

Supplemental Material Online: Additional Text and References

Unusual Tremor Syndromes: Know in Order to Recognise

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Search criteria

The review comprised of a comprehensive online search to identify the available literature of case reports and articles for each tremor syndrome not listed in Table 1. We searched Google Scholar, PubMed and Ovid databases with no date restrictions using the keyword: ‘tremor’; articles on the common tremors listed in Table 1 were excluded. An additional literature search was performed using the following keywords: ‘FXTAS’, ‘Fragile X-associated tremor/ataxia syndrome’, ‘spinocerebellar ataxia’, ‘autosomal recessive cerebellar ataxia and tremor’, ‘myorhythmia’, ‘isolated tongue tremor’, ‘Wilson’s disease and tremor’, ‘slow orthostatic tremor’, ‘peripheral trauma and tremor’, ‘tardive tremor’, ‘rabbit syndrome’, ‘hereditary chin tremor’, ‘familial geniospasm’, ‘essential chin myoclonus’, ‘bilateral high-frequency synchronous discharges’, ‘paroxysmal head tremor’, ‘limb-shaking transient ischaemic attack’, ‘bobble-head doll syndrome’, ‘spasmus nutans’, and ‘shuddering attack’. In addition, supplementation with the reference list from the articles was used as necessary.

Wilson’s disease

The onset is typically in the second or third decade with a predominantly neurological presentation and it has been suggested that patients generally fall into several broad categories with considerable overlap: a dystonic syndrome, action tremor (also known as ‘pseudosclerotic syndrome’), a parkinsonian syndrome, or an ataxic syndrome.[1 2] The most common presenting tremors reported vary but are usually in the upper limb and include postural, action and dystonic tremors with resting tremor occurring less frequently.[2-4] In the early stages, symptoms may be non-specific so clinicians must have a high index of suspicion in young adults presenting with a movement disorder because the prognosis is worse if the diagnosis is delayed.[5] A tremor predominant presentation suggests a more favourable prognosis and better response to treatment with chelation therapy, zinc, or both.[1 6]

In more advanced stages, the classic tremor observed is a postural proximal upper limb, large amplitude 'wing-beating' movement, which shares some similarities with Holmes' tremor.[2]

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Tremor induced by peripheral lesions

There is some limited evidence that peripheral trauma can cause tremor. [1-3] It is often associated with complex regional pain syndrome, paraesthesia or hyperpathia in the affected region. Trauma-induced hand tremor is described following limb or cervical injury (whiplash), although it can spread to involve other body regions including the proximal limb or contralateral side. It is usually a highly variable tremor that is asymmetric, mainly postural-kinetic with a moderate frequency of 5-7 Hz. The tremor may clinically appear like essential, parkinsonian, dystonic, or other neurological tremors.[4] Other movement disorders, such as dystonia or myoclonic-like jerking, may occasionally be associated with the tremor.[1 3] A possible predisposing factor is present in up to 65% of these cases and include a family history of essential tremor or dystonia, previous use of neuroleptics or stimulants, HIV infection, seizure disorder, or mental retardation.[3] The tremor generally starts within a few weeks after trauma, worsening over the first year, then becoming less pronounced but rarely completely resolving.[5] Diagnostic criteria for peripherally-induced tremors were initially proposed by Jankovic *et al.* [6] to include: 1) the trauma has sufficient severity to result in local symptoms lasting at least two

weeks or requiring medical attention within the first two weeks; 2) the tremor is anatomically related to the site of injury; 3) onset of the tremor occurs within one year from the trauma.

The pathophysiology of these tremors is largely unclear and in most cases functional disorders have been the final diagnosis. We believe that before accepting the possible diagnosis of tremor induced by a peripheral lesion, every patient should undergo a detailed electrophysiological assessment of the tremor, which can easily support a diagnosis of functional tremor ("Laboratory-supported definite").[7] Researchers who favour an organic cause propose two predominant theories, either the generation of a peripheral rhythm generator or an enhancement of the normal physiological tremor. The majority of peripheral trauma tremors are refractory to medical therapy, such as propranolol, clonazepam, and carbamazepine, as is the case with functional tremors.[2] Injections of botulinum neurotoxin have shown benefit albeit transiently.[4]

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Table 5

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