Is oxygen required before atropine administration in organophosphorus or carbamate pesticide poisoning? – A cohort study


Background

Early and adequate atropine administration in organophosphorus (OP) or carbamate insecticide poisoning improves outcome. However, some authors advise that oxygen must be given before atropine due to the risk of inducing ventricular dysrhythmias in hypoxic patients. Because oxygen is frequently unavailable in district hospitals of rural Asia, where the majority of patients with insecticide poisoning present, this guidance has significant implications for patient care. The published evidence for this advice is weak. We therefore performed a patient cohort analysis to look for early cardiac deaths in patients poisoned by anticholinesterase pesticides.

Methods

We analysed a prospective Sri Lankan cohort of OP or carbamate-poisoned patients treated with early atropine without the benefit of oxygen for evidence of early deaths. The incidence of fatal primary cardiac arrests within 3 h of admission was used as a sensitive (but non-specific) marker of possible ventricular dysrhythmias.

Results

The cohort consisted of 1957 patients. The incidence of a primary cardiac death within 3 h of atropine administration was 4 (0.2%) of 1957 patients. The majority of deaths occurred at a later time point from respiratory complications of poisoning.

Conclusion

We found no evidence of a high number of early deaths in an observational study of 1957 patients routinely given atropine before oxygen that might support guidance that
oxygen must be given before atropine. The published literature indicates that early and rapid administration of atropine during resuscitation is life-saving. Therefore, whether oxygen is available or not, early atropinisation of OP- and carbamate-poisoned patients should be performed.

Full text available from: [http://dx.doi.org/10.3109/15563650.2014.915411](http://dx.doi.org/10.3109/15563650.2014.915411)

**National toxicovigilance for pesticide exposures resulting in health care contact – An example from the UK’s National Poisons Information Service**


**Background**

Although there are extensive systems in place for pharmacovigilance, similar systems for detecting adverse health effects relating to pesticide exposure are rare. In 2004, the National Poisons Information Service (NPIS) pesticide surveillance study was implemented to identify cases requiring health care contact in the UK. This report describes the epidemiology of pesticide exposures reported to poison centres in the UK over a 9-year period.

**Methods**

Data on exposures were gathered through monitoring access to the NPIS's online clinical toxicology database TOXBASE® and through monitoring calls to the four NPIS units (Edinburgh, Cardiff, Newcastle and Birmingham). Severity was judged by both caller and NPIS staff.

**Results**

During the 9 years, 34,092 enquiries concerning pesticides were recorded; 7,804 cases of pesticide exposure were derived from these enquiries. Exposures were predominantly unintentional and acute (6,789; 87.0%); 217 (2.8%) and 755 (9.7%) were chronic unintentional and acute deliberate self-harm exposures, respectively. The majority of cases occurred in children, especially the 0-4 year age group. The minimum incidence of pesticide exposure requiring health care contact was 2.0 cases/100,000 population per year. Reported numbers were 6- to 25-fold greater than those picked up through other UK pesticide toxicovigilance schemes. There were 81 cases of severe toxicity and 38 cases of fatal exposure. Deliberate self-harm accounted for 62.3% of severe cases and 79% of deaths. Aluminium phosphide, paraquat, diquat and glyphosate were responsible for most severe and fatal cases.

**Conclusions**

The data gathered from this pesticide surveillance study indicate that poison centre resources can usefully monitor pesticide exposures resulting in health care contact in the UK. The NPIS may usefully be one component of the UK's response to European legislation requiring surveillance of complications resulting from pesticide use.

Full text available from: [http://dx.doi.org/10.3109/15563650.2014.908203](http://dx.doi.org/10.3109/15563650.2014.908203)
Organophosphate-pyrethroid combination pesticides may be associated with increased toxicity in human poisoning compared to either pesticide alone


**Background**

Organophosphate (OP) poisoning results in significant toxicity while pyrethroid poisoning is associated with extremely low fatality. OPs can inhibit the detoxification of pyrethroid and increase the toxicity of the combination. We assessed whether mixed OP-pyrethroid poisoning impacted outcome in human poisoning.

**Methods**

Patients were identified from a prospectively collected institutional poisoning database that incorporates demographic and outcome data of patients presenting with poisoning.

**Results**

Of the 1177 poisoned patients admitted over 2 years, 32 presented with OP-pyrethroid (50% chlorpyrifos-5% cypermethrin mixture) poisoning (Group 1), 26 consumed 20% chlorpyrifos (Group 2), and 32 took 15% cypermethrin (Group 3). Seizures occurred in 15.6% (n = 5) with chlorpyrifos-cypermethrin poisoning, 18.8% (n = 6) with cypermethrin poisoning, and 3.9% (n = 1) with chlorpyrifos poisoning. Ventilatory requirements were 53.5% (17/32), 42.3% (11/26), and 15.7% (5/32) in Groups 1–3, respectively. Ventilator-free days (Mean ± SD) was significantly lower (p < 0.006) in Group 1 (20.9 ± 9.3 days) than those in Group 2 (26.1 ± 4.4 days) or 3 (27.8 ± 0.6). The median (inter-quartile range) hospital stay was 5.5 (4–19.5), 5 (5–6), and 1 (0.65–1.5) days, respectively, in the three groups. Four patients died in Group 1 (13%). None died in the other groups.

**Conclusion**

Although confounded by the varying quantity of chlorpyrifos and cypermethrin in the different formulations, patients with mixed poisoning appear to have shorter ventilator-free days than patients poisoned by either of the pesticides alone. Further studies are required comparing patients poisoned by formulations with similar quantities of OP and pyrethroid or with analysis of blood pesticide concentration on admission.

Full text available from: [http://dx.doi.org/10.3109/15563650.2014.909933](http://dx.doi.org/10.3109/15563650.2014.909933)

Isopropanol poisoning


**Introduction**

Isopropanol is a clear, colorless liquid with a fruity odor and a mild bitter taste. Most commonly found domestically as rubbing alcohol, isopropanol is also found in numerous household and commercial products including cleaners, disinfectants, antifreezes, cosmetics, solvents, inks, and pharmaceuticals.

**Aim**

The aim of this review is to critically review the epidemiology, toxicokinetics, mechanisms of toxicity, clinical features, diagnosis, and management of isopropanol poisoning.

**Methods**

OVID MEDLINE and ISI Web of Science were searched to November 2013 using the words "isopropanol", "isopropyl alcohol", "2-propanol", "propan-2-ol", and "rubbing alcohol"
combined with the keywords "poisoning", "poison", "toxicity", "ingestion", "adverse effects", "overdose", or "intoxication". These searches identified 232 citations, which were then screened via their abstract to identify relevant articles referring specifically to the epidemiology, toxicokinetics, mechanisms of toxicity, clinical features, diagnosis, and management of isopropanol poisoning; 102 were relevant. Further information was obtained from book chapters, relevant news reports, and internet resources. These additional searches produced eight non-duplicate relevant citations.

Epidemiology
The majority of isopropanol exposures are unintentional and occur in children less than 6 years of age. Although isopropanol poisoning appears to be a reasonably common occurrence, deaths are rare.

Toxicokinetics
Isopropanol is rapidly absorbed following ingestion with peak plasma concentrations occurring within 30 min. It can also be absorbed following inhalation or dermal exposure. Isopropanol is widely distributed with a volume of distribution of 0.45–0.55 L/kg. Isopropanol is metabolized by alcohol dehydrogenase to acetone, acetaldehyde, and methylglyoxal, propylene glycol, acetate, and formate, with conversion of these metabolites to glucose and other products of intermediary metabolism. The elimination of isopropanol is predominantly renal, though some pulmonary excretion of isopropanol and acetone occurs. In one case 20% of the absorbed dose was eliminated unchanged in urine, with the remainder excreted as acetone and metabolites of acetone. The elimination half-life of isopropanol is between 2.5 and 8.0 h, whereas elimination of acetone is slower with a half-life following isopropanol ingestion of between 7.7 and 27 h.

Mechanisms of toxicity
While the exact mechanism of action of isopropanol has not been fully elucidated, brain stem depression is thought to be the predominant mechanism. While the clinical effects are thought to be mostly due to isopropanol, acetone may also contribute.

Clinical features
The major features of severe poisoning are due to CNS and respiratory depression, shock, and circulatory collapse. The most common metabolic effects are an increased osmol (osmolal) gap, ketonemia, and ketonuria.

Diagnosis
Poisoning can be diagnosed using the measurement of isopropanol serum concentrations, though these may not be readily available. Diagnosis is therefore more typically made on the basis of the patient's history and clinical presentation. An osmol gap, ketonemia, and/or ketonuria without metabolic acidosis, along with a fruity or sweet odor on the breath and CNS depression support the diagnosis.

Management
Supportive care is the mainstay of management with primary emphasis on respiratory and cardiovascular support. Hemodialysis enhances elimination of isopropanol and acetone and should be considered in very severe poisoning.

Conclusions
Severe isopropanol poisoning results in CNS and respiratory depression and circulatory collapse. Treatment primarily consists of symptom-directed supportive care. Although hemodialysis increases the elimination of isopropanol and acetone substantially, it should only be considered in severe life-threatening poisonings. Patients usually make a full recovery provided they receive prompt supportive care.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.914527
Poisonings requiring admission to the pediatric intensive care unit: a 5-year review

Background
Poisonings represent a significant number of preventable admissions to the pediatric intensive care unit (PICU), but data about poisonings requiring PICU-level care are limited.

Objectives
To identify the demographics of patients admitted with poisonings and characterize their clinical courses related to their poisoning.

Methods
All poisonings over a 5-year period (2008–2012) at an academic medical center in New England were retrospectively reviewed using electronic medical records in an observational case series. Poisonings were identified using key search terms within an admissions database.

Results
There were 273 admissions for poisonings, which represent 8% of total PICU admissions over this time period. The poisonings were unintentional in 148 (54%) cases and intentional in 125 (46%). The vast majority of poisonings occurred in patients either 3 years or below (N = 121, 44%) or 13 years or above (N = 124, 45%). Most (96%) admissions were for less than 48 h and 41% were for less than 24 h. Mean PICU length of stay was 1.2 + 0.7 days. A total of 468 substances were ingested in 54 different drug classes, with analgesics and antidepressants being the most common. Eighty-five (31%) poisonings were polypharmaceutical. The most commonly used therapies were naloxone, activated charcoal, and benzodiazepines. Twenty-seven patients (10%) received mechanical ventilation. There was one fatality, an adolescent with a polypharmacy overdose in a suicide attempt.

Conclusion
Pediatric poisonings are a significant percentage of admissions to the PICU. The majority of poisonings are non-fatal, require supportive care, close monitoring, and some specific treatment. Drug classes causing poisonings have changed to a higher percentage of opioids in younger patients and atypical antidepressants in adolescents.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.909601

Epidemiological trends in electronic cigarette exposures reported to U.S. Poison Centers

Context
The Centers for Disease Control and Prevention (CDC) has reported an increase in electronic cigarette (e-cigarette) use in both adults and adolescents. Poison Center calls provide data on exposures pertaining to e-cigarette devices and components (including nicotine-refill cartridges), potentially identifying epidemiological trends in reported exposures over time.

Objective
To characterize the trends in e-cigarette exposures reported to United States (U.S.) Poison Centers between 01 June 2010 and 30 September 2013.
Methods
We obtained data from the American Association of Poison Control Centers (AAPCC) for all exposures involving e-cigarettes reported to the National Poison Data System (NPDS) by U.S. Poison Centers and described trends in exposures over time, demographics, geographical characteristics, clinical effects and outcomes, management site, and exposure route.

Results
A total of 1,700 exposures were reported to Poison Centers during this time. The most frequent age groups were children 5 years or below with 717 (42.2%) exposures and adults ages 20–39 years with 466 (27.4%) exposures. Temporal trends showed an increase of 1.36 exposures per month [95% CI: 1.16–1.56] from June 2010 through December 2012, after which exposures increased by 9.60 per month [95% CI: 8.64–10.55] from January through September 2013. The majority of patients who were followed reported that they had only minor effects.

Conclusions
The majority of exposures to e-cigarette devices and components occurred in children of 5 years or below due to accidental exposure. Based on the available data, the reported exposures have resulted in minimal toxicity. Calls to Poison Centers regarding these products have rapidly increased since 2010, and continued surveillance may show changes in the epidemiological trends surrounding e-cigarette exposures.

Electronic cigarettes: another pediatric toxic hazard in the home?
Lowry JA. Clin Toxicol 2014; online early:
doi: 10.3109/15563650.2014.918998:

Electronic cigarettes (e-cigarettes) have recently gained attention in the media as an alternative to traditional smoking due to its increased use and lack of regulation. Originally marketed as a tobacco reduction or smoking cessation product, recreational use of e-cigarettes in adolescents and adults has doubled from 2010 to 2012. Current use in Great Britain rose from 2.7% of adult smokers studied in 2010 to 6.7% in 2012. Alternatively, adolescent data reveal that 9.3% of ever cigarette users had reported never smoking conventional cigarettes. This trend in increased use has been associated with increased calls to the U.S. Poison Control Centers as evident in the article by Vakkalanka et al. in this issue. In their study, unintentional exposures in children less than 6 years of age accounted for the most calls to poison control centers compared with other age groups. While the majority of patients followed had no more than minor effects reported, moderate, and major effects were found in this study. One fatality was reported, and, unfortunately, the clinical effects were not accounted for this age. Additionally, poison center data are unable to verify dose resulting in an incomplete account for the seriousness of these exposures. Parents and clinicians should be alarmed at these numbers and recognize the potential risk of harm to children and adolescents.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.918998
Evaluation of dexmedetomidine therapy for sedation in patients with toxicological events at an academic medical center

Introduction
Although clinical use of dexmedetomidine (DEX), an alpha2-adrenergic receptor agonist, has increased, its role in patients admitted to intensive care units secondary to toxicological sequelae has not been well established.

Objectives
The primary objective of this study was to describe clinical and adverse effects observed in poisoned patients receiving DEX for sedation.

Methods
This was an observational case series with retrospective chart review of poisoned patients who received DEX for sedation at an academic medical center. The primary endpoint was incidence of adverse effects of DEX therapy including bradycardia, hypotension, seizures, and arrhythmias. For comparison, vital signs were collected hourly for the 5 h preceding the DEX therapy and every hour during DEX therapy until the therapy ended. Additional endpoints included therapy duration; time within target Richmond Agitation Sedation Score (RASS); and concomitant sedation, analgesia, and vasopressor requirements.

Results
Twenty-two patients were included. Median initial and median DEX infusion rates were similar to the commonly used rates for sedation. Median heart rate was lower during the therapy (82 vs. 93 beats/minute, \( p < 0.05 \)). Median systolic blood pressure before and during therapy was similar (111 vs. 109 mmHg, \( p = 0.745 \)). Five patients experienced an adverse effect per study definitions during therapy. No additional adverse effects were noted. Median time within target RASS and duration of therapy was 6.5 and 44.5 h, respectively. Seventeen patients (77%) had concomitant use of other sedation and/or analgesia with four (23%) of these patients requiring additional agents after DEX initiation. Seven patients (32%) had concomitant vasopressor support with four (57%) of these patients requiring vasopressor support after DEX initiation.

Conclusion
Common adverse effects of DEX were noted in this study. The requirement for vasopressor support during therapy warrants further investigation into the safety of DEX in poisoned patients. Larger, comparative studies need to be performed before the use of DEX can be routinely recommended in poisoned patients.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.913175

Pulmonary toxicity following exposure to a tile coating product containing alkylsiloxanes. A clinical and toxicological evaluation

Context
Coating products are widely used for making surfaces water and dirt repellent. However, on several occasions the use of these products has been associated with lung toxicity.
**Objective**
In the present study, we evaluated the toxic effects of an aerosolized tile-coating product.

**Methods**
Thirty-nine persons, who reported respiratory and systemic symptoms following exposure to the tile-coating product, were clinically examined. The product was analysed chemically and furthermore, the exposure scenario was reconstructed using a climate chamber and the toxicological properties of the product were studied using *in vivo* and by *in vitro* surfactometry.

**Results**
The symptoms developed within few hours and included coughing, tachypnoea, chest pain, general malaise and fever. The physical examination revealed perihilar lung infiltrates on chest radiograph and reduced blood oxygen saturation. The acute symptoms resolved gradually within 1–3 days and no delayed symptoms were observed. By means of mass spectrometry and X-ray spectroscopy, it was shown that the product contained non-fluorinated alkylsiloxanes. The exposure conditions in the supermarket were reconstructed under controlled conditions in a climate chamber and particle and gas exposure levels were monitored over time allowing estimation of human exposure levels. Mice exposed to the product developed symptoms of acute pulmonary toxicity in a concentration-and time-dependent manner. The symptoms of acute pulmonary toxicity likely resulted from inhibition of the pulmonary surfactant function as demonstrated by *in vitro* surfactometry. Among these patients only a partial association between the level of exposure and the degree of respiratory symptoms was observed, which could be because of a high inter-individual difference in sensitivity and time-dependent changes in the chemical composition of the aerosol.

**Conclusion**
Workers need to cautiously apply surface coating products because the contents can be highly toxic through inhalation, and the aerosols can disperse to locations remote from the worksite and affect bystanders.

Full text available from: [http://dx.doi.org/10.3109/15563650.2014.915412](http://dx.doi.org/10.3109/15563650.2014.915412)

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**Exhaust fumes**


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**Acrylamide**


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Alcohol (ethanol)

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METALS
General


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Arsenic


**Arsenic**


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