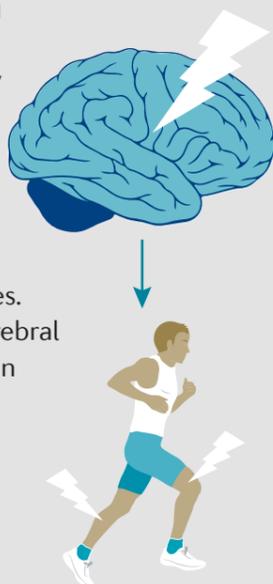


For the Primer, visit [doi:10.1038/nrdp.2015.82](https://doi.org/10.1038/nrdp.2015.82)

➔ Cerebral palsy is the most common cause of movement and posture disorders and is often also associated with disturbances in sensation, perception, cognition, communication and behaviour, as well as epilepsy and secondary musculoskeletal problems. It is not a disease in the traditional sense, but rather a clinical description of children and adults who share features of a non-progressive brain injury acquired during the antenatal, perinatal or early postnatal period of life.

MECHANISMS

Cerebral palsy is the result of processes that injure healthy brain tissue in the majority of children, but it can also be the consequence of congenital malformations of the brain. The type of lesions (liquefaction necrosis and astrogliosis), the site of lesions (the cerebral cortex, the hemispheric white matter, the basal ganglia and the cerebellum) and the response to injury all depend on the stage of brain development when the injury occurs. In general, the brain injury seems to involve several events — caused by hypoxia or ischaemia — that include cellular energy depletion, excitotoxicity and oxidative stress leading to necrosis or apoptosis. Although the primary injury takes place in the central nervous system, most clinical symptoms are observed in the peripheral neuromuscular system, especially in skeletal muscles. Muscles in children with cerebral palsy are shorter and contain fibres of reduced diameter with fewer and longer sarcomeres, hypertrophy of the extracellular matrix and fewer satellite cells.



DIAGNOSIS



The type of motor dysfunction largely depends on the location and severity of the brain lesion

! Cerebral palsy has a heterogeneous presentation; the type of movement disorder, the degree of functional abilities and limitations, and the affected body parts vary widely between patients

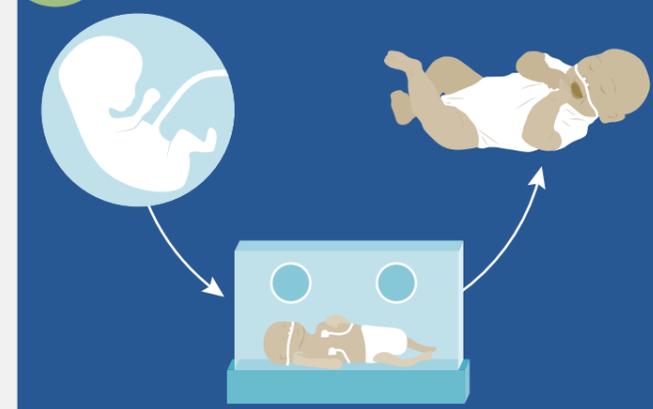
Brain imaging is recommended to confirm the clinical diagnosis

Motor dysfunction in cerebral palsy is characterized by spasticity — an excessive reaction of muscles to rapid stretch — and dystonia, which is characterized by sustained or intermittent muscle contractions or co-contractions causing abnormal and repetitive movements and/or postures

PREVENTION

Primary prevention includes the elimination of known risk factors, which include Rhesus disease, hyperbilirubinaemia and maternal iodine or thyroid hormone deficiency. Secondary prevention involves strategies that reduce premature birth and include medications that delay labour. Administration of magnesium sulfate and antenatal steroids reduce the rate and severity of cerebral palsy in premature infants, as does body cooling in high-risk full-term infants.

EPIDEMIOLOGY



Cerebral palsy has a prevalence of approximately 1 in 500 neonates, with an estimated 17 million people affected worldwide. Premature birth and difficult labour associated with neonatal asphyxia are the most important risk factors for cerebral palsy. The risk of cerebral palsy development is 50-times higher in infants born <28 weeks of gestation than in full-term infants, but a modest increase in risk is already observed as early as 38 weeks of gestation. Although premature birth is a very important risk factor, full-term infants with signs of birth depression account for the majority of cases.

MANAGEMENT

No definitive cure for cerebral palsy exists. Clinical management is directed at improving function and minimizing the effects of the factors that can make the condition worse, such as epilepsy, feeding challenges, hip dislocation and scoliosis. These management strategies include enhancing neurological function during early development; managing medical co-morbidities, weakness and hypertonia; using rehabilitation technologies to enhance motor function; and preventing secondary musculoskeletal problems.