotions to the pruritic lesions involved with chicken pox. The active drug within this compound was Benadryl® (diphenhydramine).

What treatment is indicated in this case?

Most cases of anticholinergic syndrome need only supportive measures. Physostigmine, a centrally active anticholinesterase inhibitor, has been administered in patients who present with coma, delirium, or unstable vital signs. Side effects are many and this drug should

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Continued from page 2

be reserved for single toxin exposures after consultation with a qualified toxicologist.

HEAVY BREATHING

A 3 year old presents with a three day history of fever, progressive sleepiness, and respiratory distress. Previously healthy, his parents report that he developed a fever (101) at first, for which they gave Tylenol. His behavior has become that of a lethargic and irritable child, with a "funny, fast breathing pattern". Physical examination includes vital signs: T 40C, HR 140, RR 70 and deep, BP 140/70. The patient is sleepy and the remainder of the physical examination is non-contributory. The patient exudes a "medicinal smell".

What is the initial problem list involving this patient?

This patient presents with fever, lethargy, and hyperpnea. It is important to distinguish patients who are tachypneic from those who are truly hyperpneic.

What are some of the toxins that cause hyperventilation?

P.A.N.T.

P	PCP, Pneumonitis (Chemical)
A	ASA (Salicylates)
N	Noncardiogenic Pulmonary Edema
T	Toxic Metabolic Acidosis

A review of this patient's primary survey revealed that airway, breathing, and circulation was not impaired. A standard finger stick glucose was 100 mg/dL. The initial differential diagnosis included sepsis, pneumonia, or meningitis. A careful secondary survey was performed which failed to reveal any pupillary or specific neurologic abnormalities. The family denied any significant past medical history, medication, or allergies.

What investigations were performed in the emergency department?

A complete blood count was normal. A review of the electrolytes revealed an anion gap of 30. Arterial blood gases performed in room air demonstrated a pH of 7.50, pC02 =20, pO2 =100, BE= –15. Chest radiographs were normal.

What are some of the toxins that cause anion gap acidosis?

M.U.D.P.I.L.E.S.

M	Methanol
U	Uremia
D	DKA
P	Phenformin, Paraldehyde
I	Iron, INH
L	Lactate
E	Ethanol, Ethylene Glycol
S	Salicylates

Further laboratory investigations revealed a normal serum osmolarity, a negative ethanol level, and an elevated salicylate level (66 mg/dL).

What are the characteristics of salicylate toxicity?

The classic toxic presentation of salicylate exposure is fascinating. Initially the patient presents with hyperpnea secondary to central stimulation of the respiratory center. A respiratory alkalosis will develop in most cases. Salicylates then act as cellular poisons, uncoupling oxidative phosphorylation and interrupting glucose metabolism. A profound acidosis will also develop. As the patient's clinical status deteriorates, the central respiratory alkalosis dissipates and the patient becomes profoundly acidotic.

What are the clinical manifestations of salicylate exposure?

Patients present with vomiting, hyperpnea, tinnitus, and lethargy. In severe intoxications, coma, seizures, hypoglycemia, hyperthermia, and pulmonary edema will occur. Death is due to cardiovascular collapse and central nervous system failure.

What therapy is indicated in these patients?

The use of intravenous sodium bicarbonate has the potential for increasing the blood and urine pH, ion trapping available salicylates. The clinician should attempt to keep the blood pH between 7.45 and 7.55 and the urine pH over 7.5. In severe cases hemodialysis may be indicated.

What was the toxin in this case?

Although salicylates were not found in the home nor administered by the parents, the grandparents had been administering topical oil of wintergreen liniment to this child in an attempt to provide comfort for his viral illness. Oil of wintergreen contains very high concentrations of salicylates per unit volume and can be cutaneously absorbed.

Summary

While medical conditions certainly are possible etiologies in these presentations, careful evaluation of each patient for the presence of a classic toxidrome will often point the way to an accurate diagnosis.

CNYPCC Tidbits answers:
A. 2
B. 3
C. 4
D. 1

Tox Trivia answers:
1. Pigment at 450 wavelength of light
2. It uncouples oxidative phosphorylation leading to inefficient energy production
3. Carbon monoxide

SPI CORNER TOPIC: NO_2 ABUSE

Contributed by: Margo Spain, R.N.,SPI

The following is a list of the daily diet of the LSD " 60's' drug culture Dr. Timothy Leary, who died May 1996 at 75 years of age: Forty four cigarettes, Three cups of coffee, Two glasses of wine, One beer, One marijuana joint, Tylenol PM, Two morphine pills, Twelve balloons of Nitrous oxide and Three "leary biscuits' (a cheese soaked marijuana bud on A ritz cracker).

Thirty-five years later, health care professionals continue to see the effects of the recreational use of nitrous oxide gas. Teens today easily obtain nitrous balloons, also called "whippets" at rock concerts and clubs. Nitrous oxide gas is commonly used as a medical anesthetic. It is found as a propellant gas in food aerosols and is available for purchase at head shops, bar supply stores and the Internet. It is readily used as octane booster in race cars. The NO_2 abuser will experience intoxication within minutes of inhalation. Because the intoxication effects last only a few minutes, abusers frequently seek to prolong the high by continuing to inhale repeatedly over the course of several hours, a very dangerous practice.

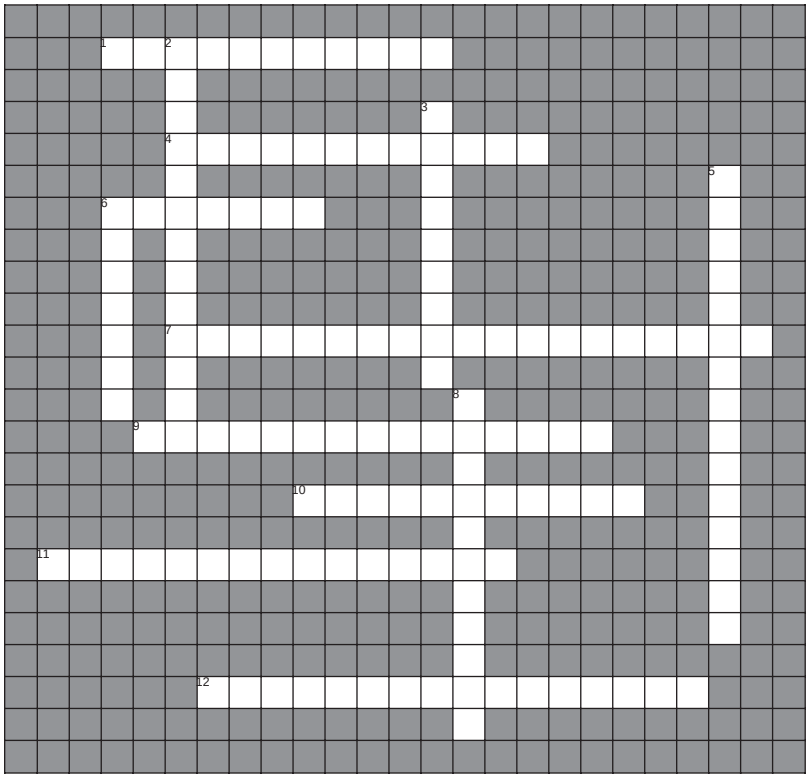
The main complications seen are due to an acute lack of oxygen. Asphyxiation occurs after repeated inhalations, resulting in symptoms including respiratory distress, headache, dizziness, excitation proceeding to CNS depression, seizures, increased intracranial pressure and death due to anoxic brain injury. Cardiac dysrhythmias may also occur, a result of hypoxia.

Treatment is aggressive, symptomatic and supportive. The mainstay of therapy includes respiratory and cardiovascular support. Oxygen, 100% should be administered and ventilations assisted/intubation initiated as needed. Careful monitoring of oxygen saturation through pulse oximetry and arterial blood gas measurements is imperative. An ECG and continuous cardiac monitoring is useful to monitor for ectopy. Cyanotic patient should be screened for methemoglobinemia. When chronic exposure is suspected, hematologic changes such as anemia may be seen on a complete blood count. There are also rare reports of myleoneuropathy after chronic inhalation. Early identification and interventions are the best ways to stop inhalant abuse. Parents, educators, health care professionals should be alert to the following signs of a serious abuse problem: (1)Drunk or disoriented appearance, including slurred speech, altered perception and motor coordination. (2) Inattentiveness, lack of coordination, irritability and depression. (3) Chemical odors on breath or clothing. (4) Stains on face, hands, and clothes. (5) Hidden empty containers, chemical soaked rags and clothing.

References:

National institute on drug abuse research report series: inhalant abuse

Arizona poison center trivia report.



Answers:
Down 2. Acetaminophen 3. Albuterol 5. diphenhydramine 6. Dextromethorphan 8. Benzonatate
Across 1. guaifenesin 4. Theophylline 6. Camphor 7. Phenylpropanolamine 9. Antichollinergic 10. Imidazoline decongestants 11. Pseudoephedrine 12. Codeine

PHARMACEUTICALS

Contributed by: Laurie Piwinski, RN, CSPI

Down

2. Hepatotoxic drug in many OTC cold and cough remedies
3. Selective beta 2 agonist used in asthma therapy
5. May make you see "little people"
6. Cough suppressant that may cause respiratory depression, especially in children
8. Antitussive with local anesthetic properties

Across

1. Non-toxic expectorant in OTC preparations
4. Methylxanthine that may cause sudden seizures refractory to standard anticonvulsant therapy
6. Mom used to rub ointment with this toxin on your chest
7. Banned by the FDA due to the risk for hemorrhagic stroke
9. Antihistamine toxidrome
10. In nasal and ophthalmic preparations, resembles clonidine in overdose
11. Decongestant of choice
12. Cough suppressant commonly abused among teenagers

Central New York Poison Center
1-800-222-1222

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Toxicology Letter

July, 2002

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Date and times for Grand Rounds
July 10, 2002 11AM
August 14, 2002, 11 AM
September 11, 2002, 11 AM

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PROGRAM ANNOUNCEMENT:

Mark your calendars for the Sixth Annual Toxicology Teaching Day, to be held on October 30, 2002 at the University Sheraton. Please watch for our brochure to be sent in early September and save the date now !! (Please call 315-464-7078 with any questions)

CNYPCC TIDBITS:

Gastrointestinal Decontamination – Match the treatment with the reported ingestion

A. Syrup of Ipecac	1. 10 times too much aminophylline intravenously
B. Orogastric lavage	2. 100 Lithium carbonate 450 mg tablets (5 min ago at home)
C. Single dose activated charcoal	3. 100 diltiazem 360 mg tablets
D. Mutiple dose activated charcoal	4. 100 digoxin 0.25mg tablets
E. Whole Bowel Irrigation	5. 100 amitryptyline 50 mg tablets

TOX TRIVIA: HISTORY

1. This was the common ingredient in many universal antidotes of antiquity?
2. Cocaine use dates back at least to: a. 300 BC, b. 1870 AD, c. 1200 AD
3. What poison may have contributed to the fall of the Roman empire?

Case History

Contributed by: Deborah Drumm, Pharm.D. Candidate and Christine M. Stork, Pharm.D., DABAT

DISCONTINUATION REACTIONS ASSOCIATED WITH SELECTIVE SEROTONIN REUPTAKE INHIBITORS AND TRICYCLIC ANTIDEPRESSANTS

Case:

A 54 year old female presents to the emergency department with new onset vertigo which caused her to fall several times in the past 24 hours. Her past medical history is significant for one episode of depression a year ago. She is maintained on no medications and has no significant social history. Upon questioning, the patient also describes difficulty sleeping and nausea in addition to vertigo for the past 48 hours. Physical examination including vital signs are non-contributory. A full medical work-up is performed with no significant findings noted. Upon further questions the patient reveals that she discontinued her antidepressant medications, paroxetine 10 mg daily and amitriptyine 25 mg at bedtime daily 3 days prior to presentation.

Can SSRIs or TCAs cause discontinuation reactions?

Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs) are commonly prescribed medications used in a variety of disorders including depression and panic. Upon discontinuation of therapy, both are associated with withdrawal symptoms. Awareness and recognition of the signs and symptoms of withdrawal is important for proper patient management.

How can you identify which SSRIs or TCAs cause the greatest risk for drug discontinuation reactions?

SSRIs and TCAs vary in their ability to cause discontinuation syndromes. Increased risk of SSRI withdrawal occurs with those drugs having shorter duration of activity and higher potency (e.g. paroxetine, sertraline). In fact, paroxetine is reported to cause discontinuation reactions in as many as 1 out of every 3 patients. Also, patients withdrawn from shorter acting drugs typically exhibit more severe withdrawal symptoms than those that occur in drugs with longer durations of activity. (e.g. citalopram, fluoxetine)

The onset of SSRI withdrawal symptoms occur as soon as after the second missed dose. Commonly reported symptoms include; disequilibrium (dizziness, lightheadedness, vertigo, ataxia), nausea, lethargy and headache. These symptoms are usually mild to moderate and self-limiting, resolving within 1 to 3 weeks or more immediately upon reinstatement of the SSRI.

TCA discontinuation symptoms can persist for as long as 4 to 5 weeks. Rates of discontinuation symptom are reported to be 100% after imipramine, 80% after amitriptyline, and 33% after clomipramine discontinuation. As with SSRIs, the shorter the duration of drug effect,

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DISCONTINUATION REACTIONS...

Continued from page 1

the more rapid and more severe the discontinuation symptoms. Common discontinuation symptoms associated with TCAs include lethargy, headache, tremor, insomnia, vivid dreams and nausea.

Why do SSRI and TCS discontinuation reactions occur?

Discontinuation reactions can occur for several reasons; noncompliance, changing from one SSRI or TCA to another, discontinuation of therapy deemed no longer medically necessary or by accidentally missing doses. In the case of discontinuation of therapy, taping doses too quickly or not at all can lead to withdrawal symptoms. Gradual tapering over at least a four-week period when discontinuing a SSRI or TCA that has been administered for 8 weeks or more should decrease the incidence of discontinuation symptoms. Many experts recommend reducing the dose by 25% every 4 to 6 weeks. It is important to note that even with gradual tapering, symptoms can occur, especially when the drug has a short duration of activity, is a TCA or has been administered for a long period of time.

What causes SSRI and TCA discontinuation reactions?

The physiologic mechanism by which SSRIs and TCAs cause withdrawal symptoms is largely unknown, although most proposed mechanisms are related to drug pharmacokinetics and phyarmacodynamics. SSRIs act to inhibit the reuptake of serotonin causing high levels of serotonin at autoreceptors and post-synaptic receptor sites. Long-term treatment with SSRIs and TCAs can lead to desensitization of these receptors. Upon discontinuation of the antidepressant there is a decreased level of available serotonin, which provides insufficient receptor agonist stimulus. Serotonin 5HT-1A receptors have been linked to motion sickness symptoms. A decreased level of serotonin at these receptors upon discontinuation is postulated to cause symptoms such as dizziness, lightheadedness, lethargy, nausea and ataxia. TCAs and to a lesser extent, paroxetine, also act to block muscarinic receptors. Upon their discontinuation, cholinergic rebound may occur, which explains symptoms such as sleep disturbances, akathisia, gastrointestinal symptoms, and anxiety. Direct or secondary effects on other neurotransmitters, such as serotonin-mediated inhibition of dopaminergic neurons, GABA, and norepinephrine may also be responsible for some of the discontinuation phenomenon.

How can SSRI and TCA discontinuation reactions be recognized?

Recognition of the key clinical features of these syndromes is important in order to prevent unnecessary medical interventions. Discontinuation reactions can be misdiagnosed as reoccurrence of depressive symptoms, ineffective or inadequate drug therapy, or adverse effects of the medication. It is important to recognize the symptoms and correlate their onset, severity, and duration. Discontinuation reactions are rare in patient treated for less than 5 weeks and symptoms are short in duration and are transient, lasting only a few days to a few weeks if left untreated.

How can discontinuation symptoms be prevented?

Patient education is extremely important in maintaining awareness of the consequences of noncompliance Before discontinuing or tapering the dose of the medication, it is important to educate the patient on the symptoms which may develop.

How should SSRI and TCA discontinuation reactions be treated?

The best approach to treatment of the symptoms is to prevent them before they occur. Gradual tapering of the SSRI or TCA should be done over at least a 4-6 week period. The severity of symptoms should determine if treatment is necessary. Continued depression coupled with discontinuation symptoms should be treated by reinstatement of the antidepressant with a more gradual tapering schedule. Insomnia associated with discontinuation syndromes can be treated with short-term use of benzodiazepines or agents such as zolpied (ambient®) or xaleplone (Sonata®). Headaches and nausea can be symptomatically treated with pharmacological agents as well. Simple reassurance is also helpful in easing the concern of a worried patient.

CNYPCC Tidbits answers:

A. 2
B. 5
C. 4
D. 1
E. 3

Tox Trivia answers:

1. Opium
2. a
3. Lead

SPI CORNER TOPIC: DRUG-INDUCED HYPERTHERMIA

Contributed by: Laurie Piwinski, R.N.,SPI

Serotonin Syndrome

Serotonin syndrome typically occurs in susceptible individuals 1-2 hours after exposure to concomitant serotonergic drugs. Common interactions include SSRIs, MAOIs, TCAs, meperidine, lithium, dextromethorphan, atypical antidepressants with serotonergic properties, and some drugs of abuse such as LSD, MDMA, and Ecstasy. Symptoms include mental status changes, restlessness, muscular rigidity, myoclonus, hyperreflexia, diaphoresis, shivering, fever and tachycardia. Treatment includes aggressive supportive care, sedation and cooling 24-48 hours after discontinuation of the offending agent. In severe cases, neuromuscular blockage may be required. Serotinerigc antagonists, such as cyproheptadine can be used but are not critical in reducing life-threatening hyperthermia.

Neuroleptic Malignant Syndrome (NMS)

NMS is characterized by fever, muscle rigidity, extrapyramidal effects, and mental status changes. It is the result of central dopamine blockade and is typically precipitated by medications including phenothiazines, butyrophenones, thioxanthines and loxapine.

Treatment includes discontinuation of the causative drug, supportive care, cooling, and sedation As with serotonin syndrome, paralysis may be indicated in severe cases, in which hyperthemia caused by muscular rigidity cannot be otherwise controlled.

Malignant Hyperthermia (MH)

MH is a genetic disease that involves muscle hypermetabolism triggered by exposure to certain anesthetic agents and succinylcholine. Symptoms include muscular rigidity, excess oxygen consumption and carbon dioxide production, continuous calcium reuptake leading to depletion of cellular ATP, and potentially life threatening hyperkalemia. Hyperthermia is one of the later findings.

Treatment consists of immediate discontinuation of the offending agent and giving dantrolene, which blocks the reuptake of calcium, reversing the muscular contractions. Good supportive care is essential. Follow up should always include referral to a malignant hyperthermia biopsy center for genetic counseling and definitive diagnosis.

FDA SAFETY SUMMARIES 4/02-6/02

Seroquel (quetiapine fumarate) - medication errors involving confusion with Serzone (nefazodone hydrochloride)

Procrit (epoetin alfa) - lot number P002641, P002384, P002970 found to contain active ingredient that is approximately 20 times lower in potency

Lioresal Intrathecal (baclofen injection) - cases of intrathecal baclofen withdrawal that can lead to life threatening sequelae

Serostim [somatropin (rDNA origin) for injection] - counterfeit lot of Serostim [somatropin (rDNA origin) for injection]. S810-1A1.

Combivir (lamivudine plus zidovudine) - bottles containing 60 tablets of Combivir (lamivudine plus zidovudine) that actually contained another medicine

PLAS+SD (Pooled Plasma, (Human) Solvent Detergent Treated) - contraindicates the use of PLAS+SD in patients undergoing liver transplant, patients with severe liver disease and known coagulopathies

Zyprexa (olanzapine) - product tampering of 10 and 15 mg bottles

Vioxx (rofecoxib) - strengthened the WARNINGS, PRECAUTIONS, and CLINICAL STUDIES sections of Vioxx to describe new cardiovascular & GI information

Heparin Sodium Injection, USP - voluntarily recalling ALL lots of Heparin Sodium Injection, due to the presence of clear crystals

Thiazolidinediones [Actos (pioglitazone HCl), Avandia (rosiglitazone maleate)] - more clearly describe the cardiovascular risks

Rapamune (sirolimus) - risk of hepatic artery thrombosis, graft loss, and death

Albuterol Sulfate - Solution for Inhalation (0.5%) recent hospital outbreaks of lower respiratory tract colonization and infection with Burkholderia cepacia attributed to contamination.

Milk Based Powdered Infant Formulas Used in Neonatal Intensive Care Units - Enterobacter sakazakii infections in neonates

Lovenox (enoxaparin sodium) - not recommended for thromboprophylaxis in patients with prosthetic heart valves.

Zerit (stavudine) - potential for lactic acidosis as a complication of therapy with Zerit (stavudine), d4T.

Kava-containing Dietary Supplements (Piper methysticum) - severe liver injury

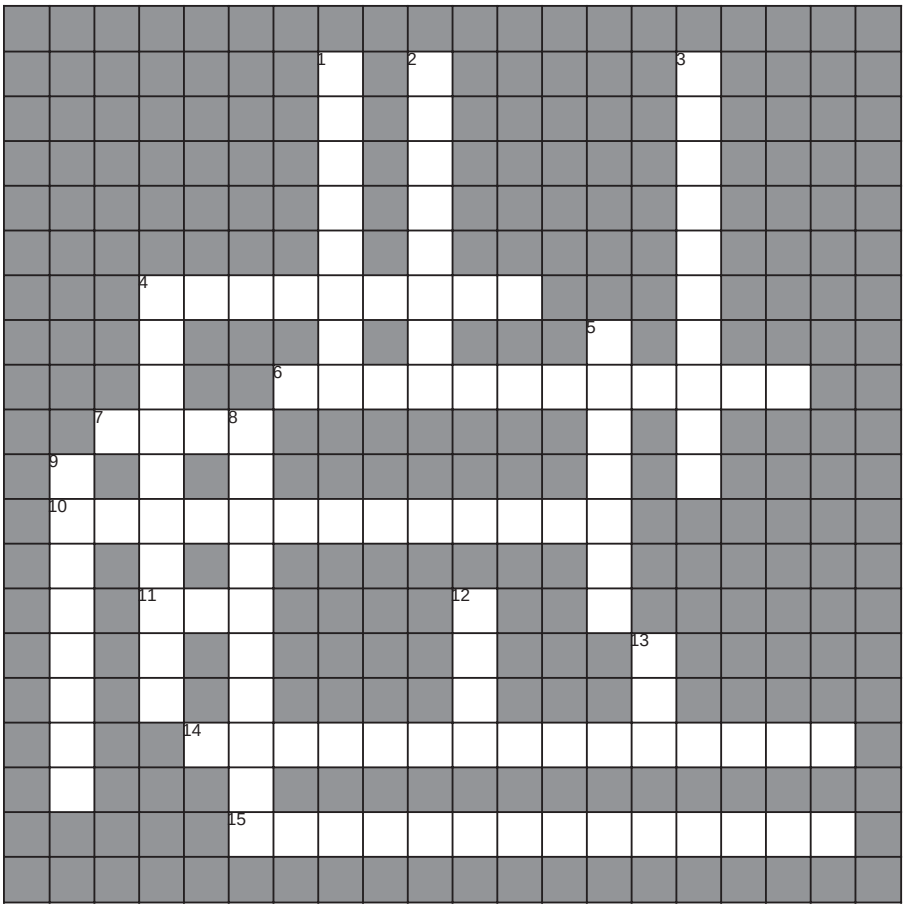
Alpha Interferons - Intron A (Interferon alfa 2b, recombinant) Rebetron Combination Therapy (Rebetrol (Ribavirin, USP) Capsules and Intron A) Roferon-A (Interferon alfa-2a, recombinant) - occurrence of neuropsychiatric, autoimmune, ischemic, and infectious disorders

Versed Syrup (midazolam HCl) CIV - potential presence of a crystalline precipitate

PC SPES, SPES (BotanicLab) - warned to stop using the dietary supplement / herbal products PC SPES and SPES capsules because they contain undeclared prescription drug ingredients

Serzone (nefazodone HCL) - Black Box Warning. Rare cases of liver failure

CROSSWORD PUZZLE



DOWN

1. SERIOUS SUNBURNS, ESPECIALLY IN CHILDHOOD CAN INCREASE THE CHANCES OF DEVELOPING THIS CANCER.
2. THIS TOXIN IS FOUND IN TOMATOES AND NIGHTSHADE BERRIES; HAS RELATIVELY LITTLE TOXICITY IN ADULTS, BUT FATAL INTOXICATIONS HAVE OCCURRED IN CHILDREN
3. THIS UNCOMMON FORM OF HEAT ILLNESS CONSTITUTES A TRUE MEDICAL EMERGENCY
4. THESE MUSHROOMS HAVE A LSD-LIKE EFFECT
5. THIS MUSHROOM ACCOUNTS FOR 95% OF MUSHROOM-RELATED FATALITIES.
8. MOST VENOMOUS FISH OF THE TEMPERATE ZONE FOUND ON THE BOTTOM IN SHALLOW WATER
9. CARRIER OF THE LYME DISEASE
12. ACUTE POISONING IN CHILDREN FROM USE OF THIS SPRAY MAY BE MISTAKEN FOR VIRAL INFECTION
13. THESE TYPE OF RAYS CAN BE HARMFUL TO THE SKIN

ACROSS

4. THIS PLANT;OFTEN REFERRED TO “LEAVES OF THREE LET THEM BE”, CAN CAUSE EDEMA, ERYTHEMA,BLISTERS AND IS OFTEN SPREAD WITH SCRATCHING OF CONTAMINATED AREAS
6. SOME CONSIDER THIS PLANT THE MOST VIOLENTLY POISONOUS OF THE NORTH TEMPERATURE ZONE.
7. THIS STICK WHICH IS OFTEN SOLD AT COUNTY FAIRS MAY CAUSE ORAL IRRITATION WHEN BITTEN INTO
10. MAY GET THIS ORGANISM FROM EATING UNDERCOOKED HAMBURGER FROM THE GRILL
11. MORE DEATHS IN THE U.S. ANNUALLY FROM THIS INSECT STING THAN FROM SNAKE BITES
14 THESE DAINTY, FRAGRANT, BELL-LIKE, WHITE FLOWERS HAVE DIGITALIS-LIKE SXS
15. THIS ILLNESS OFTEN OCCURRING IN ATHLETES IN WARM TEMPERATURES IS CHARACTERIZED BY WEKNESS, H/A, N/V, DIARRHEA, AND MUSCLE CRAMPS

ANSWERS
DOWN 1. melanoma, 2. solanine, 3. heatstroke, 4. psilocybin, 5. amanita, 8. weeverfish, 9. deer tick, 12. DEET, 13. UVA
ACROSS 4. poison ivy, 6. water hemlock, 7. glow, 10. escherica coli, 11. bee, 14. lily of the valley, 15. heat exhaustion

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CNYPCC TIDBITS:

Inhalants - Matching

A. Phosgene	1. normal pO2, false normal pulse oximeter reading
B. Carbon monoxide.	2. normal pO2, irrationally lowered pulse oximeter reading
C. Chlorine	3. delayed irritant, smells like hay
D. Methemoglobinemia	4. irritant, sodium bicarbonate may alleviate symptoms

TOX TRIVIA: COLORS

1. Causes “blue/green emesis and lobster red skin”
2. Makes blood look “chocolate”
3. Orange tinged secretions

Case History

Contributed by: Tracey H. Reilly, M.D., Jeanna M. Marraffa, Pharm.D., Christine M. Stork, Pharm.D., DABAT

SUDDEN DEATH IN A HEALTHY THIRTEEN YEAR OLD ADOLESCENT AFTER ACUTE INHALATION OF GASOLINE

Case:

A 13-year-old previously healthy male was found sitting on his ATV (all terrain vehicle) with his face into the opening of the gas tank. His mother last saw him acting normally earlier that morning. He was unresponsive and emergency medical assistance (EMS) was contacted. Upon arrival of EMS, the patient remained unresponsive and cardiopulmonary resuscitation was initiated.The electrocardiogram revealed asystole despite 11 doses of epinephrine, 4 doses of atropine and 1 dose of sodium bicarbonate. After an hour of resuscitation, resuscitative efforts were terminated.

What is the likely etiology of the cardiopulmonary arrest in this patient?

The incidence of volatile substance use for abusive purposes is increasing along with the number of deaths occurring after use of these substances.

Inhalants are chemicals that vaporize at room temperature and are intentionally inhaled to achieve a mind-altered state. Any substance that exists in a gaseous form or rapidly evaporates at room temperature, is psychoactive and is not extremely irritating has the potential to be abused by inhalation.[1]

The types of inhalants abused comprise a large and diverse group of chemicals that are mostly hydrocarbon derivatives. Fuels for engines, lighters and lamps as well as solvents for paints and adhesives are among the more common agents abused. Inhalation of volatile substances of abuse can be accomplished by “sniffing”, “huffing”, or variants of these methods. Advanced methods of inhalation abuse termed ‘bagging’ involve breathing in and out of a plastic or paper bag that contains a small amount of the substance to be abused.[1]

The concentration of the inspired volatile substance of abuse increases from sniffing to huffing to bagging.

What is the epidemiology of inhalant abuse?

Volatile substance inhalation abuse (VSIA) is typically seen in older children and young adolescents. According to the National Institute on Drug Abuse approximately 6% of US children have experimented with inhalants by the time they reach fourth grade.[2] Abuse usually peaks during grades seven through nine with only a small percentage of children going on to become chronic abusers. The inhalation of volatile substances of abuse is reported in up to 13% of teenage

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populations. Males more than females tend to abuse inhalants. Children with poor school performance, volatile family environments and poor socioeconomic status appear to be at higher risk for abuse of inhalants.[2]

What are the dangers of inhalant abuse?

Hydrocarbons are central nervous system (CNS) depressants that initially cause euphoria, intoxication and a mind altered state similar to ethanol due to their inhibition of cortical function. Very little exposure is required, only 15 to 20 inhalations can produce euphoria or intoxication that can last 3 to 6 hours.[3] All inhalational substances of abuse have to ability to cause neurologic dysfunction, pulmonary irritation, asphyxia, cardiovascular abnormalities and GI and dermal irritation.[1] Cardiac arrhythmias and death can occur with first time use. Irreversible CNS and peripheral nervous system (PNS) damage can occur after repeated exposure. Pulmonary asphyxiation can develop when vapors decrease the partial pressure of oxygen in inspired air to below 17 percent. Common gastrointestinal side effects include nausea and vomiting which can increase the risk of aspiration and pulmonary irritation especially when the abuser may have an altered mental status due to euphoria or hypoxia.

The mechanisms of death due to inhalant abuse include cardiac dysrhythmias, vagus-mediated cardiac inhibition, CNS respiratory depression, mechanical asphyxia, aspiration of gastric contents and trauma due to intoxication.[3] Cardiac dysrhythmias with or without hypoxia appears to be the most common cause of death due to inhalant abuse. However, this conclusion is difficult to assess because post-mortem findings of proposed deaths due to VSIA are typically minimal.[4] Sudden death typically occurs when an intoxicated patient performs some sort of physical exertion.

Volatile substances are thought to sensitize the myocardium to the effects of circulating catecholamines by stabilizing the myocardial cells to depolarization. Stabilization results in reduced irritability of myocardial cells. Cardiovascular depression is dose dependent and biphasic. Initially, there is peripheral vasodilation resulting in hypotension with a reflex tachycardia. This is followed by bradycardia, decreased cardiac contractility and decreased cardiac output. When the myocardium is sensitized through the use of inhalants, the variability of the irritability of the individual myocardial cells can lead to small blocks in electrical impulse that can increase the risk of ventricular arrhythmias and ectopic foci of pacemaker activity.[4-6]

Are there any considerations in the management of cardiopulmonary arrest in patients with acute intoxication of volatile substances of abuse?

Cardiac arrhythmias appear to be the most likely cause of the majority of acute deaths associated with volatile substances of abuse. Dysrhythmia induced sudden death is termed "sudden sniffing death syndrome". The mechanism of dysrhythmia appears to be sensitization of the myocardium to endogenous catecholamines.

Management of dysrhythmias should include management of electrolyte disturbances, hypoxemia, hypotension and hypothermia. Ventricular fibrillation poses a particular concern as common practice guide the use of epinephrine in this setting.

Since the myocardium is sensitized to the effects of catecholamines, sympathomimetic drugs should be avoided.[11] Beta adrenergic antagonists have been recommended to be administered to protect the sensitized heart from circulating catecholamines. However, these reduce the ionotropy of the heart and should be used with care. Other drugs to consider are lidocaine and amiodarone.[8] Several cases of successful treatment of cardiac arrhythmias from chloral hydrate toxicity with propranolol have been documented.[10] Chloral hydrate sensitizes the myocardium to circulating catecholamines and induces ventricular arrhythmias resistant to standard catecholamine based antiarrhythmics. Literature reviews suggest that ventricular arrhythmias induced by chloral hydrate are resistant to lidocaine but responsive to beta blockers. Published case studies suggest a treatment regimen that involves an intravenous bolus of a beta blocker followed by an intravenous infusion to maintain a heart rate of 50 to 70 beats per minute.[10] Selection of the beta blocker should be based on the half life of the beta blocker and the estimated half life of the toxic drug or metabolite.

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SPI CORNER TOPIC: THE TERRORIST THREAT AND THE ROLE OF THE POISON SPECIALIST.

Contributed by: Kathleen Groff, RN, CSPI

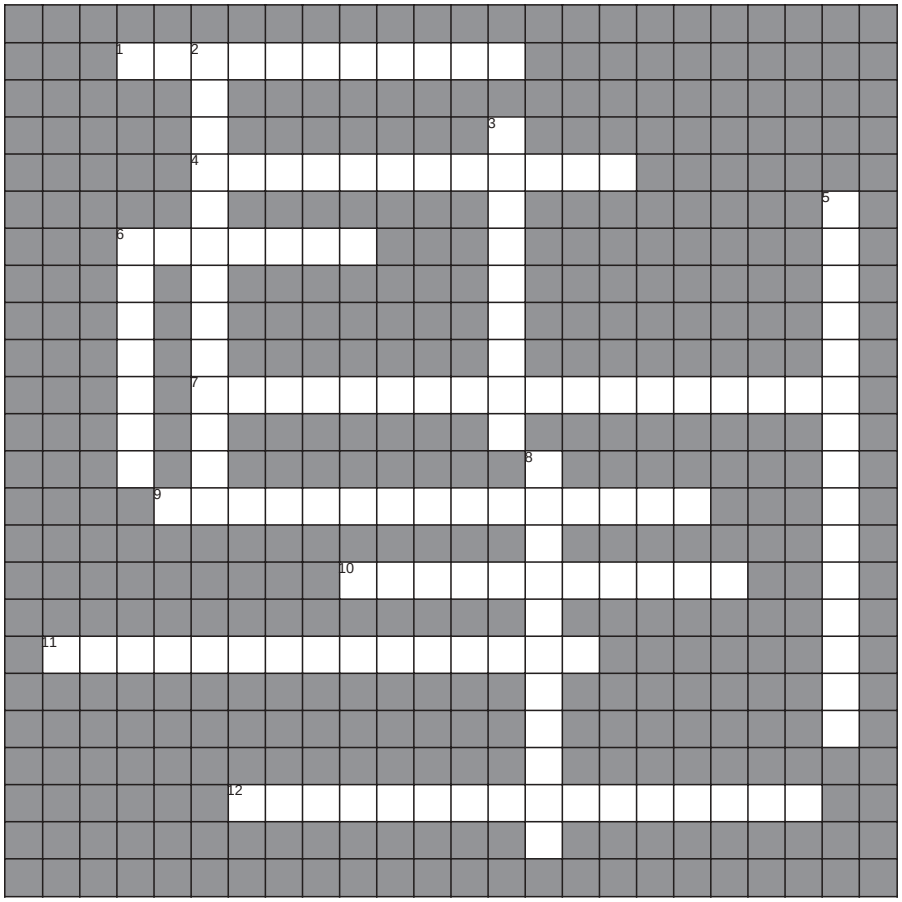
In the wake of the tragic events of 911 and the anthrax threats that followed, the nation is preparing for the worst, another attack. We all understand that domestic preparedness and medical readiness is key. The Poison center has a vital role during a WMD [weapons of mass destruction] incident. That role is to provide the most current toxicological information available. The specialists are highly qualified and will provide information on hazardous material exposures to both the general public and health care professionals. Throughout the anthrax crisis the call volume increased dramatically as the poison specialist remained a constant and credible resource able to provide concise and reassuring information to the public and health care community.

Many of the agents with potential for use as a weapon of mass destruction are found in every day life in which the poison specialists already have some experience. An example is organophosphate pesticides, commonly used in commercial grades on large farms being similar in both presentation and management to a weaponized nerve agent exposure. The method of delivery may also be similar, for example delivered by ground tank dispersal or crop dusting plane.

Poison information specialists have the knowledge to assist healthcare providers in several ways; In determining the agent- creating a toxin related differential diagnosis, By reviewing toxicological effects, Providing decontamination and treatment recommendations, Describing contamination risks for healthcare providers, Reviewing indicated labs and tests, Identifying location of appropriate lab. facility, and by facilitating a toxicological consult with the on call toxicologist.

CROSSWORD PUZZLE: COLD SEASON

Contributed by: Laureene Piwinski, RN, CSPI



DOWN:

2. Hepatotoxic drug in many OTC cough and cold remedies
3. Selective beta-2 adrenergic agonist used in asthma therapy
5. If you take too much of this, you may see “little people”
6. Cough suppressant that may cause respiratory depression, especially in children
8. Antitussive with local anesthetic properties

ACROSS:

1. Non-toxic expectorant in many OTC preparations
4. Methylxanthine that cause seizures refractory to standard anticonvulsant therapy
6. Mom rubbed this toxin on your chest
7. Banned by the FDA due to an increased risk of hemorrhagic stroke
9. Antihistamine toxidrome
10. Class of decongestants that resemble clonidine in oral overdose
11. Decongestant of choice
12. Cough suppressant commonly abused among teens

ANSWERS:
Down: *2 acetaminophen, 3 albuterol, 4 diphenhydramine, 6 codeine, 8 benzonatate*
Across: *1 guaifenesin, 4 theophylline, 6 camphor, 7 phenylpropanolamine, 9 anticholinergic, 10 imidazoline, 11 imidazoline, 11 pseudoephedrine, 12 dextromethorphan*

DID YOU KNOW?

- That the Poison Center offers programs for healthcare professionals on both biological and chemical exposures.
- The poison center has dedicated WMD- Hazardous materials coordinators.
- The poison center participates on a local and state level with emergency response planning.
- The Poison center will soon offer a WMD web page for health care professionals, to provide toxicological information quickly should conventional communication systems become overwhelmed.

The role of the Poison Specialist in the face of terrorism is that of an expert toxicological resource. The specialist can provide insight and support during the emergency planning phase. The specialist can provide reassuring information to a panicked public. The specialist can provide ongoing educational programs for both the public and the health care community. The specialist can provided essential toxicological information in an effort to reduce morbidity and mortality. Please call the Poison Center with any questions you may have, including interest in inservice/lectures.

CNYPCC Tidbits answers:

A. 3
B. 1
C. 4
D. 2

Tox Trivia answers:

1. Boric acid
2. Methemoglobinemia
3. Rifampin

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