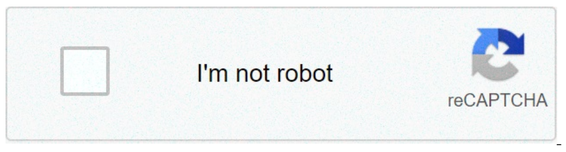

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Virchow robin space

百科分类：[病理学-神经系统]　血管周围间隙是在一个多世纪前由德国病理学家R.Virchow和法国生物学和组织学家C.P.Robin提出,后来命名为Virchow-Robin腔（VRS），也有称之为血管周围淋巴间隙。VRS将血管与周围的脑组织分离开来，是神经系统内的正常解剖结构。MRI能够在体显示这一解剖学和组织学的结构，并发现VRS的增多和扩大与多种神经疾病有着密切的关系。SI0影像园XCTMR.com　血管周围间隙是神经系统内的正常解剖结构，具有一定的生理和免疫功能。VRS增多和扩大见于老年脑、高血压、糖尿病、痴呆、脑白质病变、脑积水、多发性硬化、中枢神经隐球菌感染、儿童脑发育性疾病等。SI0影像园XCTMR.com SI0影像园XCTMR.com SI0影像园XCTMR.com Harvey B. Sarnat, Laura Flores-Sarnat, in Handbook of Clinical Neurology, 2013Inflammatory cells occur in perivascular (Virchow-Robin) spaces and the leptomeninges in resected brain tissue of epileptic children. The type of cell is usually the T-lymphocyte and macrophage, and may be identified by specific immunocytochemical cell markers. If preoperative subdural electrodes were used, the inflammation often may be attributed to it. Vascular congestion and fresh focal micro-hemorrhages often are present in addition. The significance of focal infiltrates of inflammatory cells not related to foreign bodies is uncertain, but usually does not suggest an underlying autoimmune disease or cerebral vasculitis as the pathogenesis of the epilepsy. True cerebral vasculitis, not just perivascular cuffing, is rarely demonstrated.Microglial cells are resident macrophages in the brain and are of mesodermal, not ectodermal, origin. Increased numbers of immunoreactive microglial cells occur in a variety of conditions, including ischemic infarction of brain, encephalitis, vasculitis, and in epilepsy. Microglial “activation” may be seen in both gray and white matter, and appears to be a nonspecific feature in the brain tissue of epileptic children, regardless of the presence or absence of a defined lesion of the brain. Microglial activation may occur with or without concomitant overexpression of α-B-crystallin.Infarcts of the brain are a rare finding in epilepsy surgery in children. Porencephaly secondary to a middle cerebral artery occlusion in fetal life may produce epileptic foci in the rim of the cyst. In the case of congenital infections, particularly cytomegalovirus disease (CMV) and congenital rubella, the brunt of the viral infestations is borne by the endothelial cells of both capillaries and large vessels; the endothelium may swell, narrowing the lumen, may produce fibrin deposits and thrombi, and both microinfarcts and major cerebral arterial occlusions may occur. The most extreme result, occurring in late fetal life and continuing postnatally, is multicystic encephalomalacia.Dawit G. Aregawi, ... David Schiff, in Handbook of Clinical Neurology, 2012Radiation therapy penetrates the sulci of the brain and the Virchow–Robin spaces. These areas are not easily reached by intrathecal chemotherapy and thus make radiation therapy an attractive treatment option. In addition, standard therapy for LMM has evolved to include palliative radiation therapy to sites of symptomatic or bulky disease. Radiation is typically given in doses of 30–36 Gy in 10 to 12 fractions and is the most expeditious method of relieving symptoms. Because of the significant potential for long-lived myelosuppression, only focal rather than craniospinal irradiation is generally feasible. Patients with cauda equina disease and lower extremity weakness, or with bladder or bowel dysfunction, receive lumbosacral spine irradiation alone. Patients with myelopathic findings or bulky disease require irradiation to the pertinent areas of the neuraxis. Radiation to sites of interrupted CSF flow, as demonstrated by a radionuclide flow study, also should be given before the administration of intrathecal therapy. On the other hand, patients with hydrocephalus, increased intracranial pressure, significant cranial neuropathies, or cortical deficits are believed to have diffuse metastatic disease and are consequently treated with WBRT (Mehta and Bradley, 2005).Adam L. Hartman, in Cerebrospinal Fluid in Clinical Practice, 2009It has long been recognized that CSF-containing perivascular channels, also known as Virchow-Robin spaces, surround major vessels as they course through the CNS parenchyma and maintain continuity with the subarachnoid space. Anatomical evidence suggests that as an artery enters the brain parenchyma, an extension of the pia mater surrounds it to form a definable sleeve. As they penetrate more deeply from the parenchymal surface, however, these pial layers approximate themselves with adjacent glia to the point where no definable perivascular space exists at the capillary level.5 These pial–glial cell interfaces form the adventitia of vessels and communicate with the subarachnoid space via gaps between leptomeningeal cells.5 Intracerebral veins have similar structures, but the meningeal layer around them is not as complete.5Nikolaus Deigendesch, Werner Stenzel, in Handbook of Clinical Neurology, 2018Microscopically, a dense infiltration by granulocytes/polymorphonuclear neutrophils (PMNs) can be observed in the subarachnoid space following the pia into the Virchow–Robin spaces (Fig. 16.1 B, C). The PMNs can traverse the blood–brain barrier, and enter the parenchyma, leading to cortical damage; leukocytoclastic vasculitis may be the consequence of dense perivascular cuffing, and may subsequently lead to focal areas of cortical infarction (Fig. 16.1B). The parenchyma is severely edematous, and widespread cortical neuronal damage is detectable. Sinus thrombosis, reduced flux in the sinus, as well as adjacent venous bleeding are common complications of meningitis.Daniel J. Brat, in Practical Surgical Neuropathology, 2010Histologic examination of DIAs and DIGs confirms that they are largely extracerebral in location, with variable, superficial extension of tumor into the brain along Virchow-Robin spaces. A distinctly biphasic morphology is characteristic. Collagen- and reticulin-rich regions populated principally by spindled cells in loose fascicular or storiform array dominate most examples, leading to a mesenchymal appearance that abruptly gives way to tissue of neuroepithelial character (see Fig. 7-4). The latter typically contain small cells of embryonal or astroglial appearance, densely aggregated within a reticulin-free fibrillar matrix. Fairly subtle small polygonal ganglion cells and gemistocytic cells may be seen in both fibrillar and desmoplastic regions. The presence of neuronal elements leads to the designation of DIG rather than DIA. Neuronal cells are most conspicuous in the noncollagenous portions, range considerably in size, and may include ganglion cells of fully differentiated or atypical appearance. EGBs are occasionally found, but are usually inconspicuous in comparison with classic gangliogliomas. Mitotic activity and necrosis are not usually present and are typically restricted to primitive small-cell components. Examples with high mitotic rate, microvascular proliferation, and necrosis have been documented, yet this has not translated into poor prognosis for most of these patients.42Rewati Raman Sharma, ... Shrikant Rege, in Schmidek and Sweet Operative Neurosurgical Techniques (Sixth Edition), 2012Cryptococcosis is one of the commonest CNS fungal infections in immunocompromised patients.151,152 It is ubiquitous but more common in Europe than elsewhere.18 It is a generalized systemic visceral mycosis affecting previously healthy people66; however, in 50% of cases, it has been reported in immunocompromised subjects, children, and middle-aged and older males.24 Pigeon breeders are at special risk. The causative agent, C. neoformans, is a spherical budding capsulated yeast (5-20 μm) (Fig. 149-5). It is found in soil and wood contaminated with bird excreta. It has been isolated from fruit juices and milk. C. neoformans neoformans causes disease in immunocompromised hosts, and C. neoformans gattii is the cause in immunocompetent hosts6; the portal of entry is the respiratory system. The primary focus lies in the lungs, from which secondary systemic dissemination occurs via hematogenous spread. There is a strong neurotropic tendency to involve meninges and the brain.105-113,143 CNS cryptococcal infection commonly presents with meningitis (subacute or chronic) or meningoencephalitis. Acute fatal meningitis occurs rarely in cryptococcosis. Usually, chronic meningitis present with the periods of remission and relapses. Cryptococcosis is one of the common CNS fungal infections in immunocompromised patients: nearly one tenth of patients with human immunodeficiency virus (HIV) develop cryptococcosis, and many HIV patients have cryptococcal meningitis as their initial clinical presentation.With cryptococcosis, the leptomeninges become infiltrated, thickened, and opaque (Fig. 149-6).3,8,17,18,143 The Virchow-Robin spaces around penetrating vessels are distended with organisms (Fig. 149-7).6,18 Granulomatous lesions can be found in the cerebral or spinal parenchyma. Spinal arachnoiditis may also be present. Chronic fibrosing leptomeningitis may cause hydrocephalus. Less commonly, intraparenchymatous cysts are seen (basal ganglionic regions), related to exuberant mucinous capsular material produced by the proliferating cryptococci.7,105,109,131 Rarely, fungi aggregate in an inflammatory lesion and produce small or large granulomas (cryptococcomas or torulomas) in the meninges, parenchyma, ependymal surfaces, or choroid plexuses.Microscopic examination shows three types of tissue reactions: (1) disseminated leptomeningitis, (2) granulomas, and (3) intraparenchymal cysts.3,6,18,107,143 In meningitis, there is minimal inflammatory response. The capsule of the fungus seems to impede inflammation by masking surface antigen. Inflammatory response consists of lymphocytes (mainly), plasma cells, eosinophils, fibroblasts, and multinucleated giant cells (studded with cryptococci).6,9,143 Glial reaction and associated cerebral edema are minimal. Granulomas are rarer late tissue reactions mimicking tubercles. They are composed of fibroblasts, giant cells (with fungal organisms), and necrotic areas. Multiple intraparenchymal cysts related to exuberant capsular material produced by the proliferating cryptococci create honeycomb-like cystic cerebral changes, especially in the basal ganglia. No membrane or capsule surrounds these cysts, which are well delineated from the surrounding tissue.9,143,151,152 Inflammatory response (macrophages with fungi and giant cells) around these cystic lesions is minimal. In our series of 22 cases, 21 cases presented with meningitis, and in an autopsy study in 17 cases, meningitis was confirmed in the form of thickened, hazy meninges with a characteristic slimy exudate over the superolateral surfaces and base of the brain. Among the secondary features, only 3 cases showed multiple granulomas in the cerebral hemispheres, hypothalamus, and, in 2 of these 3 cases, in the brain stem. Tiny cryptococcal cysts in the brain parenchyma containing plasma-like coagulated material were seen in 13 cases, and multiple areas of cystic degeneration destroying the brain parenchyma extensively and containing cryptococci were found in 13 cases (Fig. 149-8). In 1 case, multiple areas of demyelination with coexisting cerebral edema were seen, and in 4 cases, dilated ventricles were noted. In 2 biopsy cases, the structure of a granuloma consisted of inflammatory granulation tissue with foreign body giant cells and cryptococci. In 1 case of cerebral astrocytoma grade II, cryptococcosis was present within the tumor tissue and was well managed following surgery.CNS cryptococcosis commonly presents with nonspecific manifestations. At onset, patients usually present with headaches, nausea, vomiting, visual impairment, and papilledema; at a later stage, neck stiffness develops, followed by fever, personality changes, seizures, deterioration in sensorium, cranial nerve palsies, and hydrocephalus.7,108 In many patients, there are no physical signs. Periods of remission and relapses are noted. Cryptococcomas may produce prolonged, unresolving, focal neurologic deficits. Acute fatal meningitis in cryptococcosis is extremely rare.6,7 In our series, all 21 cases of CNS cryptococcosis presented with signs and symptoms of meningitis with raised ICP. Fever, headache, and vomiting were the commonest presenting symptoms. Altered sensorium, cranial nerve palsies, and visual symptoms were seen in 6 cases, and fatal meningitis occurred in 2 cases. Spinal cryptococcal arachnoiditis may present with progressive myelopathy or myeloradiculopathy.105-113,143The fungal capsule is transparent with cryptococcosis; therefore, the CSF appears clear, although it is mildly xanthochromic and under high pressure. The cell count may go up to 100 cells per cubic millimeter (mainly lymphocytes and polymorphs). The sugar and chloride levels are reduced, and total proteins may be raised. As the fungal capsule is transparent on routine microscopy, India ink preparation of the CSF can demonstrate a mucoid capsule. Organisms can be seen in tissues with PAS and methenamine silver stains.6,9,18 On mucicarmine and alcian blue stains, the fungal capsule is well recognized. However, routinely, CNS cryptococcosis is diagnosed by positive cryptococcal antigen titer and India ink staining of the CSF film preparation. Chest x-ray (pulmonary lesion) and CT/MRI brain scans (brain edema, hydrocephalus, basal meningitis, granuloma, and intraparenchymal cysts) are helpful (Figs. 149-9 and 149-10). The commonest patterns of CNS cryptococcosis are ventricular dilation in CT scans and Virchow-Robin space dilation in MRI. MRI is more sensitive in detecting CNS cryptococcal infection, such as Virchow-Robin space dilation and leptomeningeal enhancement. There is no significant pattern difference between immunocompromised and nonimmunocompromised patients with CNS cryptococcosis.142 The CSF culture should be done at 30°C for 5 days. This organism can be isolated in 95% of cryptococcal meningitis cases. Positive latex agglutination testing with rising serum titers of polysaccharide capsular antigen is of prognostic value.Untreated cryptococcal meningitis is generally fatal. Early aggressive therapy with combined amphotericin-B and flucytosine offers best chances of cure. Alternatively a 6-week course of amphotericin-B followed by maintenance oral fluconazole gives good results. Granulomas, cysts, and hydrocephalus are treated on their own merits. Predisposing factors should be carefully corrected. Failure of treatment raises the possibility of an underlying medical disorder, such as AIDS. Patients receiving treatment before CNS complications have a good prognosis.Matthew D. Cykowski, in Neurology Secrets (Sixth Edition), 2017173.What distinct anatomic compartments or structures may be involved by metastases?Bone (cranium or vertebrae), dura, subarachnoid space, the subpial and perivascular (Virchow–Robin) spaces, CNS parenchyma, choroid plexus, pineal gland, and pituitary gland. The involvement of some anatomic compartments is stereotypic for some tumors (e.g., breast carcinomas frequently metastasizes to dura).174.What is the approximate ratio of supratentorial to infratentorial metastases? Which primary site tumors more commonly metastasize to infratentorial locations?The ratio is approximately 3-4 to 1. Pelvic organs (colorectal, ovarian, and uterine carcinomas) and breast are overrepresented in metastases of the infratentorial compartment/posterior fossa.175.Metastases to the choroid plexus are very uncommon. Which tumor most typically does this?Renal cell carcinoma.176.What are the hemorrhagic metastases?The classic list of hemorrhagic metastases includes renal cell carcinoma, melanoma, lung cancer, and choriocarcinoma. Hemorrhagic primary tumors include glioblastoma and, most famously, oligodendroglioma.177.In the cortex, what is the typical site of metastasis that may be seen macroscopically?The most common site of metastasis is to the frontal lobe in arterial border (watershed) zones at the junction of gray and white matter.178.What features are unique to epidural metastases of the spine?Epidural metastasis presents with symptoms of cord and nerve root compression with extremity weakness and sensory loss. Epidural metastases rarely penetrate through dura to involve cord parenchyma (unlike leptomeningeal metastatic disease in which invasion into parenchyma is common). Epidural metastases may also result in a compressive myelopathy that is progressive and irreversible with cavitation and necrosis of cord parenchyma, as well as spongiosis and vacuolation in ascending/descending white matter tracts of the cord.1.The two major classes of glioma are infiltrating (diffuse) and circumscribed.2.The molecular signature of oligodendroglioma is whole-arm 1p/19q codeletion3.Glioblastoma, WHO Grade IV, is characterized by nuclear pleomorphism, mitotic activity, and either microvascular proliferation or necrosis (or both).4.Tumors with eosinophilic granular bodies include pilocytic astrocytoma, pleomorphic xanthoastrocytoma, and ganglioglioma.5.Tumors commonly seen with a history of chronic seizures include dysembryoplastic neuroepithelial tumor, ganglioglioma, pleomorphic xanthoastrocytoma, hypothalamic hamaratoma (with characteristic gelastic fits), and angiocentric glioma (not discussed here).C. Elizabeth Shaaban, ... Caterina Rosano, in Progress in Molecular Biology and Translational Science, 2019These lesions can progress over time from punctate to clustered to confluent. Punctate forms show a variety of small vessel pathologies (Fig. 1). Accentuation of perivascular (Virchow-Robin) spaces is associated with a variety of vascular pathologies including: convoluted vessels (arterioles, capillaries or venules), sclerotic vessels (with or without amyloidosis), arteriolosclerosis, hyalinosis and collagenosis.When WMH are associated with susceptibility artifacts, as detected on sequences sensitive to the magnetic properties of the iron in hemoglobin (e.g., gradient-recalled echo or susceptibility-weighted MRI), these lesions are thought to involve micro-hemorrhage with extravasation of red blood cells, pericyte erythrophagocytosis, hemoglobin degradation and hemosiderin deposition (Fig. 1C and D).33 How such changes might reflect “vascular integrity” or the blood-brain barrier remains to be fully elucidated.Some WMH are associated with complete or incomplete infarctions (Fig. 2), including conspicuous lacunar type infarcts or less obvious microinfarcts. Given limitations of sampling microinfarcts (i.e., infarctions not visible to the naked eye), it has been suggested that histopathology grossly underestimates the frequency of microinfarctions (e.g., for every microinfarct noted during routine brain sampling 500 microinfarcts are present in vivo34). Reactive gliosis (astrocytic and microglial cellular response) is the sine qua non of CNS damage and repair (Fig. 2). Its associations with WMH and perivascular changes are definitive proof that the histological changes are not fixation or processing artifacts, but are instead representative of in vivo pathology.Fig. 2. Lacunar infarction of basal ganglia. (A) Coronal section through the frontal lobe of the brain of an older adult patient. Ventricles (VEN) are mildly dilated, and there is a lacunar infarct in the head of the caudate on the right side (between arrows). (B) Hematoxylin and eosin-stained section of the lacune demonstrates a central region of infarction (INF) with tissue loss. (C) Immunostain for glial fibrillary acidic protein (GFAP) (brown) shows prominent peri-infarct astrocytosis. (D) Higher power micrograph of lacunar infarct showing intact ependyma overlying ventricle (VEN). Tissue surrounding the infarct contains numerous sclerotic blood vessels with dilated perivascular spaces. (E) Immunostain for GFAP demonstrates pronounced astrocytosis.Images were provided by Dr. Clayton Wiley (University of Pittsburgh).Myelin pallor (rarefaction) is a frequently described pathological finding associated with WMH. Despite the pathological term being vague and non-specific, it is frequently misquoted as “demyelination” or other more specific, but inaccurate, descriptors. This has resulted in substantial confusion and inappropriate assumptions about the pathogenesis of myelin pallor. Histologically it is manifest as decreased staining of white matter using any of a number of stains (e.g., H&E, luxol fast blue, staining for myelin basic protein, etc.). Like the MRI finding of WMH, this histological finding of myelin pallor can be broadly interpreted as altered tissue water content with numerous potential etiologies. Simple brain edema (vascular or cytotoxic) can expand the extra- and intracellular space leading to decreased stain density per square mm. Decreased numbers of axons (e.g., loss of fibers projecting through a region of rarefaction secondary to distal stroke) can decrease the tissue density and be associated with staining pallor. Demyelination could lead to decreased staining, but despite frequent use of this term in the literature there is no convincing evidence that primary demyelination (that is selective loss of myelin sheaths as opposed to loss of myelin sheaths secondary to loss of axons) is a true feature of WMH. Based upon measurements of select myelin proteins and alterations in vasoconstrictive endothelin-1 (EDN1) and vascular growth factors (VEGF), Love and colleagues have recently put forward the hypothesis that white matter rarefaction is the result of decreased perfusion.35 While an enticing explanation of white matter dysfunction, there is no formal proof for demyelination in aged white matter associated with WMH.Testing the hypothesis that WMH (or at least some of its components) is the result of demyelination is technically quite difficult. Love and colleagues35 have attempted to compare ratios of myelin associated glycoprotein (MAG) to proteolipid protein (PLP) as a surrogate marker of demyelination. While this is a convenient approach, there are multiple reasons other than demyelination that could lead to changes in the MAG/PLP ratio. Axons in the central nervous system become myelinated once they achieve a diameter of 0.5 μm. Since that is at the resolution limit of light microscopy, ultrastructural analysis (i.e., electron microscopy (EM)) is required to sensitively assess myelination. Most EM studies require perfusion fixed tissue, which of course is essentially impossible to get in humans. Thus, given the ultrastructural artifacts introduced by postmortem studies, the optimal human study would require brain biopsy followed by glutaraldehyde fixation, a perhaps unattainable goal in humans. Therefore, testing the hypothesized role of demyelination in WMH awaits the development of a good animal model that would permit in vivo perfusion and EM confirmation.Prashant Nagpal, ... Rajan Jain, in Handbook of Neuro-Oncology Neuroimaging (Second Edition), 2016Tumor-related stroke could also occur owing to vascular involvement by leptomeningeal spread of cancer. Leptomeningeal metastases or carcinomatous meningitis results from metastatic spread to the cerebral vessels in the Virchow–Robin spaces. These can cause stroke either by vascular occlusion/spasm or by causing vasculopathic changes in the cerebral vessels.29,30 Ischemic stroke has been described as a rare cause of focal cerebral symptoms in patients with carcinomatous meningitis.31 Imaging is very helpful in diagnosis of leptomeningeal metastases. CT is usually the first-line modality but has limited utility in diagnosis of metastases. MRI is the best investigation for diagnosis of leptomeningeal metastases and has been shown to be more sensitive (reported sensitivity of 100%) than cerebrospinal fluid (CSF) analysis for diagnosis.32 Nodular pial enhancement is one of the characteristic features of leptomeningeal metastases on gadolinium-enhanced MRI. Other features of leptomeningeal metastases that can be seen on MRI include hydrocephalus (often noncommunicating), multifocal cranial and spinal neural enhancement, and diffuse arachnoid enhancement.32 Digital subtraction angiography is rarely used for diagnosis but can show segmental narrowing and wall irregularities in multiple proximal and distal vessels in the setting of leptomeningeal metastases from carcinoma.31,33B.K. Kleinschmidt-DeMasters, Kenneth L. Tyler, in Practical Surgical Neuropathology, 2010Viral meningitis differs considerably from bacterial meningitis in the often subtle degree of inflammation present in tissue sections. Classic features of viral meningitis are those of lymphocytic meningeal infiltrates associated with perivascular lymphocytic extension along Virchow-Robin spaces (Fig. 21-4A). Microglial nodules and tissue destruction indicative of encephalitis are absent, by definition, and if identified, should suggest that the virus is producing an encephalitis in addition to the meningitis. Often the two conditions have overlapping features, and although clinicians recognize a predominance of one clinical presentation over the other for infections with certain types of viruses (see Table 21-12), at the tissue level, the division between meningitis and encephalitis may be less distinct.The subtle and minimal nature of the meningeal inflammation in some cases of bona fide viral meningitis cannot be overemphasized. Patients who succumb acutely may have little more than hyperemia, congestion, and edema of the brain, with correspondingly subtle histopathologic features. Lymphocytes and monocytes in the meninges may be few and far between. A high degree of clinical suspicion, coupled with extensive sampling to include multiple meningeal sections, may be necessary to even suggest the correct diagnosis of viral meningitis at all. At the other end of the spectrum, some cases of viral meningitis in their acute phases manifest with a CSF profile that includes considerable numbers of PMNs, such as lymphocytic choriomeningitis (LCMV) and West Nile virus encephalitis, and in these instances, neutrophils are also found in the meninges by the pathologist.