CASE REPORT

Cardiac arrest by inhalation of deodorant spray
Kelvin Harvey Kramp, Mahdi Salih, Elsbeth Thomeer, Martin Gardien

SUMMARY
Drug abuse by inhalation of volatile household product substances is uncommon, however, it can have devastating consequences. This case report describes the fatal outcome of deodorant inhalation by a 19-year-old patient in a detoxification clinic who developed a cardiac arrest after inhaling butane from a deodorant spray. Despite initial successful resuscitation, he developed a postanoxic encephalopathy with a status epilepticus resistant to extensive treatment. Inhalant abuse can be a cause of death in young patients.

BACKGROUND
Volatile substances in household products can be used in various ways as drugs to attain an altered mental state. Although the gross of medical personnel is largely unfamiliar with intoxication due to inhalant abuse, the consequences can be severe. Cardiac arrest appears to be caused by increased myocardial sensitivity to catecholamines, coronary spasms, depressed myocardium or a combination of these pathophysiological mechanisms. This case report describes a patient under treatment in a drug rehabilitation centre that relapsed in drug abuse by abusing butane, an inhalant readily available in household products. The aim of this paper is to emphasise that there might be an increased risk of inhalation-related cardiac deaths in isolated environments, such as detoxification clinics or prisons, because patients within these places are more inclined to abuse household products.

OUTCOME AND FOLLOW-UP
After stopping sedation the Glasgow Coma Score remained E1M1Vt. The patient showed persistent myoclonias despite treatment with propofol infusion and levetiracetam. This was accompanied by cerebral diabetes insipidus, suggested by increased urine production (maximum 1710 mL/hour), increased plasma sodium concentration and low urine osmolality (84 mOsm/L). Vasopressin was initiated with a consequent decrease in urine production. A brain CT-scan showed no signs of cerebral oedema or bleeding. Somatosensory evoked potential showed a bilateral present cortical negative peak at 20ms (N20) response, which did not predict a poor outcome per se. On day 3, the patient showed myoclonic jerks, for which levetiracetam and propofol was started. On day 4, an electroencephalogram (EEG) showed spike and wave complexes with high index for which diphenylhydantoin was added on. On day 5, the EEG showed generalised slow wave activity and excess of beta activity that was considered to be drug induced. Spike and wave complexes were still present but with a lower index compared with the day before. The patient had intermittent signs of an epileptic seizure for which valproate and midazolam were initiated along with the previously started propofol, levetiracetam and diphenylhydantoin. On day 7, the EEG showed an electrical status epilepticus. A barbiturate coma was induced with thiopental during two episodes of 24 hours with an initial reduction of epileptiform activity. On day 9, the second barbiturate coma ended and the patient again showed rhythmic contractions of the eyebrows, knees and no signs of (self) strangulation or other physical trauma. Initial laboratory investigation showed a lactate acidosis (table 1).

Family history did not give any clues for a heritable cause that could have led to the cardiac arrest. The chest X-ray revealed broken ribs and a haemothorax, most likely the consequence of basic life support. Toxic substance screening of the urine was negative for amphetamine, cocaine, cannabis, 3,4-methylenedioxymethamphetamine (XTC) and phencyclidine.

At arrival on the intensive care unit, the patient was placed on dobutamine, enoximon and nitroprusside. He was sedated with midazolam and remifentanil and therapeutic hypothermia was initiated (33°C during a period of 24 hours) with cold infusion and a Blanketrol III (Cincinnati Sub-Zero Products). On day 2, after reaching normothermia, the ECG had normalised and troponin levels were decreasing.
repetitive swallowing. Considering the persistent Glasgow Coma Score (E1M1Vt) after stopping sedation and the recurrent signs of a status epilepticus on physical examination and EEG, unresponsive to treatment with propofol, levetiracetam, diphenitoine and thiopental, further treatment was deemed futile. The patient was extubated and comfort care was given with morphine and midazolam. The patient died shortly thereafter.

DISCUSSION
Cardiac arrests after the inhalation of volatile substances have been reported for more than 40 years. The phenomenon of sudden cardiac death after inhalant abuse has been named ‘sudden sniffing death’ after the epidemic of 110 cardiac arrests due to the rising popularity of glue sniffing in the 1960s among American youths. The first report of sudden cardiac death after butane intoxication due to the inhalation of deodorant spray was reported in 1975 by Kamm. There have been a large number of case reports afterwards describing cardiac arrests after deodorant inhalation, but only a few have described patients who survived after treatment.16

The misuse of volatile substances is one of the least known methods to attain an altered mental state by drugs. It is estimated that in the USA it causes 100–125 deaths per year.7 Inhalant abuse has a remarkable high prevalence in younger age groups, reaching its peak incidence between 15 and 19 years. The mean age of first use is 16.3 years, the youngest age of all drugs and the prevalence decreases sharply after the first few years after high school. The use of inhalants has been correlated with a low income, chaotic, fractured and abusive households.10 11 There is a 10-fold higher change of getting help from municipal care and the highest mortality is observed in urban populations.12

There is a long list of household products that can be used for inhalation.10 The substances can be categorised into: (1) volatile solutions such as lime, paint thinner and felt tip, (2) aerosols such as hairspray, deodorant and spray paint and (3) pressurised gasses such as from a lighter or camping gas.13 Inhalant abuse is usually categorised into three modalities: sniffing, huffing and bagging.14 Sniffing is the direct inhalation of volatile substances. Huffing is spraying the volatile substance on a piece of clothing and inhaling it through the nose or mouth. A piece of clothing can be soaked in a volatile substance, or as in our case, the substance can be sprayed through a clothing placed over the nose or mouth. Bagging is using a plastic bag or balloon to transfer the inhalant to outside of the original packing. A soaked clothing can be put into the plastic bag or the bag can be placed over the head. The latter carries the risk of hypoxia as a consequence of asphyxia next to inhalant intoxication, although getting unconscious would normally release the muscle tone needed to fixate the bag over the head. Which modality is used depends on the desired intensity and on the particular substance used. Often, there is a progression from sniffing to huffing and from huffing to bagging.15 Independent of the method of inhalation

Table 1 Laboratory evaluation at admission

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Result</th>
<th>Unit</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRP</td>
<td>&lt;1</td>
<td>mg/L</td>
<td>&lt;1</td>
</tr>
<tr>
<td>WBC</td>
<td>31.2</td>
<td>10^9/L</td>
<td>4.0–10.0</td>
</tr>
<tr>
<td>Potassium</td>
<td>2.9</td>
<td>mmol/L</td>
<td>3.5–5.0</td>
</tr>
<tr>
<td>Sodium</td>
<td>136</td>
<td>mmol/L</td>
<td>135–145</td>
</tr>
<tr>
<td>Creatinine</td>
<td>133</td>
<td>µmol/L</td>
<td>64–104</td>
</tr>
<tr>
<td>Urea</td>
<td>5.3</td>
<td>mmol/L</td>
<td>2.5–6.4</td>
</tr>
<tr>
<td>eGFR</td>
<td>66</td>
<td>mL/min/1.73 m²</td>
<td>&gt;90</td>
</tr>
<tr>
<td>Corrected Calcium</td>
<td>2.33</td>
<td>mmol/L</td>
<td>2.20–2.65</td>
</tr>
<tr>
<td>ALAT</td>
<td>148</td>
<td>U/L</td>
<td>0–41</td>
</tr>
<tr>
<td>ASAT</td>
<td>140</td>
<td>U/L</td>
<td>0–37</td>
</tr>
<tr>
<td>LDH</td>
<td>356</td>
<td>U/L</td>
<td>0–250</td>
</tr>
<tr>
<td>Troponin</td>
<td>0.06</td>
<td>µg/L</td>
<td>0.00–0.04</td>
</tr>
<tr>
<td>Glucose</td>
<td>18.8</td>
<td>mmol/L</td>
<td>4.4–5.5</td>
</tr>
<tr>
<td>Arterial blood gas</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.14</td>
<td></td>
<td>7.35–7.45</td>
</tr>
<tr>
<td>Partial pressure of carbon dioxide</td>
<td>40</td>
<td>mm Hg</td>
<td>35–48</td>
</tr>
<tr>
<td>HCO3</td>
<td>14</td>
<td>mmol/L</td>
<td>21–28</td>
</tr>
<tr>
<td>Partial pressure of oxygen</td>
<td>81</td>
<td>mm Hg</td>
<td>75–100</td>
</tr>
<tr>
<td>Lactate</td>
<td>14.1</td>
<td>mmol/L</td>
<td>0.5–2.2</td>
</tr>
</tbody>
</table>

ALAT, alanine aminotransferase; ASAT, aspartate aminotransferase; CRP, C reactive protein; eGFR, estimated Glomerular Filtration rate; HCO3, Bicarbonate; LDH, lactate dehydrogenase; WBC, White blood cells.

Figure 1 ECG after return of spontaneous circulation following defibrillation.
and concentration of toxic substance inhaled, sudden death from inhalant abuse can result during a first single dose or during chronic use. It is reported that 43% of deaths are related to the first-time inhalant abuse.\[^{16,17}\]

The main toxic substance in deodorant spray inhalation is butane. Butane is one of the hydrocarbons commonly used as propellants in sprayable household products. Hydrocarbons are lipophilic and therefore easily cross the air–blood and blood–brain barrier. It dissolves into tissues with a high fat content such as the nervous system, fat tissue, liver and kidneys. Elimination is through respiration.\[^{18}\]\[^{19}\] The longer the carbon chain inhaled the higher the lipophilicity and the lower the onset threshold for the desired experience.\[^{18}\]\[^{20}\] Raines et al\[^{18}\] conducted an electrophysiological study of the effects of butane on the gamma-aminobutyric acid (GABA) receptors and observed butane has the ability to enhance the effects of agonists on the GABA receptor.\[^{18}\]\[^{21}\] Thus, butane has similar neurological disinhibiting effect as alcohol. The intention of abusers is to experience feelings of euphoria and disinhibition. Symptoms that can reveal intoxication include slurred speech, ataxia, confusion, hallucinations and a loss of consciousness at high blood concentration.\[^{19}\]\[^{22}\]

Abusers can have a distinct smell on their clothing, skin or breath. A review on the causes of death by inhalant abuse revealed that 77% of deaths could be attributed to direct effects of the inhalant, 18% by asphyxia due to the use of a plastic bag.\[^{23}\]\[^{24}\] Inhalants exert their potential lethal toxic effects primarily on the cardiovascular system. First, inhalants can sensitize the heart for adrenergic stimulation. Consequent physical activity due to adrenergic stimulation leads to increased sensitivity to counteract the sympathic stimulation of the heart.\[^{21}\]\[^{22}\] Second, myocardial dysfunction might be involved in the pathophysiological process of sudden cardiac death as Harris has shown a depression of the myocardial contractility in response to inhalant administration in rats and monkeys.\[^{22}\]\[^{23}\] Lastly, coronary spasm has also been suggested as an explanation for sudden cardiac death. There are cases wherein the ECG demonstrated patterns of cardiac ischaemia with increased cardiac enzymes as in our case, but coronary angiography showed no evidence of coronary artery stenosis or thrombosis.\[^{23}\]\[^{24}\]

As with many drugs that depress the nervous system, inhalants in high doses can cause central nervous system depression with respiratory failure,\[^{10}\] although there is only one report of respiratory arrest during inhalant abuse.\[^{25}\] Respiratory failure can also be caused by spraying directly into the pharynx. The rapid decrease of the temperature of the fluids to −20°C as a consequence of gas volume expansion can cause prolonged laryngospasm.\[^{26}\] Furthermore, the 1:100 increase in gas volume after spraying pressurised deodorant can cause alveolar hypoxia leading to cardiac arrest.

In conclusion, our case describes the fatal outcome of deodorant inhalation and subsequent cardiac arrest in a detoxification clinic. Medical personnel should be cautioned on the dangers of inhalant abuse in this population.

**Contributors** KHK was part of the medical care during admittance of the patient and wrote case presentation, outcome, follow-up and discussion of the paper. MS was and part of the medical care during follow-up and wrote case presentation, outcome, follow-up and discussion of the patient. ET took part in the neurological examination and follow-up of the patient and reviewed the final version of the article. MG supervised the medical treatment and reviewed the final version of the article.

**Funding** The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

**Competing interests** None declared.

**Patient consent** Parental/guardian consent obtained.

**Provenance and peer review** Not commissioned; externally peer reviewed.

**Learning points**

- The abuse of volatile substances in everyday household products as inhalants is popular among youngsters in puberty and adolescents and has the highest prevalence among children from troubled households.
- Medical personnel unfamiliar with inhalant abuse can be confronted with their dramatic consequences such as asphyxia, trauma, aspiration during unconsciousness and cardiac arrest.
- Patients with a history of substance abuse who reside in an isolated environment, such as a drug rehabilitation centre or prison, may be at increased risk for using inhalants.

**REFERENCES**

Unexpected outcome (positive or negative) including adverse drug reactions