Department of Medical Genetics, Mayo Clinic, 200 First Street SW, Rochester, MN 55905).

COMMENT. Examples of IEM requiring additional preliminary tests include Menkes' kinky-hair disease (serum copper and ceruloplasmin), and molybdenum cofactor deficiency (urine sulfite dipstick).

MOVEMENT DISORDERS

CLASSIFICATION OF PAROXYSMAL DYSKINESIAS

Forty six patients, ages ranging 1 to 77 years, with paroxysmal dyskinesias and classified according to precipitating factors and duration of attacks were reported from the Movement Disorder Clinic, Department of Neurology, Baylor College of Medicine, Houston, TX. Paroxysmal kinesigenic dyskinesia (PKD), occurring abruptly after a sudden movement, affected 13 patients; paroxysmal nonkinesigenic dyskinesia (PNKD) occurred spontaneously in 26; exertion-induced attacks (PED) affected 5; and episodes were precipitated only by sleep (PHD) in 1. The etiology was idiopathic in 22 and secondary to psychogenic illness in 9, to stroke in 4, trauma (3), encephalitis (2), multiple sclerosis (2), kernicterus (1), and migraine (1). Short duration (<5 min) and long-lasting attacks (>5 min) were about equal in incidence. None had loss of consciousness or other evidence of seizures and EEGs were normal in 34 tested. MRI was normal in 25 tested. Nine of 10 (90%) patients with PKD improved with medications, mainly carbamazepine, phenytoin, or clonazepam, compared to only 7 of 19 (37%) with PNKD. (Demirkiran M, Jankovic J. Paroxysmal dyskinesias: clinical features and classification. Ann Neurol October 1995;38:571-579). (Respond: Dr Jankovic, Department of Neurology, Baylor College of Medicine, 6550 Fannin, Suite 1801, Houston, TX 77030).

COMMENT. In contrast to the original (Mount and Reback, 1940) and other previous reports which empasized genetic and familial factors in etiology, the majority of the patients in the above series had sporadic and secondary paroxysmal dyskinesias. Menkes JH, in his Textbook of Child Neurology (Lea & Febiger, 1985), provides an excellent account of the various types of paroxysmal dyskinesia, classified according to 1) movement pattern - choreiform, athetoid, dystonic, or tonic; 2) familial or acquired; 3) kinesigenic or nonkinesigenic; 4) acquired etiology - perinatal asphyxia, reflex epilepsy, metabolic disorders (eg. idiopathic hyperparathyroidism), and multiple sclerosis. The specificity of response of the kinesigenic dyskinesias to anticonvulsant drugs, especially phenytoin, has been documented previously. Rare cases of hypnogenic dyskinesia, responding to lorazepam, and one patient with paroxysmal diplopia due to superior oblique myokymia following head injury, responding to carbamazepine, are described in the present series. An overlap between paroxysmal dyskinesia and epilepsy, migraine, and paroxysmal ataxia is discussed. The possible relation between migraine and paroxysmal dyskinesia mentioned in this report has not previously been noted.

HEREDITARY MYOKYMIA AND PAROXYSMAL ATAXIA

A family with autosomal dominant hereditary myokymia and paroxysmal ataxia, linked to chromosome 12p, is described from University