Assessment of drug-related movement disorders in schizophrenia

Maurice Gervin & Thomas R.E. Barnes

Conventional antipsychotic drugs remain one of the mainstays of treatment of schizophrenia and related psychotic disorders. The therapeutic efficacy of these drugs is well established, both for treatment of acute symptoms and in relapse prevention. Unfortunately, they are associated with a broad range of side-effects, the most prominent of which is the development of a variety of movement disorders (see Box 1). Compared with the conventional antipsychotic agents, the newer, atypical antipsychotics have a lower liability for the acute extrapyramidal side-effects (EPS) and, for a few of the new drugs, there is some evidence of a lower risk of tardive dyskinesia (Barnes & McPhillips, 1999). Nevertheless, even with these newer agents, movement disorders are seen in a significant proportion of patients.

The clinical management of patients receiving these drugs thus involves careful assessment to optimise the balance of potential benefits and risks. The motor disturbances are relatively common, can be disabling and distressing and may be a disincentive to compliance. Some of the motor phenomena can be misinterpreted as signs and symptoms of psychotic illness, thus confounding clinical assessment and decisions regarding adjustment of medication. The assessment of drug-related movement disorders in psychosis is further complicated by the occurrence of a range of motor disorders that are inherent in the psychotic illness. These range from a lack of coordination, simple tic-like movements and grimacing to chorea (Casey & Hansen, 1984).

A medication-related aetiology is assumed in the acute syndrome, which are temporally more closely related to drug administration and some of which respond quickly to anticholinergic medication.

Greater difficulty arises in attributing a medication-related aetiology to the chronic motor disorders, especially tardive dyskinesia. Aetiological explanations for this disorder refer to the consequences of chronic medication, the disease process of the psychotic illness and the effects of advancing age, or an interaction between these variables. Clinical investigation of these issues and of the relative contribution of each to the totality of tardive dyskinesia has been hampered by the lack of availability of control groups of non-medicated

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**Box 1 Extrapyramidal side-effects of antipsychotic drugs**

**Acute movement disorders**
- Parkinsonism
- Acute akathisia
- Acute dystonia

**Chronic movement disorders**
- Tardive dystonia
- Chronic akathisia
- Tardive dyskinesia

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patients with psychoses. The debate regarding aetiology and also the constraint placed on long-term treatment, which is partially medico-legal, has yielded a number of studies of selected groups of neuroleptic-naïve patients. These include retrospective case record reviews (Fenton et al., 1994) and studies of long-term institutionalised patients (Owens et al., 1982) and samples in community settings (McCreadie et al., 1996). While there are conflicting results, the rate of non-medication-related, or ‘spontaneous’, dyskinesia seems to be higher than previously thought. Attempting to attribute aetiological causes in an individual patient is difficult and may bias ratings of observed movements. Fortunately, most currently available rating scales for abnormal involuntary movements tend to avoid discrimination of aetiology.

The observable features of acute parkinsonism, such as limb stiffness, slowness of movement and mask-like faces, are a social and functional handicap. The same is true of the restless movements and agitation associated with acute akathisia. However, acute EPS can have less evident mental aspects: Parkinsonian patients report slowness of thinking or ‘feeling like a zombie’, while patients with akathisia describe inner restlessness and unease. These subjective phenomena are difficult to quantify, but some attempts have been made to study their relationship to the accompanying motor phenomena and their negative effects on quality of life, compliance and outcome (Hogan & Awad, 1992). While patients are often unaware of the movements in tardive dyskinesia and are usually not distressed by them, relatives often find them distressing. These movements can be very obvious to onlookers and can set patients apart socially, possibly adding to the stigma of severe psychiatric illness. An association between tardive dyskinesia and poorer quality of life has been reported (Browne et al., 1996).

Despite the difficulties in attribution of aetiology outlined above, the systematic and reliable assessment of drug-induced movement disorders is essential in both clinical and research settings. In treatment studies, it is necessary for the investigation of the side-effect profiles of antipsychotic drugs and of the relationships between motor disorders and other clinical variables. Clinically, rating scales may act as a diagnostic aid and allow more systematic monitoring of movement disorders during individual therapeutic trials.

This review describes the characteristic features of the drug-induced movement disorders, some of the problems in their assessment and the clinical utility of a few selected rating scales. Particular emphasis is placed on three of the most widely used and easily applicable scales and a combined examination procedure is described.

### Assessment

**Drug-induced parkinsonism**

The signs of drug-induced parkinsonism may develop within days of starting antipsychotic treatment; the literature records widely varying incidence figures of up to 40% in clinical practice. The condition resembles idiopathic Parkinson’s disease in its main symptoms, although some of the characteristic features may not be as common in the drug-induced condition (see Box 2).

On physical examination, rigidity of the limbs, which are resistant to passive movement, is perhaps the most obvious feature of drug-induced parkinsonism. It can occur as sustained resistance, described as ‘lead pipe’ rigidity, or as a succession of resistances that are rapidly overcome by passive movement, known as ‘cog-wheel’ rigidity. Milder forms of rigidity may be best detected on activation. The rigidity tends to become more obvious when the subject is engaged in moving the opposite limb, for example, tapping the knee with the opposite hand. The examination may be hampered by the subject’s inability to relax, or a tendency to move voluntarily with the examiner or resist passive movement. For these reasons it is better to carry out passive movements in an unpredictable way by varying the speed and order of movements around each joint.

**Rating scales for drug-induced parkinsonism**

A number of general scales have been developed for extrapyramidal symptoms which incorporate items

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**Box 2 Symptoms of idiopathic parkinsonism observed in the drug-induced condition**

**Symptoms commonly observed**
- Muscle rigidity
- Tremor
- Postural abnormalities
- Bradykinesia

**Symptoms less frequently observed**
- Festinant, hurrying gait
- 3–5 Hz resting tremor
- Reduction in the size of handwriting
- Rhythmic disturbance of handwriting, related to tremor
for rating drug-induced parkinsonism. These include Chouinard et al’s Extrapyramidal Rating Scale (Chouinard et al, 1980), the Targeting Abnormal Kinetic Effects (TAKE) scale (Wojcik et al, 1980), the Extrapyramidal Symptoms Scale (Adler et al, 1989) and the Neurological Rating Scale for Extrapyramidal Effects (Simpson–Angus scale; Simpson & Angus, 1970). The scales vary in the emphasis they place on the different features of drug-induced parkinsonism. The TAKE scale, for example, places more emphasis on akinesia and bradykinesia, while the Simpson–Angus scale places the majority of emphasis on rigidity. The choice of scale may vary according to the purposes of the clinician or investigator, but the Simpson–Angus scale, which was the first to be developed, remains the most widely used.

The Simpson–Angus scale was devised to measure drug-induced parkinsonism, providing standardised ratings for rigidity, tremor and salivation. The scale is entirely sign led. It contains 10 items, each rated on a 5-point scale (0–4), with descriptive anchors for each point and a clearly described examination procedure for each item. Six of the 10 items rate rigidity: arm dropping, shoulder shaking, elbow rigidity, wrist rigidity, leg pendulousness and neck rigidity. There is a single item for gait, which is the only measure for bradykinesia, and is in fact a compound item incorporating gait, posture and loss of arm swing. The other three items measure tremor, glabellar tap and salivation.

Simpson and Angus (1970) validated the their scale by demonstrating that it separated three groups of patients on different doses of antipsychotic medication. Interrater reliability was established by two examiners rating 14 patients on two occasions. Almost all items achieved acceptable levels of agreement, and the ratings of rigidity tended to show the highest correlation coefficients. Perhaps the main criticism of the scale is that it is not particularly comprehensive in assessing the full range of symptoms, overemphasising rigidity. Some authors view bradykinesia as the main feature of the condition and criticise the scale for only indirectly measuring this through the item on gait (Owens, 1999).

The TAKE scale rates many manifestations of bradykinesia, but few manifestations of rigidity. Bradykinesia is characterised by features such as paucity of gesture, a mask-like facies and slow monotonous speech. These features may be mistaken for retarded, or ‘akineti cs’, depression. There is also a potential overlap between these symptoms and negative features of schizophrenia such as affective flattening, poverty of speech and lack of drive (Barnes & McPhillips, 1995). Whether current assessment scales for negative features allow differentiation of affective flattening from these more subjective features of drug-induced parkinsonism is in some doubt. It may be that the concentration on more objective features, such as rigidity in the Simpson–Angus scale, confers an advantage of lack of measurement redundancy.

The examination method for assessing neck rigidity recommended in the Simpson–Angus scale is flawed both in terms of its utility in rating this item and of disrupting professional rapport with the patient. The procedure is that the patient “lies on a well padded table (if such a thing is to hand) and his head is raised by the examiner’s hand and then allowed to drop”. In normal subjects the head will fall onto the table, whereas in mild parkinsonism the movement is delayed and in severe cases it is absent. Apart from the fact that the procedure is only likely to work once in the same patient, the differential diagnosis might also include the ‘psychological pillow’ sign described in catatonic schizophrenia. Neck rigidity can be assessed just as accurately by holding the patient’s head and passively moving it around the neck joint, as outlined in the assessment method described in the Appendix.

The Simpson–Angus scale has only one item for tremor, relating to tremor of the fingers and arm or whole body tremor, but no items relating to titubation or tongue tremor. There is no mechanism for differentiating between mild Parkinsonian tremor and tremor with other causes (such as anxiety, lithium or benign essential tremor). While this may be seen as a disadvantage, it again can add to objectivity, as the examiner measures what is seen rather than changing the rating according to a presumed cause. The Simpson–Angus scale also lays emphasis on unobtrusive observation while the patient is walking into the room: this may exaggerate the score for tremor due to postural tremor, which is relatively common in psychiatric patients on medication.

Salivation is rated in a single item according to observation of pooling of saliva in the mouth, difficulty with speaking because of excess salivation or actual drooling. The mechanism of excess salivation in drug-induced parkinsonism is unknown, but one explanation is that saliva accumulates because the patient is swallowing less frequently. Excess salivation may occur for reasons other than parkinsonism. For example, it occurs in about a third of patients taking clozapine, which may lead to difficulties if this item artificially inflates the total score for parkinsonism in the Simpson–Angus scale. Analysis of results in studies involving clozapine may need to take this into account.

The conventional scoring system for the Simpson–Angus scale is to calculate a global score by summing the individual item scores and then dividing by the total number of items. Simpson and Angus considered that a final score of up to 0.3 was
“within the normal range”. This method of scoring is quite arbitrary and does not allow for separate monitoring of the features of rigidity, tremor and salivation.

**Acute and chronic akathisia**

The diagnosis and assessment of akathisia should take account of both its subjective (Box 3) and objective components.

The restless movements most commonly associated with the subjective experience of akathisia are not dyskinetic or stereotypical, but resemble normal patterns of restless movement. Most typical are lower-limb movements, such as rocking from foot to foot and walking on the spot when standing, and shuffling and trampling of the legs or swinging one leg on the other when sitting. Pacing rapidly up and down is a characteristic of severe akathisia, and in the worst cases, patients are unable to feel comfortable in any position, such as sitting, lying or standing, for more than a few minutes. In addition, trunk rocking and fidgety movements of the upper limbs may be seen. The severity of the movements in akathisia can vary according to the situation and the patient’s degree of arousal. For example, the movements may be less obvious during an interview or while concentrating on some mental task, and more evident when standing engaged in conversation on neutral topics (Barnes, 1992). Thus, limiting the assessment of akathisia to a brief, formal examination runs the risk of underestimating the presence and severity of the condition. For this reason, a period standing with the patient and engaging in undemanding conversation on neutral topics has been included in the examination instructions of some akathisia rating scales.

**Box 3 Subjective components of akathisia**

**Commonly experienced** (Halstead et al, 1994)
- Sense of inner restlessness
- Mental unease
- Unrest or dysphoria
- Feeling unable to keep still
- An irresistible urge to move the legs
- Mounting inner tension when required to stand still

**Less commonly experienced**
- Tension and discomfort in the limbs
- Parasthesiae and unpleasant pulling or drawing sensations in the muscles of the legs

Akathisia has most frequently been considered as a relatively common acute extrapyramidal problem, but it can also be a persistent problem in those receiving maintenance antipsychotic treatment (Barnes & Braude, 1985). There are no marked differences in the motor phenomena of acute and chronic akathisia, although it has been suggested that the accompanying subjective sense of restlessness may be less intense in the latter. Thus, as with acute akathisia, the motor features most commonly involve the lower limbs, and included marching in place and crossing and uncrossing the legs when sitting. Other movements include trunk rocking, respiratory grunting and complex hand movements, such as face rubbing and scratching and rubbing the thighs. In a relatively small number of individuals, repetitive, restless movements are observed that are characteristic of akathisia, but are not accompanied by any sense of inner restlessness or a compulsion to move. This presentation is referred to as pseudoakathisia. The condition seems to be more common in males and older patients with higher scores on negative symptoms and is likely to coexist with tardive dyskinesia (Barnes & Braude, 1985; Brown & White, 1991; Halstead et al, 1994).

**Rating scales for antipsychotic-induced akathisia**

For many years, akathisia was assessed by a single item within a more general scale for extrapyramidal symptoms. This tradition has been continued with the TAKE scale, which conceptualises akathisia as part of the Parkinsonian syndrome and rates it on the basis of subjective symptoms (Owens, 1999). More recently, scales specifically dedicated to the condition have been developed: the Rating Scale for Drug-Induced Akathisia (the Barnes scale; Barnes, 1989), the Hillside Akathisia Scale (Fleischhacker et al, 1989) and the Prince Henry Hospital Akathisia scale (Sachdev, 1994). All three scales include both subjective and objective items and a global item. The psychometric properties and clinical utility of these scales have been compared and contrasted in the literature (Barnes & Kane, 1994; Sachdev, 1995; Owens, 1999).

The Barnes scale is the most widely used, and both Sachdev (1995) and Owens (1999) consider that it has strong face validity, is simple and easy to use, with clear instructions for examination, and has well-defined, relevant anchor points. Unlike the other scales, the Barnes scale differentiates between the experience of restlessness and any associated distress. The global item score may be used as an overall severity measure and has a diagnostic threshold, with a score of 2 or more indicating the presence of akathisia.
Acute and tardive dystonia

Acute dystonic reactions are involuntary movements characterised by sustained muscle action (to the point of maximal contraction)(Box 4). Repetitive contorting, twisting movements are seen, which vary from fleeting disturbance to maintained abnormal postures. The incidence in acute psychiatric patients receiving conventional antipsychotics may be between 25 and 40% (Addonzio & Alexopoulos, 1988); the problem is more common in young adults and children. Owens (1999) delineates the subjective aspects of the condition, including prodromal symptoms of anxiety and a sense of something non-specific, but imminent. Awareness of motor symptoms, such as uncomfortable muscle stiffness and postural distortion, which can be painful, may make patients agitated and frightened. In clinical practice, the condition may well be missed in patients not progressing beyond the prodromal stage, or with only mild signs. Furthermore, the condition may be misdiagnosed as dissociative phenomena, malingering, seizures, tetany, posturing associated with psychosis, or an attempt to persuade doctors to prescribe anticholinergic medication.

The muscles of the head and neck are most commonly affected, and involvement of the laryngeal and pharyngeal muscles may lead to serious problems, such as respiratory distress and asphyxia, and dysphagia and choking.

Like akathisia, dystonia is generally viewed as an acute, relatively transient extrapyramidal side-effect. The occurrence of persistent, or tardive, dystonia as a distinct phenomenon in patients receiving long-term antipsychotic treatment has been accepted only in the past 20 years and its reported prevalence is about 1.5–4% (Raja, 1995). The motor presentations are similar to those seen in acute dystonia, and are clearly distinguishable from them only by their duration. The condition is apparently identical to idiopathic torsion dystonia or secondary dystonia associated with conditions such as Huntington’s disease or Wilson’s disease, and there is some overlap with the features of tardive dyskinesia, with which it may coexist (Barnes, 1990).

Rating scales for antipsychotic-induced dystonia

No rating scale has been devised specifically to assess antipsychotic-induced dystonia. This may be partly because acute dystonias are often transitory phenomena, of relatively rapid onset and rapidly responding to anticholinergic medication, and therefore not particularly suited to formal cross-sectional assessment. Also, until recently, such dystonias were perceived as relatively uncommon problems.

Burke et al (1985) have tested the reliability and validity of a scale for primary torsion dystonia called the Dystonia Movement Scale (or Fahn–Marsden Scale), which has been applied to drug-related disorder (van Harten et al, 1996). However, it is arguably appropriate only for cases of tardive dystonia that are severe and persistent.

Box 4 Common motor presentations of dystonia

Acute dystonia
- Torticallis (dystonic distortion of the neck)
- Trismus (contraction of the masticatory muscles)
- Blepharospasm (forceful eye closure)
- Dystonia of the trunk and limbs (less common)

Chronic (tardive) dystonia
- Blepharospasm
- Torticallis
- Retrocollis
- Oculogyric spasm

Tardive dyskinesia

Tardive dyskinesia is the main late-onset condition among the EPS. The diagnosis has been applied loosely to a range of involuntary movements (Box 5) occurring more commonly in older patients on antipsychotic medication (Barnes, 1990). While patients tend to be unaware of these movements, they can be quite distressing to relatives and may contribute to stigma and social handicap. The cause of these movements has been a subject of debate in the literature and a stimulus for continuing research. Explanations of the aetiopathogenesis of these movements include the effects of chronic medication, the disease process of the illness and effects of advancing age, or an interaction of these factors.

Perhaps the main difficulty in the assessment of tardive dyskinesia, especially orofacial, is that indistinguishable movements, termed spontaneous dyskinesia, are seen in 5–15% of elderly individuals who have never been medicated with antipsychotics (Kane et al, 1982). There is also recent evidence that these spontaneous movements occur in about 7% of antipsychotic-naive individuals with schizophrenia at onset of their illness (Gervin et al, 1998; Puri et al, 1999). Attempts to attribute a cause, such as spontaneous or tardive, while rating involuntary
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 Movements are problematic and may bias raters. In research studies it is better to rate what is seen and be blind to treatment status.

**Rating scales for tardive dyskinesia**

Of the instruments used to assess dyskinesia, the 12-item Abnormal Involuntary Movements Scale (AIMS; Guy, 1976) is the most popular. Several other scales are commonly used, such as the Tardive Dyskinesia Rating Scale (TDRS; Simpson et al, 1979) and Chouinard et al's (1980) Extrapyramidal Rating Scale. While generally referred to as a multi-item scale, the AIMS might best be characterised as a global impression scale, albeit with its items ordered regionally. The main advantage of these scales is that they provide a comprehensive rating of abnormal involuntary movements in various body sites. Many of them also provide a recommended examination procedure, standardising the clinical assessment and enabling comparison between different studies.

The AIMS assesses abnormal involuntary movements in three body regions: orofacial movements, rated on four separate items; extremity movements, on two separate items; and trunk movements, on one item. Each item is rated on a 5-point scale (0–4), with instructions to rate the highest severity observed and to score movements that occur upon activation one less than those observed spontaneously. Three separate items score global severity, the subject’s awareness, and incapacitation due to involuntary movements (each on a 5-point scale). Two additional items cover the subject’s dental status, as movements in the orofacial area are more obvious in edentulous patients.

There are several versions of guidelines for conducting the AIMS examination (Lane et al, 1985; Munetz & Benjamin, 1988). Gardos and Cole (1980) summarised the reliability of these multi-item scales as “variable”, but satisfactory levels of interrater reliability have been demonstrated for the more popular instruments, such as the AIMS (Smith et al, 1979; Lane et al, 1985) and the TDRS (Simpson et al, 1979). These scales take little time to complete and are applicable to most patients, making them particularly useful as screening instruments for tardive dyskinesia. Some of the problems with assessing tardive dyskinesia lie more in the variability of the disorder itself than in the scales. Individual patients may show a wide variability in site and severity of involuntary movements, related to adjustment of medication, anxiety, posture and mobility. There may also be spontaneous fluctuations from day to day or even at different times within one day. It has been suggested that repeated ratings should be carried out over time and, as far as possible, carried out at the same time of day by the same rater. Bergen et al (1984) used the AIMS to quantify spontaneous fluctuations in tardive dyskinesia and found that intrarater variability dominated intrapatient variability.

The AIMS, as with many of the other multi-item scales, is not a diagnostic instrument, but usefully quantifies involuntary movements, giving ratings that can then be used with diagnostic criteria. The use of threshold scores or diagnostic cut-off points can result in heterogeneous groups in terms of regional distribution of dyskinesia. The topographical distribution of involuntary movements may be a critical issue in the light of evidence suggesting that orofacial and limb–truncal dyskinesia are sub-syndromes of tardive dyskinesia that are pathophysiological distinct, with different clinical correlates and differential response to drug treatments (Barnes, 1990).

**Examination procedure for extrapyramidal side-effects**

The process of examining for dyskinetic movements requires methodical and close observation of specific body areas, while simultaneously scanning the body as a whole for movements occurring in other regions. It is important to differentiate the irregular, relatively stereotyped movements of dyskinesia from the regular rhythmic movements seen in tremor and the more purposeful, restless movements of akathisia.

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**Box 5 Common motor presentations of tardive dyskinesia**

**Orofacial dyskinesia (bucco-linguo-masticatory syndrome)**
- Protrusion or twisting of the tongue
- Smacking, pursing and sucking movements of the lips
- Puffing of the cheeks
- Lateral, chewing movements of the jaw
- Grimacing movements of the face

**Limb and trunk movements**
- Purposeless, jerky, stereotypical choreiform (choreo-athetoid) movement
- Athetosis of the extremities
- Limb and axial dystonias
- Abnormalities of gait
- Lordosis
- Rocking
- Shoulder shrugging
- Rotatory movements of the pelvis
Training in the examination of dyskinetic movements can be greatly aided by the use of videotaped assessments of patients, which allow repeated observation and discussion. A comprehensive examination procedure to facilitate the assessment of involuntary movements and other EPS has been described in detail by Owens (1999). Over a number of years of use, we have developed a similar, relatively brief, standardised style of examination (see Appendix). Adopting a systematic head-to-toe approach makes the routine easy to remember. Our procedure is largely based on the AIMS examination to rate tardive dyskinesia, but incorporates procedures that help identify other extrapyramidal symptoms.

Appendix

Examination procedure for assessing extrapyramidal syndromes in people with schizophrenia receiving antipsychotic medication

Either before or after completing the Examination Procedure unobtrusively observe the patient at rest (e.g. in the waiting room or while taking history) and observe the gait on entering or leaving the examination room.

The chair used in the examination should be firm, relatively high and without arms. It should be placed at a slight angle to the rater, rather than face on, to allow for more discreet observation.

Throughout the examination look out for the characteristic restless movements of akathisia.

A Examine the patient while you are both seated on chairs a sufficient distance apart to allow you to view all body areas. When examining specific body sites, continue to scan all areas.

1 Ask the patient if there is anything in his/her mouth (such as gum or a sweet) and if there is ask him/her to remove it.

2 Ask the patient about the current condition of his/her teeth. Ask if he/she wears dentures. Ask if teeth or dentures are causing discomfort.

3 Have the patient sit on the chair with hands on knees, legs slightly apart and feet flat on the floor. Look at entire body for movements in this position.

4 Ask the patient to sit with hands unsupported, hanging down by the side (opening and closing the fist once may help to relax the hands); then ask him/her to place the hands hanging over the knees. Observe hands and other body areas.

5 Asking the patient to remove shoes and socks can reveal subtle bradykinesia and allows clear observation of any subtle peripheral movements. However, such a request may be bothersome for some patients and jeopardise further cooperation; also, the procedure can be time consuming.

6 Ask the patient to open his/her mouth. Observe tongue at rest within the mouth. Do this twice.

7 Ask the patient to protrude the tongue. Observe abnormalities of tongue movement. Do this twice.

8 Ask the patient to tap his/her thumb with each finger, as rapidly as possible for 10–15 seconds; repeat for right and left hands. Tapping the fingers engages the patient in an activity that helps release involuntary movements elsewhere. Observe facial and leg movements as this is done.

For AIMS ratings, the convention is to rate movements that occur upon activation one less than those observed spontaneously.

B Approach the patient while he/she is still seated.

1 Perform the glabellar tap examination by standing behind the patient out of the line of vision and tapping gently but rapidly on his/her forehead (in the midline, between the eyebrows), asking the patient to try not to blink.

2 Examine the patient’s neck for rigidity by gently holding the head and flexing and extending the neck in coronal and sagittal planes and then gently rotating the neck.

3 Examine under the patient’s tongue for pooling of saliva. If involuntary tongue movements are observed during this, rate them on the AIMS one less than those observed spontaneously.

C Ask the patient to stand up while you are still standing beside him/her.

1 Examine the patient’s arms for rigidity by flexing and extending the elbow and the hand and rotating the shoulder.

D Ask the patient to sit on an examination couch or a desk so that his/her feet are not touching the floor.

1 Examine the patient’s legs for rigidity by first passively flexing and extending the knee while palpating the quadriceps for stiffness, then swinging the leg in a pendular motion and observing for freeness of swing.
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E With both you and the patient standing, move to a distance sufficient to allow you to view all body areas.

1 Ask the patient to turn around through 380° in four stages. View from the front, the side, the back, the other side and then face on again. Observe truncal involuntary movements while continuing to scan all areas.

2 Ask the patient to stretch both arms out in front of him/her and to spread the fingers. Observe tremor.

3 Ask the patient to extend both arms to the side, raising them to shoulder level and then allowing them to fall freely down to the sides again. Observe freeness of fall, audible contact and rebound. Do this twice.

4 To examine a patient’s natural gait it may be best unobtrusively to observe them walking to and from the examination room. Otherwise, ask the patient to walk casually along a nearby corridor and then back as if in a hurry. Observe for swinging of arms and Parkinsonian gait. Do this twice.

F While standing with the patient engage in conversation on neutral topics.

1 Observe for characteristic restless movements of akathisia.

2 Ask the patient if he/she notices any movements in the mouth, face, hands or feet. If yes, ask him/her to describe them and to what extent they usually bother the patient or interfere with activities.

3 Enquire about the patient’s subjective sense of restlessness:
   (a) non-specific inner restlessness;
   (b) awareness of a particular inability to keep the legs still, or a desire to move the legs, and/or inner restlessness aggravated specifically by being required to stand still (for example, when in a queue);
   (c) awareness of an intense compulsion to move most of the time and/or a strong desire to walk or pace most of the time.

If the patient reports any of these, determine to what extent this distresses him/her.

After the examination, rate the movements observed according to the anchors on each item of the AIMS. Other extrapyramidal side-effects such as parkinsonism and akathisia can be rated on appropriate scales.

References


### Multiple choice questions

1. The assessment of extrapyramidal side-effects may be enhanced by:

   a seating the patient in a relaxing, soft, low armchair

   b a period of unobtrusive observation

   c examining the patient’s gait as he/she takes two or three steps across the room

   d asking the patient to remove his/her shoes and socks

   e the use of standardised rating scales.

2. With reference to tardive dyskinesia:

   a it is characterised by an absence of distress associated with the orofacial movements

   b similar ‘spontaneous’ movements are seen in a small proportion of neuroleptic-naïve people with early schizophrenia

   c in individual patients, the nature and severity of the dyskinetic movements tend not to fluctuate over time

   d the Abnormal Involuntary Movements Scale (AIMS) is the most commonly used rating scale for it in clinical studies

   e manifestations of orofacial dyskinesia include the ‘bon-bon’ sign and ‘fly-catcher’ tongue movements.

3. With reference to antipsychotic-induced parkinsonism:

   a the Neurological Rating Scale for Extrapyramidal Effects is suitable for rating subtle signs of bradykinesia

   b existing rating scales allow clear differentiation between drug-induced parkinsonism and the negative symptoms of schizophrenia

   c a handwriting sample may be useful in the assessment of drug-induced parkinsonism

   d muscle rigidity in the arm can be reduced by asking the subject to move the other arm

   e sialorrhoea is probably a consequence of less frequent swallowing related to bradykinesia.

4. The following may be said about antipsychotic-induced akathisia:

   a it is primarily a subjective phenomenon, characterised by inner restlessness and unease

   b the Barnes Scale can be used to rate pseudoakathisia

   c the diagnosis of akathisia cannot be made in the absence of observable motor restlessness

   d the subjective sense of restlessness is reliably relieved by lying down and resting

   e acute akathisia is a transient problem that rarely persists longer than a few days.

5. With reference to antipsychotic-induced dystonia:

   a acute dystonia may be mistaken for drug seeking behaviour
b acute dystonia does not usually have subjective phenomena

c rating scales such as the Dystonia Movement Scale are very useful for detecting acute dystonia

d blepharospasm and torticollis are relatively common presentations of tardive dystonia

e the movements seen in tardive dystonia are quite distinct from those seen in idiopathic torsion dystonia.

MCQ answers

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Commentary

D. G. C. Owens

Listen for the sighs – not another overview of drug-related motor disorders. One is often left with the uncomfortable feeling that much of mainstream psychiatry sees this as one topic they do ‘know’ about. Furthermore, with new-generation antipsychotics advancing apace, it relates to yesterday’s problems and redundant skills.

From my own perspective as a ‘teacher’, it appears to me that not only are many clinicians still unclear about the nature and extent of the problem; they also often feel unprepared to give a confident and reliable evaluation of the wide range of clinical phenomena comprising these disorders, their complex manifestations and uncertain boundaries.

And what of these new drugs? Gervin and Barnes (2000) comment that they “have a lower liability for ... acute extrapyramidal side-effects (EPS)” and that while the published evidence does show this fact, controversy remains about its interpretation. Does this mean that the new antipsychotics possess an inherent and pharmacologically mediated advantage? Or, relative to comparator drugs and regimes, do they possess an apparent and practice-mediated benefit? And regardless of the literature, does the fact that patients may prefer these new treatments ‘prove’ that they are indeed all they are claimed to be, or could it mean that we, as a profession, have never been as skilled as we thought in the use of the old drugs? The problems remain very much of today.

Gervin and Barnes’ overview, with its emphasis on assessment, is therefore welcome – particularly in a journal devoted to ‘advances’. Drug-related adverse effects would seem ideally suited to standardised monitoring as an outcome or quality-of-care measure, and evaluation and accessible recording of extrapyramidal status could become a required core skill.

It is pleasing to see the authors commenting on the subjective component of extrapyramidal dysfunction. It has always seemed ironical that while neurology has paid a deal of attention to the mental state features of parkinsonism (Cummings, 1992; Levy et al, 1998; Aarsland et al, 1999), psychiatry has been content to view drug-related disorder as almost exclusively sign led. Over the past 20 years, such narrow vision has probably been responsible...
for an exponentially expanding literature on ‘negative’ states that has produced a proportionately diminishing understanding. Gervin and Barnes point to the difficulty in quantifying disorder of this type, to which one might add the difficulty of reliable identification. None the less, it is sufficient for clinicians to be aware of the issue and to be ready to incorporate the subjective component of EPS into all relevant differentials.

The authors’ statement that “rigidity of the limbs ... is perhaps the most obvious feature of drug-induced parkinsonism” is not immune to challenge. Rigidity in antipsychotic-treated patients is usually mild, as the authors acknowledge with their comment on the useful role of activation. It rarely, if ever, reaches the levels of severity – and hence, certainty – found in patients with idiopathic extrapyramidal disease. It is also a uniquely difficult sign to rate confidently in those whose engagement in the examination process is tenuous.

As in idiopathic Parkinson’s disease, in which bradykinesia is the ‘core’ core disorder and necessary for the diagnosis (Quinn, 1995), bradykinesia is pervasive in drug-related disorder. The fact that it may not be obvious to psychiatrists may reflect a tendency, nurtured by training, to misattribute neurological signs to mental state disorder. One can commend bradykinesia, in all its manifestations, as ‘the most obvious feature’ of drug-related parkinsonism.

As Gervin and Barnes note, dystonia is not readily rateable on standard instruments. Chouinard et al’s (1980) Extrapyramidal Symptom Rating Scale is one of the few scales to attempt separate ratings of dystonia, both acute and chronic. The reasons why this is laudable, if on this occasion unsuccessful, have been expounded elsewhere (Owens, 1999). This scale does, however, provide a sound means of rating parkinsonism.

The disadvantages of the professional divide between neurology and psychiatry over the past century is evident in rating scales for parkinsonism. Neurology produced over a dozen (Owens, 1999), all aimed at measuring treatment response, initially to surgery and later L-dopa. All, however, had the same problem – they were not much good (Diamond & Markham, 1983). This led to the development of the Unified Parkinson’s Disease Rating Scale (Fahn et al, 1987), now the international ‘gold standard’ in the field.

Psychiatry has been excluded from these developments. Unlike neurology, which acknowledged a problem and sought to address it, psychiatry remains stubbornly wedded to its own. The Neurological Rating Scale for Extrapyramidal Side-Effects of Simpson and Angus (1970) was first in the field for specifically drug-related parkinsonism.

But first is not necessarily best, and the persistence and prominence of this scale in any of its numerous incarnations are both surprising and frustrating. Gervin and Barnes present some criticisms, and there are others (Owens, 1999). In my opinion, the continued use of this scale, especially at the behest of licensing authorities, does our profession no favours.

Most recording instruments for tardive dyskinesia emerged from psychiatry. Multi-item scales, such as the Tardive Dyskinesia Rating Scale (Simpson et al, 1979), can provide detailed typologies, but are complex and force recording of data in an unphysiological fashion – e.g. when evaluating ‘tongue protrusion’, should one also rate mouth opening, an obviously necessary procedure in protruding the tongue? However, mouth opening in this situation is totally different from the imposed contortions of primary dystonia affecting jaw musculature. Global impression scales, regionally ordered (such as the AIMS, see below), are not free from this problem, but are less afflicted by it.

All the standardised scales referred to by Gervin and Barnes were devised to serve the needs of research, and whether they automatically transfer to routine practice has never been evaluated. It is likely, however, that at least some can be usefully applied in that context as well. It is important to remember that none of these methodologies is, as is often stated, objective. All are rater subjective – a distinctly different principle. Thus, all the biases relevant to patients apply to raters too. These can probably never be eradicated completely, but they can be mitigated by two prerequisites essential to their introduction – teaching and training. The first ensures that all those involved in standardised data recording are aware of, and agreed on, what precisely comprises the phenomenology to be rated. The second establishes reliabilities with the chosen instrument. Both are particularly important if multi-disciplinary rating is envisaged. Undertaking this method of data acquisition and recording without them is foolish, and it will produce a veneer of competence on a core of misleading information. Audit managers, please note!

We cannot dictate which scale(s) to choose for routine use but, as Gervin and Barnes’ proposal highlights, patient evaluations must be comprehensive, covering all the syndromal features of antipsychotic-related motor dysfunction. The Abnormal Involuntary Movement Scale (AIMS; Guy, 1976) is probably rightly the ‘market leader’ hyperkinetic rating scale for the research community and could easily fulfil a similar function in routine practice. I find its ‘one less’ rule, which Gervin and Barnes appear to accept, illogical and have argued
for its suspension (Owens, 1999). The choices for parkinsonism are less clear (especially if one excludes the Simpson–Angus scale), but a combination of AIMS and the Targeting Abnormal Kinetic Effects scale (Wojcik et al, 1980) forms a useful and undemanding package.

At a practical level, regular rating must take account of context – both ‘simultaneous’ (disorder in one area influencing rating in another), and ‘prior’ (impressions of previous individual or group disorders unduly influencing perception of present abnormality). Like most scales used in psychiatry, the majority of those for motor disorder favour a severity continuum along an uneven number of anchor points, leaving raters open to the ‘central tendency’ effect, that is to say, unwittingly rating down the middle. And finally, one must consider beforehand what is to be done with the information, in particular whether it is to be subjected to statistical evaluation, which itself presents a series of unresolved problems (Owens, 1999).

Standardised examination has much to commend it and is predicated on the simple principles of structure and practice. Successful standardised recording rests on teaching and training. Thus, the introduction of standardised methodologies into routine practice requires upfront investment and will have resource implications.

While standardised methods can be commended for routine use, they are not without problems of their own, and in recommending them awareness of their weaknesses is perhaps a greater asset than knowledge of their strengths.

References


Assessment of drug-related movement disorders in schizophrenia
Maurice Gervin and Thomas R.E. Barnes
APT 2000, 6:332-341.
Access the most recent version at DOI: 10.1192/apt.6.5.332

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