



## Inhalant abuse in New Zealand

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**Aim** To describe patterns of inhalant abuse in New Zealand and discuss management.

**Methods** Calls to the National Poisons Centre (NPC) from January 1 2003 to December 31 2004 were analysed. In addition, deaths following inhalational abuse were identified from the Institute of Environmental Science and Research Limited (ESR) database for 2001 and 2002 and available data for 2003.

**Results** Seventy calls were classified as relating to inhalational abuse incidents. In abusers whose age was known, 83% were between 11 and 20 years, and 61% were male. Over half (44/70) of the calls involved abuse of propane or butane, either alone or in combination with a synthetic pyrethroid. ESR coronial data identified 11 inhalant abuse related deaths, most commonly attributed to cardiac effects. 73% of deaths were in teenagers and all but one fatality involved propane and/or butane.

**Conclusions** Inhalant abuse is a persisting problem in New Zealand. NPC and ESR data demonstrate that teenagers are more likely to abuse inhalants than other age groups and butane and propane are the inhalants of choice. Acute management can be difficult, with significant mortality and morbidity. Continued education and other preventive measures are essential to help curb an extremely dangerous practice.

Inhalation of volatile compounds (including adhesives, solvents, fuels, and propellant or flammable gases) is a recognised cause of sudden death among those abusing these substances for “recreational” purposes.<sup>1</sup> Recent Coroners’ inquests into eight deaths related to these substances have been reported by the media, along with an estimate that “...hundreds if not thousands...” may abuse these substances daily.<sup>2</sup>

This study aims to better characterise the inhalant abuse problem in New Zealand, by both reviewing the Coronial Services Office findings to identify deaths, and evaluating records maintained by the New Zealand National Poisons Centre (NZNPC) of enquiries relating to these substances. A review of the toxic mechanisms, health impacts, and management of those affected by inhalant abuse is also undertaken.

### Methods

Calls to the NZNPC over the 2-year period from January 1 2003 to December 31 2004 were analysed. The NZNPC is the sole Poison Control Centre for New Zealand and serves a mixed population of urban and rural areas, covering a population of approximately 4 million people. The data were retrieved from the NZNPC calls collection database. Inclusion criteria were all inhalation exposures where the reason was recorded as intentional abuse. For the purposes of this study, carbon monoxide and nitrous oxide calls were excluded. Data included were the age and sex of the user, date and time of exposure (if acute), its location, substance involved, details of the incident, our assessment, caller background, and regional area where it occurred.

Deaths following intentional inhalational abuse were also sought. Eleven fatalities (see Table 3) were identified from the Institute of Environmental Science and Research Limited (ESR) Chemical Injury Surveillance System database (CISS), which includes data from the national Coronial Services Office.<sup>3</sup> While all Coronial findings for 2001 and 2002 are considered available, findings for 2003 are not, as a full accounting of deaths assessed by Coroners and retrieved by ESR will typically take up to 3 years.

## Results

The NZNPC received 27,020 and 28,357 calls in 2003 and 2004 respectively as shown in Table 1. Of total calls, 4.6% (2003) and 4.4% (2004) were inhalational exposures. Intentional inhalational exposures (excluding nitrous oxide and carbon monoxide) accounted for 70 cases in total over the 2-year period.

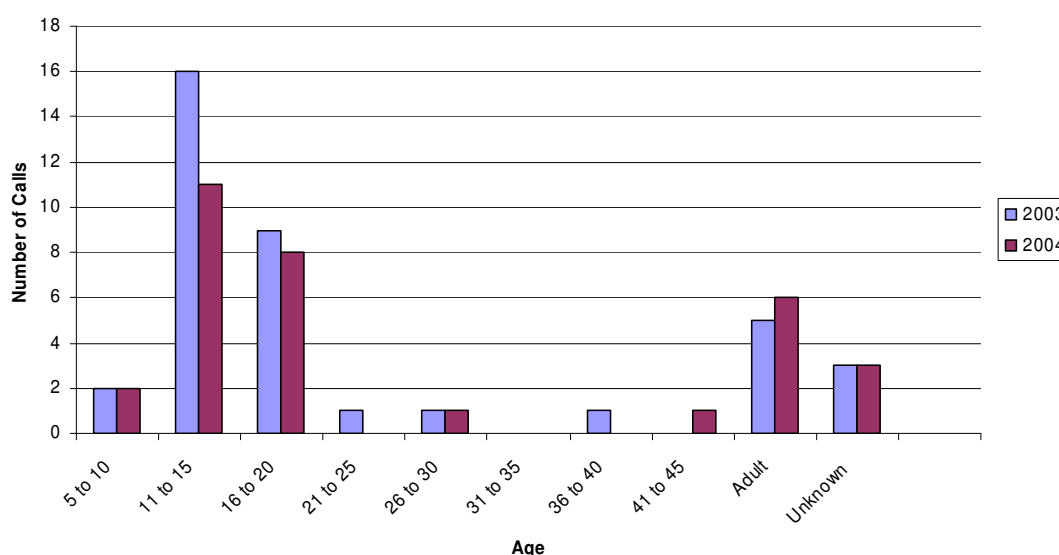
**Table 1. Total calls and inhalational exposure calls to the New Zealand National Poisons Centre (NZNPC)**

Variable	2003	2004
Total calls received by NZNPC	27,020	28,357
Inhalational exposures	1249	1241
Intentional inhalational exposures	77	72
Intentional inhalational exposures (excluding nitrous oxide and CO):	38	32
- Male	24	18
- Female	14	14

CO=carbon monoxide.

The age of users ranged from 7 to 45 years. The age distribution of inhalant abuse calls is shown in Figure 1. The patient was classified as “adult” when the age was not known other than that they were 11 years of age or over. Where age was known, 83% percent of abusers were between the ages of 11 and 20. Forty-three (61%) were male.

**Figure 1. Age of inhalant abusers**



The substance(s) the NZNPC received the most calls about was propane or butane, propellants found in common household products such as air-freshener, body sprays, and fuels in gas heaters (LPG) (as shown in Table 2). Over the 2-year period (2003

and 2004) we received a total of 30 calls in regards to their abuse. In addition, there were 14 calls regarding the abuse of synthetic pyrethroids with butane (fly spray) and 8 calls about the intentional inhalation of petrol. Calls regarding toluene, kerosene, and other hydrocarbons were less frequent.

**Table 2. Substance involved in inhalant abuse calls**

Inhalant	Household product	2003	2004
Propane/butane	Air-freshener, LPG, body sprays	15	15
Synthetic pyrethroid + butane	Fly sprays	10	4
Gasoline	Petrol	5	3
Hydrocarbons (various)	Paint	2	3
Toluene	Adhesives	2	2
Kerosene	Lighter fluid, kerosene	1	2
Trichloroethylene	Correction fluid	1	0
Mineral turpentine		1	0
Methylene chloride	Spray paint	0	1
Not specified		1	2

Deaths from inhalant abuse (as shown in Table 3), as determined by the Coroner, were most often due to cardiac dysrhythmia. In all but one case of death, propane, butane, either alone or in combination with a synthetic pyrethroid was the inhalant involved. In 73% (8/11) of cases, the deceased was a teenager. The majority involved males.

**Table 3. Substance, age of abuser, cause, and year of death due to inhalant abuse**

Inhalant	Age of deceased	Sex	Mode of death	Year
Butane and fly spray	24	M	Cardiac dysrhythmia	2001
Fly spray	18	F	Cardiac dysrhythmia	2001
Butane	16	F	Cardiac dysrhythmia	2001
Toluene	36	M	Not stated	2001
Butane and Propane	14	M	Cerebral anoxia	2002
Butane	19	M	Acute cardiac failure	2002
Liquefied Petroleum Gas	18		Asphyxia	2003
Butane	15	M	Not stated	2003
Butane	28	M	Pulmonary aspiration	2003
Aerosol propellants	16		Not stated	2003
Butane	18		Not stated	2003

## Discussion

Inhalant abuse is a persisting problem in New Zealand. Coronial data suggests it may account for at least 10% of all poisoning deaths (excluding carbon monoxide), with many of these deaths occurring in teenagers. Analysis of calls received by the NZNPC supports this finding, with a large majority of calls relating to 10 to 20 year olds. Inhalant abuse in teenagers is a common phenomenon worldwide as the household products commonly abused (including air freshener and fly spray) are inexpensive to obtain, simple to hide, and provide an easy way to get 'high'.

Our data could not provide detailed information on morbidity and outcome as we do not routinely follow up incoming enquiries (due to various reasons, including the 1993 Privacy Act). However in one prospective series in the US, ~38% of enquiries related to cases with significant effects, including loss of consciousness, seizures, metabolic acidosis, and occasionally death.<sup>4</sup> In another review, ~ 20% of cases had either moderate effects, usually requiring treatment, or were major effects or fatalities.<sup>5</sup>

Our data indicate that some abusers can be very young, as noted elsewhere.<sup>5</sup> They may be initially drawn by curiosity and the desire to imitate. The practice appeals because of the feelings of euphoria with loss of inhibitions. There may also be a sense of heightened powers, illusions, and hallucinations, along with mood swings and impulsive actions. However oral frothing, nausea, vomiting, light headedness, dizziness, slurred speech, ataxia, and cough with upper airway irritation can also develop. Heavier exposures can produce progressive nervous system depression. Nevertheless many subjects even with depressed levels of consciousness can have resolution of acute symptoms within one to two hours of cessation. This generally rapid recovery often means that abuse is not recognised by parents.<sup>4</sup>

Coronial data was insufficiently comprehensive to elucidate whether the apparent higher case fatality rate for males (reported elsewhere)<sup>4</sup> applies in New Zealand. Also unclear (internationally) is the fatality risk per abuse episode. While evidence is often circumstantial, most deaths are presumed to have a primary cardiac aetiology. A significant fraction occur in association with intense sympathetic stimulation as with running,<sup>6</sup> other exertion, auto-erotic behaviour, or agitation or startling of the abuser.

Running may be intentional (as in avoiding apprehenders) or seem coincidental, but often appears to be caused by the inhalation, and may be due in part to vivid hallucinations, at least in cases exhibiting fearful reactions.<sup>7</sup> The cardiac sensitising potency of inhaled hydrocarbons is greatly increased in the presence of high circulating catecholamine levels such as occurs with exercise or excitement<sup>6</sup> (the ability of injected adrenaline to markedly increase arrhythmia risk during solvent inhalation is well demonstrated in animal studies).<sup>8</sup>

While hypoxia and hypercapnia can also enhance cardiac sensitisation,<sup>9</sup> experimentally these do not seem such powerful influences.<sup>10</sup> Any acidosis, hypokalaemia or hypocalcaemia can also predispose to arrhythmias.

Experimentally there is evidence that solvents can inhibit cardiac inward sodium currents,<sup>11</sup> with risk of prolonged membrane depolarisation and slowed impulse propagation. (They “stabilise” myocardial cell membranes in ways that increase their resistance to normal trans-membrane ion currents.) Sinus bradycardia is typically the first rhythm abnormality seen in animal studies,<sup>12</sup> followed most commonly by AV dissociation with progressively lower escape rhythms, and finally electrical asystole or ventricular fibrillation. Bradycardia and ultimately asystole may partly arise from a direct effect on the sinoatrial node.<sup>11,12</sup> Myocardial infarction has occasionally occurred,<sup>13</sup> the postulated mechanism being coronary artery spasm.<sup>9,14</sup>

While sudden sniffing death syndrome may occur on the 1st, 10<sup>th</sup>, or 100th time a person abuses inhalants, it appears to occur most often in naïve, first-time users. (One factor may be the difficulty in regulation or “titration” of a dose with many of the delivery methods. There is also likely substantial individual variation in susceptibility,

though specific risk factors are not fully characterised.) Furthermore, the risk does not vanish immediately on cessation of inhalation, instead persisting for several hours. This is not surprising, given the high lipid solubility of solvents, which enables access into (and some persistence within) myocardial cell membranes.

Other causes of death include respiratory depression and anoxia from suffocation during the practice of inhaling substances from a plastic bag. Fatalities have also occurred due to aspiration of vomitus<sup>15</sup> and mishaps (such as drowning or motor vehicle accidents)<sup>1</sup> that can arise while functioning is still significantly impaired by the inhalant.

Butane and propane appear the most commonly abused inhalants which is not surprising, given they are found as propellants in a wide variety of household sprays. Both are recognised internationally as major causes of inhalant abuse fatalities.<sup>16</sup> Indeed, one study suggested that (especially in the context of air fresheners) they may carry a disproportionately high fatality rate.<sup>5</sup> However the data from the study, based on the toxic exposure surveillance system (TESS) database of the American Association of Poison Control Centers (AAPCC), only involved cases reported to individual PCCs, which likely represents a small proportion overall, with a potential for reporting bias.

While they are considered less narcotic and less potent cardiac sensitisers than some other propellants and solvents,<sup>17</sup> their high volatility and rapid evaporation from compressed liquid forms can result in very high exposures during abuse, increasing the risks.<sup>8</sup> Long term neurological sequelae may develop in surviving cases.<sup>13</sup>

Some methods of inhaling these compounds present additional hazards. Vagal inhibition may occur from a sudden freezing effect on the larynx and surrounding structures, due to the rapid expansion and cooling of gas produced from compressed liquid forms or even aerosols when sprayed directly into the mouth. For example, butane inhalation via cigarette lighter refills may involve releasing a jet of fluid cooled to  $\leq 20^{\circ}\text{C}$ . This can result in reflex inhibition of the heart, with bradycardia or in extreme cases, cardiac arrest.<sup>1</sup> There is also a risk of profuse mucosal oedema, burns to the throat and airways,<sup>18</sup> and laryngospasm with severe respiratory tract obstruction. Inhalation of burning (accidentally ignited) gas can cause adverse lung effects, including pulmonary oedema.

The increasing abuse of fly sprays is also concerning due to the presence of synthetic pyrethroids as the active constituent. Comparatively little is known about the effects of pyrethroids on the heart (as opposed to neurons). However, some experimental evidence suggests “type II” pyrethroids possess considerable mammalian cardiac arrhythmogenic potential.<sup>19</sup> Pyrethroids can also produce marked adrenal stimulation, with increased circulating catecholamines.<sup>20</sup> While severe allergic respiratory reactions are described with natural pyrethrum, and even its purified pyrethrin extracts,<sup>21</sup> they appear less of a risk with synthetic pyrethroids. In one case of a fly-spray abuse related death discussed with the NZNPC, lack of sputum eosinophils argued against an allergic basis. However allergic asthma has been attributed to tetramethrin.<sup>22</sup> While the role of pyrethroids as opposed to propane/butane in fly spray abuse-related deaths remains uncertain, they could be expected to increase the risk.

Acute management of abusers can be difficult, as there may be rapid onset of life-threatening effects, yet limited opportunity for early intervention, as abuse may take place in unobserved and/or remote situations. Most deaths occur outside hospital.<sup>23</sup>

All cases of acute abuse should be observed (even if without initially obvious clinical abnormalities), as a risk of sudden arrhythmia may remain for some hours after inhalation.<sup>24</sup> It is important to calm conscious victims to reduce further release of endogenous catecholamines.<sup>7</sup> Symptomatic cases should be medically observed until 8 to 12 hours post-exposure, while asymptomatic cases remaining so and without ECG abnormalities can probably be discharged safely after four hours.

Oxygen should be administered to all symptomatic patients. Severe respiratory depression requires assisted ventilation, and early airway protection has been recommended, particularly in unconscious patients, as vomiting is common.<sup>23</sup> However, intubation in the presence of laryngospasm (or for agents known to cause it) must be cautious, as this may cause excessive vagal stimulation, exacerbating bradycardia, and is not advised in patients with impending cardiac arrest.<sup>9</sup> Instead Ambu bag ventilation using an oropharyngeal airway has been successful in this situation.<sup>7,9</sup> With improvement in cardiac rhythm, endotracheal intubation, assisted by neuromuscular paralysis if necessary, can be instituted. Bronchoscopy may be required to remove aspirated material. In the event of bronchospasm, use of inhaled beta 2 adrenergic receptor agonists must be cautious, to minimise any additional risk of arrhythmia.

Resuscitation from cardiac arrhythmias has not often been successful, partly due to their rapidity of onset. It is generally not advised to administer catecholamines, as they can increase the risk of arrhythmias including ventricular fibrillation.<sup>9</sup> Standard electrical methods and early use of antiarrhythmic agents should be considered, particularly with recurrent ventricular arrhythmias.<sup>25</sup> Agents used in association with occasional successful outcomes have included amiodarone, lignocaine, and mexiletine.<sup>9,14</sup> Amiodarone has the advantage of causing little or no myocardial depression; initial doses of 300 mg IV have been used,<sup>25</sup> the same as recommended for ventricular fibrillation in other contexts.

Hypotension is largely secondary to cardiac arrhythmia or impaired contractility. It may respond to fluid replacement or management of any underlying cardiac rhythm disturbance. Careful use of an agent with inotropic (and/or chronotropic) properties may be required. Noradrenaline<sup>25</sup> and dopamine<sup>26</sup> (with additional vasopressor effects) have sometimes been used with success, despite concerns regarding arrhythmia risks. While a non-catecholamine might be theoretically preferable, there appears no reported experience with other inotropes such as glucagon. Myocardial infarction can occasionally be a factor in severe hypotension or arrhythmia, including recurrence of ventricular fibrillation.<sup>25</sup> In some cases it appears as a complication,<sup>26</sup> but might also occur as a primary event. Treatment with catecholamines may require extra caution in this situation.

The effectiveness of atropine for persistent, haemodynamically significant bradycardia appears unclear. Glucagon might be useful, and would be theoretically preferable to catecholamines in terms of safety. It directly increases automaticity at the sinoatrial and atrioventricular nodes,<sup>27</sup> and is known to have potent chronotropic

as well as inotropic actions. However there is no guidance on doses. It can also stimulate release of endogenous catecholamines.<sup>28</sup>

Beta-adrenergic blockers have been recommended to protect the catecholamine sensitised heart,<sup>9</sup> but should be used with care given their negative inotropic and chronotropic effects,<sup>25</sup> with risk of hypotension,<sup>26</sup> particularly in the presence of bradycardia. There are few reports of their use, (though a short acting compound appeared beneficial for junctional rhythm in one case).<sup>29</sup>

Seizures should be treated aggressively with benzodiazepines as their contribution to hypoxia, acidosis, and catecholamine stimulation can increase risk of arrhythmias. Rhabdomyolysis may develop, especially in cases involving seizures, prolonged immobilisation, or severe hypokalaemia. It may be a factor in cases of renal failure unexplained by circulatory impairment. Acidosis if severe requires treatment (an added advantage being this reduces risk of myoglobinuria induced renal tubular damage). Electrolyte disturbances may require correction, under close monitoring. Haemodialysis has occasionally been required for renal failure.

Chronic symptoms of inhalant abuse may include a chemical smell on the breath, poor attention to hygiene, obvious intoxication where alcohol is not a factor, personality changes, alterations in sleeping and eating behaviour and a persistently runny nose or eye irritations. A rash or acne around the nose/mouth may also be present but could be easily confused for what is a common complaint in many teenagers.

Toluene has been widely abused in the past and carries significant acute cardiac risk.<sup>30</sup> However in chronic abusers, neuropsychiatric disorders, gastro-intestinal complaints, and muscle weakness often feature.<sup>31</sup> Effects on short term memory, concentration, visuo-spatial and other executive or abstract thinking functions can be marked. Psychotic episodes may be precipitated or even initiated by high exposures.<sup>32</sup> Temporal lobe epilepsy, cranial nerve dysfunction, and peripheral neuropathy are reported (though co-exposure to other solvents can also be a factor).<sup>33</sup> Neurological effects are not always reversible. Imaging techniques have demonstrated decreased perfusion and atrophy of cerebral, cerebellar, thalamic and brainstem structures.<sup>34</sup> Jaundice is reported and abnormal liver function tests may take up to six months to normalise.<sup>35</sup> Chronic myocardial<sup>36</sup> effects have been reported, and adverse pulmonary effects may also occur.<sup>37</sup>

It can also produce renal tubular acidosis (RTA), with impaired ability to acidify the urine and thus increased risk of acidosis. Distal or less commonly proximal tubular acidosis or a mixed form can occur.<sup>38</sup> The tubular dysfunction can also result in electrolyte disturbances, including hypokalaemia, hypocalcaemia, hypophosphataemia, and hyperchloraemia.<sup>31</sup> Hypokalaemia (a risk factor for rhabdomyolysis) is more common than hyperkalaemia, especially in acute-on-chronic abuse, but the latter can occur acutely, generally as a result of rhabdomyolysis, which itself may result in acute tubular damage.<sup>39</sup> However renal insufficiency is often rapidly reversible, with reduced urine output for two or three days only after sniffing episodes.<sup>35</sup> Metabolic acidosis can be marked; when present as an acute effect<sup>38</sup> there is often an elevated anion gap component due to accumulation of toluene metabolites,<sup>40</sup> while with regular abusers, RTA is a common contributor. Maternal abuse has been associated with a foetal solvent syndrome,<sup>41</sup> postnatal persistence of growth deficiency, and electrolyte abnormalities in the newborn.<sup>42</sup>

Given the difficulties in acute management and the significant chronic morbidity, preventive measures are critical. Following acute treatment, all patients should be referred to an appropriate substance abuse program. Youthful users motivated primarily by curiosity and peer pressure may be responsive to educational campaigns,<sup>43</sup> however the best approach may be to also provide family and community counselling, residential care, and alternative recreational activities.<sup>44,45</sup> Fortunately it appears that most users ultimately abandon the practice, often before they develop physical complications, such as neurological or renal damage. However, it appears to be a gateway phenomenon among younger adolescents where children who abuse inhalants early in life are more likely later to use other illicit drugs.<sup>46</sup> Continued efforts to optimise prevention are required, while recognising that complete control of supply of all inhalants with abuse potential is not possible.

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#### References:

1. Shepherd RT. Mechanism of sudden death associated with volatile substance abuse. *Hum Toxicol.* 1989;8:287–91.
2. Masterton.co.nz. The heart of the Wairarapa. Tragic lesson in deaths parents' emotional plea. Available online. URL: <http://masterton.co.nz/modules.php?name=News&file=print&sid=366> Accessed April 2006.
3. The Institute of Environmental Science and Research Limited (ESR). Chemical Injuries Surveillance. Porirua: CSR; 2005. Available online. URL: [http://www.surv.esr.cri.nz/public\\_health\\_surveillance/chemical\\_injuries\\_surveillance.php](http://www.surv.esr.cri.nz/public_health_surveillance/chemical_injuries_surveillance.php) Accessed April 2006.
4. Spiller HA, Krenzelok EP. Epidemiology of inhalant abuse reported to two regional poison centers. *J Toxicol Clin Toxicol.* 1997;35:167–73.
5. Spiller HA. Epidemiology of volatile substance abuse (VSA) cases reported to U.S. Poison Centers. *Am J Drug Alcohol Abuse.* 2004;30:155–65.
6. Bass M. Sudden sniffing death. *J Am Med Assoc.* 1970;212:2075–9.
7. Williams DR, Cole SJ. Ventricular fibrillation following butane gas inhalation. *Resuscitation.* 1998;37:43–5.
8. Clark DG, Tinston DJ. Correlation of the cardiac sensitizing potential of halogenated hydrocarbons with their physicochemical properties. *Br J Pharmacol.* 1973;49:355–7.
9. Adgey AAJ, Johnston PW, McMechan S. Sudden cardiac death and substance abuse. *Resuscitation.* 1995;29:219–21.
10. Flowers NC, Horan LG. Nonanoxic aerosol arrhythmias. *J Am Med Assoc.* 1972;219:33–7.
11. Cruz SL, Orta-Salazar G, Gauthereau MY, et al. Inhibition of cardiac sodium currents by toluene exposure. *Br J Pharmacol.* 2003;140:653–60.
12. Taylor GJ, Harris WS. Cardiac toxicity of aerosol propellants. *J Am Med Assoc.* 1970;214:81–5.
13. Bauman JE, Dean BS, Krenzelok EP. Myocardial infarction and neurodevastation following butane inhalation [Abstract]. *Vet Hum Toxicol.* 1991;33:389.

14. Cunningham SR, Dalzell GWN, McGirr P, Khan MM. Myocardial infarction and primary ventricular fibrillation after glue sniffing. *Br Med J.* 1987;294:739-40.
15. Anderson HR, Macnair RS, Ramsey JD. Deaths from abuse of volatile substances: a national epidemiological study. *Br Med J.* 1985;290:304-7.
16. Johns A. Volatile solvent abuse and 963 deaths. *Br J Addict.* 1991; 86:1053-6.
17. Reinhardt CF, Azar A, Maxfield ME, et al. Cardiac arrhythmias and aerosol "sniffing." *Arch Environ Health.* 1971;22:265-79.
18. Oh S-J, Lee S-E, Burm J-S, et al. Explosive burns during abusive inhalation of butane gas. *Burns.* 1999;25:341-4.
19. Spencer CI, Yuill KH, Borg JJ, et al. Actions of pyrethroid insecticides on sodium currents, action potentials, and contractile rhythm in isolated mammalian ventricular myocytes and perfused hearts. *J Pharmacol Exp Ther.* 2001;298:1067-82.
20. Ray DE, Forshaw PJ. Pyrethroid insecticides: poisoning syndromes, synergies, and therapy. *J Toxicol Clin Toxicol.* 2000;38:95-101.
21. Wax PM, Hoffman RS. Fatality associated with inhalation of a pyrethrin shampoo. *Clin Toxicol.* 1994;32:457-60.
22. Vandenplas O, Delwiche J-P, Auverdin J, et al. Asthma to tetramethrin. *Allergy.* 2000;55:417-8.
23. Roberts MJD, McIvor RA, Adgey AAJ. Asystole following butane gas inhalation. *Br J Hosp Med.* 1990;44:294.
24. Ashton CH. Solvent abuse. *Br Med J.* 1990;300:135-6.
25. Edwards KE, Wenstone R. Successful resuscitation from recurrent ventricular fibrillation secondary to butane inhalation. *Br J Anaesth.* 2000;84:803-5.
26. LoVecchio F, Fulton SE. Ventricular fibrillation following inhalation of Glade air freshener.<sup>TM</sup> *Eur J Emerg Med.* 2001;8:153-4.
27. Love JN, Sachdeva DK, Bessman ES, et al. A potential role for glucagon in the treatment of drug-induced symptomatic bradycardia. *Chest.* 1998;114:323-6.
28. White CM. A review of potential cardiovascular uses of intravenous glucagon administration. *J Clin Pharmacol.* 1999;39:442-7.
29. Mortiz F, de La Chapelle A, Bauer F, et al. Esmolol in the treatment of severe arrhythmia after acute trichloroethylene poisoning. *Intensive Care Med.* 2000;26:256.
30. Carder JR, Fuerst RS. Myocardial infarction after toluene inhalation. *Pediatr Emerg Care.* 1997;13:117-9.
31. Streicher HZ, Gabow PA, Moss AH, et al. Syndromes of toluene sniffing in adults. *Ann Intern Med.* 1981;94:758-62.
32. Tarsh MJ. Schizophreniform psychosis caused by sniffing toluene. *J Soc Occup Med.* 1979;29:131-3.
33. King PJ, Morris JG, Pollard JD. Glue sniffing neuropathy. *Aust N Z J Med.* 1985;15:293-9.
34. Caldemeyer KS, Armstrong SW, George KK, et al. The spectrum of neuroimaging abnormalities in solvent abuse and their clinical correlation. *J Neuroimaging.* 1996;6:167-73.
35. O'Brien ET, Yeoman WB, Hobby JAE. Hepatorenal damage from toluene in a glue sniffer. *Br Med J.* 1971;2:29.
36. Wiseman M, Banim S. "Glue sniffer's" heart? *Br Med J.* 1987;294:739.
37. Schikler KN, Lane EE, Seitz K, Collins WM. Solvent abuse associated pulmonary abnormalities. *Adv Alcohol Subst Abuse.* 1984;3:75-81.

38. Kamijima M, Nakazawa Y, Yamakawa M, et al. Metabolic acidosis and renal tubular injury due to pure toluene inhalation. *Arch Environ Health*. 1994;49:410–3.
39. Mizutani T, Oohashi N, Naito H. Myoglobinemia and renal failure in toluene poisoning: a case report. *Vet Hum Toxicol*. 1989;31:448–50.
40. Fischman CM, Osler JR. Toxic effects of toluene: a new cause of high anion gap metabolic acidosis. *J Am Med Assoc*. 1979;241:1713–5.
41. Pearson MA, Hoyme HE, Seaver LH, Rimsza ME. Toluene embryopathy: delineation of the phenotype and comparison with fetal alcohol syndrome. *Pediatrics*. 1994;93:211–5.
42. Wilkins-Haug L, Gabow PA. Toluene abuse during pregnancy: obstetric complications and perinatal outcomes. *Obstet Gynecol*. 1991;77:504–9.
43. Dinwiddie SH. Abuse of inhalants: a review. *Addiction*. 1994;89:925–39.
44. Billington AC. Volatile substance abuse: the role of agencies in the community in prevention and counselling. *Hum Toxicol*. 1989;8:323–5.
45. Lee JT. Volatile substance abuse within a health education context. *Hum Toxicol* 1989;8:331–4.
46. Johnson EO, Schutz CG, Anthony JC, Ensminger ME. Inhalants to heroin: a prospective analysis from adolescence to adulthood. *Drug Alcohol Depend*. 1995;40:159–64.