Management of intentional overdose in A&E departments

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**Abstract**


This guided reflection article discusses the biological, psychological and social causes of intentional overdose and how the A&E nurse can treat the suicidal patient. The author shows how nurses in A&E can provide interventions that address patients’ physical and psychosocial needs. The correct acute response to the medications commonly taken in an intentional overdose case are described. These interventions promote patient survival, improve concordance with mental health follow-up and reduce recidivism.

There is an entire body of research on the attitudes of A&E nurses towards patients who have taken intentional overdose (McLaughlin, 1994). This is an aspect of nursing care that brings nurses face to face with moral questions about their ability to help someone and can sometimes result in feelings of helplessness. It is perhaps unsurprising then, that many nurses react with anger and frustration to the medications described. These interventions promote patient survival, improve concordance with mental health follow-up and reduce recidivism.

**Why do people take overdoses?**

Intentional overdose is usually an attempt to stop suffering, and anyone has the potential to consider it if they perceive their suffering to be intolerable, interminable and inescapable (Chiles and Strosahl, 1995). Those who lack the psychosocial resources that ameliorate suffering are therefore at risk. These resources include:

- Religious beliefs;
- Social support;
- Problem-solving skills.

Psychiatric disorder is a source of suffering that also undermines those important psychosocial resources, and patients with specific psychiatric diagnoses are at increased risk of successfully committing suicide (Box 1). However, the presence of psychiatric disorder does not in itself predict suicidal behaviour, indeed the vast majority of psychiatric patients will never attempt suicide (Mann, 2002).

Furthermore, half of intentional overdose cases have no specific psychiatric disorder (Chiles and Strosahl, 1995).

Those who are more likely to take an overdose tend to react more pessimistically to stressful events, and usually experience more hopelessness and depression as a result (Mann, 2002). Chiles and Strosahl (1995) state that their lives are often marked by interpersonal conflict and limited social support, producing:

- Isolation;
- Social anxiety;
- Difficulty in forming new relationships.

They often have poor problem-solving skills, coupled with an impatience for results (Chiles and Strosahl, 1995) and are likely to display aggressive and impulsive behavioural traits (Mann, 2002) that are biological as well as psychological.

Aggressive and impulsive behaviour is correlated with impaired serotonin functioning in the brain (Gilliam et al, 2000). Childhood adversity, in addition to producing psychological impairment, is suspected of impairing the anatomical development of serotonin systems in the brain (Skodol et al, 2002). This is a possible biopsychosocial mechanism by which people who suffer abuse, neglect and violence in childhood may become self-destructive adolescents and adults. Those born with impaired serotonin systems are more likely to provoke adverse experiences because of their aggressive and impulsive behaviours.

Consistent with this, most patients explain their overdose as a reaction to being in ‘a terrible state of mind’ or ‘an unbearable situation’, feeling a loss of control, wanting to die or wanting to escape from an impossible situation (Schnyder et al, 1999). Patients also commonly describe feelings of despair, emptiness, and anxiety or panic prior to overdosing (Schnyder et al, 1999).

Most deny that it was intended to simply ‘manipulate’ others, although some admit wanting people to understand how desperate they were feeling or to seek help from someone or thinking that their death would make things easier for others (Schnyder et al, 1999).

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**Learning objectives**

- Consider your attitude to patients who have taken an intentional overdose
- Be able to recognise the psychosocial factors contributing to overdose
- Gain an understanding of the principles of acute care for this patient group
- Identify the most commonly taken drugs
- Become aware of how you can meet the psychosocial needs of patients who have taken an intentional overdose

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**References**

Thus, they take intentional overdoses to escape from emotional suffering when they have exhausted all other attempts to cope. Some will be motivated by a serious intent to die, while others will be ambivalent. They may be desperate to find alternative ways of responding to their suffering, yet may die for want of an answer (Chiles and Strosahl, 1995).

**Acute interventions**

The first aim is to ensure the patient’s survival using a logical system of acute assessment and intervention (Clegg and Hope, 1999). Standard resuscitation assessment and interventions should be used (ABC – assess airway, breathing, circulation, and neurological status) (Advanced Life Support Group, 2001).

If possible, the amount and type of substance(s) taken, and length of time since ingestion should be ascertained. Four interventions are traditionally used in treating overdose (Clegg and Hope, 1999):

- Induced vomiting;
- Gastric lavage;
- Whole bowel irrigation;
- Absorbed charcoal.

However, the only routine intervention currently recommended is the use of activated charcoal (Mehta et al, 2004). This is most effective when given within one hour of ingestion. Some poisons are best treated with repeated doses of 50g of activated charcoal every four hours (Mehta et al, 2004) (Box 2, p40).

Gastric lavage may be considered in patients who have taken poisons that are not absorbed by activated charcoal – excluding petroleum distillates and corrosives (Box 3, p41). Whole bowel irrigation should only be considered after receiving advice from a national poisons centre (the electronic database can be found on the internet at www.spib.axl.co.uk/toxbase) (Mehta et al, 2004). In severe poisoning haemodialysis or haemoperfusion may be indicated (Boxes 4–5, p42–43).

**Box 1. Completed Suicide Rates for Selected Psychiatric Diagnoses versus General Population Rates (Harris and Barraclough, 1997)**

<table>
<thead>
<tr>
<th>PSYCHIATRIC DIAGNOSIS</th>
<th>SUICIDE RATE COMPARED WITH GENERAL POPULATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Past inpatient treatment for suicidal ideation</td>
<td>47 times higher</td>
</tr>
<tr>
<td>Past overdose</td>
<td>40 times higher</td>
</tr>
<tr>
<td>Past suicide attempt</td>
<td>38 times higher</td>
</tr>
<tr>
<td>Depression</td>
<td>20 times higher (different studies have found a range of 0–200 times higher than the general population)</td>
</tr>
<tr>
<td>Sedative, hypnotic or anxiolytic dependence and abuse</td>
<td>20 times higher; + alcohol 16 times; + drugs 44 times</td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td>15 times higher (range 0–133 times higher)</td>
</tr>
<tr>
<td>Opioid dependence and abuse</td>
<td>14 times higher (range 3–36 times higher)</td>
</tr>
<tr>
<td>Alcohol dependence and abuse</td>
<td>6 times higher (range 1–60 times higher)</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>8.5 times higher (range 0.8–115 times higher) (most common in age &lt;30 in first year after diagnosis)</td>
</tr>
<tr>
<td>Obsessive compulsive disorder</td>
<td>10 times higher</td>
</tr>
<tr>
<td>Panic disorder</td>
<td>10 times higher</td>
</tr>
<tr>
<td>Personality disorder</td>
<td>7 times higher</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>5 times higher</td>
</tr>
<tr>
<td>Generalised anxiety disorder</td>
<td>2.5–3.5 times higher</td>
</tr>
<tr>
<td>Huntington’s disease</td>
<td>3 times higher</td>
</tr>
</tbody>
</table>

**References**


Prescribed medication

**Antidepressants**
Antidepressants are commonly prescribed for depression and anxiety disorders (these conditions often coexist). Tricyclics are also prescribed for certain types of neuropathic pain. The majority of deaths occur within six hours of ingestion, and none occur after 24 hours have elapsed (Finnell and Harris, 2000).

Those at greatest risk on presentation are those with (Finnell and Harris, 2000):
- Arhythmias;
- Altered mental status;
- A QRS interval of greater than 0.1 seconds;
- Seizures;
- Respiratory depression;
- Hypotension.

Newer antidepressants, such as selective serotonin reuptake inhibitors (SSRIs), are less cardiotoxic than tricyclics (Mehta et al, 2004).

**Antidiabetics**
The main effect of an insulin overdose is prolonged hypoglycaemia, alongside seizures and coma. The depot effect of a large overdose can necessitate continued glucose treatment for up to four days (Spiller, 1998). Surgical excision of the depot site, while not routine, may be considered if the insulin was injected within the past three hours, was a particularly large dose, and was a long-acting preparation (Spiller, 1998).

Hypoglycaemia is increased by the additional ingestion of beta-blockers, alcohol, salicylates, fenfluramine, clofibrate, tetracycline, or monoamine oxidase inhibitors, while hepatic or renal impairment can prolong insulin’s half-life (Spiller, 1998). Prolonged hypoglycaemia can result in cerebral oedema, bradycardia, cardiovascular collapse and hypokalaemia, and if left untreated can lead to permanent neurological damage. Glucagon is likely to be ineffective, as the patient’s hepatic glycogen stores are likely to have been exhausted.

**Biguanide (metformin) overdose** produces initial symptoms of nausea, vomiting and abdominal pain, but hypoglycaemia does not usually occur (Spiller, 1998). More seriously, it induces lactic acidosis, potentially causing confusion, lethargy, coma, seizures, hypotension, tachycardia, ventricular arrhythmias and myocardial infarction (Spiller, 1998). The development of acidosis can occur a number of hours following ingestion, so observations are required for up to eight hours. Spiller (1998) recommends intubation and artificial hyperventilation to reduce circulating carbon dioxide generated by the acidosis, allowing lactate metabolism to resume. An alternative is to administer insulin intravenously, as it reduces lactate production.

The main danger from sulphonylurea overdose is the possibility of prolonged hypoglycaemia (Spiller, 1998). As with insulin overdose, this can lead to either death or permanent neurological damage.

**Antiepileptics**
Carbamazepine overdose produces drowsiness or agitation, coma, seizures, hypotension or hypertension, and respiratory depression. Adult respiratory distress syndrome, cardiovascular collapse, cardiac arrhythmias and rhabdomyolysis (reduction in skeletal muscle) are possible. Cardiac and respiratory monitoring should continue for at least 12 hours after ingestion (McCrea, 2002). Gabapentin overdose produces vomiting, dizziness, ataxia, headache, dilated pupils, faecal incontinence, tachycardia, respiratory depression, hypotension and convulsions. Patients should be observed for four hours following overdose and be given symptomatic and supportive care as required (McCrea, 2002).

Lamotrigine overdose produces nausea, vomiting, drowsiness, diplopia, nystagmus, ataxia, muscle weakness, hypertonia and tremor. It can also produce hypotension, respiratory depression, coma and convulsions. Observations should continue for six hours following overdose and symptomatic and supportive care should be given as required (McCrea, 2002). Mild phenobarbital overdose can often be characterised by drowsiness, ataxia, slurred speech and hypotension. Severe overdose produces coma, respiratory depression, myocardial depression and occasionally cer-
Phenytoin overdose produces vomiting, ataxia, slurred speech, nystagmus, altered consciousness, confusion and delirium. Occasionally, it can produce severe coma, hypotension and respiratory depression. Observations should continue for at least three hours after overdose, but symptoms can be prolonged, requiring hospitalisation (McCrea, 2002).

A mild overdose of sodium valproate results in gastrointestinal disturbance, drowsiness, dizziness, confusion, ataxia, and irritability. Severe overdose is rare, but produces a sudden deep coma, hypotonia, hyporeflexia, convulsions and respiratory depression. Cerebral oedema can occur up to 72 hours after overdose. Any cardiac effects are usually mild (for example, prolonged QTc interval). Electrolytes and blood gases should be monitored for a minimum of six hours after overdose and 12 hours for modified-release preparations (McCrea, 2002).

Antipsychotics

Antipsychotics are usually prescribed for schizophrenia or mania, while smaller doses are prescribed for anxiety. Overdose can produce dystonias (torticollis, or neck twisting; trismus or jaw lock; oculogyric crisis, or locked upward gaze) that respond to diazepam or antimuscarinic drugs. There can also be changes to conscious state and cardiac symptoms. Death following antipsychotic overdose is relatively rare (Pickford, 2000).

If arrhythmias occur, disopyramide, procainamide and quinidine should be avoided as they can have QT-prolonging effects, especially with ziprasidone. In hypotension and circulatory collapse use intravenous fluids and sympathomimetics, with preference for noradrenaline and dopamine. Cardiac symptoms – arrhythmias, heart block, ventricular fibrillation, hypotension and cardiac arrest – can take up to six hours to develop (Finnell and Harris, 2000). Cardiac symptoms – arrhythmias, heart block, ventricular tachycardia, ventricular fibrillation, hypotension and cardiac arrest – can take up to six hours to develop (Finnell and Harris, 2000). Beta blocker overdose should be managed in a similar manner to calcium channel antagonists (Nelson, 2001). Signs of toxicity usually appear within two hours, including seizures, hypotension and bradycardia, which similarly do not respond to usual interventions (Finnell and Harris, 2000).

Cardiac drugs

Calcium channel antagonists are prescribed for hypertension and angina. Overdose produces life-threatening hypotension, bradycardia, reduced cardiac output and asystole (Finnell and Harris, 2000). The sudden hypotension can produce shock. There can be confusion, agitation, drowsiness, seizures and hyperglycaemia. The latter is due to decreased insulin production, and can result in ketoacidosis. Amiodipine has the longest half-life, with a significant overdose producing a toxicity that can last 10 days (Finnell and Harris, 2000). Whole bowel irrigation can be considered following overdose with modified-release preparations.

The antidote to calcium channel antagonist overdose is intravenous calcium chloride (Finnell and Harris, 2000) or calcium gluconate (Nelson, 2001). However, digoxin levels should be checked first, as calcium salts can interact with digoxin to produce cardiac arrhythmia. Glucagon can be used to reverse cardiac instability (Finnell and Harris, 2000). Overdose-induced hypotension and bradycardia do not respond to any of the usual treatments. Dopamine, adrenaline, noradrenaline, dobutamine and isoproterenol are all specifically recommended. Insulin therapy and dextrose infusion can be given for up to four days in the case of unresponsive hypotension. Haemoperfusion may help for refractory hypotension (Finnell and Harris, 2000).

Mortality following digoxin overdose is greatest in those with heart disease (Bara, 2001). Early symptoms of overdose include anorexia, nausea, vomiting, headache, lethargy, confusion and visual disturbances (yellow or green halos around lights) (Finnell and Harris, 2000). Cardiac symptoms – arrhythmias, heart block, ventricular tachycardia, ventricular fibrillation, hypotension and cardiac arrest – can take up to six hours to develop (Finnell and Harris, 2000). The antidote is digoxin-specific Fab antibody fragments (Digibind).

Beta blocker overdose should be managed in a similar manner to calcium channel antagonists (Nelson, 2001). Signs of toxicity usually appear within two hours, including seizures, hypotension and bradycardia, which similarly do not respond to usual interventions (Finnell and Harris, 2000).

Opioids

Opioid overdose produces increasing unconsciousness, respiratory depression and pinpoint pupils. Naloxone is an antidote (Mehta et al, 2004), but its relatively short duration of action means the patient can go back into the overdose state as the naloxone is metabolised so repeated doses may be required.

With very long-acting opioids, such as methadone and dextropropoxyphene, monitoring may need to be prolonged. Dextropropoxyphene and paracetamol in combi-

**REFERENCES**


nation (coproxamol) is the prescription opioid that is responsible for most opioid overdose deaths, and is associated with acute cardiovascular collapse when taken with alcohol (Mehta et al, 2004).

Over-the-counter medication

Antihistamines

Mild antihistamine overdose produces nausea, vomiting, headache, diaphoresis. Results of biochemical tests are often normal. In the third stage, occurring between 72 and 96 hours, there is clinical deterioration, with gastrointestinal bleeding, coagulopathy, hypoglycaemia, renal failure and electrolyte imbalance. Death can occur.

Beyond this comes the fourth stage (after 96 hours), which is one of recovery.

Antidotes to paracetamol overdose include methionine and acetylcysteine. The latter protects against liver damage if given within eight hours. There have been no deaths among those given acetylcysteine within 16 hours of paracetamol ingestion (Hersh et al, 2000).

However, it can also be administered beyond 24 hours to positive effect (Hamm, 2000).

Aspirin

Ingestion of 6–10g of aspirin produces a serious overdose in an adult (Hersh et al, 2000). Initial symptoms include gastrointestinal upset, diaphoresis, fever and tinnitus (with or without hearing loss).

Later symptoms include agitation, lethargy, hallucinations, seizures, hyperthermia, metabolic acidosis and clotting inhibition. Sodium bicarbonate increases urinary excretion and haemodialysis is indicated in serious cases (Muller, 2003).

Ibuprofen

Ibuprofen has a relatively low toxicity, even in overdose. The main symptoms are gastrointestinal disturbances (nausea, vomiting, abdominal and epigastric pain) and tinnitus. Symptoms usually arise within four hours of ingestion. Haematemesis, drowsiness, ataxia, dizziness, headache, nystagmus, hypotension, hypokalaemia or hyperkalaemia, convulsions, coma and apnoea can occasionally occur (Hartley, 2003; Hersh et al, 2000).

Other drugs

Cocaine

Cocaine overdose produces hypertension and tachycardia with concurrent constriction of the coronary vessels. The reduction in blood supply in the presence of increased demand leads to myocardial ischaemia, myocardial infarct, cardiac dysrhythmia and acute pulmonary oedema (Finnell and Harris, 2000).

Administering benzodiazepines decreases the sympathomimetic action of cocaine. However in unresponsive hypertension nitroprusside or phentolamine can be used. For cocaine-induced chest pain, the patient should be oxygenated and given aspirin and diazepam, and sublingual nitroglycerine can be used for unresponsive pain.

For benzodiazepine-resistant supraventricular tachycardia, adenosine and labetalol can be used. Note that using lidocaine for ventricular dysrhythmias can worsen cocaine toxicity (Finnell and Harris, 2000).

Amphetamine

Amphetamine overdose can result in cardiac dysrhythmias, tachycardia, hypertension, angina, acute myocardial infarction, congestive heart failure, seizures, hyperthermia and intracranial haemorrhage. There can be later complications including rhabdomyolysis, renal failure and disseminated intravascular coagulation (Finnell and Harris, 2000).

Theophylline

The initial symptoms of theophylline overdose include nausea, vomiting, abdominal pain, haematemesis, diarrhoea, ataxia, agitation, tremor, hyperventilation and tachycardia (Colbridge, 2001). Occasionally there can be hallucinations and electrolyte imbalances.

In severe cases there can be hypotension, convulsions, dysrhythmias and cardiac arrest. Most theophylline preparations are modified release, so the effects of an overdose can be delayed for up to 24 hours. It should be noted that in vomiting patients it is safe to give ranitidine for excess acid, but not cimetidine as it increases theophylline’s half-life by 60 per cent (Colbridge, 2001).
Psychosocial needs
Meeting the physical needs is only one part of the nursing care required for cases of intentional overdose, as these patients are psychologically vulnerable. Common feelings that overdose survivors experience are shame, guilt and feeling ‘exposed’, with a desire to remain invisible to the health care workers around them (Wiklander et al, 2003).

A patient’s lack of communication is an obvious response to these feelings. The most immediate helpful nursing response is therefore to interact with the patient in a kind, caring and respectful way (Wiklander et al, 2003). It is important not to get anxious at not knowing the solution to the patient’s problems. Do not moralise, and do not coerce or threaten with dire consequences should they do this again (Chiles and Strosahl, 1995).

While not condoning the patient’s behaviour, the most helpful attitude to adopt is one of understanding that human suffering sometimes results in drastic and mistaken responses. This attitude is most evident in a willingness to actively listen to the patient, and to express empathy for their experience (Schnyder et al, 1999).

Psychotherapy
Clearly, the psychological traumas that patients have to live with are not going to be resolved in A&E. Since psychotherapy can help reduce repeated suicidal behaviour (Salkovskis et al, 1990), follow-up counselling or mental health intervention can be a vital determinant in the patient’s future outcome. However, mental health resources for such patients can be lacking (Kerkhof, 1994) and patients can be resistant to help even when it is available (Wiklander et al, 2003).

Given what we know about patients’ predispositions towards intentional overdose behaviour, it is not surprising that many go on to repeat the behaviour, with fatal consequences. In one study 12 per cent of intentional overdose patients repeated their overdose, almost half of these took place within three months (Boyes, 1994). Others found that one-quarter of suicidal adolescent patients repeated their suicide attempt within a year (Hulten et al, 2001).

There are two specific brief psychological interventions that can be useful in caring for intentional overdose cases:

- Solution-focused therapy (SFT) takes a future-oriented stance to identify immediate mechanisms for change (Sharry et al, 2002);
- Motivational interviewing (MI) (Miller and Rollnick, 2002) has also been developed to help clinicians address a range of health-related behaviours (Rollnick et al, 1999).

One A&E psychiatric liaison team treated 40 intentional overdose patients with SFT, of whom only one repeated the overdose behaviour within six months (Wiseman, 2003). According to Sharry et al (2002), SFT:

- Highlights patients’ strengths rather than weaknesses;
- Focuses upon exceptions to the problem rather than the problem itself;
- Seeks out solutions and practical goal-setting by using scaling questions and visualising success.

SFT is a collaborative intervention that facilitates patients’ own problem-solving and is intentionally brief in application.

Although SFT and MI were developed independently, the similarities in their approach have not gone unnoticed (Lewis and Osborn, 2004). MI is a patient-centred counselling approach, directed at resolving the ambivalence thought to lie at the heart of behaviour change. By engaging the patient in a discussion of both the perceived benefits and costs of a particular behaviour, the aim is to increase the discrepancy between the patient’s values, ambitions and ideal behaviour, and their actual current behaviour.

By avoiding an emotional and/or moralistic interaction with the patient, the nurse can treat overdose behaviour as a ‘matter-of-fact’ response to emotional suffering, explore the perceived benefits of this to the patient, and also the less positive consequences. This opens up the opportunity to discuss alternative coping responses, and how one might go about learning them.

Conclusions
Intentional overdose behaviour can lead to a fatal patient outcome and is best understood from a biopsychosocial perspective. Its nature can provoke a range of distressing reactions in nurses. However, the acute nurse who is supported and supervised and can develop a realistic awareness and acceptance of their human abilities, limitations and responsibilities can provide a range of therapeutic nursing interventions that address patients’ physical and psychosocial needs.

GUIDED REFLECTION
Each week we publish a guided reflection article to help you with your CPD. After reading the article use the following points to help you write your reflection:

- Explain why you read this article;
- Describe the last patient that you cared for following intentional overdose;
- Outline the most important points that the article makes;
- Write about how you will use this information the next time you care for a patient who has taken an overdose;
- Summarise what you have learnt and how you will follow this up.

REFERENCES
