Schizophrenia, Tardive Dyskinesia, and the Abnormal Involuntary Movement Scale (AIMS)

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The incidence of tardive dyskinesia as a side effect of antipsychotic medications is well documented in the literature on the treatment for schizophrenia. Although the new generation of atypical neuroleptics helps diminish the incidence of such side effects, a complete elimination has yet to be fully realized. Nurses continually observe and assess patients. As such, nurses in all settings can effectively contribute to both preventative and palliative care of the patient who is about to receive or is currently receiving antipsychotic medications. Familiarity with the Abnormal Involuntary Movement Scale (AIMS) and expertise in the application of AIMS in patient assessment assist in these important prevention efforts. (J Am Psychiatr Nurses Assoc [2002]. 8, 51-6.)

Schizophrenia is a severe mental disorder that affects approximately 1% of the population. Its course is generally chronic with acute psychotic exacerbations. Persons with schizophrenia require long-term therapy with antipsychotic medications, and these frequently produce a wide range of extrapyramidal side effects (EPS) that can have a negative impact on the individual's functional status (Awad, Voruganti, & Heslegrave, 1997; Herz et al., 2000). Dystonia, parkinsonism, and akathisia are the most common medication-related movement disorders (Herz et al.; Kaplan & Sadock, 1998; Keks, 1996). Tardive dyskinesia (TD) is a gradually appearing EPS that may arise as a result of sustained exposure to any antipsychotic medication, except clozapine. TD manifests as a sometimes persistent, involuntary movement disorder that can be irreversible (Egan, Apud, & Wyatt, 1997; Germer, Seraydarian, & McBrearty, 1984; Herz et al.; Whall, Engle, Edwards, Bobel, & Haberland, 1983).

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BACKGROUND
Extrapyramidal Motor System

The extrapyramidal motor system, located in the basal ganglia, is a collection of neuronal pathways that provide significant input into involuntary motor movements. Nerves from the nuclei of the putamen, globus pallidus, and caudate structures that make up the majority of the extrapyramidal motor system connect with the cerebral cortex, thalamus, midbrain structures, spinal cord, and with each other (Boyd & Nihart, 1998).

Within the midbrain are nuclei of the substantia nigra, the cells of which project to the striatum. The striatum, which contains the caudate nucleus and putamen of the basal ganglia, forms the nigrostriatal pathway. The nigrostriatal pathway has primary control over involuntary motor movements. Other parts of the midbrain, the tegmentum and the substantia nigra, are also part of the motor system. The reticular system, found within the tegmentum, influences functions, such as motor activity, sleep, consciousness, righting reflexes (maintaining the position of the head in relation to the rest of the body), and basic attention capacities. In Parkinson's disease, the cells of the substantia nigra are severely affected, producing many of the involuntary movements associated with this disease. Parkinsonism is an acute EPS and is associated with TD (Boyd & Nihart, 1998).

Tardive Dyskinesia: Mechanisms and Clinical Features

Cardoso and Jankovic (1997) acknowledged that the underlying mechanism of TD remains to be deter-

mined. They suggested that the dopamine receptor-blocking action of the antipsychotic agents and the transient improvement of TD with use of higher doses of neuroleptic medications support the notion of up-regulation of dopamine receptors in the basal ganglia. It is thought that the D_2-receptor blockade disables crucial neural pathways in the basal ganglia that are involved in the control of both voluntary and involuntary movements. Thus, D_2-receptor blockage may disinhibit the circuits that determine the clinical expression of extrapyramidal abnormalities of muscle tone and movement (Kaplan & Sadock, 1998). Other hypothesized mechanisms underlying the disarrayed loops of feedback and feed-forward regulation in the basal ganglia that manifests as TD are imbalance between D_1- and D_2-subpopulation receptors, changes in gamma-amino butyric acid (GABA, an amino acid that acts as a neurotransmitter), or free radicals generation (Cardoso & Jankovic; Kaplan & Sadock).

TD is characterized by a wide variety of involuntary movements and tics, including lip smacking, biting, clenching, chewing, sucking, tongue thrusting, and jaw movements (American Psychiatric Association, 1999; Germer et al., 1984; Kane et al., 1992; Munetz & Benjamin, 1988; Tarsy, 1984). TD is clinically expressed as stereotypes (repetitive, purposeless, but seemingly purposeful movements) and dystonias (briefly sustained or fixed abnormal postures). TD expresses with features of parkinsonism (tremor, rigidity, bradykinesia), akathisia (restlessness, pacing, rocking), and choreiform (rapid, objectively purposeless, irregular) or athetoid (slow, writhing, serpentine) movements of the upper and lower extremities (hands, fingers, wrists, arms and legs, knees, ankles, toes). In addition, there may be trunk movements with the neck, shoulders, or hips displaying rocking, twisting, or squirming. There may also be hip gyrations (Cardoso & Jankovic, 1997; Kaplan & Sadock, 1998; Tarsy; Whall et al., 1983).

Tardive Dyskinesia: Prevalence

General acceptance of the association of TD with long-term neuroleptic treatment came in the early 1970s, although the first reported cases of TD related to the administration of chlorpromazine (Thorazine) occurred earlier in the French (Delay & Deniker, 1952) and German (Schonecker, 1957) medical literature (Egan et al., 1997). With acceptance came therapeutic trials to study the prevalence of TD and the relationship of this disorder to neuroleptic exposure. Estimates of the prevalence of TD have ranged from 0.5% to 62%. The average yearly rate of developing TD from exposure to neuroleptics is 4% to 5% per year for the first several years (Egan et al.; Kopala, 1996). It is unclear whether the risk levels off after 5 years or continues to increase linearly for 10 years or longer (Egan et al.).

The 10-year risk is estimated to be 49%, and the 25-year risk is estimated to be 68%. Sustained antipsychotic medication exposure without dose reduction after the development of TD diminishes the likelihood of reversibility (Gureje, 1988). However, the severity of TD does not seem to increase with time when steady, moderate doses of antipsychotic medication are used (Herz et al., 2000).

Several authors warn that movements resembling TD are sometimes seen in unmedicated patients diagnosed with schizophrenia (Ahrens, Sramek, Herrera, Jewett, & Alcorn, 1988; Kaplan & Sadock, 1998; Kopala, 1996; Manschreck et al., 1990). From Kraepelin's diagnosis of schizophrenia in 1919 to the appearance of antipsychotics in the 1950s, it has been noted that 1% to 5% of patients diagnosed with schizophrenia manifest movements similar to those now associated with TD (Kaplan & Sadock; Manschreck et al.). A substantial number of drug-naïve patients exhibit movement disorders that could be mistaken for EPS if drugs were started without a careful baseline assessment (Kopala). Difficulties may occur in distinguishing between symptoms of schizophrenia, medication side effects, and neuroleptic-induced dysphoria because of the similarities in symptomotology. For example, persons suffering from any of these conditions may present with unpleasant feelings such as sadness, anxiety, or irritability (Browne et al., 1998).

ABNORMAL INVOLUNTARY MOVEMENT SCALE (AIMS)

The criteria for probable TD includes a history of at least 3 months of continuous exposure to antipsychotic medications; the presence of at least moderate abnormal involuntary movements in one or more body areas or at least mild movements in two or more body areas; and the absence of other conditions that might produce involuntary movements (Germer et al., 1984). Routine periodic movement examinations with a tool such as the Abnormal Involuntary Movement Scale (AIMS) (Guy, 1976) are considered by many to be the most important step in prevention of TD. Such examinations have been recommended to aid in the early detection of TD and to help both clinicians and patients evaluate either the presence or the course of drug-induced movement disorders (Ahrens et al., 1988; Germer et al.; Herz et al., 2000; Jeste & Caligiuri, 1993; Kane et al., 1992; Munetz & Benjamin, 1990). The American Psychiatric Task Force report on tardive dyskinesia (Kane et al.), in its guidelines for the avoidance and management of TD, recommended regular examinations for early signs of choreoathetosis and oral-lingual dyskinesias. Ideally, examinations that use instruments such as the AIMS should be done before the institution of neuroleptic drug therapy and then repeated on a reg-

ular basis. *The Expert Consensus Guideline Series: Treatment of Schizophrenia* (American Psychiatric Association, 1999) suggested that patients receiving conventional antipsychotic medications should be monitored for TD every 4.3 months. If patients are on a newer atypical antipsychotic (Risperidone, Olanzapine, Quetiapine, or Ziprasidone), an AIMS-type examination should be performed every 6.5 months. For patients on clozapine, an atypical antipsychotic medication distinguished by its lack of EPS, *The Expert Consensus Guideline Series* (American Psychiatric Association) suggest monitoring patients who receive clozapine with the AIMS for TD every 8.7 months. *The Practice Guidelines for the Treatment of Patients with Schizophrenia* (Herz et al.) state that evaluations for TD should occur every 3 months, or more often, independent of the type of antipsychotic.

Many patients with motor EPS are unaware of having any abnormal movements; therefore it is important for clinicians to be constantly alert for EPS (Egan et al., 1997; Germer et al., 1984; Herz et al., 2000; Larsen & Gerlech, 1996). Routine monitoring of TD is essential to track symptomatic changes and response to medications (Ahrens et al., 1988; American Psychiatric Association, 1999; Awad et al., 1997; Boyd & Nihart, 1998; Egan et al.; Germer et al.; Kaplan & Sadock, 1998; Lane, Glazer, Hansen, Berman, & Kramer, 1985; Munetz & Benjamin, 1988, 1990; Munetz & Schultz, 1986). Adding a formal movement examination, such as the AIMS, to routine clinical observation provides a way to monitor the progress of treatment and increases the likelihood of detecting TD as it emerges (Germer et al.; Kane et al., 1992; Munetz & Benjamin, 1990). Using the AIMS to examine patients for abnormal movements on a regular basis, that is, upon admission and with every 3, 6, or 12 month follow-up, would prevent potential cases of TD from progressing unnoticed and would increase TD awareness among clinical staff (Ahrens et al.; American Psychiatric Association, 1999; Egan et al.; Germer et al.; Munetz & Benjamin, 1988, 1990). Awad et al. described the specificity of the AIMS scale as well as the rating scale used to determine the presence, or absence, of TD:

> The AIMS is a scale specifically designed to measure the presence and severity of abnormal movements in seven areas of the body: muscles of facial expression, lips and perioral area, jaw, tongue, upper extremities, lower extremities, and trunk. Ratings of tremor are specifically excluded. A 5-point rating scale is used to rate the movements in each body area, overall severity of abnormal movements, incapacitation because of abnormal movements, and patients' awareness and distress because of abnormal movements, yielding a total score. (p. 23)

The AIMS yields a possible total score of 0 to 40; a score of zero is considered diagnostic of no TD. As mentioned previously, seven body areas, severity of movements, incapacitation, and patient awareness of movements are assessed, for a total of 10 components. The rating scale is 0 to 4 (5 point), with 0 indicating none or normal and 4 indicating severe. The AIMS, then, is for assessment of the presence of movement disorders, specifically TD, with score levels being arbitrarily set by various researchers for the purpose of determining severity. The importance of the AIMS is that it offers a set of criteria by which to determine the presence of TD in individuals who are prescribed antipsychotic medications (Germer et al., 1984; Munetz & Benjamin, 1988).

Conducting the AIMS Exam

Because most involuntary movements increase with anxiety, decrease with relaxation, and disappear during sleep, the patient's level of arousal is important. Taking time to converse with the patient for the purpose of creating as relaxed an atmosphere as possible before conducting the assessment should help decrease anxiety and increase the sense of comfort. If the assessment is done on an inpatient unit rather than in a community setting, patients should not be observed during sleep (Munetz & Benjamin, 1988). Another consideration is the importance of unmasking involuntary movements. Armrests on chairs tend to mask involuntary movements, therefore, when performing the AIMS, the rater should ask the patient to sit in a chair that does not support the arms. During the informal phase of the AIMS assessment, the rater should make unobtrusive observations of the patient's movement, that is, the patient is observed without knowing that a movement evaluation is taking place. Patients can temporarily mask movements of TD by purposeful effort; therefore unobtrusively watching the patient as he or she walks into the office or waiting room or even during a routine clinical interview can assist the assessment process (Munetz & Benjamin, 1988). Upon completion of this informal aspect of the AIMS assessment, the questions and observations, adapted from Munetz and Benjamin (1988), outlined in Table 1, should be used. In this table the left column provides the list of actual test questions for the AIMS, and the right column provides the rationale.

IMPLICATIONS

Fleischhacker and Hummer (1997) posited that the first step in optimizing treatment of individuals with schizophrenia is an increase in the awareness and implementation of existing treatment standards. The Schizophrenia Patient Outcomes Research Team (PORT) treatment recommendations (Lehman &

Table 1. AIMS Procedure

Instruction	Reason for the Instruction
1. Ask the patient whether there is anything in his or her mouth, such as gum or candy. If there is, ask the patient to remove it.	1. The gum-chewing patient can be incorrectly assessed as having an oral dyskinesia. Conversely, the patient with constant chewing can be thought only to be chewing gum.
2. Ask the patient about the current condition of his or her teeth. Does the patient wear dentures? Ask whether teeth or dentures bother the patient now.	2. Ahrens et al. (1988) noted that pain in the mouth or poorly fitting dentures can result in oral movements that mimic TD. Munetz and Benjamin (1988) stated that dental status is important in considering the differential diagnosis of the patient who manifests oral dyskinesias alone. Edentulous patients may develop an oral dyskinesia, which is clinically indistinguishable from the buccolingual masticatory syndrome seen in TD. Patients with tooth or gum disease may develop stereotyped oral movements as well.
3. Ask whether the patient notices any movements in his or her mouth, face, hands, or feet. If the answer is yes, ask the patient to describe the movements and to what extent the movements currently bother the patient or interfere with activities.	3. Many patients who are aware of even subtle movements are distressed, while others deny feeling distressed. Notably, 25% to 50% of patients with TD deny awareness of abnormal movements. It can be clinically important (insight) to ask the patient who denies awareness of abnormal movements if anyone else (family or friends) has pointed it out.
4. Have the patient sit in a chair with hands on knees, legs slightly apart, and feet flat on floor. Look at the entire body for movements.	4. The inability to keep hands and feet still may be an early sign of chorea. The clinician needs to differentiate choreiform toe movements from the foot-tapping movements of akathisia or anxiety.
5. Ask the patient to sit with hands hanging unsupported. For a male patient ask him to let the hands hang between his legs. For a female patient ask her to lean forward, rest her arms on her lap, and let her hands hang loosely over her knees.	5. Look at the hands specifically for choreiform finger movements. Then look systematically at each of the seven body areas for choreoathetosis. Observe respiratory pattern, looking for abnormal diaphragmatic and abdominal movements. Grunting, gasping, and sighing can be signs of respiratory dyskinesias and can increase the patient's risk of aspiration.
6. Ask the patient to gently open his or her mouth to observe the tongue at rest within the mouth. Do this observation twice.	6. Look for vermiform movements (writhing, wormlike movements) of the tongue, which may be one of the early signs of TD. Patients with more severe tongue involvement may have twisting or darting movements (fly-catcher's tongue). If the tongue is pushing against the cheek, the patient has the appearance of chewing candy (bon-bon sign). Such a sign is difficult for the patient to disguise and is therefore useful to the observing clinician in determining signs of TD.
7. Ask the patient to protrude his or her tongue. Observe abnormalities of the tongue. Do this observation twice.	7. Look for vermiform or choreoathetoid tongue movements. If patient attempts to keep the tongue protruded but it darts in and out of the mouth, the patient may have a form of choreic tongue movement. If, however, the patient needs to be repeatedly reminded to stick out the tongue, they may have a form of motor impersistence unrelated to TD.
8. Ask the patient to tap his or her thumb with each finger, as rapidly as possible for 10 to 15 seconds, first with the fingers of the right hand, then with the left. Observe facial and leg movements.	8. Finger tapping is most likely to unmask oral, facial, and lower-extremity movements. During this maneuver the examiner should pay special attention to those body areas. Activating maneuvers, such as finger tapping, can elicit otherwise covert movements or increase the frequency and amplitude of overt involuntary movements.
9. Flex and extend patient's left and right arms, one at a time. Note any rigidity.	9. Parkinsonism and TD frequently co-exist. However, parkinsonian rigidity may partly or completely mask dyskinesias, so its presence needs to be noted. The steady muscular resistance of parkinsonism (cogwheeling) is to be differentiated from dystonia or spasticity.
10. Ask the patient to stand up, with arms at side. Observe the patient both straight on and in profile, noting all body areas again, including the hips.	10. The examiner may see truncal movements for the first time when the patient stands. The examiner may also notice the fore flexed posture of parkinsonism or the marching in place of akathisia, neither of which should contribute to the AIMS score.
11. Ask the patient to extend both arms out in front with palms down. Observe trunk, legs, and mouth.	11. This is a second activating maneuver. The major goal is to look for disinhibited truncal movements (squirming, twisting, or rocking), leg movements (including choreiform foot and toe movements), and oral movements and upper face, including the eyelids. May also observe choreiform finger movements.
12. Have the patient walk a few paces, turn, and walk back to the chair. Observe hands and gait. Repeat this observation twice.	12. The final activating maneuver, walking, is most helpful if the patient has enough space to walk comfortably and encouraged to walk normally. Choreiform or choreoathetoid finger, hand, and arm movements are clearly observed most often during the walking exercise as are oral dyskinesias and truncal movements and gait abnormalities.

Note. TD = Tardive dyskinesia; AIMS = Abnormal Involuntary Movement Scale.

Steinwachs, 1998) are one example of collaborative efforts to move toward "evidence-based" practice for schizophrenia and to identify both the strengths and limitations in the current knowledge base. Despite continued efforts to determine best practices, questions remain unanswered. For example, the Texas Medication Algorithm Project procedure manual (Miller, Chiles, Chiles, Crismon, & Podawiltz, 1999) reports that there is not enough evidence to recommend a switch from typical antipsychotic to atypical for the purposes of preventing TD. Nevertheless, other guidelines, such as the American Psychiatric Association's *Practice Guidelines for the Treatment of Patients with Schizophrenia* (Herz et al., 2000) and the *Expert Consensus Guideline Series: Treatment of Schizophrenia* (American Psychiatric Association, 1999), offer best practice guidelines for assessing for EPS side effects and TD and suggest strategies for selecting medications, services, and assessment issues.

Although improvements in antipsychotic medications have lowered the prevalence of TD, prevention remains paramount. It is important for clinicians and nurses to include the AIMS examination as a component of clinical observations (Herz et al., 2000; Munetz & Benjamin, 1990). Early recognition and treatment of TD can save the patient considerable discomfort, fear, and impairment (Egan, 1997) and potentially increase patient compliance. Thus it would be useful for all nurses, particularly psychiatric mental health nurses, to develop expertise in the ability to perform AIMS examinations.

Researchers note the likelihood of side effects that patients find bothersome but that are neglected by clinicians, to cause potential compliance problems (Herz et al., 2000; Larsen & Gerlach, 1996). Nurses who are aware of the symptoms of TD and other movement disorders may choose to develop expertise in such instruments as the AIMS. If symptoms of TD are observed (because of regular administration of the AIMS), notification of the prescriber and implementation of needed medication changes or other treatments can occur sooner (American Psychiatric Association, 1999). Appropriate medication changes have the potential to positively impact the clinical care of the persons being treated with antipsychotic medication, thus enhancing the possibility of patient compliance, a known component of preventing psychotic relapse in persons with schizophrenia (Egan et al, 1997; Herz et al.; Weiden, Scheifler, McEvoy, Frances, & Ross, 1999).

SUMMARY

Epidemiologic studies have uncovered a variety of risk factors that increase the chances of developing TD (Egan et al., 1997). These factors include dose of neuroleptic, duration of treatment, presence of drug-in-

duced EPS, age (higher rates in women over 65 and in young men), the presence of diabetes, organic brain damage, and psychiatric diagnosis (patients with mood disorders have increased risk, as do patients with schizophrenia) (Egan et al.; Herz et al., 2000; Kopala, 1996). Although it is hoped that the new generation of atypical neuroleptics will eliminate tardive dyskinesia, such a promise has yet to be fully realized (American Psychiatric Association, 1999; Kopala). Therefore, it is expected that many patients will continue to develop and suffer from TD and clinicians, whether nurse or physician, will continue to have to face the therapeutic conundrum of how to minimize the risk of TD and what to do with patients once they develop it (Egan et al.). Clinicians are able to effectively contribute to both preventative and palliative care of the patient receiving, or about to receive, antipsychotic medications by gaining familiarity with the AIMS and acquiring expertise through recommended application of AIMS as a regular component of patient assessment.

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