Pediatric Neurology Part III: Chapter 183. Epileptic encephalopathy with suppression-bursts and nonketotic hyperglycinemia (Handbook of Clinical Neurology)

Olivier Dulac

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Bursts of paroxysmal activity alternating with lack of activity define the suppression-burst (SB) pattern that may be acute, in hypoxic-ischemic encephalopathy and barbiturate intoxication, or chronic in the course of early epileptic and neonatal myoclonic (NME) encephalopathies. Malformations, namely Aicardi syndrome and hemimegalencephaly, gene mutations – of ARX and MUNC18 –, and inborn errors of metabolism, namely glycine encephalopathy, are the main causes, with spasms indicating more likely a malformation whereas myoclonus indicates metabolic disorders. Although glycine encephalopathy has a very severe outcome in its classical expression, it may be transient in the neonatal period, for reasons yet not identified. Although glycine encephalopathy is the main identified cause of NME, the disorder may not cause SB, especially in cases with later onset. The biochemical bases, due to changes in one of the four proteins that compose the enzyme, are well understood, but there is no phenotype-genotype correlation. Prenatal diagnosis is based on villous biopsy. The mechanism of SB partly depends on glutamate – or glycine, the co-neurotransmitter for NMDA transmission – overflow, mainly in the immature brain but also in cases due to barbiturate intoxication. Energy supply defect may also be involved in some inborn errors of metabolism.

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