syndrome occur less often with atypical antipsychotics than with the typical agents.

**PATHOPHYSIOLOGY**

The prevailing theory of depression implicates an imbalance in various neurotransmitters and their receptors. Pharmacologic therapy has been engineered to neuromodulate these imbalances. Consequently, the signs and symptoms seen in a significant overdose of an antidepressant medication are the results of gross derangement of one or more neurotransmitters.

**TRICYCLIC ANTIDEPRESSANTS**

TCAs have similar ring structures and, with only a few exceptions, result in a related toxicity. Examples are amitriptyline (Elavil), imipramine (Tofranil), and doxepin (Sinequan). The five major pharmacologic effects of TCAs are listed in Table 147.1.

**MONOAMINE OXIDASE INHIBITORS**

Phenelzine (Nardil) and tranylcypromine (Parnate) are the two most commonly prescribed MAOIs in the United States, and they account for the majority of toxicity seen with this class of agents. The pharmacologic effects of MAOIs are listed in Box 147.1. These effects in overdose may result in a sympathomimetic toxidrome followed by profound hypotension. A therapeutic dose of an MAOI can interact with certain foods, drinks, and other pharmacologic agents and cause serious toxicity.

**SELECTIVE SEROTONIN REUPTAKE INHIBITORS**

Selective SRI antidepressants commonly prescribed are sertraline (Zoloft), paroxetine (Paxil), fluoxetine (Prozac), citalopram (Celexa), and escitalopram (Lexapro). The clinically beneficial central nervous system (CNS) effects of SRIs are thought to result from blockade of presynaptic reuptake of serotonin at 5-hydroxytryptamine type 1 (5-HT₁) receptors. This blockade leads to higher synaptic serotonin levels and hence has positive effects on mood. Overdoses of these agents are much safer than overdoses of TCAs or MAOIs, although significant morbidity and mortality may occur with significant syndrome.

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**KEY POINTS**

- Central nervous system depression is the most common sign of antidepressant or antipsychotic overdose.
- Tachycardia, hypotension, seizures, and ventricular dysrhythmias can also occur, especially after tricyclic antidepressant (TCA) overdose.
- Airway intervention, benzodiazepines, intravenous fluids, and cooling measures (especially for serotonin syndrome and/or neuroleptic malignant syndrome) are the mainstays of supportive care and treatment.
- Specific treatment options include sodium bicarbonate for TCA toxicity and crystalloid fluids or hemodialysis for lithium poisoning.
- Controversial treatment modalities include dantrolene for toxin-induced hyperthermia, cyproheptadine for serotonin syndrome, bromocriptine for neuroleptic malignant syndrome, and prophylactic magnesium for long QTc intervals without evidence of hypomagnesemia or torsades de pointes.

**EPIDEMIOLOGY**

Data reported from United States poison control centers reveal that toxic exposures from antidepressants and antipsychotic agents continue to remain significant (Figs. 147.1 and 147.2). Tricyclic antidepressant (TCA) and monoamine oxidase inhibitor (MAOI) overdoses have historically resulted in the most significant morbidity and mortality. Currently, however, these agents are prescribed much less frequently than serotonin reuptake inhibitors (SRIs), atypical antipsychotics, and lithium. Atypical antipsychotic agents have largely replaced the older typical agents because these newer agents effectively reduce hallucinations, restructure thinking, and control agitation while assisting with the negative effects of psychotic disorders (flattened affect, avolition, social withdrawal). In addition, movement disorders such as dystonia, akathisia, tardive dyskinesia, and neuroleptic malignant syndrome occur less often with atypical antipsychotics than with the typical agents.
**Fig. 147.1** Trends in exposure to atypical antipsychotics and antidepressants. (Compiled from the National Poison Data System, 2001 to 2009.)

**Fig. 147.2** Trends in deaths related to atypical antipsychotics and antidepressants. (Compiled from the National Poison Data System, 2001 to 2009.)

**BOX 147.1 Pharmacologic Effects of Monoamine Oxidase Inhibitors**

Inhibition of monoamine oxidase isoenzymes that results in excessive activity of epinephrine, norepinephrine, serotonin, and tyramine

Effects on exogenous amphetamines and methamphetamine

Depletion of norepinephrine stores

Inhibition of pyridoxine-containing enzymes

**BOX 147.2 Examples of Atypical Antipsychotic Medications**

- Clozapine (Clozaril)
- Risperidone (Risperdal)
- Quetiapine (Seroquel)
- Ziprasidone (Geodon)
- Aripiprazole (Abilify)
- Paliperidone (Invega)
- Olanzapine (Zyprexa; also Symbyax when combined with fluoxetine [Prozac])

**Table 147.1 Five Major Pharmacologic Effects of Tricyclic Antidepressants**

<table>
<thead>
<tr>
<th>PHARMACOLOGIC EFFECT</th>
<th>SYMPTOMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blockade of sodium conductance through fast channels in the myocardium</td>
<td>Prolonged phase 0 of the cardiac action potential that results in a widened QRS complex on an electrocardiogram</td>
</tr>
<tr>
<td>Blockade of potassium efflux</td>
<td>Prolonged phase 3 of the cardiac action potential resulting in an increased QTc interval that lends itself to the development of torsades de pointes</td>
</tr>
<tr>
<td>Peripheral $\alpha_1$-receptor blockade</td>
<td>Vasodilation, decreased perfusion, and hypotension</td>
</tr>
<tr>
<td>Serotonin and norepinephrine reuptake inhibition</td>
<td>Agitation, delirium, or seizure activity</td>
</tr>
<tr>
<td>Anticholinergic activity</td>
<td>Range of physical findings (coma, delirium, urinary retention, mydriasis, seizures, tachycardia, flushing hyperthermia, dry skin)</td>
</tr>
<tr>
<td></td>
<td>“Hot as a hare, dry as a bone, red as a beet, blind as a bat, mad as a hatter, fast as a cat, full as a tick”</td>
</tr>
</tbody>
</table>

overdose or, more commonly, with ingestion of an SRI in combination with ingestion of agents possessing proserotonergic activity.1

**ATYPICAL ANTIPSYCHOTICS**

The pharmacologic mechanism of action of atypical agents includes blockade at dopamine ($D_2$) receptors and serotonin (5-HT$_{2A}$) receptors.2 These agents can also cause repolarization abnormalities by blocking potassium efflux in the myocardium and thereby increasing the risk of torsades de pointes (Box 147.2).

**LITHIUM**

The lightest metal known, lithium is in the same group of elements as sodium and potassium and therefore has similar chemical properties. Since the early 1970s, lithium has been
The onset of action occurs within 8 hours, but the effects may not manifest until 24 hours and may last for several days. Agitation, delirium, seizures, coma, and muscular rigidity predominate. Late in the clinical course of a significant poisoning, depletion of catecholamines can result in asystole. In the presence of increased sympathetic tone, rhabdomyolysis may occur.

MAOI interactions with foods or beverages (aged cheeses, fava beans, ales, wines) produce early onset of signs and symptoms within minutes to hours. Because of tyramine’s short-lived action on the adrenal medulla (to increase endogenous amines), these interactions last only several hours. Interactions of MAOIs with other drugs (sympathomimetics, methylxanthines, SRIs, meperidine) also lead to elevated sympathetic tone. This effect manifests within minutes to hours and can last several hours to days.

SEROTONIN REUPTAKE INHIBITORS
Overdose of SRIs causes CNS abnormalities (sedation, agitation, delirium), peripheral alterations (tremor, hyperreflexia, rigidity), cardiovascular changes (tachycardia, bradycardia), nausea or vomiting, and lightheadedness. The patient with citalopram or escitalopram overdose should be observed for seizures and QTc and/or QRS interval lengthening. Although isolated SRI ingestions frequently result in only mild toxicity, severe overdose or concomitant ingestion of proserotonergic medications can lead to serotonin excess and serotonin syndrome (Fig. 147.3). A history of ingestion of serotonergic agents, altered mental status, autonomic instability, and peripheral signs of rigidity or hyperreflexia are usually present.

ATYPICAL ANTIPSYCHOTICS
Patients usually present within a few hours of atypical antipsychotic overdose with signs of CNS depression (sedation,
confusion, coma). Hypotension and reflex tachycardia from peripheral vasodilation may also occur. Miosis may lead the examiner to consider opioid poisoning. QTc prolongation can be seen in therapeutic use, as well as in overdose. Other adverse effects, which are less commonly seen with the newer agents, are acute dystonias, akathisia, and tardive dyskinesia.

The most significant extrapyramidal effect is neuroleptic malignant syndrome (NMS).\textsuperscript{12} NMS results when dopamine-blocking agents yield “dopamine-depleted” activity at D\textsubscript{2} receptors in the CNS. Although NMS can occur after an intentional overdose, it usually arises after an increase in dose or after the addition of agents with similar activity (e.g., lithium inhibition of dopamine secretion). Manifestations of NMS include CNS abnormalities (sedation, agitation, delirium), peripheral alterations (tremor, hyperreflexia, rigidity), and cardiovascular changes with autonomic instability (tachycardia, bradycardia, hyperthermia) much like those seen in serotonin syndrome. Unlike serotonin syndrome, in which onset of symptoms is normally rather quick, NMS occurs insidiously. Historical information and medication lists are often required to differentiate between the two conditions (Table 147.2).

**LITHIUM**

The clinical effects of lithium overdose are gastrointestinal (nausea, vomiting, and diarrhea), neurologic (tremor, confusion, ataxia, weakness), and cardiovascular (QTc prolongation, bradycardia, T-wave flattening or inversion, bundle branch blocks). Adverse effects include nephrogenic diabetes insipidus, polyuria, psoriasis, alopecia, edema, and leukocytosis. Gastrointestinal distress is usually one of the first manifestations of lithium toxicity. Many presentations are chronic and result from continued lithium administration in the presence of dehydration (decreased fluid intake or vomiting and diarrhea). Physiologically, lithium cannot be differentiated from sodium and is retained by the kidney in patients with dehydration. The results are greater CNS levels and subsequent toxicity. Additionally, changes in a patient’s renal function may decrease lithium clearance.

### Table 147.2 Comparison of the Manifestations of Serotonin Syndrome and Neuroleptic Malignant Syndrome

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>SEROTONIN SYNDROME</th>
<th>NEUROLEPTIC MALIGNANT SYNDROME</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Drug(s) with serotonergic activity</td>
<td>Dopamine-blocking agents</td>
</tr>
<tr>
<td>Time of onset</td>
<td>Hours</td>
<td>Days</td>
</tr>
<tr>
<td>Mental status</td>
<td>Agitation to coma</td>
<td>Agitation to coma</td>
</tr>
<tr>
<td>Tone</td>
<td>Rigidity, greater in lower than in upper extremities</td>
<td>“Lead-pipe” rigidity</td>
</tr>
<tr>
<td>Vital signs</td>
<td>Hypertension, tachycardia, and hyperthermia</td>
<td>Hypertension, tachycardia, and hyperthermia</td>
</tr>
</tbody>
</table>

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Any sedating agent (e.g., opioids, ethanol, benzodiazepines) should be considered in the differential diagnosis of most antidepressant and antipsychotic overdoses. Serotonin syndrome (e.g., SRIs, ecstasy, meperidine, lithium, dextromethorphan, t-tryptophan), neuroleptic malignant syndrome (e.g., antipsychotics such as phenothiazines), malignant hyperthermia (e.g., volatile anesthetic agent use), sympathomimetic overdose (e.g., cocaine, amphetamines), and MAOI overdose or drug-food interaction should also be considered (Box 147.3).

**TRICYCLIC ANTIDEPRESSANTS**

The differential diagnosis of TCA overdose should be broader and should include anticholinergic and antihistamine products (e.g., diphenhydramine) and agents that can poison fast sodium channels, thereby lengthening the QRS interval (e.g., type I antidysrhythmics, cocaine, diphenhydramine, propoxyphene, carbamazepine, cyclobenzaprine, phenothiazines). Life-threatening features are hyperthermia associated with mental status changes, autonomic instability, and tremors, clonus, and rigidity. Life-threatening toxicity should be anticipated in an adult who has ingested TCA doses of 10 mg/kg or greater. Qualitative urine screens for TCAs are of no diagnostic benefit. Although quantitative serum levels of TCAs greater than 1000 ng/mL (therapeutic, 50 to 300 ng/mL) have been correlated with severe toxicity, quantitative testing may not be available in a timely fashion. In addition, depending on the time from ingestion, the type of TCA taken, and the chronicity of dosing, patients may be very ill with serum levels much lower than 1000 ng/mL. An electrocardiogram (ECG) is the diagnostic test of choice.\textsuperscript{14} Normal ECG findings do not fully exclude TCA poisoning, but QRS prolongation greater than 120 msec should be a threshold for treatment (Fig. 147.4).\textsuperscript{14} Cardiac monitoring helps discern the severity of...
study showed that the sensitivity of a QRS interval longer than 100 msec can be matched by two other parameters: the terminal 40 msec of lead aVR measuring longer than 3 mm (R wave in aVR > 3 mm), and a ratio of R-wave to S-wave amplitude in the aVR lead greater than 0.7.\(^4\) (Fig. 147.5).

toxicity. Maximal limb-lead QRS interval duration is a sensitive indicator of illness.\(^5\) Generally, QRS intervals longer than 100 msec are associated with a 33% incidence of seizure activity, and QRS intervals longer than 160 msec are associated with a 50% incidence of ventricular dysrhythmia. One

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**Fig. 147.4** Electrocardiogram showing signs of poisoning by a tricyclic antidepressant. Tachycardia and severe sodium channel poisoning are evidenced by a significantly widened QRS interval.

**Fig. 147.5** The electrocardiogram of a patient poisoned by a tricyclic antidepressant. The tracing shows mild tachycardia and QRS interval lengthening in addition to a noticeable terminal R wave in lead aVR.
**SECTION XV  ■  TOXICOLOGIC EMERGENCIES**

**MONOAMINE OXIDASE INHIBITORS**
A clinical diagnosis (largely based on historical facts) is required for MAOI overdose. No laboratory (urine or blood) test is readily available to make a diagnosis of MAOI poisoning.

**SEROTONIN REUPTAKE INHIBITORS**
The diagnosis of SRI toxicity is purely clinical. The criteria for serotonin syndrome are met through focused elicitation of historical information and the finding of signs and symptoms consistent with the disorder (Table 147.3).

**ATYPICAL ANTBIPSYCHOTICS**
Blood or urine testing plays no role in the diagnosis of atypical antipsychotic overdose. Although QTc prolongation can occur after both therapeutic and toxic ingestions, this finding is not specific for these agents. NMS is a clinical diagnosis coupled with diligent history taking.

**LITHIUM**
Clinical suspicion, historical features, and serum lithium concentrations form the basis of the diagnosis of lithium poisoning. Serum concentrations must be interpreted in the context of chronicity and timing of ingestion. Because long-term users of this drug have higher CNS levels, ill effects occur at lower serum levels. Additionally, lithium has a long distribution time after absorption. In light of this feature, measurement of a serum lithium level less than 6 hours after the last dose may yield an excessively elevated concentration (therapeutic, 0.6 to 1.2 ng/mL). Generally, serum lithium levels greater than 4 ng/mL in acute ingestion and greater than 2.5 ng/mL in chronic poisoning are considered significant.

### Table 147.3 Criteria to Determine Serotonin Syndrome and Toxicity

<table>
<thead>
<tr>
<th>Sternbach’s diagnostic criteria for serotonin syndrome</th>
<th>Hunter’s criteria for serotonin toxicity (context of serotonergic medications)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Recent addition or increase of proserotonergic medication</td>
<td>1. If patient has spontaneous clonus, serotonin toxicity present</td>
</tr>
<tr>
<td>2. At least three of the following:</td>
<td>2. If no spontaneous clonus, one of the following needed for a diagnosis of serotonin toxicity:</td>
</tr>
<tr>
<td>• Agitation</td>
<td>• Inducible clonus and agitation or diaphoresis</td>
</tr>
<tr>
<td>• Ataxia</td>
<td>• Ocular clonus and agitation or diaphoresis</td>
</tr>
<tr>
<td>• Diaphoresis</td>
<td>• Tremor and hyperreflexia</td>
</tr>
<tr>
<td>• Diarrhea</td>
<td>• Temperature &gt; 38°C and ocular clonus or inducible clonus</td>
</tr>
<tr>
<td>• Hyperreflexia</td>
<td></td>
</tr>
<tr>
<td>• Hyperthermia</td>
<td></td>
</tr>
<tr>
<td>• Mental status changes</td>
<td></td>
</tr>
<tr>
<td>• Myoclonus</td>
<td></td>
</tr>
<tr>
<td>• Shivering</td>
<td></td>
</tr>
<tr>
<td>• Tremor</td>
<td></td>
</tr>
<tr>
<td>3. Neuroleptic agent not added or dose increased before the onset of symptoms</td>
<td></td>
</tr>
<tr>
<td>4. Diagnosis of infections, withdrawal, and other poisoning or metabolic disruptions excluded</td>
<td></td>
</tr>
</tbody>
</table>

**TREATMENT**

**TRICYCLIC ANTIDEPRESSANTS**
The treatment of TCA overdose depends on symptoms and is most effectively judged from the ECG (Fig. 147.6). Decontamination is best done early after the overdose. Gastric lavage can be considered after life-threatening ingestion and early presentation (<1 hour). The mainstay of decontamination is activated charcoal. The risks of each technique should be weighed against the potential benefits. Unruly behavior, seizure activity, decreased mental status, and loss of airway reflexes are poor predictors of success and thus raise the risk of aspiration.

Focused therapy consists of serum alkalinization with intravenous sodium bicarbonate (NaHCO₃). Administration of boluses of 1 to 2 mEq/kg is accompanied by close examination of the QRS interval. Boluses should be repeated every 5 minutes until QRS widening resolves, dysrhythmias occur, or blood pH exceeds 7.55. Rarely, hypertonic saline solution can be considered for prolonged QRS intervals and severe alkalemia. NaHCO₃ drips—3 ampules added to 1 L of 5% dextrose in water (D₅W) and given at rates of 2 to 3 mL/kg/hour—and hyperventilation with ventilatory support are considered adjuncts to NaHCO₃ bolus therapy. Serum potassium levels should be monitored with this therapy, and potassium losses should be replaced as necessary.

Seizure activity should be managed with sedatives such as benzodiazepines and barbiturates. If muscle paralysis is necessary, continuous electroencephalographic techniques should be used to measure occult seizure activity. Lidocaine is an alternative to NaHCO₃ therapy for dysrhythmias. Class IA, IC, and III antidysrhythmics, beta-antagonists, and calcium channel blockers are contraindicated in the patient with TCA overdose. Flumazenil and physostigmine are also poor treatment strategies because they have been reported to cause seizure activity and asystolic arrest, respectively.

**MONOAMINE OXIDASE INHIBITORS**
Decontamination techniques in patients with MAOI overdoses should include activated charcoal and, possibly, gastric lavage for significant ingestions without contraindication. No specific antidote is effective. Supportive care should be provided for hemodynamic compromise and/or hyperthermia. Beta-blockers and calcium channel blockers for treatment of tachycardia or hypertension should be avoided because of the theoretical concern of unopposed peripheral α₁-adrenergic vasoconstriction and worsening hypertension or the development of hypotension and bradycardia, respectively. Rather, phentolamine (bolus of 5 mg in adults and 0.02 mg/kg to 0.1 mg/kg in children, repeated in 5 to 10 minutes as needed) or nitroprusside (0.3 mcg/kg/minute titrated to effect) should be considered for hypertensive emergencies. Ventricular...
**SEROTONIN REUPTAKE INHIBITORS**

Standard activated charcoal decontamination should be employed for SRI overdose. Supportive care of the airway, breathing, and circulation encompasses the majority of treatment. In cases of serotonin syndrome, aggressive cooling measures for hyperthermia, benzodiazepines for agitation, and intravenous fluids are usually the only interventions
Table 147.4  Treatment of Non–Tricyclic Antidepressant Overdoses

<table>
<thead>
<tr>
<th>DRUG</th>
<th>GASTROINTESTINAL DECONTAMINATION</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lithium</td>
<td>Consider whole-bowel irrigation for sustained-release formulations</td>
<td>IV fluids at 1.5-2× maintenance levels</td>
</tr>
<tr>
<td></td>
<td>(acute ingestions)</td>
<td>Hemodialysis in severe cases</td>
</tr>
<tr>
<td>Monoamine oxidase inhibitors</td>
<td>Activated charcoal</td>
<td>For hypertension: IV phentolamine 2-5 mg infused over several minutes or</td>
</tr>
<tr>
<td></td>
<td>For significant ingestions, lavage before charcoal</td>
<td>nitroprusside 0.3 mcg/kg/min titrated to effect</td>
</tr>
<tr>
<td></td>
<td></td>
<td>For dysrhythmias: lidocaine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cooling with mist and fan</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Benzodiazepines for agitation</td>
</tr>
<tr>
<td>Selective serotonin reuptake</td>
<td>Activated charcoal</td>
<td>Supportive care</td>
</tr>
<tr>
<td>reuptake inhibitors</td>
<td></td>
<td>Aggressive cooling</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Benzodiazepines for cooling and/or agitation</td>
</tr>
<tr>
<td>Atypical antipsychotics</td>
<td>Activated charcoal</td>
<td>For life-threatening hyperthermia: sedation, paralysis, and ventilation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

I/V, Intravenous.

required to decrease the risk of death. Life-threatening hyperthermia should be treated with sedation, neuromuscular paralysis, and ventilatory care. The use of cyproheptadine (8 mg by mouth; up to 24 mg per day) for serotonin syndrome has been described but remains controversial. Bromocriptine has been reported to worsen serotonin syndrome, and dantrolene (proven effective for malignant hyperthermia) should be considered only for life-threatening hyperthermia and significant rigidity.

**ATYPICAL ANTIPSYCHOTICS**

Acute overdose of atypical antipsychotic agents is managed with supportive care because no specific antidote exists. These agents bind to activated charcoal, use of which should be the standard mode of decontamination. Correction of electrolyte (potassium, magnesium, and calcium) disturbances helps prevent widening or further lengthening of the QTc interval. The prophylactic use of magnesium to prevent a widened QTc interval from degenerating into torsades de pointes has no proven benefit when magnesium concentrations are normal. Treatment of NMS is for the most part identical to that of serotonin syndrome (aggressive cooling, benzodiazepines for agitation, fluids, and ventilatory support as warranted). Bromocriptine, an antihistamine with dopaminergic activity, has been used without consistent benefit. Dantrolene, which works peripherally to inhibit release of calcium from the sarcoplasmic reticulum, has never been proven to be of benefit in NMS but should be considered for the patient with significant rigidity and life-threatening hyperthermia.

**LITHIUM**

Treatment of lithium poisoning depends on the clinical context. Activated charcoal is contraindicated because lithium does not bind to it, and whole-bowel irrigation is considered only with ingestions of sustained-release products in patients with no contraindications. Sodium polystyrene sulfonate has been shown to bind to lithium in vitro, but in clinical use it requires excessive dosing at the risk of potentially causing hypokalemia.

Enhancing elimination through hemodialysis is a controversial topic in the setting of lithium poisoning. The patient likely to benefit is the one with acute ingestion, mental status abnormalities, and/or significant renal dysfunction or pulmonary edema. Many patients experience lithium “redistribution” and an asymptomatic period after hemodialysis. This result often leads to further hemodialysis sessions, but whether this approach has an ultimate beneficial outcome remains controversial. The more common approach, barring any of the preceding abnormalities, is fluid hydration. Lithium’s clearance depends on the glomerular filtration rate, which, in turn, depends on volume status. Dehydrated patients continue to reabsorb, rather than eliminate, lithium because of its physical characteristics. Crystalloids given at two times maintenance doses should suffice. Forced diuresis or diuretic therapy has no role in this situation.

Table 147.4 summarizes treatment of overdoses involving antidepressant and antipsychotic agents other than TCAs.

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION**

Any patient with a deliberate overdose of an antidepressant or antipsychotic medication or with clinical symptoms should be admitted to the hospital. Patients who are asymptomatic 6 hours after ingestion can be medically cleared for psychiatric evaluation. The exceptions are patients with elevated serum lithium values, who warrant further observation and subsequent lithium measurements, and patients with overdose of an
MAOI agent, which may not manifest for 24 hours. Patients should be admitted to the intensive care unit for any mental status changes that require close observation for loss of airway reflexes or seizure activity. In addition, patients with cardiovascular abnormalities, especially those requiring treatment in the emergency department with NaHCO₃, lidocaine, or other cardiovascular drugs, merit disposition to a critical care unit.

### TIPS AND TRICKS

- Although normal electrocardiographic findings do not fully exclude tricyclic antidepressant (TCA) poisoning, QRS prolongation to more than 100 to 120 msec should be a threshold for treatment.
- Generally, QRS duration longer than 100 msec is associated with a 33% incidence of seizure activity, and a QRS greater than 160 msec is associated with a 50% incidence of ventricular dysrhythmia. Cardiac monitoring is a valid way of discerning the severity of toxicity in TCA poisoning.
- Devastating hemodynamic instability follows seizure activity in 13% of patients with TCA poisoning.
- Rightward deviation of the terminal 40-msec QRS axis (R wave in aVR > 3 mm) should be a cause for concern in a patient with TCA overdose.
- Significant, late monoamine oxidase inhibitor poisoning results in asystole from depletion of catecholamines, so affected patients should be monitored for 24 hours.
- Atypical antipsychotic toxicity can be associated with sedation or coma and miosis and therefore can be confused with opiate intoxication.
- Lithium toxicity is often precipitated by dehydration or worsening renal function.
- A serum lithium value obtained less than 6 hours after the last dose may result in excessively elevated concentrations (therapeutic, 0.6-1.2 mg/mL).

### SUGGESTED READINGS


### REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES