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REVIEW PAPER

The ventricular system of the brain: a comprehensive review of its history, anatomy, histology, embryology, and surgical considerations

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Abstract
Introduction The cerebral ventricles have been recognized since ancient medical history. Their true function started to be realized more than a thousand years later. Their anatomy and function are extremely important in the neurosurgical panorama.
Methods The literature was searched for articles and textbooks of different topics related to the history, anatomy, physiology, histology, embryology and surgical considerations of the brain ventricles.
Conclusion Herein, we summarize the literature about the cerebral ventricular system.

Keywords Ventricles · Brain · Cerebrospinal fluid

Introduction

The presence of a "hollow" inside the head had been known since ancient times. The origin, content, and function of this hollow, however, was ambiguous. In the third century BC, Herophilus and Erasistratus were able to perform human dissections for the first time in history. Their work lead to a better understanding of the ventricles and gave full anatomic description of four "small stomachs," the ventricles, and their communications. However, they believed that the function of these ventricles was to convert the vital spirit (*pneuma zooticon*), which circulates with the blood from the heart, to the animal (from *anima* means soul) spirit (*pneuma psychikon*). This process was believed to produce thoughts, feelings, and emotions. This belief became more established at the time of Galen. Besides that, Nemesius of Emesa attributed the imagination and its connection with the five senses to the ventricle of the frontal lobe. The ventricles, which became known as "*cellulae*" until the Renaissance, were counted from three to five in number, and many medical illustrations attempted to demonstrate their appearance in the brain. One of these was by Albertus Magnus in 1506, which represented three cavities surrounded by thin brain tissue. He believed that the functions of these cavities were, from anterior to posterior, imagination, reasoning, and memory. Around 1504, Leonardo da Vinci was the first to make an accurate depiction of the ventricles by performing the first known ventriculography by injecting molten wax into the ventricles of an ox. However, he also followed the same older belief regarding the functions of the ventricles with only slight modifications. Following Da Vinci's illustrations of the ventricles, anatomists of the Renaissance started to give more attention on studying these cavities, but even in Vesalius's *Fabrica*, the ventricles were described as air containing spaces that fill during inspiration, and contain the animal spirit. In the same period of time, some anatomists, including Nicolo Massa in 1569, opposed the widely held belief of the content of the ventricles. It took almost a century until Constanzo Varolio and Francis Glisson described humor content instead of the classical spirit. This did not end the debate, however. In 1764, Domenico Felice Antonio Cotugno was the first to discover cerebrospinal fluid and to describe the continuity between the ventricles and subarachnoid space. His findings were later confirmed by

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François Jean Magendie, whose contribution to the discovery of the foramen of Magendie will be described below in addition to the history of ventricular communications [1].

Anatomy

The ventricular system of the brain consists of four freely communicating, cerebrospinal fluid (CSF) filled cavities: the two lateral ventricles, the third ventricle, and the fourth ventricle (Figs. 1 and 2).

The lateral ventricles are C-shaped cavities that lie deep in each cerebral hemisphere. This shape is thought to be a related to the developmental expansion of the frontal, parietal, and occipital lobes that displace the temporal lobe inferiorly and anteriorly [2].

Lateral ventricles

Each lateral ventricle is divided into a body and atrium, as well as anterior (frontal), posterior (occipital), and inferior (temporal) horns. Each of these parts has medial and lateral walls, a roof, a floor, and an anterior wall [3].

The body occupies the parietal lobe and extends from the posterior edge of the foramen of Monro to the point where the septum pellucidum disappears and the corpus callosum and fornix meet. The lateral wall is formed by the caudate nucleus superiorly and the thalamus inferiorly, separated by the striothalamic sulcus, the groove in which the stria terminalis, and the thalamostriate vein course. The medial wall is formed by the septum pellucidum superiorly and the body of the fornix inferiorly. The floor is formed by the thalamus and the roof by the body of the corpus callosum [2–6].

The body widens posteriorly were it becomes continuous with the atrium. The atrium communicates with the body anteriorly and above the thalamus, with the inferior horn anteriorly and below the thalamus and with the posterior horn posteriorly. The atrium and the occipital horn form a triangular cavity, with the apex in the occipital lobe and the base anteriorly on the pulvinar of the thalamus, which constitutes the anterior wall of the atrium. The roof of the atrium is formed by the body, splenium, and tapetum of the corpus callosum. The floor is formed by the collateral trigone, a triangular area overlying the posterior end of the collateral sulcus. The medial wall is formed by superior and inferior prominences named the bulb of the corpus calllosum and calcar avis, respectively. The superior prominence (bulb of the corpus) is formed by the large bundle of fibers called the forceps major, and the inferior prominence (calcar avis) overlies the deepest part of the calcarine sulcus. The anterior part of the lateral wall is formed by the caudate nucleus and the posterior part by fibers of the tapetum of the corpus callosum [2, 3, 5–7].

The occipital (posterior) horn curves posteromedially from the atrium towards the occipital lobe to form a triangular or diamond-shaped cavity. It may vary in size from absent to extending far into the lobe and may vary from side to side. The medial wall is formed by the two prominances: the bulb of the corpus callosum superiorly and the calcar avis inferiorly. The roof and lateral wall are formed by the tapetum of the corpus callosum, overlaid laterally by the optic radiation, and then the inferior longitudinal fasciculus. The floor is formed by the collateral trigone [3, 5, 6].

The temporal (inferior) horn, the largest part of the lateral ventricles, extends downward and posteromedially around the pulvinar, and then turns anteriorly in the medial part of the temporal lobe to end 2.5 cm of the temporal pole, just behind the amygdaloid nucleus, which constitutes the anterior wall. The floor is formed by the hippocampus or pes hippocampus and laterally by the collateral eminence, the prominence overlying the collateral sulcus. The medial part of the roof is formed by the inferior surface of the thalamus and the tail of the caudate nucleus separated by the striothalamic sulcus. The lateral part is formed by the tapetum of the corpus callosum, which also sweeps inferiorly to form the lateral wall of the temporal horn. The medial wall is formed by the choroidal fissure, a narrow cleft situated between the inferolateral part of the thalamus, and the fimbria of the fornix [2, 3, 5, 6].

The frontal (anterior) horn extends anteriorly from the interventricular foramen into the frontal lobe. It has a roughly triangular shape on the coronal section. The medial wall is formed by the septum pellucidum, separating the frontal horns on both sides. The anterior wall and roof are formed by the genu of the corpus callosum. The lateral wall is formed by the head of the caudate nucleus, and the narrow floor by the rostrum of the corpus callosum [2, 3, 5, 6].

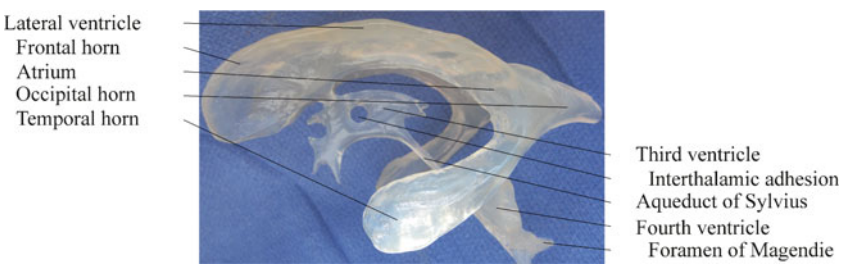

Fig. 1 Lateral view of a glass cast of the ventricular system (lateral view)

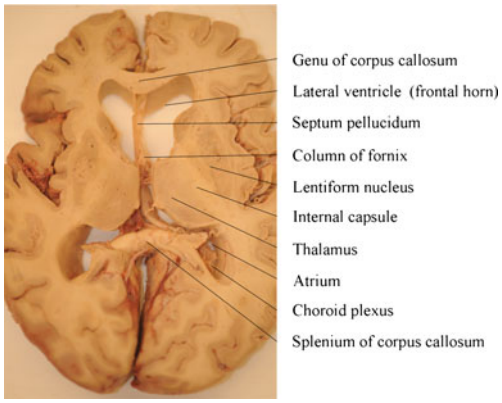

Fig. 2 Cadaveric dissection of the brain at the level of the basal ganglia and thalamus (axial section)

Kremen et al. [8], in 2010, revised and reported on previous studies on the heritability of the size of the lateral ventricles in twin cases. They concluded that lateral ventricular volume is heritable, and that its heritability does increase with age from childhood to at least late middle age.

As with the case of the cerebral hemispheres themselves, asymmetry of the lateral ventricles exists. The incidence of this asymmetry is 5–12 %, which make it relatively a common entity. Asymmetry of the lateral ventricles can be expected with an underlying pathological condition, but in the absence of underlying pathology, the presence of this asymmetry raises many questions [9]. However, the underlying cause of this asymmetry is still obscure. In a study by McRae et al. [10] in 1968, they found that the left occipital horn was longer than the left in 57 % of cases, and the right occipital horn was longer in 13 %. According to them, this, in most, is related to brain dominance, as the left occipital horn is longer in right-handed adults, and the right is longer in left-handers. Strauss and Fitz [11], in 1983, studied the occipital horns of 75 subjects, age 5 months to 18 years. They found that in 29 (38.7 %) of the cases, the left horn was longer than the right and the right was longer in 13 (17 %) of the cases. Furthermore, they reported an association between early brain lesions in the first year of life and this asymmetry, mainly in the cases of a longer right occipital horn, as 85 % of them reported history of brain injury in early life, compared to 46 % with longer left horns. In 1983, in a sonographic examination of brains of 66 human neonates with gestational ages of 28–44 weeks, Horbar et al. [12], reported asymmetry in the body of the lateral ventricle. This asymmetry could be found as early as the first postnatal day in infants with a gestational age as low as 28 weeks. The body of the left lateral ventricle was larger in 31.8 % of the neonates, whereas the right body was larger in only 6.1 %. According to them, this asymmetry could result from intrauterine or postnatal compression of the skull. Erdogan et al. [13] studied the effects of gender and handedness on the size and asymmetry of the lateral, third, and fourth ventricle. They stated that both handedness and gender-handedness interaction were significant factors influencing the volumes of the right and left lateral, third, and fourth ventricles, but gender was not significant. The mean volumes of the right lateral and third ventricles were significantly larger in right-handers than in left-handers, but the mean volumes of the left-lateral and fourth ventricles did not exhibit any significant handedness difference. Degree and character of right-to-left asymmetry also changes with various kinds of diseases and abnormalities. For instance, in patients with autism, highly localized reductions in volume in the left frontal and occipital horns of the lateral ventricles have been reported [14]. In addition, the degree of asymmetry may change in patients with schizophrenia in relation to sex [15]. Kiroglu et al. [9] studied the association of the asymmetry of the lateral ventricle with clinical and structural pathologies, and they concluded that an asymmetric lateral ventricle represents a normal anatomic variation and has a relatively lower risk for accompanying disorders in cases of mild or moderate degree asymmetries without septal deviation or diffuse enlargement. However, with severe degree of asymmetries and the presence of septal deviation or diffuse enlargement, it should not be considered normal.

Foramen of Monro

The interventricular foramen (of Monro) is named, according to most authors, after Alexander Monro Secundus (1733–1817). Some authors [16], however, ascribe it to Alexander Monro Primus (1697–1767). In his earliest work, in 1783, Monro (Secundus) [17] stated that this foramen has been described before, but never illustrated. However, a thorough investigation by Sharp [18] in 1961 doubts his claim. At the end of his study, Sharp concluded, "The present writer feels strongly that there is no justification for the retention of the eponymous term 'foramen of Monro' as a synonym for the interventricular foramen of the modern terminology for two reasons: firstly, because Monro added nothing of value to the pre-existing description of the foramen, and secondly because he actually misinterpreted the nature of the communication between the third and lateral ventricles" [18].

The interventricular foramen forms the communicating canal between the lateral ventricle on either side and the third ventricle at the junction of the roof and the anterior wall. It has a diameter of 3–4 mm, and is bounded anteriorly by the junction of the body and the columns of the fornix and posteriorly by the anterior pole of the thalamus, and has a posterior concavity. The size and shape of the foramina of Monro depend on the size of the ventricles: If the ventricles are small, each foramen is a crescent-shaped opening bounded anteriorly by the concave curve of the fornix and posteriorly by the convex anterior tubercle of the thalamus. As the ventricles enlarge, the foramen on each side becomes rounder. The structures that pass through the foramen are the choroid

plexus, the distal branches of the medial posterior choroidal arteries, and the thalamostriate, superior choroidal, and septal veins [3, 6]. Congenital atresia of the foramen of Monro is infrequently reported in literature [19–21].

Third ventricle

The third ventricle is a narrow, funnel-shaped, unilocular, midline cavity located at the center of the head. It communicates with the lateral ventricles through the interventricular foramen of Monro on its anterosuperior aspect, and with the cerebral aqueduct of Sylvius on its posteroinferior aspect.

The roof of the third ventricle forms a gentle upward arch, extending from the foramen of Monro anteriorly to the suprapineal recess posteriorly. It has four layers: one neural layer, the uppermost layer, formed by the body of the fornix anteriorly and by the crura and the hippocampal commissure posteriorly, the septum pellucidum is attached to the upper surface of the body of the fornix. Below the neural layer, there are two thin membranous layers of tela choroidea and a layer of blood vessels between the sheets of tela choroidea. The anterior wall of the third ventricle is formed, from superior to inferior, by the diverging columns of the fornix, foramina of Monro, the transversely orientated anterior commissure, lamina terminalis, optic recess, and optic chiasm [3].

The anterior half of the floor is formed by diencephalic structures, and the posterior half is formed by mesencephalic structures. When viewed from below, the structures forming the floor include, from anterior to posterior, optic chiasm and its recess, the infundibulum of the hypothalamus and its narrow funnel-shaped recess, the tuber cinereum of the hypothalamus, the mamillary bodies, the posterior perforated substance, and most posteriorly the part of the tegmentum of the midbrain located above the medial aspect of the cerebral peduncles. At the junction of the floor and the anterior wall, a small, angular, optic recess extends into the optic chiasm [2, 3].

In their experimental study on the normal anatomy and some pathological changes of the third ventricle, Corrales et al. [22] have described three portions in the floor of the third ventricle: The first or premammillary portion is the thinnest and extends from the infundibulum to the premammillary sulcus. The second or interpeduncular portion is slightly thicker and extends from the postmammillary recess to the posterior border of the interpeduncular space. The third or peduncular portion is the thickest segment and corresponds to the portion lying on the cerebral peduncles and it extends to the aqueduct of Sylvius.

The optic recess is a small, angular cavity situated between the junction of the anterior wall and the floor of the third ventricle. Its floor or posteroinferior wall is formed by the optic chiasm, which at the same time separates its cavity from the infundibulum. Its anterior or anterosuperior wall is formed by the lamina terminalis, which has a posterior curving insertion in the superior aspect of the optic tract. The insertion of the lamina terminalis may be more anteriorly or posteriorly located in the superior surface of the chiasm. The optic recess extends laterally in both sides following to a small extent the posterior lateral curving insertion of the lamina terminalis in the optic tract. This fact makes the optic recess broader as compared with the infundibulum. The lateral border of the optic recess is formed by the inferior part of the "area praeoptica" of the hypothalamus. At its superoposterior aspect, the optic recess communicates with the cavity of the third ventricle [22]. Normal variations in the anatomy of the optic recess and subsequently optic chiasm have been reported [23]. In addition, conditions of persisting embryonic infundibular recess, albeit rare, have been recognized [24].

The posterior wall of the third ventricle is formed, from above to below, by the suprapineal recess, the habenular commissure, the pineal body and its recess, the posterior commissure, and the aqueduct of Sylvius. The suprapineal recess projects posteriorly between the upper surface of the pineal gland and the lower layer of tela choroidea in the roof. The pineal recess projects posteriorly into the pineal body between the two laminae of the pineal gland [3].

Suprapineal recess is usually a small cavity that lies above the habenular commissure. Posteriorly, it extends near the corpus callosum to the vicinity of the quadrigeminal cistern. It is 2–3 mm in height and length. Dilation of the suprapineal recess is the most common variation in the third ventricle, and according to Krokfors et al., it is present in 4 % of the cases they studied. Occasionally, both the height and the length may exceed 10 mm, and lengths even up to 40 mm have been encountered. A large suprapineal recess has not been considered to have any pathological significance. In the case of hypertensive hydrocephalus, the suprapineal recess may become greatly distended, and it has indeed been termed the "pressure diverticulum" of the third ventricle [25].

The upper part of the lateral wall of the ventricle is formed by the medial surface of the anterior two thirds of the thalamus, and the lower part is formed by the hypothalamus anteriorly and the subthalamus posteriorly. The boundary between the thalamus and hypothalamus is marked by the ill-defined hypothalamic sulcus, which extends horizontally on the ventricular wall between the interventricular foramen of Monro and the cerebral aqueduct [2]. The superior limit of the thalamic surfaces is marked by narrow, raised ridges, known as the striae medullaris thalami. These striae extend forward from the habenulae along the superomedial surface of the thalamus near the attachment of the lower layer of the tela choroidea [3]. The upper halves of the lateral walls of the third ventricle are joined by an interthalamic adhesion, or massa intermedia, a band of gray matter, which extends from one thalamus to the other. It is present in approximately 75 % of brains and located 2.5–6.0 mm (average, 3.9 mm) posterior to the foramen of Monro [2, 3].

Aqueduct of Sylvius

As with most of the brain structures, the first introduction of a canal between the third and fourth ventricles was made by Galen. According to some authors [26], the first recent illustration of the cerebral aqueduct can be traced by to Leonardo da Vinci. However, the first description of this canal was done by Berengarius Carpensis in 1521. Later on, Vesalius clearly described the aqueduct in his book, *Fabrica*, in 1543. Jacobus Sylvius, a famous French anatomist and a blind follower of Galen, was born in 1478, and he was the teacher of Vesalius. His book *Isagoge* was published in 1555, and in the book, we can find a clear description of the aqueduct. This later publication of his work has obscured the merits of Sylvius, who had probably seen the cerebral aqueduct much earlier than his student Vesalius. In 1663, a Dutch anatomist named François de le Böe (or Franciscus Sylvius in the Latinized form) also described the same structure in his book *Disputationes* and probably the term aqueduct of Sylvius (aka aqueductus Sylvii) can be ascribed to him. The term aqueduct, derived from the latin word aqueductus, was first used by Arantius in 1587 [27].

In 1855, and based on his histological studies, von Gerlach [28] subdivided the Sylvian aqueduct into three parts and proclaimed that the shape of the aqueduct is different from site to site on cross sections. Turkewitsch [29, 30], on the other hand, recognized five portions, namely, the aditus ad aquaeductum or aditus aquaeducti, anterior part, ampulla, genu, and posterior part. The anterior part and the genu form the first and second constrictions surrounding the enlarged ampulla, and formed by the superior and inferior colliculi, respectively [26, 31]. Woollam and Millen [32] considered this subdivision rather elaborate and suggested a division into three parts: Pars Anterior, Ampulla, and Pars Posterior [33]. The aqueduct is the narrowest part of the ventricular system and the most common site for blockade, and it is approximately 18 mm long [4]. The antrum of the aqueduct is triangular in shape, with the floor located dorsally, and formed by the posterior commissure; the other two limbs are formed by the central gray matter of the midbrain, mainly by the two red nuclei [3, 32]. Broman [34] and Bickers and Adams [35] stated the size of the lumen of the aqueduct decreases progressively from the second fetal month to birth due to the effect of the surrounding nuclear system and neural fibers development. Spiller [36] found that the aqueductal lumen continues to decrease in size with age much like the central canal of the spinal cord. Flyger et al. [33], and based on experimental study, concluded that the CSF pressure within the lumen of the aqueduct does not allow any shrinkage and that the size increases with age. They did not mention, however, whether this is due to general old age atrophy or to a reduction in the size of the surrounding nuclear masses and fiber tracts. They also included that the cross-sectional area of the aqueduct differs from site to site and patient to patient and may range from 0.40 to 9.84 mm^2 [33]

Fourth ventricle

The fourth ventricle is a broad, tent-shaped midline cavity located at the center of the posterior fossa between the brain stem and the cerebellum. It lies ventral to the cerebellum, dorsal to the pons and upper half of the medulla, and medial to the cerebellar peduncles. It is connected rostrally with the cerebral aqueduct, caudally with the central canal of the spinal cord, inferoposteriorly with the cisterna magna through the foramen of Magendie, and laterally with the cerebellopontine angles through the foramena of Luschka. The fourth ventricle has a roof, a floor, and two lateral recesses. Its widest part is at the pontomedullary junction where a lateral recess on both sides extends to the lateral border of the brain stem [2, 37].

The apex of the tent-shaped cavity extends from its narrow rostral end at the level of the aqueduct to its narrow caudal end at the level of the foramen of Magendie. Between these two ends, the roof expands laterally and posteriorly to reach its peak (fastigium) at the level of the lateral recesses, before it tapers again caudally. The fastigium divides the roof into superior and inferior parts. The superior part is formed by the medial borders of the two superior cerebellar peduncles and a connecting sheet of white matter called the superior medullary velum, which is continuous with the white matter of the cerebellum and is covered dorsally by the lingula of the superior vermis. The inferior part of the roof is formed by the inferior medullary velum, which consists of a thin sheet devoid of nervous tissue and formed by the ventricular ependyma and its posterior covering of the pia mater of the tela choroidea. At the level of the inferior medullary velum, just below the nodule of the cerebellum, a median aperture known as the foramen of Magendie, connects the ventricle with the cisterna magna [2, 4, 37].

The floor of the fourth ventricle is diamond, or rhomboid in shape, called the rhomboid fossa. The rostral two third of the floor lies behind the pons, and the caudal third lies behind the superior half of the medulla. The upper tip of this diamond cavity is located at the level of the aqueduct and the inferior end, the obex, at the antrum of the central canal, opposite to the foramen of Magendie. The lateral angles open through the lateral recesses and the foramina of Luschka. The floor is divided into three parts: superior or pontine part, intermediate or junctional part, and inferior or medullary part. The superior part is triangular in shape, it apex is formed by the aqueduct, the base is formed by imaginary line connecting the two cerebellar peduncles, and the two limbs are formed by the medial surface of the two cerebral peduncles. The intermediate part is a strip between the lower margin of the cerebellar peduncles and the site of insertion of the tela choroidea at the taenia of the fourth ventricle just below the lateral recesses. The inferior part is triangular in shape, its apex lies caudally and opposite to the foramen of Magendie, the base of the triangle is at the lower margin of the intermediate part, and the

lateral limbs are formed by the taenia of fourth ventricle. The floor is divided longitudinally from the rostral tip to the caudal tip into symmetrical halves by the median sulcus. On each side of this sulcus, there is an elevation, the medial eminence, which is bounded laterally by another sulcus, the sulcus limitans. Lateral to the sulcus limitans, there is an area known as the vestibular area overlying the vestibular nuclei. The sulcus limitans is discontinuous, and it is more prominent at two points, or dimples, in the pontine and medullary parts of the floor. The pontine dimple is called the superior fovea, and the medullary dimple is called the inferior fovea. At the level of the superior fovea, each medial eminence is represented by the facial colliculus, a slight swelling produced by the fibers from the motor nucleus of the facial nerve looping over the abducens nucleus. Caudal to the inferior fovea, each medial eminence has three triangular areas: The most medial is the hypoglossal triangle, overlying the hypoglossal nucleus. Lateral to this is the vagal triangle, overlying the dorsal motor nucleus of the vagus. The third is the area postrema, a narrow area between the vagal triangle and the lateral margin of the ventricle, just rostral to the opening into the central canal. The inferior part of the vestibular area also lies lateral to the vagal triangle. Strands of nerve fibers, the stria medullaris, derived from the arcuate nuclei, emerge from the median sulcus and pass laterally over the medial eminence and the vestibular area and enter the inferior cerebellar peduncle to reach the cerebellum. At the rostral tip of the sulcus limitans, at the lateral margin, there is a bluish-gray area, produced by a cluster of nerve cells containing melanin pigment and norepinephrine; the cluster of cells is called the substantia ferruginea and is more commonly known as locus coeruleus [2, 4, 37].

The lateral recesses were discovered by Bochdalek in 1849 and mistakenly thought to be blind extensions of the fourth ventricle, but they were later correctly revealed as channels communicating with the subarachnoid space by Luschka in 1855. The lateral recesses are narrow, curved pouches formed by the union of the roof and the floor of the fourth ventricle. They extend laterally below the cerebellar peduncles, which open through the foramina of Luschka into the cerebellopontine angle along the V-shaped cerbellopontine fissure. The ventral wall of each lateral recess is formed by the junctional part of the floor and the rhomboid lip, a sheetlike layer of neural tissue that extends laterally from the floor and attach to the tela choroidea to form a pouch at the center extremity of the lateral recess. The dorsal wall is formed by the peduncle of the flocculus interconnecting the inferior medullary velum and the flocculus. As the lateral recess extends towards the cerebellopontine angle, the contribution of the inferior medullary velum and tela choroidea reduces and the rhomboid lip takes its place. The rostral wall is formed by the caudal margin of the cerebellar peduncles, and the caudal wall is formed by the tela choroidea [37, 38].

In 1828, François Magendie [39] presented his findings regarding the cerebrospinal fluid at the Academy of Sciences in Paris, titled *Mémoire Physiologique sur le Cerveau*. His descriptions included an opening in the midline of lower part of the roof of the fourth ventricle, later named after him, that is variable in size and wide enough to admit the point of the finger [40, 41]. The presence of this opening has been confirmed later by Luschka [42], Key and Retzius [43], and others. However, it has been also denied by many authors, including Todd [44] in 1847, Virchow [45] in 1854, Reichert [46] in 1861, and many others. In 1931, Rogers [40] documented this controversy and confirmed the presence of this aperture, but stated that it is not a normal foramen, but rather a deformity in the roof of the ventricle. He believes, however, that his finding does not contradict with Magendie's [40].

Hubert von Luschka, a German anatomist, was the first to describe the foramina of Luschka in 1855 [42]. Luschka commented and confirmed the presence of the foramen of Magendie and described an open communication between the fourth ventricle and the subarachnoid space in the cerebellomedullary and cerebellopontine cisterns at the outer margins of the fourth ventricle. His findings, although denied by come authors, were later confirmed by many anatomists including Key and Retzius [43] in 1875 [38, 47]. The rhomboid lip, which is initially caudal, becomes the medial lip of the foramen of Luschka. Immediately anterior to the foramen sites the origins of the glossopharyngeal and vagus nerves, whereas the acoustic striae, cochlear nerve, and flocculus are just anterosuperior [48].

Histology

The term ependyma, which was first introduced by Virchow more than a hundred years ago, is used to describe the special type of cuboidal to columnar epithelial cells that line the ventricular system of the brain and central canal of the spinal cord [49]. The ependymal lining of the cerebral ventricles constitutes a layer of heterogeneous cells that are variably modified. In the lateral ventricles, two main types of ependymal cells can be identified: ciliated and nonciliated cells. These cells lack the characteristic tight junctions presenting in the choroid plexus. In the ventrolateral wall of the third ventricle, a third type of cells can also be identified, the tanycytes, which were first described by Horstmann in 1954. Tanycytes, of which almost four types have been identified, have unique elongated cytoplasmic processes that extend from the ventricular lining toward the surrounding neuropils, where they enwrap blood vessels or terminate on neurons, glia, or the external glial limitans. Their function was of interest to the scientists after their suggested role in neuroendocrine connection between the CSF and the hypophysial-portal vasculature to affect the adenohypophesial function along with the

hypothalamus. This function was mainly attributed to the tanycytes located along the floor and lateral walls of the fundibular recess. Some authors suggested that the hypothalamic hormones first go the CSF and then to the portal system by way of the tanycytes. Others suggested a complementary function to the hypothalamus, as the tanycytes may selectively absorb certain substances from the CSF (e.g., endorgan hormones) and transport them to vessels or neurons that affect the function of the adenohypophysis. A counter-transport function may also present, where substances from the neuronal or vascular secretion may be transported to the CSF. However, the definitive evidence and importance of this function are still not clear. Other possible function of the tanycytes in the brain may include modifying the ionic concentrations in the extracellular space of the periventricular zone and, hence, the membrane properties of neuronal processes. Beneath the ependymal layer, the subependymal glial cells are found, where the ependymal cells exhibits numerous infoldings that interdigitate with adjacent astrocyte processes and blood vessels and contributes to the formation of the blood–brain barrier. However, this barrier does not present in ever part of the ventricular system. In certain parts along the third and fourth ventricles, the barrier is ineffective or totally absent, which allows almost free communication between these structures and the blood. These structures, which are collectively known as circumventricular organs, include the pineal gland, median eminence, subfornical organ, area postrema, subcommissural organ, organum vasculosum of the lamina terminalis, and posterior lobe of the pituitary gland [50–53].

Embryology

Following neural tube closure, three dilatations, the primitive brain vesicles, are formed on the cephalic end of the neural tube. These vesicles, from cephalic to caudal, are the prosencephalon (forebrain), mesencephalon (midbrain), and rhombencephalon (hindbrain). At the fifth week of gestation, the prosencephalon gives rise to the telencephalon (cerebral hemispheres) and the diencephalon. The rhombencephalon also gives rise to the metencephalon (cerebellum and pons) and the myelencephalon (medulla oblongata). Cavities remain inside these developing vesicles, communicating with the lumen of the neural tube and represent the future ventricles. The cavity of the rhombencephalon is the fourth ventricle, the cavity of the diencephalon is the third ventricle, and those of the telencephalon are the lateral ventricles. The lumen of the mesencephalon connects the third and fourth ventricles and later becomes narrower and known as the cerebral aqueduct [54].

During the early phase of their development, the ventricles undergo massive expansion with faster growth compared to the surrounding brain tissue. Upon reaching the maximum ventricle/brain ratio, with the ventricle assuming the adult size, the brain begins to outpace the ventricular growth and leads to a change of the ventricular configuration to the adult form. This process proceeds in a caudal to rostral direction [55].

The precise position and unique shape of the brain ventricles is proposed to be controlled directly or indirectly by a number of patterning genes and their products. These genes received more extensive study in animal models, including chick embryos and zebrafish. One of these genes is the ventral neural signaling morphogen Sonic Hodgehog (*Shh*) that is secreted by the notochord. Early separation of the notochord from the brain may lead to loss of Shh expression and subsequent ventricular collapse. Other examples include *h1x1* gene, and *zic* family of genes. The mechanism by which these genes might affect the ventricular pattern is not well understood. One theory, introduced by His, proclaimed that the shape of the ventricles is a result of uneven cellular proliferation, migration, and differentiation throughout the neural tube, and controlled by the patterning genes intrinsic to different brain regions. Programmed cell death has also been found to counter the effect of cellular proliferation and control the proper ventricular formation. Beside this spatial morphogenesis of the ventricles, much specific tissue changes are also genetically regulated. This includes cells shape changes and cytoskeleton formation, development of the intercellular adhesion and tissue maintenance, and the epithelial anchoring and support with the extracellular matrix formation. The embryonic CSF, which is suggested to be secreted by neuroepithelial and non-neuroepithelial (vascular) sources, constitutes another important factor in the development of the ventricles. It plays this role mainly by creating a continuous intraluminal pressure that inflates the ventricles. It also contains large number of proteins and growth factors that affects the surround cells proliferation and differentiation [55].

Surgical considerations

Access to the ventricular system is one of the most utilized surgical approaches in neurosurgery. Access is indicated whenever there is a need to assess intracranial pressure, decompress the ventricular system of CSF, gain access to the ventricular system as a route for delivery of medication, and gain access as a route for access to intraventricular pathology.

Ventricular access

Access to different parts of the ventricles is achieved using one of the following anatomical landmarks, which all were described prior to the development of modern neuroradiology (Fig. 3).

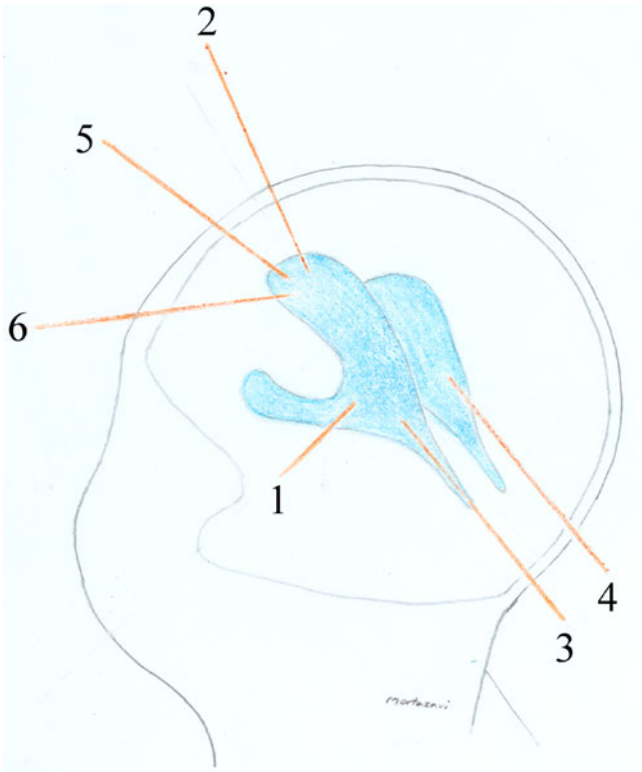

Fig. 3 Schematic illustration of all the extra-calvarial ventricular access points: *1* Keen, *2* Kocher, *3* Dandy, *4* Frazier, *5* Kaufman, and *6* Tubbs

Keen's point

Keen [56] was the first to describe the external anatomical landmarks for ventricular puncture in 1890. Keen's, or the posterior parietal point, is located 2.5–3 cm posterior and 2.5–3 cm above the pinna of the ear. It is used as an entry point for the parietal burr hole through which the catheter is placed to reach the trigone of the lateral ventricle. To reach this point, the catheter is placed perpendicular to the cortex, with a slight cephalic direction, for about 4–5 cm [57, 58].

Kocher's point

Although not clear, this term might be named after the Swiss physician Emil Theodor Kocher (1841–1917). However, the first known ventriculostomy through this point is attributed to Tillmanns [59] in 1908. Since then, many efforts have been directed towards enhancing the accuracy of this procedure using guided methods [60]. Kocher's, or the coronal point, is the most common site for drain placement. It is located 1–2 cm anterior to the coronal suture in the midpapillary line, or 11 cm posterior from the glabella and 3–4 cm lateral from midline [61, 62]. The laterality is to be a safe enough distance from the bridging veins. It can also be defined by the intersection of the two perpendicular lines: anteroposterior midpapillary line and a horizontal line line starting at the midpoint between the external auditory meatus and the lateral canthus of the ipsilateral eye (Fig. 4). This allows direct access to the frontal horn of the lateral ventricle from a point that lies anterior to the motor strip and posterior enough to avoid incision on the forehead [61]. The right side is still preferred even in cases of left-sided hemorrhages. In patients with midline shift, drain placement tends to be more accurate if the surgical side was ipsilateral toward the midline shift [63]. To reach the target, the catheter is inserted towards the intersection between two imaginary lines: a line that runs backward from the ipsilateral medial canthus and a line extending coronally from the ipsilateral tragus. It is inserted from 5 to 6 cm [64]. The position of the tip into the frontal horn or near the foramen of Monro decreases the risk of obstruction by the choroid plexus [58].

Dandy's point

In 1918, Dandy [65] described a technique involving occipital ventricular horn puncture for air ventriculography in pediatric cases. Dandy's point is a common entry point for an occipital burr hole. It is located 3 cm above and 2 cm lateral to the inion. In the pediatric population, this can be related to the lamboid suture at the midpapillary line. The catheter is placed perpendicular to the cortex, with a slight cephalic direction, for about 4–5 cm. This trajectory has a higher risk of visual impairment [57].

Frazier's point

Frazier's or the parieto-occipital point is probably named after Frazier and Gardner who, in 1928, described a burr hole for

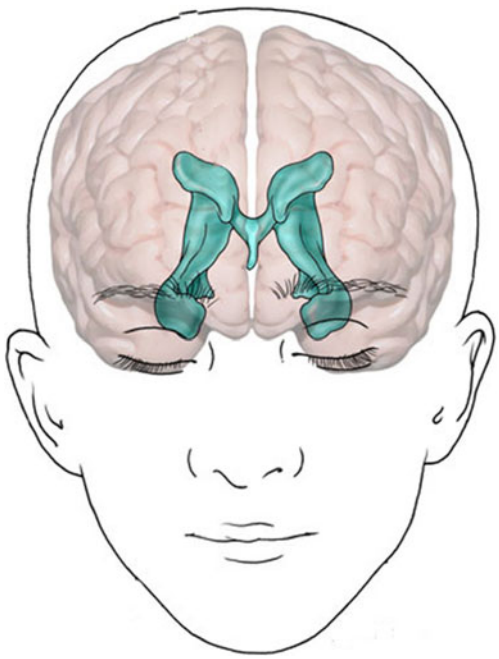

Fig. 4 Frontal view of the ventricular system in relationship to important facial landmarks serving as a reference for ventricular access

extradural trigeminal transection [66]. Frazier's point is located on the parietal side of the limb of the lambdoid suture, at the junction of the parietal and occipital bones [64]. This is 6 cm above and 4 cm lateral to the inion [67]. It allows the insertion of the catheter down the body of the lateral ventricle [61]. The catheter is inserted perpendicular to the cortex for about 4 cm. In clinical practice of pediatric neurosurgery, Frazier's point is often mixed with Dandy's point. Again, Dandy's point is basically on the lambdoid suture and is topographically lower and medial to Frazier's point. Frazier's point allows an easier advancement of the catheter into the whole body of the ipsilateral lateral ventricle.

Kaufman's point

Forehead access to the frontal horn was initiated by Kaufmann et al. [68] in 1970. It involves the introduction of the catheter from a point located 4 cm superior to the nasion and 3 cm lateral to the midline. Then, the catheter is directed toward the midline, 3 cm above the external occipital protuberance, and is inserted for 6–7 cm. Cosmetic concerns, although minimal, have decreased the use of this technique. However, in a study by Park et al., the accuracy rate of forehead access exceeded that of Kocher access. This is mainly due to use of a more proximal landmark, CT guidance, and reserving a place for a future potential shunt [60].

Paine's point

Paine's point was first described by Paine et al. in 1988. They proposed the use of this point for intraoperartive access to the frontal horn of the lateral ventricle after dural opening. This point is located at the intersection of a line 2.5 cm above the floor of anterior cranial fossa (or superior to the lateral orbital roof) and a line 2.5 anterior to the Sylvian fissure marked by the Sylvian veins. This can be defined by the apex of an isosceles triangle, whose base is 3.5 cm and lies along the Sylvian veins, and each of the two limbs are 2.5 cm. A catheter is placed perpendicular to the brain convexity and is inserted for 4–5 cm depending on the size of the ventricles [69].

Menovsky's point

In 2006, Menovsky et al. described an access point for keyhole surgeries performed through supraorbital craniotomy via an eyebrow incision, to overcome the need for a second incision. During this procedure, the cerebral puncture point is situated directly under the key burr hole at the base of the frontal lobe and the catheter is directed 45° to the midline and 20° up from an imaginary line parallel to the orbitomeatal line. To reach the ventricle, the catheter is inserted for about 5 cm from the cortex [70].

Hyun's and Park's modifications on Paine's point

The proximity of Paine's point to Broca's area on the dominant side and violation of the caudate nucleus and its related complications lead Hyun et al. in 2007 and Park and Hamm in 2008 to describe new intraoperative landmarks during pterional craniotomies. Hyun et al. [71] suggested that the new point should be extended approximately 2 cm posterior along the anterior limb of Paine's triangle, and the catheter is inserted only about 5 cm. Park's point, is located 2.5 cm superior to the lateral orbital roof and 4.5 cm anterior to the Sylvian fissure, and is actually a 2-cm anterior extension of the posterior limb of Paine's triangle. Perpendicular puncture at this point provides the trajectory of the catheter between the caudate nucleus and the corpus callosum. The ventricle can be accessed at about 3.5 cm from the surface [72]. In summary, Hyun's point is a 2-cm further posterior extension of the anterior limb of Paine's triangle, and Park's point is a 2-cm anterior extension of the posterior limb of Paine's triangle. Hence, Hyun's point is most posterior, Paine's point is in the middle, and Park's point in the most anterior among these three points.

Tubbs' point

In 2009, Tubbs et al. [73] described an external landmark for performing emergent transorbital access to the frontal horn of the lateral ventricle. Their technique involved accessing the frontal horn from a point just medial to the midpapillary point through the roof of the orbit. A trajectory aimed 45° from a horizontal line and 15–20° medial to a vertical line is taken. The depth of catheter insertion from skin to the level of foramen of Monro ranges from 7–8.5 cm (mean, 8 cm; Fig. 5).

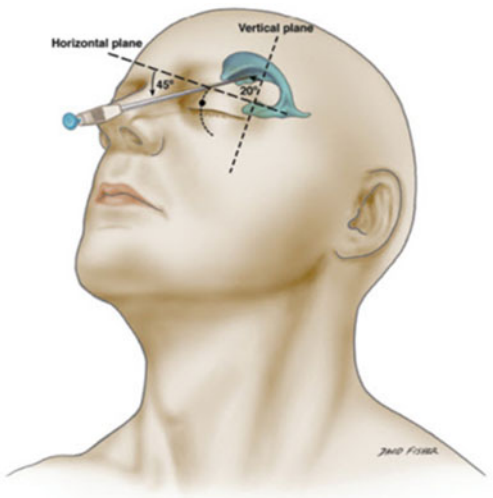

Fig. 5 Schematic illustration of access to the frontal horn through Tubbs' point

Treatment of acute hydrocephalus

Hydrocephalus is derived from the Greak terms *Hydro* - meaning water and *Cephalus* meaning head; therefore, it literally means water in the head. It was first described by Hippocrates in the fifth century BC, followed by Galen in the second century AD, as a disease caused by a superficial, extraaxial accumulation of water rather than enlargement of the ventricles [74, 75]. The first description of surgical evacuation of the intracranial fluid in children was made by the Muslim physician Al-Zahrawi in the tenth century AD, using a special instrument he invented [75]. The Muslim's knowledge of disease and treatment moved back to Europe, and was further developed during the renaissance. The first accurate description of fluid accumulation in the ventricles as a cause of hydrocephalus was made by Vesalius in 1551, based on human necropsy of a 2-year-old girl [76]. However, most of the bedside techniques used and developed later for treatment of childhood hydrocephalus relied on single and repeated ventricular puncture, and were associated with over drainage, ventricular collapse, and death [74].

External ventricular drain (also known as ventriculostomy)

The first reported attempt to use continuous and effective external ventricular draining was made by the French surgeon and anatomist Laude-Nicolas le Chat (1700–1768). Le Chat invented a special canula with a stopple that is inserted into the lateral ventricle and drain small amounts of the CSF at a time. Similar techniques were later reported in the nineteenth century, and began to gain more popularity thereafter using more sterile and less fatal techniques. These include the methods used by Wernicke in 1881, Pollock in 1884, Zrenner in 1886, von Bergmann in 1888, and Broca in 1891. Nevertheless, Oppenheim warned in 1902 that the use of these techniques is particularly dangerous and should be avoided. In 1911, Krause reported a successful use of a drain for 8 weeks with no documented complications. In 1941, Ingraham was the first to describe the use of a closed draining system [74, 76].

The external ventricular drain (EVD), or bedside ventriculostomy, is one of the most common procedures performed in neurosurgery. It is also considered the most accurate, reliable, and cost-effective method for intracranial pressure monitoring in adults. It defines a catheter that is inserted percutaneously at the bedside into the ventricular system and is connected to an external strain gauge transducer. It facilitates the measurement of intracranial pressure and therapeutic drainage of cerebrospinal fluid [77, 78]. The sites of EVD insertion have been described above.

In a study by Kakarla et al. [63] that involved 346 consecutive patients, they found that the indication of EVD use was subarachnoid hemorrhage in 153 (44 %), trauma in 64 (18 %), intracerebral or intraventricular hemorrhage in 63 (18 %), tumor in 51 (15 %), hydrocephalus in nine (3 %), and other diagnoses in six (2 %) patients. The most common site of insertion was the right frontal in 286 patients (83 %), followed by left frontal in 46 (13 %), right parietooccipital in 10 (3 %), and left parietooccipital in 4 (1 %) patients [63]. Other indications of EVD placement may include measuring of intracranial pressure, CSF sampling, and administration of medication into the CSF [79].

In determining the accuracy of EVD placement, a new grading system using postprocedural CT scans has been proposed and applied by Kakarla et al. [63] on 28 patients selected randomly. Grade I represented optimal placement with the tip in the ipsilateral frontal horn or third ventricle through the foramen of Monro. Grade 2 represented functional placement into the contralateral lateral ventricle or noneloquent cortex. Grade 3 represented suboptimal placement into eloquent cortex or nontarget cerebrospinal fluid space, with or without functional drainage. Grade I placement was seen in 77 % of his reported the patients, grade II in 10 %, and grade III in 13 %. Similar results were reported by Park et al. [60] in 2011 and other authors [63], and less optimal results were reported by Huyette et al. [80] in 2008. Optimal placement was mainly seen in subarachnoid hemorrhage patients, while suboptimal was mainly seen in trauma patients [63]. The accuracy of EVD placement may significantly increase using a Ghajar or image guide technique, with a success rate of 96 and 95 %, respectively. Nevertheless, they use of these techniques is uncommon (<3 % for Ghajar and 1 % for the image guide in one study) except in extreme cases like head trauma [60]. Ghajar guide was described by Ghajar [79] in 1985. It consists of a molded plastic tripod that is applied to the patient's Burr hole and guides the catheter in a path perpendicular to the skull and directly toward the frontal horn. Thus, it provides a higher level of accuracy [79, 81].

Infection is the most common complication of EVD, with rates ranging from 0 to 45 % [82]. According to Lo et al., the main risk factor of EVD infection was multiple insertions of EVD, with the second or third EVD being more prone to infection. The duration of the drain was not an independent risk factor, and in most cases, the infection occurred in a mean of 5.5±0.7 days postinsertion [83]. The same findings were reported by other authors [84, 85]. However, Park et al. [60] found less risk of infection associated with early conversion of EVD to ventriculoperitoneal shunt (VPS), which reflects the importance of timing. Hemorrhage is the second most common complication associate with insertion or removal of EVD. It occurs in 0–15 % of EVD placement [63], but may reach up to more than 40 % of the cases. However, this hemorrhage is usually insignificant and rarely needs surgical intervention, as only 1 patient out of 77 needed surgical evaluation in one study by Gardner et al. [86]. Other complications may include obstruction, malfunction, overdrainage,

neurological injury, hyponatremia, tension pneumocephalus, and intracranial calcifications [77, 87, 88].

Transorbital access ad modum Navarro

Acute and rapidly progressive hydrocephalus is a life-threatening condition that needs to be released at once. Various methods have been proposed for emergent relief of the inraventricular pressure. These include using an 18-gauge needle to drill the forehead, as described by Rifkinson et al. [89] in 1973. In 1975, Verdura et al. [90] described the use of a battery-operated drill for rapid access to frontal horn. In 1981, Navarro et al. were able to penetrate the orbital roof using an 18-gauge needle for rapid access to the frontal horn. They recommended the use of this technique as an emergency measure in rapidly deteriorating comatose patients whose signs are suggestive of acute cerebral or cerebellar herniation due to hydrocephalus when the drill is not readily available [91]. In this technique, the periorbital skin and conjunctiva are prepared, the upper eyelid is retracted forward and backward using a gauze, then a 18-gauge needle is inserted in the rostral third of the orbital roof, directed and advanced posteriorly towards the coronal suture and midline for almost 3 cm, until clear CSF can be seen [91, 92]. Possible complications of this technique may include injury to the supraorbital neurovascular bundle, damage to frontal lobe vessels, and CSF leak into the orbit. These complications can be minimized or avoided using the external landmark described by Tubbs et al. in 2009 (see above) [73].

Lumbar drainage

Although the first lumbar puncture was performed by Corning in 1885, the earliest application of lumbar puncture for treatment of hydrocephalus was by Quincke in 1891. However, complications due to use in obstructive hydrocephalus were reported and remained until differentiation between communicating and noncommunicating hydrocephalus could be made with the introduction of pneumoencephalography [76]. In patients with acute communicating (absorptive) hydrocephalus, usually caused by intracerebral or intraventricular hemorrhage, the use of EVD is often recommended. Although controversial, time is a determining factor in the use of EVD, as it needs to be replaced (usually <10 days) due to occlusion or infection. In cases of acute communicating hydrocephalus, a simple, noninvasive, and effective extracorporal CSF drainage can be achieved by insertion of a lumbar drain, which replaces repeated EVD, decreases the need for a ventriculoperitoneal shunt, and extends the time for recovery of the Pacchionian granulations. In one study, EVD with fibrinolytic therapy followed by lumber drain represented promising, safe, and feasible approach in the therapy of acute communicating hydrocephalus following severe ventricular hemorrhage [93, 94]. In a series of three consecutive patients treated with lumbar drain, none experienced complications. However, as in any other neurological procedures, a risk of anesthesia, parenchymal or subdural bleeding, infection, or symptomatic seizure is still possible [94].

Treatment of chronic hydrocephalus

For the purpose of nonacute hydrocephalus treatment, numerous types of shunts that divert the CSF flow into a cavity or organ in the body have been used.

Torkildsen shunt

Torkildsen, or ventriculocisternal shunt is a shunt that diverts CSF from the lateral ventricles to the cisterna magna in cases of third ventricle, aqueduct of Sylvius, or fourth ventricle obstruction [95]. It was one of the first shunts used in the treatment of hydrocephalus and was first described by the Norwegian neurosurgeon Arne Torkildsen [96] in 1939. This procedure became widely accepted and used as a treatment of hydrocephalus, mainly in the 1940s and 1950s, until ventriculoatrial, and later, ventriculoperitoneal shunts were introduced. At that time, the procedure was conducted without neuroradiological evaluation by CT and MRI and in severely ill patients. This contributed to a high mortality rate of 30 %. The main complications were inflammatory reaction, migration of the shunt catheter, and formation of a subdural fluid collection [95, 96]. One of the important advantages of Torkildsen shunt is keeping CSF within the nervous system, which, in case of cancer, decreases the risk of spread to the blood or peritoneal cavity [97]. In a recent study by Morota et al. [95] that involved 217 hydrocephalic patients treated with 494 surgical procedures, only five patients underwent Torkildsen shunt surgery. All five patients had lesions in and around the third and fourth ventricles, which precluded endoscopic third ventriculostomy or other endoscopic procedures. Two of the five patients had the shunt as a first surgical choice. No complications were recorded, and only one patient experienced a nonfunctioning shunt, where temporary ventricular drainage was added [95]. Modified techniques of internal shunts were also reported, including ventriculocervical subarachnoid shunt, ventriculo-epidural shunt, ventriculosubdural shunt, and ventriculolumbar shunts [76, 98].

Ventriculoperitoneal shunt

VP shunt is the most commonly used and usually the first choice shunt in hydrocephalic patients. The first introduction of the VP shunt use was by Kausch in 1905 and reported in 1908. He led a rubber tube from the lateral ventricles into the

peritoneal cavity. However, the patient died a few hours later due to CSF overdrainage. In 1910, Hartwell reported a more successful application of this method using a silver wire as a ventriculoperitoneal wick. The child died 2 years later due to stress fracture of the wire in the neck. This use of this technique faded thereafter and was revived during the Second World War by Cone, who never published his results, and Scarff who reported on hydrocephalus arrest in 55 % of cases in 1963. Since 1960s, the technique has not changed much and became the main shunting procedure replacing the ventriculoatrial shunt [76, 99].

Shunt obstruction is the most common cause of shunt failure; it can be classified into proximal, valve-related, or distal obstruction, depending on the segment affected. Proximal obstruction remains the most common, and it depends on several factors including the accuracy of catheter tip placement. Factors that may occlude the catheter tip include CSF contents, blood products, particulate brain parenchyma, cellular debris, ependymal cells, glial tissue, connective tissue, and leptomeninges. The size of the target ventricle also affects the rate of occlusion, as small, narrow ventricles may collapse and lead to catheter occlusion. Insertion of the catheter tip anterior to the foramen of Monro, in the atrium or occipital horn of the lateral ventricle may increase the risk of tip obstruction. The valve is an infrequent site of obstruction, which may be associated bacterial proliferation, the development of an immune-mediated cellular reaction, and from contamination with clot, parenchyma, or debris upon insertion. Distal obstruction is often seen in cases of malpositioning during the initial procedure, infection, or malabsorption. Malpositioning usually results from confusion between the preperitoneal fat and the omentum with improper placement of the catheter in the preperitoneal space. This might be evident later with the formation of subcutaneous CSF collection. Placement of the catheter at sites of intreabdominal adhesion may also result in distal obstruction. Low-grade infection, even if not symptomatic, may result in intraabdominal loculation or pseudocyst formation with resultant distal obstruction. The absorption of CSF by the peritoneum is a major factor in VP shunt function. In cases of limited absorptive function of the peritoneum, due to tumor seeding, mucopolysaccharidosis, or immune reactions, the CSF will accumulate in the abdomen causing pseudocyst or generalized ascites [100]. Although the operative exploration has been the gold standard for evaluation of shunt function, a new method has been proclaimed. In their study, Rocque et al. [101] performed shunt taps prior to 68 operative explorations in 51 patients. They found that in patients with poor flow on shunt tap, 93 % had proximal shunt obstruction. In patients with good flow on shunt tap with high opening pressure, proximal obstruction was found in 8 % of these shunts, and a distal obstruction in 92 %, and none of them showed good flow on surgical exploration. In patients with good flow on shunt tap with low opening pressure, 64 % had proximal obstruction and 7 % had distal obstruction, and only 29 % had no obstruction. From their findings, it is seen that only poor flow on shunt tap is highly predictive for shunt obstruction [101].

Shunt infection is a common complication that occurs in 1–41 % of the cases, with an average of 10–15 %. It is defined as infection of the shunt device, CSF, distal site, or overlying wound. The most common organism is *Staphylococcus* epidermidis in 40 % of cases, followed by *Staphylococcus aureus* in 20 %. Factors that increase the risk of infection include postoperative CSF leak, patient prematurity or young age, exposure to breached surgical gloves, poor conditions of the skin, intercurrent sites of infection, duration of procedure, time of procedure, and experience of the surgeon. In one study, factors that may decrease the risk of infection included restricting operating room personnel, scheduling procedures early in the day, soaking the shunt system in antibiotics, and using prophylactic intravenous antibiotics. The latter is controversial. Clinical picture ranges from totally asymptomatic to sever ventriculitis, sepsis, and death. Fever and obstructive symptoms, including headache, nausea, vomiting, and irritability are common [100].

Other complications include overdrainage, which is seen in 10–12 % of the cases; shunt fracture, disconnection, and migration, the latter may occur proximally or distally; seizures, which depends on the site of insertion and may range from 5 to 49 % of the cases, however, it still controversial; and pneumocephalus, a rare condition that is mainly associated with head trauma. Allergic reaction and rejection has also been reported [100].

Abdominal complications are not common and might include injury to viscous organ: gallbladder, urinary bladder, scrotum, testes, and uterus. Intraabdominal adhesion, and its consequences, has been reported as related to repeated procedure [100]. Although rare, it might cause infertility in women [102]. Liver abscess formation has also been reported [103].

Ventriculoatrial shunt

VA shunt is currently used by some surgeons as a second choice a after VP shunt, especially when the peritoneal cavity cannot be used as a distant target due to adhesions or infection. Although the VA shunt could be first used the 1940s, it received more attention during 1950s. In 1957, Pudenz et al. [104] described their experiments on use of silicone as a shunt tubing material, which they used for the first time in VA shunting on a child with aqueduct atresia. In the same year, Holter, a precision machinist, who obviously was unaware of Pudenz studies, developed a valve for shunting into the internal jugular vein or right atrium as an attempt to save his hydrocephalic child. Holter used a double silicone slit valve, which was first implanted by Spitz in 1956, and became known as "Spitz–Holter" valve [99, 105]. During the 1950s

and 1960s, the VA shunt became the preferred shunt for communicating and noncommunicating hydrocephalus. Thereafter, the use of this shunt was almost abandoned when the patients began to experience serious complications, including infections, thromboses, pulmonary emboli, and technical problems. In the 1960s and 1970s, the VP shunt became the mainstay treatment [99].

The conventional route of VA shunt insertion includes access to the vena caval system and consequently the right atrium. It is accomplished by a lateral right-sided neck dissection and isolation of the facial or the external jugular vein in the Farabauf's triangle to introduce the atrial catheter into the internal jugular vein. In this case, a revision of the shunt is very difficult, as another dissection is required, and a second route is preferred. Another method of percutaneous catheterization of the subclavian, internal jugular vein, or another functional vein, with or without ultrasound assist, has gained popularity, and is the preferred method for VA shunt. In contrast to the conventional method, it is considered quick and easy to perform, and with minimal complications [106–109]. The ideal placement of the tip of the distal catheter is in the midway between the entrance into the right atrium and the tricuspid valve [100].

Besides the proximal complications mentioned above in VPS section, distal compilations of the VA shunt may include misplacement, which may precipitate in arrhythmias, myocardial perforation, kinking of tubing, thrombus formation, and subsequent obstruction. Cardiac and pulmonary thromboembolic attack may result in thrombus formation. Multiple attempts of vascular access may also lead to tissue scarring, vessel sacrifice, or vessel thrombosis. Disconnection or fracture of the distal catheter may also occur and result in migration of the fragments to the heart or pulmonary vasculature. Patients who cannot tolerate additional vascular fluid load may develop congestive heart failure. Infection is also not uncommon [100].

The association between infected VA shunts and glomerulonephritis was first reported on two children by Black et al. [110] in 1965, and was later described in many other case reports. In response to the shunt infection, antigen–antibody complexes may be formed and precipitate in the glomeruli, which result in impairment of renal function [100, 111]. The incidence of shunt nephritis range from 0.7 to 2.25 %, and the time from disease onset and diagnosis range from 1 month to 8 years later with average about 3 years [112].

Ventriculopleural shunt

Insertion of the distal catheter into the pleural space was first reported by Heile [113] in 1914. In 1950s, Ransohoff and colleagues revived this technique and, in 1970, reported an almost 65 % success rate of his treatment of 85 patients with this shunt. However, long-term follow-up revealed that most of the patients developed shunt obstruction within 3 years. Therefore, the ventriculopleural (VPl) shunt has been used with limited indications and is a useful and effective alternate shunt when the peritoneal cavity or vascular system are unavailable, such as in cases of peritoneal infection, adhesion, ascites, or in previous vascular procedures, thromboses, or cardiovascular problems [114, 115]. Milhorat and Hoffman in 1978 and 1982, respectively, recommended that VPl shunt not to be used in children <3 and 8 years old, respectively, due to risk of pleural effusion. Nevertheless, Jones et al. [115] proclaimed in 1988 that using a differential pressure valve with regular supervision, the VPl shunt may be used in patients younger than 4 years old. Willison et al. [116] also mentioned that although this technique is not the ideal choice in children, but it is safe to be used temporarily to control obstructive hydrocephalus until a definitive diversion procedure may be performed.

In almost all patients with VPl shunts, a small pleural effusion evident by obliteration of costophrenic angle on chest X-ray can be seen as a sign of functioning shunt. However, in cases of inflammation, infection, or very young age that might limit the absorptive function of the shunt, and symptomatic pleural effusion may develop. Pneumothorax and subcutaneous emphysema may also occur during shunt placement; it is, however, not clinically significant and may resolve without additional treatment. In patients with mechanical ventilation, the intrathoracic positive pressure may lead to shunt obstruction. Shunt migration is an unusual complication, and some patents may experience transitory diaphragmatic pain [100, 114].

Lumboperitoneal shunt

The lumboperitoneal (LP) shunt is a shunt that arises from the lumber subarachnoid space below the level of conus medullaris into the peritoneal cavity. The first introduction of the LP shunt was made by Ferguson in 1898. He treated two patients using a silver wire that passed through a hole in the lumbar vertebra. The patients died 3 months later of surgical complications [99]. The use of percutaneous insertion of the shunt decreased the need for laminectomies and its related complications [117]. The physiology of draining is different between LP and VP. In the latter, the draining function is attributed to the pressure difference between the inlet and ending points of the catheter, which creates a siphoning suction pressure. In the LP shunt, the inlet and outlet points are at the same level, and hence, if the shunt was inserted in the upright position, inadequate draining will happen in the supine position and vice verse. Thus, the valve used for lumbar shunts must function in a dynamic state to adjust to these changes in body position. For this purpose, two valves have been used to work in two pressures, depending on the body position [118]. Indications of LP shunt include communicating hydrocephalus, pseudotumor cerebri, postoperative

pseudomeningoceles, spontaneous and postoperative rhinorrhea and otorrhea, slit ventricle syndrome, and obstruction distal to the spinal subarachnoid space [117].

The main advantage of LP shunt use over the VP shunt is the lower risk of infection, at about 0.5 % compared to as high as 41 % in VP shunts. Shunt failure and overdrainage and consequent ventricular collapse are also significantly less common. The fact that no catheter needs to be inserted in the brain can be related to decrease risk of hemorrhage and seizures. Other advantages include shorter operation time, lower incidence of obstruction, lower incidence of shortening due to child growth, and possible use of spinal anesthesia [117, 119]. The major disadvantage is the significant difficulty in assessing the intracranial pressure, which in some conditions (e.g., pseudotumor cerebri) needs to be done often. The incidence of scoliosis and arachnoiditis has decreased following the use of percutaneous methods. Hindbrain herniation secondary to Chiari I malformation is an uncommon complication that might be related to very young age, underlying pathology, and the type of shunt used [117]. Other disadvantages include the difficult use in patients with vertebral deformities, risk of epidural hematoma, radiculopathy and myelopathy, and pneumocephalus. However, these are very uncommon manifestations [119].

Endoscopic Third Ventriculostomy

The first cases of endoscopic neurosurgery were reported by Lespinasse in 1910. He was an urologist who used a rigid cystoscope to perform choroid plexus ablation in two hydrocephalic children. In 1927, Dandy performed the first subfrontal approach for an open third ventriculostomy. However, he had to abandon this procedure due to its high mortality rate. The first case of endoscopic third ventriculostomy (ETV) was reported in 1923 by Mixter [120], a urologist who used a urethroscope to perform the third ventriculostomy in a child with obstructive hydrocephalus. With some modifications on the urethroscope made and described by Putnam [121] in 1934, besides the use of the valve-regulated shunt, the ETV remained almost unchanged for around 30 years. McNickle, in 1947, introduced the method of percutaneous third ventriculostomy, which had decreased the complication rate and improved the success rate. In 1978, Vries described the use of endoscope in third ventriculostomy, and since then, the ETV became one of the main operations in treatment of hydrocephalus [122, 123]. Although the ETV can be applied in both communicating and obstructive hydrocephalus, it is mainly indicated in the latter. It can also be used in previously shunted patients who experience shunt failure; however, some authors prefer performing shunt revision due to ETV complications. The use of ETV in neonates, infants, and young children is controversial, as most of the authors have reported higher rates of ETV failure, and recommend shunt use in this age group [122, 124].

Complications rate in ETV may range from 2 to 15 %. Beside possible intraoperative complications like misplacement and bradycardia, early (within the first 4 weeks) and late complications may exist. Early complications include infection, hematoma, blockage, CSF leak, diabetes insipidus, weight gain, precocious puberty, and abnormal prolactin level. Other rare early complications may include hyperkalemia, severe parkinsonism, acute respiratory alkalosis, and tachypnea. The major late complication is stoma block, which needs long-term follow-up [122].

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