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Is alternating hemiplegia a mitochondrial disease?

The syndrome of alternating hemiplegia of childhood (AHC) was first described in 1971¹ and has usually been thought to be a variant of migraine. A recent report from Paris describes 22 children and is an excellent review of the clinical findings and differential diagnosis.² The cardinal features are: (1) repeated episodes of hemiplegia affecting each side of the body in turn, and sometimes both together, and occurring at least once a month; (2) onset before the age of 18 months; (3) recovery from hemiplegia during sleep; (4) paroxysmal dystonia, nystagmus, and autonomic disturbance; and (5) non-paroxysmal choreo-athetosis, ataxia, and global learning disorder. The French workers point out that some of the features of AHC are similar to those of MELAS (mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes) a known mitochondrial disorder.

A paper from Montreal (Douglas L Arnold and colleagues, *Annals of Neurology* 1993; 33: 604–7) provides evidence of mitochondrial dysfunction in AHC. They examined four children with the disease using magnetic resonance spectroscopy to measure phosphorus and its compounds in resting gastrocnemius muscle and found reduced phosphorylation potential (a measure of mitochondrial energy storage ability (as ATP)), reduced amounts of phosphocreatine, and increased amounts of inorganic phosphate and ADP.

The authors interpret their findings as indicating mitochondrial dysfunction in muscle. They state that they can not be sure whether this dysfunction is primary or secondary but suggest that the clinical features of AHC taken together with these findings are consistent with a primary disorder of mitochondrial function.

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