CHAPTER 1
Distinguishing Clinical Features of Hyperkinetic Disorders

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Introduction

Movement abnormalities can be dichotomized into the two broad categories of hypokinetic and hyperkinetic syndromes. The hallmark of hypokinesias is the loss of voluntary and automatic movements (akinesia), which is combined with slowness (bradykinesia) and stiffness or increased muscle tone (rigidity) in akinetic-rigid or parkinsonian syndromes [1]. In contrast, hyperkinesias are manifested by abnormal, uncontrollable, and unwanted movements. This term should not be confused with “hyperkinetic disorders” used in ICD 10 [2] to describe a behavioral abnormality—typically labeled attention deficit disorder with hyperactivity, occurring particularly in children and often associated with attention deficit and a tendency to move from one activity to another without completing any one. This is often associated with disorganized, ill-regulated, and scattered activity and thinking. This is not the only inconsistency between terminology in adult and childhood disorders, and efforts have been recently undertaken to unify the nosology and diagnostic recommendations in pediatric and adult movement disorders [3].

Hyperkinetic movement disorders include six main phenotypic categories, which can appear in isolation or in variable combinations: tremor, chorea, tics, myoclonus, dystonia, and stereotypies. In addition to these six categories there are other abnormalities of motor control that are also included within the field of movement disorders, such as akathisia, amputation stumps, ataxia, athetosis, ballism, hyperekplexia, mannerisms, myorhythmia, restlessness, and spasticity. The term “dyskinesia” is commonly used to indicate any or a combination of abnormal involuntary movements, such as tardive or paroxysmal dyskinesias or levodopa-induced dyskinesia, but more specific phenomenological categorization should be used whenever possible. In addition, there is a large and important group of peripherally-induced movement disorders, exemplified by hemifacial spasm [4], although any hyperkinetic movement disorder can be triggered or induced by peripheral injury [5].

Some conditions combine hypokinetic and hyperkinetic features, as exemplified by the coexistence of bradykinesia and tremor in Parkinson disease (PD) often referred to by the oxymora “gait disorder with acceleration” [6] or “shaking palsy” [7]. Probably the best examples of coexistent hyper- and hypokinesia is levodopa-induced dyskinesia in patients with PD and chorea or dystonia in patients with Huntington disease, many of whom have an underlying hypokinesia [8].

We describe here the hallmark features and phenomenology of the main hyperkinetic disorders, which are listed according to the time of their medical recognition.

**Historical background**

The importance of recognizing the appropriate phenomenology, not only as a guide to diagnosis but also as a means to study the pathophysiology of the disorder, is highlighted by the following statement attributed to Sir William Osler: “To study the phenomenon of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all” [9].

The characterization and classification of the various hyperkinetic disorders has evolved over a long period of time (Table 1.1). Tremor was a common language word before becoming a medical term. In ancient Greek, the root TRE is a lexical unit to indicate at the same time fear and shaking. Tremor was defined by Galen as an “involuntary alternating up and down motion of the limbs.” Involuntary movements present during action or at rest were also mentioned by Sylvius [10]. Parkinsonian tremor was later described by James Parkinson [7] and further differentiated from kinetic “intentional tremor” by Charcot [11]. The familial occurrence of postural action tremor was recognized shortly afterwards [12].

Epidemics of “dancing mania” emerged in central Europe in the late Middle Ages as local phenomena [13] or in connection with pilgrimages. Coincident with the Black Plague in 1348–50, St Vitus was called upon to intercede, leading to the term “chorea Sancti Viti” (St Vitus dance) to indicate at the same time a request for intercession and a means to expiate. This terminology has entered medical literature after Paracelcus described this syndrome among one of the five that “deprive man of health and reason.” He adopted the term “chorea” into medical jargon and proposed using the expression “chorea lasciva” to describe the epidemics [14]. One century later, Thomas Sydenham observed an epidemic affecting only children which he called “chorea minor” [15] and was later recognized to be a manifestation of rheumatic fever. Adult-onset hereditary chorea was described in the 19th century [16] and later renamed Huntington chorea.

The term “tic” arose in France in the 17th century to describe shivers in horses, particularly of certain breeds, which affect primarily the muscles of the pelvic region, pelvic limbs, and tail [17]. The word was later used by French doctors by analogy. The first medical report on human tics is probably the description of the Marquise of Dampierre, who started having tics at 7 years of age [18]. Later, Trousseau listed tics among choreatic disorders [19] and Gilles de la Tourette provided a separate taxonomic categorization of these phenomena [20].

Essential myoclonus was first described by Friedrich [21], who reported a 50-year-old man with a 5-year history of multifocal muscle jerks affecting both sides of the body symmetrically, but asynchronously. The syndrome was defined as “paramyoclonus multiplex” because of the reported symmetry. Forms of myoclonic epilepsy were later described and Lundborg [22] proposed a classification of myoclonus that remains largely in use today. Asterixis was observed in patients with hepatic encephalopathy [23] and later recognized to be a form of negative myoclonus.

Dystonia was the last main hyperkinetic disorder to be recognized: its name derives from a supposed alteration of muscle tone in patients with generalized distribution [24]. The hereditary nature was noted at about the same time [25].

**Table 1.1** Chronology of first description of the main hyperkinetic disorders.

<table>
<thead>
<tr>
<th>Date</th>
<th>Name</th>
<th>First usage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ancient Greece</td>
<td>Tremor</td>
<td>τρεμω (to tremble, to fear)</td>
</tr>
<tr>
<td>XI Century</td>
<td>Chorea</td>
<td>Choreomania (ritual dance)</td>
</tr>
<tr>
<td>XVII Century</td>
<td>Tic</td>
<td>French horse breeders</td>
</tr>
<tr>
<td>1871</td>
<td>Athetosis</td>
<td>Hammond [71]</td>
</tr>
<tr>
<td>1881</td>
<td>Myoclonus</td>
<td>Friedreich [21]</td>
</tr>
<tr>
<td>1885</td>
<td>Ballism</td>
<td>Kussmaul [72]</td>
</tr>
<tr>
<td>1911</td>
<td>Dystonia</td>
<td>Oppenheim [24]</td>
</tr>
<tr>
<td>1953</td>
<td>Asterixis</td>
<td>Adams [23]</td>
</tr>
</tbody>
</table>
Phenomenology and classification

Although at first sight involuntary movements resemble each other, each hyperkinetic disorder has a specific phenomenology (signature) that can be identified by direct observation of the patient or videotaped examination. Duration, rhythmicity, topography, and other features must be carefully analyzed and noted in order to make a specific phenomenological diagnosis [26] (Table 1.2).

Tremor

Tremor is an involuntary, rhythmic, oscillation of a body region about a joint axis. It is usually produced by alternating or synchronous contractions of reciprocally innervated agonistic and antagonistic muscles that generate a relatively symmetric velocity in both directions about a midpoint of the movement [27, 28]. The oscillation produced by tremor can be represented by a sinusoidal curve; it is generated by rhythmical discharges in an oscillating neuronal network and maintained by feedback and feed-forward loops. The resulting movement is patterned and rhythmic, characteristics that distinguish tremor from other hyperkinesias [29].

Tremor varies when different voluntary movements are performed or postures are held: it is labeled as a rest tremor, postural tremor, or action tremor according to the condition of greatest severity. Intention tremor, typically associated with cerebellar dysfunction, is characterized by the worsening of tremor on approach to a target, as in a finger-to-nose maneuver. The typical rest tremor of PD has a frequency of 4 to 6 Hz, and is most prominent distally. Its characteristic appearance in the hand is also referred to as a pill-rolling tremor. Parkinsonian rest tremor also typically involves the chin, jaw, and legs, but almost never involves the neck. Indeed, head oscillation should suggest essential tremor or dystonic tremor rather than PD. True rest tremor, however, disappears during complete rest, such as sleep, and is reduced or disappears with voluntary muscle contraction, or during movement. Postural tremor is present with the maintenance of a particular posture, such as holding the arms outstretched in front of the body. It is commonly seen in physiological and essential tremor. Re-emergent tremor refers to a postural tremor that occurs after a variable latency period during which time no observable postural tremor is present [30]. This typically occurs in the setting of PD, and most likely represents a parkinsonian rest tremor that has been “reset” during the maintenance of a posture [31].

Task-specific tremor occurs only during execution of a particular task, such as writing, and is considered by many to be a variant of dystonic tremor. Dystonic tremor may occur in the setting of dystonia, and is a rhythmic, oscillation-like, dystonic movement [32]. Position-specific tremors only occur when the affected body part is placed in

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Tremor type</th>
<th>Amplitude</th>
<th>Prevalent site</th>
<th>Relation to voluntary movement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–4 Hz</td>
<td>Cerebellar tremor</td>
<td>Medium–high</td>
<td>Limbs</td>
<td>Postural, action</td>
</tr>
<tr>
<td>3–5 Hz</td>
<td>Task-specific tremor</td>
<td>Low–medium</td>
<td>Hand</td>
<td>Writing, feeding, playing an instrument</td>
</tr>
<tr>
<td>4–5 Hz</td>
<td>Parkinsonian tremor</td>
<td>Medium–high</td>
<td>Limbs, jaw</td>
<td>Rest</td>
</tr>
<tr>
<td>5–8 Hz</td>
<td>Essential tremor</td>
<td>Medium–high</td>
<td>Limbs, head, voice</td>
<td>Postural</td>
</tr>
<tr>
<td>8–12 Hz</td>
<td>Physiologic tremor</td>
<td>Medium</td>
<td>Limbs</td>
<td>Postural</td>
</tr>
<tr>
<td>14–16 Hz</td>
<td>Orthostatic tremor</td>
<td>Low–medium (may not be visible, but can be palpated or auscultated)</td>
<td>Legs, trunk</td>
<td>Standing</td>
</tr>
</tbody>
</table>
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a particular position or posture. Orthostatic tremor is an example of a position-specific tremor, and refers to a fast (14–16 Hz) tremor, mainly affecting the trunk and legs, that occurs after standing for a certain period of time [33].

**Chorea**

Chorea is an irregular, unpredictable, involuntary random-appearing sequence of one or more, discrete, involuntary jerk-like movements or movement fragments. Movements appear random due to the variability in timing, duration, direction, or anatomic location. Each movement may have a distinct start and end point, although these may be difficult to identify since movements are often strung together, one immediately following or overlapping another. Movements may, therefore, appear to flow randomly from one muscle group to another, and can involve trunk, neck, face, tongue, and extremities. Infrequent and mild chorea may appear as isolated, small-amplitude brief movements. It may resemble restless, fidgety, or anxious behavior. When chorea is more severe, it may appear to be almost continuous, flowing from one site of the body to another (Figure 1.1).

Although chorea may be worsened by movement, it usually does not stop with attempted relaxation. Chorea is distinguished from tremor and dystonia by its lack of rhythmicity and predictability. Chorea may be difficult to differentiate from myoclonus, but the latter is more intermittent rather than continuous. Chorea is typically a fluent disorder involving contiguous body parts in variable order and direction. It may be associated with hypotonia, hung-up and pendular reflexes, and motor impersistence (inability to maintain a sustained contraction). Examples of impersistence include an inability to maintain prolonged tongue protrusion or handgrip (“milkmaid grip”). The term “parakinesia” refers to the incorporation of the involuntary movements into semipurposeful movements, in a semiconscious attempt to camouflage the chorea. Examples of parakinesia include touching one’s face, adjusting glasses, and other mannerisms that often served to delay the recognition of the involuntary movement.

Ballism is characterized by high amplitude, almost violent, movements that mainly involve the proximal limb joints. It is considered an extreme phenomenological expression of the spectrum of chorea that affects proximal joints such as shoulder or hip. This leads to large amplitude movements of the limbs, sometimes with a flinging or flailing quality. As patients recover from acute ballism, frequently associated with a stroke in the contralateral subthalamic nucleus, the ballistic movements often gradually evolve into chorea or dystonia (see Chapters 10 and 11).

**Tics**

Tics are repeated, individually recognizable, intermittent movements or movement fragments that are almost always briefly suppressible and are usually associated with the awareness of an urge to perform the movement, the so-called “premonitory sensation.” Motor tics often result in either a simple jerk-like movement such as a blink, facial grimace, head jerk, or shoulder shrug, or more complex, stereotyped, semivoluntary, intermittent movements. Tics are usually abrupt in onset, fast and brief (clonic tics), slow and sustained (dystonic tics), or manifested by sudden cessation of movement because of isometric muscle contractions (tonic tics), or inhibition of voluntary movement (blocking tics). The duration of each tic movement is characteristic of that tic, and the duration does not generally vary between different repetitions [34]. Tics can occur during all stages of sleep.

Characteristic features include predictability of both the nature of the movement and its onset, suggestibility, exacerbation during excitement or stress and also after stress (rebound), and brief voluntary suppressibility. Complex motor tics may resemble normal motor acts or gestures, but are generally inappropriately intense and timed [34]. The movements can appear purposeful, such as touching, throwing, hitting, jumping, and kicking, or non-purposeful, such as head shaking or trunk bending. Occasionally tics can be so severe as to cause neurological sequels, with reports of compressive cervical myelopathy resulting from recurrent head thrusting and violent neck
hyperextension tics [35]. Complex motor tics can also include copropraxia (grabbing or exposing one’s genitals) or echopraxia (imitating gestures).

Motor tics are almost invariably accompanied by vocal or phonic tics and many experts view motor and phonic tics as having the same pathophysiological mechanism. Simple phonic tics can involve brief occurrences of sniffing, throat clearing, grunting, screaming, coughing, blowing, or sucking sounds. Pathological laughter has also been reported as a manifestation of a simple phonic tic [36]. In contrast, complex phonic tics are semantically meaningful utterances and include coprolalia, or shouting of obscenities, profanities, or other insults. Other

Figure 1.1 This photographic sequence (1.5 frames per second) permits an appreciation of the rapid flow of chorea motor fragments in a patient with Huntington disease.
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complex phonic tics include echolalia (repeating someone else’s words or phrases) and palilalia (repeating one’s own utterances, particularly the last syllable, word, or phrase in a sentence). Rarely, tics may be continuous and disabling, resulting in a so-called “tic status” [37] or in severe, self-injurious, even life-threatening behaviors, so called “malignant Tourette syndrome” [38]. Because of the broad expression of Tourette syndrome, manifested not only by motor and phonic tics but by a variety of behavioral comorbidities (such as attention deficit with hyperactivity, obsessive-compulsive disorder, and impulsivity), the management depends on establishing an appropriate hierarchy of the various symptoms and targeting the therapeutic strategies to the most troublesome problems [39]. (See Chapters 12 and 13).

Athetosis

Athetosis is a slow, continuous, involuntary writhing movement that (1) prevents the maintenance of a stable posture; (2) involves continuous smooth movements that appear to be random and are not composed of recognizable movement fragments; (3) typically involves the distal extremities (hands or feet) more than the proximal and can also involve the face, neck, and trunk; and (4) may worsen with attempts at movement or posture, but can also occur at rest.

Athetosis rarely occurs in isolation but is much more commonly associated with chorea and dystonia. In fact, it is considered a variant of distal chorea or dystonia. Phenomenologically, athetosis is at the opposite end of ballism, resulting in a slow, gentle, and distal motion, resembling slow chorea. The recognition of athetosis often leads to consideration of cerebral palsy or paroxysmal choreoathetosis. Pseudoathetosis refers to a severe distal sensory loss syndrome whereby involuntary, slow, writhing movements are due to loss of proprioception [40].

Myoclonus

Myoclonus consists of repeated, often non-rhythmic, brief shock-like jerks due to the sudden involuntary contraction or relaxation of one or more muscles. These “lightning-like” movements differ from epileptic myoclonus and do not affect consciousness [41]. Myoclonus may be synchronous (several muscles contracting simultaneously), spreading (several muscles contracting in a predictable sequence), or asynchronous (several muscles contracting with varying and unpredictable relative timing). When myoclonus affects more than one muscle in an apparently random and varying pattern it is called multifocal; it is called generalized when many muscles through the body are involved simultaneously. Myoclonus is characterized by a sudden unidirectional movement due to agonist contraction (positive myoclonus) or by sudden brief muscle relaxation (negative myoclonus) [42]. The latter is exemplified by asterixis, which typically presents in patients with hepatic and other encephalopathy.

The distinction between myoclonus and other involuntary disorders – particularly tics, chorea, and different varieties of jerks – is not always clear. Tics are usually associated with a generalized, conscious, urge or local premonitory sensation to move and a feeling of relief of tension after the movement. In addition, many tics are suppressible, in contrast to myoclonus. Brief muscle movements in dystonia are often associated with dystonic posturing. Mild chorea may be difficult to distinguish from myoclonus. Sometimes myoclonus is rhythmic and can resemble tremor. When myoclonus is repeated rhythmically it is also called “myoclonic tremor”, but this is a misnomer as rhythmic myoclonus, such as palatal myoclonus [43], is caused by contractions of agonists only, not alternating contractions of antagonist muscles as seen in tremor.

Myoclonus can be caused or worsened by movement and can sometimes occur during sleep. Myoclonus can be categorized as action myoclonus, postural myoclonus, or rest myoclonus on the basis of the condition in which it is observed [44]. It can also be categorized on the basis of the presumed anatomic origin as cortical, subcortical, brainstem, propriospinal, or spinal. Myoclonus may coexist with dystonia (as in myoclonus-dystonia syndrome) or with tremor (as in essential myoclonus) [45]. (See Chapters 14, 15, and 16).
Dystonia
In dystonia, involuntary sustained or intermittent muscle contractions cause twisting and repetitive movements, abnormal postures, or both. The combination of postures and dystonic movements is typical of dystonia [46].

Dystonic postures are repeated and particular patterns or postures are characteristic of each patient at a given point in time. Similar dystonic postures may occur in different patients. Postures can be sustained, particularly at the peak of dystonic movements, or may occur during very brief intervals. Dystonic postures are often triggered by attempts at voluntary movement or voluntary posture, and in some cases they are triggered only in particular body positions or by particular movements as may occur in task-specific dystonia. With the exception of certain seizure disorders [47], dystonic movements or postures are not typically seen during sleep, possibly due to inhibition of movements by spinal mechanisms [48]. Postures tend to occur at intervals determined by voluntary movement and can be sustained for variable lengths of time. Relaxation may be impaired so that the dystonic posture may be maintained well beyond the end of the attempted voluntary movement that triggered it. There may be multiple dystonic postures in the same patient, so that different dystonic postures may be combined.

Dystonic movements may vary in terms of speed, amplitude, rhythmicity, forcefulness, and distribution in the body, but the same muscles are usually involved; hence the term “patterned” movement disorder. Dystonia may occur at rest, during activity or only during a specific motor movement or posture, so-called task- or position-specific dystonia (Figure 1.2) [49]. The most common adult-onset upper limb task-specific dystonia is writer’s cramp [50]. Musician’s cramp occurs while playing a musical instrument [51]. Embouchure dystonia affects the control of the lip, jaw, and tongue muscles, and may be seen in woodwind and brass players [52].

The term “fixed dystonia” is used to indicate persistent, abnormal posture, without a dynamic component. When present but untreated for weeks or longer, dystonia may lead to fixed contractures. Fixed dystonia is often associated with painful contracture, as in post-traumatic, chronic regional pain syndrome [53] or sustained voluntary contraction as in psychogenic dystonia. (see Chapter 24).

Dystonia is typically associated with the occurrence of gestes antagonistes (or sensory tricks), mirror phenomena and overflow [54–56]. Their recognition supports the clinical diagnosis of dystonia [46]. Dystonia can affect any body part, with a wide range in severity from very mild to extremely severe cases (see Chapters 8 and 9).

Stereotypies
Stereotypies are involuntary or unvoluntary (in response to or induced by inner sensory stimulus or unwanted feeling), coordinated, patterned, repetitive, rhythmic, seemingly purposeless movements or utterances [57]. Although stereotypies typically occur in children with autism or other pervasive developmental disorders, they can also occur in adults. Typical motor stereotypies encountered in children with autism include body rocking, head nodding, head banging, hand washing and waving, covering ears, fluttering of fingers or hands in front of the face, repetitive and sequential finger movements, eye deviations, lip smacking, and chewing movements, pacing, object fixation, and skin picking. Phonic stereotypies include grunting, moaning, and humming. In adults, stereotypies are...
usually encountered in patients with tardive dyskinesias. In this setting stereotypies are usually in the form of orofacial or lingual chewing movements, pelvic rocking movements and other repetitive coordinated movements. They are often accompanied by akathisia, manifested by motor and sensory restlessness (see Chapters 21 and 22).

Non-motor features
Psychiatric morbidity is higher in patients with hyperkinetic movement disorders than in community samples or in patients with other forms of chronic disease. Behavioral abnormalities have been reported in patients with Tourette syndrome [58], Wilson disease [59], dystonia [60], essential tremor [61], Sydenham chorea [62] and Huntington disease gene carriers [63]. Age at onset is likely to be an important determinant of susceptibility to psychiatric morbidity in many of these conditions.

Given the complexity of basal ganglia functions, it is not surprising that hyperkinetic disorders are frequently associated with behavioral or psychological changes that, in many cases, are considered to have a pathogenic commonality with the motor disturbance. Basal ganglia pathology engenders a wide spectrum of neuropsychiatric symptoms [64], which are thought to involve the associative circuit (focused on the dorsolateral caudate nucleus and the caudalventral putamen) and the emotional circuits (centered in the ventral caudate nucleus, the nucleus accumbens, and the amygdala) [65, 66].

Particularly chorea, tics, and dystonia are coincident with obsessive-compulsive traits, anxiety, or depression in different combinations and with variable severity. Such coincidence may be due to an underlying basal ganglia dysfunction producing both motoric and behavioral expressivity. Of particular interest is the finding that depression, attention-deficit hyperactivity disorder and vocal tics are significantly more common in children with Sydenham chorea, compared to children who had rheumatic fever without Sydenham chorea [67]. Medication-related adverse effects may be an additional source of depression or anxiety in patients with hyperkinetic movement disorders and cause akathisia or additional hyperkinesias [68–70].

Behavioural features associated with hyperkinetic disorders should not be confounded with psychogenic movement disorders, which are abnormal movements thought to be due to pre-existing psychological or psychiatric disturbances. The borderland between movement disorders and psychiatry is a difficult diagnostic area. It is remarkable that most movement disorders were initially considered psychogenic due to the inexplicability of their phenomenology, such as the paradigmatic case of primary dystonia, featuring bizarre postural abnormalities, relief by gestes antagonistes, task specificity, and normal brain morphology. The organic nature of primary hyperkinetic movement disorder is now unequivocally recognized, although they may not always be easily differentiated from psychogenic hyperkinesias. Chronicity, social impairment, and stigma, however, can affect the ability of patients with hyperkinetic disorders to develop or continue many of their key social roles, such as marital or employment status, thus engendering reactive depression or other secondary behavioral consequences.

Clinical examination and medical recording
Although the expert clinician can quickly attempt to recognize the features of hyperkinetic disorders (Figure 1.3) it is necessary to accomplish a thorough documentation of the observed features to avoid mistakes and allow review and comparison of the phenotype [26, 57].

Examination of patients with a hyperkinetic movement disorder must include a full examination for associated neurological findings. It must also include an assessment of the effect of the movement disorder on overall motor function and quality of life. Observation of the disorder itself should include several components, including the phenomenology of the disorder, the time-course, triggers and suppressibility, and the somatic distribution (focal, segmental, multifocal, and generalized). The phenomenology should be described in terms of duration, speed, amplitude, jerkiness, repeatability, or stereotyped quality, and
the number of different identifiable movements or postures. The time-course should be described in terms of rhythmicity, whether it is intermittent with intervening more normal movement, whether movements are sustained or ongoing, and whether there are discrete submovements or movement fragments or whether the movement appears to be continuously flowing. Possible triggers should be assessed from the history and examination, including attempted movement, posture, rest, and emotional state. Suppressibility can be tested in clinic or assessed from the history, and the presence of an urge to move should be determined.

Distractibility evaluates whether unrelated mental or physical tasks (as opposed to asking the patient to voluntarily suppress) result in movement suppression. Distractibility can be seen in tics, stereotypies, and psychogenic movements.

Given the patient’s consent, it is valuable to take a video of the clinical interview and medical examination. This allows the examiner to review the phenomenology of the hyperkinetic disorder, to seek expert consultation and visually compare phenomenology changes during natural course of the condition. It is particularly important to show as clearly as possible on the video clip the features listed

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**Table 1.3** General features of hyperkinetic disorders.

<table>
<thead>
<tr>
<th>Movement</th>
<th>Regularity</th>
<th>Rhythmicity</th>
<th>Speed</th>
<th>Suppressibility</th>
<th>Duration</th>
<th>Triggerability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tremor</td>
<td>Yes</td>
<td>Yes</td>
<td>1–16 Hz</td>
<td>Sometimes briefly</td>
<td>Transient or permanent</td>
<td>Position, posture, voluntary movement</td>
</tr>
<tr>
<td>Chorea</td>
<td>No</td>
<td>No</td>
<td>Fast</td>
<td>No</td>
<td>Transient or permanent</td>
<td>Voluntary movement</td>
</tr>
<tr>
<td>Tics</td>
<td>No</td>
<td>No</td>
<td>Slow or fast</td>
<td>Usually</td>
<td>Transient or permanent</td>
<td>Sensory stimuli, stress, thoughts</td>
</tr>
<tr>
<td>Myoclonus</td>
<td>Sometimes</td>
<td>Sometimes</td>
<td>Fast</td>
<td>No</td>
<td>Transient or permanent</td>
<td>Sensory or proprioceptive stimuli</td>
</tr>
<tr>
<td>Dystonia</td>
<td>No</td>
<td>Rarely</td>
<td>Slow or fast</td>
<td>Partial or only briefly</td>
<td>Transient, permanent or paroxysmal</td>
<td>Specific motor tasks</td>
</tr>
</tbody>
</table>

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**Figure 1.3** Flow chart for a quick orientation in the differential diagnosis of the five main hyperkinetic disorders.
in Tables 1.3 and 1.4, allowing the specifics of the observed phenomena to be visually evaluated. A well-constructed video recording can convey more accurate information than standard clinical notes.

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