

## Pediatric Neurology Part III: Chapter 181. Defects in amino acid catabolism and the urea cycle (Handbook of Clinical Neurology)

Georg F. Hoffmann, Stefan Kölker

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Symptoms in patients with defects in amino acid catabolism and the urea cycle usually develop because of intoxication of accumulating metabolites. The cumulative prevalence of these disorders is considerable (at least>1:2000 newborns). Timely and correct intervention during the initial presentation and during later episodes is most important. Evaluation of metabolic parameters should be performed on an emergency basis in every patient with symptoms of unexplained metabolic crisis, intoxication, and/or unexplained encephalopathy. A substantial number of patients develop acute encephalopathy or chronic and fluctuating progressive neurological disease. The so-called cerebral organic acid disorders present with (progressive) neurological symptoms: ataxia, myoclonus, extrapyramidal symptoms, and "metabolic stroke." Important diagnostic clues, such as white matter abnormalities, cortical or cerebellar atrophy, and injury of the basal ganglia can be derived from cranial magnetic resonance imaging (MRI). Long-term neurological disease is common, particularly in untreated patients, and the manifestations are varied, the most frequent being (1) mental defect, (2) epilepsy, and (3) movement disorders. Successful treatment strategies are becoming increasingly available. They mostly require an experienced interdisciplinary team including a neuropediatrician and/or later on a neurologist.

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