Pediatric Neurology Part III: Chapter 186. Monoamine neurotransmitter deficiencies (Handbook of Clinical Neurology)

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Pediatric neurotransmitter disorders refer to a constellation of inherited neurometabolic syndromes attributable to disturbances of neurotransmitter synthesis, degradation, or transport. Monoamine deficiencies represent defects in synthesis of dopamine, serotonin, norepinephrine, and epinephrine or in availability of tetrahydrobiopterin, an important cofactor for monoamine synthesis. Some disorders do not manifest peripheral hyperphenyalaninemia and require CSF neurotransmitter metabolite assay for diagnosis. These include Segawa dopa-responsive dystonia and enzymatic deficiencies of aromatic amino acid decarboxylase, tyrosine hydroxylase, and sepiapterin reductase. The first, autosomal dominantly inherited GTP cyclohydrolase deficiency, has a satisfying response to therapy at any age with benefits maintained over time. The others have more severe and treatment-refractory phenotypes, typically with manifestations well beyond movement disorders. Disorders detectable by elevated serum phenylalanine are deficiencies of GTP cyclohydrolase (homozygous), pterin-carbinolamine dehydratase, dihydropteridine reductase, and pyruvoyl-tetrahydropterin synthase. The latter is the most prevalent and heterogeneous but typically has infantile onset with extrapyramidal as well as bulbar, hypothalamic, limbic, and epileptic manifestations. There are therapeutic roles for neurotransmitter supplementation, and dopaminergic agonists. Basal ganglia calcifications in dihydropteridine reductase deficiency are reversible with folinic acid. Deficiencies of monoamine degradation lead to cognitive, behavioral, and autonomic disorders.

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