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Introduction

This manual is divided into two parts. Part 1 contains recommendations and guidelines for prescribers who treat consumers with schizophrenia. Part 1 is based on evidence in the psychiatric literature, and, where the evidence is as yet inconclusive, expert opinion. Part 2 is targeted toward organizations that employ or contract with multiple prescribers for the treatment of consumers with schizophrenia. Part 2 is a compilation of the tools that mental health care providers have developed and used to achieve the basic goals of MedMAP: systematic and evidence-based selection and use of medications, measurement of outcomes, methods of documentation that sustain the first two goals, and shared decision-making between prescriber and consumer. Organizations are encouraged to choose from amongst the tools in Part 2 the ones that most closely fit with their resources and mission. These choices are best made collaboratively, involving all key stakeholders.
Recommendations

The recommendations for medication management of schizophrenia that are listed below have been taken from four sources:


- The Mount Sinai Conference on the Pharmacotherapy of Schizophrenia. Schizophrenia Bulletin, 2002; 28(1) 5-16), and a conference of experts to update the schizophrenia TMAP algorithms held in January 2002 in San Antonio, TX (publication in preparation).

Each set of recommendations was based on expert review of the existing literature. Where evidence in the literature was absent, inconsistent, or weak, the authors used the expert consensus method. Since the TMAP update conference was most recent and included many of the participants in the Mt. Sinai conference, the recommendations reflect the group consensus at that time with regard to issues where newer data were available (e.g. safety of ziprasidone). Following the style of the Mt. Sinai Consensus Conference, the recommendations are posed as questions, followed by the consensus answer, and a brief synopsis of the rationale. More detailed discussion can be found in Marder et al., 2002. A brief section on drug interactions follows the recommendations.
**Question 1. Should conventional agents still be considered first-line agents?**

*Consensus Opinion*: No.

First generation antipsychotics (FGA’s) are those conventional antipsychotic medications that preceded clozapine’s entry into the antipsychotic armamentarium. Second-generation antipsychotics (SGA’s) include clozapine and those agents brought to market following clozapine. At this time in the USA, these agents include clozapine, risperidone, olanzapine, quetiapine, and ziprasidone. Although the SGA’s are discussed as a group, they are a heterogeneous group of medications with different side effect profiles. However, when prescribed at effective doses, all of the SGA’s share the property of being associated with less extrapyramidal side effects (EPS) than FGA’s. This tolerability advantage is the primary reason for recommending SGA’s other than clozapine as first line agents. The evidence for SGA superiority with regard to tardive dyskinesia, cognitive deficits and negative symptoms is discussed later in this section.

FGA’s may be appropriate selections for the following groups of consumers: (1) Individuals who have a history of responding well to a conventional antipsychotic without experiencing EPS; (2) Individuals who have a history of a better response to FGA’s than to SGA’s; (3) Consumers who have responded better to a long-acting FGA depot when compared to oral antipsychotics.

**Question 2. Should ziprasidone be a first line agent?**

*Consensus Opinion*: Yes.

The package insert for ziprasidone warns of Q-T interval prolongation, potentially resulting in fatal arrhythmias. At the time of the Mt. Sinai conference, the number of consumer exposures to ziprasidone was insufficient to judge how great the actual risk of sudden death was. By January 2002, however, the number of consumers who had received the drug was up to about 150,000, without evidence of an increased incidence of sudden death. Therefore, the recommendation was to add ziprasidone as a first line agent.

**Question 3. What is an adequate antipsychotic trial duration?**

*Consensus Opinion*: Four to twelve weeks, with the possible exception of clozapine.

It is important to distinguish the duration of a trial needed to convincingly establish non-response from the duration of a reasonable trial for an acute exacerbation. It takes at least four weeks on full therapeutic doses to establish that a consumer is a non-responder. For partial responders, the trial should be extended to as long as twelve weeks. During acute exacerbations, it is often not feasible to wait four weeks and it may be reasonable to switch antipsychotics after as little as one week on a
Clozapine may take longer to have its full effects, though some evidence indicates that, once a therapeutic dose is reached, response will be evident in four weeks. Since clozapine is the last, best hope for consumers with treatment-refractory schizophrenia, it seems wise to err on the side of longer trials with efforts to maximize response by checking blood levels in poor responders.

It is worth noting that the time courses of response reported in the psychiatric literature are for positive symptoms. There are few data on time course of response of, for example, cognitive deficits or functional impairments in schizophrenia, though many clinicians believe, based on their observations, that the time to maximum improvement of these parameters is considerably more prolonged than for positive symptoms.

**Question 4. What is the relative effectiveness of clozapine and other second-generation agents for treatment refractory consumers? How many failed trials, of what, should consumers have before they receive clozapine?**

*Consensus Opinion:* Clozapine is still the treatment of choice for treatment-refractory consumers. Clozapine appears to be the most effective antipsychotic for treatment-refractory consumers. For this reason, consumers should not be considered partial responders or non-responders until they have had an adequate trial with clozapine. Clinicians should assess a consumer’s response to at least one second-generation antipsychotic before beginning clozapine.

**Question 5. Is there sufficient evidence to conclude that second-generation antipsychotics have a lower TD risk?**

*Consensus Opinion:* Yes

There is sufficient evidence to conclude that SGA’s are less likely to cause TD than FGA’s.

**Question 6. Are there characteristics of individuals that should influence antipsychotic selection?**

*Consensus Opinion:* No, with regard to efficacy (other than prior medication failures or non-adherence). Yes, with regard to side effects. In terms of efficacy considerations, there is no evidence that personal or demographic characteristics should guide drug selection. For non-adherent consumers, both depot and SGA’s should be considered before FGA’s. Side effect concerns should be central to medication selection.
Question 7. Are there differences among antipsychotics – FGA’s or SGA’s – in their effectiveness for positive, negative, neurocognitive, aggressive, and mood symptoms?

Consensus Opinion:

- For positive symptoms, there is no convincing evidence of differences among the antipsychotics, with the exception of clozapine’s greater effectiveness in treatment-refractory consumers.
- Some SGA’s produce greater improvement in negative symptoms than FGA’s, but the evidence is not conclusive as to whether these changes are due to improvements in primary or secondary negative symptoms or to improvements in both.
- Clozapine shows mixed effects on neurocognition; other SGA’s may offer benefits for neurocognition, but the evidence is still preliminary and awaits randomized double-blind trials.
- Clozapine may be more effective than conventional antipsychotic medications in reducing aggression. There is insufficient evidence to determine the ability of other SGA’s to reduce aggression.
- Some SGA’s, including clozapine, are more effective than FGA’s for relieving mood symptoms.

Question 8. What should a clinician monitor when prescribing FGA’s and SGA’s?

Consensus Opinion: The SGA’s have focused attention on the need for monitoring metabolic, endocrine and cardiovascular parameters. Recommendations for monitoring and treating traditional antipsychotic side effects, such as EPS and TD, are based on long experience and use generally accepted assessments and frequency of assessments. Recommendations for monitoring weight, glucose, lipids, sexual/endocrine side effects, lens opacities, and cardiac conduction are less firmly grounded in experience with psychiatric populations and are evolving as the levels of risk become more clearly established. To some degree, in the absence of clear evidence or clear expert consensus on what to monitor and how often, clinicians must make rather arbitrary decisions, based on their assessment of the appropriate balance between perceived risk (safety), direct costs of monitoring, and indirect costs of monitoring (such as prescriber or consumer avoidance of what might be an excellent treatment).

Question 9. What are reasonable dose ranges for antipsychotics?

Consensus opinion: The table below lists usual dose ranges for some commonly used antipsychotics. In some instances, there are safety reasons for exceeding, only with caution, the upper end of the dose range (e.g. clozapine and seizures). In most cases, the recommended dose ranges are those found to work well for the majority of consumers. This is not to deny that some consumers do better on doses above or below the recommended range, but it does mean that there should be strong evidence that consumers treated with doses outside of these ranges are indeed more benefited by them than by usual doses.
Antipsychotic Medications

<table>
<thead>
<tr>
<th>Drug</th>
<th>Starting Dose</th>
<th>Usual Dose Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpromazine</td>
<td>50-100mg/d</td>
<td>300-1000mg/d</td>
</tr>
<tr>
<td>Clozapine</td>
<td>12.5mg/d</td>
<td>150-600mg/d</td>
</tr>
<tr>
<td>Fluphenazine</td>
<td>5mg/d</td>
<td>5-20mg/d</td>
</tr>
<tr>
<td>Fluphenazine D</td>
<td>12.5-25mgIM/2-3weeks</td>
<td>6.25-50mgIM/2-4weeks</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>2-5mg/d</td>
<td>2-20mg/d</td>
</tr>
<tr>
<td>Haloperidol D</td>
<td>25-50mgIM/2weeks</td>
<td>50-200mg/2-4weeks</td>
</tr>
<tr>
<td>Loxapine</td>
<td>20mg/d</td>
<td>50-150mg/d</td>
</tr>
<tr>
<td>Molindone</td>
<td>20mg/d</td>
<td>50-150mg/d</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>5-10mg/d</td>
<td>10-20mg/d</td>
</tr>
<tr>
<td>Perphenazine</td>
<td>4-8mg/d</td>
<td>16-64mg/d</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>25mgbid</td>
<td>300-800mg/d</td>
</tr>
<tr>
<td>Risperidone</td>
<td>1-2mg/d</td>
<td>2-6mg/d</td>
</tr>
<tr>
<td>Thiothixene</td>
<td>5-10mg/d</td>
<td>15-50mg/d</td>
</tr>
<tr>
<td>Ziprasidone</td>
<td>20-40mg bid</td>
<td>40-160mg/d</td>
</tr>
</tbody>
</table>

Question 10. Do intermittent dosing strategies work as well as regular dosing for maintenance?

Consensus Opinion: No. Multiple controlled studies have shown that regular administration of antipsychotics is preferable to targeted, intermittent dosing for prevention of relapses.

Question 11. When are plasma levels of antipsychotics useful?

Consensus Opinion: The clearest evidence is for using plasma levels to achieve clozapine concentrations above 300-400 ng/ml in non-responders. There is evidence for a therapeutic window for haloperidol of 3-15 ng/ml. Plasma levels may be of value when medication non-adherence is suspected as a cause of poor medication response.
Question 12. Should anti-Parkinson agents be used prophylactically?

Consensus Opinion: For most consumers there are now effective alternatives to medications which have a high likelihood of producing EPS. For consumers who, for clinical reasons, are considered best treated with an oral or depot FGA, prophylactic anti-Parkinson agents are warranted if they have a history of EPS.

DRUG INTERACTIONS

Since symptoms of depression are common in schizophrenia, antidepressant medications are frequently combined with antipsychotics. Several antidepressant agents have the potential to inhibit antipsychotic metabolism, thus raising the blood level of the antipsychotic. This section primarily discusses pharmacokinetic interactions between antidepressants and antipsychotics. It also provides some general information on the pharmacokinetics of select SGA’s. Information on drug interactions is subject to rapid change, based upon new research findings and clinical experiences. Clinicians are encouraged to consult current references for current drug interactions information. A useful, frequently-updated website for this information is maintained by Dr. David Flockhart at Indiana University (http://medicine.iupui.edu/flockhart).

There are many drug interactions between antidepressants and antipsychotics. Of particular concern with regard to drug toxicity are the inhibitory effects of some antidepressants on clozapine metabolism, leading to increased levels and risk of seizures. Fluvoxamine (Luvox) can cause large increases in levels of clozapine and should be avoided. Other serotonin reuptake inhibitors (SSRIs) and nefazodone may cause clinically significant increases in clozapine levels and should also be used carefully in clozapine treated consumers. Clozapine serum levels should be monitored when adding one of the above antidepressants to clozapine. Because bupropion itself has an inherent risk of seizures, a pharmacodynamic interaction exists with clozapine. Therefore, the combination of clozapine and bupropion should be avoided.

In order to avoid troublesome drug interactions, the following table can be consulted whenever an antidepressant is added to an antipsychotic or whenever either component of an antipsychotic-antidepressant combination is being changed.
### Antidepressant/Antipsychotic Interactions

<table>
<thead>
<tr>
<th>INHIBITOR</th>
<th>SUBSTRATE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>(Inhibits substrate)</strong></td>
<td><strong>(Drug metabolized by pathway)</strong></td>
</tr>
<tr>
<td></td>
<td><strong>1A2</strong></td>
</tr>
<tr>
<td>Bupropion (Wellbutrin)</td>
<td>Phenothiazines (some) Clozapine* Olanzapine*</td>
</tr>
<tr>
<td>Citalopram (Celexa)</td>
<td>Phenothiazines Clozapine* Olanzapine*</td>
</tr>
<tr>
<td>Fluoxetine (Prozac)</td>
<td>Phenothiazines THIORIDAZINE Clozapine* Olanzapine*</td>
</tr>
<tr>
<td>Fluvoxamine (Luvox)</td>
<td>CLOZAPINE HALOPERIDOL OLANZAPINE THIOTHIXENE</td>
</tr>
<tr>
<td>Nefazodone (Serzone)</td>
<td></td>
</tr>
<tr>
<td>Paroxetine (Paxil)</td>
<td>PHENOTHIAZINES THIORIDAZINE Clozapine* Olanzapine*</td>
</tr>
<tr>
<td>Sertraline (Zoloft)</td>
<td>Phenothiazines Clozapine* Olanzapine*</td>
</tr>
</tbody>
</table>

Venlafaxine (Effexor) increases haloperidol levels, but not by Cytochrome P450 interaction.

**Regular type** = small changes in levels (low probability of clinically significant interaction)

**Bold type** = moderate changes in levels (moderate probability of clinically significant interaction)

**BOLD CAPS** = very large changes in levels (high probability of clinically significant interaction)

* Minor pathway

** Fluvoxamine has been shown to inhibit the metabolism of thioridazine but it is unclear whether the interaction occurs at CYP 1A2 and/or CYP 2C19. (Carrillo JA, Ramos SI, Herraiz AG et al., Pharmacokinetic interaction of fluvoxamine and thioridazine in schizophrenic consumers. J Clin Psychopharmacol 1999;19(6): 494-9.)
Risperidone is metabolized through CYP 2D6 to 9-OH-risperidone. However, both risperidone and its metabolite are equally potent, and the sum of the two remains the same with CYP 2D6 inhibition, usually resulting in no change in clinical effect and no need for reduction of the risperidone dose. There are currently no known inducers of CYP 2D6.

Quetiapine is a Cytochrome P450 3A3/4 substrate and, because of the medication’s low bioavailability, clinicians need to be aware of drug interactions that occur through this pathway. It may be necessary to increase the quetiapine dose above 800 mg per day when quetiapine is used with 3A3/4 inducers such as carbamazepine, phenytoin, phenobarbital, etc.

Ziprasidone is metabolized in the liver, primarily through the aldehyde oxidase enzyme system. These enzymes metabolize approximately two-thirds of ziprasidone, they are not known to be significantly inhibited or induced by other medications. Less than one-third of ziprasidone’s metabolism is attributable to the cytochrome P450 enzyme system; therefore it should be safe to combine ziprasidone with most other medications, including the SSRIs. The package insert warns against combining ziprasidone with medications that significantly prolong the QT interval. The drugs to be avoided are listed in the most current package insert and include mesoridazine, thioridazine, chlorpromazine, droperidol, pimozide, quinidine, dofetilide, sotalol, moxifloxacin, and sparfloxacin (not a complete list). The package insert also warns about avoiding the use of ziprasidone in conditions in which there may be QT interval prolongation, such as hypokalemia and hypomagnesemia.

**COMMON QUESTIONS AND PROBLEMS**

This Section and Chapters 2-9 provide information that addresses questions and problems commonly encountered when prescribing medication for persons with schizophrenia. These topic foci are not as amenable to firm recommendations as those addressed in the previous sections but are areas in which knowledge of evidence should influence clinical decisions. The topics that are included were based on a survey of practicing clinicians.

**Next-step strategies for partial and non-responders to initial treatment**

*Adjust the dose based on plasma concentrations*

A number of circumstances can lead to consumers having inadequate blood levels of a drug. The most obvious -- and perhaps the most common explanation -- is that the consumer is not taking the medication as prescribed. In addition, blood levels can be low when the consumer is an efficient metabolizer of a drug or when interactions occur between the antipsychotic and other drugs the
consumer is receiving. In the cases of some antipsychotics – including haloperidol trifluoperazine, perphenazine, risperidone, olanzapine, and clozapine – ordering a plasma concentration of the drug can be helpful when these options are being considered (Marder 2000). The best data is for clozapine (Bell, McLaren et al. 1998), for which a level below 350 ng/ml has been associated with an inadequate response. The evidence is weaker for other agents.

*Increase the dose above the usual range*

Treatment with high doses of antipsychotic was widely studied in consumers receiving conventional antipsychotics. It is instructive to note that during the 1970’s and 1980’s, the practice of prescribing high doses for treatment resistant consumers was very popular. This practice was evaluated in a number of studies that randomized treatment resistant consumers to either high doses or standard doses of drug. The results (reviewed in Thompson 1994)) indicated that higher doses were not associated with additional improvement, but they were associated with more side effects. Unfortunately, there are no controlled studies of higher dose treatment in consumers who have failed to respond to a second-generation agent. Nevertheless, case reports (e.g. Mountjoy, Baldacchino et al. 1999; Reich 1999) and one open-label trial (Lindenmayer, Volavka et al. 2001) indicate that clinicians have identified individual consumers who appear to have responded well when newer agents were raised above the usual level. Other case reports have pointed to adverse effects at higher doses (Bronson and Lindenmayer 2000).

*Change to an antipsychotic from a different class*

Evidence from studies of conventional antipsychotics indicates that if a consumer’s symptoms fail to respond to one antipsychotic, the symptoms will likely fail to respond to other conventional antipsychotics (Kolakowska, Williams et al. 1985). On the other hand, a number of studies have compared second-generation antipsychotics to haloperidol or other conventional agents in consumer’s symptoms that were treatment resistant with conventionals. These studies have found that consumers demonstrated greater improvement on the newer agent (reviewed in Lindenmayer 2000; Chakos, Lieberman et al. 2001). However, one study, comparing olanzapine to chlorpromazine in severely ill, treatment-refractory consumers, found that response rates were low on both agents (Conley, Tamminga et al. 1998). Thus far, published studies have focused on risperidone and olanzapine, and no published, controlled trials have evaluated quetiapine or ziprasidone in consumers whose symptoms have failed to respond to other antipsychotics.
Change to clozapine

Clozapine has been found to be effective for severely ill consumers whose symptoms have failed to respond to other antipsychotics. The best study compared clozapine and chlorpromazine in well-documented refractory consumers (Kane, Honigfeld et al. 1988). Clozapine was associated with greater improvements on a wide range of psychotic and nonpsychotic symptoms. Other studies (reviewed by Chakos, Lieberman et al. 2001) – including a VA Cooperative Study (Rosenheck, Cramer et al. 1997) that compared one year of treatment with clozapine or haloperidol – have confirmed clozapine’s role in these consumers. A review by Chakos et al. (Chakos, Lieberman et al. 2001) concluded that the data supporting clozapine’s effectiveness in treatment-refractory consumers are stronger than the data for other drugs. Although clozapine is clearly effective for treatment-refractory consumers, clinicians are often inclined to select other second generation antipsychotics due to clozapine’s side effect profile and the need for a system for blood monitoring.

Add a second antipsychotic

Issues in antipsychotic polypharmacy are presented in Chapter 6.

Summary

Although clinicians commonly use a number of strategies when a consumer’s symptoms fail to respond to an antipsychotic, most are only supported by relatively weak evidence. The strongest evidence supports switching the consumer to clozapine. A number of controlled studies support the use of other second-generation drugs, if the consumer’s symptoms have failed a trial with a conventional agent.
REFERENCES


How Best to Switch Antipsychotic Medications

INTRODUCTION

Practice guidelines and medication algorithms typically recommend changing from one antipsychotic medication (AP) to another as the preferred initial step for managing inadequate response or intolerable side effects. Details of how to manage switches are often not addressed. Switching APs too rapidly may increase the risk of discontinuation syndromes or relapse while prolonged overlap of medications may unnecessarily expose consumers to synergistic and cumulative adverse effects.

SUMMARY OF KEY INFORMATION

- Both successful outcomes as well as relapses have been reported to follow AP switches that were abrupt (medications substituted) or gradual (cross-tapered, i.e. overlapped use of APs with progressive decrease of “old” agent and initiation at full dose or progressive increase of “new” agent).

- Factors considered to favor a more gradual approach include clinical instability, stable response to clozapine, and high doses of “old” agent.

- Research evidence to directly guide how to optimally switch APs is limited. Good outcomes have been reported following switches where the period for cross-tapering was limited to 1-week and 1-2 months (with the “old” medication being clozapine, duration depended on dose used). (See Discussion.)

- Abrupt discontinuation of APs can be associated with withdrawal symptoms such as nausea, sweating and muscle aches, increased motor symptoms, and relapse of psychotic symptoms. These problems may be mitigated by:
• tapering periods of at least 3 weeks,
• extending anticholinergic medication (when present initially) for at least a few days beyond the last dose of AP,
• substituting new AP. It has been suggested that substitution of agents with overlapping neuropharmacological profiles (e.g. similar relative potency, 5-HT-2 blockade) may provide greater mitigation of discontinuation problems.

**DISCUSSION**

Research evidence is limited in terms of providing guidance on how to optimally switch APs. We are only aware of one study that featured randomization to different switching strategies. To date, this study has not been published by itself, but has been summarized in a review paper. Two hundred twenty-nine consumers whose symptoms were judged to be inadequately responsive to or who were intolerant of prior treatment with olanzapine, risperidone or traditional APs were switched to ziprasidone. This was accomplished by starting ziprasidone at 80 mg per day with randomization to either abrupt discontinuation of the original medication or a one-week tapering period starting at either 50% or 100% of the initial dose. Overall results of the switches were positive and were not reported to differ across the 3 strategies. While not a comparison of strategies, another study reported successfully switching 18 of 20 consumers from clozapine to olanzapine utilizing a cross-titration procedure. Olanzapine was initiated at a daily dose of 5 mg. After 7 days, clozapine tapering began at a rate of 25 mg every other day until it was discontinued. While clozapine was being tapered down, olanzapine was increased to 10 mg with further dose adjustments as indicated. It is recognized that clinical research studies typically feature more frequent monitoring and, sometimes, more stable populations than non-research practice settings.

There is a more extensive research literature, referred to above, that addresses discontinuation of APs. Clearly this issue is distinct from switching. Discontinuation is only recommended with caution, following stable remissions in select circumstances (see first section of manual). However, this more developed literature would seem informative to potential problems of switching. For example, the withdrawal symptoms listed above are attributed to cholinergic rebound. One might be more likely to encounter these during a rapid switch from an agent that is high in anticholinergic activity (e.g. clozapine, traditional low potency neuroleptics) to one that is low in anticholinergic activity (e.g. haloperidol, risperidone, ziprasidone). A reduced risk of relapse has been demonstrated for tapering periods of 3 to 4 weeks compared to abrupt discontinuation. Therefore, one might extrapolate that a gradual cross-tapering strategy would be appropriate when switching APs in an individual who is considered susceptible to relapse. However, the effect of using cross-tapering strategies of different lengths on relapse rates has not yet been demonstrated.
REFERENCES

Assessment and Treatment of Psychiatric Co-morbidity

When treating co-morbid psychiatric symptoms in consumers with schizophrenia, schizophrenia should be thought of as the primary condition that necessitates long-term maintenance therapy while, in general, the co-morbid symptoms should be thought of as more acute symptoms that require shorter-term treatment.\(^1\) One challenge that clinicians face is determining whether or not a consumer’s non-psychotic symptoms are manifestations of his/her primary psychotic symptoms. Weiden has developed the following approach to help clinicians decide whether or not to add an adjuvant medication to treat a consumer’s non-psychotic psychiatric symptoms.\(^2\)
Six common non-psychotic symptoms that can occur in schizophrenia are as follows:

- Depression
- Anxiety
- Obsessions and Compulsions
- Mood Instability
- Insomnia
- Aggression/Hostility

The remainder of this article will focus on the assessment and treatment of these co-morbid conditions.

**Depression**

In treating a consumer with schizophrenia who has symptoms of depression, the clinician must consider the differential diagnosis. A variety of physical illnesses and the prescription medications used to treat them, as well as substances of abuse, can precipitate depression. Assuming that such factors have been ruled out, the clinician should then ask whether the symptom is a temporary reaction to disappointment/stress or the prodrome of a new psychotic episode. Watchful waiting may provide an answer in either case and, in the latter event, the clinician should adjust the consumer’s antipsychotic (AP) medication.

If the depression persists and the consumer’s psychosis is stable, the clinician should then evaluate the consumer’s AP regimen. If a conventional AP is being used, the clinician should be aware that these agents can produce depression-like symptoms through either extrapyramidal side effects (EPS; e.g., akinesia or akathisia) or, directly, through neuroleptic-induced dysphoria. Four possible treatment alternatives are: 1) reducing the dose of the conventional agent (when possible); 2) initiating or increasing the dose of anti-Parkinsonian medication; 3) initiating or increasing the dose of anti-akathisia medication; 4) switching the consumer from a conventional to an atypical antipsychotic.

In the case of persistent depression in a stable consumer who is already on an atypical antipsychotic, the literature offers fewer answers. Clinicians should consider the first three of the four options listed above, especially if the consumer is on risperidone. (In general, it would not be advisable to add an anti-Parkinsonian agent to clozapine since clozapine has high anticholinergic activity and a very low propensity to cause EPS.) If these interventions are not possible or are ineffective, an adjuvant antidepressant medication should be considered. Although most examples in the literature involve the combination of a conventional AP and a tri-cyclic antidepressant, the SSRI’s have also
shown benefit, when used as adjuncts, to treat depression-like symptoms in consumers with schizophrenia. Clinicians must consider potential drug interactions when combining antidepressant and antipsychotic medications. (See Drug Interactions section in Chapter 1.)

**Anxiety**
The adjunctive use of a benzodiazepine is recommended for the treatment of persistent anxiety in consumers who are in the maintenance phase of treatment. Benzodiazepines are also used as adjunctive anxiolytics and sedatives in consumers experiencing an acute psychotic episode. (There is also evidence to suggest that benzodiazepine augmentation of APs may help control core psychotic symptoms.) Alprazolam (Xanax) may have an activating effect in certain consumers and should not be used in consumers with psychotic agitation. Clinicians should not overlook the possibility that akathisia is the underlying problem and that adding a benzodiazepine treats the akathisia. In this instance, it may be preferable to switch to an AP with less potential for causing akathisia.

Some studies have found that the anxiolytic effects of the benzodiazepines diminish after a few weeks, possibly due to tolerance. Clinicians should be cautious about using benzodiazepines in treating consumers with substance abuse issues and in those at risk of abruptly discontinuing the medication and going into withdrawal. Also, consumers whose schizophrenia is complicated by developmental disabilities or traumatic brain injury are more susceptible to benzodiazepine-induced disinhibition, which can lead to a worsening of psychosis.

Buspirone, a non-benzodiazepine antianxiety medication, is an alternative anxiolytic. While buspirone is not thought to be as effective an anxiolytic as the benzodiazepines, its advantage is that it does not have a withdrawal liability.

**Obsessions and Compulsions**
Consumers with schizophrenia often have obsessive-compulsive symptoms (OCS). Although it is easy to attribute these symptoms to the consumer’s psychosis, current evidence suggests that the OCS that consumers with schizophrenia experience are similar to those of non-schizophrenic consumers with obsessive-compulsive disorder. Treatment of OCS in consumers with schizophrenia has not been well studied. Adjunctive serotonergic agents (clomipramine or the SSRI’s) have demonstrated efficacy and safety in open label studies and, in the case of clomipramine and fluvoxamine, one controlled clinical trial. Because of its tolerability problems and potential lethality in overdose, clinicians should exercise caution when prescribing clomipramine.

Another issue in the treatment of OCS in consumers with schizophrenia is the reported emergence or exacerbation of OCS in consumers being treated with atypical APs. Contradictorily, atypical APs have shown efficacy as adjuncts in the treatment of non-schizophrenic consumers with OCD refractory to monotherapy with an anti-obsessional medication. If a consumer with schizophrenia experi-
ences the emergence or exacerbation of OCS during treatment with an atypical antipsychotic, the clinician should either lower the dose of the antipsychotic or wait several weeks to see if the symptoms remit spontaneously. If neither strategy works, an adjunctive serotoninergic agent should be added. As mentioned in the depression section, clinicians must consider potential drug interactions when combining antidepressant and antipsychotic medications. Of special concern is fluvoxamine’s (Luvox) ability to inhibit the metabolism of clozapine and cause toxic clozapine levels.

**Mood Instability**

Adjuvant mood stabilizers are thought to improve manic symptoms such as labile affect, agitated/excited behavior, etc. in selected consumers suffering from schizophrenia. Lithium, carbamazepine, and valproate are the mood stabilizers most commonly used for this purpose and, of the three, valproate is generally preferred. Lithium has shown effectiveness as an adjunct and is well studied but there are several disadvantages to its use. In addition to its intrinsic side effects, lithium can aggravate cognitive problems and EPS. One disadvantage to using carbamazepine is its potential to induce cytochrome P450 enzymes in the liver, which can result in a decrease in antipsychotic serum levels and an exacerbation of psychosis. Concomitant carbamazepine and clozapine use is contraindicated because both agents can cause blood dyscrasias. At present, little is known about the long-term effectiveness of using adjuvant medications to treat mood instability in consumers with schizophrenia.

**Insomnia**

Insomnia can occur during schizophrenia as an acute symptom of psychosis or a more chronic problem related to poor sleep hygiene (daytime naps, caffeinated beverages in the evening, etc.) Benzodiazepines are the most commonly used hypnotic. Other options include zolpidem (Ambien), trazodone (Desyrel), diphenhydramine (Benadryl), hydroxyzine (Atarax, Vistaril) and zaleplon (Sonata). There have been some reports of transient psychotic symptoms occurring in the middle of the night after the use of short-acting sedatives such as triazolam (Halcion) or zolpidem (Ambien). Clinicians should also be aware of the risk of priapism associated with trazodone, but it has the advantage of not being addictive or habituating.

**Aggression/Hostility**

While antipsychotic medications are the mainstay of the management of violent behavior, adjuvant agents are also used. The following paragraphs describe some of the medications that have been used with APs to treat aggression in consumers with schizophrenia.

Lithium has been reported to have an anti-aggression effect but is no longer thought of as the augmenter of choice. Carbamazepine has also been reported to reduce aggressive episodes in violent
consumers with and without EEG abnormalities. As mentioned in the section on mood stabilization, the use of carbamazepine is complicated by its tendency to reduce serum levels of other psychotropic agents. Of the three first-line mood stabilizers, valproate is most commonly used as an adjunctive anti-aggression agent. Mood stabilizers are particularly effective when aggression is caused by an underlying affective disorder.

High-dose propranolol is also thought to have an anti-aggression effect in consumers with psychiatric disorders, especially those who are mentally retarded or have suffered a traumatic brain injury. (It should be noted that some attribute propranolol’s anti-aggression effect to its treatment of unrecognized akathisia.) Clinicians should also be aware that the concomitant use of propranolol and APs might result in elevated blood levels of the AP. The abrupt discontinuation of propranolol is dangerous and can, in rare cases, lead to arrhythmias and sudden death.

Clozapine’s anti-aggressive effect deserves special mention. Several reports have demonstrated a substantial reduction in hostile, aggressive, and violent behavior in consumers treated with clozapine. A decreased need for seclusions and restraints and prn medications has also been shown. A trial of clozapine should be considered in consumers with persistent aggression.

REFERENCES


Shared Decision-Making

Shared decision-making is a process by which consumers and practitioners consider treatment options, outcomes and preferences in order to reach a health care decision based on mutual agreement. Evidence-based practices do not usually identify one particular treatment as the best, but provide options and alternatives from which to choose. The shared approach to the decision permits an open exchange of information that allows the practitioner to present and the consumer to consider all alternatives, thereby enhancing the quality of the decision made. This dialogue promotes adherence because the consumer has participated in the treatment decision process.\textsuperscript{1,3}

Practitioners have technical knowledge while consumers have ideas and preferences about treatments that are grounded in previous experiences or discussions and cultural beliefs. The knowledge, preferences and beliefs of the two parties need to come together in consultation to determine the most effective care that will result in improved health. Shared decision-making involves at least two participants. Both parties contribute to the process and it must be a complementary exchange, with the practitioner sharing his/her technical expertise and the consumer sharing his/her preferences and beliefs. Finally, a treatment decision is made and both parties agree to endorse and take responsibility for it.
NECESSARY ELEMENTS OF SHARED DECISION-MAKING

The essential characteristics of shared decision-making are the exchange of information, building a consensus and reaching an agreement on the treatment to implement.

Practitioners:

- must be receptive to the views that the consumer has about the available treatment options and must create an atmosphere that allows the consumer to express those views.
- must elicit the consumer's preferences about the treatment and discuss options that are compatible with his/her lifestyle, values and beliefs.
- must be able to explain his/her technical knowledge and information about the risks, benefits and side effects in an unbiased, clear and simple manner.
- need to probe to determine if the consumer's assumptions about the treatment and the preferences that they have are based in fact.
- should share the treatment recommendation they have with the consumer and affirm the consumer's preferences.2,3

Consumers:

- must have the willingness to be a part of the process and take responsibility for discussing preferences, asking questions, and determining their treatment preferences.2,3

BARRIERS TO THE SHARED DECISION-MAKING PROCESS

Willingness

If the consumer is willing and wants to participate, but the practitioner does not, then the model cannot work. Conversely, if the practitioner is willing, but the consumer is not, then the process cannot be a shared one.

Communication

Consumers can be reluctant to discuss their health care decisions due to the novelty of the situation, a lack of understanding of their illness and its treatment options, and/or because they are from a social/cultural background that discourages the questioning of authority in any setting.

Practitioners can sometimes be uncomfortable in their ability to turn complex technical material into information that is readily understood by the consumer, and therefore are skeptical about using this model.
**Time**
Practitioners can be under strict time constraints with each consumer and are not given the opportunity to thoroughly discuss treatment options in a satisfactory way.

**Timeliness**
Emergent situations most often require treatment decisions without the benefit of time for discussion and consideration of the consumer's preferences.

**Ability**
In severe mental illness, the consumer may present cognitively unable to make choices based in fact about treatment options. Some treatment regimens are so complex that the consumer may not be capable of executing them successfully, even though they may have discussed the treatment and agreed to try.

**REMEDIES TO THE BARRIERS**

Many of the barriers to this model can be remedied. Some practitioners under-estimate the number of consumers who want involvement in decisions that affect their health and therefore do not attempt to engage them in the decision-making process. Keeping an open line of communication is a start for allowing the shared decision-making process to unfold. Consumers may need a little time to educate themselves about the options and determine how the different options would affect them, and which would fit best into their lifestyles. Depending upon their background and upbringing, some consumers may take several encounters and encouragement to begin to feel comfortable enough to participate in the process. It may be important to consider using a triad approach, rather than a dyad, especially when a consumer is cognitively impaired. A family member or significant other can be very beneficial in the shared decision-making process and in the success of the treatment that is agreed upon. Practitioners may need to become more proficient in interviewing and discussing treatment options and outcomes in terms that are easily understood by the consumer. Complex treatment regimens can be reevaluated to see if they can be simplified. Agencies may have to address appointment scheduling to allow enough time for the process to be used. Training non-physician practitioners to assist in the delivery of educational and informational material can help with physician practitioner time constraints. Preparing educational and informational material based on the options and steps used in a systematic approach to medication management would be useful to help the practitioner in explaining, as well as, assisting the consumer as he/she considers the options.
Research indicates that many consumers, for whatever reasons, do not want to have total control over health care decision-making, but also that many do not like having no say at all. Shared decision-making offers a viable alternative.¹

REFERENCES


Antipsychotic Side Effects

The antipsychotics vary significantly in their side effect profiles and in their predilection to produce specific side effects (Crismon, 2002). Table 1 outlines the relative side effect profiles of the atypical antipsychotics and the typical agents, haloperidol and chlorpromazine. One of the primary advantages of the atypical agents is their lower incidence of extra-pyramidal side effects (EPS), including acutely-occurring EPS such as dystonia, pseudo-Parkinson’s, and akathisia, as well as the chronically occurring adverse effect, tardive dyskinesia (Crismon 2002; Crismon, in press; Miller 2000; Miller 2001). Even among the atypical agents, the risk of EPS varies, with risperidone having the greatest risk, particularly at doses exceeding 6 mg daily. The risk of EPS is dose-related, and the lowest possible dose to effectively treat psychotic symptoms should be used to minimize the risk of EPS, as well as many other adverse effects.

Although the atypical antipsychotics produce a lower incidence of EPS, they are more likely to produce some other systemic side effects than haloperidol. For example, as a class, atypical agents are more likely to cause weight gain, with clozapine and olanzapine being most commonly implicated (see Table 1; Crismon, in press; Miller 2001; Kapur 2001). In some consumers, weight gain can be profound, and body weight should usually be obtained before beginning antipsychotics and at each clinic visit. In order to minimize the risk of weight gain, consumers should be educated regarding healthy diets and encouraged to exercise regularly (McIntyre 2001a; Wetterling 2001).

To varying degrees, atypical agents have been associated with producing glucose dysregulation (Crismon 2002; Crismon in press; Newcomer 2002). In some cases, diabetic ketoacidosis has been reported (Koller 2002). Although there are more case reports with clozapine and olanzapine, the relative risk of glucose dysregulation is still debated. Although attempts have been made to relate the risk of glucose dysregulation with weight gain, the mechanism of glucose intolerance is not entirely clear (Crismon in press; Lindenmayer 2001; Miller 2001; Newcomer 2002). Hyperlipidemias have
also been reported with atypical antipsychotics, and this may be more common among those agents more likely to produce glucose intolerance (Crismon in press; McIntyre 2001b; Meyer 2001).

Hyperprolactinemia is a laboratory abnormality commonly associated with typical antipsychotics as well as with risperidone (Crismon 2002; Crismon in press). Although elevated serum prolactin has been associated with such side effects as galactorrhea, and perhaps sexual dysfunction, attempts to develop a relationship between degree of prolactin elevation and specific side effects have been unsuccessful (Conley 2001; Kleinberg 1999).

The potential effects of antipsychotics on cardiac function have long been of concern. As a class, antipsychotics have the potential, to varying degrees, to produce orthostatic hypotension secondary to alpha blockade (Crismon 2002; Crismon in press). The effects of antipsychotics on cardiac conduction are of particular concern (Crismon in press; Glassman 2001; Miller 2001). The typical antipsychotic, thioridazine, has the greatest potential to cause a significant prolongation in the QTc interval. Among the atypical agents, ziprasidone has been shown to be most likely to prolong the QTc. However, the clinical significance of this effect in consumers with no underlying risk factors is unclear (Carnahan 2001; Crismon in press; Miller 2001). A baseline EKG before starting antipsychotics should be considered in those consumers who have risk factors for EKG abnormalities.

Other common side effects of antipsychotics include sedation and anticholinergic side effects (e.g., dry mouth, blurred vision, constipation, urinary retention) (Crismon 2002; Crismon in press). Although the risk appears lower with atypical than typical antipsychotics, neuroleptic malignant syndrome is a potential adverse effect of all antipsychotics (Crismon 2002). Lowered seizure threshold has been associated with antipsychotic use (Crismon 2002). Among the atypicals, this appears to be most common with clozapine.

Because of its risk of agranulocytosis, clozapine is reserved for consumers who have demonstrated treatment resistance to other antipsychotics (Crismon 2002, Crismon in press; Miller 2000). Routine monitoring of the white blood cell count is mandated in the approved product labeling for clozapine. Sialorrhea or drooling is another peculiar side effect, which is associated with clozapine use.

Consumer characteristics such as co-morbid general medical disorders, concomitant medications, and age should be carefully considered in antipsychotic drug selection (Miller 2000). A consumer’s past experiences with medication side effects should also be reviewed in determining which antipsychotic to use. While none of the available atypical antipsychotics has a perfect side effect profile, customizing treatment to individual consumer characteristics and consumer preference can be useful in achieving the lowest side effect burden in a given consumer.

This is not intended to be an exhaustive discussion of antipsychotic side effects. For more detailed information, the clinician should consult clinical psychopharmacology and pharmacotherapy reference sources as well as the FDA approved product labeling.
### Table 1. Comparative Side Effect Risk of Antipsychotic Agents

<table>
<thead>
<tr>
<th></th>
<th>CPZ</th>
<th>HPD</th>
<th>Clozapine</th>
<th>Olanzapine</th>
<th>Quetiapine</th>
<th>Risperidone</th>
<th>Ziprasidone</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anticholinergic</strong></td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>++</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>EPS</strong></td>
<td>+++</td>
<td>+++</td>
<td>0</td>
<td>+</td>
<td>+/-</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td><strong>Orthostasis</strong></td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td><strong>Hyperprolactinemia</strong></td>
<td>++</td>
<td>+++</td>
<td>0</td>
<td>+</td>
<td>0</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td><strong>QTc prolongation</strong></td>
<td>++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Sedation</strong></td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Tardive Dyskinesia</strong></td>
<td>+++</td>
<td>+++</td>
<td>0</td>
<td>+</td>
<td>?</td>
<td>+</td>
<td>?</td>
</tr>
<tr>
<td><strong>Weight gain</strong></td>
<td>++</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>++</td>
<td>++</td>
<td>+/-</td>
</tr>
<tr>
<td><strong>Glucose intolerance</strong></td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>?</td>
<td>+</td>
<td>?</td>
</tr>
</tbody>
</table>

**Key**
- CPZ = chlorpromazine
- HPD = haloperidol
- +/- = Negligible
- + = Minimal risk of occurrence
- ++ = Low risk of occurrence
- +++ = Moderate risk of occurrence
- ++++ = Highest risk of occurrence
- ? = Inadequate data to assess relative risk

[adapted from Carnahan (2001); Crismon ML (2002); and Miller (2000)]
REFERENCES


Issues in Antipsychotic Polypharmacy

The use of combinations of antipsychotic medications is increasing since the advent of second-generation antipsychotics, despite the extreme paucity of evidence for (or against) this practice. In the initial decades, after the introduction of first generation antipsychotics in North America, it was quite common to combine antipsychotics, such as high-potency with sedating low-potency. Over the course of time, it became clear that there was little, if anything, to be gained from this practice (Hollister 1982; Meltzer & Kostakoglu 2000).

It is important to distinguish between short- and long-term polypharmacy. When switching antipsychotics, most clinicians choose to overlap or cross-titrate the two, resulting in a purposely-brief period of combination treatment. Other clinicians, especially in inpatient settings, view first-generation antipsychotics as temporarily useful adjuncts to second-generation antipsychotics in treating acute illness exacerbations (see Figure 3, Ereshefsky 1999). In each of these instances, the long-term goal is monotherapy, but combinations are used to achieve short-term goals (reduce risks of switching medications or promote more rapid resolutions of acute symptom exacerbation). While the empirical bases for these short-term uses of combination antipsychotics is not particularly strong, there are rationales that are grounded in clinical experience and involve relatively brief exposure to the risks of combinations that are detailed below.

The rest of this section will elaborate on the following points concerning antipsychotic polypharmacy:

- Very little evidence supports the use of combination antipsychotic therapy.
- Most of the existing evidence involves adding another antipsychotic agent to clozapine.
- Disadvantages of antipsychotic polypharmacy include increased risk of side effects and drug-drug interactions, potential for decreased consumer adherence to more complicated medication regimens, and increased financial burden.
Several authors have reviewed and discussed the use of combination antipsychotics in the treatment of schizophrenia (Stahl 1999; Canales et al. 1999; Weiden and Casey 1999; Kingsbury et al. 2001). The largest body of evidence is on combining clozapine with other agents, with the hope of enhancing clozapine’s efficacy. In regard to antipsychotics other than clozapine, the consensus is that, except in cases where a consumer has failed adequate monotherapy trials of several antipsychotics including clozapine, antipsychotic polypharmacy has little support in the medical literature. In individual cases, however, clinicians and consumers may serendipitously hit upon effective combinations.

As stated above, the largest body of evidence on combination antipsychotics is on combinations with clozapine, to enhance efficacy. This generally positive literature includes one controlled trial (Shiloh et al. 1997) and a number of open label trials. Shiloh and colleagues conducted a 10-week, randomized, double-blind trial of the combination of clozapine-sulpiride versus clozapine-placebo in 28 in-consumers partially responsive to clozapine monotherapy. They found that the average reduction in the Brief Psychiatric Rating Scale (BPRS) score was 20.7% in the clozapine-sulpiride group compared to 5.4% in the clozapine-placebo group (p < 0.05). Interestingly, consumers in the clozapine-sulpiride cohort fell into two major subgroups, with half demonstrating a mean reduction in BPRS score of 42.4% (“responders”) and a little over a third showing a reduction of less than 5% (“non-responders”). A limitation of the study was that, in spite of the randomization, consumers in the clozapine-placebo group had a significantly longer total duration of previous hospitalization at baseline (p < 0.05). Sulpiride is not available in the United States.

Buckley et al. (2001) recently reviewed the clozapine augmentation literature. Buckley and colleagues examined the combination of clozapine and numerous psychotropics to treat the target symptoms of schizophrenia in clozapine non- or partial-responders. The adjunctive agents reviewed included first and second-generation antipsychotics, mood stabilizers, selective serotonin reuptake inhibitors (SSRIs), glycineergic agents, and electroconvulsive therapy (ECT). The authors concluded that, while none of the above agents stands out as an obvious first-line choice for augmentation, these adjuncts are probably the clinician’s best option considering that very little evidence supports discontinuing clozapine in the hope of achieving an improved response with a different atypical. Clinicians must always assess potential drug-drug interactions when combining medications with clozapine (see Drug Interactions section in Chapter 1). The rationale for efforts to augment clozapine is based on two observations: (1) clozapine is the best medication available for treatment-refractory schizophrenia, but (2) about half of consumers treated with clozapine do not respond adequately (Lieberman et al. 1994). Therefore, no other monotherapy is likely to benefit consumers who respond inadequately to clozapine, putting the clinician in the position of having to resort to combination treatments to try to achieve at least some response.

Why not combinations? The arguments against using combination antipsychotics, except when monotherapies, including clozapine, have failed, seem compelling. (1) Other than combinations
with clozapine, there is an absence of evidence to support the practice. (2) The likelihood of problematic side effects is increased. (3) The likelihood of problematic pharmacokinetic interactions is increased. (4) The likelihood of harmful pharmacodynamic interactions is increased. (5) Consumers are less adherent to complex medical regimes than simpler regimes (Chen 1991). The costs are greater when second generation antipsychotics are combined in usual doses. The risks of tardive dyskinesia may be as great on the combination of a first and second-generation antipsychotic as on a first generation antipsychotic alone. The clinician often has no basis for deciding what dose adjustments in which ingredient of the combination to make in response to increased symptoms or side effects.

Given all the reasons for not using combination antipsychotics, why are they used so often? Conceptually, there are four routes to long-term treatment with combination antipsychotics: (1) all reasonable monotherapies, including clozapine, have been failures or have been refused by the consumer, (2) a combination which was intended to be short-term is not discontinued, (3) a combination is instituted for lack of efficacy, even though further monotherapy trials would be reasonable, and (4) a combination is used to partially deal with a particular problem of monotherapy. The rationale and evidence for scenarios 2-4 are discussed below.

A second major route to long-term combination antipsychotics is the result of a clinical “decision” to extend a temporary combination indefinitely. Clinically, the circumstances that most often produce this result are (1) the continuation of a combination produced by cross-titration of two antipsychotics when the combination appears to be beneficial and (2) the continuation of the component of a combination that was originally begun for short-term reasons. As noted above, it is a fairly common practice in inpatient settings to supplement a second-generation antipsychotic with a first-generation antipsychotic (sometimes parenterally) in an effort to treat symptoms such as aggression and agitation and to achieve a more rapid response. If the consumer is discharged on this combination, it may be unclear to the outpatient provider when, if ever, the first generation antipsychotic should be discontinued. In both these instances, the central issue is whether the need for the combination has been demonstrated in the individual consumer. Clinicians should always be on the lookout for serendipitously good treatment results, and combination treatments can fall into this category. Given the problems with combinations, however, it is incumbent on the prescriber to demonstrate that the apparent benefits of the combination were not due to the new drug alone, were not limited to the period of an acute exacerbation, and were not merely a fortunate coincidence. This can only be done by progressing to a reasonable trial of monotherapy before re-instituting the combination, if the evidence still suggests that the consumer was better off on the combination.

The third route to combinations of antipsychotics is the decision to use them in preference to further monotherapy trials, even though reasonable monotherapies have not yet been tried. Often this is done when clozapine has not yet been tried, but there is reluctance on the part of the consumer, the physician, or both, to undertake a trial of clozapine. Sometimes the decision to use a combination
is more rooted in the desire to further improve outcomes in a partial responder to monotherapy. This latter approach is often accompanied by a pharmacological rationale, such as adding a stronger dopamine receptor antagonist if the consumer has residual positive symptoms or adding a stronger serotonin receptor antagonist if the consumer is still troubled by negative symptoms. Such rationales, while appealing, have no clinical trials to support them and rely on theories about the properties and mechanisms of action of antipsychotic medications that are tentative and evolving. Particularly for consumers in the early years of their illness, one must question the potential costs, in terms of illness progression, of the decision to use unproven combination treatments in preference to the single treatment that has been shown to be most effective for treatment-refractory schizophrenia.

A fourth route to antipsychotic combinations is based on safety and tolerability considerations. One published instance of this approach addressed the problem of clozapine-induced weight gain and hyperglycemia by partially substituting quetiapine for clozapine, finding that there were improvements in both weight and glucose parameters without loss of efficacy (Reinstein et al. 1999). Replication of this study would be useful. Not uncommonly, clinicians combine a second generation antipsychotic with a depot first-generation antipsychotic. There are two rationales for this, both based on the premise that the consumer will not be adequately adherent to oral monotherapy. First, some clinicians partially replace the depot medication with one less likely to produce extrapyramidal symptoms or tardive dyskinesia, an oral second-generation antipsychotic, to improve the safety and tolerability of the depot preparation. Second, when consumers are good responders to oral second-generation antipsychotics, but repeatedly fail to take them regularly when not under close supervision, some clinicians add depot antipsychotics, as a kind of safety net to prevent a precipitous return of psychosis if the consumers miss doses of their oral medications.

A problem with the first rationale is that, if the consumer is not going to adhere to oral therapy, he or she may be left on a subtherapeutic dose of depot medication and therefore be vulnerable to psychotic decompensation. A problem with the second rationale is that adding a first-generation to a second-generation antipsychotic may reverse the atypical profile of the second-generation medication (Kapur 1998). In this instance, adding back the first-generation medication may negate the newer medication’s lower incidence of EPS and tardive dyskinesia. As for the future, several depot second-generation antipsychotics are in development and it will be interesting to see to what extent their advent reduces the use and/or study of the combination of a first-generation depot antipsychotic and a second-generation oral antipsychotic.
REFERENCES


Prescribing During Pregnancy

The table below describes the potential toxicities of various psychotropic agents during the stages of gestation. FDA pregnancy categories and facts and guidelines for using antipsychotic agents during pregnancy follow the table.

<table>
<thead>
<tr>
<th>Medication</th>
<th>1st Trimester</th>
<th>2nd Trimester</th>
<th>3rd Trimester</th>
<th>FDA Category*</th>
<th>Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricyclic antidepressants</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>D</td>
<td>Possible association between 1st trimester and limb malformation by some case reports but further studies showed no association. Perinatal syndromes: antidepressant withdrawal with jitteriness and irritability</td>
</tr>
<tr>
<td>Desipramine</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Clomipramine</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Serotonin Selective Agents</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>B/C**</td>
<td>Fluoxetine has been the most studied. No higher rates of major congenital malformation those who took fluoxetine in the 1st trimester than the general population.</td>
</tr>
<tr>
<td>Other Antidepressants</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>C</td>
<td>Teratogenicity was not revealed in animals even at much higher doses than that used in humans.</td>
</tr>
<tr>
<td>Bupropion</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Medication</td>
<td>1st Tri-</td>
<td>2nd Tri-</td>
<td>3rd Tri-</td>
<td>FDA Category*</td>
<td>Summary</td>
</tr>
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<td>---------------------</td>
<td>---------</td>
<td>---------</td>
<td>---------</td>
<td>---------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Lithium</td>
<td>ø</td>
<td>+</td>
<td>±</td>
<td>D</td>
<td>Associated with cardiac anomalies when used in 1st trimester. Prematurity associated with use in 2nd &amp; 3rd trimester. Watch for maternal lithium toxicity after delivery due to volume change-need to decrease dose by half before delivery. Lithium levels may be increased in neonates-risk of “floppy baby” &amp; hypothyroidism</td>
</tr>
<tr>
<td>Valproic acid</td>
<td>ø</td>
<td>ø</td>
<td>ø</td>
<td>D</td>
<td>Associated with neural tube defects/1-5% risk of spina bifida</td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>D</td>
<td>0.5-1% risk of spina bifida</td>
</tr>
<tr>
<td>Other Anticonvulsants</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>C</td>
<td>Gabapentin, lamotrigine, &amp; topiramate were not teratogenic in animal studies but some malformations were observed.</td>
</tr>
<tr>
<td>Typical antipsychotics</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>C</td>
<td>Most common malformations reported include cardiac, genital, skeletal (3.5%). Use of high potency agents is recommended. Avoid low potency agents due to decrease BP &amp; uteroplacental blood flow. Use in 3rd trimester associated with neonatal associated extrapyramidal effects such as agitation, tremor, poor sucking, swallowing, primitive reflexes, and hypertonicity/DC drugs 5-10 days prior to delivery to allow fetal drug level to decrease.</td>
</tr>
<tr>
<td>Haloperidol</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Chlorpromazine</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Fluphenazine</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Loxapine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Mesoridazine</td>
<td></td>
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<tr>
<td>Thioridazine</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Thiothixene</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atypical antipsychotics</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>C</td>
<td>Little information on atypical antipsychotics.</td>
</tr>
<tr>
<td>Clozapine</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Medication</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; Tri-</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; Tri-</td>
<td>3&lt;sup&gt;rd&lt;/sup&gt; Tri-</td>
<td>FDA Category*</td>
<td>Summary</td>
</tr>
<tr>
<td>------------------</td>
<td>---------------------</td>
<td>---------------------</td>
<td>---------------------</td>
<td>---------------</td>
<td>------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Propranolol</td>
<td>±</td>
<td>+</td>
<td>±</td>
<td>C</td>
<td>It has been used to treat pregnancy-induced hypertension and does not appear to be associated with malformations. Neonatal adverse effects have included hyperbilirubinemia, bradycardia, respiratory depression, and low birth weights.</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>∅</td>
<td>±</td>
<td>±</td>
<td>D</td>
<td>Increase risk of cleft palate in 1&lt;sup&gt;st&lt;/sup&gt; trimester, especially diazepam &amp; alprazolam. 3&lt;sup&gt;rd&lt;/sup&gt; trimester exposure leads to tremors, hypertonicity, failure to feed, cyanosis and apnea. Best avoided but if needed use lorazepam (prn only).</td>
</tr>
<tr>
<td>Buspirone</td>
<td>±</td>
<td>±</td>
<td>±</td>
<td>B</td>
<td>Little information is available</td>
</tr>
</tbody>
</table>

* Based on Drugs in Pregnancy and Lactation, 5<sup>th</sup> edition; see Table of FDA Categories below.
∅ Use is not recommended
+ May be used—least risk
± May be used if no other alternative available
** Package Insert and Drugs in Pregnancy and Lactation, 5<sup>th</sup> edition differ
**FDA Categories**

<table>
<thead>
<tr>
<th>Pregnancy Category</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category A</td>
<td>Controlled studies in women fail to demonstrate a risk to the fetus in the first trimester and no evidence of a risk in later trimesters. The possibility of fetal harm appears remote.</td>
</tr>
<tr>
<td>Category B</td>
<td>Studies in animals have not demonstrated a fetal risk but there are no controlled studies in pregnant women or animal-reproduction studies have shown adverse effect that was not confirmed in controlled studies in women in the first trimester</td>
</tr>
<tr>
<td>Category C</td>
<td>Studies in animals have revealed adverse effects on the fetus and there are no controlled studies in women or studies in animals and women are not available. Drugs should be given only if the benefit justifies the potential risk to the fetus.</td>
</tr>
<tr>
<td>Category D</td>
<td>There is positive evidence of human fetal risk, but the benefits from use in pregnant women may be acceptable despite the risk</td>
</tr>
<tr>
<td>Category X</td>
<td>Studies in animal or women have demonstrated fetal abnormalities or there is evidence of fetal risk based on human experience or both, and the risk of the use of the drug in pregnant women clearly outweighs any possible benefit. The drug is contraindicated in women who are or may become pregnant.</td>
</tr>
</tbody>
</table>

**Antipsychotic agents in pregnancy**

- A number of studies have shown no increase in malformations after first trimester exposure to antipsychotic drugs.
- Two studies found an increase in nonspecific congenital anomalies after exposure to phenothiazines during early pregnancy.
- Available data show no effect of *in utero* antipsychotic exposure on IQ in humans.
- A mild, transient neonatal withdrawal syndrome of hypertonia, tremor, and poor motor maturity can result after antipsychotic use in late pregnancy.
Withdrawal dyskinesia, which may include irritability, abnormal hand and trunk posturing, tongue thrusting, and a shrill cry, is a rare reaction to antipsychotic exposure. These symptoms resolve spontaneously over several months with normal subsequent motor development.

Anticholinergic side effects can be seen in the fetus, neonate, or the pregnant woman.

Very little information is available concerning the use of atypical antipsychotics during pregnancy.

Atypical antipsychotics that are prolactin-sparing make implementation of effective contraceptive counseling for seriously ill consumers more urgent.

Glucose intolerance is a problem in pregnancy and the risk may increase with the use of antipsychotics; especially olanzapine and clozapine.

There are increased risks in pregnancy with the use of clozapine: glucose intolerance in the mother and possible fetal macrosomia, increased anticholinergic type side effects (constipation) in the mother, increased fatigue and sedation, hypotensive risk in the mother, and neonatal risk for agranulocytosis.

Guidelines for using antipsychotic agents during pregnancy

- Agents of choice are haloperidol and trifluoperazine, due to being relatively well studied and having the fewest pregnancy-associated side effects. Atypicals are a possibility, but there are limited data.

- Avoid use during first trimester if possible.

- Use only when benefit clearly outweighs the risk.

- For withdrawal dyskinesias in the newborn, diphenhydramine elixir can alleviate symptoms.

- It is recommended that pregnant women on antipsychotics be given calcium supplementation, which has been shown to reduce EPS, but no other prophylaxis for EPS is indicated.

- Avoid long-acting (depot) preparations of the high-potency group in order to limit the duration of any possible toxic effect in the neonate.

REFERENCES


*Bupropion Pregnancy Registry.* Glaxo Wellcome


*Lamotrigine Pregnancy Registry.* Glaxo Wellcome


*Olanzapine Pregnancy Registry.* Eli Lilly and Company


Racial/Ethnic Variation in Tolerance, Sensitivity, Metabolism/Clearance and Therapeutic Response

Researchers have discovered genetic polymorphisms for the cytochrome P-450 isoenzymes 2C9, 2C19, and 2D6. Of these, 2D6 and 2C19 metabolize several medications used to treat psychiatric conditions. While some studies have been performed in individuals of African descent, the bulk of research on genetic polymorphism has been conducted in the Caucasian and Asian populations (Poolsup et al, 2000).

Of the second-generation antipsychotics, only risperidone is principally metabolized by a polymorphic isoenzyme, CYP 2D6. This is not thought to be clinically significant, however, because of the equal effectiveness of risperidone and its major active metabolite, 9-OH risperidone.

When assessing the effects of medication, it is important to keep in mind that several factors affect drug response. Other variables to consider are adherence, drug interactions, age, diet, and smoking status (See Special Populations Appendix in the User’s Guide to MedMAP).

CYP 2D6

At this writing, researchers have identified three possible phenotypes for the CYP 2D6 enzyme. Individuals can be either (1) poor metabolizers (PM), (2) extensive metabolizers (EM), or (3) ultra rapid metabolizers (URM). Poor metabolizers are unable to synthesize the active form of the CYP 2D6 enzyme. When treated with standard doses of medications primarily metabolized by CYP 2D6, these individuals achieve higher than expected blood levels or become toxic. On the other hand, ultra rapid metabolizers possess several active copies of the CYP 2D6 gene. When treated with standard doses of CYP 2D6 substrates, these individuals display subtherapeutic blood levels, which
Clinicians may wrongly attribute to nonadherence. The metabolic ability of extensive metabolizers, the most common phenotype, lies somewhere between the two extremes. The metabolic capacity of extensive metabolizers depends on whether they are homozygous or heterozygous for an allele that produces a functional 2D6 enzyme. (Coutts and Urichuk, 1999)

Studies show that approximately 7% of Caucasians and 1% of Asians (Chinese, Japanese, and Koreans) are poor metabolizers. Although Asians have a low incidence of the poor metabolizers phenotype, studies have shown that, compared to Caucasians, they require lower doses of haloperidol and the TCAs. This is due to the fact that, in general, the metabolic capacity of Asian extensive metabolizers is less than that of Caucasian extensive metabolizers. (Poolsup et al, 2000)

In addition to psychotropic medications (some of which are listed below), many cardiovascular agents, codeine, and dextromethorphan are metabolized by CYP 2D6. The following psychotropics are CYP 2D6 substrates: haloperidol, perphenazine, fluphenazine, risperidone, chlorpromazine, nortriptyline, amitriptyline, clomipramine, desipramine, imipramine, fluoxetine, and paroxetine. Genetic polymorphism is usually not a critical issue in the metabolism of medications that have a wide therapeutic index (fluoxetine and paroxetine.)

**CYP 2C19**

The only known phenotypes for 2C19 are poor metabolizers and extensive metabolizers. As with extensive metabolizers of 2D6, the metabolic capacity of extensive metabolizers varies depending on the genotype of the individual. While it is possible to be a poor metabolizer of both 2D6 and 2C19, an individual’s phenotype at one isoenzyme is independent of his or her phenotype at another.

Approximately 12-22% of Asians are 2C19 PMs while only 3% of Caucasions have the poor metabolizer phenotype. Additionally, Asian extensive metabolizers tend to have less metabolic capacity than Caucasian extensive metabolizers. Not surprisingly, studies have shown that Asians require lower doses of diazepam (a CYP 2C19 substrate) than Caucasians. (Coutts and Urichuk, 1999)

Commonly used substrates of CYP 2C19 include imipramine, diazepam, omeprazole, and phenytoin.

**REFERENCES**


Adherence with Antipsychotic Medications

Adherence to medication treatments is an issue in all medical conditions, not just psychiatry, and not just with antipsychotic medications (Cramer, 1998). Non-adherence is important because it is associated with increased rates of relapse and hospitalization. We prefer the term “adherence” rather than “compliance” because the latter can imply a paternalistic relationship in which the consumer is a subordinate receiver of medical orders, rather than a full partner in his or her own treatment who elects to adhere to a particular medication regime.

**Key points**

- Many factors have been studied in association with antipsychotic medication non-adherence, but the evidence is inconsistent. Methodologic differences complicate comparisons across studies.

- Factors linked most consistently with antipsychotic medication non-adherence are history of previous non-adherence and comorbid substance abuse/dependence

- More research is needed before definitive evidence-based recommendations for interventions can be made. Cognitive-behavioral approaches currently appear most promising. Psychoeducational programs have been mostly unsuccessful at improving medication adherence.

**Evidence Base**

**Factors associated with adherence**

The psychiatric literature reports many factors that are inconsistently linked with medication adherence. A sampling includes: personal history of medication non-adherence (Buchanan, 1992; Olfson et al, 2000; Ruscher et al, 2000); co-morbid substance abuse or dependence (Ayuso-Gutierrez et al, 1997; Olfson et al, 2000); consumers’ attitudes toward medication (Ayuso-Gutierrez et al, 1997), presence of side effects (Falloon, 1984; Ayuso-Gutierrez et al, 1997; Olfson et al, 2000) and subjective response to medication (Agarwal el al, 1998; Garavan et al, 1998; Van Putten & May 1978; Van

**Interventions**

Interventions to improve adherence range in their specificity (whether medication adherence is the primary outcome or one of several outcome measures), target population (high risk for non-adherence or more general population), type of program (psychoeducational, behavioral) target audience (individual, group or family), and length of follow-up. A sample of these studies is listed below.

Evidence shows that psychoeducational programs inconsistently improve adherence, whether the programs are focused on families or on individual consumers. Although these programs generally increased knowledge about medications, results on medication adherence behavior were mixed (Boczkowski, 1985; McPherson, 1996; Seltzer, 1980). Cognitive-behavioral programs improved medication adherence when they focused on behavioral tailoring, for example using a special pill box with medications counted out and linking taking medication to other behaviors (Azrin, 1998; Boczkowski, 1985; Kelly, 1990) or motivational interviewing, which involves identifying consumers’ goals and examining how taking medication may help them meet those goals (Kemp, 1998). Social skills training had less impact. Matching interventions with individual consumers’ needs may prove to be important, but the evidence base for doing so is not yet available.

**REFERENCES**


Frank AF & Gunderson JG. The role of the therapeutic alliance in the treatment of schizophrenia: Relationship to course and outcome. *Archives of General Psychiatry*, 47:228-236, 1990.


Chapter 11 presents and compares recent guidelines and algorithms for medication management of schizophrenia. Each guideline/algorithm attempts to integrate evidence from the literature and expert opinion into a set of recommendations on the sequence of use of antipsychotics in persons with schizophrenia.

The recommendations differ, in part, because they were developed at different times. Antipsychotics, which are now widely used, may have been unavailable or only recently approved at the time of promulgation of the algorithm or guideline. These differences illustrate a recurring dilemma in updating medication recommendations. When new medications become available through the FDA approval process they have been tested in limited populations (e.g. excluding substance abusers) and there is often little information about critical questions, such as optimal switching strategies and efficacy in persons who have failed other medications, for the same indication. Moreover, rare but very serious side effects may not be detected or fully appreciated until many thousands of persons have received the new medication. These issues argue for a conservative approach to incorporating the new treatment into the guideline or algorithm. On the other hand, evidence-based guidelines and algorithms are supposed to be useful to practitioners. If a new treatment has advantages over existing ones, it is not helpful to the practitioner if recommendations for its use are not incorporated into the guideline/algorithm expeditiously.

At the level of implementation, the guidelines and algorithms discussed below differ greatly in the degree to which they specify key variables such as recommended doses, duration of treatment, outcome assessments, response criteria, definitions of treatment failure, etc. Strictly speaking, algorithms are more specific than guidelines, in that in an algorithm, by definition, the results of each step are used to determine the next step, whereas guidelines may have multiple alternatives and
less specific directions for measuring outcomes and adequacy of outcomes. In practice, if organizations want to monitor guideline implementation, they must have rules as to what parameters should be measured and what constitutes full, partial, and inadequate adherence to the guideline’s recommendations. That is to say, characterizing adherence to guidelines and algorithms must be based on specific criteria, whether taken from the guideline/algorithm itself or derived from other sources such as expert opinion, the medical literature, or consensus of the organization’s prescribers.
Comparison of Medication Guidelines and Algorithms


Since 1996 there has been a proliferation of treatment algorithms and guidelines for schizophrenia.1-14 Two factors that have contributed to this trend are (1) the approval and marketing of four atypical antipsychotics (risperidone, olanzapine, quetiapine and ziprasidone) since 1994 and (2) the high costs of atypical antipsychotics relative to typical or conventional antipsychotics. With increased choices and cost has come greater emphasis on appropriate and efficient use of these newer treatment alternatives. Moreover, systematic reviews of treatment of schizophrenia in public mental health facilities have shown how frequently the use of antipsychotic medication (a) does not follow expert recommendations, (b) is not responsive to residual symptoms and side effects, and (c) is poorly documented.

This Chapter briefly presents some of the most widely used and cited guidelines and algorithms for the pharmacotherapy of schizophrenia. Historically, in psychiatry and the other branches of medicine, physicians have individually made decisions regarding medication management, outcome measures, and criteria for adequacy of response. For payers and consumers, this system presents two potential problems. First, for a minority of physicians, the medication management practices are frequently inconsistent with evidence-based recommendations and/or vary widely from the practices of their peers. These “outliers” present increased risks of inferior care to consumers and
risks of legal liability and overuse of resources to the organizations responsible for them. To the degree that algorithms/guidelines specify and define “good” medication management practices, they can be helpful in identifying “bad” practices for closer scrutiny and, when necessary, corrective actions. The second problem that arises from complete individualization of medication decision-making is that consumer transitions between prescribers result in abrupt changes and significant inconsistencies in treatment. In consumers with chronic relapsing illnesses, unsystematic provider specific medication management approaches likely pose at least as much a challenge to optimizing treatment as do aberrant handling of medications by an individual prescriber.

These guidelines are part of a general effort in the health care field to cope with an ever-increasing number of therapeutic alternatives through the development of evidence-based practice recommendations. The evidence upon which to base a number of key clinical decisions about drug treatment of schizophrenia is remarkably scanty. In these cases, the practitioner must rely on clinical judgment and expert opinion. Moreover, clinical judgment is always a critical factor in optimizing treatment for the individual patient. Thus, the oft-expressed fear that the use of guidelines and algorithms promotes a rote approach to treatment has no basis in the current reality of treatment for schizophrenia.

Conceptually, the choices of medications for schizophrenia are related to the phase of illness (e.g., acute, resolving, maintenance), to the target symptoms (positive, negative, and cognitive), and to the patient’s past history of response to medications. Current guidelines vary somewhat in the degree to which they differentiate between phase of illness and target symptoms, but the underlying premise of all of them is that schizophrenia is a chronic illness in which antipsychotics are central to the treatment of all phases of the illness and each of its core components. Table 1 summarizes the comparison of medication guidelines and algorithms that have been widely promulgated in North America.
### Table 1. Medication Guideline/Algorithm Recommendations

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<tr>
<td>First line atypicals</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>First line typicals</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Second choice</td>
<td>A,T</td>
<td>A,T</td>
<td>A,T</td>
<td>A,T,C</td>
<td>A,T</td>
<td>A</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Third choice</td>
<td>C</td>
<td>A</td>
<td>C</td>
<td>C</td>
<td>C</td>
<td>C</td>
<td>A</td>
<td>C/A,T</td>
</tr>
<tr>
<td>Fourth choice</td>
<td>-</td>
<td>C</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>C+</td>
<td>C/C+</td>
<td>A,T/C/C+</td>
</tr>
<tr>
<td>Combinations</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>CF</td>
<td>CF</td>
</tr>
<tr>
<td>Response criteria</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Y</td>
<td>Y</td>
</tr>
</tbody>
</table>

* Expert Consensus Guidelines for the Treatment of Schizophrenia
† Texas Medication Algorithm Project
‡ Department of Veterans Affairs
§ American Psychiatric Association
║ Canadian Psychiatric Association

Y=yes, N= no, A= atypical antipsychotics, T= typical antipsychotics, C= clozapine, C+= Clozapine augmentation, CF= clozapine failure.

From the viewpoint of an organization responsible for the delivering of mental health care services, it is critical to recognize that the decision to adopt a medication algorithm or guideline is multi-layered, at the level of implementation. There are a related series of questions:

- Who needs to participate in the decision?
- Will there be the opportunity for local modification?
- Who will monitor prescriber adherence, and how?
- What resources will go into training?
- Who will be trained?
- Who will train new employees, and how?
- Who will have overall responsibility and what authority will they have?
Will the scope of the effort include:
- recommended sequences of medications?
- measures of symptoms? Side effects? Functioning?
- criteria for adequacy of response?
- medication education materials and programs?
- consumer oriented programs to promote medication adherence and shared decision-making?

What changes in the organization of delivery of clinical care need to be made?

What administrative changes need to be made?

How do medical records need to be reorganized?

What new forms will be needed to support the practice?

Is the formulary consistent with the guideline or algorithm?

Are there practical or administrative impediments to implementation?

REFERENCES


Lehman AF: Improving treatment for persons with schizophrenia. Psychiatr Q 70:259-272, 1999


Lehman AF, Steinwachs DM: Translating research into practice: The schizophrenia PORT treatment recommendations, Schizophr Bull 24:1-10, 1998


Guidelines and Algorithm Resources

**Texas Medication Algorithm Project (TMAP)**

**Texas Implementation of Medication Algorithms (TIMA)**

TMAP began in 1996 as collaborative research effort in the state of Texas to develop, implement and evaluate medication algorithm-driven treatment. The medication management in TMAP consists of evidence-based, consensually agreed upon medication treatment algorithms, clinical and technical support to implement, patient and family education programs, and documentation of patient care and outcomes.

TIMA is the ongoing statewide implementation phase of TMAP occurring in the Texas Department of Mental Health and Mental Retardation facilities.

http://www.mhmr.state.tx.us/centraloffice/medicaldirector/TMAP.html

http://www.mhmr.state.tx.us/centraloffice/medicaldirector/TIMA.html

**1999 Expert Consensus Guidelines Series**


American Psychiatric Association Guidelines

This practice guideline, published in April 1997, was developed by psychiatrists who are in active clinical practice. The guideline has been reviewed by members of APA as well as by representatives from related fields. Contributors and reviewers were asked to base their recommendations on an objective evaluation of the available evidence.

http://www.psych.org/psych_pract/treatg/pg/prac_guide.cfm

Canadian Psychiatric Association

The Canadian guidelines were written by a working group that included psychiatrists and psychologists expert in the assessment and treatment of schizophrenia. Recently published guidelines were used as source documents. Some recommendations are based upon research evidence and substantial experience; others are based upon expert opinion and consensus.


http://www.cpa-apc.org/Professional/Guidelines/Guidelines.asp

Department of Veterans Affairs


http://www.oqp.med.va.gov/cpg/psy/psy_base.htm
Evaluation Tools

This Chapter discusses domains of outcome in schizophrenia such as positive and negative symptoms, cognitive deficits, and functional impairments. This Chapter also provides information on assessment tools used for diagnostic evaluation and rating symptom severity and functional outcomes in persons with psychotic illness. The instruments featured were selected based on their established use in evaluating treatment of psychotic illnesses as well as practical applicability. Brief instruments developed by the Texas Medication Algorithm Project and forms for tracking clinical information that facilitate identifying critical decision points are included.

OUTCOMES IN SCHIZOPHRENIA

Treatments of schizophrenia typically target specific symptoms or problems for improvement. Domains of outcome frequently targeted in schizophrenia include positive symptoms, negative symptoms, cognitive deficits, and functional impairments. Each area is distinct in terms of its response to treatment and its impact on the overall course of illness. This section will briefly review these outcome domains and the role of medications in their treatment.

Positive symptoms are prominent during acute episodes of the illness. These include hallucinations and delusions. Positive symptoms are usually responsive to antipsychotic medications and can be greatly diminished or eliminated for long periods of time. It is recommended that the level of antipsychotic symptoms be assessed with scaled and reliable measures and the results used as a key indicator of antipsychotic efficacy. This approach allows different clinicians to use common reference points to decide if positive symptoms are improving, unchanging, or worsening.
Improvement in negative symptoms is also a goal of medication treatment of patients with schizophrenia. Although there is evidence of improvement of negative symptoms with antipsychotic treatment (often reported to be greater with second generation antipsychotics), much of the evidence is from short-term studies of treatment of acute exacerbations. Of much greater clinical relevance are long-term changes in negative symptoms that potentially improve the patient’s quality of daily life. There is considerably less data on enduring effects of antipsychotics, antidepressants, or stimulants on negative symptoms, though there is limited evidence for long-lasting benefits from some of the second-generation antipsychotics.

Studies of negative symptoms have identified multiple components. Three of these are incorporated into DSM-IV criteria for schizophrenia: alogia, avolition, and flat affect. Both because of their diagnostic significance and their important association with impaired functioning, it is important to document the severity of negative symptoms and their response to treatment. However, there is controversy over whether core negative symptoms of schizophrenia respond to medications, making it unclear whether persistent high levels of negative symptoms should be used clinically as an indication for changing medication treatment. Clinical considerations in making this decision include addressing the following questions: (1) Could the negative symptoms be secondary to medication side effects? (2) Are there elements of depression? (3) Have the negative symptoms been relatively invariant across multiple medication trials? (4) Is the patient motivated to reduce his/her negative symptoms? (5) What are the risks of failure on a new medication?

On average, patients with schizophrenia fall substantially below population means on a wide array of tests of cognitive functioning. Recent evidence suggests that the second-generation antipsychotics can improve cognitive test performance, though further studies are needed to rule out alternative explanations for their better results compared with first generation antipsychotics. Moreover, the improvements only partially remedy the deficits, and it remains to be shown that improved test performance results in improved task performance or functioning. With regard to evaluation of cognitive functioning in schizophrenia, clinically practical assessment tools are lacking, though several are in development. The brief scales used in dementia are too insensitive to the deficits found in schizophrenia, while standard neuropsychological test batteries are too lengthy and require too much technical expertise to administer to be useful in monitoring treatment effects on cognition in schizophrenia.

Finally, the bottom line in many patients with schizophrenia is impaired functioning in work, relationships, and activities of daily living. A variety of psychosocial interventions are intended to improve patient functional status. Medications per se do not directly affect functioning, but they may well change the potential for new learning, skill acquisition etc. that could lead to improved functioning. While there are a number of tests of functional abilities, clinically feasible tests that
would detect an increased potential to improve functioning would be very useful to have available. If medication treatments could produce increased potential to improve functioning, these tests could help guide selection and allocation of psychosocial interventions and resources. Such tests are not currently available.

**INTRODUCTION TO THE OUTCOME MEASURES**

The table of outcome assessments, below, is intended to give users much of the information they will need in order to decide about the suitability of the tests for their particular system or practice. Two general principles should be noted with regard to selection of assessments: (1) the lengthier assessments are usually less variable across observers and across time, but their time of administration makes them not feasible for routine use in systems with limited resources; (2) the global measures have the value of brevity, but their variability across observers and across time can be large and they do require the rater to spend enough time talking with the consumer to form an accurate global impression. That is to say, it takes only a minute to record the global measures, but it takes 10-20 minutes of interviewing to form the basis for the rating. Of the assessments listed in the following table, the four-item PSRS and BNSA are provided in this document. The other items are publicly available.
## MedMAP Outcome Assessments

<table>
<thead>
<tr>
<th>Assessment Category</th>
<th>Assessment Title</th>
<th>Abbr</th>
<th>Desired response</th>
<th>Clinical Utility</th>
<th>Time to administer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td>Brief Psychiatric Rating Scale</td>
<td>BPRS</td>
<td>Decrease ≥ 20%</td>
<td>Symptom change (in response to treatment interventions)</td>
<td>20-30</td>
</tr>
<tr>
<td></td>
<td>Positive &amp; Negative Syndrome Scale</td>
<td>PANSS</td>
<td>Decrease ≥ 20%</td>
<td>Symptom change (in response to treatment interventions)</td>
<td>30-40</td>
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<td></td>
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<td>Decrease</td>
<td>Symptom change (in response to treatment interventions)</td>
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<td></td>
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<td>SAPS</td>
<td>Decrease</td>
<td>Symptom change (in response to treatment interventions)</td>
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<td>Positive Symptom Rating Scale (4 items)</td>
<td>PSRS</td>
<td>Decrease to ≤ 6</td>
<td>Symptom change (in response to treatment interventions)</td>
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<td>BNSA</td>
<td>Decrease to ≤ 12</td>
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<td>Decrease</td>
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<td>Decrease</td>
<td>Symptom change (in response to treatment interventions)</td>
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<td>Decrease</td>
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<td>Monitors EPS</td>
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<td>Measures drug induced akathisia</td>
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<td>Absent</td>
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<td>Absent</td>
<td>Elicits adverse events</td>
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<td>HoNOS³</td>
<td>Decrease</td>
<td>Overall functioning</td>
<td>15-30</td>
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| Cognitive Measures                          | Mini-Mental State Exam                                  | MMSE | Increase         | Tracks cognitive impairment                                                       | 5-10               |
|                                             | Neurobehavioral Cognitive Status Exam                   | NCSE | Increase         | Tracks functioning across multiple cognitive domains                             | 5-10               |
|                                             | Brief Cognitive Rating Scale                           | BCRS | Decrease         | Staging integrity of cognitive abilities                                         | 15                 |
|                                             | Drug Attitude Inventory                                | DAI  | ≥ 0              | Identify at risk for non-adherence to medications                               | 10                 |
|                                             | Approaches to Schizophrenia Communication Self Report  | ACS-SR | Absent | Engages patient and treatment team to deal with problematic SE’s                    | < 5                |

* Anchored rating scale  º Global rating scale
4-Item Positive Symptom Rating Scale (PSRS)
Brief Negative Symptom Assessment (BNSA)
Texas Department of Mental Health and Mental Retardation

Version (5.0)
Revised September 6, 2001

The 4-item PSRS was adapted from the Expanded Version of the BPRS developed by:


The Brief Negative Symptom Assessment was adapted from the Negative Symptom Assessment and the Scale for the Assessment of Negative Symptoms developed respectively by:


In the past 7 days...

4-ITEM POSITIVE SYMPTOM RATING SCALE (VERSION 5.0)

SCALE ITEMS AND ANCHOR POINTS

1. SUSPICIOUSNESS
Expressed or apparent belief that other persons have acted maliciously or with discriminatory intent. Include persecution by supernatural or other nonhuman agencies (e.g., the devil). Note: Ratings of “3” or above should also be rated under Unusual Thought Content.
Do you ever feel uncomfortable in public?

Does it seem as though others are watching you?

Are you concerned about anyone’s intentions toward you?

Is anyone going out of their way to give you a hard time, or trying to hurt you?

Do you feel in any danger?

[If patient reports any persecutory ideas/delusions, ask the following]:

How often have you been concerned that [use patient’s description]?

Have you told anyone about these experiences?

1  Not Present

2  Very Mild
Seems on guard. Reluctant to respond to some “personal” questions. Reports being overly self-conscious in public.

3  Mild
Describes incidents in which others have harmed or wanted to harm him/her that sound plausible. Patient feels as if others are watching, laughing, or criticizing him/her in public, but this occurs only occasionally or rarely. Little or no preoccupation.

4  Moderate
Says others are talking about him/her maliciously, have negative intentions, or may harm him/her. Beyond the likelihood of plausibility, but not delusional. Incidents of suspected persecution occur occasionally (less than once per week) with some preoccupation.

5  Moderately Severe
Same as 4, but incidents occur frequently, such as more than once per week. Patient is moderately preoccupied with ideas of persecution OR patient reports persecutory delusions expressed with much doubt (e.g., partial delusion).

6  Severe
Delusional -- speaks of Mafia plots, the FBI, or others poisoning his/her food, persecution by supernatural forces.
7  Extremely Severe
   Same as 6, but the beliefs are bizarre or more preoccupying. Patient tends to disclose
   or act on persecutory delusions.

2.  UNUSUAL THOUGHT CONTENT
   Unusual, odd, strange or bizarre thought content. Rate the degree of unusualness, not the
degree of disorganization of speech. Delusions are patently absurd, clearly false or bizarre
ideas that are expressed with full conviction. Consider the patient to have full conviction if
he/she has acted as though the delusional belief were true. Ideas of reference/persecution
can be differentiated from delusions in that ideas are expressed with much doubt and con-
tain more elements of reality. Include thought insertion, withdrawal and broadcast. Include
grandiose, somatic and persecutory delusions even if rated elsewhere. Note: If Suspicious-
ness is rated “6” or “7” due to delusions, then Unusual Thought Content must be rated a “4”
or above.

Have you been receiving any special messages from people or from the way things are arranged
around you?

Have you seen any references to yourself on TV or in the newspapers?

Can anyone read your mind?

Do you have a special relationship with God?

Is anything like electricity, X-rays, or radio waves affecting you?

Are thoughts put into your head that are not your own?

Have you felt that you were under the control of another person or force?

[If patient reports any odd ideas/delusions, ask the following]:

How often do you think about [use patient’s description]?

Have you told anyone about these experiences?

How do you explain the things that have been happening [specify]?
1 **Not Present**

2 **Very Mild**
   Ideas of reference (people may stare or may laugh at him), ideas of persecution (people may mistreat him). Unusual beliefs in psychic powers, spirits, UFOs, or unrealistic beliefs in one’s own abilities. Not strongly held. Some doubt.

3 **Mild**
   Same as 2, but degree of reality distortion is more severe as indicated by highly unusual ideas or greater conviction. Content may be typical of delusions (even bizarre), but without full conviction. The delusion does not seem to have fully formed, but is considered as one possible explanation for an unusual experience.

4 **Moderate**
   Delusion present but no preoccupation or functional impairment. May be an encapsulated delusion or a firmly endorsed absurd belief about past delusional circumstances.

5 **Moderately Severe**
   Full delusion(s) present with some preoccupation OR some areas of functioning disrupted by delusional thinking.

6 **Severe**
   Full delusion(s) present with much preoccupation OR many areas of functioning are disrupted by delusional thinking.

7 **Extremely Severe**
   Full delusions present with almost total preoccupation OR most areas of functioning are disrupted by delusional thinking.

3. **HALLUCINATIONS**
   Reports of perceptual experiences in the absence of relevant external stimuli. When rating degree to which functioning is disrupted by hallucinations, include preoccupation with the content and experience of the hallucinations, as well as functioning disrupted by acting out on the hallucinatory content (e.g., engaging in deviant behavior due to command hallucinations). Include “thoughts aloud” (“gedankenlautwerden”) or pseudo-hallucinations (e.g., hears a voice inside head) if a voice quality is present.
Do you ever seem to hear your name being called?

Have you heard any sounds or people talking to you or about you when there has been nobody around?

[If hears voices]: What does the voice/voices say?

Did it have a voice quality?

Do you ever have visions or see things that others do not see?

What about smell — odors that others do not smell?

[If the patient reports hallucinations, ask the following]:

Have these experiences interfered with your ability to perform your usual activities/work?

How do you explain them?

How often do they occur?

1  Not Present

2  Very Mild
   While resting or going to sleep, sees visions, smells odors, or hears voices, sounds or whispers in the absence of external stimulation, but no impairment in functioning.

3  Mild
   While in a clear state of consciousness, hears a voice calling the subject’s name, experiences non-verbal auditory hallucinations (e.g., sounds or whispers), formless visual hallucinations, or has sensory experiences in the presence of a modality-relevant stimulus (e.g., visual illusions) infrequently (e.g., 1-2 times per week) and with no functional impairment.

4  Moderate
   Occasional verbal, visual, gustatory, olfactory, or tactile hallucinations with no functional impairment OR non-verbal auditory hallucinations/visual illusions more than infrequently or with impairment.
5 **Moderately Severe**
Experiences daily hallucinations OR some areas of functioning are disrupted by hallucinations.

6 **Severe**
Experiences verbal or visual hallucinations several times a day OR many areas of functioning are disrupted by these hallucinations.

7 **Extremely Severe**
Persistent verbal or visual hallucinations throughout the day OR most areas of functioning are disrupted by these hallucinations.

4. **CONCEPTUAL DISORGANIZATION**
Degree to which speech is confused, disconnected, vague or disorganized. Rate tangentiality, circumstantiality, sudden topic shifts, incoherence, derailment, blocking, neologisms, and other speech disorders. Do not rate content of speech.

1 **Not Present**

2 **Very Mild**
Peculiar use of words or rambling but speech is comprehensible.

3 **Mild**
Speech a bit hard to understand or make sense of due to tangentiality, circumstantiality or sudden topic shifts.

4 **Moderate**
Speech difficult to understand due to tangentiality, circumstantiality, idiosyncratic speech, or topic shifts on many occasions OR 1-2 instances of incoherent phrases.

5 **Moderately Severe**
Speech difficult to understand due to circumstantiality, tangentiality, neologisms, blocking, or topic shifts most of the time OR 3-5 instances of incoherent phrases.

6 **Severe**
Speech is incomprehensible due to severe impairments most of the time. Many PSRS items cannot be rated by self-report alone.

7 **Extremely Severe**
Speech is incomprehensible throughout interview.
1. **PROLONGED TIME TO RESPOND (a measure of Alogia)**
   Observed throughout communication with the patient. After asking the patient a question, he or she pauses for inappropriately long periods before initiating a response. Delay is considered a pause if it feels as though you are waiting for a response or if you consider repeating the question because it appears that the patient has not heard you. He or she may seem “distant” and sometimes the examiner may wonder if he has even heard the question. Prompting usually indicates that the patient is aware of the question, but has been having difficulty in developing his thoughts in order to make an appropriate reply. Rate severity on the frequency of these pauses.

   1. **Normal**
      No abnormal pauses before speaking.

   2. **Minimal**
      Minimal evidence of inappropriate pauses (brief but not abnormally lengthy pauses occur) may be extreme of normal

   3. **Mild**
      Occasional noticeable pauses before answering questions. Due to the length of the pause, you feel the need to repeat yourself once or twice during the interview.

   4. **Moderate**
      Distinct pauses occur frequently (20-40% of responses).

   5. **Marked**
      Distinct pauses occur most of the time (40-80% of responses).

   6. **Severe**
      Distinct pauses occur with almost every response (80-100% of responses).

2. **EMOTION: UNCHANGING FACIAL EXPRESSION; BLANK, EXPRESSIONLESS FACE (a measure of Flat Affect)**
   The patient’s face appears wooden, mechanical, frozen. Facial musculature is generally expressionless and unchanging. The patient does not change expression, or change is less than normally expected, as the emotional content of discourse changes. Because of this, emotions may be difficult to infer. Disregard changes in facial expression due to abnormal involuntary movements, such as tics and tardive dyskinesia. The two dimensions of importance when making this rating are degree of emotional expression and spontaneity.

Items adapted from NSA and SANS
1. Normal
Spontaneous displays of emotion occur when expected. Normal degree of expressiveness of emotions is present.

2. Minimal
Spontaneous expressions of emotion occur when expected. However, there is a reduction in degree or intensity of the emotions expressed. May be extreme of normal.

3. Mild
Spontaneous expressions of emotion occur infrequently. When emotions are expressed there is a reduction in degree or intensity displayed.

4. Moderate
Obvious reduction in spontaneous expressions. Spontaneous expressions of emotion may occur very rarely during interaction and only when discussing topics of special interest or humor to the subject.

5. Marked
Facial expression is markedly decreased. There are no spontaneous expressions of emotion unless prompted or coaxed by the interviewer.

6. Severe
There are no expressions of emotion even when attempts are made to elicit an emotional response. The subject’s face remains blank throughout the interview.

3. REDUCED SOCIAL DRIVE (a measure of Asociality)
This item assesses how much the subject desires to initiate social interactions. Desire may be measured in part by the number of actual or attempted social contacts with others. If the patient has frequent contact with someone (e.g., family member) who initiates the contact, does the patient appear to desire the contact (i.e., would he or she initiate contact if necessary)? In making this rating, probe the desire to initiate social interactions, number of social interactions and the ability to enjoy them.
Assessed by asking the patient questions like:

*How have you spent your time in the past week?*

*Do you live alone or with someone else?*

*Do you like to be around people?*

*Do you spend much time with others?*

*Do you have difficulty feeling close to others?*

*Who are your friends?*

*How often do you see them?*

*Did you see them this past week?*

*Have you called them on the phone?*

*When you get together, who decides what to do and where to go?*

*When you spend time with others, do you ask them to do something with you or do you wait until they ask you to do something?*

*Is anyone concerned about your happiness or well-being?*

1. **Normal**
   Normal desire to initiate and normal number of contacts. Social contacts are enjoyable.

2. **Minimal**
   Minimal reduction in either the desire to initiate social contacts or the number of social relationships. May initially seem guarded, but has the ability to establish relationships over time. Social relationships are enjoyable.

3. **Mild**
   Reduction in desire to initiate social contacts. The patient has few social relationships and these social contacts are enjoyable.

4. **Moderate**
   Obvious reduction in the desire to initiate social contacts. The patient has few relationships toward which he or she feels indifference. However, a number of social contacts are initiated each week.
5. **Marked**
Marked reduction in desire to initiate social contacts. The patient has very few relationships toward which he or she feels indifference. The patient does not initiate social contacts but may maintain a few contacts (such as with family).

6. **Severe**
Patient does not desire social contact. Actively avoids social interactions.

4. **GROOMING AND HYGIENE (a measure of Amotivation)**
Observed during interaction with the patient. The patient displays less attention to grooming and hygiene than normal. The patient presents with poorly groomed hair, disheveled clothing, etc. Do not rate grooming as poor if it is simply done in what one might consider poor taste (e.g., wild hairdo or excessive makeup). In addition to observation, one must ask the patient about regularity of bathing, brushing teeth, changing clothes, etc. This is particularly important with outpatients, as the patient may present his or her best grooming and hygiene at their clinic visit. Two dimensions to keep in mind when making this rating are current appearance and regularity of grooming behaviors.

Assess the patient by asking questions like:

_How many times in the past week have you taken a shower or bath?_

_How often do you change your clothes?_

_How often do you shower and brush your teeth?_

1. **Normal**
Patient is clean (e.g., showers every day) and dressed neatly.

2. **Minimal**
Minimal reduction in grooming and hygiene, may be at the extreme end of the normal range.

3. **Mild**
Apparently clean but untidy appearance. (e.g., may shower or brush teeth only 3 to 4 times per week). Clothing may be mismatched. Patient may shower less often than every other day, or may brush teeth less than everyday.

4. **Moderate**
There is an obvious reduction in grooming and hygiene. Clothes may appear un-
kempt, rumpled, or the patient may look as if he or she just got out of bed. The patient may to without shower or bathing for two days at a time. The patient may go for two days without brushing their teeth.

5. **Marked**
   There is a marked reduction in grooming and hygiene. Clothing may appear dirty, stained or very unkempt. The subject may have greasy hair or a body odor. The patient may go 3 days at a time without showering or 3 or 4 days without brushing their teeth.

6. **Severe**
   Clothing is badly soiled. Patient has a foul odor. Patient may go more than 4 days in a row without showering or more than 4 days in a row without brushing his/her teeth. Poor hygiene may present a health risk.
SCORE SHEET

for 4-ITEM POSITIVE SYMPTOM RATING SCALE AND BRIEF NEGATIVE SYMPTOM ASSESSMENT

4-Item Positive Symptom Rating Scale

Use each item’s anchor points to rate the patient.

1. Suspiciousness
   NA 1 2 3 4 5 6 7
2. Unusual Thought Content
   NA 1 2 3 4 5 6 7
3. Hallucinations
   NA 1 2 3 4 5 6 7
4. Conceptual Disorganization
   NA 1 2 3 4 5 6 7 SCORE: _____

4-Item Brief Negative Symptom Assessment

Use each item’s anchor points to rate the patient.

1. Prolonged Time to Respond
   1 2 3 4 5 6
2. Emotion: Unchanging facial expression; blank, expressionless face.
   1 2 3 4 5 6
3. Reduced Social Drive
   1 2 3 4 5 6
4. Grooming and Hygiene
   1 2 3 4 5 6 SCORE: _____

Source of Information (check all applicable) Explain here if validity of assessment is questionable:

_____ Patient
_____ Symptoms possibly drug-induced
_____ Parents/Relatives
_____ Underreported due to lack of rapport
_____ Mental Health Professionals
_____ Underreported due to negative symptoms
_____ Chart
_____ Difficult to assess due to formal thought disorder
_____ Other
_____ Patient uncooperative

Confidence in assessment

_____1=Not at all – 5=Very confident
Documentation

This Chapter begins with charts that compare various sites currently using a systematic approach to medication management. The sites are compared in terms of the content of their data collection forms and the assessments of outcome that they use. Sample forms from the various sites follow the charts.

Chart of Medication Tracking and Data Collection from Texas, New Mexico, Ohio, and New Hampshire Sites

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<th>TMAP</th>
<th>TIMA</th>
<th>UHS</th>
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<td>✓</td>
</tr>
<tr>
<td>Assessment Used at the Texas, New Mexico, Ohio and New Hampshire Sites</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------------------------------------------------</td>
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<td></td>
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</tr>
<tr>
<td></td>
<td>TMAP</td>
<td>TIMA</td>
<td>UHS</td>
<td>NMPI</td>
<td>OMAP</td>
<td>DMHT</td>
</tr>
<tr>
<td>SAS Simpson Angus Scale</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>AIMS Abnormal Involuntary Movement Scale</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>GAF Global Assessment of Functioning Scale</td>
<td>✓</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>MSE Mental Status Exam</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
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<tr>
<td>POS Positive Symptoms</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>NEG Negative Symptoms</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>PANSS Positive and Negative Syndrome Scale</td>
<td>✓</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>BNS Brief Negative Scale</td>
<td></td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>IDS-SR Inventory of Depressive Symptomology self report</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>IDS-C Inventory of Depressive Symptomology clinician admin</td>
<td>✓</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>BPRS Brief Psychiatric Rating Scale</td>
<td>✓</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td>TMAP</td>
<td>TIMA</td>
<td>UHS</td>
<td>NMPI</td>
<td>OMAP</td>
<td>DMHT</td>
</tr>
<tr>
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<td>-----</td>
<td>------</td>
<td>------</td>
<td>------</td>
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<tr>
<td>ALTMAN</td>
<td>✓</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CGI Clinical Global Impression</td>
<td></td>
<td></td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*TMAP  Texas Medication Algorithm Project  
TIMA  Texas Implementation of Medication Algorithms Project  
UHS  University Health System of Texas Clinical Forms  
NMPI  New Mexico Pharmacotherapy Initiative  
OMAP  Ohio Medication Algorithm Project  
DMHT  Dartmouth Medication History and Treatment Forms
Dartmouth Psychiatry: Interim Evaluation for Medication Management

Date:   Name:   ID#:   Time Spent:

**Diagnosis:**

**Medications:**

date/nature of recent change(s) -

**Subjective:**

Symptoms, side effects -

Function -

Stressors/circumstances -

EtOH/drugs – yes, no

Adherent to therapy – yes, no

Current, relevant medical problems -

**Observations:**

(Mental status)

PE/lab -
Assessment of severity/therapeutic response:
Principal recent/ongoing target symptoms: (rate* or check)
___ depressed mood ___ unusual thought content
___ concentration difficulties ___ suspiciousness
___ change in appetite ___ hallucinations
___ loss of libido ___ disorganization
___ insomnia ___ withdrawal
___ irritability ___ hostility
___ elevated mood
___ worry
___ phobic anxiety
___ panic attacks
___ obsessions/compulsions
___ intrusive reexperiencing
___ impulsivity
other:
Symptoms interfering with usual activity: not at all ___, mildly ___, moderately ___, markedly ___
Global Rating of Severity* ___

*Rating anchors: 1-No Sxs, 2-Minimal (no impairment, little concern), 3- Mild, 4-Moderate (frequent and distressing, some interference with function), 5-Moderately Severe, 6-Severe (incapacitated in at least 1 area, symptoms causing substantial distress), 7- Extreme (incapacitating, among the most severely ill)
Global Rating of Change (since time of change of intervention noted in Medications section) __
1-Marked Improvement, 2-Moderate Improvement, 3-Minimal improvement, 4-No Change, 5- Minimally Worse, 6- Much Worse.

**Assessment of tolerance:**
Current side effects: None __, Weight gain __, Sexual interference __, Sedation __, Activation __, Anticholinergic __, Motor: EPS __, akathisia __, TD __.
Other ________________ ________________

Global rating of side effect burden __
1-minimal or no side effects, no impact, 2-noticeable, minimal distress, no effect on function, 3-moderately distressing, some impact on functioning, 4-very distressing, impairing.

**Assessment of risk** (suicidal or homicidal) (circle): no, unchanged, yes
(comment):

**Further impressions/plan/rationale:**

**Signature** __________________________________________________________

**Additional Comments:**
## MEDICATION HISTORY FORM

<table>
<thead>
<tr>
<th>Prescriber Init.</th>
<th>Medication Type</th>
<th>Date Started M/Yr: (299)</th>
<th>Reason/Indication* (see footnote)</th>
<th>Date d/c M/Yr: (599)</th>
<th>Dose</th>
<th>Response (Improvement)</th>
<th>Toler. (Side effects)</th>
<th>Side Effects</th>
<th>Overall Medication Compliance</th>
</tr>
</thead>
<tbody>
<tr>
<td>TM Q1</td>
<td>Clonazapine</td>
<td>400</td>
<td>2 (New)</td>
<td>500</td>
<td>20 mg. 30 mg</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Q2</td>
<td>Clonazapine</td>
<td></td>
<td></td>
<td></td>
<td>40 mg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>Clonazapine</td>
<td></td>
<td></td>
<td></td>
<td>40 mg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q4</td>
<td>Clonazapine</td>
<td></td>
<td></td>
<td></td>
<td>40 mg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SJB Q1</td>
<td>Risperidone</td>
<td>900</td>
<td>3 (Agitation)</td>
<td>700</td>
<td>10 mg 30 mg</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Q2</td>
<td>Quetiapine</td>
<td></td>
<td></td>
<td></td>
<td>20 mg 40 mg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>Quetiapine</td>
<td></td>
<td></td>
<td></td>
<td>40 mg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q4</td>
<td>Quetiapine</td>
<td></td>
<td></td>
<td></td>
<td>40 mg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Reason Codes: 1 = Pos. Sympt.; 2 = Neg. Sympt; 3 = Agitation; 4 = Aggression; 5 = Depression; 6 = Anxiety; 7 = ...; 8 = ...; 9 = ...; 10 = other.
# Psychopharmacology Treatment Flow Sheet

**Pl. Name:**

**Prescriber:**

**Prior Known Psychiatric Medications:**

<table>
<thead>
<tr>
<th>Date</th>
<th>Medication (dose)</th>
<th>Date Started (Mo/Yr.)</th>
<th>Principal Indication(s)*</th>
<th>Date Discont. (Mo/Yr.) (if any)</th>
<th>Response Indicators</th>
<th>Tolerance Indicators</th>
<th>Overall Medication Compliance</th>
<th>Tardive Dyskinesia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>TM 7/22</strong></td>
<td>Olanzapine 10mg</td>
<td>4/99</td>
<td>P, N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lithium 600 mg</td>
<td>87</td>
<td></td>
<td>No, 99</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>29-Sep</strong></td>
<td>Olanzapine 20mg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Nov-99</strong></td>
<td>Risperidone 5 mg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vapomate 1000 mg</td>
<td>5/00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Indication Codes: (target symptoms) - P = Positive symptoms of psychosis; N = Neg. Sympt; A = Agitation; Agg = Aggression; D = Depression; M = Manic; A = Anxiety; In = Insomnia, (other) = SE = management of side effects

**Severity Ratings:** 1 = none; 2 = very mild; 3 = mild; 4 = moderate; 5 = moderately severe; 6 = severe; 7 = very severe (see instruction sheet for target symptom and global descriptors/endorse)

**Side Effects:** EPS, S = Sedation, A = Activation, W = weight gain, S = sexual dysfunction, O = Other (specify)
### Patient Clinic Visit

**Physician Review Form**

<table>
<thead>
<tr>
<th>Local Case #</th>
<th>Component/Clinic #</th>
<th>Date of Visit: mm/dd/yy</th>
<th>Visit Number:</th>
</tr>
</thead>
</table>

#### All Medications In Last Week (Prescription and OTC)

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>Was the medication taken as prescribed?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mostly Yes</td>
</tr>
<tr>
<td></td>
<td>Mostly Yes</td>
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<tr>
<td></td>
<td>Mostly Yes</td>
</tr>
<tr>
<td></td>
<td>Mostly Yes</td>
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<td>Mostly Yes</td>
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</tr>
<tr>
<td></td>
<td>Mostly Yes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>Was the medication taken as prescribed?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mostly Yes</td>
</tr>
<tr>
<td></td>
<td>Mostly Yes</td>
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<td>Mostly Yes</td>
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<td>Mostly Yes</td>
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<td>Mostly Yes</td>
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<td></td>
<td>Mostly Yes</td>
</tr>
<tr>
<td></td>
<td>Mostly Yes</td>
</tr>
<tr>
<td></td>
<td>Mostly Yes</td>
</tr>
</tbody>
</table>

#### Clinical Rating Scales:

- POS SX: 
- NEG SX: 
- PANNS-N: 
- BPRS: 
- IDS-C: 
- IDS-SR: 
- Altman: 

**Overall Patient Global (self report):**

Symptom Severity: [ ] 0 [ ] 1 [ ] 2 [ ] 3 [ ] 4

Side Effects: [ ] 0 [ ] 1 [ ] 2 [ ] 3 [ ] 4

**Staff Time (for this visit):** 

(In minutes)

**Patient/Family Education:**

Done at this visit? [ ] Yes [ ] No

Between last visit and this visit? [ ] Yes [ ] No

Patient Education Activity Log Completed? [ ] Yes [ ] No

#### Most Recent Drug Levels:

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>Date Drawn</th>
<th>Serum Level</th>
</tr>
</thead>
</table>

**Comments:**

__________________________
__________________________
__________________________
__________________________
__________________________
__________________________

**Termination Visit?** [ ] Yes [ ] No

If No, Next Appointment Date: mm/dd/yy

**Clinical Coordinator Signature:**

__________________________
<table>
<thead>
<tr>
<th>Patient Clinic Visit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Record Form</td>
</tr>
<tr>
<td>Service Activity Code:</td>
</tr>
<tr>
<td>Site/Clinic #:</td>
</tr>
<tr>
<td>Physician Code:</td>
</tr>
<tr>
<td>Start Time:</td>
</tr>
<tr>
<td>Stop Time:</td>
</tr>
<tr>
<td>Duration:</td>
</tr>
</tbody>
</table>

**Primary Current Dx:**
- [ ] MDD-NP
- [ ] MDD-P
- [ ] BPD-M
- [ ] BPD-D
- [ ] SCZ
- [ ] SCZ-A
- [ ] Other (specify): |

**Use for all physician's ratings below:**
- 0=No Symptoms
- 1=Borderline
- 2=Mild
- 3=Moderate
- 4=Marked
- 5=Severe
- 6=Extreme

**Core Symptoms:**
- [ ] Mania
- [ ] Depression
- [ ] Positive Sx or Psychoses
- [ ] Negative Sx

**Other Symptoms:**
- [ ] Irritability
- [ ] Mood Lability
- [ ] Insomnia
- [ ] Agitation
- [ ] Anxiety
- [ ] Other (specify): |

**Overall Side Effect Severity:** |

**Is patient presently suicidal?**
- [ ] Yes
- [ ] No

**homicidal?**
- [ ] Yes
- [ ] No

**Overall Functioning:** (1-100) |

### Prescription Information

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>New/Continuing/Discontinue</th>
<th>Please provide information on starting dose, frequency, duration the medication is to be taken, start and stop date (if applicable), and any other pertinent information describing this medication.</th>
<th>Indication check all that apply?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>[ ] S</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>[ ] S</td>
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<td>[ ] S</td>
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<td></td>
<td></td>
<td></td>
<td>[ ] S</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>[ ] S</td>
</tr>
</tbody>
</table>

- [ ] Meds Targeted at specific syndromes. OS=Meds targeted at other symptoms. SE=Meds for side effects of S or OS.
- [ ] Are serum levels needed?  [ ] Yes  [ ] No  (if yes, specify below)

Comments: |

Is a change from the algorithm recommended?  [ ] Yes  [ ] No

Algo Stage: |

If yes, check all that apply:  [ ] No options left (ALGO ran out).  [ ] Next step not acceptable to patient.  [ ] Patient previously failed next (or 1st) step.

Algo Version: |

Side effect algo implemented?  [ ] Yes  [ ] No

Other symptom algo implemented?  [ ] Yes  [ ] No

Return to clinic: _______ weeks

Physician Signature: |

------
# TIMA Texas Implementation of Medication Algorithms

## Outpatient Intake Form

**Date of Visit:**

**MHMR Physician Code:**

**Age:** __

**Gender:**

- [ ] Female
- [ ] Male

**Ethnic or Racial Group** (please check only one response):

- [ ] White
- [ ] Hispanic
- [ ] African American
- [ ] Asian or Pacific Islander
- [ ] American Indian or Alaskan Native
- [ ] Other

**Principal Diagnosis (DSM-IV Axis I code):**

**Age at Onset:** __________

**# of Episodes:** __________

**Onset of Current Episode:**

**Other current diagnoses not including principal diagnosis:**

**Axis I:** __________________________________________

**Axis II:** __________________________________________

**Alcohol/Substance Abuse:**

- [ ] No
- [ ] Yes

  *if yes, [ ] Current [ ] Past*

**Axis III (Current General medical conditions, check all that apply):**

- [ ] Hypertension
- [ ] Hypothyroidism
- [ ] Head injury
- [ ] HIV
- [ ] CHF
- [ ] Diabetes
- [ ] Seizure Disorder
- [ ] Cancer
- [ ] Heart Disease
- [ ] Endocrine (Other)
- [ ] Stroke
- [ ] Chronic Lung Disorder
- [ ] Cardiac (Other)
- [ ] Asthma
- [ ] Neurological (Other)
- [ ] Allergies (If yes, explain below)
- [ ] Other

**Significant Systemic Illness (specify):**

**Additional Information:**

________________________________________________________________________________________

---

**Any family members with a history of any of the following (please check all that apply):**

<table>
<thead>
<tr>
<th>Parent</th>
<th>Depression</th>
<th>Schizophrenia</th>
<th>Bipolar</th>
<th>Substance Abuse</th>
<th>Suicide</th>
<th>Other</th>
<th>Effective Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sibling</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Children</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aunt/Mnicie</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grandparent</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

**Number of Psychiatric Hospitalizations (best estimate):**

- [ ] Past Year: __________
- [ ] Past 5 Years: __________
- [ ] Lifetime: __________

**Past and Current Psychoactive Medications (Patient Self-Report/Records):**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Taken for this episode?</th>
<th>Dose</th>
<th>Freq.</th>
<th>Start/Stop (Mo/YY)</th>
<th>Response</th>
<th>Well Tolerated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>7.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>8.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>[ ] Yes [ ] No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Signature / Title:** __________

**Date:** __________
### TIMA Texas Implementation of Medication Algorithms
#### OutPatient Clinic Visit

**Clinical Record Form**

**Date:**

**Physician Code:**

**Service Activity Code:**

**Start Time:**

**Stop Time:**

**Primary Current Dx:**
- [ ] MDD-NP
- [ ] MDD-P
- [ ] BPD-M
- [ ] BPD-MX
- [ ] SCZ-A (BP)
- [ ] SCZ-A
- [ ] Other (specify):

**Stage:**

**Weeks in this stage:**

**Vital Signs:**
- BP
- Pulse
- Temp
- Weight
- Height (if needed)

**Most Recent Drug Levels:**

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>Date Drawn</th>
<th>Serum Level</th>
<th>WHL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Medications taken as Prescribed:**
- [ ] Yes/Mostly
- [ ] No/Inadequate

**Any other medications taken during the past week:**
- [ ] No
- [ ] Yes (Specify below)

---

**Patient Global Self Report (0-10) 0 = No symptoms 5 = moderate 10 = extreme**

**Symptom Severity:**

**Side Effects:**

**Clinical Rating Scales**

**POS SX:**

**NEG SX:**

**IDS-SR:**

**Altman:**

**OTHER:**

**Use for all physician's ratings below (0-10):**

**0 = No symptoms**

**5 = moderate**

**10 = extreme**

**Core Symptoms:**
- Mania
- Depression
- Positive Sx or Psychoses
- Negative Sx

**Other Symptoms:**
- Irritability
- Mood Lability
- Insomnia
- Agitation
- Anxiety
- Level of Interest
- Appetite
- Energy Level
- Other (specify):

**Overall Side Effect Severity:**

**Overall Functioning:**

**If yes, in progress note:**

---

**Prescription Information**

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>Change from previous visit?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>[ ] No [ ] Yes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>New/ Continuing/ Discontinue</th>
<th>Prescription Information describing this medication</th>
<th>Indication (check all that apply)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td><img src="image.png" alt="Image" /></td>
</tr>
</tbody>
</table>

* Mdes = Meds targeted at core syndrome. OS = Meds targeted at other symptoms. SE = Meds for side effects of 5 or OS.

TIMA CRF-Outp
Final Version
12/27/00
Page 1 of 2
TIMA Texas Implementation of Medication Algorithms

Are serum levels needed? □ Yes □ No (if yes, specify in progress note)

Medication Response: □ Full □ Partial □ Minimal □ None □ Symptoms Worsening

Reason for Medication Change (Include Dose Changes):
□ Critical Decision Point Indicates Change Necessary □ Insufficient Improvement □ Patient Preference
□ Side Effects Intolerable □ Symptoms Worsening □ Diagnosis Change □ Other:

Reason for Antidepressant Choice: □ SE Profile □ Pattern of Associated Sx □ Past Response □ Other:

Reason for Antipsychotic Choice: □ SE Profile □ Pattern of Associated Sx □ Past Response □ Other:

Reason for Mood Stabilizer Choice: □ SE Profile □ Pattern of Associated Sx □ Past Response □ Other:

Reason for Augmentation Choice: □ SE Profile □ Pattern of Associated Sx □ Past Response □ Other:

Patient/Family Education:
Done at this visit? □ Yes □ No
Between last visit and this visit? □ Yes □ No

Progress Note: (☐ Check here if note was dictated. Date of dictation ___/___/___)

Subjective (Sleep, appetite, side effects, medication efficacy, other patient reports.)

Objective (Orientation, appearance, rapport, speech patterns, suicidal or homicidal ideations, psychotic thought content & process, mood, affect, insight, judgement, cognitions, other observations)

Assessments (Diagnosis, clinical progress, formulations, problems, prognosis, other appraisals.)

Plan (Current direction for biopsychosocial treatment, discharge planning, placements, other needs.)

Return to clinic: _______ weeks

Signature/Title:

TIMA CRF-Output
Final Version

12/27/00
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TIMA Texas Implementation of Medication Algorithms

Outpatient Interim Contact Form

Case #: ___________  Date: ___________

Primary Diagnosis: [ ] MDD-NP  [ ] BPD-M  [ ] BPD-D  [ ] SCZ
[ ] MDD-P  [ ] BPD-MX  [ ] BPD-A (BP)  [ ] SCZ-A  [ ] Other (specify): ___________

Type of Contact: [ ] Telephone  [ ] Office Visit

All Prescription Medications In Last Week

Medication Name – Please provide information on dosing, frequency and any other pertinent information.

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>Was the medication taken as prescribed?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>[ ] Yes  [ ] No</td>
</tr>
<tr>
<td>2.</td>
<td>[ ] Yes  [ ] No</td>
</tr>
<tr>
<td>3.</td>
<td>[ ] Yes  [ ] No</td>
</tr>
<tr>
<td>4.</td>
<td>[ ] Yes  [ ] No</td>
</tr>
<tr>
<td>5.</td>
<td>[ ] Yes  [ ] No</td>
</tr>
</tbody>
</table>

Adherence to medication treatment? [ ] Yes  [ ] No  If no, document in progress note.

Significant Side Effects Reported? [ ] Yes  [ ] No  If yes, describe:

__________________________________________________________________________

Overall Patient Global (self report): 6=none 5=moderate 10=extreme
Symptom Severity: (0-10) Side Effects: (0-10)

Is patient currently suicidal? [ ] Yes  [ ] No  Is patient homicidal? [ ] Yes  [ ] No

Progress Note

__________________________________________________________________________

__________________________________________________________________________

__________________________________________________________________________

__________________________________________________________________________

Stage: _______  Weeks in Stage: _______  Change to Treatment Recommended? [ ] YES  [ ] NO

IF yes, schedule physician visit.  Appointment Date: _______/_____/______

Signature/Title: ____________________________________________________________________
Clinical Inpatient Record
Progress Note

Check here if note was dictated
Date and Time of Dictation: ____________________________
Date and Time of Exam: ____________________________

Patient seen and chart reviewed? □ Yes □ No
Level of Service □ Low □ Medium □ High
Review Frequency: □ Daily □ Weekly □ Monthly □ Quarterly □ Other

1. SUBJECTIVE FINDINGS:

- Appetite: □ Normal □ Good □ Poor □ Overeating
- Sleep: □ Normal □ Good □ Poor □ Fair
- Side Effects: □ Involuntary Movements □ Appetite □ Tremors □ GI □ Akathisia □ Sexual □ Other:
- Medication Efficacy: □ Excellent □ Poor □ Good □ Fair

Comments: ________________________________________

2. OBJECTIVE FINDINGS:

- Orientation: □ Person □ Place □ Time □ Situation
- Rapport: □ Appropriate □ Hostile □ Distant □ Inattentive □ Poor Eye Contact
- Appearance: □ Appropriately Dressed □ Poorly Dressed □ Disheveled □ Bodily Odor
- Mood: □ Euthymic □ Depressed □ Angry □ Irritable □ Elated □ Labile
- Affect: □ Appropriate □ Depressed □ Blunted □ Flat
- Speech: □ Coherent □ Appropriate □ Incoherent □ Slurred

Thought Content & Process:
- □ Appropriate □ Thought Insertion □ Paranoia □ Phobias □ Hopelessness □ Self Deprecation
- □ Goal Directed □ Broadcasting □ Grandiose □ Suicidal Ideation □ Worthlessness □ Hallucinations
- □ Delusional □ Obsessions □ Pseudopsychosis □ Suicidal Plan □ Auditory Hallucinations
- □ Persecution □ Obsessions □ Passional Psychosis □ Homicidal Ideation □ Visual Hallucinations
- □ Reference □ Compulsions □ Captusional Psychosis □ Homicidal Pas

Insight: □ Excellent □ Good □ Poor □ Grossly Impaired
- Judgment: □ Excellent □ Good □ Poor □ Grossly Impaired
- Cognitive: □ No Gross Cognitive Deficit □ Concentration Problems □ Abstract □ Easily Distracted
- Psychomotor Activity: □ Normal □ Restless □ Retarded
- Memory: □ Good □ Fair □ Impaired

Comments: ________________________________________

3. ASSESSMENTS:

Psychiatric condition is generally: □ Improving □ Unchanged □ Deteriorating

4. PLAN:

____________________________________________________________________________________________________

Are serum levels needed? □ Yes □ No

Medication Name Date Drawn Serum Level

Labs WNL? □ Yes □ No If no, describe below.

Pertinent Lab Data: ____________________________

□ Initial Certification: Patient could receive proper treatment in a SNF, but no bed is available.
Psychiatric Hospital Services continues to be medically necessary for:

□ Treatment which can reasonably be expected to improve the patient’s condition and/or □ Diagnostic Study

Physician Signature: ____________________________

Revised: 12/99
Approved/Reviewed by the Medical Records Committee: 7/99

TINA MHRS 5-2.1 (Front)
Clinical Inpatient Record
Progress Note

COMPLETE THIS SECTION IF PATIENT IS AN ALGORITHM CLIENT

Stage: ___________________________ Weeks in this stage: ___________________________

Patient Education Completed? □ Yes □ No

Primary Current Dx: □ MDD-NP □ BPD-M □ BPD-D □ SCZ □ Other (specify) ___________________________

(Check one) □ MDD-P □ BPD-MX □ SCZ-A(BP) □ SCZ-A ___________________________

Use for all physician’s ratings below: (0-10) 0 = No symptoms 5=Moderate 10=Extreme Leave blank if they do not apply

Core Symptoms: __________ Mania __________ Depression __________ Positive Sx of Positive Psychosis __________ Negative Sx of Psychosis

Other Symptoms: __________ Irritability __________ Mood Lability __________ Insomnia __________ Agitation __________ Anxiety

__________________________________________ __________________________

Psychotropic Medication Information

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>Dosing Information</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Document any new or discontinued medications or dosage change of established medications. Please provide information on titration, dose, dose frequency, duration the medication is to be taken, start and stop date (if applicable) and any other pertinent information describing this medication.</td>
<td>(Check all that apply.)</td>
</tr>
<tr>
<td>-----------------</td>
<td>--------------------</td>
<td>------------</td>
</tr>
<tr>
<td>□ New Change □ D/C</td>
<td></td>
<td>□ S</td>
</tr>
<tr>
<td>□ New Change □ D/C</td>
<td></td>
<td>□ OS</td>
</tr>
<tr>
<td>□ New Change □ D/C</td>
<td></td>
<td>□ SE</td>
</tr>
<tr>
<td>□ New Change □ D/C</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

□ Medication unchanged from before

S=Meds Targeted at core syndrome OS=Meds targeted at other symptoms SE=Meds for side effects of S or SO

Deviation from medication algorithm recommended? □ Yes □ No (If yes, check all that apply)

□ Patient previously failed next step □ Next step not acceptable □ Next step not available at this site

□ Next step not medically safe for this patient □ No options left □ Other ___________________________

Reason for Medication Choice: □ SE Profile □ Pattern of Associated SX □ Past Response

□ Other ___________________________

__________________________________________ __________________________

Patient Global Self Report (0-10) 0=No symptoms 5=Moderate 10=Extreme

Symptoms Severity: ___________________________ Side Effects: ___________________________

Clinical Rating Scales

<table>
<thead>
<tr>
<th>MMSE</th>
<th>AIMS</th>
<th>POS SX</th>
<th>NEG SX</th>
<th>IDS-SR</th>
<th>Altman</th>
<th>Other</th>
</tr>
</thead>
</table>

__________________________________________ __________________________

Revised: 12/99

Approved/Reviewed by the Medical Records Committee: 9/99

TMA
Thought Disorder Clinic
Progress Notes

**Clinical Guideline Reference:** Thought Disorder

**Reason for visit:**  
- [ ] Follow-up
- [ ] Unscheduled visit for:

**Current Medications:**

**Patient's status since last visit:**

---

**MENTAL STATUS EXAM (Use the following ratings to complete MSE):**

- **0=None**
- **1=Very Mild/Questionable**
- **2=Mild**
- **3=Moderate**
- **4=Moderate Severe/Marked**
- **5=Severe**
- **6=Extremely Severe/Gross Disability**

<table>
<thead>
<tr>
<th>ASSESSMENT CATEGORY</th>
<th>AREA OF ASSESSMENT</th>
<th>RATING</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance</td>
<td>Patient dresses in an unusual manner or does other strange thing to alter his/her appearance.</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Mood and Affect</td>
<td>Depressed Mood (Sadness, hopelessness, helplessness, worthlessness)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Excitement-Patients heightened emotional tone, agitation, increased reactivity</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Affect</td>
<td>Blunted Affect—Patients reduced emotional tone, reduction in normal intensity of feelings, flatness.</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Elevated or expansive mood and/or optimistic attitude toward the future which lasted at least several hours and was out of proportion to the circumstances.</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Sensorium</td>
<td>Disorientation—Confusion or lack of proper association for person, place or time (Rate errors: 1 mild, 3-6 moderate, 6 or more severe)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Orientation</td>
<td>Spell WORLD forwards and backwards (Rate Errors: 1 mild, 2 errors moderate, 3 or more severe)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Recent Memory</td>
<td>Registration: Examiner will name 3 objects (1 second each). Ask patient to repeat all three words. Repeat as necessary until patient learns all 3 (up to 6 trials).</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Thought Coherence</td>
<td>Conceptual Disorganization—Patients thought processes confused, disconnected, disorganized, disrupted</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Perceptions</td>
<td>Hallucinatory Behavior—perceptions without normal external stimulus correspondence.</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Hallucinations</td>
<td>Unusual Thought Content—unusual, odd, strange, bizarre thought content</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Content</td>
<td>Suicidal tendencies, including preoccupations with thoughts of death and suicide</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hostility—Patients animosity, complaints, belligerence, disdain for others</td>
<td>NA</td>
<td></td>
</tr>
</tbody>
</table>

**Additional Comments:**

**PROGRESS NOTES THOUGHT DISORDER CLINIC**

- [ ] I INPATIENT
- [ ] I OUTPATIENT

**Chart Order:**

IP 365 OP
### Lab Results:

| □ None | EXC Results: | □ None |

### Other:

- Current Drug Use (last 4 weeks) □ None □ Yes: Type __________ Quantity: __________ Frequency: __________
- Current Alcohol Use (last 4 weeks) □ None □ Yes: Type __________ Quantity: __________ Frequency: __________

### A:

- AXIS I: MMSE (score) (date)
- AXIS II: GAF (score) (date)
- AXIS III: AIMS (score) (date)

### Medication Side Effects:

- □ Akathisia □ Pseudoparkinsonism □ Stiffness □ Acute Dystonia □ Sexual Dysfunction
- □ Dry Mouth □ Constipation □ Drooling □ Weight Gain (wt.: ______) □ None □ Other: ____________________________

### Severity of Illness:

|------------------|--------------------------|---------------|-----------|---------------|-------------|------------|----------------------------------------|

### Continue Current Medications:

### Medications Changed / Added:
## University Health System
### Schizophrenia Rating

<table>
<thead>
<tr>
<th>Date of Survey (month-day-year)</th>
<th>Next Flu Visit Date (month-day-year)</th>
<th>Next Critical Visit Date (mm-dd-yyyy)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Stage:**
- 1: Admission
- 2: Outpatient MH
- 3: Emergency Center
- 4: Discharge
- 5: Outpatient ATC

**Clinical Guideline Reference: Schizophrenia**

<table>
<thead>
<tr>
<th>Rating</th>
<th>Not Assessed</th>
<th>Not Present</th>
<th>Very Mild</th>
<th>Mild</th>
<th>Moderate</th>
<th>Moderately Severe</th>
<th>Severe</th>
<th>Extremely Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>NA</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>1</td>
<td>NA</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>NA</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>NA</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<td>4</td>
<td>NA</td>
<td>0</td>
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<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
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<td>NA</td>
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<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>NA</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

**Rate items on the basis of patient's self-report during interview. Item 2 is also rated on observed behavior during the interview. Item 4 is rated on the basis of observed behavior and speech.**

1. **Suspiciousness**
   - NA: 0
   - Not Assessed: 1
   - Not Present: 2
   - Very Mild: 3
   - Mild: 4
   - Moderate: 5
   - Moderately Severe: 6
   - Severe: 7
   - Extremely Severe: 8

2. **Unusual Thought Content**
   - NA: 0
   - Not Assessed: 1
   - Not Present: 2
   - Very Mild: 3
   - Mild: 4
   - Moderate: 5
   - Moderately Severe: 6
   - Severe: 7
   - Extremely Severe: 8

3. **Hallucinations**
   - NA: 0
   - Not Assessed: 1
   - Not Present: 2
   - Very Mild: 3
   - Mild: 4
   - Moderate: 5
   - Moderately Severe: 6
   - Severe: 7
   - Extremely Severe: 8

4. **Conceptual Disorganization**
   - NA: 0
   - Not Assessed: 1
   - Not Present: 2
   - Very Mild: 3
   - Mild: 4
   - Moderate: 5
   - Moderately Severe: 6
   - Severe: 7
   - Extremely Severe: 8

5. **Prolonged Time Response**
   - NA: 0
   - Not Assessed: 1
   - Not Present: 2
   - Very Mild: 3
   - Mild: 4
   - Moderate: 5
   - Moderately Severe: 6
   - Severe: 7
   - Extremely Severe: 8

6. **Emotion**
   - Unchanging facial expression blank, expressionless face.
   - NA: 0
   - Not Assessed: 1
   - Not Present: 2
   - Very Mild: 3
   - Mild: 4
   - Moderate: 5
   - Moderately Severe: 6
   - Severe: 7
   - Extremely Severe: 8

7. **Reduced Social Drive**
   - NA: 0
   - Not Assessed: 1
   - Not Present: 2
   - Very Mild: 3
   - Mild: 4
   - Moderate: 5
   - Moderately Severe: 6
   - Severe: 7
   - Extremely Severe: 8

8. **Poor grooming and hygiene**
   - NA: 0
   - Not Assessed: 1
   - Not Present: 2
   - Very Mild: 3
   - Mild: 4
   - Moderate: 5
   - Moderately Severe: 6
   - Severe: 7
   - Extremely Severe: 8

**Physician's Signature**

245
NMPI Intake Form

Local Case # ___________ Physician Code ___________ Component / Clinic # ___________
Date of Visit: _____ / _____ / _____

Coordinator Name: __________________________

Age: _______ Gender: □ Female □ Male Ethnic or Racial Group (please check only one response): □ White □ Hispanic
□ African-American □ American Indian or Alaskan Native □ Asian or Pacific Islander □ Other

Principal Diagnosis (DSM-IV Axis I Code): ___________ • ___________ Age at Onset: _______

Other current diagnosis not including principal diagnosis:
Axis I: ___________ • ___________ _______

Alcohol / Substance Problem (within last 6 months): □ Yes □ No

Axis III (Current General medical conditions, check all that apply): □ Tardive Dystonias AIMS Score ______
□ Hypertension □ Diabetes □ HIV Disease □ Closed Head Injury with Loss of Consciousness
□ Heart Failure □ Hypothyroid □ Cancer □ Head Injury □ Ischemic Heart Disease □ Arthritis
□ Chronic Lung Disease □ Seizure Disorder □ Other Significant Systemic Illness (specify): _______________________

Have any family members been treated for the following (please check all that apply):

<table>
<thead>
<tr>
<th>Father</th>
<th>Depression</th>
<th>Schizophrenia</th>
<th>Bipolar</th>
<th>Alcohol Abuse</th>
<th>Drug Abuse</th>
<th>Suicide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sister(s)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brother(s)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Son(s)</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Daughter(s)</td>
<td></td>
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<td></td>
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<td></td>
</tr>
</tbody>
</table>

Number of Psychiatric Hospitalizations (best estimate): Past Year: _____ Past 5 Years: _____ Lifetime: _____

Does Patient Have Primary Care Access: □ Yes □ No

Please provide medications for the past two years, record the highest dose given.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Current</th>
<th>Dose</th>
<th>Freq.</th>
<th>Weeks on</th>
<th>Response *</th>
<th>Reason for Discontinuation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Yes</td>
<td>No</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
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<td></td>
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<tr>
<td>3</td>
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<tr>
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</tbody>
</table>

Date Visit #1 is Scheduled For: _____ / _____ / _____ (can be the same date as visit, i.e., intake)

* F = Full P = Partial N = None U = Unknown

Reason for Enrollment: ___ New/recent diagnosis ___ Improve treatment effectiveness ___ Reduce side effects ___ Economic need
N MPI
Patient Clinic Visit
Rev. 9/6/99

Date: ___/___/___  Patient Initials: __________  Patient #: __________

Site/Clinic #: ______/____ MD #: ___ Visit #: ___ Algor. Stage: ___


Primary Current Dx:  ____ SCZ  ____ SCZ-A (Depressed Type)


CGI: ___/___  SAS (if assessed): ___ AIM (if assessed): ___ Weight: ___

Is patient presently suicidal? ___Y___N  Homicidal? ___Y___N
Substance use since last visit? Alcohol: ___ Y___N  Illicit drug: ___ Y___N

Urine TOX screen (circle):  +  or  -

### Prescription Information

<table>
<thead>
<tr>
<th>Medication Name</th>
<th>New/Continuing/Discontinue</th>
<th>1. Titration, dose, dose frequency, duration</th>
<th>2. Dosage taken if different from prescribed</th>
<th>Compliance F=Full, P=Partial, U=Unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>New</td>
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<td>Cont.</td>
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<td>D/C</td>
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<td>U</td>
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</tbody>
</table>

### Rx for Patient Education / Support:

**WHO**
(MD, CC, PA, PEC)

**WHAT**
(Indiv., Family, Group)

**FOCUS**
(i.e. acceptance, med. info, dx. info., support)

### Notes and Comments
- Other side effects being monitored (i.e. weight, sexual dysfunction);
- Lab results; major events (i.e. jail, hospitalization); response to patient education/support; status

### Is a change from the algorithm recommended?

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
<th>Algo Stage:__________________</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Next step not acceptable to patient.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Next step not medically safe for this patient.</td>
</tr>
</tbody>
</table>

Return to clinic _______ weeks

Peer Advocate: ___________________  Physician: ___________________

Patient Ed. Coord.: ______________  Clinical Coord.: ______________
<p>| Date | 29 | 30 | 31 | 32 | 33 | 34 | 35 | 36 | 37 | 38 | 39 | 40 | 41 | 42 | 43 | 44 | 45 | 46 | 47 | 48 | 49 | 50 | 51 | 52 | 53 | 54 | 55 | 56 | 57 |
|------|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| 4-Item | BPRS |
| 28 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 26 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 24 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 22 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 20 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 18 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 16 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 14 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 12 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 10 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 8  |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
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| 4  |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| BNSA |     |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
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| 22 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 20 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 18 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 16 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 14 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 12 |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
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| 6  |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| 4  |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |</p>
<table>
<thead>
<tr>
<th>LOCAL CASE #</th>
<th>SITE/Clinic #</th>
<th>DATE</th>
<th>PROVIDER</th>
<th>RECIPIENTS</th>
<th>TIME in minutes</th>
<th>MATERIALS USED</th>
</tr>
</thead>
<tbody>
<tr>
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<td>1: Phenytoin</td>
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<td>2: Phenytoin</td>
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<td></td>
<td></td>
<td></td>
<td>3: Phenytoin</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Completed by: [Signature]  
Provider Code: [Code]  
Date: [Date]
**Site Name:** QCM, Inc.

**Client Name:**

**Physician Code:**

**Age:**

**Gender:** □ Female  □ Male

**Ethnic or Racial Group (please check any one):**  □ White □ Hispanic □ African-American □ Asian or Pacific Islander □ American Indian or Alaskan Native □ Other

**Chief Complaint:**

**HPI (include past H’ history):**

**Number of Psychiatric Hospitalizations (best estimate):**

Past Year:  
Past 5 Years:  
Lifetime:  

**Psychosocial:** (living situation, marital status, children, school, job, military, legal history)

**Family psychiatric and substance use history (diagnosis? suicide? treatment?):**

**Current Medications: (psychiatric, non-psychiatric, and herbal):**

| 1. |  |  |  |  |
| 2. |  |  |  |  |
| 3. |  |  |  |  |
| 4. |  |  |  |  |
| 5. |  |  |  |  |
| 6. |  |  |  |  |
| 7. |  |  |  |  |
| 8. |  |  |  |  |
| 9. |  |  |  |  |
| 10. |  |  |  |  |

07/17/00
Physician Progress Note Form

Site Name: QC/M, Inc.

Client Name: __________________

Physician Code: _______ Start time: _______

Dx: (check one) □ SCZ □ SCZ-A □ SCZ-A (BP) □ BPD-M □ BPD-D □ BPD-MX □ MDD-NP
□ MDD-P □ Other (specify): __________

Current OMAP Stage (if applicable): _______ Weeks in this stage: _______

Clinical Rating Scales (if applicable):

Patient Global Self Report (0-10) 0 = No Symptoms 5 = Moderate 10 = Extreme

Symptom Severity: __________ Side Effects: __________

Physician Assessment (0-10) 0 = No Symptoms 5 = Moderate 10 = Extreme

Overall Functioning: __________ Overall Side Effect Severity: __________ (0-10)

Core Symptoms: □ Mania □ Depression □ Positive Sx or Psychoses □ Negative Sx

Other Symptoms: □ Irritability □ Mood Lability □ Insomnia □ Agitation
□ Anxiety □ Level of interest □ Appetite □ Energy Level □ Other

Is the patient presently suicidal? □ yes □ no □ Homicidal? □ yes □ no
(If yes, provide detail in progress note.)

Medications taken as prescribed? □ Yes/Mostly □ No/Inadequate

Any new medications taken during the past week? □ No □ Yes (Explain below.)

Current Medications: (psychiatric, non-psychiatric, and herbal)

| 1. |
| 2. |
| 3. |
| 4. |
| 5. |
| 6. |
| 7. |

*Full, partial, minimal, none, symptoms worsening*

Pertinent Laboratory Tests/Med. Levels (if applicable):

______________________________________________________________

______________________________________________________________

Results Discussed with Patient? □ Yes □ No

Current Medical Issues/Pertinent ROS (specify if new from last visit):

______________________________________________________________

pregnancy: □ Yes □ No

Vital Signs: (if needed) BP _____/_____ Pulse _____ Temp _____ Weight _____ Height _____

New Psychosocial Issues Since Last Visit: (if applicable)

______________________________________________________________

Substance Use: (include ETOH, THC, other substances (specify), tobacco)

______________________________________________________________

OMAP – Ohio Medication Algorithm Project
07/19/00
Progress Note:

Subjective (sleep, appetite, side effects, medication efficacy, other patient reports.)

Objective (orientation, appearance, rapport, speech patterns, suicidal or homicidal ideations, psychosis thought content & process, mood, affect, insight, judgment, cognition, other observations)

Assessments (diagnosis, clinical progress, formulations, problems, prognosis, other appraisals)

Plan (medication choice and rationale*, direction for biopsychosocial treatment, discharge planning, placements, other needs)

*for rationale, report if medications are targeted for core symptoms, targeted for other symptoms, used to treat side effects, or chosen for side effect profile or past response.

Reason for Medication or Dose Change (if applicable):
- Critical Decision Point Indicates Change Necessary
- Insufficient Improvement
- Patient Preference
- Side effects intolerable
- Symptoms Worsening
- Diagnosis Change
- Other

Explained to patient reasons for medication choices and possible side effects, risks, benefits, and alternative treatment: ☐ Yes ☐ No

Patient/Family Education Done at This Visit: ☐ Yes ☐ No

OMAP Stage at End of Session: __________

Return to the clinic: _____ weeks  Next appointment date: _____/_____/_____

Signature/Title:

OMAP - Ohio Medication Algorithm Project
07/19/00