Tremor in Childhood

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Tremor in childhood is not commonly described in the literature; but it is also likely underappreciated. The etiology of childhood tremor encompasses a wide variety of pathologic processes. Tremor may occur in isolation, or in association with other neurologic findings or systemic disorders. This article aims to provide an overview of tremorogenic mechanisms with respect to neuroanatomy and neurophysiology, particularly as they relate to children. Classification of tremors, diagnostic entities in childhood, and treatment will also be discussed. With improved recognition and characterization of childhood tremors, we may gain a better understanding of the pathophysiology of the disease and determine more age-appropriate treatment strategies.

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Tremor

Tremor is a rhythmic oscillatory movement resulting from the action of antagonist muscles, affecting one or more limbs. Such oscillations may be the result of normal or accentuated muscular and postural processes; but more often, visible tremor is a sign of a pathologic process. Although not commonly described in childhood,1,2 tremor is likely underappreciated, especially in the context of childhood neurologic diseases. Children with tremor comprise 10%-20% of those with pediatric movement disorders.3 The etiology of tremor in childhood encompasses a wide variety of pathologic processes. Tremor may occur in isolation or in association with other neurologic findings or systemic disorders. The goal of this article is to provide an overview of tremorogenic mechanisms with respect to neuroanatomy and neurophysiology, particularly as they relate to children. Classification of tremors, diagnostic entities in childhood, and treatment will also be discussed.

Neurophysiology of Tremor

Maintenance of body position or of a limb in space requires a balance between agonist and antagonist muscles, with a steady flow of information both to and from the particular muscle groups of interest. Similar processes occur during the execution of both skilled and reflexive movements. Sensory afferents provide proprioceptive input, as well as ongoing information relating to muscle stretch. These sensory pathways are integrated with higher levels of the motor system to allow for accurate and precise execution of motor activities. Disruption or disturbance of movement can be the result of abnormalities affecting hind-brain, midbrain, and forebrain structures. Therefore, it is important to examine all levels of the neuraxis to obtain a complete understanding of the anatomic and physiological origins of tremor.

Oscillatory activities of antagonistic muscles may be produced by peripheral and/or centrally mediated processes.4-6 There is an inherent instability when a limb is maintained against gravity. This instability may be exaggerated by cardiac activity and is exquisitely sensitive to the weight of the limb.7 Using sensitive methods of detection, oscillatory behavior with a frequency of 20 Hz can be detected in the finger, with correspondingly lower frequencies in the hand, forearm, and shoulder.8 To underscore the peripheral origin of this activity, it has been shown that the tremor frequency will decrease with loading of the limb. Such a phenomenon is the chief characteristic of physiological tremor.

Reflex activity involved in maintenance of posture may also give rise to tremor.9 Muscle stretch receptors provide information regarding muscle length and tension, allowing for fine adjustments of posture or limb position. Under certain conditions, an increase in reflex gain may result in a perturbation manifesting as tremor. Such augmentation appears under the influence of epinephrine and excess thyroid hormone.

Tremor can also result from the action of groups of neurons that manifest rhythmic discharges. The thalamus, infe-
rhythmic myoclonias that clinically appear oscillatory. An example of this would be the motor disturbance seen in opsoclonus-myoclonus. Finally, psychogenic tremor, although often a diagnosis of exclusion, manifests with a particular set of features, including the following: sudden onsets and remissions; unusual combinations of rest and intention tremors that vary in amplitude, frequency, and distribution; and historical or neurologic examination findings that suggest a functional origin. Examples of each of these tremor types will be discussed later in the text.

**Evaluation of the Child with Tremor**

When assessing a tremor, older children and adolescents are usually able to cooperate with a neurologic examination similar to that of adults. It is important to determine the distribution of tremor with respect to which body parts are involved, and to characterize stimuli or situations, which either amplify or ameliorate the tremor. Finally, ascertainment of the presence of other neurologic signs and symptoms, particularly other movement disorders, is of major importance, as this may help identify specific disorders of which tremor is only one component.

During clinical examination, maneuvers may be used to elicit tremor and to illustrate any impairments of function. Maintenance of the affected limb(s) in a particular position will help to identify postural tremor. For example, an effective maneuver for the examination of the hands is to have the child flex the elbows while abducting the arms, simultaneously holding the fingertips in near apposition. Asking the child to bring the fingertips as close together as possible, without touching, will often elicit wrist and finger tremor that is easily observable. Simple finger-to-nose testing can elicit action tremor in the arms and hands, as can toe-to-finger pointing in the legs and feet. Such activities as drinking from a full cup, eating cereal with a spoon, or threading beads on a string are easily accomplished during the examination with a willing child. Additionally, handwriting and drawing an Archimedes spiral are often illustrative.

For younger children and those with cognitive impairments, some of these procedures may not be appropriate because of lack of cooperation or failure to have achieved the necessary motor skills. In these instances, the examiner must endeavor to observe the child performing activities that have been observed by the family or caregivers, as these same behaviors are, in all likelihood, the reason for neurologic consultation. Building towers with cubes, tracing a simple figure with a crayon or pencil, or even drawing a path through a maze will provide clear evidence of the presence of a tremor, depending on the developmental level of the child. If, for some reason, a child’s tremor is not adequately observed, the clinician can have the family record a video of the child at home or play setting, performing the activities in which the tremor is most obvious.

As part of the formal characterization of childhood tremor, the examiner must assess the presence or absence of rest, postural, and kinetic components. Furthermore, kinetic tremor...
should be determined to be of either an action or intention type. Finally, an attempt should be made to ascertain the frequency and amplitude of tremor. Conventional terms for frequency include slow (<4 Hz), intermediate (4-7 Hz), and fast (>7 Hz) tremors. Amplitude (large, intermediate, or small) tends to vary inversely with frequency. There is also a developmental component to consider, as physiological tremor frequencies have been shown to vary proportionally with age. At the age of 2, the frequency is approximately 3 Hz and increases by about 0.25 Hz per year. Precise characterization of frequency should rely on more quantitative methods, such as surface electromyography. However, in practice, clinical approximation of tremor frequency is often adequate for characterization.

**Etiology of Childhood Tremor**

There is a wide range of conditions that may include tremor as part of the clinical phenotype. Table 1 shows a list of some of the scenarios in which tremor can appear. The following sections will examine the more common clinical entities.

**Essential Tremor**

Considered the most common movement disorder in adults, childhood essential tremor (ET) has not engendered a significant amount of published clinical articles. With the exception of the reports of Paulson and Louis et al., few reports in the literature have addressed the pediatric presentation of ET. This is somewhat remarkable, considering that 50% of individuals diagnosed with ET report an onset during childhood. This can probably be explained by the minimal impact of the tremor on function until adulthood. However, ET is not uncommon in a pediatric movement disorder clinic, and familiarity with this entity is of value to neurologists caring for children.

ET was characterized as a hereditary action tremor by Dana. In the 19th century, the descriptor “essential” was applied to any condition that was felt to be idiopathic or intrinsic to an individual. Because ET was typically an isolated condition with only an occasional tendency to progress, and was determined to segregate in families, it was also described as benign familial tremor. ET is inherited in an autosomal dominant fashion, but with variability of expression within families. However, the penetrance is believed to approach 100% after age 60. The genetics of ET remain elusive but at least 3 different loci have been identified for familial ET, which are EMT1, EMT2, and a locus on 6p23. The EMT1 locus located at 3q13 was identified in a large Icelandic pedigree. Within this locus, the DRD3 gene codes for dopamine D3 receptor. A Ser9Gly variant of this gene has been found to be associated with an increased risk of ET. The EMT2 locus on chromosome 2p22-25 has also been identified in multiple studies of familial and sporadic ET in American and North American patients.

Large epidemiologic studies of ET have been reported, with some refinement of the clinical features as well as variant presentations. Consensus diagnostic criteria have been adopted, and consist of the following: (1) a monosymptomatic disorder characterized as tremor alone, typically with a frequency of 4-11 Hz, and (2) distribution most commonly in the hands and arms or head alone. Exclusionary criteria include presence of other neurologic findings suggestive of another process; (2) an atypical course, or one that is relatively static or slowly progressive; (3) a history of exposure to tremorogenic drugs or toxins; and (4) tremor that is

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**Psychogenic**

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orthostatic or occupational, or isolated to the tongue, chin, voice, or leg. Other features of ET that aid in the diagnosis include ethanol responsiveness and a positive family history. In over half of adult ET patients, they report a significant improvement in symptoms after a moderate intake of alcohol. Obviously, this is a feature that is seldom observed in children, but pertinent family history of ethanol responsiveness may be elicited.

Studies to examine the pathophysiology of ET indicate the cerebellum as the likely anatomic site of the disorder. Although gross brain pathology in ET is normal\(^\text{38,39}\) in vivo imaging has been performed in a number of studies. Functional magnetic resonance imaging (fMRI) has shown increased neuronal activity in cerebellum, red nucleus, and globus pallidus contralateral to the affected limb in ET.\(^\text{40}\) Similarly, positron emission tomography\(^\text{41,42}\) in patients with ET show an increase in activity in the cerebellum with intention or action tremor, and further increases in the thalamus and basal ganglia with postural tremor compared to those in controls. Finally, two studies of magnetic resonance spectroscopy have been reported in ET,\(^\text{13,43}\) both demonstrating altered neuronal metabolism in the cerebellum.

There is some controversy regarding ET, in that some authors consider the disorder to be monosymptomatic\(^\text{44,45}\) whereas others emphasize the possibility of comorbid features.\(^\text{46}\) ET usually manifests as an action tremor but has been described with associated rest tremor in almost 20\% of adults.\(^\text{46}\) Other conditions described with ET include buccofacial dyskinesias and myoclonus,\(^\text{47}\) and other tremors may be misdiagnosed as ET.\(^\text{46}\) To better clarify this issue, the Committee for the Classification of Tremor has purposed the entity of indeterminate ET, or a tremor with the typical appearance of ET but in the context of some other neurologic disorder or syndrome. This type of tremor may be quite common in children, but the relative frequency compared to that of ET is not known.

In childhood, the usual presentation of ET is that of a postural and action tremor of insidious onset and prolonged duration. The frequency of the tremor has been reported to vary with age and limb position,\(^\text{49}\) but the tremor is typically between 5 and 9 Hz. The distribution is overwhelmingly centered on the hands, with a minority manifesting head tremor on examination. Voice tremor was not reported in either of the 2 series of patients with childhood ET. If a careful history is obtained, family members may recall tremor from infancy, although this type of retrospective is of uncertain reliability. In terms of age of onset, the series of Louis et al\(^\text{50}\) reported ET to begin at 6.8 years, but this is solely reliant on parental history. Of note, however, is that referral for evaluation seems to cluster around 2 epochs of childhood. Some children present to the neurologist in the latter half of the first decade, and in all cases this is because of the perception of the tremor by a parent, teacher, or the child’s physician. In these cases, the children are seldom disabled or otherwise adversely affected by the tremor. Although the young child with ET will characteristically manifest a tremor when drinking from a full cup or will have poor handwriting, it is unusual for the complaint to include impairments in other activities.

Another group of children present in their teens, and in these patients, the chief complaint often centers on similar activities to that of younger children, but the perceived impairment is considerably greater. In these patients, they often relate a longstanding history of tremor but indicate that the tremor is more obvious in social or occupational settings. Notably, the tremor severity parallels emotional state, so it is more apparent when the child is nervous, upset, or anxious. It is difficult to gauge whether the progression of tremor is truly a worsening or a subjective perception of deterioration. This is of importance from various perspectives. Foremost among these is that a progressive tremor may actually indicate the likelihood of some other diagnosis than that of ET, as in adult-onset ET the course of progression is quite long. Another issue is whether childhood ET is a manifestation of genetic anticipation, an issue of some relevance to what is known about trinucleotide repeat disorders. In published studies, there is no indication of genetic anticipation in ET, but few studies have addressed this issue. Finally, a common concern among families who have a child with ET is the long-term prognosis. Although it may be presumed that duration of tremor would correlate with ultimate severity and disability, this has not been thoroughly studied.

The differential diagnosis of ET in childhood is not particularly lengthy, especially if consensus criteria are met. When confronted with a child exhibiting a postural or action tremor that primarily involves the hands and head, of long duration and minimal impairment, with no other concomitant neurologic findings, the likelihood of other disorders becomes low. By definition, historical information regarding exposure to tremorgenic agents, such as anticonvulsants and \(\beta\)-adrenergic agonists should be obtained, and the presence of signs of hyperthyroidism should be determined. Wilson disease (see later in text) has been reported to present initially with tremor, but rest tremor is more typical. In short, an otherwise normal child or adolescent with hand tremor as the sole manifestation is probably suffering from ET.

However, there are no published studies of treatment of childhood ET. Keeping in mind that younger children typically have minimal impairment, it should not be a common practice to consider pharmacologic intervention in preadolescents. Rather, specific remedies or adaptations should be sought to address the particular problems experienced by these children. As mentioned above, handwriting and some fine motor tasks are impaired. Interventions such as weighting of the wrists\(^\text{50}\) or an evaluation by an occupational therapist to apply adaptive strategies for fine motor problems can be quite beneficial.

On the other hand, adolescents who present with ET have usually accommodated to their condition for some years; and by the time they present to a neurologist, a common interest is the desire to minimize the tremor. This often necessitates medication therapy, and it is appropriate to approach treatment similar to that in adults. First-line medications have included primidone and propranolol, both of which have demonstrated efficacy in studies performed on adults.\(^\text{31-34}\)
Because of problems with primidone availability, propranolol or other β blockers have been considered the first-line therapy for childhood ET. In adolescents, dosage guidelines would be identical to those for adults. A starting dose of 30 mg once a day, then changing to 30 mg twice a day, is often helpful in ameliorating hand tremor. Care should be taken to ascertain whether a patient suffers from reactive airway disease, as β blockers are contraindicated. Many childhood ET patients will respond to doses in the 60–80 mg range, and long-acting preparations (Inderal, LA) are taken once per day to promote compliance. Other β blockers (atenolol, timolol, etc.) can be tried, as they have proved to be successful in some cases.

Occasionally, because of lack of efficacy, side effects, or both, other therapeutic options may be necessary. In instances of episodic aggravation of ET when, for example, there is a particular situation that provokes tremor, short-term treatment with a benzodiazepine, such as clonazepam or lorazepam, can be quite helpful. Adolescents who can tolerate clonazepam or lorazepam can be given doses of 0.5 to 1 mg per day. In adolescents who are sensitive, clonazepam can be given in divided doses of 0.25 mg, and lorazepam can be given in divided doses of 0.5 mg.

In summary, ET in childhood has many of the same features of adult-onset ET. Indeed, the clinical phenotype and response to therapy are identical. It is not known, however, what the prevalence of childhood ET is, nor is it known whether childhood onset ET is a particularly severe form of ET. These questions will require inclusion of children in epidemiologic ET studies.

### Tremor in Association With Other Conditions

According to consensus guidelines, indeterminate ET is tremor that has the clinical appearance of ET but is not an isolated neurologic finding, or occurring in the context of another neurologic disorder. The most obvious example of this entity has been described in carriers of the Fragile X (FRAX) premutation, caused by an expansion of untranslated CGG trinucleotide repeats.68–72 The clinical presentation of tremor is usually in adulthood, consisting primarily of an action or intention tremor. Because FRAX premutations are common in the general population, it has been postulated that some cases of ET are actually caused by CGG expansions. Interestingly, another X-linked mental retardation syndrome with action tremor has been reported,73 additionally characterized by short stature, hypogonadism, muscle wasting, and localizing to Xq24–q25. Other genetic conditions in which tremor has been described include 47, XYY syndrome and mutations of the MECP-2 gene.78,79 An ET-like tremor has also been described in a patient with X-linked Charcot-Marie-Tooth disease.58 One family has been reported with hemiplegic migraine, nystagmus, and tremor with onset in childhood, and is presumed to be a form of multisystem degeneration.80 Another family has also been described with a distal amyotrophy, akin to Charcot-Marie-Tooth disease, progressive ataxia, and tremor.81 Whether either of these families represents a distinct nosologic entity is unclear.

Action and/or postural tremor have been described as part of the presentation of a variety of illnesses affecting all parts of the neuraxis. As an example, Dawood and Moosa studied hand tremor in spinal muscular atrophy, correlating their clinical observations with an “ECG tremor.” They describe a coarse tremor corresponding to the presence of muscle potentials on limb leads of the electrocardiogram. This may actually represent polyminimyoclonus, but further study is needed. Peripheral nerve injury may cause tremor in the context of complex regional pain syndrome,83,84 and it has been described as both a postural and a rest tremor. Tremor because of stroke occurs in childhood,85–87 similar to what has been described in adults. In children, cysts in the thalamus and pineal gland have also been reported to present with tremor.88,89 Mitochondrial encephalopathies can at times manifest as movement disorders. Macaya et al reported 22 of 34 patients with Leigh syndrome who had evidence of a movement disorder. Although dystonia and rigidity were the most common, 2 of these patients exhibited tremor. Frequent traumatic brain injury is a cause of tremor, particularly in the convalescent stages up to 18 months after the event.82 Fortunately, it has been reported to resolve spontaneously in approximately half of patients.

Tremor as a component of other movement disorders has been described in numerous conditions. Juvenile onset occurs in fewer than 10% of patients with Huntington disease and is the consequence of a marked expansion of a CAG trinucleotide repeat motif in the translated 5’ end of the huntingtin gene. The clinical phenotype of this condition differs from the adult-onset variety, in that there is a preponderance of parkinsonism and rigidity.90,91 Tremor, when it occurs in this context, is most often during rest. On the other hand, one family with benign hereditary chorea has been reported in association with intention tremor.92 Primary parkinsonism, juvenile Parkinson disease, is rare in childhood and usually presents with dystonia, Bradykinesia, and rigidity. In contrast to the adult form of the disease, it rarely involves tremor.93 Another basal ganglia disorder that may present with a prominent tremor is that of Wilson disease, or hepatolenticular degeneration. In children, this disorder of
copper metabolism presents primarily as a disturbance of hepatic function, although there are rare reports of isolated neurologic symptoms.\textsuperscript{96} Intention tremor as an initial finding of Wilson disease was seen in 1 patient in the study by Slovis et al.\textsuperscript{97} Tongue tremor with dysarthria has also been reported.\textsuperscript{97,98} Dystonia is another movement disorder that has been reported in association with tremor. In the so-called dystonic tremor, most patients suffer from cervical dystonia. Large series by Jankovic\textsuperscript{99} and Rondot\textsuperscript{100} indicate a prevalence of tremor of 71\% and 21\%, respectively. In Jankovic’s series, however, most of the tremors involved the head and neck, whereas 27\% had tremor identical to ET. The possible link between focal or segmental dystonia and tremor has also been illustrated by one report of torsion dystonia and writing tremor in a family,\textsuperscript{101} and by the phenomenon of task-specific tremor coexisting with task-specific dystonia.\textsuperscript{102}

As is obvious from the previous discussion, the topic of secondary tremors and indeterminate ET is somewhat broad, especially given the lack of detail with respect to the types of tremor that occur in various disease states. Therefore, a discussion of a treatment algorithm is problematic. In some cases that this author has observed, a secondary tremor or indeterminate ET may resolve on its own or be of minimal severity, not requiring treatment. More commonly, though, empiric trials of agents used to treat tremor, such as primidone, propranolol, clonazepam, and topiramate, have been ineffective in ameliorating the condition. This is an area that is poorly understood, and greater efforts are needed to develop effective therapeutic strategies for these children. In cases of severe, disabling tremor secondary to trauma, surgical therapy has been beneficial,\textsuperscript{103} but moderate gains are offset by significant morbidity.

### Head Tremors in Childhood

The subject of oscillatory movements of the head in childhood requires a separate discussion, as there are several entities presenting in childhood. Head nodding is a relatively slow head movement in a “yes-yes” direction seen during infancy, and is often first noticed in the first few months of life as head control is attained. The frequency of this movement is usually about 2 Hz, and reflects the mechanical and viscoelastic properties of the head and neck.\textsuperscript{104} Nodding may be pathologic in the context of weakness and an inability to maintain the head in a stable position, such as that seen in myopathic states,\textsuperscript{105} or it can be a compensatory phenomenon in relation to congenital nystagmus.\textsuperscript{104,106,107}

Spasmus nutans is another disorder of infancy, typically described as the triad of head tremor (usually in a “no-no” direction), head tilt, and nystagmus.\textsuperscript{108-109} The head tremor is of a variable frequency, and may be vertical, horizontal, or a combination of both. The nystagmus is asymmetric, and (similar to head movement) may be horizontal, vertical, or rotatory. It has been postulated that the head tremor in spasmus nutans is a compensatory response to the nystagmus to aid visual fixation.\textsuperscript{110-112} Similarly, abnormal head movements have been described in congenital nystagmus,\textsuperscript{106,113} but this entity is distinct from spasmus nutans, as the former is usually present from birth, while the latter appears at approximately 4-12 months of age. The constellation of findings in spasmus nutans is certainly indicative of a process involving posterior fossa structures or at least a central process. Indeed, both infra- and supratentorial lesions have been described in children presenting with spasmus nutans, and cranial imaging studies are warranted.\textsuperscript{114-118} However, in the absence of a discernible lesion, spasmus nutans resolves within months in most cases.

Finally, some mention is necessary of the “bobble-head doll” syndrome. This head movement is usually in a “yes-yes” direction, with a higher frequency and lower amplitude than head nodding, and may be intermittent. First described as a clinical concomitant of an intraventricular tumor,\textsuperscript{119} later reports indicate that the findings may also be seen in the context of hydrocephalus as well as other supratentorial lesions.\textsuperscript{120-126} Because of the association with tumors of the central nervous system, there may be other associated findings, including gait abnormalities, incoordination, oculomotor disturbances, and even behavioral changes.\textsuperscript{127} Should there be an effective treatment of the underlying cause, the head tremor usually resolves.\textsuperscript{124,128,126}

### Tremor Related to Drugs

There is a long list of agents that can produce tremor as a side effect.\textsuperscript{3} Drugs that modify dopaminergic neurotransmission, such as neuroleptics, typically produce a parkinsonian rest tremor. Action or postural tremors can also be caused by medication exposure, and there are numerous drugs that are frequently used in childhood, which should be taken into consideration. Anticonvulsants are commonly prescribed in childhood epilepsy, and valproic acid has been reported to cause an action tremor in 15\% of patients.\textsuperscript{129} Other antiepileptics associated with childhood tremor include phenytoin, 14\% and carbamazepine, 1\%. Lamotrigine overdose has been reported to manifest in part with a generalized tremor of short duration.\textsuperscript{130} Adult studies indicate that tremor occurs in 9\% of patients taking tiagabine and 12.5\% of those taking gabapentin. Seldom, though, is anticonvulsant-induced tremor a cause for cessation of the medication, nor is it a significant contributor to disability.

Another common childhood condition is reactive airways disease, commonly treated with \(\beta\)-adrenergic agents, corticosteroids, or a combination of the two agents. \(\beta\)-adrenergic agents are particularly prone to cause tremor,\textsuperscript{131-133} but corticosteroids are rarely reported to be associated with clinically significant tremor.\textsuperscript{134}

Finally, bone marrow and solid organ transplantation have become more common in the pediatric age group, resulting in a population that is chronically immunocompromised. Although there are few studies characterizing the neurologic complications of pediatric cancer patients,\textsuperscript{135} tremor has been described in approximately 1\%. Solid tumors in brain can cause tremor\textsuperscript{136} but are rare. In the context of a pediatric oncology patient, the cause of tremor is usually attributed to cyclosporin or FK-506, both of which are used regularly in pediatric transplant medicine.\textsuperscript{137-139}
Metabolic and Toxic Causes of Tremor

Nutritional deficiency has long been known to present with tremor, particularly vitamin B12. Periodic case reports of infants diagnosed with B12 deficiency describe an action tremor that appears after treatment of the deficiency has begun. In developed countries, an infant with megaloblastic anemia typically suffers from an isolated lack of B12; but in underdeveloped nations, the context is one of a more pervasive nutritional impairment. "Infantile tremor syndrome," reported primarily in India, is described as a fine "shivering" of high frequency, and a predominantly generalized distribution. The clinical context is one of the concomitant nutritional deficiencies, and later authors have considered the condition to be a manifestation of B12 deficiency and perhaps other vitamins or minerals. An apparently similar entity, "kwashiorkor," has also been described in Africa and the Caribbean in children who have undergone correction of severe malnutrition.

Metabolic disorders may present with a variety of neurologic symptoms, including movement disorders and tremor. In a study from Spain, 10%-30% of patients with phenylketonuria developed kinetic or positional tremors. Pyruvate carboxylase deficiency and homocystinuria have also been associated with tremor. In most metabolic disorders, the tremor presents in a constellation with other symptoms and is not the sole presenting complaint.

Various intoxications with heavy metals may cause tremor, but usually as part of a more generalized parkinsonian presentation. Mercury poisoning, however, can result in a coarse action or postural tremor, as can intoxication with diethyltoluamide, commonly found in insect repellants.

Benign Tremors of Childhood

In the immediate postpartum period, concerns are often raised regarding excessive tremulous behavior. Usually referred to as "jitteriness," the tremor involves the entire body, and is state-sensitive, becoming more prominent with handling or distress. Studies examining the prevalence and course of jitteriness indicate that as many as 44% of healthy newborns will manifest at least a "mild" amount of tremulous behavior. The long-term course, though, is relatively benign, with most patients experiencing a resolution within 2-5 months. Futagi has reported a series of 24 patients with neonatal tremor. Although it was not clearly indicated whether these children manifested jittery behavior, most patients had a benign outcome with the exception of those who had sustained some type of perinatal insult.

Another benign tremor of childhood is that of shuddering or distending or distress. Studies examining the prevalence and consistency of amplitude, frequency, and distribution of affected limbs or muscles should certainly raise suspicions of a conversion disorder. Unusual combinations of rest, postural, or intention tremor are common, and in most cases marked attenuation of tremor with distraction may be observed. The clinician who suspects a psychogenic tremor should attempt to ascertain the presence of recent stressors, behavior changes, or other concomitants that are often associated with this disorder.

Psychogenic Tremor

Conversion disorders are well described in pediatric patients, not uncommonly presenting with neurologic complaints. Tremor as a conversion symptom has been described in adults, and rarely in childhood. Fortunately, psychogenic tremor is fairly easy to distinguish from other tremor types, precisely because the clinical features of tremor do not correspond to those seen as part of pathologic processes. No adequate information exists regarding the prevalence or typical clinical presentation of psychogenic tremor in childhood. However, tremor of sudden onset with inconsistent amplitude, frequency, and distribution of affected limbs or muscles should certainly raise suspicions of a conversion disorder. Unusual combinations of rest, postural, or intention tremor are common, and in most cases marked attenuation of tremor with distraction may be observed. The clinician who suspects a psychogenic tremor should attempt to ascertain the presence of recent stressors, behavior changes, or other concomitants that are often associated with this disorder.

Conclusions

Clinically, tremor in childhood has many of the same features as in adults. In indeterminate or symptomatic tremors, the underlying causes may be particular to childhood diseases, but the phenomenology of tremor remains indistinct through the lifespan. Despite these similarities, however, a great deal remains to be ascertained regarding tremor in childhood that could shed light not only on brain development but on disease mechanisms. For example, although there have been investigations of physiological tremor in the young, recent assessments of pathologic states have focused on age-related changes in mature individuals. Because physiological tremor frequencies change with maturation into adulthood, the contribution of this dynamic process to the manifestation of pathologic tremors could possibly play a role in tremor expression and disability. Furthermore, there may be an impact on treatment strategies as well. However, comprehensive investigations into childhood pathologic tremors are lacking, hence, inadequate understanding and treatment of these conditions.

In the future, a better recognition of childhood tremor will be needed, with appropriate classifications as well. As better identification and clarification of tremor occurs, we can begin to develop age-appropriate management strategies and take advantage of the progress being made in adult tremor disorders.
References

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