The Causes of Underdiagnosing Akathisia

by Shigehiro Hirose

Abstract

This article reviews what causes clinicians to overlook or underdiagnose akathisia. The causes are considered to be related to both the patient's symptoms and the clinician's attitude toward akathisia. The patient factors include mild severity of akathisia, lack of apparent motor restlessness, no voluntary expression of inner restlessness, no clear communication of inner restlessness, restlessness in body parts other than the legs, atypical expressions of inner restlessness, other prominent psychic symptoms, and absence of other extrapyramidal signs. The clinician factors include emphasis on objective restlessness, failure to consider akathisia during antipsychotic therapy, failure to fully implement antiakathisia treatments in ambiguous cases, and strict adherence to research diagnostic criteria. Akathisia is likely to be overlooked or underdiagnosed when both patient and clinician factors are present. Currently, there may be two major problems with underdiagnosis: (1) symptoms that fulfill the diagnostic criteria for akathisia are overlooked, and (2) conditions that do not fulfill the diagnostic criteria but can still benefit from antiakathisia measures are underdiagnosed.

Keywords: Antipsychotic-induced akathisia, overlooking, underdiagnosis, undertreatment, diagnostic threshold.

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Antipsychotic-induced acute akathisia is a distressing side effect of antipsychotic medication for patients (Kalinowsky 1958; Editorial 1986; Blaisdell 1994). It has been reported that there is a high incidence of overlooking akathisia (Weiden et al. 1987), and even experienced psychiatrists can make errors in the diagnosis of akathisia (Sachdev 1995*a*). One of the causes for the wide discrepancy in the reported incidence of akathisia is believed to be underdiagnosis (Van Putten and Marder 1987; Weiden et al. 1987; Adler et al. 1989). There is a significant difference between studies in the incidence of risperidone-induced akathisia (6.7% to 50%) (Murasaki et al. 1993; Ceskova and Svestka 1996; Kopala et al. 1997; Tran et al. 1997; Miller et al. 1998; Moller et al. 1998; Rosebush and Mazurek 1999; Wirshing et al. 1999).

Clinicians today may be less focused on akathisia because of the increasing use of newer or atypical antipsychotics that are less likely to produce neurological side effects. Nonetheless, atypical antipsychotics can produce akathisia, although the incidence may be lower than that of conventional antipsychotics (Henderson and Goff 1996; Miller et al. 1998; Wirshing et al. 1999): risperidone, 6.7 to 50 percent; olanzapine; 2.8 to 16 percent (Beasley et al. 1996; Tollefson et al. 1997; Tran et al. 1997; Gomez et al. 2000; Ishigooka et al. 2001); quetiapine, 2 to 5 percent (Arvanitis and Miller 1997; Small et al. 1997; McManus et al. 1999; Copolov et al. 2000; Emsley et al. 2000); clozapine, 0 to 39 percent (Claghorn et al. 1987; Cohen et al. 1991; Safferman et al. 1992; Miller et al. 1998); and haloperidol, 5.9 to 39 percent (Murasaki et al. 1993; Beasley et al. 1996; Tollefson et al. 1997; Rosebush and Mazurek 1999; Gomez et al. 2000; Ishigooka et al. 2001). However, the lower incidence may increase the rate of akathisia overlooking, as clinicians will be less likely to consider akathisia as a possible side effect. The rate of akathisia overlooking in clinical practice today is not known. The problem is that overlooking in itself is usually undetected by the clinician who overlooks. Consequently, the prevalence of overlooking is underestimated.

Trouble with overlooked akathisia has often been reported following the introduction of antipsychotic therapy (Kalinowsky 1958; Raskin 1972; Van Putten 1975; Shen 1981; Ratey and Salzman 1984; Siris 1985; Drake and Sederer 1986; Weiden et al. 1987; Kaplan and Sadock 1995; Barnes and McPhillips 1999). Overlooked or under-

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diagnosed akathisia, which can lead to either continued antipsychotic treatment or elevation of the dose (Shen 1981; Ratey and Salzman 1984; Siris 1985; Editorial 1986; Szabadi 1987; Ayd 1992), prolongs or worsens the distress in the patient. Akathisia can be severe enough that some patients want to die to escape the unrelenting distress (Shear et al. 1983; Drake and Ehrlich 1985; Sachdev and Loneragan 1992; Hirose 2000). In addition, akathisia can lead to treatment noncompliance, ultimately producing relapse of the primary illness (Van Putten 1974).

Therefore, measures to prevent the overlooking or underdiagnosing of akathisia are needed. However, a review focused on this issue has not been published. To avoid the overlooking and subsequent undertreating of akathisia, the causes need to be examined. This article, based on the literature, reviews what causes akathisia overlooking or underdiagnosing from the point of view of the patient's condition as well as the clinician's attitude toward akathisia and discusses measures to prevent the undertreatment of akathisia in clinical practice.

Within current rating scales (Chouinard et al. 1980; Braude et al. 1983; Barnes 1989; Fleischhacker et al. 1989; Sachdev 1994*a*) and research diagnostic criteria in *DSM–IV* (APA 1994), both objective and subjective components of akathisia have to occur concomitantly in order for a diagnosis of akathisia to be made. These include (1) subjective inner restlessness, particularly referable to the legs and (2) objective motor restlessness appearing primarily as restless leg movements, which occur following initiation of antipsychotic therapy, increase in the dosage of the antipsychotic, switching of the patient to a high-potency antipsychotic, or reduction or withdrawal of concomitant antiakathisia agents (APA 1994). Consequently, it has been commonly considered that patients are diagnosed as having akathisia only if they manifest both components. However, it has been reported that there is considerable variation in symptoms between patients with akathisia. Some of these are not described in the various rating scales or do not fulfill the research diagnostic criteria for akathisia.

Patient Conditions That May Contribute to Underdiagnosis

Mild Severity of Akathisia. The incidence and degree of akathisia with antipsychotics have been reported to increase in proportion to the dosage (McClelland et al. 1976; Braude et al. 1983; Van Putten et al. 1990), rate of dose escalation (Miller et al. 1997), and potency (Ayd 1961, 1983; Van Putten et al. 1984; Miller et al. 1998). The patient is often given oral anticholinergics and benzodiazepines concurrently in clinical practice to treat or prevent parkinsonism and insomnia, respectively. However, both agents may ameliorate but not completely eliminate akathisia (Braude et al. 1983; Sachdev and Loneragan 1991). Consequently, the severity of akathisia induced by a low dose of an antipsychotic or a lower potency antipsychotic with these concomitant drugs may be mild. In addition, the intensity of akathisia fluctuates over the course of a day (Gibb and Lees 1986; Sachdev 1995*a*) and can be altered by the patients' position—whether sitting on a chair, lying, or standing (Braude et al. 1983; Barnes and Braude 1985; Sachdev and Kruk 1994). Thus, the intensity can be mild at times.

When the degree of akathisia is mild, the typical symptoms of akathisia can be less apparent than in severe akathisia (Editorial 1986; Van Putten and Marder 1986; Lang 1988). For example, typical motor restlessness in severe akathisia is manifested as rocking from foot to foot while standing, swinging the legs, pacing, and being unable to be still for a short period of time (Barnes and Braude 1986; Van Putten and Marder 1986), whereas patients with mild akathisia do not manifest such motor restlessness (Van Putten 1975; Editorial 1986; Van Putten and Marder 1986; Lang 1988).

Lack of Apparent Motor Restlessness. A patient will not present with motor restlessness if akinesia is present (Van Putten and Marder 1986; Tuisku et al. 2000). Furthermore, akinesia is often overlooked in clinical practice (Van Putten and Marder 1987; Weiden et al. 1987). Patients with motor restlessness can voluntarily stay still for a certain period of time (Ratey and Salzman 1984; Barnes 1989; Sachdev 1994*b*), such as during a consultation. Consequently, a lack of apparent motor restlessness in the consulting room may be a common feature of akathisia. In addition, patients who manifest motor restlessness at times other than consultation and their family members often do not voluntarily report this to the clinician.

No Voluntary Expression of Inner Restlessness. Patients with akathisia can usually acknowledge their inner restlessness once the term "inner restlessness" is suggested to them and thereafter can express the term (Van Putten 1975; Munetz and Cones 1983; Hirose 2000). However, initially, most akathisia patients do not voluntarily express feelings of subjective restlessness to clinicians, and some never express it unless they are questioned (Hirose 2000), perhaps because they are not skilled in verbalizing their inner experiences and do not know which symptoms to disclose. A clinician may not ask the patient about inner restlessness if motor restlessness is absent at the time of consultation. Indeed, a wide difference in the incidence between clinician-solicited akathisia

and spontaneously reported akathisia has been reported with both atypical and conventional antipsychotics (Tollefson et al. 1997).

No Clear Communication of Inner Restlessness. Patients with severe excitement, severe mental retardation (Kumar 1979; Ratey and Salzman 1984; Sachdev 1995*b*), dementia (Lang 1988), or brain damage (Ratey and Salzman 1984) usually do not communicate their subjective inner feelings to the clinician. Thus, the clinician cannot identify the subjective restless sensation in the patient. In such cases, observable restless movements are referred to if the clinician suspects akathisia in the patient. However, restless movements may not be attributed to akathisia but to the excited behavioral state of the illness itself.

Restlessness in Body Parts Other Than the Legs. Restlessness in the legs has been considered a common sign highly characteristic of akathisia (Van Putten and Marder 1987; Adler et al. 1989; Barnes 1989; APA 1994; Sachdev 1994*a*). Therefore, restlessness referable to the legs has been considered a pathognomonic sign of akathisia, distinguishing it from restlessness due to other etiologies (Table 1) (Braude et al. 1983). Consequently, the research diagnostic criteria in *DSM–IV* and akathisia rating scales stipulate restlessness in the legs as requisite for akathisia.

However, Gibb (Gibb and Lees 1986) and Sachdev (Sachdev and Kruk 1994) found leg restlessness in only 27 percent and 55 percent of akathisia cases, respectively. It has been reported that manifestations of restlessness in akathisia can occur to varying degrees in body parts other than the legs, including the head or neck (Gibb and Lees 1986; Hirose 2001), chest (Burke et al. 1989; Hirose 2000), abdomen (Ratey and Salzman 1984), and arms (Barnes and Braude 1985; Walters et al. 1989; Sachdev and Kruk 1994). It has also been reported to originate psychically (Gibb and Lees 1986; Sachdev and Kruk 1994). Consequently, akathisia can be overlooked if only restlessness in the legs is considered.

Atypical Expressions of Inner Restlessness. Despite expression of their inner feelings, patients' subjective experience of inner restlessness may not be spontaneously articulated by the typical expression of "I feel restlessness in the legs" but expressed in a variant form. The most common variant expression of inner restlessness of akathisia is anxiety (Kendler 1976; Van Putten et al. 1984; Gibb and Lees 1986; Adler et al. 1989; Lipinski et al. 1989; Casey 1993; Tonda and Guthrie 1994; Casey 1995; Holloman and Marder 1997; Collaborative Working Group 1998; Hirose 2000). This expression can be easily misinterpreted as being related to the primary illness itself, for instance, schizophrenia (e.g., delusional mood), bipolar disorder, anxiety disorder, or personality disorder.

Agitation or anxiety was commonly reported as a side effect of antipsychotics, e.g., risperidone (Borison et al. 1992; Chouinard et al. 1993; Murasaki et al. 1993; Marder and Meibach 1994; Owens 1996; Moller et al. 1998; Dawkins et al. 1999), quetiapine (Borison et al. 1996; Arvanitis and Miller 1997; Small et al. 1997; Copolov et al. 2000), and olanzapine (Beasley et al. 1996; Tran et al. 1997; Ishigooka et al. 2001). Although the reported agitation and anxiety were classified as psychic side effects of antipsychotics (Hoyberg et al. 1993; Peuskens 1995) or regarded as being related to the original illness (Borison et al. 1996; Arvanitis and Miller 1997; Small et al. 1997), it has been suggested that they are subtle manifestations of akathisia (Kalinowsky 1958; Hirose 2000).

Inner restlessness may also be expressed as impatience, apprehension, dysphoria, irritation, anger or rage, tension, confusion, fear, vague somatic complaints, and dyspnea (Van Putten et al. 1974; Van Putten and Marder 1987; Halstead et al. 1994; Hirose 2000). For example, a subtle manifestation of inner restlessness is the inability to watch a television program without continually changing the channel or the inability to read a book (Lang 1988; Sachdev 1995*a*). These forms of inner restlessness are also expressed as "difficulty in concentrating" (Murray et al. 1977; Anderson et al. 1981). Most patients that have such an atypical form of inner restlessness usually acknowledge this once the term "inner restlessness" is suggested by the clinician (Van Putten 1975; Hirose 2000). Incidentally, difficulty in concentrating was also reported as a psychic side effect of atypical and conventional antipsychotics (Claus et al. 1992; Hoyberg et al. 1993; Peuskens 1995; Daniel et al. 1996), where a possibility of underdiagnosed akathisia was not ruled out.

Other Prominent Psychic Symptoms. Some patients with akathisia manifest prominent psychic symptoms different from those of akathisia, while their akathisia symptoms are not noticeable. Exacerbation of hallucinations or delusions (Van Putten et al. 1974; Van Putten 1975), anger to the point of violence (Keckich 1978), manic activity (Kumar 1979; Lipinski et al. 1984), disruptive behavior (Shen 1981), panic attack (Anderson et al. 1981; Maltbie and Cavenar 1997; Hirose 2000), acting out (Siris 1985), suicidal attempt (Shear et al. 1983; Drake and Ehrlich 1985) or suicidal ideation (Schulte 1985; Shaw et al. 1986; Teicher et al. 1990; Hamilton and Opler 1992; Wirshing et al. 1992), and depression (Kalinowsky 1958; Lipinski et al. 1984; Ratey and Salzman 1984; Van Putten et al. 1984) have been reported as manifestations of akathisia. Seductiveness and public masturbation (Siris 1985), sexual craving (Van Putten

Table 1. Differences between antipsychotic-induced akathisia and restless legs syndrome[1]

Feature	Antipsychotic-induced akathisia	Restless legs syndrome
Current or past exposure to antipsychotics	Present	Absent
Parts of body involved	Predominantly the legs, but involvement of other parts of the body is not uncommon	Restricted to legs in most cases
Positive sensory symptoms in calves, e.g., pins and needles sensation, creeping and crawling, aching, burning, or coldness	Absent	May be present
Myoclonic jerks	Rare	Usual
Sleep disturbance	Rare	Usual
Effect of movements	Some relief with movements of affected parts and worse when being still	Worse when legs at rest
Diurnal variation	Occasional fluctuation in intensity	Symptoms worse or exclusively in evenings
Familial occurrence	Unknown	Present
Effective treatment	Withdrawal or reducing dose of causative drugs	Administration of benzodiazepines, L-dopa, and bromocriptine
	Administration of anticholinergics, beta-antagonists, and benzodiazepines	
	Change to antipsychotic agent with decreased liability to akathisia	

[1] Modified from Sachdev and Loneragan 1991 and Sachdev 1995*a*.

1975), and self-destructive behaviors such as head banging (Van Putten 1975; Hirose 2000) have also been reported. These symptoms have been considered to be caused or exacerbated by akathisia induced by the pharmacotherapy of the primary illness (Van Putten et al. 1974; Duncan et al. 2000). Usually, subjective inner restlessness and objective motor restlessness may be identified even in the above cases if the patients are carefully examined and the prominent symptoms can be eliminated by antiakathisia treatment (Van Putten 1975; Hirose 2000). Generally, the patients are cooperative during the examination and accept the treatment by clinician as they perceive the distress of akathisia as alien to themselves (Van Putten 1975; Hirose 2000).

However, the aforementioned symptoms can be common in the acute phase or postpsychotic depression of schizophrenia, manic or depressive episodes in mood disorders, and impulsivity or inappropriate emotions in Cluster B personality disorders in *DSM–IV* or mental retardation. These symptoms can be attributed to the original illness instead of akathisia, leading to inappropriate treatment. Thus, these symptoms could mask akathisia and prevent the clinician from considering akathisia.

Furthermore, if the patient is given drugs other than antipsychotics that induce akathisia, such as amoxapine (Ross et al. 1983), selective serotonin reuptake inhibitors (Lipinski et al. 1989), tricyclics, and so on (Sachdev 1995*b*), the possibility of akathisia may be less likely to be considered by the clinician, as the incidence of akathisia with these agents is generally considered to be low (Sachdev 1995*b*).

Absence of Other Extrapyramidal Signs. Some studies have found that akathisia is commonly accompanied by signs of parkinsonism, such as tremor, rigidity, or akinesia (Van Putten 1975; Braude et al. 1983; Sandyk and Kay 1990; Sachdev 1995*b*). Consequently, it has been thought that the appearance of parkinsonism following antipsychotic medication might serve as a predictor for the development of acute akathisia (Sachdev and Kruk 1994). However, the occurrence of akathisia in the absence of parkinsonism is also common (Lang 1988; Sachdev 1995*a*; Van Harten et al. 1997; Brune 1999). Therefore, the preconception that parkinsonism accompanies akathisia may cause the clinician to fail to consider the possibility of akathisia when parkinsonism is absent in the patient.

Clinician Attitudes That May Contribute to Underdiagnosis

Emphasis on Objective Restlessness. Akathisia was originally considered to be fundamentally a subjective condition (Munetz and Cones 1983; Lang 1988; Casey 1995). Indeed, the majority of akathisia patients report that restless movements are voluntary, a response to perceived inner restlessness (Sachdev 1995*a*). However, there has been disagreement about whether subjective inner restlessness or objective motor restlessness is the primary feature of akathisia (Van Putten 1975; Kendler 1976; Barnes and Braude 1985; Stahl 1985; Editorial 1986; Sachdev 1994*b*). Motor restlessness can be objectively evaluated by the clinician, while subjective restlessness cannot, so clinicians tend to favor motor restlessness (Hirose 2000). In addition, subjective restlessness can be considered a nonspecific symptom in psychiatric patients (Sachdev and Kruk 1994). Although restless movements also may represent nonspecific symptoms in psychiatric disorders (Gibb and Lees 1986), rocking from foot to foot or pacing is considered specific to the motor restlessness of akathisia (Van Putten and Marder 1987; Adler et al. 1989; Barnes 1989; APA 1994; Sachdev 1994*a*)—another reason why motor restlessness has been emphasized in diagnosing akathisia. Indeed, motor restlessness is a reliable indicator of severe akathisia. Originally, however, rocking from foot to foot or pacing was considered unique in only cases of moderate to severe akathisia (Van Putten and Marder 1986). Nevertheless, these signs may have been overemphasized in diagnosing not only severe akathisia in patients who cannot communicate verbally but mild akathisia in patients who can communicate their inner restlessness but do not have apparent motor restlessness.

Authors stressing objective restlessness have reported a lower incidence of akathisia than have those stressing subjective restlessness (Gibb and Lees 1986; Adler et al. 1989; Miller and Fleischhacker 2000). This suggests that objective restlessness is less sensitive than subjective restlessness in detecting a broader range of akathisia. In fact, objective motor restlessness is often less obvious or is absent, as mentioned above. Furthermore, even in severe akathisia, a patient can voluntarily remain still for a certain time (Ratey and Salzman 1984; Barnes 1989) when good manners are expected. This suggests that the emergence of motor restlessness depends on the patient's capacity for patience, which could decrease the objectivity of motor restlessness as a reliable sign. Videotaping during relaxed times could compensate for this problem (Barnes and Braude 1986; Sachdev 1995*a*) in some patients, but not those with akinesia and milder akathisia. Beyond that, videotaping is impractical in the clinical setting.

Thus, placing primary emphasis on the presence of objective signs may lead to underdiagnosis of akathisia. Although akathisia with no apparent motor restlessness may be mistakenly judged by the clinician to be of no distress to the patient, the patient's subjective distress is usu-

ally severe enough to be unendurable (Van Putten 1975; Van Putten and May 1978). I recommend stressing motor restlessness only when patients cannot fully communicate their inner feelings to the clinician and their akathisia is severe enough to manifest motor restlessness.

Failure to Consider Akathisia During Antipsychotic Therapy. To avoid overlooking akathisia, clinicians should always suspect akathisia in any patients during antipsychotic therapy, some researchers believe (Raskin 1972; Shen 1981). This is because some patients with akathisia do not spontaneously or voluntarily present typical symptoms of akathisia in the presence of the clinician. Akathisia in these cases may be identified by only a clinician who seeks to detect akathisia by questioning the patient and family members about akathisia symptoms despite their apparent absence. The incidence of akathisia detection would increase once the clinician considers it in daily clinical practice.

Failure To Fully Implement Antiakathisia Measures in Ambiguous Cases. When the patient does not fully communicate with the clinician or does not clearly distinguish inner restlessness from anxiety, irritation, and difficulty concentrating and does not manifest typical motor restlessness, such as rocking from foot to foot, akathisia cannot be diagnosed or ruled out. The medication history prior to the appearance of the symptoms should be discerned. Some amelioration of the symptoms following activating procedures, for instance, finger tapping (Munetz and Cones 1983; Fleischhacker et al. 1993; Sachdev 1995*a*) or strong motor exercise of affected parts of the body—such as rapid walking (Kendler 1976)—may lead to a diagnosis of akathisia. The result of pharmacological interventions for the symptoms would also reduce the underdiagnosing of akathisia.

Oral antiakathisia agents such as beta-blockers, anticholinergics, and benzodiazepines have been reported to be effective against akathisia (Table 2) (Fleischhacker et al. 1990; Tonda and Guthrie 1994; Miller and Fleischhacker 2000). However, they often do not completely eliminate akathisia (Braude et al. 1983; Sachdev and Loneragan 1991; Adler et al. 1993). Therefore, the failure of a patient to respond to oral agents does not necessarily help a clinician to rule out akathisia.

Concerning parenteral preparations, Sachdev and Loneragan (1993) reported that intravenous propranolol was not effective against akathisia, although the dosage used in the study was very low (1 mg). The intravenous administration of benzodiazepines (Gagrat et al. 1978; Hirose and Ashby 2002) is effective in treating akathisia but would not be helpful in differentiating akathisia from anxiety due to other illnesses, as these agents produce nonselective sedation.

Van Putten and Mutalipassi (1975) reported that intramuscular biperiden could be used to diagnose akathisia. I know of no report where parenteral anticholinergics were not adequately effective against akathisia; all authors (Ayd 1960, 1961; Medina et al. 1962; Eckmann 1963; Kline et al. 1974; Van Putten et al. 1974; Ross et al. 1983; Sachdev and Loneragan 1993; Hirose and Ashby 2000) employing parenteral anticholinergics for akathisia reported adequate efficacy. This is in contrast to authors reporting that anticholinergics given orally do not produce an adequate response (Kruse 1960*b*; Braude et al. 1983; Adler et al. 1987; Adler et al. 1988). This discrepancy in the results with oral versus parenteral anticholinergics, which is likely due to the differences in the serum level of the drugs, applies also for diazepam (Gagrat et al. 1978; Braude et al. 1983; Adler et al. 1985; Hirose and Ashby 2002). Therefore, intravenous or intramuscular anticholinergics could be recommended in ambiguous cases for diagnostic treatment.

However, there may be rare individuals who do not respond to the parenteral administration of anticholinergics; this lack of responsiveness may occur if the dosage is inadequate. Nonetheless, even in these cases, a considerable reduction of the antipsychotic dose or, more desirably, discontinuation (Munetz and Cones 1983; Drake and Ehrlich 1985; Lang 1988), if clinically possible, usually leads to a disappearance of symptoms in about 1 week (Braude et al. 1983).

There are reported cases of acute-onset akathisia that lasts for months or years following discontinuation of antipsychotic treatment (Kruse 1960*a*; Weiner and Luby 1983*a*; Burke et al. 1989); this is referred to as acute per-

Table 2. Treatment recommendations for antipsychotic-induced acute akathisia

1. Diagnosis or consideration of the likelihood of akathisia
2. Parenteral anticholinergics or benzodiazepines for cases of distressing akathisia requiring rapid relief
3. Reduction of the dosage or withdrawal of antipsychotic if clinically feasible
4. Oral administration of anticholinergics, beta-adrenergic antagonists, or benzodiazepines. (These drugs can be administered concomitantly using high dosages of each agent.)

sistent akathisia (Barnes and Braude 1985; Sachdev and Loneragan 1991). Late-onset persistent akathisia—that is, tardive akathisia (Braude and Barnes 1983; Weiner and Luby 1983*b*)—also does not respond to discontinuation of antipsychotic drugs or typical antiakathisia measures (Miller and Fleischhacker 2000). Therefore, a diagnostic treatment cannot be employed to rule out this type of akathisia over a short period of time. The symptoms and the course may be helpful in identifying such cases. It has been suggested that the level of distress in chronic akathisia is less than that in acute akathisia (Barnes and Braude 1985; Sachdev 1995*a*). However, a detailed characterization of this type of akathisia has not been reported (Miller and Fleischhacker 2000).

Strict Adherence to Research Diagnostic Criteria. Significant variation in the clinical features and disagreement as to which are pathognomonic in akathisia may have led to confusion in diagnosing akathisia (Munetz and Cones 1983; Adler et al. 1989; Sachdev 1994*b*). However, recent akathisia rating scales (Barnes 1989; Fleischhacker et al. 1989; Sachdev 1994*a*) and the research diagnostic criteria in *DSM–IV,* which were constructed to increase diagnostic accuracy, have led to a higher diagnostic threshold. Indeed, these may help provide more homogeneous samples for research purposes but may not be suitable for daily clinical practice.

Strict adherence to diagnostic criteria may lead to underdiagnosis of subthreshold cases of akathisia (e.g., akathisia without objective restlessness, akathisia with restlessness in parts of the body other than the legs). As mentioned above, the manifestations of akathisia can be subtle with low-potency antipsychotics. With newer antipsychotics, the manifestation of akathisia may be more subtle than with conventional antipsychotics. The strict diagnostic criteria may be appropriate to an era where older agents were used in higher dosages.

Drug exposure is an essential criterion for diagnosing akathisia (Sachdev 1994*b*). However, if a subthreshold condition appeared following akathisia-inducible pharmacotherapy and disappeared following antiakathisia measures, it might have been valid to diagnose and treat the condition as akathisia. The other extreme may be regarding the condition as akathisia and implementing antiakathisia measures only when the research diagnostic criteria are met. Underdiagnosis related to the high diagnostic threshold could lead to undertreatment or failure to treat akathisia—thus leading to the same result as in cases of overlooked akathisia.

One could argue that the diagnosis of akathisia with a lower threshold is not exact and thus akathisia may be overdiagnosed. However, this very attitude may increase the overlooking and undertreatment of akathisia. This is a problem because the distress in overlooked akathisia is usually more severe than that of the original illness for which the patient was treated (Kalinowsky 1958). The distress of akathisia can persist for an extended period once one clinician overlooks akathisia—until another clinician finally diagnoses it.

Conclusion

Underdiagnosis or undertreatment of akathisia is likely with the co-occurrence of patient and clinician factors. There are two major points concerning the underdiagnosis of akathisia. One is the failure to identify the conditions that fulfill the diagnostic criteria of akathisia, leading to incorrect or lack of treatment. These conditions include motor restlessness at times other than consultation, lack of voluntary expression of the inner restlessness, atypically expressed inner restlessness, and prominent psychic symptoms besides those of akathisia. Akathisia in these conditions may not be manifested or obvious in unquestioned patients but can be identified following careful questioning by the clinician. The second point is that underdiagnosis may occur because the symptoms presented by the patient do not fulfill the diagnostic criteria or are not present in the rating scales for akathisia. These symptoms, which can be ameliorated by antiakathisia treatments, include inner restlessness unaccompanied by motor restlessness, inner restlessness and motor restlessness appearing in areas other than the legs, unidentified inner restlessness due to lack of communication, and other ambiguous akathisia conditions. As the use of newer antipsychotics increases, clinicians should be more vigilant about subtle manifestations of akathisia.

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