Co-funded by the European Union

Specialized and updated training on supporting advance technologies for early childhood education and care professionals and graduates

MODULE III.1

Childhood brain damage

Teacher

Elvira Mercado Val

Department of Educational Sciences

University of Burgos

and-EarlyCare-T

Table of contents

“Specialized and updated training on supporting advance technologies for early childhood education and care professionals and graduates”, e-EarlyCare-T, reference 2021-1-ES01-KA220-SCH-000032661, is co-financed by the European Union's Erasmus+ programme, line KA220 Strategic Partnerships Scholar associations. The content of the publication is the sole responsibility of the authors. Neither the European Commission nor the Spanish Service for the Internationalization of Education (SEPIE) is responsible for the use that may be made of the information disseminated herein”

I. Introduction

Childhood brain injury is a sudden injury to the brain that involves a change in neuronal activity that causes partial or generalized deficits with varying severity, altering the physical, cognitive, emotional and social dimensions, conditioning the psychosocial adjustment of the child and his family. It can be temporary or permanent. This chapter will address the different types of acquired brain damage and the repercussions arising from them.

II. Objectives

1. Addressing the concept of brain damage in children.
2. Know the most prevalent types of brain damage in children.
3. Know the most frequent neuropsychological alterations in brain damage, to assess the consequences of the injury in a developing brain.

III. Specific contents of the topic

3.1. Childhood brain injury (CBI)

CBI causes a sudden injury to the brain that generates a change in neuronal activity altering the physical, metabolic and functional integrity of the NS cells that occurs after birth.

A greater number of school-age children survive severe brain injuries due to accidents and neurological diseases, but return to school (Table 1) with multiple cognitive, behavioral, communicative and physical sequelae that will have to be evaluated academically (Cámara-Barrio et al, 2020., Hayes et al, 2017).

Unlike the lesions produced in adults, in the child we find a developing brain, with a large number of functions still unacquired, which will lead to alterations much more diffuse and complex to specify.

As Carrillo et al (2015) point out in the balance of severity and prognosis of brain damage, they will counterbalance, among other things, brain plasticity at this stage of development, the location and extent of the lesion. In most cases, as the child advances in age and brain maturation, functions that were initially absent can be enabled and altered functions improved. But, on the other hand, difficulties may appear that did not appear at first. It will be then that it is possible to outline more accurately a general profile of functioning of the child who suffers brain damage.

Table 1: Main neuropsychological and psychosocial alterations in children suffering from ICD. Cámara Barrio et al, 2020.

	Most common alterations
Cognitive area	Alteration in processing speed Faster troubleshooting Alteration in memory (storage, retention and evocation of information) Short-term memory and new learning Language (its development does not keep up with the expected pace) Alterations in attention (concentration, sustained attention, selective)
Executive functions	Behavior planning and monitoring Difficulties in judgment and formation of concepts regarding what would correspond to their age and educational level
Emotion and behavior	Difficulty showing empathy or regret Low frustration tolerance with frequent mood swings
Social aspects	Loss of friendships Poor academic performance Poor performance in social skills Social isolation within the school environment

3.2. Early brain damage

There is a general consensus that injuries acquired during early stages were considered to have less serious and shorter consequences than if they occurred at later stages. (Junqué et al, 2009).

However, challenges in comprehensive intervention after acquired brain damage cause children to return to their new reality to face the future of completing child development with an injured brain.

Therefore, a proper understanding of the real problem of the child must take into account both the context of the brain injury and the moment of development in which it is. If we consider the context, it must include all the environmental variables that may affect your cognitive, emotional, functional and social functioning, as well as the time elapsed since the injury occurred and the specific treatments you have received.

If the moment of development of the child when suffering from ABI is assessed, it is considered that recovery will depend on the age at which the injury has occurred, highlighting three basic critical reference periods:

1. Before the first year of life.

2. Between the first and fifth year of life.

3. After five years.

If the ABI occurs in the previous months, before the first year of life and bilateral lesion appears in the cerebral cortex during the period of **neurogenesis** (*miotic division of stem cells in the neural tube that form neurons and glia*) that will be completed by the fourth or fifth month of embryonic development, recovery will be total, Due in part to the fact that the process of intact division of stem cells still continues, which would allow the brain to replace the cells damaged by the injury and redistribute the existing healthy ones, managing to continue performing this miotic division (Junqué et al, 2009).

Shortly after *neurogenesis* begins, *neuronal migration* begins, which will continue for several weeks afterwards then initiating *the differentiation process*, where cells become more specialized, becoming different types of neurons. This differentiation will end at birth, although neuronal maturation (growth of dendrites, axons and synapse formation) will occur for years and in some regions (medulla oblongata and hippocampus) will continue until adult life.

If from the fourth month of life, when the massive displacements of neurons or precursor cells *(cell migration)* and differentiation for the basic formation of neuronal circuits completed *around the eighth month of birth occurs,* if neurons are altered or destroyed, the connectivity between different brain regions (cortical, corticosubcortical and subcortical) will be permanently affected, since at this stage, the brain is especially sensitive to injury (Junqué et al, 2009).

With respect to the lesions *that occur in the first year of life,* they produce greater functional alterations than those produced at later ages and will be related to lower intelligence quotients (IQ). In addition, those children who have suffered severe harm are at risk of suffering what Cámara Barrio et al (2020) call "cognitive stagnation" in phases after their recovery.

This has its importance in the learning processes because it will mean a stop or a slowdown in the stages of cognitive, social or motor development beyond this first year of life, despite the fact that there is a significant recovery of the premorbid level.

In turn, lesions that occur *around the first and fifth year* of postnatal life usually **have a certain degree of reorganization of brain function**. This reorganization is possible because dendrites and axons are still developing, having the ability to overcome the obstacles of the lesion achieving their synaptic objectives using other alternative routes. This adaptation mechanism allows to create functional connections when there is an alteration in its normal development.

Finally, injuries occurring *after the fifth year of life* usually have minimal or no functional recovery. If once the migration is established and the differentiation of the circuits has been carried out, they are damaged, the capacity to reorganize neural connectivity is already very limited.

As Junqué et al (2009) point out, it is very likely that some type of functional recovery will occur as a result of the reorganization of local circuits in the directly or indirectly affected areas**.**

3.3.Types of childhood brain damage:

3.3.1 Head injury in the paediatric population

The causes of traumatic brain injury (hereinafter TBI) in childhood differ from adults and even within the same pediatric population, varying the causes significantly according to the age of the child suffering this type of injury (Enseñat et al, 2015). In the infant stage there is a greater risk of suffering TBI due to falls and abuses due to the greater activity of children and the absence of awareness of danger (Anderson et al, 1997). Older children and adolescents tend to be victims of sports accidents and accidents (Enseñat et al., 2010). The consequences of brain damage suffered at an early age have been considered different from adults both quantitatively and qualitatively.

The lesions produced by a TBI can affect various brain areas causing motor, sensory and neuropsychological alterations (Solís-Marcos et al, 2014). This type of injury is the leading cause of brain damage in children and young people. TBI will cause neuropathological changes as a consequence of primary damage and secondary damage. (Table 2).

TBI is defined as an injury to the brain caused by an external force, blow, or wound (open or closed wound) to the head that causes alteration or loss of consciousness. With respect to the open wound, it causes skull fracture and instead, in the closed wound, injuries occur by non-penetrating wound in which there is no skull fracture.

Be that as it may,the trauma will cause primary injuries, originated at the time of impact and secondary injuries that occur will go after a period of time as a result of complications (Enseñat et al., 2015).

With respect to primary damage, a consequence of the mechanical component of trauma (movements, product of brain acceleration and deceleration after impact) that results in stretching, twisting and ruptures of axons and cerebral capillaries causing microhemorrhages. Primary lesion involves local lesion and diffuse lesion (Roig-Rovira et al, 2011)

The focal lesion within the primary lesion causes direct cortical contusions with a **more frequent location in the frontal and temporal lobes area.** Within this classification we also find injuries by blow/backlash mechanism.

Table 2. Neuropathological processes of traumatic injury Junqué, 2008

	Focal	Diffuse
Primary injury	Focal cortical contusion Deep cerebral hemorrhage Extracerebral hemorrhage	Diffuse axon injury Bleeding in white matter
Secondary injury	Delayed neuronal injury Microvascular injury Focal ischemic lesion-hypoxia Herniation Regional and diffuse hypometabolism	Delayed neuronal injury Microvascular injury Focal ischemic lesion-hypoxia Diffuse hypometabolism

On the other hand, diffuse lesions correspond to the mechanism of injury present in this type of trauma, being 40-50%. This term refers to the presence of multiple lesions located in specific regions, produced after long-term and high-speed brain decelerations. The most frequent locations are in parasagittal white matter, corpus callosum and the pontinomesencephalic junction adjacent to the superior cerebellar peduncles. (Roig-Rovira et al, 2011).

The secondary lesion causes cerebral edema, hematomas and ischemia (Junqué, 2008). It can increase brain damage established at the same time of injury.

Tertiary damage corresponds to deeper changes that generate modifications in neurotransmitters, ionic homeostasis and neuronal membrane.

With regard to the assessment of TBI severity, it is evaluated (initial *assessment at the initial moment of patient evaluation*) on the score obtained on the Glasgow Coma Scale (GCS) (Table 3) which classifies the severity according to three types of response; ocular response, motor response and verbal response.

The ocular response includes: 4. Spontaneous response, 3. Verbal order. 2. Pain. 1. No response. **The motor response**: 6. Obey verbal command. 5. Locate the pain. 4 Remove and bend. 3. Abnormal flexion. 2. Extension. 1. No response. **The verbal response**: 5. Oriented and preserved. 4. Disoriented and talking. 3. Inappropriate words. 2. Incomprehensible sounds. 1. No response. Maximum score: 15. Depending on the total response obtained by adding these three types of response, the TCE can be classified as mild, moderate and severe.

A mild TBI is one that has obtained a score between 13-15; a moderate TBI is between 9-12 points, and severe between 3-8 points. (Table 3)

Table 3. Severity of TBI taking into account the Glasgow Coma Scale. Excerpted from León Carrión and Domínguez-Morales, 2005.

GCS Score	Severity	Neuropsychological deficit	Evolution
15-13	Lightweight	Lightweight	Positive 1-6 months
12-9	Moderate	Moderate	Reserved 1-15 months
8-3	Grave	Grave	Months of rehabilitation

The loss of consciousness at the time of injury followed by time in a coma always occurs with diffuse lesions, due to rotational mechanisms that cause stretching and rupture of axons, subsequently producing neuronal death.

White matter lesions can interrupt the normal functioning of the frontal lobe ascending activator reticular system, leading to alterations of frontal-executive semiology**: attention and motivation.** In addition to attentional deficits, diffuse lesions are characterized by the presence of concentration difficulties, mnestic processes, slowing in the speed of information processing, fatigue, irritability and lack of initiative.

If we consider the neuropsychological alterations (Table 4) that appear as a consequence of TBI, we find **a cognitive profile** where alterations in processing speed predominate (white matter injury, corpus callosum), memory (hippocampus/prefrontal area), attention, executive alterations and alteration in the ability to acquire new learning. Process that causes a decrease in the ability to make new learning (anterograde amnesia, plus short-term memory dysfunction) vital for those children who are of school age (Cámara-Barrios, et al, 2020; Junqué, 2008).

The alteration of **frontal/executive** functions is a constant and is explained by the fact that these functions require the integrity of all circuits affected by LAD (Junqué, 2008). The cognitive, behavioral and emotional consequences of people who have suffered a mild TBI usually resolve before six months and even within the first month (León-Carrión and Domínguez, -Morales, 2005).

Table 4. Neuropsychological alterations in TBI. Based on Enseñat et al, 2015.

Neuropsychological process	Alteration	Injury
Attention	Attention deficit Selective, sustained attention (increase in number of omissions) Incomplete visual search, crawling	Prefrontal injury Diffuse axonal damage depending on the location of the lesion
Processing speed	Slowing (may interfere with other processes, attention, memory, language, visualconstruction, motor and precision.	Diffuse axonal damage Focal basal ganglia lesions
Language	Aphasia, anomie, verbal fluency, pragmatics of language	Focal lesions or diffuse lesions
Memory	Alteration processes encoding and evocation of new information. Impact on learning ability	Loss of hippocampal volume Involvement of neuronal structures Frontal lobe damage
Executive functions	Lack of initiative, difficulty controlling impulses, disinhibition, inability to seek alternatives, inflexibility, poor planning skills and low tolerance for frustration	Prefrontal lesions
Emotion and behavior	Difficulty managing behavior, egocentrism, perseverance and impairment of social skills, emotional instability, aggressiveness	moderate, severe TBI

3.3.1.1. Neuroimaging of TBI

Neuroimaging techniques and lesional studies used to identify the neural basis and characterize the effects of TBI provide important structural and functional data. This allows to delimit the acute diagnosis and the long-term structural sequelae. (Junqué, 2009). Within neuroimaging techniques, structural magnetic resonance imaging and computed tomography (CT) are mainly used.

With magnetic resonance imaging, microhemorrhages can be visualized. It has a better resolution to detect areas of contusion or diffuse lesions of the white matter being this technique more accurate in the diagnosis during the acute phase.

However, CT has clear advantages in its use for several reasons:

1. With CT you can better visualize, bleeding in the acute phase.
2. Detects fractures, ventricular dilation and its correlation with the degree of cortical atrophy
3. It is relatively fast and has greater availability and facilitates rapid monitoring of the patient, especially in the acute phase.

In the acute phase, CT shows brain compression, reduction in ventricular size and tissue changes showing edema and presence of microhemorrhages.

This is important because it was traditionally believed that children recovered better from TBI. Children may even show better resolution of motor and sensory deficits. However, these differences are not met for cognitive functions and many studies have demonstrated the presence of long-term neuropsychological deficits after severe TBI in children (Enseñat et al, 2015).

Yuan et al, 2007, on a sample of children aged 6 to 9 years with TBI of at least one year of evolution, observed decreases in the size of the corpus callosum appreciating that the alterations of connectivity were not reversible.

This leads to the conclusion that pediatric TBI causes a reduction in size and microstructural changes in the posterior regions that indicate an interruption in neurodevelopment and altered myelination (Junqué, 2008). If magnetic resonance imaging is used, there are three parameters to classify the degree of diffuse axonal injury. (Table 5).

Table 5. Classification parameters of diffuse axonal injury Junqué, 2009.

DEGREE	DIFFUSE AXONAL INJURY
I	White matter and gray matter injury
II	Focal lesions in the corpus callosum
III	Additional brain damage to the brainstem

The most relevant quantitative measures in neuropsychology that reflect the impact of diffuse brain damage are the volume of the ventricular system, the surface of the corpus callosum, the volume of the hippocampus and that of the basal ganglia.

Both the volume of the ventricular system and the surface of the corpus callosum are an indirect measure of diffuse axonal damage. On the other hand, hippocampal and basal ganglia volume reflect diffuse neuronal loss of highly vulnerable brain structures. (Junqué, 2008).

The size of both surface and volume of these structures is related to the most frequent cognitive losses in TBI and that are the objective of intervention in neuropsychological rehabilitation, domains such as attention, learning capacity (vital for academic performance) and mental processing speed. The quantification of the atrophy of brain structures (loss of global brain volume) may have some interest to assess the impact of these sequelae on higher cognitive processes.

3.3.1.2. Educational needs of children who have undergone TBI:

One of the most common problems that appear in children who have suffered a TBI (Table 4) is the slowdown in the **speed of information processing,** which implies that the student cannot continue learning at an adequate pace. You may need to present information more easily or you may also need more time to understand the information before responding (Carney et al, 2013)

Another aspect of vital importance is related to how the child with a TBI **assimilates the learning of school.** In general, the children did not forget what they had learned before suffering the trauma, and the pre-injury learning was relatively intact.

On the other hand, new difficulties will arise when new knowledge has to be stored, a mnesic process altered due to the ECT. We see that the child shows important limitations that affect their academic performance, so it will be necessary to take into account when considering the curricular adaptations that must be taken into account, how is the student's ability to learn new skills and be able to overcome the respective academic years.

If we assess the alterations in **the mnestic processes** produced by the injury, we have a type of student with alterations in memory both in the coding phase and evocation of new information, which will have an impact on their learning capacity (Enseñat et al, 2015).

If we focus on **the attentional processes** altered after the injury, children who have suffered TBI are especially vulnerable to presenting attentional alterations, since these capacities are in continuous development during childhood. (Enseñat et al., 2015).

Attentional deficits are associated with frontal lobe lesions or diffuse axonal injury. The attentional manifestations will vary according to the type and location of brain damage. The most frequent attentional alterations are both in **sustained and selective attention,** showing a greater number of omissions (oversights) that increase over time. Attentional deficits of alternating type and divided attention are more evident in the later stages of development.

3.3.2 Childhood brain infections

Among the CNS infections that can occur in children, are those that are caused by viruses or bacteria, which invade the central nervous system (CNS) producing inflammation of the brain (encephalitis and meningitis). These CNS infections arise as a result of invasions by viruses and bacteria in the brain and spinal cord, through transmission via the nose, ears or mouth resulting in a wide number of neurological sequelae ranging from severe disability to complete recovery, through subtle alterations. (Enseñat el al 2005).

3.3.2.1 Viral encephalitis

Encephalitis is an inflammatory process of the brain of viral origin that produces a neurological dysfunction characterized by the presence of fever, headache and altered consciousness being infectious, autoimmune, etc. Others include acute cognitive dysfunction, behavioral changes, **focal neurological signs** and seizures. (Huanca et al, 2012),

Most cases of viral encephalitis are caused by Herpes simplex virus (HSV) types 1 and 2, varicella-zoster, Epstein Barr virus (EBV), measles, mumps and enterovirus. However, this will depend on the continent and environmental factors.

The herpes simplex virus mainly affects the brain parenchyma in the temporal lobes, and in some cases, frontal and parietal area. The mumps virus can cause acute viral encephalitis or postinfectious encephalitis.

The influenza virus causes diffuse cerebral edema as the main component in pathogenesis and for the varicella zoster virus the vasculitic process predominates.

The mechanism by which the virus crosses the blood-brain barrier explains the pathogenesis of any viral encephalitis. The usual neurotropic pathway consists of the penetration of the virus into the motor or sensory nerve terminals reaching the ganglion cells or motor neurons. HSV-1 encephalitis occurs during primary infection in younger children, however, in older children and adults, the most common mechanism is viral reactivation from the latent phase in which the viruses are located at the level of olfactory bulb and brainstem (pons and medulla oblongata). Huanca et al, 2012.

3.3.2.1.1 Symptoms of encephalitis:

The symptoms are often similar to those of a flu-like picture (fever, headache, lack of energy), although in severe cases there are serious neurological disorders (altered speech and hearing, diplopia, hallucinations, changes in personality, loss of consciousness, loss of sensation in some parts of the body, muscle weakness, partial paralysis in the arms and legs, sudden severe dementia, impaired judgment, seizures, and memory loss).

In babies it is especially important to also pay attention to symptoms such as vomiting, body stiffness, tense or protruding fontanelle and /or constant crying and hypoactivity. Guaman, 2018.

3.3.2.1.2. Neuropsychological disorders caused by encephalitis

With regard to neuropsychological alterations, we find a cognitive profile where alterations are observed in memory processes (retrograde amnesia) and mainly alterations in executive functions (attention, planning, supervision of behavior). (Mogollón et al, 2010).

3.3.2.2 Meningitis

Meningitis involves inflammation of the meningeal membranes being this pathology relatively common in childhood. The most frequent symptoms are, **headache, fever, stiffness, vomiting, confusion and lethargy, and** may progress to a loss of consciousness with seizures unless treatment is instituted quickly. (Enseñat et al, 2015).

Meningitis can be caused by either a viral or bacterial infection, with viral infection being the most common, but it is the most difficult to diagnose. On the other hand, bacterial meningitis is easier to detect and already with the use of vaccination in children under five years of age the incidence of this disease has dropped considerably.

The treatment of bacterial meningitis requires the isolation and identification of the pathogen, as well as treatment with antibiotic therapy and in some cases, the use of anticonvulsant medication is necessary. In the acute phase of the disease there is an interruption in the dynamics of the cerebrospinal fluid (CSF) that causes a series of processes that increase intracranial pressure, causing hydrocephalus, cerebral edema and subdural effusions. (Enseñat et al, 2015).

All these events secondary to infection can negatively influence causing an increase in intracranial pressure (ICP) obstructing the flow of CSF in the ventricular system causing herniation (pressure that displaces structures) within the pons, medulla oblongata and cerebellum, affecting part of the cranial nerves that are located in these anatomical areas, *which is why vestibular alteration and hearing loss* are more frequent sequelae in children suffering from meningitis (Enseñat et al, 2015).

3.3.2.2.1. Neuropsychological alterations caused by meningitis.

It is known that suffering from the disease before 12 months, is a risk factor for suffering neuropsychological and neurological sequelae, Although most of the problems associated with meningitis are resolved over time, there is a percentage of children to whom it does not happen, leaving them with permanent sequelae.

The sequelae caused by meningitis include a series of alterations in the main cognitive processes involved in memory, processing speed and alterations in language. It has been shown that children who have suffered bacterial meningitis obtain an IQ on average at a low or even below average, in more than one standard deviation (Enseñat et al, 2015).

3.3.2.3. Brain tumors.

Brain tumors represent the most frequent type of solid tumor in pediatric age, being the second in general frequency after the group formed by leukemias and lymphomas. (López-Aguilar et al, 2011).

The signs and symptoms of neurological dysfunction in a child with a brain tumor vary and will depend on both the child's age and developmental level, as well as the location and origin of the tumor. The most prevalent brain tumors in childhood are **medulloblastomas and primitive cerebellar neuroectodermal tumor** , whose onset is located in the posterior fossa.

The usual age of diagnosis is between 3 and 9 years of age, being more common in boys than in girls. There are different treatment options, but the irreparable damage that radiotherapy can cause on the developing CNS of children is also recognized,

which is why we try to delay its application whenever possible until 5 and even 8 years (Enseñat et al, 2015).

Neuroscientific research postulates that the neurotoxic effects of these treatments will lead to the presence of alterations in hippocampal neurogenesis (fundamental for mnestic processes), destruction of CNS neuron precursors (oligodendrocytes and alterations in white matter).

This can cause cognitive and behavioral alterations associated with chemotherapeutic and radiotherapeutic treatment. If we take into account specific cognitive skills, it is observed that, in these children, there is a decrease in general intellectual capacity, with lower scores on the manipulative quotient versus the verbal quotient. In addition, there are alterations in attentional processes (sustained attention), low speed in information processing, alterations in expressive and compressive language, abstract reasoning and the ability to store new information.

This has its relevance in terms of the academic assessment of children who return to classes, seeing that academic difficulties will appear in both reading and writing, as well as calculation, processes necessary for the acquisition of knowledge. (Enseñat et al, 2015).

Table 6. Main most common alterations in brain tumors (Grau Rubio and Cañete, 2002)

	Sensory	**Motor**	**Cognitive**	**Emotional**
Type of alteration	Uni- or bilateral perceptual deafness Total or partial blindness, temporary or homonymous hemianopsia, ocular motor impairment, nystagmus and mydriasis	Hemiplegia and hemiparesis, spasticity, ataxia, adiadochokinesia and paresthesia	Attentional disturbances, drowsiness, mental clumsiness (fog), mnestic difficulties and decrease in IQ scores Aphasia, dysarthria, akinetic mutism.	Mental fog, self-esteem issues and social skills

3.3.2.4. Neonatal ischemia-hypoxia

Perinatal asphyxia remains one of the major causes of neurological morbidity and mortality. **Hypoxic-ischemic neonatal encephalopathy** is a major cause of brain damage, affecting 1-3 in 1,000 newborns in a moderate-severe manner and posing a high risk of permanent neurological deficits. The only current therapeutic approach consists of moderate hypothermia, whose efficacy, although proven, does not always provide a total functional recovery (Moral et al, 2019).

Fetal asphyxia decreases cerebral and systemic blood flow with decreased oxygen and glucose supply, reversal of aerobic to anaerobic metabolism, decreased energy production, and apoptosis with or without permanent neuronal damage. Three forms of clinical presentation have been described. The mild form is characterized by total recovery in three days and without, or minimally, sequelae of neurodevelopment without body hypothermia. Moderate and severe forms lead to permanent

neurological deficits and neurodevelopmental disturbances (48%) or death (27%) after treatment with body hypothermia (Papazian, 2018).

Perinatal hypoxic-ischemic encephalopathy (HIE) presents a set of clinical and neuropathological manifestations that occur in the NB after an episode of asphyxia, being necessary to clearly differentiate asphyxia from encephalopathy, since physiopathologically they are different, even if they are sequential events: asphyxia is cause, while encephalopathy is effect; however, asphyxia does not always produce HIE, nor is the choking factor found in all injuries. (Rizzo Ortega, 2017).

This syndrome of neurological dysfunction affects newborns older than 35 weeks, with an estimated incidence of 1-3/1000 live births. It is currently known that this pathology has a wide spectrum of symptoms characterized by the presence of motor alterations, movements, muscle tone, auditory dysfunctions with or without hearing loss, oculomotor alterations and tooth enamel dysplasia among others. Neonatal encephalopathy should not be seen as a causal risk factor for CP, but as a more reliable isolated prognostic factor in children born at term and near term. Perinatal asphyxia causing brain damage and subsequent sequelae is invariably caused by acute encephalopathy. (Barcia de la Cruz et al, 2021).

Summary

In this item III. 1 The concept of brain damage in children has been addressed and the different types of brain damage most prevalent in the child population have been reviewed. The neuropsychological function of the neuroanatomical and neurobiological structures involved, as well as their most frequent alterations in brain damage, has been reviewed to know the possible impact associated with the injury on a developing brain.

Glossary

Aconitic mutism: the estate in which a person is virtually unable to speak (mutism) or move (akinetic). Akinetic mutism often occurs due to damage to the lower frontal lobe of the brain.

Adiadochokinesia: The ability to perform rapid and repetitive opposite movements; this is the lack of coordination of body movements.

Anosognosia: neuropsychological disorder that generates in the patient an inability to have a state of full awareness about his disease or deficit, product of brain damage or a neurodegenerative process.

Apoptosis: programmed cell medicine.

Cell migration: the optimization of movements or massive displacements of nerve cells, or precursor cells, in order to establish differentiated populations of nerve cells (layers of the cerebral cortex, subcortical nuclei).

Cerebral parenchyma: brain mass consists mainly of neurons, glial cells and blood vessels, which together give a structure and intensity.

Differentiation: The process by which cells become more specialized. In the early stages of embryonic development, the cells are similar to each other, but they specialize later. Nerve cells acquire specific characteristics by being part of different structures of the nervous system.

Diffuse axonal injury (DAI): A form of traumatic brain injury. It occurs when the brain moves rapidly within the skull when an injury occurs. The long connecting fibers in the brain called axons are cut as the brain rapidly speeds up and decelerates within the hard bone of the skull.

Enamel dysplasia: Qualitative or quantitative structural disorders that occur during the period of amelogenesis (formation of tooth enamel). Dysplasias are included within the dentinal anomalies that imply that the teeth present an opalescent, bluish-gray opaque and amber appearance

Encephalopathy: malfunction of the brain. In current medical use, encephalopathy refers to a syndrome of brain dysfunction, which can be caused by multiple medical conditions.

Focal neurological signs: signs that appear by the presence of neurological disease and whose appearance, allows to locate the lesion in the cerebral area where the clinical findings are observed. In meningitis, Kernig's sign (resistance to passive extension of the knee) is observed

Myelination: Balancing axons with a myelin sheath in order to allow adequate transmission of nerve impulses.

Neurogenesis: also called proliferation, this being a process that consists of the miotic division of stem cells in the neural tube to later produce neurons and glia. It leads to the formation of brain regions at precise times. It begins with the caudal cortical regions to end with the most complex structures of the cerebral cortex.

Synaptogenesis: The establishment of synaptic connections as neuronal tissue develops and axons and dendrites grow.

Bibliography

Barcia de la Cruz, S. F., Intriago Macias, M. D., Mera Rivas, J. D., Bazurto Zambrano, A. V. (2021). Risks and symptoms of neonatal encephalopathy RECIMUNDO, (Vol. 5) *; 261-270, DOI: 10.26820/recimundo/*5.(*ESP.1).Nov.2021.261-270*

Cámara Barrio, S., Esteso Orduña, B., Vara Arias, M. T., Rodríguez Palero, S., Fournier del Castillo, M. C. (2020). Neuropsychological approach in a pediatric unit of acquired brain damage of the public health system. *Neurology*, 8 , 1-8.

Guaman, E. (August 27, 2022). *Encephalitis, diagnosis and prevention.* Elsevier. https://www.elsevier.com/es-es/connect/medicina/encefalitis-sintomas,-diagnostico-y-prevencion

Huanca, D. (2012). *Handbook of Neuropediatrics - Evidence-Based CPG.* File. IIDENUT.

Forsyth, R. J. (2010). Back to the future: rehabilitation of children after brain injury. *Arch Dis Child*, 95 (7); 554-559.

Junqué, C. (2008). Assessment of diffuse axonal damage in traumatic brain injury. *Writings of Psychology.* 2-1, 54-64.

León-Carrión, J., Domínguez Morales, M. R. (2005). Assessment of the mental and psychiatric sequelae derived from traumatic brain damage: when, what and how. Spanish Journal of Neuropsychology. 7.1, 35-49.

López-Aguilara, And. , Sepúlveda-Vildósola, A.C., Rioscovian-Soto, A. P., Pérez-Ramírez, J. P., Siordia-Reyese, G. (2011). Brain tumors in pediatrics. Current status of diagnosis and treatment. *GAMO,* Vol. 10 (1), 41-45

Mogollón, P., Negrete, J. (2010), Neuropsychological profile of a patient with herpetic encephalitis. In:http://biblioteca.usbbog.edu.co:8080/Biblioteca/BDigital/66228.pdf

Papazian, O. (2018). Neonatal hypoxic-ischemic encephalopathy. *Medicine*, 78(2), 36-41.

Rizzo Ortega, A. A. (2017). Neonatal encephalopathy in University Hospital between 2014 - 2015. University of Guayaquil. Faculty of Medical Sciences. Medical career.

Solís-Marcos, I., Castellano-Guerrero, A. M., Machuca-Murga, F., Domínguez-Morales, R., León-Carrión, J. (2014). Predictors of cognitive functional recovery in patients with traumatic brain injury. *Revneurol,* 58, (7): 296- 302

Verger, K., Serra-Grabulosa, J. M., Junqué, C., Álvarez, A., Bartrés-Faz, D., Mercader, J. M. (2001). Study of the long-term sequelae of traumatic brain injuries: evaluation of declarative and procedural memory and its neuroanatomical substrate. *Revneurol,* 33 (1): 30-34.

Resources

Web

Childhood brain damage: https://neurointegra.com/dano-cerebral-adquirido-infantil/

Spanish Federation of brain damage: https://fedace.org/

Active training in Early Care Pediatrics: https://fapap.es/articulo/304/atencion-temprana-recursos-criterios-de-derivacion

Foundation to help newborns with neurological problems: https://www.neurologianeonatal.org/