ARACHNOID CYSTS

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Professor Zdeněk Novák, M.D., Ph.D. et al.

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maxdorf jessenius

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FOREWORD

Probably every neurosurgeon was asked to operate on a patient with radiologically verified arachnoid cyst of various size referred for neurosurgical therapy with an idea that neurosurgical intervention will relieve all pleomorphic and polytopic problems. Moreover patients in whom complete cyst regression does not lead to any change of the patients problems as well as patients with complete symptoms remission after surgery without any change of the cyst size are well known to all neurosurgeons having at least moderate experience with arachnoid cyst treatment.

When considering neurosurgical treatment in patients with arachnoid cyst the following principal questions are asked by the treating surgeon:

- the relationship of the patient's problems to the arachnoid cyst, the necessary diagnostic workup
- indication for surgery
- the type of surgical procedure

CT and MRI offer nearly unequivoical diagnostic possibilities for the definitive diagnosis of arachnoid cyst at present time, maybe with the exception of some unique rare situations, when the differentiation between arachnoid cyst, cystic brain tumor, dermoid – or epidermoid tumor, posthaemorrhagic or postsurgical pseudocyst is not easily made. On the other side the difficult question of patient problems and neurological symptoms correlation with the radiologically verified arachnoid cyst should be answered. Arachnoid cyst may be only one of clinical manifestations of rare inherited diseases. Also the headache in patients with arachnoid cyst cannot be attributed only to intracranial hypertension threatening patients with large cysts or local cyst expansion. And finally the results of surgical treatment of patients with arachnoid cyst manifesting with epileptic seizures prove more complex relationship between the cyst operated on and seizure disordes implicating that not always successful cyst treatment leads to seizures elimination.

From the point of treatment procedure choice the following possibilities are usually considered: cyst drainage, eg. to the peritoneal cavity (cystoperitoneal shunt), microsurgical resection of cyst walls and neuroendoscopic cyst fenestration (to basal cisterns in the majority of cases). Generally speaking neuroendoscopic surgery combines the positive features of shunt surgeries (limited surgical approach only from skull trephination), and microsurgical cyst resection (surgery under direct vision) and eliminates the disadvantages – no need for external cyst drainage, no need for craniotomy for cyst walls resection.

The aim of the presented book is to submit data considered by the authors to be essential for diagnostic and therapeutic considerations, surgical treatment and postsurgical follow up of a patients with arachnoid cyst. The book contains experience gained during 10 years study of a large group of patients with arachnoid cysts of the most different location enabling the authors to declare their preference of neuroendoscopic technique as primary treatment modality for patients with arachnoid cysts.

The primary aim of the books is to share the author's experience with home and foreign neurosurgical community for inspiration and further discussion that may lead to optimalization of treatment possibilities in patients with arachnoid cysts.

PŘEDMLUVA

Snad každý neurochirurg se ve své praxi setkal s nemocným s různě rozsáhlou radiologicky prokázanou arachnoidální cystou, odeslaným k neurochirurgické terapii s představou, že neurochirurgická operační terapie odstraní veškeré polymorfní a polytopní potíže nemocného. Podobně je možné identifikovat nemocné, kdy při kompletní regresi cysty po operačním zákroku nedochází ke zlepšení subjektivních potíží nemocného, a naopak jsou popisováni i nemocní, u kterých je překvapivým nálezem při kompletní regresi potíží prakticky stacionární obraz cysty po provedeném výkonu.

Při terapeutické úvaze u nemocného s arachnoidální cystou si neurochirurg pokládá tyto základní otázky:

- vztah potíží nemocného a arachnoidální cysty, další nutná diagnostika
- indikace operačního výkonu
- způsob provedení operačního výkonu

CT a především MRI vyšetření v současnosti představují téměř suverénní diagnostické metody pro diagnózu arachnoidální cysty, snad s výjimkou spíše ojedinělých situací, kdy je nutné rozlišovat mezi arachnoidální cystou, cystickým tumorem, tumorem typu epidermoidu a pseudocystou po operaci nebo intracerebrálním krvácení. Velmi obtížná je naopak otázka korelace klinické symptomatologie a neurologického nálezu u nemocného s prokázanou arachnoidální cystou. Arachnoidální cysta může být jen jedním z projevů vzácných genetických onemocnění. Bolesti hlavy uváděné některými nemocnými s arachnoidální cystou nejsou otázkou pouze intrakraniální hypertenze u nemocných s rozsáhlou cystou. Podobně výsledky operací arachnoidálních cyst u nemocných s epilepsií poukazují na komplexnější vztah operované cysty a epileptických záchvatů, z čehož plyne skutečnost, že ne vždy technicky dokonalé řešení cysty znamená vymizení záchvatů.

Z hlediska chirurgického řešení jsou obvykle zvažovány následující možnosti operačního výkonu: zavedení drenáže cysty například do břišní dutiny (cystoperitoneální shunt), mikrochirurgická resekce stěn cysty a neuroendoskopická operace s fenestrací cysty nejčastěji do cisteren na spodině mozku. Velmi zjednodušeně je možno říci, že neuroendoskopický výkon spojuje pozitivní rysy shuntových operací – minimální přístup, dostačuje pouze trepanace – a mikrochirurgických resekcí – výkon prováděný pod kontrolou zraku – a eliminuje nevýhody těchto technik – není nutná drenážní hadička vedoucí z dutiny cysty do břicha a není nutná kraniotomie k resekci stěn cysty.

Cílem knihy je prezentovat data, která považujeme za zásadní pro diagnosticko terapeutickou rozvahu, operační řešení a pooperační sledování u nemocných s arachnoidální cystou. Prezentujeme zkušenosti získané v průběhu 10 let v rozsáhlém souboru nemocných s arachnoidálními cystami nejrůznějších lokalizací, které autorům umožňují preferovat neuroendoskopickou techniku jako prvotní pro ošetření arachnoidální cysty. Autoři si kladou za další cíl tyto zkušenosti předat neurochirurgické komunitě domácí i zahraniční k inspiraci a další diskusi vedoucí k optimalizaci terapeutického postupu u nemocných s arachnoidálními cystami.

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1.

INTRODUCTION

ÚVOD

CHAPTER 1

Introduction

The unstoppable progress in all branches of science has become evident also in the field of neurosurgery. The coming of stereotaxy and imaging techniques forming together a new diagnostic – treatment complex has changed the content of the original classic neurosurgery characterized by courageous and even heroic surgeries into minimally invasive neurosurgery. Surely the development has been aided by the progress in neurosurgical instrumentation and supporting medical devices, especially in the treatment of neurooncological and cerebrovascular diseases.

But there are new tendencies towards operating in deep and eloquent brain structures and especially in the cerebrospinal fluid space. The idea to treat non communicating hydrocephalus and cerebral arachnoid cysts by means of direct communication with the cerebrospinal fluid pathways to became again the natural part of cerebrospinal fluid space was finally asserted.

The diagnostic workup in pathological processes interfering with cerebrospinal fluid movements inside brain ventricles and subarachnoid space was definitive after the introduction of CT and MRI. The site of obstacle or disorder was reliably identified and optimum trajectory reaching these lesions was calculated with the help of ingenious stereotactic software.

It was not earlier before neuroendoscope became a new surgical device navigated to the site of the lesion able to inspect the lesion directly with organic substrate identification, to understand its effect and make adequate decision regarding the optimum surgical procedure.

Therefore a new minimally invasive discipline was established in neurosurgery – neuroendoscopy.

The process of diagnostic workup in cerebrospinal fluid disorders with associated symptoms has been almost solved. The characteristic message is surgical technique determining the treatment results and forming theoretical foundation of the branch.

This is the reason for paying attention to neuroendoscopic surgeries of arachnoid cysts in different location of treated lesions in the submitted monography.

KAPITOLA 1

Úvod

Nezadržitelný pokrok v každém vědním oboru se projevil i v oblasti neurochirurgie.

Nástup stereotaxe a zobrazovacích metod, které společně vytvořily nový diagnosticko-léčebný komplex, změnil obsah původní klasické neurochirurgie charakterizovaný odvážnými až heroickými operacemi na neurochirurgii minimálně invazivní. Jistě tomu napomohl i pokrok v nástrojových a přístrojových prostředcích neurochirurgie, zvlášť v léčbě nádorových a cévních onemocnění mozku.

Ale v současné neurochirurgii vznikly i nové tendence operování v hlubokých a funkčních strukturách mozku a především v jeho likvorových prostorech. Konečně se prosadila myšlenka léčit nekomunikující hydrocefalus a arachnoidální cysty mozku fyziologicky propojením do likvorových cest, aby se znovu staly přirozenou součástí likvorového prostoru.

Diagnostika poruch pohybu likvoru v mozkových komorách a v subarachnoidálních cisternách se stala vyšetřením CT/MR bezpečně definitivní. Spolehlivě se zjistilo místo překážky či poruchy a stereotakticky pomocí důmyslných programů se podařilo vypočítat optimální trajektorii k jejich dosažení.

Novým operačním nástrojem se však stal až neuroendoskop, který se naviguje do místa poruchy a umožní spatřit zrakem její povahu, pochopit její působení a rozhodnout se pro odpovídající léčebný postup.

V neurochirurgii tak vznikla nová minimálně invazivní disciplína, endoskopická neurochirurgie.

Diagnostika poruch v likvorovém prostoru a na ní závislá symptomatologie je téměř bezproblémová. Charakteristickým posláním je však operační technika, ze které se v praxi odvíjí léčebný výsledek i teoretický základ oboru.

To je důvod, proč jsme hlavní pozornost v předkládané monografii o subarachnoidálních cystách mozku a míchy věnovali způsobu endoskopických zákroků při jejich různé lokalizaci.

2.

ORIGIN AND DEVELOPMENT OF ARACHNOID CYSTS

VZNIK A VÝVOJ ARACHNOIDÁLNÍCH CYST

CHAPTER 2

Origin and development of arachnoid cysts

Arachnoid cysts are associated with the development of brain coverings.

2.1 MECHANISM OF ARACHNOID STRUCTURES FORMATION

Soon after neural tube has formed from the foetal ectodermal tissue widening appears at its cranial end in the form of cerebral vesicle sharing common cavity. The protective envelope consisting of two leaves is formed from the mesenchymal layer during the same time. The outer leaf is the thick pachymeninx and the inner leaf – more delicate leptomeninx carries the primitive vascular network to the brain vesicle. The growth of neural elements, primarily dependent solely on the tissue fluid inside the brain vesicle, is therefore greatly facilitated by blood circulation. At the same time pachymeninx forms two infoldings into the primordial brain vesicle directed transversally and longitudinally in a shape of duplicature forming cerebral falx separating cerebral and partially cerebellar hemispheres and tentorium separating cerebellum from brain hemispheres. The connection of cerebellar structures to the supratentorial brain hemispheres is provided by brainstem, passing through tentorial incisura. As a result two lateral ventricles are formed in cerebral hemispheres, connected by two interventricular foramina with the unpaired third ventricle and by means of Sylvian aquaeduct with the partially intracerebellar fourth ventricle. Spinal cord central canal is the continuation of the entire central nervous system cavitary system.

The remaining pachymeningeal structure becomes the basis for intracranial dura mater and elastic spinal dural sac. Leptomeninx invaginates together with infolding of pachymeningeal duplicature on both falcine sides and definitive cerebral arteries and veins are formed in the network of primitive brain vessels [Ognev 1950]. The apposition of leptomeninx and the thinned walls of the brain ventricles wall forms intraventricularly located choroid plexus and cerebrospinal fluid formation is initiated. As soon as the newly formed cerebrospinal fluid fills in brain ventricles entirely, the pressure of the cerebrospinal fluid produced by the choroid plexus inside the IV-th ventricle causes its opening in the 8-th gestational week and subsequent leptomeningeal splitting into two leaves – inner pia mater together with blood vessel covering directly cerebral and spinal surfaces and outer layer of thin arachnoid membrane. The formation of membranes from the original leaves gives rise to separated spaces. There is virtual subdural space between dura mater and arachnoid, subarachnoid space between arachnoid and pia mater, and subpial space between pia mater and cerebral or spinal surface. In the cranial cavity dura mater confluences with the periosteum of cranial vault (desmogenic ossification process), and in the area of skull base with the periosteum of skull base (chondrogenic ossification) therefore only arteficial separation of dura from the bone forms the epidural space. On the contrary spinal epidural space containing fat tissue, blood vessels and spinal nerve roots remains wide.

Chambers of varying size and shape forms in the subarachnoidal space from the rest of leptomeningeal tissue between pia mater and arachnoid tissue and these chambers provide pathways for cerebrospinal fluid flow. Chamber walls system augments the anchoring of the brain and spinal cord. At the site of great vessels and at the base of the brain the voluminous chambers are named cisterns with the largest being cisterna magna forming the junction of the superficial cerebrospinal fluid system of the brain and spinal cord [Baron 1949].

The changes of leptomeningeal tissue undergoing the process of infolding together with tentorium are slightly different because leptomeningeal tissue is found above as well as below tentorial structures, whereas leptomeninx is found on both falcine sides and the subarachnoid space chambers are superimposed one on another.

Tentorial infolding widens the gap between cerebral hemispheres and cerebellum (fissura telodiencephalica). The upper, supratentorial leptomeninx substantiates symmetrical formation of the large choroid plexus located in lateral brain ventricles. Blood supply for both sides is provided by means of posterior chorioidal lateral artery except for temporal horn fed by anterior chorioidal artery.

Infratentorial leptomeningeal tissue gives rise to the choroid plexus located in the third ventricular roof supplied by medial posterior chorioidal artery. Because leptomeninx overleaps tentorial margins, choroid plexus structures of lateral and third ventricles join each other in interventricular foramina.

Chambers forms in the subarachnoid space above third ventricular roof and these chambers may give origin to cavum Vergae located anteriorly and the continuous confluence of the remaining leptomeningeal tissue forms velum interpositum, which may become the basis for dorsally located velum interpositum cyst. Both cysts types share the name dorsal third ventricular cysts. Supratentorial space extends posteriorly as far as the quadrigeminal plate, is closely related to the space formed by cisterna ambiens in the region of tentorial incisura and extends to the proximity of intepeduncular cistern and skull base cisterns. These spaces are connected to the entire cerebral subarachnoid space, so that in triventricular hydrocephalus spontaneous communication between third ventricle and subarachnoid space may form through the dorsal third ventricle to ambient cisterns.

Similar changes of leptomeningeal coverings are seen in posterior fossa. However cerebellar volume increases gradually due to recruitment of pathways from the vestibular brainstem nuclei located in corpus restiforme, pathways from the spinal cord via brachia conjunctiva and cerebral pathways via brachia pontis. Therefore only marginal plates have remained from the original fourth ventricular roof on its upper and lower borders – anterior medullary velum and posterior medullary velum, supporting the oldest cerebellar nodulofloccular part – archicerebellum. Lateral recess of the fourth ventricle extends into this structure. The recess was the first structure in contact with leptomeninx and gave rize to the choroid plexus of the fourth ventricle (fig. 2.1) [Nádvorník 1965].

Choroid plexus attachements in the form of plexus taenias are directed toward the obex of the fourth ventricle. There are three sites where cerebrospinal fluid start the process of leptomeningeal splitting to pial and arachnoid layer – the paired lateral foramina Luschkae and the midline unpaired foramen of Magendii. Free pathway to form another cerebrospinal fluid space is established here. The pathway continues from here not only to the skull base and convexity but also to the spinal cerebrospinal fluid space.

Smaller lateral foramina opened in the area of both lateral recesses of the fourth ventricle may support cerebrospinal fluid circulation. Cerebellar basal cisterns have formed in their proximity, especially cerebellomedullary cistern with cerebellopontine angle arachnoid cyst, prepontine and intercrural arachnoid cisterns.

Craniocervical junction subarachnoid chambers at foramen magnum area may also gave rise to arachnoid cysts of varying size and shape. These cerebrospinal fluid filled spaces together with parafalcine chambers were able to form arachnoid cysts in the direction towards fourth ventricle or midline cerebellar plane extending as far as sinuum confluence. Arachnoid cyst may also form in spinal subarachnoid space.

2.2 THE INFLUENCE OF OTHER DEVELOPMENTAL ANOMALIES

The original Baron microscopic study of the development of the inner brain coverings forms an acceptable reactive explanation of posterior fossa anomalies when compared with the most diverse mechanic and other theories, supported by the final appearance of the malformed hindbrain [Hackel 1996]. Cysterna magna subarachnoid cysts influenced the genesis of the lower vermis and the neighboring cerebellar hemispheres in relation to their size, or midline subarachnoidal cysts, named as cisterna magna permagna, changed the shape and size of the entire cerebellum. The possible role of cerebellar structures primary violation as classified by Dandy Walker scale during the same time is unclear [Hackel 1999].

It can be accepted that arachnoid cyst formation can be enhanced by skeletal changes, as well as by the developmental failure of some cerebral structures, for instance temporal lobe agenesis in some extensive temporal arachnoid cysts or callosal agenesis, enabling marked enlargement of cavum Vergae towards interhemispheric fissure in the form of dorsal third ventricular cysts. Under normal circumstances, the formation of corpus callosum structures begins from genu area backwards as far as the infolding falcine duplicature reaches the weakened spot of lateral ventricular walls. That is the reason why choroid plexus is not formed here and the weakened walls of both lateral ventricles under corpus callosum fuse to form single septum pellucidum or may maintain the ependymal cavity between them.

2.3 ARACHNOID CYST GROWTH

The idea of cyst development and growth originated from the observation of frontobasal injury with brain coverings laceration. The elevation of pressure inside paranasal cavities during coughing or sneezing causes air influx intracranially. Although the process of air resorption is relatively fast (50 % during 24 hours), the amount of intracranial air may increase because of repeated air influx.

An assumption can be accepted for the cerebrospinal fluid space that cerebrospinal fluid may enter the subarachnoid chambers, but it does not flow backwards, because there in an obstacle in the chamber ostium. Explanation was similar to cerebrospinal fluid circulation obstacle with hydrocephalic dilatation forming above the block. The obstacle can be perceived like ventil or valve, blocking cerebrospinal fluid flow and the critical site can be depicted by means of CT/MRI.

It was not earlier than after the advent of neuroendoscopic technique that the dynamic effect of cerebrospinal fluid in circulation pathways was put forward, because for instance the visualisation of cerebrospinal circulatory pathways block together with the consequencies can be achieved by means of CT/MRI, but only the direct endoscopic inspection reveals the nature of such obstacle and the effect on cerebrospinal fluid circulation is discovered [Novak 2005].

In 13 years old male patient complaining of headaches with gradually increasing severity MRI revealed block at the level of left interventricular foramen, with resulting dilatation of the left lateral ventricle. Therefore the presurgical diagnosis was univentricular hydrocephalus caused by left foramen of Monro blockage. However when the endoscope was inserted into the left lateral ventricle along the preplanned trajectory (Praezis Plus software), it became apparent, that hypertrophic lobular flat membranaceous structure protruded from choroid plexus, periodically opening and closing foramen of Monro in the rhythm of heart beats.

During systolic phase the valve inclined towards the orifice closing it but during diastolic phase the flap turned away disclosing the entire opening. The movements copied the pulsatory rhythm of heart beats. It was considered to be justified to search the driving force in pressure relationships between the volumes of the larger lateral ventricle and the smaller third ventricle. During systolic phase the volume of brain tissue increases by the volume of blood and cerebrospinal fluid pressure in the lateral ventricles rises, therefore acting as secondary heart driven by heart activity.

Under normal circumstances cerebrospinal fluid systolic pressure in the lateral ventricles increases and the appropriate amount of cerebrospinal fluid is ejected from the lateral ventricle (the site of cerebrospinal fluid slow formation) through the interventricular foramen. Therefore the unidirectional cerebrospinal fluid flow is in fact periodical and driven by heart pulsations.

During the diastolic phase the pressure inside the lateral brain ventricle decreases as well as brain tissue volume, because the defined blood volume returns back to the heart by means of venous system. Also the fraction of cerebrospinal fluid already ejected into the third ventricle tends to return into the lateral ventricle because the pressure inside the third ventricle overweights the pressure in the lateral ventricles for a short period.

The pressure relationships inside brain ventricles are the driving forces for valvular movements. Intracranial pressure is maintained by the steady exchange of blood and cerebrospinal fluid volume during defined time period between the two systolic phases. This value is determined by the difference of pressures inside the participating brain ventricles p_1 and p_2 and the area of interventricular foramen S according to equation defining the actual influence of constant *k*:

$$\Delta V / \Delta t = \pm k (p_2 - p_1) S$$

This valve mechanism in cerebrospinal fluid pathways responsible for univentricular dilatation is able to provide an explanation for some cases of triventricular hydrocephalus, when the valve mechanism acts in the posterior part of the third ventricle or at the level of aqueductal orifice.

In 39 years old patient complaining of vertigo and memory problems CT investigation has shown triventricular hydrocephalus and therefore aqueductal stenosis was suspected. However when neuroendoscope was introduced into posterior third ventricle, it became evident that aqueductal orifice was blocked by the posterior commissure, moving in synchrony with heart beats. The result was alternation of widening and narrowing of the slit like aqueductal orifice. The movements of commissural structures therefore resembled lip – shaped valve. The impact of pulsatory cerebrospinal fluid flow from the upper three ventricles opened the valve during systolic phase, therefore a portion of intraventricular cerebrospinal fluid passed through the Sylvian aqueduct to the IV-th ventricle, but during diastolic phase the unidirectional flow of cerebrospinal fluid was interrupted and a fraction of the cerebrospinal fluid compartment could flow back.

Similar mechanism was found during surgery for interhemispheric cyst associated with callosal agenesis. 47 years old male patient began to complain of lasting paraesthesias of bosth upper and lower limbs with concomittant hypaesthesia. The cause was giant interhemispheric cyst extending from frontal area as far as quadrigeminal plate. The medial surfaces of both frontal lobes were separated by a wide gap and also third ventricular walls were separated one from another so that anterior commissure was the only structure connecting them. Agenesis of corpus callosum was also described. Cerebrospinal fluid pathways were freely patent, therefore it was possible to assume that the structural origin of the cyst is cavum Vergae cystic dilatation. Right sided endoscopic approach permitted direct inspection of huge cyst cavity. The floor was covered by membranes, separating the cavity from the third ventricle with brain vessels, third ventricular choroid plexus and posteriorly located brown-grey pineal body visible at the bottom. When cyst fenestration was established in the membranes extending between cerebral convolutions to the subarachnoid space the diameter of the orifice opened and closed in the rhythm of heart beats. The movement resembled valve mechanism, responsible for arachnoid cyst formation and growth between the arachnoid leaves.

As a result of the analysis, there are two patterns of cerebrospinal fluid flow: first, steady and continuous, related to slow formation of cerebrospinal fluid, passing unidirectionally through the cerebrospinal fluid circulation system. Second movement type is related to blood pulsations and subsequent pulsatory movements of the upper three brain ventricles acting as secondary heart. The direction of cerebrospinal fluid flow is alternating during the systolic and diastolic phase of the cardiac cycle.

2.4 SEPTAL STRUCTURES INSIDE ARACHNOID CYSTS

The origin of intracystic or endocystic septal structures causing multilocular cyst appearance remains to be explained.

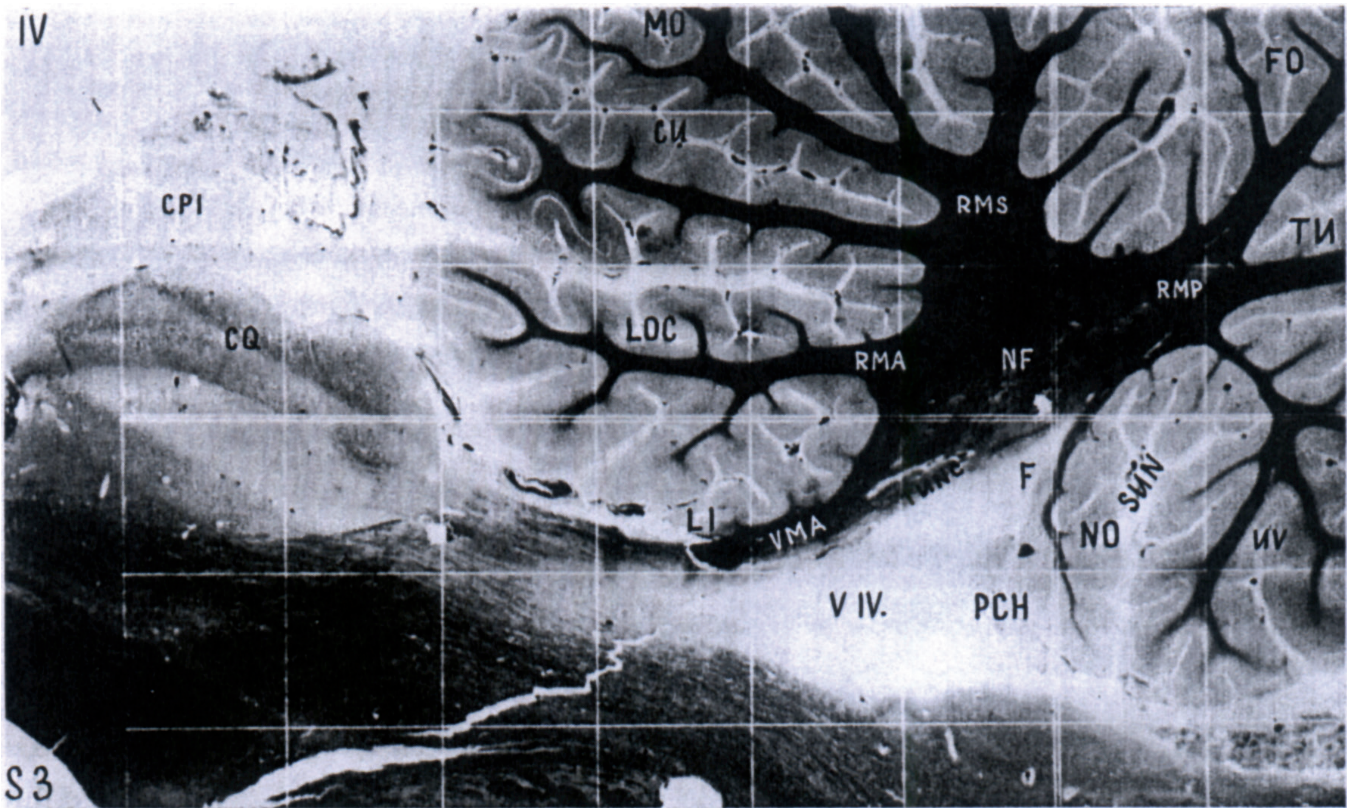
The mechanism responsible for cyst growth is probably the confluence of subarachnoid chambers based on mutual connections forming one definitive cyst. The connections resemble valve. It can be accepted that the cysts, appearing independently and forming cluster- like formation gradually form intercyst connections. But the residuals of cyst walls may remain apparent inside the final cavity in the form of septal structures. The presence of septal structures is a rule in large cysts, and it is possible that drainage surgery fails to drain the entire cyst volume. It may be one of the causes of shunt failure. Therefore the fenestration of septal leaves is a necessity during neuroendoscopic surgery.

But some septations may form in relationship to vascular structures, especially in cyst located in their proximity. The original embryonic vascular network penetrating leptomeningeal structures gives rise to the definitive brain vessels, and the superfluous original brain vessels vanish in apoptotic manner. But remnants of the original vascular network may be present in the form of intracystic septa facilitating cyst enlargement especially in the proximity of large cerebral arteries and veins. This holds especially true for temporal cysts that occasionally grow towards frontal, parietal and occipital regions accompanying middle cerebral artery branches, main cortical veins and deep venous system of Galenic vein with tributaries, occasionally reaching huge size. This is confirmed not only by the extent of the cyst, but also by the presence of delicate blood vessels located on the cyst surface and in septal structures, but also by the endoscopic observation of blood vessels passing through the cyst or freely floating inside the cyst cavity. Because these blood vessels are found in tension cysts (increased intracystic pressure and cerebrospinal fluid escaping under high pressure after cyst opening) such findings elicit consideration about causality.

2.5 THE IMPACT OF ARACHNOID CYSTS

There is no doubt that true arachnoid cysts of brain and spine are established during the embryonic period and the process of cyst formation depends on the splitting of the original brain coverings into arachnoid and pia mater. It is supported by the finding of lamina interna or the entire bone thinning or skull bulging at the site of the forming cyst, especially in temporal region, where the desmogenic bone is weaker. The pressure activity is also evident on the compressible brain tissue with resulting cyst impression, brain vessels dislocation and subsequent morphological abnormity reaching even the form of suspected agenesis. The feature is especially prominent in temporal and frontal lobes or in posterior fossa in cerebellar vermis and hemispheres development.

The majority of arachnoid cyst affects single site of extraventricular cerebrospinal fluid space; only exceptionally multiple cysts are found and there are nearly no cases of symmetrical cysts.



■ **Obr. 2.1** Stereotaktická sagitální mapa IV. mozkové komory s jejím chorioidálním plexem a otvorem foramen Magendie, kterým komorový systém ústí do subarachnoidálního prostoru mozku a mozečku

■ **Fig. 2.1** Stereotactic map of IV-th ventricle (sagital plane) with its choroid plexus and foramen Magendie, where the system of brain ventricles communicates with cerebral and cerebellar subarachnoid space