Module III.1

Pathologies at an early age

Brain damage

Co-funded by the European Union

e-EarlyCare-T

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"This project has been funded with support from the European Commission. This publication [communication] reflects the views only of the author, and the Commission cannot be held responsible for any use which may be made of the information contained therein. "

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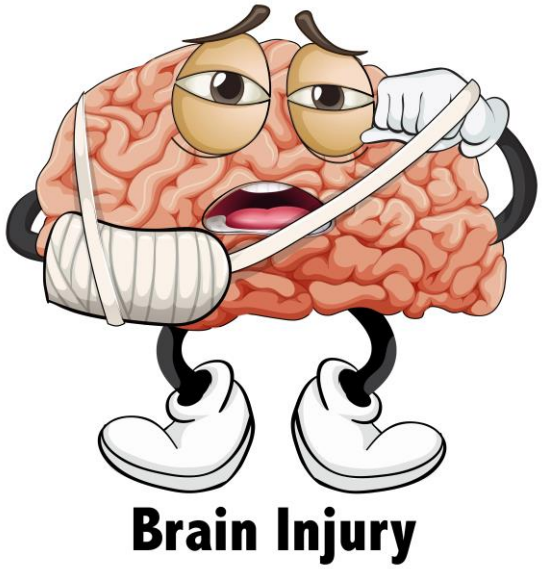
Pathologies at an early age: Brain damage

- **1.1. Concept of childhood brain damage**
- Childhood DCA **is a sudden injury to the brain** that involves a change in neuronal activity that affects the physical integrity, metabolic activity or functional capacity of brain cells.
- Occurs after birth (non-congenital, hereditary or degenerative origin)
- The etiology includes:
- Traumatic brain injury (TBI).
- ischemic stroke /haemorrhagic stroke
- Brain infections.
- Hypoxia and Neonatal Ischemia (Cámara-Barrio et al, 2020).



1.1. Concept of childhood brain damage

The sequelae associated with DCA can present with varying severity, be temporary or permanent, cause partial or generalized deficits in the physical, cognitive, emotional and social dimensions, affecting children's and their families' psychosocial adjustment (Cámara-Barrio et al, 2020).

Unlike lesions in adulthood, children's brains are still developing, with many functions not yet acquired, leading to much more diffuse and complex alterations.

Evaluating the severity and prognosis of brain damage needs the assessment of brain plasticity, location, extent of the injury and the point in brain development when the damage occurs.

1.1. Concept of brain damage

As children age and mature, functions that were initially absent can be enabled, improving altered functions. However, new difficulties may appear. It is at that point that a general profile of functioning can be outlined for children suffering from brain damage.

1.1. Early brain damage:

There is a general consensus that injuries acquired during early stages have less severe and less long-lasting consequences than if they occurred later in life (Junqué et al, 2009).

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

Unrealistic expectations of recovery can cause subsequent events to be perceived as a functional, academic, and work "failure" (Forsyth, 2010).

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1.1. Childhood brain damage

Proper understanding of children's problems in the context of brain injury needs consideration of both the context and the point in the child's development.

Context includes all the environmental variables that may affect cognitive, emotional, functional and social functioning, as well as the time elapsed since the injury and the specific treatments received.

Considering when in their development a child suffers from DCA, recovery will depend on the age at which the injury occurs, there are three critical reference periods:

1. Before the first year of life

2. Between the first and fifth year of life

3. After the age of five.

1.1. Childhood brain damage

Before the first year of life

If DCA occurs in the previous months, **before the first year of life** and bilateral lesion appears in the cerebral cortex during the period **of neurogenesis** (myotic division of stem cells in the neural tube that will subsequently form neurons and glia) that will be completed towards the fourth or fifth month of embryonic development, recovery will be total. Due in part to:

The ongoing process of intact division of intact stem cells, which would make it possible for the brain to replace the cells damaged by the injury and redistribute existing healthy cells that could continue to perform this myotic division. (Junqué et al, 2009)

Shortly after starting neurogenesis, neuronal migration begins, which will continue for several weeks, before the process of differentiation begins (cells become more specialized, becoming different types of neurons)

1.1. Childhood brain damage

Before the first year of life

If there is alteration or destruction of neurons **from the fourth month of life—**at which time the massive displacements of neurons or precursor cells (cell migration) and differentiation for the basic formation of neural circuits completed **around the eighth month of birth** occurs—connectivity between different brain regions (cortical, corticosubcortical and subcortical) **will be permanently affected**, since at this stage, the brain is especially sensitive to injury.

This differentiation ends at birth, although neuronal maturation (growth of dendrites, axons and synapse formation) will occur for years and in some regions (medulla oblongata and hippocampus) continues into adult life.

1.1. Childhood brain damage:

2. Between the first and fifth year of life:

Lesions in the first year of life produce greater functional alterations than those at later ages and are related to lower (IQ).

Children who suffer severe damage are at risk of suffering "cognitive stagnation" in post-recovery phases.

This is important in the learning processes because it means a stop or a slowdown in the stages of cognitive, social or motor development beyond this first year of life, despite significant recovery to the premorbid level.

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1.1. Childhood brain damage:

2. Between the first and fifth year of life:

Injuries occurring between the first and fifth year of postnatal life usually mean a certain degree of reorganization of brain function.

This reorganization is possible because **dendrites and axons** are still developing and can overcome the obstacles of the injury, reaching their synaptic targets using alternative routes.

This adaptation mechanism allows functional connections to be created when normal development is affected in any way.

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1.1. Childhood brain damage:

3. After the age of five.

Injuries **after the fifth year** of life usually **have minimal or no functional recovery.**

If neurons are damaged after migration and circuit differentiation, there is very limited capacity to reorganize **neuronal connectivity.**

There will very probably be some kind of functional recovery due to reorganization of local circuits in the areas directly or indirectly affected. **(Brain plasticity)**

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1.2. Types of brain damage

Traumatic brain injury (TBI)

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.

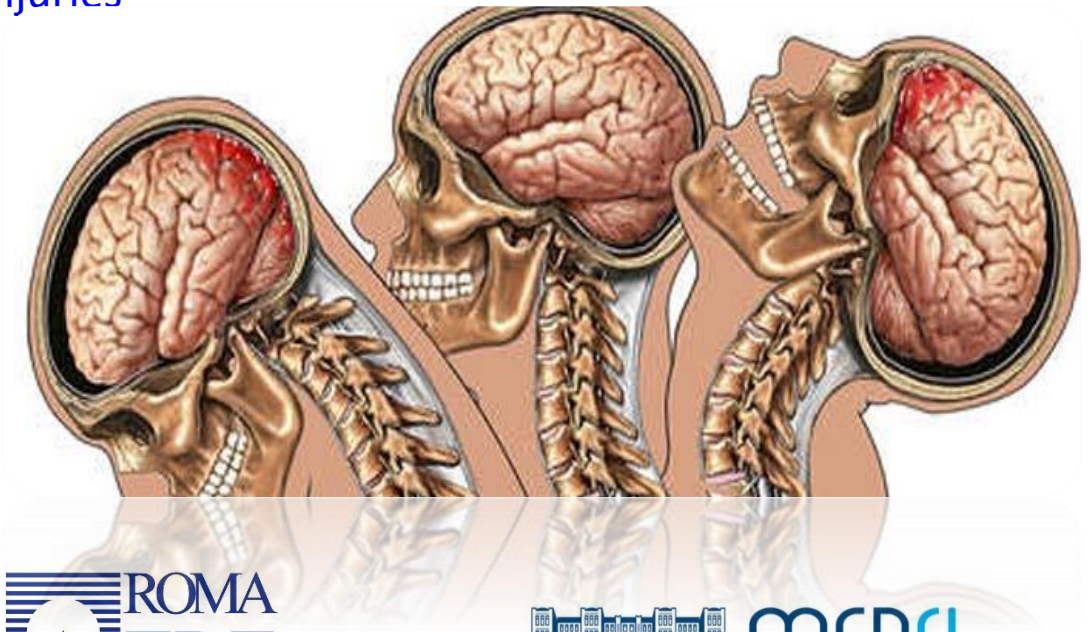
TBI will cause primary injuries (moment of impact) and secondary injuries (occur later as a result of complications)

Injuries can affect various brain areas causing alterations:

. **Motor**

. **Sensory**

. **Neuropsychological**



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1.2. Types of brain damage

Traumatic brain injury (TBI)

The main cause of brain damage in children and young people.

Primary damage: consequence of the mechanical damage of the trauma, a product of acceleration-deceleration.

Causing stretching, twisting and ruptures of axons and cerebral capillaries.

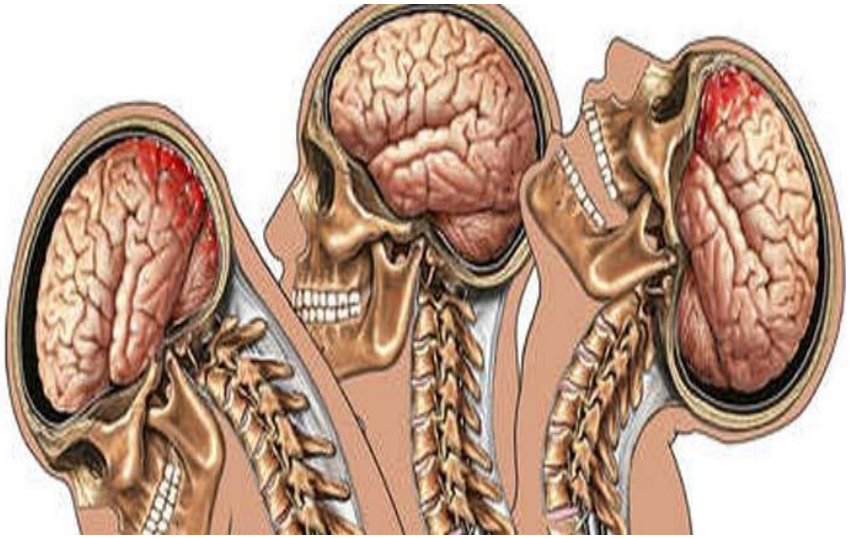
Microhemorrhagias.

Primary injury involves focal injury and diffuse injury.

Focal injury: cortical contusions (blow/backlash injury)

More frequent in frontal and temporal lobes

Diffuse lesion: white matter, pontinomesencephalic corpus callosum, cerebel



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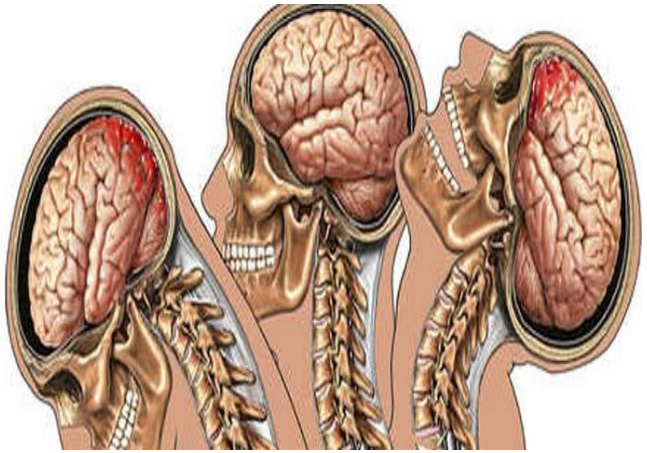
1.2. Types of brain damage

Traumatic brain injury (TBI)

Junqué, 2008

Secondary damage: cerebral edema, bruising and ischemia.

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



Tertiary damage:
Modifications in neurotransmitters, ionic homeostasis
Neuronal membrane

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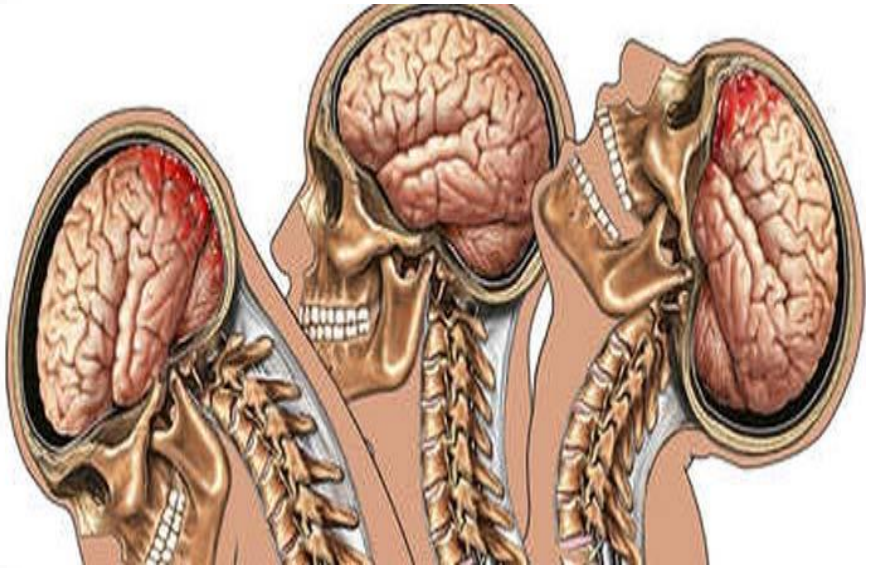
1.2. Types of brain damage

Assessment of TCE severity: Glasgow coma scale

TABLE 38-2
Glasgow Coma Scale

BEHAVIOR	RESPONSE	SCORE
Eye opening response	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best verbal response	Oriented to time, place, and person	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response	Obeys commands	6
	Moves to localized pain	5
	Flexion withdrawal from pain	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	No response	1
Total score:	*Best response*	15
	Comatose client	8 or less
	Totally unresponsive	3

Mild TBI: 13-15 points
Moderate TCE: 9-12 points
Severe TBI: 3-8 points



1.2. Types of brain damage

Assessment of TCE severity: Glasgow coma scale

Loss of consciousness at the time of injury

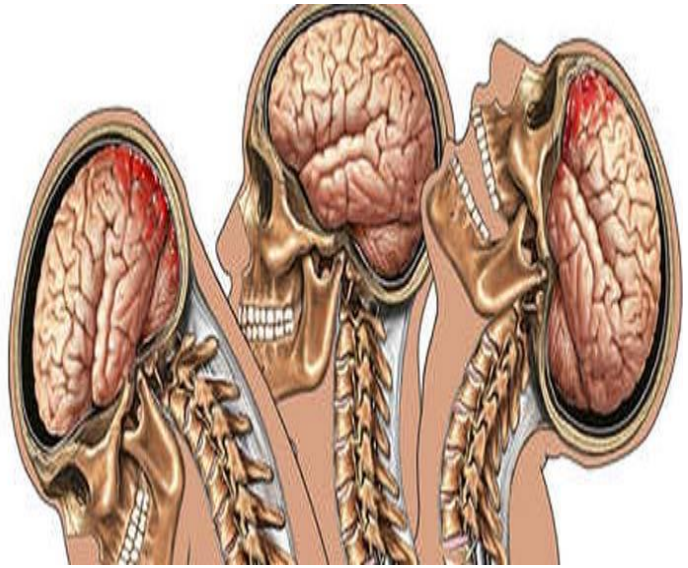
Time in a coma (diffuse lesions)

Rotating mechanisms (stretching and rupture of axons) apoptosis

White matter lesions can cause alteration in the activating mechanisms

Reticular activator system (frontal lobe) disejecutive syndrome (attention and motivation)

Mild TBI: 13-15 points
Moderate TCE: 9-12 points
Severe TBI: 3-8 points



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Pathologies at an early age: Brain damage

Neuropsychological alterations associated with TBI:

Alterations in processing speed (white matter injury, corpus callosum)

memory (hippocampus/prefrontal area)

Attention, executive alterations and alteration in the ability to acquire new learning.

Decrease in ability to perform new learning (anterograde amnesia, more short-term memory dysfunction) vital for school-age children (Cámara-Barrios, et al, 2020; Junqué, 2008).

Alteration of frontal/executive functions is a constant and is explained by the fact that these functions require the integrity of all cortico-cortical and cortico-subcortical circuits, circuits affected by LAD (Junqué, 2008).

Non-awareness of the deficit (anosognosia) apathy, verbal disturbances (lexical access).

The cognitive, behavioral and emotional consequences of children with mild TBI are usually resolved before six months and even within the first month (León-Carrión et al, 2005).

Pathologies at an early age: Brain damage

Neuropsychological process	Alteration	Injury
Attention	Attention deficit Selective, sustained attention (visual search, crawl)	Prefrontal injury Diffuse axonal damage (location of the lesion)
Processing speed	Slowing (interferes with other processes, attention, memory, language, visuoconstruction, motor and precision.	Diffuse axonal damage Focal lesions basal ganglia Focal lesions or diffuse lesions
Language	Aphasia, anomie, verbal fluency, pragmatic	Diffuse axonal damage Focal lesions basal ganglia Focal lesions or diffuse lesions
Memory	Coding processes and evoking new information. Impact on ability to learn	Loss of hippocampal volume Involvement of neuronal structures Frontal lobe damage
Executive functions	Lack of initiative, impulsivity, disinhibition, inability to seek alternatives, inflexibility, poor planning and low tolerance for frustration	Prefrontal lesions
Emotion and behavior	Difficult behavior, self-centeredness, perseverance and affectation social skills, emotional instability, aggressiveness	Moderate, severe TBEs

Pathologies at an early age: Brain damage

Neuroimaging of TBI:

Provides important structural and functional data.

In the acute phase, brain comprehension, reduction of ventricular size and tissue changes (edema and presence of microhemorrhages) are observed.

It allows an outline of acute diagnosis and long-term structural sequelae (Junqué, 2009).

Two neuroimaging techniques are used: CT and magnetic resonance imaging.

Computed tomography (CT) scan

CT has clear advantages:

1. Better visualization of hemorrhages in the acute phase.

2. Detects fractures, ventricular dilation and there is a correlation with the degree of cortical atrophy

3. It is relatively fast, facilitates rapid monitoring, especially in the acute phase.

Pathologies at an early age: Brain damage

TBI Neuroimaging: Magnetic Resonance Imaging:

Three parameters classify the degree of diffuse axonal injury.

DEGREE ⇨	**DIFFUSE AXONAL INJURY**
I	**Injury to white matter and gray matter**
II	**Focal lesions in the corpus callosum**
III	**Additional brain damage to the brain stem**

Quantitative measures of importance in neuropsychology for diffuse brain damage are

The volume of the ventricular system **(indirect measure of diffuse axonal damage)** ventricular dilation

Surface of the corpus callosum, volume of the hippocampus (**Diffuse neuronal loss of cortical structures)**

Surface of the basal ganglia. (**Diffuse neuronal loss of cortical structures).**

Size and surface area, **volume are related to the most frequent cognitive losses in TBI.**

Pathologies at an early age: Brain damage

Educational needs of children with TBI

School and brain damage

Significant curricular adaptations based on these alterations

Cognitive impairments present after TBI

Learning and processing speed

Cognitive functioning (IQ)

Memory and new learning

Attention and academic performance (fatigue)

Behavioral changes and mood.

- Deficit awareness (aware of the limitations/alterations caused by the brain damage).

1.2.2. Childhood brain infections

These are infections of the CNS caused by viruses or bacteria that can cause inflammation of the brain or meninges (encephalitis and meningitis).

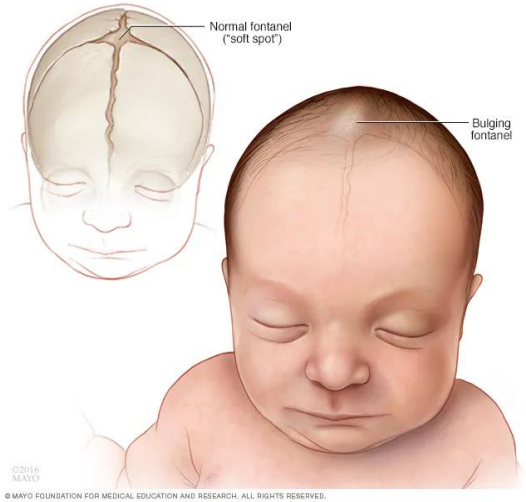
They result from infection through the nose, ears or mouth and can lead to a large number of neurological sequelae that go:

From severe disability to full recovery through subtle alterations.

Viral encephalitis

Meningitis

Neuropsychological alterations



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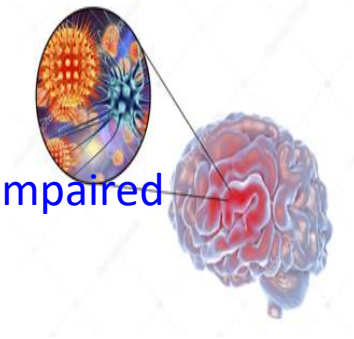


1.2.2 Viral encaphalitis

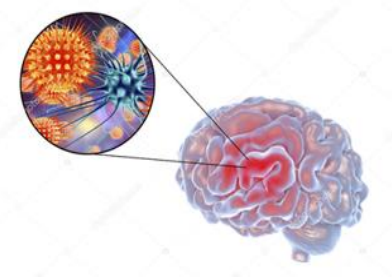
A viral inflammation which produces neurological dysfunction characterized by fever, cephalea, and impaired consciousness, caused by infection, autoimmune response, etc.

Other alteration include intense **cognitive dysfunction**, behavioral changes, **focal neurological signs**, and convulsive seizures.

Most viral encephalitis is caused by the Herpes Simplex Virus (HSV) type 1 and 2, varicela-zoster, Epstein Barr virus (EBV), measles, mumps, and enteroviruses (dependin on the continent and environmental factors).

Herpes simplex mainly affects the cerebral parenchyma in the termporal lobes, and in some cases, the fronal and parietal areas.

Pathologies at an early age: Brain damage



Viral encephalitis

The mumps virus can cause acute viral encephalitis or post-infectious encephalitis.

The influenza virus causes diffuse cerebral edema as the main component in pathogenesis.

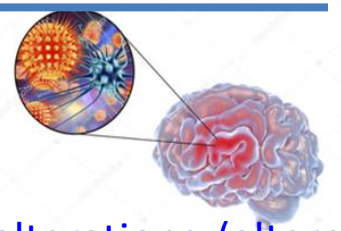
For the varicella zoster virus, the vasculitic process predominates.

The usual neurotropic pathway consists of the penetration of the virus into the motor or sensory nerve terminals reaching the ganglion or motor neuron cells.

HSV-1 encephalitis occurs during primary infection in younger children.

In older children and adults, the most common mechanism consists of viral reactivation from the latent phase in which viruses are located at the level of olfactory bulb and brainstem (protuberance and medulla oblongata).

Pathologies at an early age: Brain damage



Symptoms of encephalitis

Flu-like symptoms (fever, headache, lack of energy), in severe cases there are serious neurological alterations (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, impaired judgment, seizures, and memory loss)

In babies, it is important to pay attention to symptoms such as vomiting, body stiffness, tense or protruding fontanel and/or constant crying,

Neuropsychology of encephalitis

Alterations in memory, (retrograde amnesia) and mainly alterations in executive functions (attention, planning, supervision of behavior).

Meningitis

This involves inflammation of the meningeal membranes; it is 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 treated quickly. (Enseñat et al, 2015).

Meningitis can be caused by either a viral or bacterial infection, viral being the most common, but the most difficult to diagnose.

Bacterial meningitis is easier to detect and with vaccination in children under five years of age, the incidence of this disease has decreased considerably.

Treatment involves isolation and identification of the pathogen, as well as antibiotic treatment and in some cases, anticonvulsant medication.

Meningitis

In the acute phase, interruption in the dynamics of the CSF and spinal brain that causes elevation of intracranial pressure, hydrocephalus, cerebral edema and subdural strokes. (Enseñat et al, 2015).

The loss of self-regulation can interfere with cerebral blood circulation leading to hypoperfusion or hyperfusion especially affecting the **middle cerebral artery.**

This vascular event can alter the functionality of the pre- and postcentral gyrus and the inferior and superior parietal lobes, as well as the middle and upper temporal gyrus.

These secondary vascular events can have negative impacts (increased intracranial pressure (ICP) obstruction of the CSF in the ventricular system) causing herniation (displacing structures**) protuberance, medulla oblongata** and **cerebellum,** affecting part of the localized cranial nerves, which is why vestibular alteration and hearing loss are more frequent sequelae in children suffering from meningitis.

Meningitis

Neuropsychological alterations caused by meningitis

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 resolve over time, a proportion of children are left with permanent sequelae.

The sequelae caused by meningitis include a series of alterations in the main cognitive processes:

1. Memory
2. Processing speed
3. Language alterations

Children who have suffered from bacterial meningitis have been shown to have low, or lower than average IQs, by more than one standard deviation.

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1.1. Brain tumours

These are the most frequent type of solid tumor in pediatric ages, the second most common after the group of leukemias and lymphomas.

- The signs and symptoms will depend on both children's ages and levels of development, as well as the location and origin of the tumor.
- The most common brain tumors in the childhood stage are:
- **Medulloblastomas**
- **Cerebellar primitive neuroectodermal tumor** (located in the posterior fossa)
- Usual age of diagnosis (3 to 9 years more in boys than in girls)
- Neurotoxic effect of chemotherapeutic treatments (hippocampus, oligodendrocytes and alterations in white matter) attentional alteration, IQ, memory, processing speed, language
-

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Pathologies at an early age: Brain damage

Main 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, alteration of ocular motor skills, nystagmus and mydriasis	Hemiplegia and hemiparesis, spasticity, ataxia, adiadochokinesia and paresthesia.	Attentional disturbances, drowsiness, mental awkwardness (haze), mnesic difficulties and decrease in IQ scores Aphasia, dysarthria, akinetic mutism.	Mental nebulosity, self-esteem problems and social skills.

Pathologies at an early age: Brain damage

Neonatal ischemia-hypoxia

Perinatal asphyxia remains one of the major causes of neurological morbidity and mortality.

Derived neonatal encephalopathy is a major cause of brain damage, affecting 1-3 out of every 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 achieve a total functional recovery (Moral et al, 2019).

Fetal asphyxia decreases cerebral and systemic blood flow with decreased oxygen and glucose, reversal of aerobic to anaerobic metabolism, decreased energy production, and apoptosis with or without permanent neuronal damage.

Pathologies at an early age: Brain damage

Neonatal ischemia-hypoxia

Three forms of clinical presentation.

The mild form is characterized by total recovery in three days and without, or with minimal, sequelae of neurodevelopment without bodily hypothermia.

Moderate and severe forms lead to permanent **neurological deficits and alterations in neurodevelopment** (48%) or death (27%) after treatment with body hypothermia (Papazian, 2018).

Perinatal asphyxia causing brain damage and subsequent sequelae causes acute encephalopathy.

Neonatal encephalopathy: Neurological dysfunction syndrome affects newborns over 35 weeks of age, with an incidence estimated at 1-3/1000 live births.

Hypoxic–ischemic perinatal encephalopathy (HIE)

Set of clinical and neuropathological manifestations that occur in the RN after an episode of asphyxia. The asphyxia needs to be differentiated from the encephalopathy, since patho-physiologically they are different, although they are sequential events:

Asphyxia is a cause, while encephalopathy is an effect, asphyxia does not always produce HIE, nor is the suffocation factor found in all lesions.

This pathology has a wide spectrum of symptoms (motor alterations, muscle movements and tone, auditory dysfunctions with or without hearing loss, oculomotor alterations and dysplasia of tooth enamel).

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.

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Pathologies at an early age: Brain damage

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/

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Imagery

Image 3 https://www.vecteezy.com/vector-art/519871-man-brain-injury-character
Image 5 https://www.bbmundo.com/bebes/cero-seis/8-senales-con-las-que-un-bebe-se-comunica-antes-de-hablar/
Image 7 https://thegreattalesofthegilliams.blogspot.com/
Image 13::https://www.mercuryrx.com/post/2017/01/26/series-whats-under-the-dementia-umbrela
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Image 23:https://www.mayoclinic.org/es-es/diseases-conditions/encephalitis/symptoms-causes/syc-20356136
Image 24: https://sp.depositphotos.com/162675776/stock-photo-viral-encephalitis-illustration.html
Image 25: https://sp.depositphotos.com/162675776/stock-photo-viral-encephalitis-illustration.html
Image 26: https://sp.depositphotos.com/162675776/stock-photo-viral-encephalitis-illustration.html

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Pathology at an early age: Brain damage

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