

# Intramuscular Ziprasidone: Moving Beyond the Conventional in the Treatment of Acute Agitation in Schizophrenia

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The appropriate management of schizophrenia and schizoaffective disorder requires effective, safe antipsychotic agents for use across a continuum of treatment, from control of acute psychotic episodes to prevention of relapse. Intramuscular (IM) formulations are the method of choice for administering antipsychotics to schizophrenic patients who require emergency treatment but cannot take oral medication. Atypical antipsychotics are now widely acknowledged as the first-line choice for the management of patients with schizophrenia. However, use of these agents in acutely agitated psychotic patients has been limited by the lack of an IM formulation. Ziprasidone is the first, and currently only, atypical antipsychotic to be available in a rapid-acting IM formulation. This review focuses on studies evaluating the efficacy and tolerability of IM ziprasidone. In agitated psychotic patients, IM ziprasidone reduces agitation as early as 15 minutes after administration, with improvement sustained for  $\geq 4$  hours. In patients with acute psychosis, with or without agitation, IM ziprasidone has been demonstrated to be superior to IM haloperidol in improving overall symptom severity. During the critical IM-to-oral transition, efficacy and tolerability are maintained with ziprasidone. IM ziprasidone represents an important advance over older, conventional IM agents in the treatment of the acutely ill patient with schizophrenia.

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Schizophrenia requires effective strategies across a continuum of care, from emergency management of acute psychotic episodes to long-term maintenance therapy. Intramuscular (IM) formulations of antipsychotics are an important pharmacologic option for the management of acute psychotic symptoms. Through bypassing the gastrointestinal tract and first-pass metabolism, IM formulations offer the advantage of faster onset of therapeutic action and more rapid bioavailability. Until recently, acutely ill patients requiring IM medication have been treated with conventional antipsychotics. Although these agents are effective in controlling the positive symptoms of schizophrenia, they are associated with a wide range of adverse effects.<sup>1</sup> In particular, conventional antipsychotics frequently cause movement disorders such as acute dystonia, parkinsonism, akathisia, rigidity, and tremor and are associated with catatonia and hypotension.<sup>1</sup> Benzodiazepines have often been used concomitantly with antipsychotics in agitated patients. These agents, especially at higher dosages, have been associated with

drowsiness, behavioral changes, ataxia, and respiratory depression (if combined with alcohol or other sedatives).<sup>1</sup> They also have been known to increase agitation and cause assaultive behavior in some patients.

Atypical antipsychotics are now considered first-line agents for the treatment of psychotic disorders,<sup>1</sup> including schizophrenia.<sup>2</sup> They are at least comparable to conventional antipsychotics in the management of schizophrenia and are associated with a lower incidence of movement disorders. However, the use of atypical antipsychotics in the most acutely ill agitated patients has been limited by the lack of an IM formulation.

Ziprasidone is the first, and currently only, atypical antipsychotic available in a rapid-acting IM formulation. IM ziprasidone uses  $\beta$ -cyclodextrin sulfobutyl ether to solubilize the drug by forming a complex.<sup>3</sup> IM ziprasidone achieves peak serum concentration within approximately 30 minutes of administration and has 100% bioavailability with dose-proportional exposure.<sup>4</sup> This pharmacokinetic profile supports the use of IM ziprasidone in the acutely agitated patient, in whom rapid onset of maximal therapeutic effect is desirable. Ziprasidone exhibits a short elimination half-life ( $< 3$  hours),<sup>4</sup> indicating that there is no need for a washout period prior to initiating oral treatment.

Controlled trials reviewed here have shown that ziprasidone rapidly reduces agitation in psychotic patients. In comparative trials versus IM haloperidol, IM ziprasidone

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**Table 1. The 7-Point Behavioral Activity Rating Scale<sup>a</sup>**

Score	Behavior
1	Difficult or unable to rouse
2	Asleep, responds normally to verbal or physical contact
3	Drowsy, appears sedated
4	Quiet and awake (normal level of activity)
5	Signs of overt (physical or verbal) activity, calms down with instruction
6	Extremely or continuously active, not requiring restraint
7	Violent, requires restraint

<sup>a</sup>Adapted with permission from Swift et al.<sup>7</sup>

has demonstrated superior efficacy in reducing overall symptom severity and anxiety, and has exhibited a more favorable tolerability profile with respect to movement disorders than has haloperidol. In clinical trials evaluating the transition from IM to oral treatment, efficacy and tolerability were maintained.

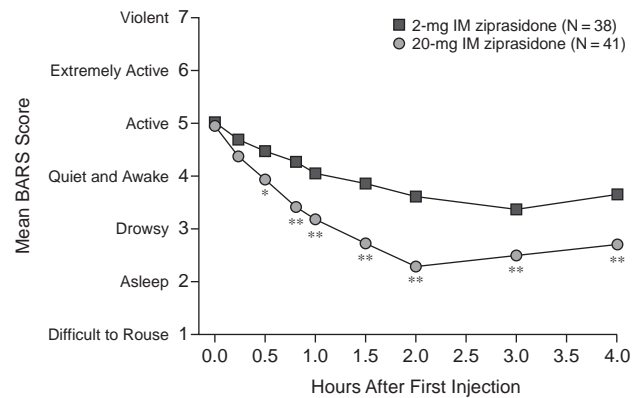
**INTRAMUSCULAR ZIPRASIDONE IN CONTROLLING ACUTE AGITATION**

The tolerability and efficacy of the IM formulation were initially evaluated in a pilot study in which 12 patients with acute schizophrenia (agitation was not an entry criterion) were treated for 3 days with fixed-dose IM ziprasidone (10–60 mg/day) and transitioned to oral ziprasidone (40–160 mg/day).<sup>5</sup> Intramuscular ziprasidone was associated with rapid improvement in overall psychopathology. Numerical improvements (no formal statistical analyses were conducted in this study) in mean Brief Psychiatric Rating Scale (BPRS) total score occurred on day 1 and continued throughout the study. Similarly, there were improvements in the BPRS agitation cluster items anxiety, tension, hostility, and excitement. Although ziprasidone had a tranquilizing effect that, with 20 mg, was apparent within 30 minutes, no excessive sedation was observed. No incidents of extrapyramidal syndrome (EPS), dystonia, or postural hypotension, which are commonly associated with conventional antipsychotics, were reported.

Subsequently, two 24-hour, double-blind, randomized trials were conducted in patients with acute agitation with psychosis.<sup>2,6</sup> These trials were very similar in design, differing mainly in the dose evaluated.

In the first study, agitated adult inpatients with schizophrenia, schizoaffective disorder, bipolar disorder with psychotic features, or other psychotic disorder were randomized to 24 hours of double-blind treatment with 20 mg (N = 41) of IM ziprasidone or a subtherapeutic control dose of 2 mg (N = 38).<sup>6</sup> Following the initial dose, up to 3 additional doses could be administered at ≥ 4-hour intervals as necessary. No benzodiazepines were allowed during the study period. The primary efficacy measure was the 7-point Behavioral Activity Rating Scale (BARS) (Table 1),<sup>7</sup> which was administered before and at frequent defined intervals following dosing. Secondary measures

**Figure 1. Mean BARS Scores 0 to 4 Hours Following Administration of 2 mg (control) or 20 mg of IM Ziprasidone<sup>a</sup>**



<sup>a</sup>Adapted with permission from Daniel et al.<sup>6</sup>

\*p < .01 vs. 2 mg.

\*\*p < .001 vs. 2 mg.

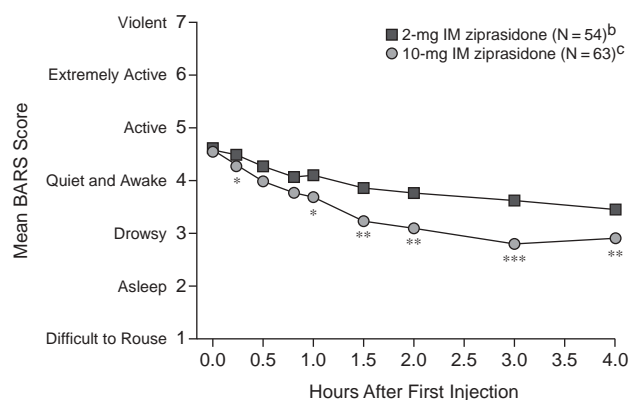
Abbreviations: BARS = Behavioral Activity Rating Scale, IM = intramuscular.

included the 7-point Clinical Global Impressions-Severity of Illness scale (CGI-S), the Clinical Global Impressions-Improvement scale (CGI-I), and the Positive and Negative Syndrome Scale (PANSS) total score and agitation items subscore (the sum of the anxiety, tension, hostility, and excitement item scores).<sup>6</sup>

Improvement in mean BARS score in the 20-mg group was numerically greater than that in the 2-mg control group at 15 minutes after injection of the first dose, and it was significantly greater at 30 minutes after the first injection (p < .01) and at all subsequent time points, from 45 minutes to 4 hours (Figure 1). Improvement in mean BARS score was maximal at 2 hours postinjection,<sup>6</sup> with a significantly greater percentage of patients in the 20-mg group than in the 2-mg group rated as BARS responders (defined as a ≥ 2-point reduction in BARS score) (90.2% vs. 34.2%, respectively; p < .001). Significantly greater improvements in mean CGI-S, CGI-I, and PANSS agitation subscale scores were observed at 4 hours in patients treated with 20 mg of IM ziprasidone versus those treated with 2 mg (p < .05).

Tolerability was comparable in the 2 treatment groups. Only 1 patient (in the 2-mg group) developed mild EPS.<sup>6</sup> Neither akathisia, dystonia, respiratory depression, nor excessive sedation were observed in either treatment group, nor any consistent change in blood pressure or pulse rate. There were no clinically relevant electrocardiographic (ECG) changes, and there was a mean increase in the corrected QT interval (QTc) of 3.6 ms in patients treated with 2 mg of IM ziprasidone and a mean decrease of 1.3 ms in patients treated with 20 mg. No clinically significant changes in QTc were noted in either group.

Figure 2. Mean BARS Scores 0 to 4 Hours Following Administration of 2 mg (control) or 10 mg of IM Ziprasidone<sup>a</sup>



<sup>a</sup>Adapted with permission from Lesem et al.<sup>2</sup>

<sup>b</sup>At baseline, N = 54; at 2 hours, N = 54; at 4 hours, N = 45.

<sup>c</sup>At baseline, N = 63; at 2 hours, N = 62; at 4 hours, N = 55.

\* $p < .05$  vs. 2 mg.

\*\* $p < .01$  vs. 2 mg.

\*\*\* $p \leq .001$  vs. 2 mg.

Abbreviations: BARS = Behavioral Activity Rating Scale, IM = intramuscular.

In the second 24-hour study, 54 patients were treated with 2 mg of IM ziprasidone and 63 were treated with 10 mg of IM ziprasidone.<sup>2</sup> Ziprasidone could be injected at 2-hour intervals (up to 4 injections) as deemed necessary. Lorazepam (up to 8 mg/day) was allowed for agitation and temazepam (up to 30 mg/day) was allowed for insomnia during the 24-hour treatment period, but both were prohibited in the 4 hours before baseline assessment and immediately afterward.

At 15 minutes after the first injection, a significant improvement in mean BARS scores was observed in patients receiving 10 mg of IM ziprasidone versus those receiving the 2-mg dose ( $p < .05$ ). Mean BARS scores in the 10-mg group continued to improve (decrease) up to 2 hours after the first injection and were significantly lower than in the 2-mg group at all subsequent time points from 60 minutes onward (Figure 2). Mean BARS scores remained significantly lower in the 10-mg group than in the 2-mg group ( $p < .01$ ) among patients with assessments at 3 and 4 hours postdose. Mean improvements from baseline in secondary endpoints (i.e., reductions in PANSS total and agitation subscale scores, CGI-I, and CGI-S) were similar in the 2 treatment groups. In this study, the BARS responder rate for 10 mg was 57.1%, which was significantly higher than the 29.6% observed with 2 mg ( $p \leq .001$ )<sup>2</sup> but not as high as that seen with 20 mg (90.2%).<sup>6</sup>

Ziprasidone 10 mg and 2 mg were comparably well tolerated. The most frequently reported adverse events (> 10% of patients) were mild-to-moderate headache and injection-site pain in both groups.<sup>2</sup> In the 10-mg group, 1 patient experienced akathisia, 1, agitation, and 2, dizzi-

ness. In the 2-mg group, 1 patient had EPS, 2 had agitation, and 2 reported dizziness. There was no pattern of clinically important changes in blood pressure or pulse and no clinically important changes in ECG measurements. Mean changes in QTc were  $-3.7$  ms in the 2-mg group and  $-1.8$  ms in the 10-mg group.

## COMPARATIVE TRIALS VERSUS HALOPERIDOL

A randomized, open-label, multicenter study compared sequential IM/oral ziprasidone with sequential IM/oral haloperidol in the treatment over 7 days of 132 inpatients with acute psychotic agitation.<sup>8</sup> Ninety patients were treated for up to 3 days with flexible-dose IM ziprasidone (initial 10-mg dose plus subsequent doses of 5 to 20 mg every 4 to 6 hours as necessary; maximum daily dose of 80 mg). Forty-two patients were given IM haloperidol (initial 2.5- to 10-mg dose, followed by 2.5- to 10-mg doses every 4 to 6 hours as needed; maximum daily dose of 40 mg). The mean ( $\pm$  SD) total IM doses on day 3 for ziprasidone and haloperidol were 27.6 ( $\pm$  21.2) mg and 11.0 ( $\pm$  10.2) mg, respectively. After 3 days, all patients were transitioned to oral ziprasidone (80–200 mg/day) or haloperidol (10–80 mg/day), which continued for an additional 4 days. The mean ( $\pm$  SD) last daily oral dose for ziprasidone was 90.5 ( $\pm$  44.9) mg and 14 ( $\pm$  10.1) mg for haloperidol.

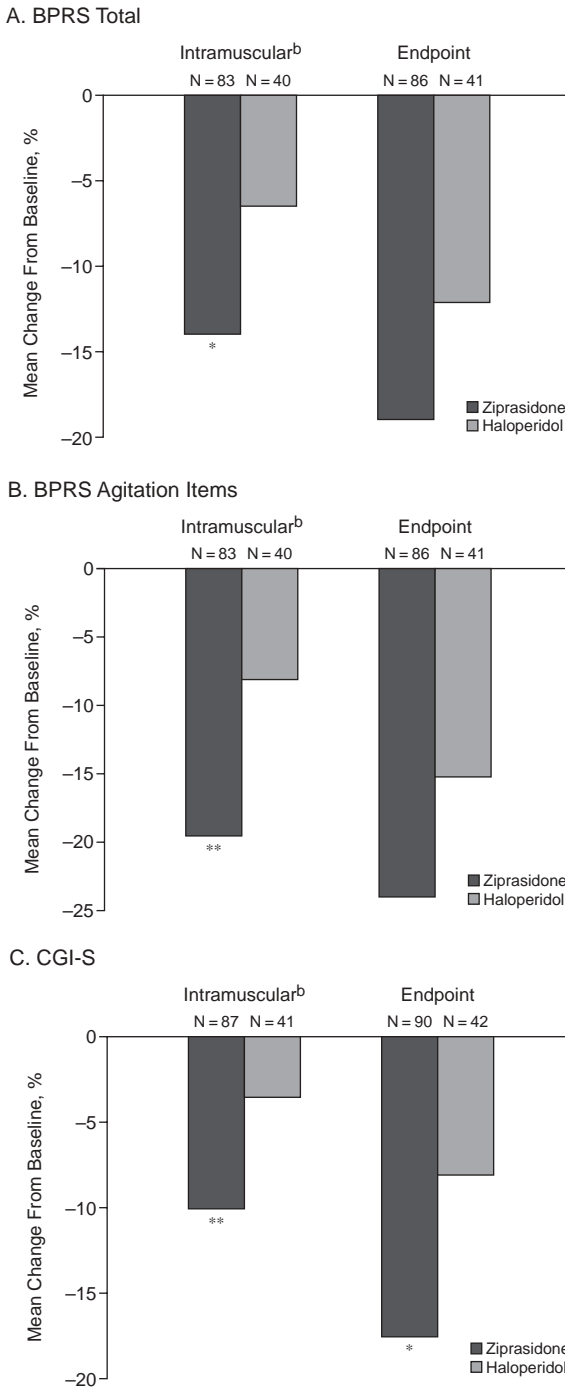
Mean improvements in BPRS total and agitation items scores as well as in CGI-S scores were significantly greater in patients treated with IM ziprasidone than in those assigned to IM haloperidol (Figure 3).<sup>8</sup> Further improvements in all 3 measures were observed following transition to and completion of 4 days of oral treatment. The percentage of patients in the ziprasidone group who required concomitant anticholinergic medication for movement disorders (14.4%) was about one third of that in the haloperidol group (47.6%).

A second 7-day, open-label, multicenter study reported by Swift and colleagues<sup>9</sup> also compared the efficacy of sequential IM/oral ziprasidone with that of sequential IM/oral haloperidol. Two hundred six hospitalized patients with psychotic disorders were treated for 3 days with IM ziprasidone in 3 different fixed doses (up to 80 mg/day) followed by transition to oral ziprasidone (40–200 mg/day). One hundred patients received IM haloperidol (up to 40 mg/day) followed by oral haloperidol (initial dose equal to the last IM dose) with clinical adjustments.

Within 30 minutes of first IM administration, the mean improvement in BARS score was greater in all ziprasidone treatment groups than in the haloperidol group.<sup>9</sup> Tolerability and efficacy were sustained following transition to oral treatment.

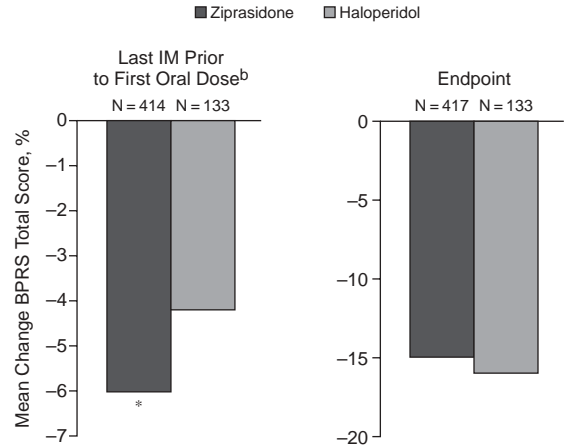
In a 6-week, randomized, parallel-group, rater-blind, flexible-dose study, Brook and colleagues<sup>10</sup> compared the efficacy, safety, and tolerability of sequential IM/oral

**Figure 3. Mean Change From Baseline in (A) BPRS Total and (B) BPRS Agitation Items Subscores and (C) CGI-S Scores After IM Ziprasidone or IM Haloperidol Treatment and at Endpoint (all subjects, observed cases)<sup>a</sup>**



<sup>a</sup>Reprinted with permission from Brook et al.<sup>8</sup>  
<sup>b</sup>Intramuscular denotes last observations after IM injection and before oral administration. The number of patients included in the analysis (N) represents patients who were assessed at baseline and had ≥ 1 postbaseline assessment on IM treatment and at endpoint.  
 \*p < .05.  
 \*\*p < .01.  
 Abbreviations: BPRS = Brief Psychiatric Rating Scale, CGI-S = Clinical Global Impressions-Severity of Illness scale, IM = intramuscular.

**Figure 4. Mean Change From BPRS Total Score at End of IM Treatment and at Endpoint (ITT, LOCF) in a 6-Week Trial of Sequential IM/Oral Ziprasidone Versus Haloperidol<sup>a</sup>**



<sup>a</sup>Reprinted with permission from Brook et al.<sup>10</sup>  
<sup>b</sup>Last IM injection on day 1, 2, or 3.  
 \*p < .004 vs. haloperidol.  
 Abbreviations: BPRS = Brief Psychiatric Rating Scale, IM = intramuscular, ITT = intent to treat, LOCF = last observation carried forward.

ziprasidone and IM/oral haloperidol in patients with acute exacerbation of schizophrenia or schizoaffective disorder. Patients were randomized to IM ziprasidone (10 or 20 mg initially; additional doses of up to 40 mg/day for ≤ 3 days; N = 417) or IM haloperidol (2.5 or 5 mg initially; additional doses of up to 10 mg/day for ≤ 3 days; N = 133). After a minimum of 2 IM doses and completion of applicable rating instruments, patients who were taking IM ziprasidone were transitioned to oral ziprasidone (40 mg b.i.d. initially; adjusted to 40–80 mg b.i.d. as necessary); patients who initially received IM haloperidol were switched to oral haloperidol (5–20 mg b.i.d.).

At completion of the IM phase, in last-observation-carried-forward analysis of the intent-to-treat population, significantly greater improvements in mean scores on the BPRS total (Figure 4) and Covi Anxiety scales were observed with IM ziprasidone compared with IM haloperidol (p < .01).<sup>10</sup> Improvements in mean CGI-S and CGI-I scores were comparable between the 2 treatment groups at the end of IM dosing. Transition to oral dosing was accompanied by comparable clinical improvements in psychometric indices in the 2 groups at the 6-week study endpoint.

Ziprasidone-treated patients were significantly less likely than haloperidol-treated patients to exhibit EPS or akathisia at all study visits (p < .001).<sup>10</sup> Adverse events occurring in > 10% of haloperidol patients were akathisia, dystonia, EPS, hypertonia, and insomnia. In the ziprasidone group, adverse events occurring in > 10% of patients were anxiety, insomnia, and somnolence. No

clinically meaningful changes in QTc interval were noted with either drug.

### TRANSITION TO ORAL TREATMENT

To ensure optimal outcomes across the continuum of treatment, the transition from IM to oral medication must be associated with sustained efficacy and tolerability. Daniel and associates<sup>11</sup> reviewed data from the 3 blinded-assessment multicenter studies discussed above<sup>8-10</sup> in which a total of 1005 inpatients were randomly assigned to sequential IM/oral ziprasidone or sequential IM/oral haloperidol. Results from the two 7-day studies and the one 6-week study support the continued efficacy and tolerability of ziprasidone during this critical transition period.<sup>11</sup> In all studies, sustained efficacy or improvements in measures of disease severity were observed in both drug groups, and no discontinuations because of lack of efficacy occurred during the IM treatment phase.

Importantly, review of the data from the two 7-day studies indicates that no prominent safety issues were associated with the transition from IM to oral ziprasidone.<sup>11</sup> Overall, comparable proportions of patients in both drug groups discontinued for reasons related to the transition from IM to oral study drug in these studies (8.1% of ziprasidone patients vs. 5.6% of haloperidol patients). No significant change in the pattern of safety-related discontinuations was observed with transition for either ziprasidone or haloperidol; however, a greater percentage of patients in the haloperidol group than in the ziprasidone group discontinued treatment (for all causes) during the oral phase (7.5% vs. 3.7%, respectively).

In the 6-week study, fewer patients discontinued ziprasidone than haloperidol during the first 2 weeks of oral therapy.<sup>11</sup> During the first week following transition from IM to oral therapy, the rate of discontinuation for both ziprasidone and haloperidol was < 10%. Also, there were no evident changes in the pattern of safety-related discontinuations during week 1.

### PATIENT ACCEPTANCE

Patient satisfaction with antipsychotic therapy may predict future treatment adherence. During the 6-week IM-to-oral transition trial described above,<sup>10</sup> a subjective 10-question Drug Attitude Inventory (DAI) was administered. Compared with haloperidol, significantly greater improvements were observed with ziprasidone in DAI total ( $p < .01$ ), subjective total ( $p < .001$ ), and subjective positive scores ( $p < .01$ ) after 1 week of treatment, and in DAI subjective total scores at the end of IM dosing (days 1 to 3) ( $p < .05$ ).<sup>12</sup> The authors concluded that patients with acute schizophrenia have better subjective feelings about using ziprasidone than haloperidol, particularly during treatment outset (i.e., IM phase through transition to oral therapy).

### EMERGING PHARMACOECONOMIC INSIGHTS

A model developed using clinical trial data comparing IM ziprasidone ( $N = 90$ ) with IM haloperidol ( $N = 42$ ) in acutely psychotic patients in the emergency department setting suggests that despite the higher acquisition costs of IM ziprasidone in the United States, treatment with IM ziprasidone is more cost-effective than treatment with haloperidol.<sup>13</sup> The excess cost associated with IM haloperidol was largely attributable to emergency department treatment for acute EPS and dystonia.

Many acutely agitated patients require restraint and close monitoring to minimize the possibility of injury. The need for extensive supervision during acute psychotic episodes imposes a high burden, both in terms of time and overall costs, on hospital staff and health care systems. A recent study of the use of ziprasidone in the psychiatric emergency service yielded preliminary data suggesting that introduction of the agent in this setting may reduce restraint time.<sup>14</sup> Mean duration of restraint use in ziprasidone-treated patients ( $N = 69$ ) was half that recorded for a group of agitated patients who had received conventional antipsychotics in the month before study initiation ( $N = 80$ ).

### DISCUSSION

Although atypical antipsychotics are replacing conventional antipsychotics as first-line treatment for schizophrenia, their use for rapid control of acute psychotic agitation is limited considerably by the lack of IM formulations. IM conventional antipsychotics and benzodiazepines are still the mainstay in the acute management of agitated psychotic patients.

At this writing, ziprasidone is the only atypical antipsychotic available in an IM formulation. Cumulative data from the trials presented above show that IM ziprasidone rapidly controls agitation and psychotic symptoms and has a low liability for the adverse effects (particularly EPS) seen with conventional antipsychotic agents. These studies showed that ziprasidone produces dose-related improvements in BARS scores, with the 20-mg dose rapidly producing significantly greater improvements than the 10- or 2-mg (control) doses. Significantly greater improvements in mean CGI-S, CGI-I, and PANSS agitation scores were also seen with 20 mg versus 2 mg IM ziprasidone; improvements in these secondary parameters were comparable between the 2- and 10-mg doses. Current recommendations state that IM ziprasidone 10 mg may be administered every 2 hours as needed and 20-mg doses may be administered every 4 hours up to a maximum of 40 mg/day.<sup>15</sup> Administration of IM ziprasidone for > 3 consecutive days has not been studied.

Intramuscular ziprasidone is also associated with significantly greater mean improvements in BPRS total and

agitation items scores, as well as CGI-S and Covi Anxiety scores, than IM haloperidol, and ziprasidone-treated patients are less likely than haloperidol-treated patients to exhibit EPS or akathisia. No clinically meaningful changes in QTc interval were noted with ziprasidone in these clinical studies.

Furthermore, the transition from IM to oral ziprasidone dosing is well tolerated and sustains symptom control. In comparison with conventional antipsychotics, the improved side effect profile of ziprasidone appears to have a positive effect on patient satisfaction with therapy and may therefore improve compliance with subsequent long-term therapy. IM ziprasidone thus represents a welcome alternative to conventional IM agents in the treatment of patients at the crisis or acute-illness end of the schizophrenia continuum.

*Drug names:* haloperidol (Haldol and others), lorazepam (Ativan and others), temazepam (Restoril), ziprasidone (Geodon).

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