

Guidelines for the medical management of patients with methamphetamine-induced psychosis



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1 Background

These guidelines for the management of patients with methamphetamine-induced psychosis have been prepared to aid emergency, general, medical and psychiatric staff in the treatment of these patients in the emergency setting. These guidelines have been developed in consultation with and guidance from experts from the fields of drug and alcohol treatment, emergency medicine and psychiatry. They are based on the national and international literature and on clinical experience with methamphetamine-induced psychosis patients in Australia. The lack of evidence based guidelines for the management of methamphetamine-induced psychosis was the major impetus to the development of these guidelines. Given this, much of the evidence-base for these guidelines is drawn from the literature addressing other psychotic conditions, particularly schizophrenia.

These guidelines are structured to provide an overview of the current literature on methamphetamine psychosis, followed by suggested treatment guidelines for acute presentations of methamphetamine psychosis. An abbreviated version of the guidelines is also provided.

1.1 Aims

The major aims of the guidelines are:

- to update and incorporate experience with new medications to improve the medical treatment of this condition
- to provide advice based on the latest evidence and expert opinion
- to provide consistent care for patients across different jurisdictions
- to promote productive and effective treatment alliances between mental health, emergency medicine and drug and alcohol professionals leading to good clinical practice for this group of patients

These guidelines should be considered in conjunction with the document: *Management of Patients with Psychostimulant Toxicity: Guidelines for Emergency Departments*¹.

2 Amphetamines

Amphetamines are central nervous system stimulants with sympathomimetic and adrenergic agonist activity. Amphetamine administration increases mood, arousal and physical activity²⁻⁵ and produces an increase in heart rate and blood pressure.^{2,6-9} Both methamphetamine and its principal metabolite amphetamine are indirectly acting sympathomimetic drugs with both central and peripheral actions. They act by releasing and inhibiting the reuptake of three neurotransmitters: dopamine, noradrenaline and serotonin.^{10,11} Additionally, amphetamine displaces newly synthesised neurotransmitters from their respective vesicular stores by acting on the vesicular monoamine transporter, thereby increasing the pool of cytoplasmic transmitter available for release. Amphetamines can also inhibit the metabolism of these neurotransmitters by inhibiting the action of monoamine oxidase.¹²

2.1 Pharmacokinetics

Both methamphetamine and amphetamine are readily absorbed from the gastrointestinal tract and the nasal mucosa, and freely penetrate the blood brain barrier.¹³⁻¹⁵ Both are mainly excreted unchanged in the urine via the organic cation transport system.^{16,17} Via this route, amphetamines are excreted more rapidly in an acidic environment¹⁸. Following oral administration, the oral bioavailability of methamphetamine is around 67%¹⁶ and the elimination half-life around 10 hours.¹⁹ In addition to elimination of unchanged drug via the urine, both amphetamine and methamphetamine are metabolised by Phase I (oxidation) and Phase II (conjugation) reactions. In Phase I metabolism, both amphetamine and methamphetamine – as substrates for the cytochrome P450 systems – undergo N-dealkylation and hydroxylation reactions. Peak plasma concentrations occur at 2–4 hours following oral ingestion of amphetamine.^{20,21} Maximum cardiovascular effects of orally administered amphetamine occur at one hour and maximum observer-rated activation and subjective effects at 1½ to 2 hours post-ingestion. However, subjective and behavioural effects then decline despite substantial amphetamine concentrations.^{7,22}

2.2 Route of administration

Both vapour inhalation (smoking), and intravenous injection of amphetamine are increasingly common routes of administration in different populations of users.²³⁻²⁶ Both routes of administration produce rapid and intense pharmacologic effects that provide strong positive reinforcement for repeated use. Intravenously administered methamphetamine has a mean plasma half-life of 11–12 hours.^{14,16} Both inhalation of methamphetamine vapour and injection of methamphetamine show similar parameters.¹⁶

2.3 Neurotoxicity associated with methamphetamine use

Human and animal models of dose dependent dopamine transporter loss associated with methamphetamine use have been developed.²⁷⁻²⁹ Specifically, controlled studies in methamphetamine users have identified significant dopamine transporter reductions in the striatum³⁰ and in the orbitofrontal cortex, dorsolateral prefrontal cortex, and amygdala.³¹ In the latter study, dopamine transporter reduction in the orbitofrontal cortex and the dorsolateral prefrontal cortex was positively related to the duration of methamphetamine use and with the severity of persistent psychiatric symptoms. Volkow and colleagues³⁰ identified a reduction in dopamine transporters in the striatum of methamphetamine users who had been abstinent for at least 11 months. This was associated with motor slowing and memory impairment. Further studies have indicated that these effects may be partially reversible with protracted abstinence (12–17 months).³² However, the extent of recovery may not be sufficient to restore full cognitive function.³³

2.4 Prevalence of amphetamine use in Australia

Of Australians aged 14 years and older, 3.2% had used amphetamines in the previous 12 months.³⁴ Of this group, 10.8% used amphetamines at least once a week, and 16.1% had used about once a month. Using the multiplier-benchmark method, McKetin and colleagues³⁵ estimated that the number of regular methamphetamine users in Australia was 102 600, or 10.3 per 1000 persons aged 15–49 years. Of these regular methamphetamine users, it was estimated that there were 72 700 dependent methamphetamine users, or 7.3 per 1000 population aged 15–49 years.³⁵ It is important to be aware that amphetamine use is likely to be a part of a polydrug use pattern. For example, in the National Drug Strategy Household Survey,³⁴ recent users of amphetamines (used in the previous 12 months) had used alcohol (87.2%) cannabis (67.6%) and ecstasy (49.4%) (on at least one occasion) concurrently with amphetamine. Similarly, studies of methamphetamine users in other jurisdictions have identified alcohol and cannabis as the principal drugs used in conjunction with methamphetamine.^{36–40}

2.4.1 Prevalence of amphetamine use among mental health patients

Substance abuse and dependence are common among psychiatric patients.^{41,42} In addition to the negative consequences of substance use, patients with both an Axis I disorder and a substance use disorder may experience an exacerbation of their psychiatric symptoms.⁴³ Additionally, substance use disorders in mental health patients may compromise their ability to derive benefit from treatment. For example, patients with comorbid substance use and psychiatric disorders are more likely to be non-compliant with prescribed medication or to experience negative side effects resulting from mixing prescribed medications and alcohol or illicit drugs.^{44,45}

2.4.2 Amphetamine-related hospital attendances

The most recent data from the South Australian arm of the Illicit Drug Reporting System (IDRS) showed that amphetamine-related attendances to the Royal Adelaide Hospital (RAH) emergency department have increased, with the number of attendances in 2004–05 for amphetamines being higher than for any other illicit drug. Additionally, attendances for drug-induced psychosis have doubled in the last year. Although, the present coding system does not allow the quantification of drug-induced psychosis by drug,⁴⁶ it is likely that amphetamine-induced psychosis forms a substantial proportion of these episodes.

3 Amphetamine-induced psychiatric symptoms

Psychiatric symptoms are common among methamphetamine users. Almost half (49%) of a sample of current amphetamine users interviewed had been diagnosed or treated for a mental health problem and these problems had occurred commonly after the commencement of regular amphetamine use.⁴⁷ A recent study of 309 regular methamphetamine users (used at least monthly) found that 13% of participants screened positive for psychosis, and 23% had experienced a clinically significant symptom of suspiciousness, unusual thought content or hallucinations in the past year. Methamphetamine users who were dependent on the drug were three times more likely to have experienced psychotic symptoms than non-dependent methamphetamine users.⁴⁸

3.1 Methamphetamine psychosis

Methamphetamine psychosis is characterised by persecutory delusions, auditory or visual hallucinations, strange or unusual beliefs, thought reading, ideas of reference and delusions of reference and thought insertion. There may be high levels of suspiciousness in a state of clear consciousness. The patient may lack insight and high levels of fear may lead to aggressive behaviour, particularly where persecutory beliefs are held.⁴⁹⁻⁵¹ The incidence of methamphetamine psychosis is related to high frequency use of high doses.⁵² However, incidence and severity of the psychosis experienced by patients are related to dosage and route of administration, particularly where rapid routes of administration such as injecting and inhalation of vapour are used.⁵³⁻⁵⁵ Sleep deprivation is also believed to exacerbate psychotic symptoms⁵⁶ and, in patients using methamphetamine on a 'run', may also increase the severity of symptoms. In addition, as previously mentioned, patients with co-morbid psychiatric and substance use disorders may experience an exacerbation of their psychiatric symptoms.⁴³

3.2 Diagnosis of methamphetamine psychosis

The current empirical and clinical evidence does not provide a clear distinction between methamphetamine psychosis and psychosis due to other factors. Particularly in the case of an acute presentation, distinguishing between methamphetamine-induced psychosis and another psychotic illness such as schizophrenia is very difficult and it is often only in retrospect that a diagnosis can be made. Accurate diagnoses of methamphetamine-induced psychoses rely on information that patients or their families provide about methamphetamine use. However, in acute situations, this information may be unavailable.

There is a general consensus that presentations of methamphetamine psychosis closely resemble those of paranoid schizophrenia and can mimic mania.^{51,56} The positive symptoms of methamphetamine psychosis are similar to those of paranoid schizophrenia, consisting mainly of delusions (particularly of persecution, but also ideas of reference) and hallucinations. The recurrent nature of methamphetamine psychosis is also suggested as another apparent similarity, as recurring methamphetamine psychosis may mimic the clinical course of endogenous schizophrenia.⁵⁷ Delusions of persecution are almost universally noted to be characteristic of methamphetamine psychosis, and findings of ideas of reference are also common.^{58,59} Auditory hallucinations are reported by some research to be more prevalent in this disorder than visual,^{60,61} and tactile hallucinations.⁵³ The absence of thought disorder in many published reports of methamphetamine psychosis was thought to be a major distinguishing feature of methamphetamine psychosis.⁶⁰ However, this symptom was observed in later studies of methamphetamine psychosis.⁶² The variation in the clinical presentations of methamphetamine psychosis in the literature compounds the already difficult task of differentiating this disorder from schizophrenia.

3.3 Duration of methamphetamine psychosis

Methamphetamine psychosis usually abates rapidly (within days) with the cessation of amphetamine intake, restorative sleep and the elimination of amphetamines.⁶² However, in some cases psychosis may persist for several years⁶³ and around 5–15% of the users who develop methamphetamine psychosis fail to recover completely.⁶⁴ Furthermore, once the psychotic state develops with methamphetamine use, recurrence can happen in response to psychological stressors even in the absence of further methamphetamine use.⁶¹ Psychotic symptoms may also be a feature of the amphetamine withdrawal syndrome. For example, in a recent study of 21 patients undergoing treatment for methamphetamine withdrawal, two developed auditory hallucinations within the first four days of abstinence.⁶⁵

3.4 Recurrence of methamphetamine psychosis

The recurrent nature of methamphetamine psychosis has been well documented.^{63,66,67} The triggers for methamphetamine psychosis relapse reported include methamphetamine⁶⁷ or other drug use (including alcohol),⁵⁷ psychosocial stressors,⁶⁸⁻⁷¹ or other nonspecific stimuli.⁶⁶ Notably, if methamphetamine use is recommenced, a significantly shorter period of abuse is apparently sufficient to reproduce the psychotic state than that which produced the initial episode.⁷² Recurrences of methamphetamine psychosis are reported to closely resemble previously experienced episodes.⁶¹

3.5 Medical management of methamphetamine-induced psychosis

There is little empirical evidence on which to base treatment approaches to methamphetamine-induced psychosis.⁵⁰ Most of the published evidence on methamphetamine psychosis treatment consists of case reports or small open label studies. The results of two early studies in amphetamine users show that agitation and some psychotic symptoms may abate within an hour after antipsychotic injection. In the first study of eight amphetamine psychotic patients, the symptoms of excitement and paranoid ideation were significantly decreased within 60 minutes of haloperidol intramuscular administration.⁷³ In the second study, the results of a randomised trial in 146 acutely agitated methamphetamine users showed that droperidol, a butyrophenone, can sedate patients significantly faster than lorazepam at 10, 15, 30 and 60 minutes after the intravenous administration.⁷⁴ Case studies of methamphetamine psychosis have also reported good responses to olanzapine,⁷⁵ risperidone⁷⁶ and quetiapine.⁷⁷

A study of methamphetamine psychosis in South Australia found that each interviewed patient was treated with both antipsychotic medication and benzodiazepines during their inpatient stay. Olanzapine was the most commonly used atypical agent (76% of patients), and of the typical antipsychotics, haloperidol was most commonly administered. Some patients also received more than one type of antipsychotic or benzodiazepine during their admission.⁷⁸ Clinical reports and the limited research evidence available suggest that the most commonly used agents for the acute treatment of methamphetamine-induced psychosis are benzodiazepines and antipsychotics.

3.6 Pharmacology of antipsychotics

All currently effective antipsychotic drugs provide some degree of dopamine receptor blockade and are generally more effective toward positive than negative and cognitive symptoms of schizophrenia.⁸⁰ Antipsychotic medications can be divided broadly into two categories: 'typical' or first generation and 'atypical' or second generation antipsychotics. Table 1 shows the currently available atypical antipsychotics in Australia.

Table 1 Atypical antipsychotics currently available in Australia

Generic Name	Brand Name	Preparation
Amisulpride	Solian ®	Tablet, liquid
Aripiprazole	Abilify ®	Tablet
Clozapine	Clopine ®	Tablet
Clozapine	CloSyn ®	Tablet
Clozapine	Clozaril ®	Tablet
Olanzapine	Zyprexa ®	Tablet, injection
Olanzapine	Zyprexa Zydis ®	Wafer
Quetiapine (fumerate)	Seroquel ®	Tablet
Risperidone	Risperdal ®	Tablet, liquid, wafer
Risperidone	Risperdal Consta ®	Depot injection

Data in Table 1 derived from: Rossi S. *Australian Medicines Handbook* 2006. Adelaide, South Australia: Australian Medicines Handbook Pty Ltd; 2006.

Typical antipsychotics include the butyrophenones (e.g. haloperidol), phenothiazines (e.g. chlorpromazine) and thioxanthenes (e.g. zuclopenthixol). Haloperidol is considered to be the prototypic first generation antipsychotic and is generally used as a comparator in evaluation studies of antipsychotic therapy. The second generation or atypical antipsychotics include olanzapine, clozapine, quetiapine, and risperidone. Atypical antipsychotics are a heterogeneous group of drugs and most have a multireceptor profile.

In a review of the onset of action of antipsychotics, Agid and colleagues⁷⁹ concluded that the onset of the antipsychotic effect occurs within 24 hours. Moreover, the antipsychotic effect is specific to antipsychotic medications and can be distinguished from behavioural sedation. This onset of antipsychotic action occurs with either oral or parenteral administration of either typical or atypical antipsychotics. While most of the antipsychotic effects occur within the first two weeks of treatment, the greatest improvement occurs in the first month of treatment.

When administered at clinically effective doses for the treatment of psychotic disorders, typical antipsychotics induce elevated levels of serum prolactin and extrapyramidal adverse effects. Tardive dyskinesia can occur with long-term treatment. The adverse effects associated with typical antipsychotics are listed below:

- Dystonias, such as oculogyric crisis, torticollis, opisthotonos or laryngeal dystonia. These can occur soon after antipsychotic drug therapy is commenced.
- Parkinsonism or rigidity may occur within the first days or weeks after starting antipsychotic therapy.
- Akathisia or restless legs may occur as well as agitation.
- Tardive dyskinesia, characterised by repetitive involuntary movements, usually of the mouth and tongue is often irreversible.
- Hyperprolactinemia leading to galactorrhea.

In contrast to first generation antipsychotics, second generation antipsychotics are 'atypical' because, as a number of studies have shown, they provide effective control of psychotic symptoms while eliciting low or negligible levels of extrapyramidal adverse effects and tardive dyskinesia.⁸¹ Of the second generation antipsychotics, olanzapine and risperidone can be considered high potency as they exert their antipsychotic effects at relatively low doses in comparison to other atypical antipsychotics such as clozapine and quetiapine which require relatively higher dose due to their lower potency.⁸²

There is a large body of evidence showing that second generation antipsychotics produce fewer extrapyramidal adverse effects in comparison to haloperidol.⁸³⁻⁸⁶ Further, the atypical antipsychotics have demonstrated equivalent or superior control of psychotic symptoms in comparison to typical antipsychotics.^{84,86,87} Because of the superior efficacy of the newer antipsychotics and the better side effect profile, atypical antipsychotics have become first line treatments for psychotic disorders, particularly in the case of first episode psychosis.⁸⁹ One exception to the preference for atypical medications for the treatment of psychotic disorders is clozapine which has been associated with the risk of fatal agranulocytosis during long-term therapy. However, there are still significant side effects associated with long-term use of the atypical antipsychotics. Olanzapine and clozapine in particular have been associated with substantial weight gain and the increased risk of diabetes.^{90,91} However, the balance of evidence suggests that of the atypical antipsychotics, the superior efficacy and better side effect profile of the atypical antipsychotics risperidone and olanzapine make them the preferred first line treatment of acute psychosis in inpatients with schizophrenia.^{82,89}

Current theories suggest that the 'atypicality' of second generation antipsychotics is a function of their low affinity for the dopamine D₂ receptor in comparison to first generation antipsychotics. That is, atypical antipsychotics are loosely bound to and rapidly released from D₂ receptors. Importantly, atypical antipsychotics bind more loosely to the D₂ receptor than the endogenous ligand (dopamine) while typical antipsychotics bind more tightly.⁸¹ The choice of dose and type of antipsychotic medication rests on achieving a balance between effective symptom control and the minimisation of adverse effects. The clinical efficacy of antipsychotic medications is associated with a block of 60–80% of D₂ receptors⁹² while extrapyramidal adverse effects are associated with a D₂ receptor block in excess of 70%.⁹³

Although atypical antipsychotics are grouped together in comparison to typical antipsychotics, it is clear that these second generation antipsychotics are a heterogeneous group of drugs that vary in their efficacy and side effect profile. While the current research evidence has identified generally greater efficacy, more head-to-head studies would clarify treatment choices. What is clear from the evidence is that all atypical antipsychotics have a lower risk of extra pyramidal adverse effects and hyperprolactinemia in comparison to typical antipsychotics. The advantages of atypical compared with typical antipsychotics can be summarised as follows:

- Greater therapeutic effect in some treatment-resistant patients
- Greater efficacy for negative symptoms and cognitive deficits
- Reduced risks of extrapyramidal adverse effects including long-term effects
- Reduced risk of hyperprolactinemia
- Improvement in depressive symptoms
- Increased medication compliance

The combination of greater efficacy and reduced risk of adverse effects are important considerations which may balance the substantially greater cost associated with atypical antipsychotic therapy.

4 Proposed treatment guidelines

The proposed treatment guidelines outlined below are based on currently available evidence and expert opinion in the field. The key principles underlying this document are the safety and well-being of individuals affected by psychosis, their families and associates as well as the health of other professionals involved in their care.

4.1 Initial contact

Assessment of the psychotic patient will vary with the setting and degree of behavioural and cognitive disturbance. Where the degree of disturbance is such that a standard medical assessment may be made, a drug and alcohol history should form part of that assessment. Where the patient is fearful and agitated, the goal of treatment should be to ensure their immediate safety as well as the safety of other individuals in the vicinity. For all patients exhibiting symptoms of psychosis, the initial goal of treatment should be to provide reassurance and engage the patient in the therapeutic relationship.

Symptoms of agitation will prevent a complete assessment and may substantially interfere with treatment. Therefore, the initial goal is to reduce agitation and gain cooperation in order to facilitate a complete assessment. Organic causes such as alcohol intoxication, or delirium due to other causes may need to be excluded. Agitated patients should be approached in a low-key manner with the main aim of reducing the potential for an exacerbation of symptoms.

4.2 Assessment

The initial phase includes an assessment of immediate risks to the patient and to others. Reassurance and specific feedback should be provided at this stage. Where this first level of intervention is successful in gaining the patient's cooperation, a more complete assessment may be made. This assessment should address:

1. Vital signs, blood sugar level, oximetry
2. Orientation
3. Insight
4. Delusional thinking
5. Types and nature of any hallucinations
6. History of psychiatric illness
7. Medical history
8. Current medications
9. Drug use history, specifically:
 - number and types of different drug types used (both licit and illicit)
 - changes in recent use patterns, particularly escalation
 - time and amount of most recent use of each drug
 - route of administration
 - presence of injection sites or 'track marks'
10. Recent sleep patterns

4.3 Investigations

The choice of investigation will depend on the setting and available resources as well as other practical considerations such as expense. Organic screening tests for medical illness may vary from none to extensive, depending on the individual clinical presentation. This decision is based on clinical history and examination. Investigations may include a drug screen to confirm drug-related presentations especially when patients deny using substances. Consider urine or saliva drug screen, blood or breath alcohol concentration. However, drug screening is imperfect and can be confounded by false positives and negatives. If there is a history of drug or alcohol use then there may be no need to confirm recent drug use by testing.

4.4 Treatment

The first hour of care is critical to a successful outcome for patients presenting with methamphetamine psychosis. Early expert clinician input at this stage will often de-escalate the situation and allow the initiation of assessment and treatment. At this stage, many patients will accept medication where it is later refused. This initial phase is therefore a critical time in the management of these patients. Nurse-initiated guidelines for an oral regime of benzodiazepine administration (see Section 4.6 Step 1) should be commenced at this stage depending on the level of agitation (see Appendix 1). It is also important to encourage the patient to eat and drink to restore deficits from overactivity and self-neglect. This is particularly important, as it is possible that the patient will enter into a period of extended sleep as recovery takes place. A team approach is often required. This may include a nurse, medical officer, appropriately trained security guard, psychiatric team and drug and alcohol workers.

4.5 Medication

The aims of the medication protocol should be the safe containment and management of the disturbed individual and further to facilitate a restorative sleep. This may be particularly helpful where sleep deprivation is a feature of the presentation. Because of their safety and efficacy, the expert panel proposed oral benzodiazepines, particularly lorazepam as the first choice of medication for methamphetamine psychosis. Surveys of emergency room patients with psychotic disorders show that benzodiazepines are preferred to other forms of sedation.^{94,95} Antipsychotic medication should only be administered if benzodiazepines prove ineffective. Where use of an antipsychotic becomes necessary, the expert panel identified olanzapine as the most suitable because of its availability as an oral or intramuscular preparation. However, an alternative oral atypical antipsychotic could be utilised if that was the local preference. Utilisation of the sedative properties of antipsychotic medication is the main indication for their use. Antipsychotic medication should be continued until the sedative effect is evident and the patient is either asleep or calm enough for a more thorough assessment. Physical restraint should only be used as a last resort and only until chemical restraint is achieved. For many patients, methamphetamine psychosis is a transitory state that will resolve with restorative sleep, medication and the elimination of the drug from the body. Continuation of antipsychotic medication beyond this acute phase is usually unnecessary after 72 hours and should be avoided longer term.

4.6 Stepped care approach

1. Offer oral medication early using a nurse initiated protocol. Initial dose: oral lorazepam 2mg up to 4mg within an hour and repeat again an hour later if necessary. Dosing should be prompted by the score on the Level of Agitation (LOA) Scale (see Appendix 1). Medication should be offered if the score is over 4.
2. If the patient is highly agitated, the LOA >4, and oral medication is refused, administer midazolam 5mg i.m. Repeat in 10 minutes if behaviour remains uncontrolled.
3. If either of these steps fail to achieve control with two appropriate doses, give olanzapine. Antipsychotics should be given if the LOA >4 after benzodiazepine management. Begin with olanzapine 10mg oral or if oral refused give i.m. Repeat one hour later if agitation is still present.
4. If steps 1–3 fail, consideration needs to be given to repeating the steps for i.m. midazolam (see Step 2).
5. Use physical restraints only as a last resort if the steps above have failed. Appropriate close supervision is necessary. Sudden unexpected deaths are more common when physical, rather than chemical restraint is used.
6. If an antipsychotic is used, its use must be reviewed within three days. Given the (usually) short-term nature of amphetamine-induced psychosis, and the potential for harmful side effects associated with this group of medications, routine ongoing administration of antipsychotic medication can usually be avoided.

Management of these patients should ideally be in a quiet place with constant observation and preferably with consistency of staffing. Patients should be monitored in a low stimulus environment and should remain visible at all times.

4.7 Duration of treatment

1. The aim of initial treatment is to settle behaviour.
2. Once compliant when taking oral medication only, benzodiazepines should be used until the diagnosis is clarified. This may take up to five days of observation.

Once behaviour is contained and the patient is no longer psychotic, referral to a drug and alcohol facility should be considered. In many instances the involvement of these services will be as an outpatient.

4.8 Once the agitation resolves

Initiate the assessment protocol (see section 4.2 and 4.3) to elucidate psychotic symptoms and differentiate methamphetamine-induced psychosis or psychosis of another origin. This evaluation will help to clarify the diagnosis. Scales such as the Brief Psychiatric Rating Scale,⁹⁶ and the Positive and Negative Symptom Scale⁹⁷ may be used to augment the assessment in clinical practice.

The presentation of the patient at the emergency department provides an opportunity to link the patient with appropriate health services, such as drug and alcohol, and general medical services. Health risk assessments (blood-borne virus screens, general health screens etc.) can also be performed at this time.

An empathic, educative approach that summarises the facts elicited in the history and physical examination and encourages patients to link their presenting physical and psychosocial problems and the biochemical findings to their drug use will, in most cases, result in an acceptance of the diagnosis. Establishing a supportive, non-judgmental relationship that encourages active participation facilitates this. Motivation to change is reinforced by helping patients weigh up the costs and benefits of their continued drug use and by stressing the benefits of a drug-free lifestyle. Actual change requires the development of a clear, mutually acceptable treatment plan that structures specific interventions to meet the needs of the individual. The involvement of drug and alcohol staff can help to facilitate this process.

4.9 Follow-up

A comprehensive discharge summary and treatment plan will contribute substantially to effective treatment on future presentations. Ideally, this plan should be available to all those likely to be involved in the patient's care such as GPs, acute hospital emergency staff, drug and alcohol services and mental health services. Information detailing effective management of the patient, the pattern of presentation and recovery should be included in the documentation.

4.10 Limitations of the evidence

Antipsychotics for agitation have only been studied in patients with schizophrenia, not drug-induced psychosis, and no randomised controlled trials have investigated treatment and agitation resolution in patients with methamphetamine-induced psychosis.

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Appendix 1

Level of Agitation Scale

Item	Score
Patient is asleep.	1
Patient is awake but calm, without verbal aggression or agitation.	2
Patient is angry, but this is primarily focused on the situation, and requests are not delivered in an obviously threatening or aggressive manner.	3
Patient is awake and agitated with some verbal outbursts but no physical aggression.	4
Patient is severely agitated with extreme verbal outbursts and/or physical aggression.	5

Reproduced from the Royal Adelaide Hospital Emergency Department *Policy and Protocol for the Use of the Safe/Seclusion Rooms* form.