

# *Current Awareness in Clinical Toxicology*

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## **CURRENT AWARENESS PAPERS OF THE MONTH**

### **Paracetamol (acetaminophen) attenuates *in vitro* mast cell and peripheral blood mononucleocyte cell histamine release induced by *N*-acetylcysteine**

**Coulson J, Thompson JP. Clin Toxicol 2010; 48: 111-4.**

#### ***Introduction***

The treatment of acute paracetamol (acetaminophen) poisoning with *N*-acetylcysteine (NAC) is frequently complicated by an anaphylactoid reaction to the antidote. The mechanism that underlies this reaction is unclear. We used the human mast cell line 1 (HMC-1) and human peripheral blood mononucleocytes (PBMCs) to investigate the effects of NAC and paracetamol on histamine secretion *in vitro*.

#### ***Method***

HMC-1 and human PBMCs were incubated in the presence of increasing concentrations of NAC ± paracetamol. Cell viability was determined by the Trypan Blue Assay, and histamine secretion was measured by ELISA.

#### ***Results***

NAC was toxic to HMC-1 cells at 100 mg/mL and to PBMCs at 67 mg/mL. NAC increased HMC-1 and PBMC histamine secretion at concentrations of NAC from 20 to 50 mg/mL and 2.5 to 100 mg/mL, respectively. NAC-induced histamine secretion by both cell types was reduced by co-incubation with 2.5 mg/mL of paracetamol.

#### ***Conclusion***

Paracetamol (acetaminophen) is capable of modifying histamine secretion *in vitro*. This may explain the clinical observation of a lower incidence of adverse reactions to NAC *in vivo* when higher concentrations of paracetamol are present than when paracetamol concentrations are low. Paracetamol (acetaminophen) attenuates *in vitro* mast cell and PBMC cell histamine release induced by NAC.

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## **Medicine, poison, and mystic potion: a personal perspective on paracetamol. Louis Roche lecture, presented at the EAPCCT XXIX International Congress, 12-15 May 2009, Stockholm, Sweden**

**Bateman DN, Dear J. Clin Toxicol 2010; 48: 97-103.**

### ***Background***

Paracetamol poisoning has been a clinical problem for over 40 years. This article reflects the content of the Louis Roche lecture given on this topic in 2009.

### ***Historical context***

Initially key work illustrated the relationship between plasma paracetamol concentration and risk of liver injury facilitating the development of antidote strategies. Much of this work was done in the Edinburgh clinical toxicology unit.

### ***Discussion***

The antidote most widely used, acetylcysteine, was developed in Edinburgh and subsequently shown to be associated with adverse effects. Studies on this aspect and on key problems remaining in patient management are described. These include the identification of patients at greatest risk of toxicity, in order that they may be targeted for appropriate early intervention. More recent work suggests that the inflammatory response to paracetamol-induced liver injury may offer new targets for drug therapy in this major poisoning.

### ***Conclusion***

Paracetamol poisoning remains a challenging problem, but new approaches to treatment seem possible based on recent experimental studies in animals and humans.

## **Outcomes of liver transplantation for paracetamol (acetaminophen)-induced hepatic failure**

**Cooper SC, Aldridge RC, Shah T, Webb K, Nightingale P, Paris S, Gunson BK, Mutimer DJ, Neuberger JM. Liver Transpl 2009; 15: 1351-7.**

### ***Abstract***

Paracetamol (acetaminophen) hepatotoxicity, whether due to intentional overdose or therapeutic misadventure, is an indication for liver transplantation in selected cases. However, there is a concern that long-term outcomes may be compromised by associated psychopathology that may predispose patients to further episodes of self-harm or poor treatment adherence.

We therefore undertook a retrospective analysis of patients transplanted for paracetamol-induced fulminant hepatic failure (FHF) to determine their long-term outcomes, psychiatric problems, and compliance and whether these issues could be predicted from pretransplant information. Records from patients undergoing liver transplantation for paracetamol-associated liver failure in this unit and 2 comparison groups (patients undergoing liver replacement for FHF from other causes and for chronic liver diseases) were examined.

Of 60 patients transplanted for paracetamol-induced FHF between 1989 and 2007, 44 (73%) survived to discharge. Currently, 35 patients (58%) are surviving at an average of 9 years post-transplantation. The incidence of psychiatric disease (principally depression) and 30-day mortality were greatest in the paracetamol group, but for those who survived 30 days, there was no difference in long-term survival rates between the groups. Adherence to follow-up appointments and compliance with immunosuppression were lowest in the paracetamol overdose group. Poor adherence was not predicted by any identifiable premorbid psychiatric conditions. Two patients grafted for paracetamol FHF died from self-harm (1 from suicide and 1 from alcoholic liver disease after 5 years).

This study suggests that, notwithstanding the shortage of donor liver grafts, transplantation is an appropriate therapy in selected patients, although close follow-up is indicated.

## Oxygen consumption is depressed in patients with lactic acidosis due to biguanide intoxication

Protti A, Russo R, Tagliabue P, Vecchio S, Singer M, Rudiger A, Foti G, Rossi A, Mistraletti G, Gattinoni L. *Crit Care* 2010; 14: R22.

### Introduction

Lactic acidosis can develop during biguanide (metformin and phenformin) intoxication, possibly as a consequence of mitochondrial dysfunction. To verify this hypothesis, we investigated whether body oxygen consumption ( $VO_2$ ), that primarily depends on mitochondrial respiration, is depressed in patients with biguanide intoxication.

### Methods

Multicentre retrospective analysis of data collected from 24 patients with lactic acidosis (pH  $6.93 \pm 0.20$ ; lactate  $18 \pm 6$  mM at hospital admission) due to metformin (n = 23) or phenformin (n = 1) intoxication. In 11 patients,  $VO_2$  was computed as the product of simultaneously recorded arterio-venous difference in  $O_2$  content [ $C_{a-v}O_2$ ] and cardiac index (CI). In 13 additional cases,  $C_{a-v}O_2$ , but not CI, was available.

### Results

On day 1,  $VO_2$  was markedly depressed ( $67 \pm 28$  ml/min/m<sup>2</sup>) despite a normal CI ( $3.4 \pm 1.2$  L/min/m<sup>2</sup>).  $C_{a-v}O_2$  was abnormally low in both patients either with ( $2.0 \pm 1.0$  ml  $O_2$ /100 ml) or without ( $2.5 \pm 1.1$  ml  $O_2$ /100 ml) CI (and  $VO_2$ ) monitoring. Clearance of the accumulated drug was associated with the resolution of lactic acidosis and a parallel increase in  $VO_2$  (P < 0.001) and  $C_{a-v}O_2$  (P < 0.05). Plasma lactate and  $VO_2$  were inversely correlated ( $R^2$  0.43; P < 0.001, n = 32).

### Conclusions

$VO_2$  is abnormally low in patients with lactic acidosis due to biguanide intoxication. This finding is in line with the hypothesis of inhibited mitochondrial respiration and consequent hyperlactatemia.

## Randomized controlled trial comparing the effectiveness and safety of intranasal and intramuscular naloxone for the treatment of suspected heroin overdose

Kerr D, Kelly A-M, Dietze P, Jolley D, Barger B. *Addiction* 2009; 104: 2067-74.

### Aims

Traditionally, the opiate antagonist naloxone has been administered parenterally; however, intranasal (i.n.) administration has the potential to reduce the risk of needlestick injury. This is important when working with populations known to have a high prevalence of blood-borne viruses. Preliminary research suggests that i.n. administration might be effective, but suboptimal naloxone solutions were used. This study compared the effectiveness of concentrated (2 mg/ml) i.n. naloxone to intramuscular (i.m.) naloxone for suspected opiate overdose.

### Methods

This randomized controlled trial included patients treated for suspected opiate overdose in the pre-hospital setting. Patients received 2 mg of either i.n. or i.m. naloxone. The primary outcome was the proportion of patients who responded within 10 minutes of naloxone treatment. Secondary outcomes included time to adequate response and requirement for supplementary naloxone. Data were analysed using multivariate statistical techniques.

### Results

A total of 172 patients were enrolled into the study. Median age was 29 years and 74% were male. Rates of response within 10 minutes were similar: i.n. naloxone (60/83, 72.3%) compared with i.m. naloxone (69/89, 77.5%) [difference: -5.2%, 95% confidence interval (CI) -18.2-7.7]. No difference was observed in mean response time (i.n.: 8.0, i.m.: 7.9 minutes; difference 0.1, 95% CI -1.3-1.5). Supplementary naloxone was administered to fewer patients who received i.m. naloxone (i.n.: 18.1%; i.m.: 4.5%) (difference: 13.6%, 95% CI 4.2-22.9).

### ***Conclusions***

Concentrated intranasal naloxone reversed heroin overdose successfully in 82% of patients. Time to adequate response was the same for both routes, suggesting that the i.n. route of administration is of similar effectiveness to the i.m. route as a first-line treatment for heroin overdose.

## **The course of chronic solvent induced encephalopathy: a systematic review**

**van Valen E, Wekking E, Van der Laan G, Sprangers M, van Dijk F. Neurotoxicology 2009; 30: 1172-86.**

### ***Background***

Worldwide millions of workers are exposed to organic solvents. Long term exposure leads in some workers to the development of Chronic Solvent induced Encephalopathy (CSE). The first reports about CSE came from the European Nordic countries in the 1970s. In spite of decades of experience with this disease, little is known about the course and prognostic factors of CSE.

### ***Objective***

To provide an overview of the evidence about the course and prognostic factors of CSE.

### ***Methods***

A systematic review was conducted. Databases PubMed, PsycINFO (1970-2008) and Embase (1980-2008) were searched with the search strategy: solvent AND follow up AND (encephalopathy OR chronic intoxication). Inclusion criteria were: written in English, study population of CSE patients, follow-up time of at least 1 year. Included articles were assessed on methodological quality.

### ***Results***

Sixty unique articles were retrieved of which sixteen met the inclusion criteria. Data extraction provided information about domains of neurology, neuropsychology, physical and mental health perceptions, and social consequences. In a number of studies no significant changes, and in other studies improvement of functioning could be measured. Prognostic factors resulting from included studies were summarized for each domain indicating a potential positive influence of younger age and lower exposure variables.

### ***Discussion***

Due to the large heterogeneity of methodology no levels of evidence could be obtained. This review shows that there is a need for future research that addresses a variety of domains of functioning, hopefully resulting in an overall prognostic model for CSE.

### ***Conclusion***

Studies in this review are in agreement about CSE being a non-progressive disease in which no severe deterioration of functioning occurs after diagnosis. In a number of studies no significant changes, and in other studies improvement of functioning could be measured. Presumably cessation of exposure might be one of the causal factors for the non-progressive character of the disease as has been found. Future studies are needed to clarify the role of various prognostic factors on the course of CSE.

## **Pediatric ingestions of hand sanitizers: debunking the myth**

**Mrvos R, Krenzelok EP. Pediatr Emerg Care 2009; 25: 665-6.**

### ***Objective***

Poison centers frequently receive calls concerning children who are exposed to hand sanitizers. These exposures can occur while the product is being used correctly or when a child has unsupervised access to the container. In 2007, the use of ethanol-containing hand sanitizers in the pediatric population came under media scrutiny owing to an Internet urban legend that resulted in a greater awareness of the potential toxicity of these sanitizers based on their high ethanol content.

### ***Methods***

A retrospective review of all exposures to hand sanitizers in children younger than 6 years

reported to a regional poison information center from January 1, 2000 to March 30, 2007 was performed. Data reviewed included substance, age, sex, and outcome. Data were analyzed using descriptive statistics.

### **Results**

Six hundred forty-seven cases were identified including 324 females and 323 males. Ages ranged from 1 month to 5 years with a mean of 1.89 years and a median of 2 years. Outcome data included 31 patients (4.8%) with no effect, 26 (4%) with a minor effect, 372 cases (57.5%) coded as nontoxic - expect no effect, 208 cases (32.1%) with minimal clinical effects possible, and 10 cases (1.6%) where the symptoms were judged to be unrelated to the exposure. There were no moderate or major outcomes and no fatalities.

### **Conclusions**

Children in this age group have frequent hand-to-mouth activity and environmental curiosity making the application or availability of a hand sanitizer the perfect situation for an exposure to occur. Although ethanol-based hand sanitizers have the potential to cause toxicity, the benefits of prevention of illness outweigh the hazards when used in a supervised situation.

## **A new tool for the early diagnosis of carbon monoxide intoxication**

**Piatkowski A, Ulrich D, Grieb G, Pallua N. *Inhal Toxicol* 2009; 21: 1144-7.**

### **Abstract**

Invasive measurement of carboxyhemoglobin (COHb) by blood gas analysis (BGA) is accepted as the standard diagnostic procedure in diagnosis of inhalation injury and carbon monoxide (CO) intoxications.

The main disadvantage of BGA with COHb testing is the unavailability in pre-hospital rescue conditions. The non-invasive SpCO analysis using pulse CO oximetry (Rad57, Masimo Corp., USA) represents an easy-to-handle device to facilitate the diagnosis of CO intoxication.

Between January 2006 and August 2008, 20 patients who were admitted with CO intoxication to our burn centre were included in this study. Blood gas analysis including COHb testing was performed on the first day, hourly. At the same time, SpCO was determined using the Rad57 pulse CO oximeter. Patients received inhaled oxygen according to the parameters of blood gas analysis or hyperbaric oxygenation if COHb > 10%. Five young healthy volunteers served as control group. The SpCO of the volunteers was cross-checked against their COHb levels, which were measured by blood gas analysis.

Results of pulse CO oximetry revealed a mean error of approximately 3.15% from the results achieved by blood gas analysis. If COHb resulted in values higher than 10%, the bias remained approximately the same (3.43%/precision 2.362%). When different blood gas analyzers in our department were tested with the same patient sample, a mean error of 2.4% was found. This is only 1% lower compared to the mean error of pulse CO oximetry.

Therefore, pulse CO oximetry represents a reliable measurement technique that is easy to handle and could facilitate the early diagnosis of CO intoxication in pre-hospital rescue conditions.

## **A prospective observational study of the clinical toxicology of glyphosate-containing herbicides in adults with acute self-poisoning**

**Roberts DM, Buckley NA, Mohamed F, Eddleston M, Goldstein DA, Mehrsheikh A, Bleeke MS, Dawson AH. *Clin Toxicol* 2010; 48: 129-36.**

### **Context**

The case fatality from acute poisoning with glyphosate-containing herbicides is approximately 7.7% from the available studies but these have major limitations. Large prospective studies of patients with self-poisoning from known formulations who present to primary or secondary hospitals are needed to better describe the outcome from acute poisoning with glyphosate-containing herbicides. Furthermore, the clinical utility of the glyphosate plasma concentration for

predicting clinical outcomes and guiding treatment has not been determined.

### **Objective**

To describe the clinical outcomes, dose-response, and glyphosate kinetics following self-poisoning with glyphosate-containing herbicides.

### **Methods**

This prospective observational case series was conducted in two hospitals in Sri Lanka between 2002 and 2007. We included patients with a history of acute poisoning. Clinical observations were recorded until discharge or death. During a specified time period, we collected admission (n = 216, including five deaths) and serial (n = 26) blood samples in patients. Severity of poisoning was graded using simple clinical criteria.

### **Results**

Six hundred one patients were identified; the majority ingested a concentrated formulation (36%, w/v glyphosate). Twenty-seven percent were asymptomatic, 63.7% had minor poisoning, and 5.5% of patients had moderate to severe poisoning. There were 19 deaths (case fatality 3.2%) with a median time to death of 20 h. Gastrointestinal symptoms, respiratory distress, hypotension, altered level of consciousness, and oliguria were observed in fatal cases. Death was strongly associated with greater age, larger ingestions and high plasma glyphosate concentrations on admission (>734 µg/mL). The apparent elimination half-life of glyphosate was 3.1 h (95% CI = 2.7-3.6 h).

### **Conclusions**

Despite treatment in rural hospitals with limited resources, the mortality was 3.2%, which is lower than that reported in previous case series. More research is required to define the mechanism of toxicity, better predict the small group at risk of death, and find effective treatments.

## **Pro-2-PAM therapy for central and peripheral cholinesterases**

**Demar JC, Clarkson ED, Ratcliffe RH, Campbell AJ, Thangavelu SG, Herdman CA, Leader H, Schulz SM, Marek E, Medynets MA, Ku TC, Evans SA, Khan FA, Owens RR, Nambiar MP, Gordon RK. *Chem Biol Interact* 2010; Online early: PM:20156430:**

### **Abstract**

Novel therapeutics to overcome the toxic effects of organophosphorus (OP) chemical agents are needed due to the documented use of OPs in warfare (e.g. 1980-1988 Iran/Iraq war) and terrorism (e.g. 1995 Tokyo subway attacks).

Standard OP exposure therapy in the United States consists of atropine sulfate (to block muscarinic receptors), the acetylcholinesterase (AChE) reactivator (oxime) pralidoxime chloride (2-PAM), and a benzodiazepine anticonvulsant to ameliorate seizures. A major disadvantage is that quaternary nitrogen charged oximes, including 2-PAM, do not cross the blood brain barrier (BBB) to treat brain AChE.

Therefore, we have synthesized and evaluated pro-2-PAM (a lipid permeable 2-PAM derivative) that can enter the brain and reactivate CNS AChE, preventing seizures in guinea pigs after exposure to OPs. The protective effects of the pro-2-PAM after OP exposure were shown using a) surgically-implanted radiotelemetry probes for electroencephalogram (EEG) b) neurohistopathology of brain, c) cholinesterase activities in the PNS and CNS, and d) survivability.

The PNS oxime 2-PAM was ineffective at reducing seizures/status epilepticus (SE) in diisopropyl-fluorophosphate (DFP)-exposed animals. In contrast, pro-2-PAM significantly suppressed and then eliminated seizure activity. In OP-exposed guinea pigs, there was a significant reduction in neurological damage with pro-2-PAM, but not 2-PAM. Distinct regional areas of the brains showed significantly higher AChE activity 1.5 h after OP exposure in pro-2-PAM treated animals compared to the 2-PAM treated ones. However, blood and diaphragm showed similar AChE activities in animals treated with either oxime, as both 2-PAM and pro 2-PAM are PNS active oximes.

In conclusion, pro-2-PAM can cross the BBB, is rapidly metabolized inside the brain to 2-PAM, and protects against OP-induced SE through restoration of brain AChE activity. Pro-2-PAM represents the first non-invasive means of administering a CNS therapeutic for the deleterious effects of OP poisoning by reactivating CNS AChE.

## Factors for determining survival in acute organophosphate poisoning

**Kang E-J, Seok S-J, Lee K-H, Gil H-W, Yang J-O, Lee E-Y, Hong S-Y. Korean J Intern Med 2009; 24: 362-7.**

### *Background/aims*

Organophosphate poisoning has a high mortality rate. Recently, differences among organophosphorus insecticides in human self-poisoning were reported. This study investigated the prognostic risk factors and the mortality of different organophosphates following acute organophosphate poisoning.

### *Methods*

This retrospective study included 68 patients with acute organophosphate poisoning. We investigated patient survival according to initial parameters, including the initial Acute Physiology and Chronic Health Evaluation (APACHE) II score, serum cholinesterase level, and hemoperfusion and evaluated the mortality according to organophosphate types.

### *Results*

Thirteen of the 68 patients died. The agents responsible for mortality were different. The APACHE II score was a significant predictor of mortality (odds ratio [OR], 1.194;  $p < 0.01$ ; 95% confidence interval [CI], 1.089 to 1.309) and respiratory failure (OR, 1.273;  $p < 0.01$ ; 95% CI, 1.122 to 1.444). The mortality was 0% for dichlorvos, malathion, chlorpyrifos and profenofos. However, other organophosphates showed different mortality (16.7% for O-ethyl-O-4-nitrophenyl phenylphosphonothioate, 25% for phenthoate, 37.5% for phosphamidon, 50% for methidathion). The usefulness of hemoperfusion appears to be limited.

### *Conclusions*

The initial APACHE II score is a useful prognostic indicator, and different organophosphates have different mortality.

## Neurobehavioural effects of pesticides with special focus on organophosphorus compounds: which is the real size of the problem?

**Colosio C, Tiramani M, Brambilla G, Colombi A, Moretto A. Neurotoxicology 2009; 30: 1155-61.**

### *Abstract*

The risk of neurobehavioural impairment as a consequence of a prolonged, low dose exposure to neurotoxic pesticides is not clearly demonstrated despite numerous publications addressing the topic.

We reviewed the 24 papers published on human neurobehavioural effects of organophosphorus and/or carbamates pesticides up to May 1st 2008. Variables evaluated were compound/s addressed, number of subjects, approach to measure or estimate exposure, characteristics of control groups and presence of confounders, methodological approach, and type of alteration, taking into account cognitive, sensory-motor, psychological, and psychomotor measures.

A total of 6 papers considered the whole spectrum of functions, the studies yielding positive or uncertain results were 13 (68%) for cognitive function, 11 (69%) for psychomotor function, 11 (65%) for sensory-motor function, and 11 (65%) for psychological function impairment. In 46% of the positive studies a previous severe acute poisoning was reported. Exposure levels were measured only in 5 studies, and very often there were problems in the selection of controls, and firm conclusions on the risk of neurobehavioural effects cannot be reached yet.

The main limits of the available data are: limited number of studies and compounds addressed, significant differences in the approach among studies, poor concordance of the results of different studies, and difficulties in controlling confounding factors. Nevertheless, there are sufficient data to conclude that neurobehavioural impairment might be the consequence of an acute poisoning, and possibly the consequence of relatively high and prolonged exposures.

## **Is there a relationship between the WHO hazard classification of organophosphate pesticide and outcomes in suicidal human poisoning with commercial organophosphate formulations?**

**Peter JV, Jerobin J, Nair A, Bennett A. Regul Toxicol Pharmacol 2010; Online early: doi: 10.1016/j.yrtph.2010.01.004: 1-4.**

### **Abstract**

The WHO classification of pesticides by hazard is based primarily on the acute oral and dermal toxicity to rats. In several Asian countries there is no legislation against the sale of Class I insecticides.

We evaluated if there was an association between the WHO hazard Class I, II or III organophosphate compound and outcomes in human poisoning. Two-hundred and fifty-one patients with mean (SD) age of 30.4 (11.8) years, admitted with symptomatic poisoning and treated with atropine and supportive care, were followed up until death or hospital discharge.

The admission pseudocholinesterase level of 818.8 (1368) IU/L indicated significant suppression of cholinesterase activity. Class I compounds were ingested by 126, Class II by 113 and Class III by 12 patients. The hospital mortality rate was 16.7%, 5.3% and 0% with Class I, II and III organophosphate compounds, respectively ( $P = 0.01$ ). Ventilatory requirements were higher with Class I compared with Class II poisoning (77.0% vs. 54.9%,  $P < 0.001$ ). Patients with Class I poisoning needed mechanical ventilation for a longer period (10.55 (7.4) vs. 7.0 (5.2) days,  $P = 0.002$ ).

The linear relationship between the WHO hazard class and mortality in acute organophosphate poisoning mandates the restriction of the sale of organophosphate compounds associated with higher lethality amongst humans

## **Aspects of *Datura* poisoning and treatment**

**Krenzelok EP. Clin Toxicol 2010; 48: 104-10.**

### **Introduction**

*Datura* species, especially *Datura stramonium* (e.g., jimsonweed), are the focus of scores of case reports that chronicle the toxidrome of anticholinergic toxicity.

### **Mechanisms of toxicity**

Toxicity occurs because of the presence of up to 28 belladonna alkaloids, predominated by atropine and scopolamine. There are significant interspecies differences in the ratio of the belladonna alkaloids, atropine to scopolamine, and the presence of at least 26 other related alkaloids will vary, even between and among specimens of the same species. The differences may account for unexpected dose-response effects that are observed in some patients and even different profiles of toxicity. All parts of the *Datura* species contain belladonna alkaloids; in decreasing order, the belladonna alkaloid content is generally the greatest in the petioles (flowers), stem, fruit (seeds), leaves, and roots.

### **Features**

The most prominent symptoms are due to the blockade of peripheral muscarinic receptors that innervate exocrine glands, smooth muscle, and cardiac tissue. Therefore, the primary toxic manifestations include mydriasis, which is due to the blockade of papillary sphincter muscle and iris muscle; dry mouth, secondary to parasympathetic blockade of salivary secretion; tachycardia, caused by competition at muscarinic receptors in postganglionic parasympathetic neurons and

blockade of receptors in the SA node; and fever and erythema, because of vasodilation and inhibition of sweating.

### ***Direct ocular exposure***

Well known to produce mydriasis after ingestion, *Datura* is also notorious for producing maladies such as "gardeners eye" and "cornpickers eye," which is a testament to the high concentrations of belladonna alkaloids in the entire plant. When the sap or dried plant material from a member of the *Datura* species enters the eye directly, the papillary sphincter and ciliary muscles are affected resulting in mydriasis and cycloplegia. Pupil dilation is sudden in onset and often profound. In a dose-response fashion, the mydriasis and its persistence will be dependent on the alkaloidal content and quantity of the sap. The mydriasis can be bilateral, but most commonly it is unilateral, which is frightening to the victim and to the clinician and suggestive of significant cerebral pathology.

### ***Management***

As with any toxic exposure, the foundation of patient management is supportive care and patient reassurance. Because of the central nervous system effects of the belladonna alkaloids, the patient may be agitated, combative, confused, and disoriented. Initial intervention focuses on addressing those issues that protect the patient and their caregivers. In severe cases, physostigmine, a cholinesterase inhibitor, should be used to reverse anticholinergic toxicity. Physostigmine should be given intravenously to an adult in a dose of 0.5-2.0 mg at a rate of no more than 1 mg/min; a second dose may be administered if necessary. Children should receive 0.02 mg/kg intravenously and the rate should not exceed 0.5 mg/min. Extracorporeal elimination and forced diuresis of the belladonna alkaloids are not viable options.

### ***Conclusions***

Members of the *Datura* species contain abundant amounts of belladonna alkaloids that can produce both local and systemic anticholinergic toxicity. Fatalities because of *Datura* species exposure are rare, but adverse effects are very common. Treatment is supportive with the use of physostigmine in more severe cases of poisoning.

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## CHEMICAL WARFARE, BIOLOGICAL WARFARE AND RIOT CONTROL AGENTS

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