For the full versions of these articles see bmj.com

CLINICAL REVIEW

Managing acute organophosphorus pesticide poisoning

Darren M Roberts,[1] Cynthia K Aaron[2]

[1]South Asian Clinical Toxicology Research Collaboration, Australian National University

[2]Regional Poison Center, Children's Hospital of Michigan, Detroit, MI 48201, USA

Correspondence to: C K Aaron
caaron@dmc.org

BMJ 2007;334:629-34
doi: 10.1136/bmj.39134.566979.BE

Reviewers' comments and authors' responses are on bmj.com

This article is an abridged version; the full version is on bmj.com: doi=10.1136/bmj.39134.566979.BE.

Organophosphorus pesticides are used widely for agriculture, vector control, and domestic purposes. Despite the apparent benefits of these uses acute organophosphorus pesticide poisoning is an increasing worldwide problem, particularly in rural areas. Organophosphorus pesticides are the most important cause of severe toxicity and death from acute poisoning worldwide, with more than 200 000 deaths each year in developing countries.[1] Although the incidence of severe acute organophosphorus pesticide poisoning is much less in developed countries, many patients with low dose acute unintentional or occupational exposures present to health facilities.[2 3] We provide an evidence based review of the management of acute organophosphorus pesticide poisoning.

Box 1 | Sources of organophosphorus pesticides

Domestic

- Garden sheds—in particular insecticidal preparations but also other products that are marketed as fertilisers but contain some organophosphorus pesticides, available as solid or liquid formulations
- Surface and room sprays
- Baits for cockroaches and other insects (for example, chlorpyrifos)
- Shampoos against head lice (for example, malathion)
- Pet preparations (for example, pet washes, collars)

Industrial or occupational

- Crop protection and livestock dipping
- Large scale internal control, including fumigation

Terrorism or warfare (nerve agents)

Sarin, for example, was used in the Tokyo subway attack, and both tabun and sarin were used during the Iraq-Iran conflict. Although nerve agents share a similar mechanism of toxicity with organophosphorus pesticides, their treatment is a specialised topic and not dealt with in this review

Why do I need to know about acute organophosphorus poisoning?

Household and agricultural products containing organophosphorus pesticides are prevalent (box 1), allowing many opportunities for acute poisoning. Since the correlation between intent, dose, and severity of toxicity after acute poisoning is poor, each exposure requires a thorough review.[2] A structured approach to risk assessment of exposed patients is necessary.[5] Long term complications occasionally occur so a rigorous assessment is required given potential legal implications in unintentional, criminal, or occupational exposures.

Patients with moderate to severe organophosphorus pesticide poisoning usually require management in an intensive care unit.[2 6 7] Mortality from severe poisoning is high (10%)[1] compared with the overall mortality from pharmaceuticals (0.5%).[w1] Current evidence suggests that prompt and appropriate management optimises outcomes (LB).

What is the pathophysiology of acute organophosphorus poisoning?

The effects of organophosphorus compounds on human physiology are multiple and complex. Organophosphorus compounds inhibit numerous enzymes, of which esterases seem to be the most clinically important. Inhibition of acetylcholinesterase leads to the accumulation of acetylcholine at cholinergic synapses, interfering with normal function of the autonomic, somatic, and central nervous systems. This produces a range of clinical manifestations, known as the acute cholinergic crisis (box 2).[8 9] The organophosphorus-esterase complex subsequently undergoes multiple spontaneous reactions (fig 1).

Although clinicians commonly think of organophosphorus compounds as an interchangeable class, noticeable differences are observed between them in the clinical manifestations of acute poisoning.[8 9 11-13] This may result from differences in pharmacokinetics,[8 13 14] additional mechanisms of toxicity such as oxidative stress,[w3] dynamic physiological adaptations after prolonged stimulation,[8 12 14] differences between patients,[13 15] or potency of enzyme inhibition.[8 16]

How is organophosphorus poisoning diagnosed?

A history of acute exposure to an organophosphorus pesticide and development of characteristic clinical effects (box 2) is diagnostic of organophosphorus poisoning. When the history is not forthcoming, however, the differential diagnosis is broad and may

Sources and selection criteria

We searched several resources to identify relevant information on the diagnosis and management of acute organophosphorus poisoning: Medline, Embase, the Cochrane Library, and the Chemical Safety Information for Intergovernmental Organizations database (www.inchem.org/pages/pds.html); websites for registration of clinical trials, including the Current Controlled trials website (http://controlled-trials.com/) using the *m*RCT search feature; personal archives; and attendance at, and review of abstracts from, workshops and conferences on pesticide poisoning.

Levels of evidence in the review

The evidence supporting specific therapeutic approaches to patients with acute organophosphorus poisoning is listed after each management recommendation. We have adopted the classification used in the BMJ publication *Clinical Evidence*[6]:

- Beneficial (B)
- Likely to be beneficial (LB)
- Trade-off between benefits and harms (TO)
- Unknown effectiveness (UE)
- Unlikely to be beneficial (UB)
- Likely to be ineffective or harmful (LIH)

include intoxication with carbamates, other poisons, or pontine haemorrhage. Therefore a good history, high index of suspicion, and a detailed clinical examination are essential. Although there are differences in the manifestations of individual organophosphorus compounds,[13] the mnemonic DUMBELS and other clinical features described in box 2 are useful prompts for the various signs and symptoms that should be considered. Onset of clinical toxicity is variable; however most patients who will develop severe toxicity usually have symptoms within six hours. Patients remaining asymptomatic for 12 hours after ingestion are unlikely to develop major clinical toxicity.[2 9] Exceptions exist with some highly lipophilic organophosphorus compounds (most importantly fenthion), which produce only subtle cholinergic features initially then progressive muscle weakness, including respiratory failure requiring intubation for several days.[6 12 13]

When the diagnosis is in question or there is doubt about the significance of an organophosphorus exposure, quantification of butyrylcholinesterase or acetylcholinesterase activity is helpful. Cholinesterase activity that is less than 80% of the lower reference range is probably indicative of a significant exposure to an organophosphorus compound [w3]; with severe clinical toxicity, the erythrocyte acetylcholinesterase activity is less than 20% of normal.[9 14 18] Butyrylcholinesterase has no relation to the severity of clinical toxicity.[14 17 19] Several methods for classifying the severity of acute organophosphorus poisoning have been developed using clinical data or laboratory investigations, but to date few of these methods have been validated or widely adopted. Figure 2 shows a simplified method for considering the severity of organophosphorus poisoning. This classification is intended to guide clinical management rather than to be used for prognostication and is based primarily on clinical variables so it can be used in resource poor environments with limited access to clinical investigations.

What is the initial management of patients with acute organophosphorus poisoning?

The initial management of acute exposures to organophosphorus is immediate assessment and management of disturbances in airway, breathing, and circulation (LB). Further steps in ongoing management are based on risk assessment and observations during continuous clinical monitoring (fig 2[5 7 11]) including the dose ingested, time since ingestion, clinical features, patient factors, and available medical facilities.[5] When antidotal therapy is indicated, it should be given rapidly, as this may be lifesaving (LB). Although the amount ingested according to the history seems to be a poor predictor of the amount absorbed,[15 19] all patients with a history of deliberate ingestion should be initially managed as a severe poisoning. In concert with immediate assessment and resuscitation, all patients should undergo some degree of skin decontamination. Simply removing exposed clothing reduces the risk of toxin exposure in patients and staff.

What antidotes are used in the management of acute organophosphorus poisoning?

The three most widely used classes of antidotes are muscarinic antagonists (usually atropine) (LB), oximes (usually pralidoxime or obidoxime) (UE), and benzodiazepines (LB).[4 7] Atropine is carefully titrated to reverse muscarinic effects (box 3).[4 7] Atropine has no effect on the neuromuscular junction and muscle

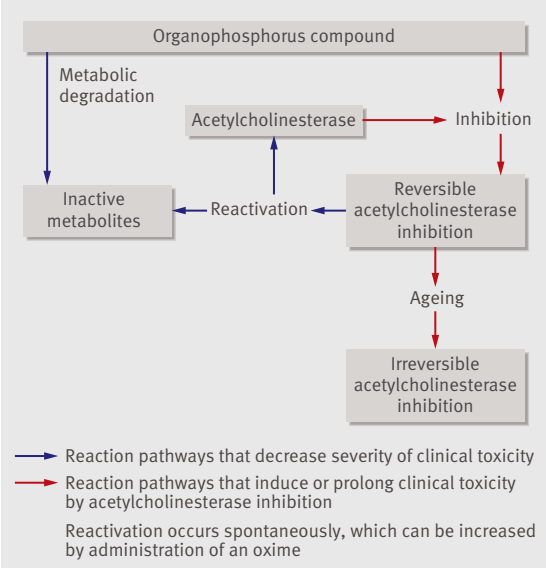

Fig 1 | Interactions of organophosphorus compounds in vivo; relative rates of each reaction vary between organophosphorus compounds[8]

Suggested symptom based treatment recommendations for organophosphorus poisoning

Sign or symptom	Recommended therapy
Excessive salivation, lacrimation, nausea and vomiting, diarrhoea	Atropine, glycopyrrolate
Bronchorrhoea, bronchospasm	Atropine, ipratropium, glycopyrrolate
Hypotension	Fluids, atropine, vasopressors, inotropes
Bradycardia	Atropine, glycopyrrolate
Eye pain	Mydriatics, cycloplegics
Muscle weakness	Oximes
Respiratory failure	Intubation and ventilation, oximes
Seizures	Benzodiazepines

weakness so oximes are used clinically to reverse neuromuscular blockade by reactivating the inhibited acetylcholinesterase before ageing. Oximes should be given as early as possible to limit the degree of ageing (fig 1).[8] The evidence supporting the efficacy of oximes and the dosing regimen is limited (UE).[20] A recent randomised controlled trial using pralidoxime iodide concluded that high doses were more effective than a lower dose although further studies were recommended.[21] Despite limitations in the current data and based on clinical experience, we recommend oxime use in patients with moderate to severe organophosphorus poisoning (UE)[4 8] and benzodiazepines for patients with agitation and seizures (LB).[4] The table lists organophosphorus triggered signs and symptoms and some suggested therapies in conjunction with antidotal treatment (also see box 3 and figure 2).

Other antidotes and treatments have been proposed for acute organophosphorus poisoning, but data supporting their efficacy are currently too limited to recommend their routine use.[4] Several clinical trials are in progress in Asia to tackle the limited evidence on the efficacy of treatments for organophosphorus poisoning.[22]

Auto-injectors, developed for use in exposures to organophosphorus nerve agents, are available with a fixed dose of muscarinic antagonist and oxime. The role for these auto-injectors in the management of acute organophosphorus pesticide poisoning is limited, but they can be utilised when other forms of antidotes are not available. Clinical experience suggests that the dose of atropine must be carefully titrated, and doses higher than those included in these formulations are usually required.[7] HI-6 is an oxime that is increasingly being included in auto-injectors because it is a more effective reactivator of acetylcholinesterase inhibited by nerve agents compared with pralidoxime and obidoxime.

What considerations are needed for patients with mild or no clinical toxicity, including skin exposures?

Patients who present with a history of unintentional poisoning who are asymptomatic or have mild symptoms, often do not require hospital admission (fig 2). Management priorities for these patients are rapid triage, a detailed risk assessment, and consideration of forensic implications. If the exposure is considered trivial, the patient does not need medical review and can be observed at home or in the workplace. Other patients should be decontaminated and monitored clinically for a minimum of 6-12 hours. If possible, cholinesterase activity should be measured to confirm whether the exposure is significant. A normal cholinesterase activity six hours after exposure may be sufficient to exclude a major ingestion, although this approach has not been sufficiently assessed.

Patients with a single acute skin exposure rarely develop major clinical effects and probably do not require medical assessment. Volunteer studies document the risk of a skin exposure leading to significant clinical toxicity to be far below that of ingestion. Although the rate of organophosphorus absorption across the skin is slower than across the gut, patients who are asymptomatic at 12 hours are unlikely to develop toxicity. Such patients should be given instructions to present for medical review if there is a noticeable worsening of signs and symptoms. If there is concern about a skin exposure, testing for changes in cholinesterase activity is recommended.

Box 2 | Clinical features of acute organophosphorus poisoning[8-10]

Acute cholinergic crisis

The acute cholinergic crisis is caused by the accumulation of acetylcholine at cholinergic synapses. The particular clinical features depends on the type of receptors stimulated and their location:

- Muscarinic receptors: diarrhoea, urinary frequency, miosis, bradycardia, bronchorrhoea and bronchoconstriction, emesis, lacrimation, salivation (DUMBELS), and hypotension. Cardiac arrhythmias have also been reported
- Nicotinic receptors: fasciculations and muscle weakness, which may progress to paralysis and respiratory failure,* mydriasis, tachycardia, and hypertension
- Central nervous system: altered level of consciousness, respiratory failure,* and seizures; the relative contribution of cholinergic and other neurotransmitters is not well characterised

*Respiratory failure occurs as a result of centrally or peripherally mediated mechanisms. It may manifest either during the acute cholinergic crisis (type I paralysis) or during an apparent recovery phase (intermediate syndrome, or type II paralysis). Weakness of neck flexors is an early sign of significant muscle weakness and may be useful for predicting the onset of respiratory failure.[11 12]

Organophosphorus induced delayed polyneuropathy

Unrelated to acetylcholinesterase inhibition, this occurs because of inhibition of other enzymes, in particular neurotoxic target esterase.
Organophosphorus induced delayed polyneuropathy is characterised by demyelination of long nerves, when neurological dysfunction occurs 1-3 weeks after an acute exposure, particularly motor dysfunction but also sensory dysfunction, which may be chronic or recurrent

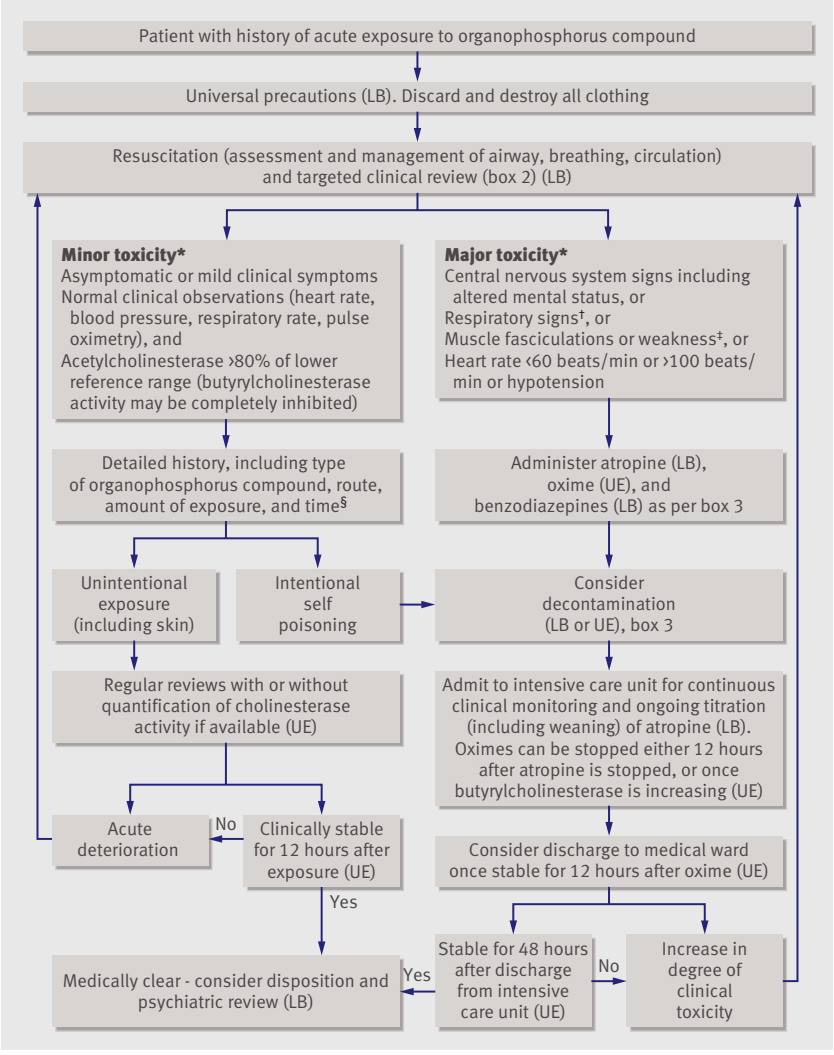

Fig 2 | **Decision tree for management of patients presenting with history of acute organophosphorus poisoning[5 7-9 14] *Patients may have variable degrees of miosis, salivation, diaphoresis, urinary frequency, or lacrimation, which may assist in diagnosis of organophosphorus poisoning. Because these manifestations are not considered to influence outcome they are not included in this decision tree. †Assessment of respiratory status includes respiratory rate and depth, presence of adventitious sounds such as rales and rhonchi, presence of bronchorrhoea, and objective measurements of pulse oximetry, arterial blood gases, and forced vital capacity or forced expiratory volume in one second. ‡Muscle weakness: difficulty in mobilisation or reduced forced vital capacity on spirometry before development of paralysis and respiratory failure. §Caution with patients with a history of exposure to fenthion (or highly fat soluble organophosphorus compounds). Patients with fenthion poisonings are usually characterised by minimal or absent cholinergic symptoms for 24-48 hours, after which they develop increasing muscle weakness and respiratory failure**

Box 3 | **Specific treatments for the routine management of acute organophosphorus poisoning[2 4 7 8 w6]**

Atropine (LB)

For poisoning in adults initially give 1-3 mg atropine intravenously (0.02 mg/kg in children). The main end points of atropinisation are a clear chest on auscultation with resolution of bronchorrhea (focal crepitations or wheeze may be noted when there has been pulmonary aspiration) and a heart rate of more than 80 beats/min. If these targets are not achieved by 3-5 minutes, double the intravenous dose. Continue to double the dose and give intravenously every 3-5 minutes until atropinisation has been achieved. Large doses (hundreds of mg) may be required in some patients.

Maintain atropinisation by infusion, starting with 10%-20% of the loading dose every hour. Regular clinical observations are necessary to ensure that atropinisation is achieved without toxicity (delirium, hyperthermia, and ileus)

Oximes (UE)

Several oximes have been developed, but two are more commonly used for treatment of acute organophosphorus poisoning. They are administered as an infusion which should be continued until recovery (12 hours after stopping administration of atropine or once butyrylcholinesterase is noted to increase)

Pralidoxime chloride—loading dose of 30 mg/kg intravenously over 20 minutes, followed by an infusion of 8 mg/kg/h. In adults it is usually given as a 2 g loading dose followed by 500 mg/h. Various salts are available and their dose is determined by converting this dose into equivalent dosing units—for example, 1 g pralidoxime iodide is roughly equal to 650 mg pralidoxime chloride

Obidoxime—loading dose of 4 mg/kg over 20 minutes, followed by an infusion of 0.5 mg/kg/h. In adults it is usually given as 250 mg loading dose followed by 750 mg every 24 hours

Benzodiazepines (LB)

Benzodiazepines are usually given intravenously as required for agitation or seizures—with doses starting at: 5-10 mg diazepam (0.05-0.3 mg/kg/dose), lorazepam 2-4 mg (0.05-0.1 mg/kg/dose), or midazolam 5-10 mg (0.15-0.2 mg/kg/dose)

Decontamination

Dermal spills—wash pesticide spills from the patient with soap and water and remove and discard contaminated clothes, shoes and any other material made from leather (LB)

Gastric lavage—consider for presentations within 1 or 2 hours, when the airway is protected. A single aspiration of the gastric contents may be as useful as lavage (UE)

Activated charcoal without cathartic—50 g may be given orally or nasogastrically to patients who are cooperative or intubated, particularly if they are admitted within one or two hours or have severe toxicity (UE)

What considerations are needed for patients with moderate and severe poisoning?

Patients with moderate or severe organophosphorus poisoning should be admitted to an intensive care unit after resuscitation to allow careful titration of antidotes (fig 2), intubation, ventilation, and inotropes or vasopressors if required.[7] Specialist advice from a clinical or medical toxicologist is recommended; one can be contacted through the local poisons information centre in many regions. Close observations are also required because rapid clinical deterioration and death are reported in patients who seemed to be recovering from the acute cholinergic crisis.[6 11] High quality general medical and nursing care is a priority for patients given that the duration of admission may be long and that secondary complications are an important cause of morbidity and mortality. If the facility is

SUMMARY POINTS

Acute organophosphorus poisoning may induce multisystem toxicity leading to severe toxicity and death

Poisoning is diagnosed on the basis of history and clinical examination; biochemical investigations can have a role for confirming the diagnosis

Management consists of prompt resuscitation, antidotes as required (particularly atropine, oximes, benzodiazepines), and selective decontamination

Ongoing monitoring and high quality supportive care are essential

Healthcare staff treating exposed patients should exercise standard precautions

UNANSWERED RESEARCH QUESTIONS AND ONGOING RESEARCH

Many other treatments have been trialled for use in patients with acute organophosphorus poisoning, but at present high quality data are insufficient to make evidence based recommendations. Examples include[4]:

- Activated charcoal (UE): a randomised controlled trial comparing a single dose or multiple doses of activated charcoal with placebo (ISRCTN02920054) was completed in 2005.[25] The full analysis is expected to be reported in 2007
- Oximes (UE), including both optimal dose and clinical efficacy[20]: randomised controlled trials were recently completed,[21] are in progress (ISRCTN55264358), or are being planned
- α-2 adrenergic receptor agonists—for example, clonidine (UE)
- Butyrylcholinesterase replacement therapy (UE)
- Gastric lavage (UE): a randomised controlled trial comparing single with triple gastric lavage was planned to start in 2006 (ISRCTN24754520)
- Extracorporeal blood purification, including haemodialysis, haemofiltration, and haemoperfusion (UE)
- Magnesium sulphate: ISRCTN50739829 (UE)
- Organophosphorus hydrolases (UE)
- Blood alkalinisation—for example, sodium bicarbonate (UE)

unable to provide this level of care or does not have ready access to antidotes, then the patient should be rapidly transported to a more appropriate healthcare facility by staff able to provide advanced life support. The rate of recovery from severe organophosphorus poisoning, and therefore the duration of stay in intensive care and requirement for antidotes, varies widely depending on the patient, dose and type of organophosphorus pesticide, and provision of advanced supportive care.

What precautions are needed for staff treating patients with acute organophosphorus poisoning?

The risk of nosocomial poisoning to staff and family members exposed to patients with acute organophosphorus poisoning is of concern. Few, if any, cases have been documented where a significant exposure was confirmed. Universal precautions using nitrile gloves are most likely to provide sufficient protection for staff.[23,24] Symptomatic or concerned staff members can be treated as for minor exposure (fig 2).

How does the management of carbamate poisoning differ from that of organophosphorus?

Carbamate pesticides also induce an acute cholinergic crisis, but the inhibited acetylcholinesterase does not age, allowing spontaneous reactivation and restoration of normal nervous function.[14] Carbamates are considered to cause milder poisoning of shorter duration than organophosphorus pesticides. However evidence is mounting that severe toxicity and death occur with some carbamates, in particular carbosulfan and carbofuran. Atropine and benzodiazepines are given in the same manner as for organophosphorus pesticides (fig 2) (LB). Because carbamate inhibited acetylcholinesterase does not age, the role for oximes seems limited, but controversial. It is not unreasonable for oximes to be given to patients with an unknown exposure and evidence of acetylcholinesterase inhibition (UE).

ADDITIONAL EDUCATIONAL RESOURCES

BMJ Clinical Evidence (www.clinicalevidence.com)

Contains information on acute organophosphorus poisoning

Cochrane Collaboration (www.thecochranelibrary.org)

Includes systematic reviews on the management of organophosphorus poisoning

Organophosphorus and carbamate pesticides (www.aic.cuhk.edu.hk/web8/pesticides.htm)

Summarises the mechanism of toxicity and clinical considerations for the treatment of acute organophosphorus pesticide poisoning

Recognition and management of pesticide poisonings (www.npic.orst.edu/RMPP/rmpp_ch4.pdf)

Contains a chapter on organophosphate insecticides

Useful websites

TOXBASE (www.toxbase.org)

May require registration

TOXINZ (www.toxinz.com)

May require registration

HyperTox (www.hypertox.com)

Free trial for 21 days

Information for patients

Medline plus (www.nlm.nih.gov/medlineplus/ency/article/002837)

Discusses clinical effects and management of diazinon poisoning

Medline plus (www.nlm.nih.gov/medlineplus/ency/article/003358.htm)

Provides general information on cholinesterase assays

We thank Nick Buckley for helpful comments. DMR is funded by a scholarship from the National Health and Medical Research Council (Australia). The South Asian Clinical Toxicology Research Collaboration is funded by a Wellcome Trust and National Health and Medical Research Council international collaborative research grant (No GR071669MA).

Contributors: DMR drafted the initial manuscript which was then improved after discussion between DMR and CKA. Both authors are guarantors.
Competing interests: None declared.

1 Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. *Q J Med* 2000;93:715-31.
2 Roberts DM, Fraser JF, Buckley NA, Venkatesh B. Experiences of anticholinesterase pesticide poisonings in an Australian tertiary hospital. *Anaesth Intensive Care* 2005;33:469-76.
3 Lai MW, Klein-Schwartz W, Rodgers GC Jr, Abramsky L, Haber DA, Bronstein AC, et al. 2005 annual report of the American Association of Poison Control Centers' national poisoning and exposure database. *J Toxicol Clin Toxicol* 2006;44:803-932.
4 Eddleston M, Singh S, Buckley NA. Organophosphorus poisoning (acute). *Clin Evid* 2005 Aug;(16):1744-55.
5 Daly FFS, Little M, Murray L. A risk assessment based approach to the management of acute poisoning. *Emerg Med J* 2006;23:396-9.
6 Sungur M, Güven M. Intensive care management of organophosphate insecticide poisoning. *Crit Care* 2001;5:211-5.
7 Eddleston M, Dawson A, Karalliedde L, Dissanayake W, Hittarage A, Azher S, et al. Early management after self-poisoning with an organophosphorus or carbamate pesticide—a treatment protocol for junior doctors. *Critical Care* 2004;8:R391-7.
8 Eyer P. The role of oximes in the management of organophosphorus pesticide poisoning. *Toxicol Rev* 2003;22:165-90.
9 Namba T. Cholinesterase inhibition by organophosphorus compounds and its clinical effects. *Bull World Health Organ* 1971;44:289-307.
10 Lotti M, Moretto A. Organophosphate-induced delayed polyneuropathy. *Toxicol Rev* 2005;24:37-49.
11 Karalliedde L, Baker D, Marrs TC. Organophosphate-induced intermediate syndrome: aetiology and relationships with myopathy. *Toxicol Rev* 2006;25:1-14.
12 Eddleston M, Mohamed F, Davies JOJ, Eyer P, Worek F, Sheriff MHR, et al. Respiratory failure in acute organophosphorus pesticide self-poisoning. *Q J Med* 2006;99:513-22.
13 Eddleston M, Eyer P, Worek F, Mohamed F, Senarathna L, Von Meyer L, et al. Differences between organophosphorus insecticides in human self-poisoning: a prospective cohort study. *Lancet* 2005;366:1452-9.
14 Lotti M. Cholinesterase inhibition: complexities in interpretation. *Clin Chem* 1995;41:1814-8.
15 Eyer F, Meischner V, Kiderlen D, Thiermann H, Worek F, Haberkorn M, et al. Human parathion poisoning: a toxicokinetic analysis. *Toxicol Rev* 2003;22:143-63.
16 Worek F, Diepold C, Eyer P. Dimethylphosphoryl-inhibited human cholinesterases: inhibition, reactivation, and aging kinetics. *Arch Toxicol* 1999;73:7-14.
17 Carlock LL, Chen WL, Gordon EB, Killeen JC, Manley A, Meyer LS, et al. Regulating and assessing risks of cholinesterase-inhibiting pesticides: divergent approaches and interpretations. *J Toxicol Environ Health B Crit Rev* 1999;2:105-60.
18 Thiermann H, Szinicz L, Eyer P, Zilker T, Worek F. Correlation between red blood cell acetylcholinesterase activity and neuromuscular transmission in organophosphate poisoning. *Chem Biol Interact* 2005;157-158:345-7.
19 Nolan RJ, Rick DL, Freshour NL, Saunders JH. Chlorpyrifos: pharmacokinetics in human volunteers. *Toxicol Appl Pharmacol* 1984;73:8-15.
20 Buckley NA, Eddleston M, Szinicz L. Oximes for acute organophosphate pesticide poisoning. *Cochrane Database Syst Rev* 2005 Jan 25;(1):CD005085.
21 Pawar KS, Bhoite RR, Pillay CP, Chavan SC, Malshikare DS, Garad SG. Continuous pralidoxime infusion versus repeated bolus injection to treat organophosphorus pesticide poisoning: a randomised controlled trial. *Lancet* 2006;368:2136-41.
22 Buckley NA, Roberts D, Eddleston M. Overcoming apathy in research on organophosphate poisoning. *BMJ* 2004;329:1231-3.
23 Roberts D, Senarathna L. Secondary contamination in organophosphate poisoning. *Q J Med* 2004;97:697-8.
24 Little M, Murray L. Consensus statement: risk of nosocomial organophosphate poisoning in emergency departments. *Emerg Med Australas* 2004;16:456-8.
25 Eddleston M, Juszczak E, Buckley NA, Senarathna L, Mohamed F, Sheriff MHS, et al. Randomised controlled trial of routine single or multiple dose superactivated charcoal for self-poisoning in a region with high mortality. *Clin Toxicol* 2005;43:442-3.

A spot of bother

That Easter weekend I was on four night shifts. The first night was busy, and I went to sleep early the next day, but typically I was awake again within an hour and a half: a fire alarm had been set off by someone cooking in the hospital accommodation. "Good start," I thought.

I eventually got back to sleep, but when I woke up that evening I felt the familiar scratchy throat and slight ache that generally heralds a viral illness. I went to work hoping it would pass, but, as the evening progressed, I rapidly got worse. My main anxiety was that I had two more nights which would be impossible to cover.

Needless to say, that night was one of the worst I've ever had—culminating in the transfer to Birmingham of a boy who had come in moribund with intussusception and the resuscitation of a hypothermic baby an hour before handover. During this time I'd been popping paracetamol and shivering my way through.

In the morning I was in a bad state, and it was clear that I wouldn't be able to cover my last two nights. My colleague was extremely supportive and told me to go home and leave the rest to her, which I gratefully did.

By this time I was feverish with temperature in the high 39s and a bad sore throat. The following day there was a rash on my forehead and nose, the fevers continued, and I developed sore and ulcerated mucous membranes. The third day, with persisting fever and an exquisitely sore throat that made it impossible to eat or drink, I visited the emergency general practitioner. He gave me amoxicillin, which I gladly took (against my medical common sense).

By the fourth day my eyes were very sore, and my own general practitioner acknowledged that I looked extremely sick and took blood samples. I requested measles serology, as I was becoming suspicious despite having no obvious contacts. My overall condition was not good. I couldn't take anything orally, not even water, without a big dose of Difflam. Later that day, after the rash had spread all over my body and my eyes had become so sore that I couldn't keep them open, the diagnosis seemed certain.

As soon as I suspected measles I informed occupational health, and (as I found out later) chaos ensued. It seemed that I had been everywhere and seen everyone during my infective period, including several oncology patients, sick neonates, and transfers to two tertiary centres. Patients were isolated, colleagues were sent home. Everyone had blood tests, and many received the measles, mumps, and rubella (MMR) vaccine.

It was only when I went to the local chemist to pick up a prescription that I saw the headline in the local paper: "Measles shuts children's ward." I felt guilty for the trouble I was causing and guilty for having so much time off work, and I still felt systemically dreadful. Gradually, however, the symptoms settled, and the diagnosis was confirmed by serology.

The whole experience was hard, but enlightening. As doctors, we feel guilty for being ill and almost feel compelled to soldier on. I was touched by the supportiveness of my colleagues, and I learnt a lot about public health. I am now even more steadfast in my views on the importance of MMR.

Simon Pirie paediatric specialist registrar, Gloucester Royal Hospital
pirie_simon@hotmail.com