What the pediatric lateral ventricles tell us.

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Learning objectives

The pediatric brain is increasingly being studied through a wide range of technics. The lateral ventricles, which could give us extra-information, are very important to make the correct diagnosis of the subjacent cause. To study the lateral ventricles, we have almost all the radiology techniques at our disposal: ultrasound (US), computed tomography (CT) and magnetic resonance imaging (MRI).

We propose to attend these objectives:

1. Review the anatomy of the pediatric lateral ventricles.
2. Show the lateral ventricles’ role in CSF dynamics.
3. Analyze the spectrum of pathologies related to the disturbances in the anatomy and function of the pediatric lateral ventricles.
Background

The pediatric brain is increasingly being studied through a wide range of technics. The lateral ventricles, which could give us extra-information, are very important to make the correct diagnosis of the subjacent cause. In this work, we are going to show the different morphologies that can be found in our daily routine. Fig. 1 on page 8

Anatomy of lateral ventricles:

Each lateral ventricle resembles a C-shaped structure that begins at an inferior horn in the temporal lobe, travels through a body in the parietal lobe and frontal lobe, and ultimately terminates at the interventricular foramina where each lateral ventricle connects to the central third ventricle. Along the path, a posterior horn extends backward into the occipital lobe, and an anterior horn extends farther into the frontal lobe.

Each lateral ventricle has three horns also called cornus. They can be referred to by their position in the ventricle, or by the lobe that they extend into.

The anterior horn of lateral ventricle or frontal horn, passes forward and to the side, with a slight inclination downward, from the interventricular foramen into the frontal lobe, and curves around the front of the caudate nucleus. Its floor is formed by the upper surface of the reflected portion of the corpus callosum, the rostrum. It is bounded medially by the front part of the septum pellucidum, and laterally by the head of the caudate nucleus. Its apex reaches the posterior surface of the genu of the corpus callosum.

The posterior horn of lateral ventricle or occipital horn, passes into the occipital lobe. Its direction is backward and lateral, and then medial ward. Its roof is formed by the fibers of the corpus callosum passing to the temporal and occipital lobes. On its medial wall is a longitudinal eminence, the calcar avis (hippocampus minor), which is an involution of the ventricular wall produced by the calcarine sulcus. Above this the forceps posterior of the corpus callosum, sweeping around to enter the occipital lobe, causes another projection, termed the bulb of the posterior cornu. The calcar avis and bulb of the posterior cornu are extremely variable in their degree of development; in some cases they are ill-defined, in others prominent.

The inferior horn of lateral ventricle or temporal horn, is the largest of the horns. It traverses the temporal lobe, forming a curve around the posterior end of the thalamus. It passes at first backward, sideward, and downward, and then curves forward to within 2.5 cm. of the apex of the temporal lobe. Its direction is fairly well indicated on the brain surface by the superior temporal sulcus. Its roof is formed chiefly by the inferior surface of the tapetum of the corpus callosum, but the tail of the caudate nucleus and the stria terminalis also extend forward in the roof of the temporal horn to its extremity; the tail of the caudate nucleus joins the putamen. Its floor presents the following parts: the
hippocampus, the fimbria hippocampi, the collateral eminence, and the choroid plexus. When the choroid plexus is removed, a cleft-like opening is left along the medial wall of the temporal horn; this cleft constitutes the lower part of the choroidal fissure.

The body of the lateral ventricle is the central portion, just posterior to the frontal horn. The trigone of the lateral ventricle is a triangular area defined by the temporal horn inferiorly, the occipital horn posteriorly, and the body of the lateral ventricle anteriorly. The cella media is the central part of the lateral ventricle. Ependyma cover the inside of the lateral ventricles and are epithelial cells. Fig. 2 on page 8

Cerebrospinal fluid dynamics in lateral ventricles.

Cerebrospinal fluid (CFS) acts as a cushion or buffer for the brain's cortex, providing basic mechanical and immunological protection to the brain inside the skull. The CSF also serves a vital function in cerebral autoregulation of cerebral blood flow.

The brain produces roughly 500 mL of cerebrospinal fluid per day. This fluid is constantly reabsorbed, so that only 100-160 mL is present at any one time.

CSF circulates within the ventricular system of the brain. Ependymal cells of the choroid plexus produce more than two thirds of CSF. From here, the CSF passes through the interventricular foramina to the third ventricle, then the cerebral aqueduct to the fourth ventricle. The fourth ventricle is an outpouching on the posterior part of the brainstem. From the fourth ventricle, the fluid passes through three openings to enter the subarachnoid space. The subarachnoid space covers the brain and spinal cord. There is connection from the subarachnoid space to the bony labyrinth of the inner ear making the cerebrospinal fluid continuous with the perilymph.

It has been known that CSF returns to the vascular system by entering the dural venous sinuses via the arachnoid granulations. But there is also some little absorption on interstitial perivascular spaces of brain parenchyma. This flow may play a substantial role in CSF reabsorption, particularly in the neonate, in which arachnoid granulations are undeveloped.

Morphological and functional alteration of the pediatric lateral ventricles:

Big size

It is an increased volume of ventricular system. It can be defined with bifrontal horn to intracranial diameter ratio > 0.33 or temporal horn width > 3 mm. But in the specific case of fetus we use the measurement from the atria of posterior horn to the inner ependymal layer.

The hydrocephalus is an abnormal accumulation of cerebrospinal fluid. It can be generally classified in two categories: communicating or non-communicating. Non-communicating
hydrocephalus Fig. 3 on page 9 Fig. 4 on page 10 Fig. 5 on page 11 is due to an obstruction inside the ventricle system, and , communicating hydrocephalus takes place when CFS can exit the ventricular system to the subarachnoid space without obstruction.

Little size

In new-born and infants, lateral ventricles could be small virtually undetectable but there are two main pathologies which have overall decrease of lateral ventricles volume: an increased intracranial pressure (usually diffuse brain edema), or postshunting Fig. 6 on page 12.

Deformation

In brain parenchyma it could exist cystic cavities which causes deformity in ipsilateral ventricle by expanding it. It may occur for instance in abscess in progress. In neonate this is particularly the case of citrobacter meningitis such as the case exposed Fig. 7 on page 13. In the late phase there are multiple, often septated cavities, usually bilateral, with extensive T2 hyperintensity within WM and variable edema, mass effect. Eventually, cavities may contract, causing profound WM loss and the deformation of lateral ventricles.

Intraventricular tumors could also expand and cause a one side deformation. Fig. 9 on page 15

Impression

Anything that create a mass effect in a cerebral hemisphere will compress the ipsilateral ventricle decreasing its volume and flattering it. An example which may be included in this group is the impression proced by mass effect of a massive epidural hematoma in a haemophilic patient Fig. 10 on page 16 or a supratentorial hemispheric tumor.

Traction

The lateral could suffer traction from surrounding parenchyma cavities which are connected to ventricle, subarachnoid space or both. Cases of ventricular traction could appear in aporencephalic cyst after periventricular ischemic injury and in an open lip schizencephaly. The key difference is that schizencephaly is an abnormal cleft lined with grey matter that form the ependyma of the cerebral ventricles to the pia mater. These clefts can occur bilaterally or unilaterally. Although exact pathogenesis is uncertain, it is thought most likely to be the result of abnormal neuronal migration. Some authors propose an early in utero vascular insult as the cause. The cleft extends from the ependymal surface of the brain to the pia mater, and both layers meet in the cleft: the
so-called pial-ependymal seam. The grey matter that lines the cleft is abnormal, usually representing polymicrogyria Fig. 11 on page 17.

Lobulation

The lateral ventricles could show border irregularities as the evolution of hypoxic ischemic hemorrhagic neonatal. Chronic hypoxic ischemic lesions are defined as a white matter volume loss (especially corpus callosum), with undulated ventricular borders, and secondary ventriculomegaly, which could associate cortical and deep gray or pontine and cerebellar volume loss Fig. 12 on page 18 Fig. 13 on page 19.

Primary alteration of the morphology of the walls

Some pathologies could affect specifically the ventricles' walls by changing the normal morphology. This is the case of tuberous sclerosis which associate subependymal nodules (hamartomas). It is also important to consider the giant cell astrocytoma (15% of Tuberous Sclerosis) or, for example, subependymal tumor spread in medulloblastoma.

Midline herniation

Ventricles morphology could express a midline shift as a consequence of a unilateral mass effect from pathology in the frontal, parietal or temporal region, such as acute cerebral medial artery infarct, extra or intracranial haemorrhage or tumour Fig. 15 on page 21. As consequences, it could happen contralateral hydrocephalus due to obstruction of the foramen of Monro or anterior cerebral artery territory infarct due to compression of its branches.

Congenital anomalies

Lateral ventricles morphology depends principally on adjacent parenchymal structures. Some alterations give specific morphologies as partial or complete absence of corpus callosum that produce parallel separate lateral ventricles and colpocephaly Fig. 16 on page 22.

Atrial diverticulum could also modify the morphology, in which a massive ventricular dilatation causes stretching and dehiscence of the fornix with formation of unilateral or bilateral pial pulsion diverticula of the inferior medial wall of the atrium. Enlargement of the pial pouch creates a dramatic subarachnoid cyst that may herniate downward through the incisura into the lateral mesencephalic, precentral cerebellar, and superior vermian cisterns where it displaces the brain stem, vermis, and fourth ventricle.

Post-surgical deformity
Surgical interventions could affect normal morphology. Decompressive craniectomy could allow brain parenchyma to expand to reduce intracranial pressures. It affects especially to the anterior horn in frontal craniectomy, expanding them Fig. 17 on page 23 Fig. 18 on page 24.
Fig. 1

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Fig. 2

CT of two normal infants showing the wide range of normal volumes.

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Fig. 3

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Fig. 4

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Fig. 5

Posterior fossa medulloblastoma causing IV ventricle compression with supratentorial hydrocephalus and transependymal edema.

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Small size

Hydrocephalus (caused by a posterior fossa pylocytic astrocytoma) treated with ventricular drainage. We see collapse of lateral ventricles due to a hyperfunction shunt, producing a slit ventricle syndrome.

Fig. 6

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Fig. 7

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Fig. 8

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Fig. 9

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Fig. 10

Massive epidural hematoma in a haemophilic patient after trauma producing collapse of ipsilateral ventricle.

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Cleft extending from the ependyma to the pia mater entirely lined by heterotopic grey matter. Open lip schizencephaly.

Fig. 11

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Fig. 12

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Brain MR shows the evolution of hypoxic ischemic neonatal injury. It develop hydrocephalus secondary to haemorrhagia, treated with ventriculoperitoneal shunt (red arrow). Brain parenchima with white matter loss. The morphology of the lateral ventricles is lobulated.
Two cases of multicystic periventricular leukomalacia. The morphology of the lateral ventricles is lobulated due to white matter volume loss.

Fig. 13

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Fig. 14

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Fig. 15

CT after a severe brain injury showing intraventricular and intraparenchymal bleed producing midline herniation toward contralateral side.

Massive epidural hematoma causing a midline herniation after a mild trauma in a patient with a severe coagulopathy.

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Lateral ventricles show a parallel disposition and enlargement of the occipital horns, due to a hypogenesis of corpus callosum (yellow arrow).

Fig. 16

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Fig. 17

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Fig. 18

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Findings and procedure details

Lateral ventricles could be studied through the three main techniques. Ultrasounds is the optimal one for screening and some parenchymal pathology in new-born. Computed tomography scan allows better spatial resolution and a complete study of the head. Magnetic resonance gives better soft tissue contrast. We use essentially T2weighted sequences to examine the ventricular system.
Conclusion

As it has been shown, the lateral ventricles adopt a broad range of patterns. That is why it is necessary for the radiologist to know which they are and what they represent.
References


