

Clinical Algorithms

Tremor

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Tremor is unwanted movement in which rhythmical oscillations occur. This rhythmical character distinguishes tremor from other involuntary movements. When formally analysed most tremors contain a range of frequencies, varying from 1–2 Hz up to 12 Hz or higher. This article considers certain common tremors that interfere with the use of the arms and legs. Clonus, a tremor that is seen most clearly when a muscle is passively stretched, is not considered, and rarer forms of tremor have been described elsewhere.¹

In clinical practice tremors are usually identified by simple clinical examination, without the use of apparatus for recording and analysing their frequencies. The algorithm suggests a scheme for such identification, requiring observation of the tremor at rest, on sustained posture, and on active use of the limb. Postural tremor is usually shown when the patient holds out his arms with the fingers abducted or holds his finger to his nose with the arm abducted at the shoulder. Tremor on active use can be shown if he carries out an everyday task such as picking up and drinking from a glass. On the examination couch tremor is usually shown by the finger to nose or heel to shin tests.

Whatever its cause, tremor is commonly made worse by anxiety. This may lead patients, relatives, and even doctors wrongly to ascribe the tremor to emotional causes. Conversely, successful efforts to reduce anxiety, through reassurance, alcohol, or other sedative drugs, are likely to reduce the severity of tremor regardless of its cause.

Individual tremors and their treatments

Parkinsonian tremor is typically evident when the affected limb is not in use—for example, in the leg when a patient is sitting or lying and in the arm when he lets his arm hang by his side. It is less evident on active use but may persist during sustained posture. At the start tremor may affect one limb or one side alone but in severe cases may be generalised. Frequency is commonly around 4–5 Hz. The diagnosis can usually be confirmed by the presence of other parkinsonian features, especially bradykinesia and the characteristic stooped posture. If the tremor does not appreciably interfere with the use of the limb treatment with drugs may not be necessary. The patient may feel some social embarrassment, but drugs will rarely reduce the tremor enough to prevent this. If important bradykinesia is also present preparations containing levodopa, such as Madopar or Sinemet, are likely to be given but are usually of less benefit to tremor than to bradykinesia. Anticholinergic drugs, such as benzhexol, are worth using for tremor and can readily be combined with levodopa. If tremor is severe and responds inadequately to drugs stereotactic surgery should be considered. Anticholinergic drugs may also help if the parkinsonian tremor is drug induced, but the possibility of reducing or replacing the provoking drug should be considered. Prochlorperazine (Stemetil) is an example of a phenothiazine that can cause parkinsonism. This is commonly forgotten when the drug is used for nausea or vomiting.

*Increased physiological tremor*²—A tremor of the hands of low

amplitude is present in all normal subjects. Frequency is in the range 6–12 Hz, tending to fall with increasing age.³ Increased amplitude can be readily seen if the hands are held out with the fingers abducted. This type of postural tremor is of smaller amplitude than others; hence the distinction between fine and coarse in the algorithm. When possible the underlying cause should be treated. If immediate symptomatic suppression is needed β blockers, such as propranolol, are usually effective.

*Drug related tremor*¹—A tremor comparable to essential tremor occurs in 40% of patients treated with lithium for manic depressive states. A similar tremor is sometimes seen with other drugs, including imipramine and amitriptyline. It is treated by reducing the dose or changing the drug.

Asterixis, or “flapping” tremor, is seen when the hands are outstretched: the posture is broken by a brief movement of flexion at wrist and fingers and is as quickly restored. Other forms of postural tremor may occur in the same circumstances.⁴ Although classically associated with hepatic disease, asterixis may be seen in several metabolic disturbances, including renal and respiratory failure.⁵ Treatment is of the underlying metabolic cause.

Essential tremor is a pure postural tremor of a frequency similar to that of physiological tremor² but of much greater amplitude. It may not develop until late in life (senile tremor) and tends to increase in severity with time. It is commonly hereditary, being inherited as an autosomal dominant condition, but many apparently sporadic cases are also seen. A striking feature, at times of some diagnostic help, is the extent to which it may improve with modest quantities of alcohol. Patients with essential tremor are often misdiagnosed as having Parkinson's disease. This may lead to distress and to treatment with antiparkinsonian drugs, to the disappointment of patient and doctor. If the criteria given in the algorithm are followed this error should seldom occur. Treatment is difficult. Alcohol or other sedation may suppress the tremor in the short term but is probably best reserved for occasions of particular social pressure as continued use may lead to addiction. Primidone has been reported to be helpful,⁶ but my experience has not been encouraging.

Cerebellar action and intention tremors—The classical intention tremor is most clearly seen towards the end of the movements in the finger to nose test. In some patients cerebellar tremors may be more prominent during the middle ranges of the movement and may also persist during sustained posture.^{4,5} These tremors occur, for example, with lesions of the cerebellar pathways in multiple sclerosis, with brain stem vascular lesions, and with tumours. Symptomatic treatment is unsatisfactory. Choline has produced conflicting results.⁵ If possible the underlying cause should be treated.

¹ Rondot P, Jedynek CP, Ferrey G. Pathological tremors: nosological correlates. In: Desmedt JE, ed. *Progress in clinical neurophysiology*. Vol 5. Basel: Karger, 1978: 95–113.

² Marsden CD. The mechanisms of physiological tremor and their significance for pathological tremors. In: Desmedt JE, ed. *Progress in clinical neurophysiology*. Vol 5. Basel: Karger, 1978: 1–16.

³ Marshall J. The effect of ageing upon physiological tremor. *J Neurol Neurosurg Psychiatry* 1961;24:14–7.

⁴ Marshall J. Tremor in Vinken and Bruyn. *Handbook of clinical neurology*. Vol 6. Amsterdam: North-Holland, 1968:809–25.

⁵ Findlay LJ, Gresty MA. Tremor. *Br J Hosp Med* 1981;26:16–32.

⁶ O'Brien MD, Upton AR, Toseland PA. Benign familial tremor treated with primidone. *Br Med J* 1981;282:178–80.

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