REVIEW ARTICLE

JIACM 2023; 24 (1): 49-57

Approach to A Patient with Tremor

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Abstract

Tremor is a common complaint and a frequently observed entity in neurology clinics. Given the various generation sites and the anatomical substrates which can give rise to tremor, the diagnosis of tremor syndrome and its localisation requires a good history taking and a prudent examination approach. This review focusses on the 'clinical pearls' for diagnosis of various tremor syndromes and their possible aetiological correlates.

Key words: Approach to tremor.

Introduction

Tremor, the most common neurological disorder[1] is defined as a rhythmic involuntary movement produced by either alternating or synchronous contractions of reciprocally innervated antagonistic muscles of one or several regions of the body[2]. The key feature of tremor is its rhythmicity, which is not easy to identify because, despite a fixed frequency, the variable amplitude may give an erroneous impression of variable frequency. Evaluation of tremor requires a comprehensive history and neurological examination. In this review, clinical methods for evaluating various tremor syndromes are discussed.

Classification of tremor

Tremor can be classified based on frequency, amplitude, and body part affected. However, clinically, the most important classification is phenomenological classification into rest and action tremor[3] (Fig. 1). Rest tremor is said to be present when the body part is not voluntarily activated, which may require complete support of body part against gravity. Action tremor is said to occur when it occurs in a body part that is voluntarily activated – whether in maintaining posture (Postural Tremor) or in performing an activity (Kinetic Tremor). Kinetic tremor is further subdivided depending on the range of movement – simple kinetic tremor when tremor is same throughout the movement and intention tremor when tremor markedly increases in terminal portions of the movement.

Approach to a patient with tremor

According to a 2018 classification proposed by the International Parkinson and Movement Disorder Society[3], there are 3 important questions that need to be answered in a patient being evaluated for tremor: is it really tremor, what the tremor syndrome is (Axis 1) and what is the aetiology of tremor syndrome. First and foremost it is important to determine the age of onset of tremor [infancy (birth to 2 years); childhood (3 - 12 years); adolescence (13 - 20 years); early adulthood (21 - 45 years); middle adulthood (46 - 60 years); and late adulthood (> 60 years)], the time course of onset and evolution of symptoms over time with respect to body parts involved and activation conditions, any associated medical conditions or drugs that could exacerbate or precipitate tremor, family history of any movement disorder and presence of any accompanying neurological deficits including but not limited to slowness, stiffness and pulling sensations or pain that may indicate a combined tremor syndrome. The clinician should enquire about activation conditions of tremor, and open-ended questions like "What type of tremor do you have?" may not always be helpful. It may be more prudent to ask specific questions like, whether the tremor occurs at rest as while lying down with arms relaxed, while walking or while sitting with arms supported (indicating tremor in a resting position); or whether tremor occurs while maintaining posture (e.g., while holding things such as glasses, laser pointer, mobile, etc.) or whether it occurs while performing some action (indicating kinetic tremor) such as writing, pouring liquids in cups. One should also determine, whether tremor occurs immediately on holding a thing or occurs after some time (indicating re – emergent tremor). It is also essential to note whether tremor occurs in a specific position or while performing a specific action. One should also ask whether the patient is aware or unaware of the tremor because some patients with essential tremor may be unaware of neck tremor. Finally, clinician should determine how much the activities of daily living are affected, because of tremor. Tremor should be differentiated

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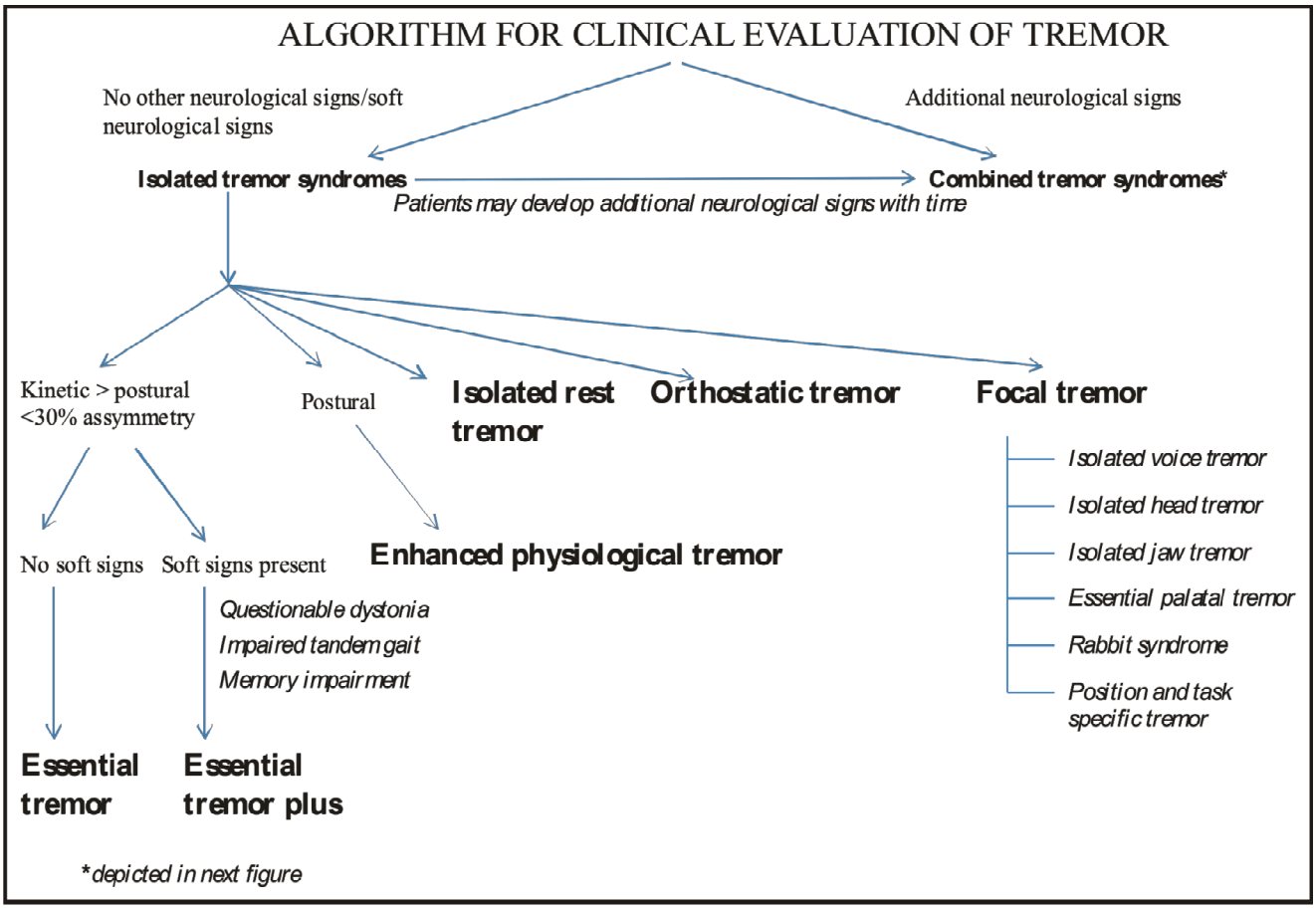

Fig. 1: *Approach to tremor.*

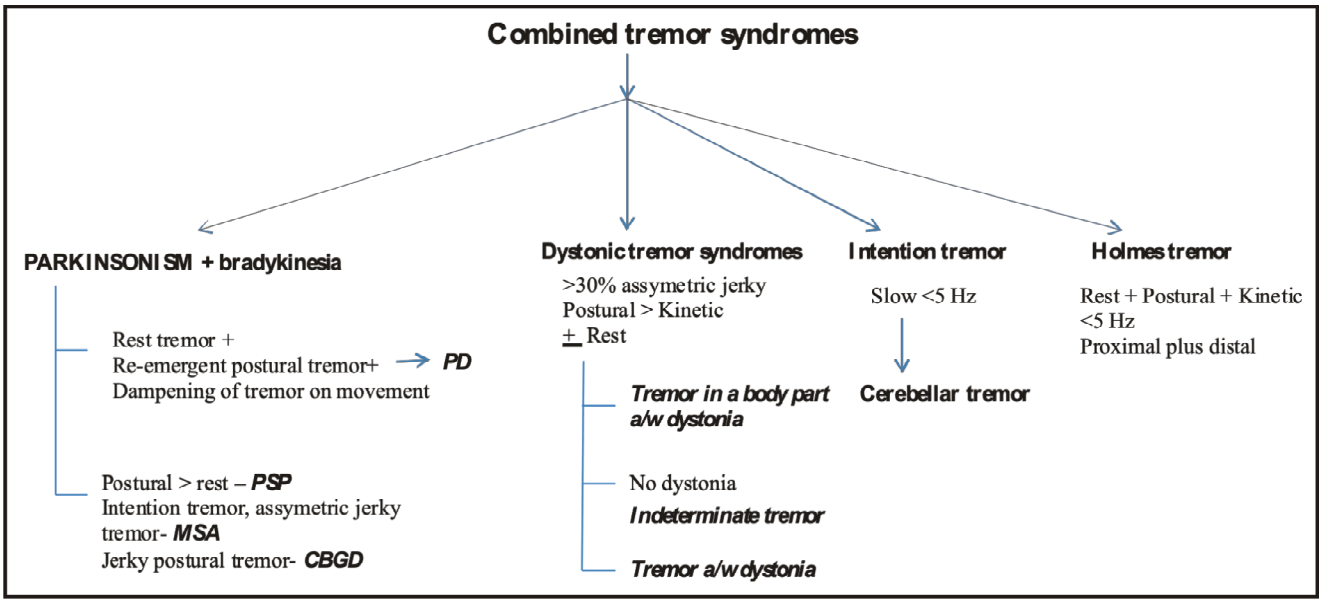

Fig. 2: *Approach to combined tremor syndromes.*

from other movement disorders such as chorea, myoclonus and dystonia[4] (Table I).

Table I: Differentiating features of different movement disorders[43].

Characteristics	Tremor	Chorea	Dystonia	Myoclonus
Jerky character	–	+	–	+ (Jerky irregular "tremor" is usually a manifestation of myoclonus)
Rhythmicity	Rhythmicity is the hallmark	–	–	– Rhythmicity is a feature of segmental myoclonus
Patterned	+	–	+	– Segmental myoclonus-patterned
Sustained character	–	–	+	–
Flowy nature	–	+	–	–
Paroxysmal/continual/continuous*	Continuous	Continual	Dystonic movements-continual Dystonic postures-continuous	Arrythmic myoclonus-continual Rhythmic myoclonus-continuous
Suppressibility**	+	++++	++	–

**Continual-over and over again, continuous-occurs without stopping.*

***Magnitude of suppressibility- No. of '+' denotes the magnitude of suppressibility, highest suppressibility being for stereotypies (++++++) followed by tics and akathisia (+++++).*

Examination of tremor: Examine the patient with his or her arms relaxed, ideally on the arms of a chair or on patient's lap, half pronated completely supported against gravity[5]. Since rest tremor may be intermittent, it may not be apparent immediately in all patients and even severe tremor may temporarily disappear. Certain provocative manoeuvres may be used to make a rest tremor apparent: walking, counting backwards from 100 loudly, motor tasks with contralateral hand or foot, and stroop test[6]. It has been seen that maximum effect of these provocative tests may be seen at about 2 - 3 minutes, so wait for a few minutes before concluding absence of rest tremor. Tremor examination under provocation can potentially distinguish between an overflow of postural tremor into the resting condition and real resting tremor. One should also note across which joints movements are occurring and in what directions. Next, to examine postural tremor, ask the patient to sit with arms stretched out in front and fingers open. Ask the patient to close his or her eyes and count backward again. One should observe latency of onset of tremor after attaining the posture (to look for re-emergent tremor) and any subtle posturing or spooning of the fingers (tendency during arm extension to flex the wrist and hyperextend the metacarpophalangeal and phalangeal joints) that may be indicative of dystonic tremor. Next the patient is asked to hold the arms in different positions (to look for positional variability and position specificity) – in a wing position with the hands facing inward, but not touching each other, arms abducted at the shoulder and bent at the elbows, arms in the same position but with the forearms pronated or supinated), arms outstretched in a karate-chop position, or any other position in which the patient has noticed tremulousness[7]. For kinetic tremor, patient should be asked to do the finger-nose-finger test, draw spirals, vertical and horizontal lines, write a sentence, pour water between cups, or drink from a cup[7]. One should look for any dampening of tremor with movement, and any exacerbation of tremor as the limb approaches the target (intention tremor) and the relative severity of postural and kinetic component. Attempts should be made to distinguish dysmetria (overshoot or undershoot) from intention tremor. Finally, one should look for any cranial tremors: head (i.e., neck) (while seated and while lying), jaw (with mouth closed and then while open), face, chin, tongue, and voice (during sustained phonation)[8]. To test for isometric tremor, ask the patient to push against a wall, flex the wrist against a table, or make a fist. To determine task specific tremor, patient can be asked to perform the particular task that provokes tremor, such as writing. At the end of a neurological evaluation, the clinician should be able to formulate the type of tremor syndrome – isolated (when tremor is the only neurologic manifestation), or combined (when other systemic or neurologic signs co-exist with the tremor); and rest, postural, kinetic or a combination of the three. The next step would be to determine the aetiology, which will depend on the neurological examination and targeted investigations. Brief clinical features of isolated and combined tremor syndromes are discussed.

Isolated Tremor Syndromes: Isolated tremor syndromes include essential tremor, focal tremors, task and position specific tremors, and orthostatic tremors.

Essential tremor: It is the most common cause of an isolated tremor syndrome. ET is a heterogenous disorder and there is considerable variability in the character of the tremor, activation conditions and association with other neurologic deficits. ET has a bimodal peak of onset, most commonly occurring in late life (> 60 years), but early onset before the age of 40 years with slow progression over several years is also seen[9]. The cardinal feature of essential tremor is action tremor (postural > kinetic tremor) which may be observed during a variety of activities on neurologic

examination (extending arms, spiral drawing, pouring water between two cups, finger-nose-finger maneuver). The tremor usually starts bilaterally but may start unilaterally in about 20% of cases. When it starts unilaterally, it progresses to involve the other limb in 2 - 3 years but some amount of asymmetry (about 30%) may persist and in 5% of patients the tremor is markedly asymmetric or unilateral[10]. Classically the tremor is absent on rest, but immediately appears as soon as arms are held outstretched. On movement, tremor may decrease, but again reappears as the target is reached (terminal tremor). This is different from intention tremor, in which tremor oscillations increase steadily before arriving at the target rather than at the termination of goal-directed activity. About 50% of patients with ET have intention tremor which is not limited to arms and can be seen in neck[11] as well as lower limbs[12,13], but clinically visible cerebellar symptoms are generally unusual. The postural tremor may be out of phase in the limbs, which accounts for the observation that functionality may improve when two hands are used, rather than one hand (e.g., while holding a cup)[7]. The tremor has greatest amplitude at the wrist joint, rather than more proximal or distal joints, and generally involves wrist flexion-extension rather than rotation/supination[12]. The tremor of ET is regularly recurrent and directionally symmetrical which can be easily observed on spiral drawings and this helps in differentiating from dystonic tremor[12,14]. Generally, there is no rest tremor, but in 10 - 15% of advanced cases, there may be rest tremor[15] which is most probably because the patient is not able to completely relax the limb[16]. Differentiating from rest tremor of PD may be difficult in these advanced cases but differentiating points in favour of essential tremor are: absence of pill rolling tremor, absence of re-emergent tremor, absence of signs of bradykinesia and rigidity and absence of dampening of tremor on movement. Although head (34%), lower limbs (20%), voice (12%), face and trunk (5%) may be involved, arm tremor in ET is always be more severe than tremor elsewhere[17]. Patients with ET may have a dominant family history of tremor. There may be some improvement with alcohol, but this feature is neither sensitive nor specific[5]. Diagnosis of ET requires absence of other neurological signs, such as dystonia, ataxia, or parkinsonism (Table II). Tremor with the characteristics of ET and additional neurological signs of uncertain significance such as impaired tandem gait, questionable dystonic posturing, memory impairment, or other mild neurologic signs, including rest tremor of unknown significance that do not suffice to make an additional syndrome classification or diagnosis, is now referred to as ET plus[3].

Enhanced Physiological Tremor: All normal people have a very low-amplitude, high-frequency physiologic tremor of approximately 10 to 12 Hertz (Hz) with a much lesser amplitude that sometimes get enhanced because of numerous factors such as fatigue and anxiety – Enhanced Physiological Tremor. It can be demonstrated by holding a piece of paper on the outstretched hand when shaking of the paper may be obvious even though tremor is not grossly visible or by using a laser pointer on a distant screen. Some medications and medical problems can also cause EPT[18]. The main differential is essential tremor (Table III) and treatment is mainly reassurance because it is usually not symptomatic except in fine motor tasks requiring extreme precision (e.g., microsurgery, jewellery making).

Table II: Diagnostic criteria of essential tremor[3].

1. Isolated tremor syndrome of bilateral upper limb action tremor.
2. At least 3 years' duration.
3. With or without tremor in other locations (e.g., head, voice, or lower limbs).
4. Absence of other neurological signs, such as dystonia, ataxia, or parkinsonism.

Table III: Clinical cues for differentiation of essential tremor and enhanced physiological tremor[7].

Features	Essential tremor	Enhanced physiological tremor
Frequency of postural and kinetic component	Lower (5 - 10 hz)	Higher (8 - 12 hz)
Amplitude of tremor	Higher	Lower
Intentional component of tremor	May be prominent (although needs to be differentiated from terminal tremor)	Absent
Body regions involved	May have involvement of voice, limb and head	Head tremor not a part of spectrum

Isolated focal tremors: Patients with isolated focal tremor, without accompanying dystonia, pose a diagnostic challenge of whether the tremor is part of an incomplete phenomenology of dystonia (so-called *"formes frustes"*) or similar in pathophysiology to ET.

Head tremor is a common focal tremor, that can be seen in the context of ET, cervical dystonia and cerebellar disorders. Isolated head tremor is not likely to be ET, because it has been observed that head tremor in ET is often seen in the presence of arm tremor[19] and is more common in women[20]. Moreover, tremor may precede the onset of dystonic postural abnormalities and may remain isolated for extended periods and even for the whole disease course making it a form fruste of cervical dystonia rather than ET. Head tremor in cervical dystonia more often persists when a patient lies down, whereas in ET head tremor usually dampens on lying down[21].

Isolated Voice Tremor: Voice tremor can be seen in PD, ET, ataxic dysarthria, and spasmodic dysphonia. Patients with

visible and/or audible tremor of the vocal apparatus and no signs of dystonia in the vocal apparatus and no tremor, dystonia, or other neurological signs elsewhere are considered to have isolated voice tremor (also labelled as essential voice tremor). Whether this is part of the clinical spectrum of ET[22] or a form fruste of dystonia is still debated[23]. Voice tremor of spasmodic dysphonia is often associated with voice breaks or strangulated speech in contrast to essential tremor.

Jaw tremor: Jaw tremor is a recognised feature of PD (often seen at rest), ET (often seen when the mouth is held open or during speech); hereditary geniospasm; neuroleptic treatment and in normal situations, such as shivering. In all these conditions, jaw tremor is associated with tremor or other abnormal involuntary movements affecting additional body parts, and the tremor frequency usually does not exceed 12 Hz[24]. A high-frequency idiopathic isolated jaw tremor of 14 -16 Hz has also been described[25]. It has been speculated that it could be a focal variant of primary orthostatic tremor affecting the masseter muscles[26].

Palatal Tremor: Essential palatal tremor presents with the symptom of an ear click, mostly attributed to rhythmic contraction of the tensor veli palatini characterised by rhythmic movement of the roof of soft palate at 0.5 to 5 hz. It is not associated with any other neurological abnormality and disappears in sleep which differentiates it from symptomatic palatal tremor[27].

Rabbit Syndrome: This was first described by Villeneuve[28] and is used to describe orofacial movement like that of a rabbit eating and is often associated with a popping sound. It classically occurs after long-term use (months to years) of dopamine receptor blocking agents and imipramine, citalopram, paroxetine, methylphenidate, and phenol intoxication. There are fine rhythmic movements (5 Hz) at rest involving only the vertical axis of the oral, perinasal and masticatory muscles[29]. It is essential to differentiate rabbit syndrome from tardive dyskinesia because unlike tradive dyskinesia, rabbit syndrome usually shows improvement with anticholinergic agents. Differentiating points favoring rabbit syndrome include: absence of tongue involvement, restriction of lip movements in the vertical plane (in contrast to chewing and lip smacking in tradive dyskinesia), and suppressibility.

Position and Task Specific Tremors: Isolated task and position specific tremors are usually focal and occur during a specific task or posture. Isolated task and position specific tremors can be confused with similar syndromes that occur in combination with other neurological signs, such as dystonia (e.g., writer's cramp with dystonic tremor) and parkinsonism (e.g., young-onset PD with dystonia). The most common entity that presents as task specific tremor is writing tremor (previously also called primary writing tremor). It is characterised by prominent pronation/ supination wrist movements that occurs predominantly or exclusively during writing without accompanying dystonia. There may be associated mild postural and terminal kinetic tremor. the frequency of primary writing tremor is like that seen in patients with essential tremor (i.e., 4 hz to 8 hz) and it is relieved by ethanol consumption in 30% to 50% of cases. Writing tremor has been variably classified as an independent entity, an ET variant, a focal dystonia, or an overlap between ET and dystonia[30]. Positional tremors arise only when a tremor is brought on during specific positioning of the involved body part and need to be differentiated from postural tremor, wherein a tremor is elicited in any posture, though it may be more prominent in one posture.

Orthostatic Tremor (OT) syndrome is a rare tremor disorder of the legs and trunk that occurs on standing and dissipates with walking or sitting down[31]. On standing, OT increases in a crescendo fashion over seconds to minutes to the point that the patient cannot continue to stand and must either sit or walk. Patients tend to stand with a wide base, but can walk with a narrow base, tend to avoid standing in queues because of OT. Primary OT has a very high frequency and it may not be visible by naked eye but can be heard with a stethoscope with a sound resembling a distant helicopter during auscultation (helicopter sign)[32]. Diagnosis needs confirmation with EMG recordings that reveal a 13 - 18 Hz tremor[33]. If there are additional signs of dementia, parkinsonism or ataxia it should be labelled as primary orthostatic tremor plus[34]. On the other hand some patients may have a slower orthostatic tremor; often in association with other neurological signs, and have been labeled as slow orthostatic tremor, tremor in orthostatism, and pseudo-orthostatic tremor[35]. Pseudo orthostatic tremor has a frequency of 4 - 6 Hz, may be assymetrical and is seen in conditions such as Parkinson's disease, Lewy body dementia and SCA3.

Isolated Rest Tremor: There is a group of patients with asymmetric rest and postural tremor, mostly resembling a somewhat irregular parkinson tremor, but often do not have other signs of parkinsonism and may show normal dopaminergic striatal innervation measured with Fluorodopa-PET. Various terms have been used for this group of patients - scans without evidence of a dopaminergic deficit' (SWEDDs), Benign Tremulous Parkinson's Disease, monosymptomatic tremor at rest and isolated rest tremor. Its exact aetiology is not yet clear. It has been seen that almost 15% of patients who present with an asymmetric rest tremor resembling PD have a normal dopamine transporter scan[36] and of these only 8 - 13% converted to have an abnormal scan up to 5 years later[37]. Others developed clinical features of advanced

Table IV: Clinical cues for differentiation of essential tremor, dystonic tremor and parkinsonian tremor[44].

Clinical features	Essential tremor	Dystonic tremor	Parkinsonian tremor
Anatomy	Hands > head > voice > others Wrist tremor > metacarpal joint tremor At wrist, flexion extension > rotational	Head >hands>others	Hands > others Metacarpal joint tremor > wrist tremor At wrist, rotational > flexion extension
Symmetry	Largely symmetrical, gross asymmetry (5%)	Asymmetrical	Asymmetrical
Activation	Posture > kinetic > rest	Posture > kinetic > rest	Rest > posture
Tremor suppression at movement onset	No	Rare	Most cases
Mental concentration	Tremor increases	Variable	Tremor decreases
Writing	Tremor increases	Variable	Tremor decreases
Walking	Tremor decreases	Variable	Tremor increases
Sensory trick	None	Most cases	None
Presence of null point*	None	Most cases	None
Alcohol intake	Supresses	Rarely	Rarely
Treatment	Beta blockers, primidone, dbs, botulinum toxin	Botulinum toxin, dbs	Levodopa, dbs

**Relief of tremor occurs by allowing the abnormal dystonic posture to develop without resistance.*

disease by the final third of their disease course and were then indistinguishable from classical Parkinson's disease. As a result, this is a heterogenous group of patients that may have different clinical outcomes including but not limited to: dystonic tremor, early PD, essential tremor plus, PD with striatal dopaminergic deficiency without nigral degeneration, Holmes tremor or atypical parkinsonism. There are certain important caveats, that the clinician must keep in mind while evaluating a patient with isolated rest tremor. First, bradykinesia is the *sine qua non* for parkinsonism and it requires progressive decline in amplitude and velocity of movements (motor decrement). Slowness of initiation of movement or reduced amplitude of movements without motor decrement may be seen in dystonia or pyramidal slowness and this can be one of the reason for patients with dystonic tremor being diagnosed as PD with normal DAT scans. Secondly, although Unified Parkinson's disease rating scale requires to test rapid alternating movements for 10 movements, this may not be enough to rule out bradykinesia in patients presenting with rest tremor. Ideally the patient should be asked to do 64 movements for 15 - 30 seconds before commenting on the absence of bradykinesia[38]. Finally, even if the clinician believes that tremor is isolated, dopamine transporter imaging should be done to exclude a parkinsonian condition, given that some early tremor-dominant PD patients (monosymptomatic tremor at rest) may exhibit no appreciable bradykinesia or rigidity early in the disease. It is best to label these patients as isolated rest tremor and terms such as SWEDDs and Benign Tremulous Parkinson's Disease should be avoided.

Combined Tremor Syndromes: Combined tremor syndromes include syndromes in which tremor is accompanied by other neurological signs such as parkinsonism, dystonia and cerebellar features. The aetiology of combined tremors is exhaustive. The common combined tremor syndromes are discussed.

Tremor combined with parkinsonism: Tremor combined with parkinsonism (bradykinesia and rigidity) is typically a 4- to 7-Hz rest tremor of the hand ("pill-rolling" tremor), lower limb, jaw, tongue, or foot. This is called classic parkinsonian tremor. It is noteworthy that other types of tremor may coexist in patients with parkinsonism, such as postural or kinetic tremor with the same (type 1) or different frequency (type 2) as the rest tremor[1]. Besides bradykinesia, other features that point to PD as the cause of tremor are: cessation or marked dampening of tremor on voluntary movement and its re-emergence on cessation of activity and maintenance of posture. Rest tremor and bradykinesia/rigidity in PD may progress independently and with advancing disease, increasing rigidity of the limbs may obscure the tremor. The tremor-dominant PD may be associated with earlier age at onset, less cognitive decline, and slower progression than the type of PD that is dominated by postural instability and gait difficulty (PIGD)[39]. Rest tremor is uncommon in other parkinsonian conditions such as MSA, corticobasal degeneration, and PSP and a postural, kinetic, dystonic or intention tremor may be more common.

Dystonic Tremor syndromes

These syndromes have a combination of tremor and dystonia as the leading neurological signs. When tremor occurs in a body part that is dystonic – it is referred to as

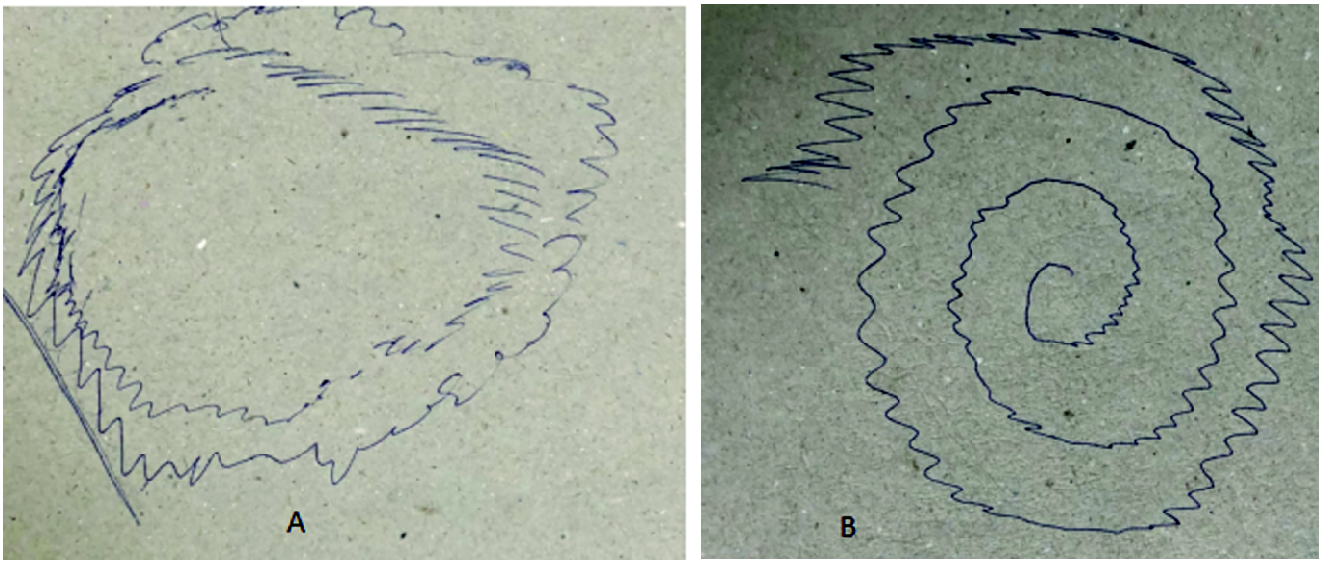

Fig. 2: *Spiral drawings in Dystonic tremor and essential tremor (A) Dystonic Tremor: The axis of tremor is variable and in essential tremor. (B) The axis remains the same throughout of the spiral.*

dystonic tremor, e.g., head tremor seen in cervical dystonia or segmental tremulous dystonia affecting the head and upper limbs. Tremor in dystonia manifests during posture or voluntary movements (action tremor), even though some dystonic patients may have tremor at rest. It is frequently unilateral,in patients with bilateral tremor it is often asymmetric and may be jerky rather than rhythmic[7]. Dystonic tremor may be relieved by sensory tricks (*geste antagoniste*); allowing the abnormal dystonic posture to develop without resistance ("null point") and is worsened by an attempt to maintain certain postures, which may account for the position sensitivity of dystonic tremor. If dystonia and tremor are found in different body parts, this is called tremor associated with dystonia[3], for example tremor in upper limbs in a patient with cervical dystonia. There is an additional group of patients, who have an asymmetric jerky tremor that has features of dystonic tremor mentioned above, but do not have any signs of dystonia anywhere in body. This group of patients may develop dystonia later and probably represent a form fruste of dystonia. Till such time, they develop signs of overt dystonia, it is best to label them as Indeterminate Tremor, thereby avoiding misclassification as ET or premature classification as dystonic tremor. Dystonic tremor may be primary dystonic tremors for example cervical dystonic tremor and hand tremor in primary writer's cramp, genetic [for example anoctamin 3 (ANO3)] or secondary to Parkinsonism, Wilson disease, Neurodegeneration with brain iron accumulation or other neurodegenerative conditions.

A pictorial differentiation from ET is shown in Fig. 3. The clinical pointers to help differentiate ET, PD and Dystonic tremors are summarised in Table IV.

Intention tremor syndromes

Intention tremor syndromes consist of intention tremor at < 5 Hz, with or without other localising signs usually signifying a lesion in the cerebellothalamic pathway. Intention tremor is characterised by usually side-to-side movements perpendicular to the line of travel, predominantly proximal more than distal and amplitude of oscillations increases toward the end of movement[4]. Tremor needs to be differentiated from irregular movements that occur in the line of the travel which are due to dysmetria. Cerebellar intention tremor usually is of low frequency, around 2 - 4 Hz[40].

Holmes Tremor

Holmes tremor is generally a unilateral tremor that has three components: rest, postural and kinetic intention tremor with the relative severity generally being such that kinetic tremor is greater than postural tremor, which is greater than rest tremor. There is both a proximal and distal component and the frequency is usually < 5 Hz[41]. Patients may have other neurologic signs including mild dystonia, oculomotor abnormalities, hemiparesis or ataxia. Common causes include cerebrovascular accident and multiple sclerosis, with a possible delay of 2 weeks to 2 years[42] in tremor onset and the occurrence of lesions. The anatomical localization is in the brainstem in the vicinity of red nucleus causing disruption of both nigrostriatal dopaminergic and cerebellothalamic systems causing the combination of a parkinsonian rest tremor and a cerebellar intention tremor.

Functional Tremor

Functional tremor is often present at rest, on posture and during action, something that is unusual in organic tremor other than seen in Holmes Tremor. Onset is usually abrupt with maximal disability at onset followed by a static or fluctuating course. The key features in differentiating functional tremor from organic tremor are: entrainment (ability of the examiner to alter the rhythm of the patient's tremor by having it match the rhythm of a tremor the examiner produces); exacerbation of tremor on weight loading; pause in tremor with a ballistic movement; distractibility and suggestibility[4].

An algorithm for clinical evaluation of tremors is depicted in Figs. 1 and 2.

Conclusion

Although tremor is the most common movement disorder, its diagnosis is often challenging. The approach to tremor involves a history and careful neurologic examination, focused on the nuances of clinical phenomenology and careful examination of associated findings such as bradykinesia, dystonia and ataxia. There is significant overlap between the common tremor syndromes and the exact nosology of various syndromes such as isolated rest tremor and tremor associated with dystonia is still not clear. If the phenomenology of tremor syndrome is not clear, it is best to label it as Indeterminate Tremor and keep the patient in observation.

References

1. Gövert F, Deuschl G. Tremor entities and their classification: an update. *Curr Opin Neurol* 2015; 28 (4): 393-9.
2. Puschmann A, Wszolek Z. Diagnosis and Treatment of Common Forms of Tremor. *Semin Neurol* 2011; 31 (01): 065-77.
3. Consensus Statement on the classification of tremors. from the task force on tremor of the International Parkinson and Movement Disorder Society. *Mov Disord* 2017; 33 (1): 75-87.
4. Bhidayasiri R. Differential diagnosis of common tremor syndromes. *Postgrad Med J* 2005; 81 (962): 756-62.
5. Edwards MJ, Deuschl G. Tremor Syndromes. *Contin Lifelong Learn Neurol*. 2013; 19: 1213-24.
6. Raethjen J, Austermann K, Witt K *et al*. Provocation of Parkinsonian tremor: Parkinsonian Tremor Provocation. *Mov Disord* 2008; 23 (7): 1019-23.
7. Louis ED. Diagnosis and Management of Tremor. *Continuum (Minneap Minn)* 2016; 22 (4): 1143-58.
8. Elble R, Comella C, Fahn S *et al*. Reliability of a new scale for essential tremor. *Mov Disord Off J Mov Disord Soc* 2012; 27 (12): 1567-9.
9. Louis ED, Dogu O. Does Age of Onset in Essential Tremor Have a Bimodal Distribution? Data from a Tertiary Referral Setting and a Population-Based Study. *Neuroepidemiology* 2008; 29 (3-4): 208-12.
10. Louis ED, Ford B, Pullman SL. Prevalence of asymptomatic tremor in relatives of patients with essential tremor. *Arch Neurol* 1997; 54 (2): 197-200.
11. Leegwater-Kim J, Louis ED, Pullman SL *et al*. Intention tremor of the head in patients with essential tremor. *Mov Disord Off J Mov Disord Soc* 2006; 21 (11): 2001-5.
12. Louis ED. Twelve clinical pearls to help distinguish essential tremor from other tremors. *Expert Rev Neurother* 2014; 14 (9): 1057-65.
13. Poston KL, Rios E, Louis ED. Action Tremor of the Legs in Essential Tremor. *Parkinsonism Relat Disord* 2009; 15 (8): 602-5.
14. Alty J, Cosgrove J, Thorpe D. How to use pen and paper tasks to aid tremor diagnosis in the clinic. *Pract Neurol* 2017; 17 (6): 456-63.
15. Cohen O, Pullman S, Jurewicz E. Rest tremor in patients with essential tremor: prevalence, clinical correlates, and electrophysiologic characteristics. *Arch Neurol* 2003; 60 (3): 405-10.
16. Papengut F, Raethjen J, Binder A. Rest tremor suppression may separate essential from parkinsonian rest tremor. *Parkinsonism Relat Disord* 2013; 19 (7): 693-7.
17. Elble RJ. Diagnostic criteria for essential tremor and differential diagnosis. *Neurology* 2000; 54 (11 Suppl 4): S2-6.
18. Shah B, Mall S, Chhetri R *et al*. A Review of Clinical Approach to a Patient with Tremor Disorder. *J Alzheimer's Dis Park [Internet]* 2017 [cited 2018 Sep 1];7(5). Available from: https://www.omicsonline.org/open-access/a-review-of-clinical-approach-to-a-patient-with-tremor-disorder-2161-0460-1000386-94699.html
19. Louis ED, Dogu O. Isolated head tremor: part of the clinical spectrum of essential tremor? Data from population-based and clinic-based case samples. *Mov Disord Off J Mov Disord Soc* 2009; 24 (15): 2281-5.
20. Louis ED, Ford B, Frucht S. Factors associated with increased risk of head tremor in essential tremor: a community-based study in northern Manhattan. *Mov Disord Off J Mov Disord Soc* 2003; 18 (4): 432-6.
21. Agnew A, Frucht SJ, Louis ED. Supine head tremor: a clinical comparison of essential tremor and spasmodic torticollis patients. *J Neurol Neurosurg Psychiatry* 2012; 83 (2): 179-81.
22. Patel A, Frucht SJ. Isolated vocal tremor as a focal phenotype of essential tremor: a retrospective case review. *J Clin Mov Disord* 2015; 2 (1): 4.
23. de Moraes BT, de Biase NG. Laryngoscopy evaluation protocol for the differentiation of essential and dystonic voice tremor. *Braz J Otorhinolaryngol* 2016; 82 (1): 88-96.
24. Palla S, Ash MM. Frequency analysis of human jaw tremor at rest. *Arch Oral Biol* 1979; 24 (10-11): 709-18.
25. Gonzalez Alegre P, Kelkar P, Rodnitzky RL. Isolated high-frequency jaw tremor relieved by botulinum toxin injections. *Mov Disord* 2006; 21 (7): 1049-50.
26. Schrag A, Bhatia K, Brown P. An unusual jaw tremor with characteristics of primary orthostatic tremor. *Mov Disord Off J Mov Disord Soc* 1999; 14 (3): 528-30.
27. Zadikoff C, Lang AE, Klein C. The 'essentials' of essential palatal tremor: a reappraisal of the nosology. *Brain* 2006; 129 (4): 832-40.
28. Villeneuve A. The rabbit syndrome. A peculiar extrapyramidal reaction. *Can Psychiatr Assoc J* 1972; 17 (2): Suppl 2: SS69-72.
29. Catena Dell'Osso M, Fagiolini A, Ducci F *et al*. Newer antipsychotics and the rabbit syndrome. *Clin Pract Epidemiol Ment Health CP EMH* 2007; 3: 6.
30. Rana AQ, Vaid HM. A Review of Primary Writing Tremor. *Int J*

Neurosci 2012; 122 (3): 114-8.

31. Heilman KM. Orthostatic tremor. *Arch Neurol* 1984; 41 (8): 880-1.
32. Benito-León J, Domingo-Santos Á. Orthostatic Tremor: An Update on a Rare Entity. *Tremor Hyperkinetic Mov [Internet]* 2016 Sep 22 [cited 2018 Aug 30];6. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5039949.
33. Deuschl G, Bain P, Brin M. Consensus statement of the Movement Disorder Society on Tremor. *Ad Hoc Scientific Committee Mov Disord Off J Mov Disord Soc* 1998; 13 (Suppl 3): 2-23.
34. Gerschlager W, Münchau A, Katzenschlager R *et al*. Natural history and syndromic associations of orthostatic tremor: a review of 41 patients. *Mov Disord Off J Mov Disord Soc* 2004; 19 (7): 788-95.
35. Thomas A, Bonanni L, Antonini A *et al*. Dopa-responsive pseudo-orthostatic tremor in parkinsonism. *Mov Disord Off J Mov Disord Soc* 2007; 22 (11): 1652-6.
36. Erro R, Schneider SA, Stamelou M. What do patients with scans without evidence of dopaminergic deficit (SWEDD) have? New evidence and continuing controversies. *J Neurol Neurosurg Psychiatry* 2016; 87 (3): 319-23.
37. Batla A, Erro R, Stamelou M *et al*. Patients with scans without evidence of dopaminergic deficit: A long-term follow-up study: Patients with Swedds. *Mov Disord* 2014; 29 (14): 1820-5.
38. Abdo WF, van de Warrenburg BPC, Burn DJ. The clinical approach to movement disorders. *Nat Rev Neurol* 2010; 6 (1): 29-37.
39. Konno T, Deutschländer A, Heckman MG *et al*. Comparison of clinical features among Parkinson's disease subtypes: A large retrospective study in a single center. *J Neurol Sci* 2018; 386: 39-45.
40. Charles PD, Esper GJ, Davis TJ. Classification of Tremor and Update on Treatment. *Am Fam Physician* 1999; 59 (6): 1565.
41. Raina GB, Cersosimo MG, Folgar SS *et al*. Holmes tremor: Clinical description, lesion localisation, and treatment in a series of 29 cases. *Neurology* 2016; 86 (10): 931-8.
42. Bhidayasiri R, Tarsy D. Holmes' Midbrain Tremor. In: Bhidayasiri R, Tarsy D, editors. Movement Disorders: A Video Atlas: A Video Atlas [Internet]. Totowa, NJ: Humana Press; 2012 [cited 2018 Aug 31]. p. 56-8. (Current Clinical Neurology). Available from: https://doi.org/10.1007/978-1-60327-426-5_27.
43. Fahn S, Jankovic J, Hallett M. Principles and Practice of Movement Disorders E-Book. *Elsevier Health Sciences* 2011; p.33-5.
44. Sternberg EJ, Alcalay RN, Levy OA. Postural and intention tremors: a detailed clinical study of essential tremor vs. Parkinson's disease. *Front Neurol* 2013; 4: 51.

Mrs. Uma Bansal – Prof. B.C. Bansal Best Paper Award (Journal-2022)

1st Prize for Best Original Article – " Comparison of Non-Invasive Scoring Systems with Ultrasound and Liver Elastography in Predicting Non-Alcoholic Fatty Diseases in Health Population" – Dr Kartik Balankhe, Dr Rishabh Ramu Nayak, Dr Rajesh Kumar Modi, Dr Pulin Kumar Gupta, Department of Medicine, ABVIMS and Dr RML Hospital, Baba Kharak Singh Marg, New Delhi - 110 001.

2nd Prize for Best Review Article – "Rickets in Renal Tubular Acidosis: A Clinical Appraisal" Dr Chhavi Agrawal, Dr Partha Pratim Chakraborty, Department of Endocrinology and Metabolism, Medical College and Hospital Kolkata, 88, College Street, Kolkata - 700 073 (WB).

3rd Prize for Best Case Report – "Covid-19- Retated Multisystem Inflammatory Syndrome in Adults; An Uncommon Case" – Dr Ashok Kumar Aggarwal, Dr BM Singh Lamba, Dr Vasudha Kumari, Dr Atul Kaushik, Department of Medicine, SMS & Sharda Hospital, Sharda University, Greater Noida - 201 308 (UP).