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Editorial Note on Improving Outcomes in Cerebral Palsy

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Introduction

Cerebral palsy (CP) is a term used to describe a group of lifelong mobility and postural abnormalities that limit activity and are caused by non-progressive anomalies in the developing foetus or infant brain [1]. Other motor and non-motor areas are frequently impacted, as are lesions to the sensorimotor cortex, subcortical axon pathways, and subplate. Hypoxia, stroke, infection, trauma, and hereditary factors are all factors that contribute to the aetiology, which is complicated and o en multivariate [3-6]. Corticospinal axons have in ltrated the spinal grey matter by the end of the second trimester, and thalamic a erents have penetrated the higher layers of the neocortex. ese systems emerge in response to their activities. Descending pathways are interrupted a er an early brain lesion, resulting in aberrant re ex and corticospinal circuitry development. Movement irregularities are initially mild, but they become more noticeable over time [7]. CP is probably caused by abnormal post-lesional plasticity. It is a fallacy to believe that developmental systems are self-correcting.

Understanding activity dependent ne tuning of brain circuitry during normal development, and promoting desired plasticity while limiting negative consequences following developmental injuries are the challenges. However, before recommending therapies, we must enhance our ability to anticipate outcomes. Cerebral palsy a ects 2/1000 live births, which is several times higher than the prevalence of spinal cord injury (SCI) and amyotrophic lateral sclerosis (ALS), both of which impact the corticospinal system. However, a Web of Science literature search for the years 2010–2014 using the terms "cerebral palsy" (excluding supranuclear palsy), "spinal cord injury," and "amyotrophic lateral sclerosis" yielded fewer papers for CP (6653) than for SCI (16147) or ALS (amyotrophic lateral sclerosis) (8258). As a result, CP, which causes lifelong and o en severe disability, receives less attention from neuroscientists and neurologists than other disorders.

Hadders-Algra sets the stage with a comprehensive discussion of early brain development and the e ects of injuries, as well as the implications for early detection and intervention [8]. Marcro look at how movement recognition technology is being used to characterise spontaneous general movements in high-risk neonates. Allievi continue the subject of technology-assisted assessment by focusing on the use of instrumented toys and robot-assisted assessment tools with functional MRI to map functional brain activity in health and disease even in infancy [9]. Douglas-Escobar investigate the potential value of two blood indicators of brain injury and neurodevelopmental outcomes in neonates with hypoxic ischemic encephalopathy (HIE), namely UCH-LI and GFAP, in neonates with HIE.

e increase in neural precursor cell growth and proliferation in the sub ventricular zone following injury is dependent on insulin-like growth factor receptor signalling as well as EGRF. ey talk about how the characteristics of the culture media employed may have disguised this signi cant discovery until now. In the context of deteriorating acidemia during labour, Frasch investigates the function of adenosine monophosphate kinase (AMPK) in triggering adaptive foetal brain shut-down and reducing pro-in ammatory responses. e work by Xu, which investigates the complicated link between prior chronic foetal hypoxia, acute and increasing acidosis, the timing and length of adaptive brain shut-down, and the degree of brain in ammation in an ovine model, is accompanied by this opinion paper. ey propose that EEG monitoring, in addition to foetal heart rate monitoring, be used during delivery to detect newborns at risk of severe acidosis early. Although the ovine model sheds insight on the human condition, extrapolations across species should always be done with caution. In a review of the appropriateness of several animal models for evaluating early intervention techniques in CP, Clowry [10] examine this problem in depth.

Moving from physiology to histology and detailed longitudinal neuroimaging, Kostovic describe white matter lesions in preterm infants in terms of the developmental dynamics of "cellular compartments in the cerebral wall," demonstrating how the axonal pathways a ected can be predicted if the precise location and timing of the insult are known. Neuroimaging is also used by Mackey to understand outcome, albeit in the context of established unilateral CP. In this case, di usion weighted MRI-based fractional anisotropy in the internal capsule's posterior limb correlates with functional assessments of the upper limb. ey also show that patients with impaired upper limb function have abnormalities in intra cortical and inter hemispheric inhibition.

Gonzales-Portillo look at the possibility for stem cell therapy in newborn HIE and the unresolved clinical concerns, whereas Li look at umbilical cord blood cell therapies in preterm babies, with an emphasis on white matter damage. Non-invasive techniques in newborns with unilateral brain injury are discussed in the other two articles. Friel present an overview of current information about corticospinal tract development, including genetic and activity-dependent variables, as well as interventional techniques that may be applied to hemiplegic CP. Finally, a clinical approach, detailing the issues with hemiplegic CP, standard care options and their limitations, and baby therapies now being studied.

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None to declare

References

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