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Biochemical key factors originating in cell death or cell process loss, observed in hypoxic–ischemic as well as inflammatory conditions, are excessive production of proinflammatory cytokines, oxidative stress, maternal growth factor deprivation, extracellular matrix modifications, and excessive release of glutamate, triggering the excitotoxic cascade. CP is most often the result of environmental factors, which might interact with genetic vulnerabilities, and could be severe enough to cause the destructive injuries visible with standard imaging (i.e., ultrasonographic study or MRI), predominantly in the white matter in preterm infants and in the gray matter and the brainstem nuclei in full–term newborns.

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Cerebral palsy (CP), defined as a group of nonprogressive disorders of movement and posture, is the most common cause of severe neurodisability in children. Interruption of oxygen supply to the fetus or brain asphyxia was classically considered to be the main causal factor explaining later CP. However several ante–, peri–, and postnatal factors could be involved in the origins of CP syndromes. Only two strategies have succeeded in decreasing CP in 2–year–old children: hypothermia in full–term newborns with moderate neonatal encephalopathy and administration of magnesium sulfate to mothers in preterm labor.

Assessment of foot deformities in individuals with cerebral palsy using weight–bearing CT.

Wellenberg RHH, et al. *Skeletal Radiol*. See all similar articles

Cited by Association between gestational levels of toxic metals and essential elements and cerebral palsy in children.

Dendrimer–Conjugated Glutamate Carboxypeptidase II Inhibitor Restores Microglial Changes in a Rabbit Model of Cerebral Palsy. PMID: 37662050 Free PMC article. 2013.2006.2009.2006.2012.2010.2023.2023.2023.2022.2023