

## Cerebral Functional Venous Anatomy

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### Let's start with backgrounds.

Functional anatomy is the study of anatomy in its relation to function. The brain vasculature consists of three types of vasculatures: namely, artery, capillary, and vein. Everyone knows that brain is a blood-consuming organ, where 15% of the blood volume is distributed, although the brain consists of only 2.5% of the whole-body weight. But the fewer recognized that 70-80% of the intracranial blood surprisingly lies in the venous system[1]. Accordingly, functional venous anatomy is essential to understanding the upstream brain physiology and pathological condition. However, the functional anatomy of the vein has not been well-understood compared to those of the artery and capillary for the three following reasons. First, the intracranial venous system has numerous variations, and its morphology is so different from case to case. Besides, the brain venous channels are valveless[2]. The blood can move freely within them, and the flow direction is not fixed. Hence, the function of the specific vein is different from case to case, and, even in an individual body, the venous function is not always identical. Second, few radiological modalities can precisely evaluate the blood volume or velocity in the intracranial veins. Without precise evaluation, understanding its function cannot be achieved. The last, the function of the vein always synchronizes with that of upstream brain tissue. Therefore, if one can precisely evaluate the flow parameters of one vein, one cannot readily assume its upstream function. The observed values are somewhat consequence rather than the origin of the upstream brain function. In these circumstances, most researchers have investigated the functional venous anatomy mainly from each vein's morphology and embryological background in the literature.

### Embryology of cerebral venous system

We seldom consider embryology in the daily clinical, and most clinicians may not recognize its importance. However, embryology indeed influences the morphology and function of the cerebral venous system, and it helps us understand the current issues.

Several previous studies have described the embryology of the cerebral venous system [3–6]. Based on the literature, the intracranial venous embryology is schematically illustrated in figure 1. If you compare the embryology of cerebral veins with those of arteries

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or neuronal tissues, you may notice the critical difference between them. The arteries can develop as segmental or pharyngeal arch arteries or ones along with the neuromere or cranial nerve. They always run together, and each facilitates the development of its counterpart. The veins develop more passively; they are initially numerous channels consisting plexiform network. Each channel grows, shrinks, or diminishes according to the venous drainage demands to sustain the dramatically growing brain tissues. The final venous morphology is determined by this selection process, which explains their myriad variations and why they don't show the segmental or metameric disposition as the arteries do.

At the end of the 4<sup>th</sup> week of fetal life appears the first venous channel of the brain and head, namely, the primary head sinus. The brain is covered by the sparse connective tissues (primitive meninges), from which future cranial bones, dura, and meninges will develop. The three (anterior, middle, and posterior) dural plexus form in the primitive meninges as an extension of the primary head sinus. Later, the pial and intraparenchymal vein develops on the brain's surface at the 5<sup>th</sup>-6<sup>th</sup> weeks of fetal life. The superficial future dural sinuses and the deep future pial veins are connected with the numerous pia-arachnoid veins (future bridging veins). During the 7<sup>th</sup> week of fetal life, primitive meninges thicken and start to differentiate, resulting in space enlargement between dural and pia layers. The numerous pia-arachnoid veins are forced to elongate and converge into a fewer number of bridging veins. At the end of the 9<sup>th</sup> week of fetal life, the basic morphological formation of the intracranial dural sinus is already attained. Indeed, at this moment, the robust cranium and dura are gradually differentiated and developed, which doesn't allow the drastic morphological change of venous channels within them[6]. However, later than this period, the minor modifications still continue to occur, even after the birth, according to the demand of the growing brain and the systemic circulation changes along with the transition from fetoplacental to pulmonary circulation and posture changes from lying to upright.

In summary of embryology, the veins developed from the numerous primitive channels in a relatively passive way to meet the demand, being influenced by the growing brain and surrounding tissues.

### **Cerebral venous anatomy**

Based on each anatomical disposition, intracranial veins are classified into four: parenchymal veins, subpial veins, subarachnoid veins, including bridging veins, and dural sinuses (Figure 2). Histologically, the veins are classified into veins and venules. Venules are usually located inside the brain or in the subpial space.

#### **(Intra-) Parenchymal veins**

Parenchymal veins are divided into superficial and deep parenchymal veins, depending on whether they empty into the superficial or deep veins. Then, depending on where they reach, they are further subdivided into subependymal, medullary, subcortical, and intracortical veins [7].

Duvernoy et al. described the morphological anatomy of the superficial parenchymal veins[8]; veins penetrating the brain's surface are only one-fourth of penetrating arteries, which means that the area one vein perfuses is four times bigger than that of arteries. Some veins have many tributaries and reach deep inside the brain, termed "principal veins." Intraparenchymal arteries anastomoses are frequently observed, while intraparenchymal veins are seldom anastomosed[8]. Hence, parenchymal veins seem more anatomically vulnerable to its injury than arteries.

Intra-parenchymal veins are efficiently extended from superficial pial and deep subependymal veins. The border between the superficial and deep drainage is usually located in the subcortex. Developmental venous anomaly (DVA) is the extreme variation of this arrangement, in which a superficial or deep medullary vein is well-developed and perfuses the larger territory of the brain. A recent study indicates this extreme variation develops postnatally [9]. However, why this variation is induced after birth has not been well clarified. As mentioned above, anastomoses between intraparenchymal veins are rarely observed. The thrombosis or occlusion of DVA sometimes results in catastrophic venous congestion, hypertension, and hemorrhage[10].

### **Cortical veins ((sub)pial veins and subarachnoid veins)**

The subpial veins run under the pia mater, while the subarachnoid veins run in the subarachnoid space. They seemed to run in different anatomical layers. However, the pial membrane leaves the brain's surface along with the vessel, and the pial sheath covers the subarachnoid vessels in the whole circumference. In other words, the pial veins never penetrate the pial membrane. Accordingly, the pial and the subarachnoid veins are essentially the same from the histological viewpoint. Their minor difference is how far they are from the brain surface. Hence, they are generally together referred to as a "cortical vein."

The gap between the subarachnoid vessels and pial sheath represents perivascular space. It is continuous to the perivascular space (Virchow Robinson Space; VRS) of the intraparenchymal vessels. The parenchymal arteries accompany the pial sheath deeply, while the parenchymal veins don't. On the brain's surface, the veins always run under the arteries. The larger collecting veins, including the superficial middle cerebral vein, the vein of Labbe, and the vein of Trolard, run in the subarachnoid space above the arteries. Generally, the number of the pial veins is greater than that of the pial arteries. However, their anastomoses between each other can be less frequently seen than that of arteries[8].

## **Bridging vein**

Bridging veins are subarachnoid veins between the surface cortical veins and dural venous sinuses. The veins play a crucial role in connecting the cerebral venous system and dural venous sinuses and are involved with intracranial pathologies; Traumatic tearing of the bridging veins causes an acute subdural hematoma. The dural arteriovenous fistula's retrograde venous reflux to the bridging vein causes cerebral venous hypertension, congestion, and hemorrhage. The brain has 54.1 bridging veins on average (45.0 veins for the cerebrum and 9.1 veins for the cerebellum)[11]. They are frequently observed adjacent to the large dural sinuses (such as superior sagittal sinus, tentorial sinuses, transverse sinuses, and cavernous sinuses). The location of the bridging veins is the consequence of embryological modification of numerous bridging veins, as aforementioned. The fate of each bridging vein depends on the flow demand during embryological stages in each patient. Hence, they can be theologically formed anywhere on the brain's surface. Indeed, we occasionally see an unusual bridging vein connected to the convexity where no dural sinuses exist. For example, a previous report describes an extreme variation of the superficial middle cerebral vein (SMCV); The SMCV is bridged to the veins penetrating the lateral part of the sphenoid bone and directly continuous to the subcutaneous deep facial vein[12].

The bridging vein running in the subarachnoid space and exposed to the open CSF space seems vulnerable to intracranial pressure (ICP). However, they never collapse even in the extremely high ICP[13]. Why do they never collapse? Some researchers suggest that the bridging veins have a constricted part called an outflow cuff[14] or sphincter[15] at the junction between the cortical bridging vein and the dural sinuses. In this stenotic compartment, the encircled[14] or spiraled[15] collagen fibers enclose the veins, while the paralleled longitudinal collagen fibers cover the other part of the veins. This stenotic part restricts the venous outflow and constantly sustains the intravenous pressure higher than ICP by 5-25 cm H<sub>2</sub>O[16] and dural sinuses [16]. This mechanism separates the intradural venous system from the other part of the venous system. It plays a crucial role in preventing the bridging vein collapse and stabilizing the intradural venous pressure. The encircled or spiraled collagen fiber is also assumed to function as spiring to damper the traumatic mechanical stress on the bridging veins[14, 15]. We can also see a similar structure in the spinal radicular veins as well[17].

## **Dural venous sinus and diploic veins**

The dural sinuses and diploic veins are valveless. Therefore, the blood can move freely from the higher to lower intravenous pressure parts. Accordingly, similarly to the cortical veins, the function of the dural sinuses (= distribution of the dural sinuses drainage area) manifests

considerable individual differences from each other. As mentioned above, the dural sinuses and the intradural veins can be considered separated venous system. The intravenous pressure in dural sinuses remains stable (2-6 mmHg) unless downstream venous stenosis or arteriovenous shunt exists[16]. The mechanism also prevents reflux to the cortical venous system from the dural sinuses; the retrograde flow to the cortical veins can be seen when the intra-sinus pressure goes beyond the cortical venous pressure due to the presence of the arteriovenous flow. Since only the thin dural membrane interposes between dural sinuses and subarachnoid space, the sinus wall is directly influenced by the ICP. Accordingly, the morphology of the sinus wall changes along with ICP; when ICP increases, the wall is deflated[18], and when ICP drops, the wall is distended[19].

Another essential function of the dural sinuses and the diploic veins is rerouting the cerebral venous drainage; The blood in the dural sinuses or diploic veins returns to the heart mainly via the internal jugular or vertebral venous system. The former is dominant in the lying position while the latter is in the standing position[20-22]. The dural sinuses and diploic veins, particularly at the level of the cranial cervical junction, have rerouting functions for these two main cerebral venous drainages. San Millan described that the anterior condylar confluence (ACC) and the veins connected with ACC play an essential role in the redirection of cerebral blood[23]. The recent articles further investigated the osseous venous channels in this region[24, 25], They emphasized that these osseous channels, along with the surrounding venous channels, is considered venous network functioning as a venous reservoir and a rerouting vessel to stabilize the cerebral intracranial pressure and venous pressure during postural changes[26].

## **The summary**

To summarize this small review, I must mention two essential features of the cerebral venous system. First, many textbooks describe that owing to the abundant anastomoses among intracranial veins, sacrificing a sole venous channel doesn't always result in venous congestion or hemorrhage[27]. However, according to the past literature, the statement seems incorrect since venous anastomoses are generally less frequently observed than arteries. Nevertheless, we sacrifice the cerebral veins during neurosurgery, but nothing occurs except the so-called dangerous veins[28]. How does the brain sustain venous perfusion without anastomoses? At this moment, I don't have a clear answer for it. But I assume that the veins have high compliance and can readily dilate and shrink to meet demand, possibly enabling the adjacent veins to supplement the venous drainage of the affected area after sacrificing without anastomoses.

Second, I emphasize the influence of surrounding environmental factors, i.e., the intracranial tumor increases the local ICP and intravenous pressure, impairing the adjoining venous function. In addition, the postural change and cardiac output can also influence

intracranial venous function. Then, the classical functional anatomy emphasizing the morphology and/or the embryological background is essential but not enough to evaluate each vein's function precisely. Recently, the modern radiological techniques using 4D-CTA or 4D-MRA enable us to directly measure the velocity or blood volume of each cerebral vein[29, 30]. These advancements in imaging may further facilitate our understanding of the cerebral venous functional anatomy.

## **Conclusion**

The intracranial veins are not merely pipes connecting the capillaries and the heart. They have complicated morphology and function. To understand intracranial functional venous anatomy, we should consider the morphology, embryology, and many surrounding environmental factors.

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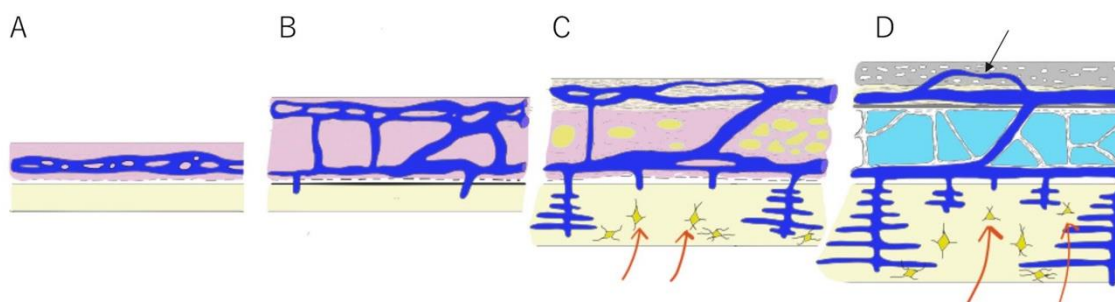
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**Figure 1.** Intracranial veins at the 4<sup>th</sup> embryological weeks (A), at the 5-7<sup>th</sup> embryological weeks (B), at the 8<sup>th</sup> embryological weeks (C), and at the 9<sup>th</sup> embryological weeks (D).

The veins are initially a plexiform network (A). The numerous bridging veins connect the superficial and deep veins (B). Along with the differentiation and growing of the primitive meninges, the bridging veins are elongated and converged into a fewer number of them (C and D).

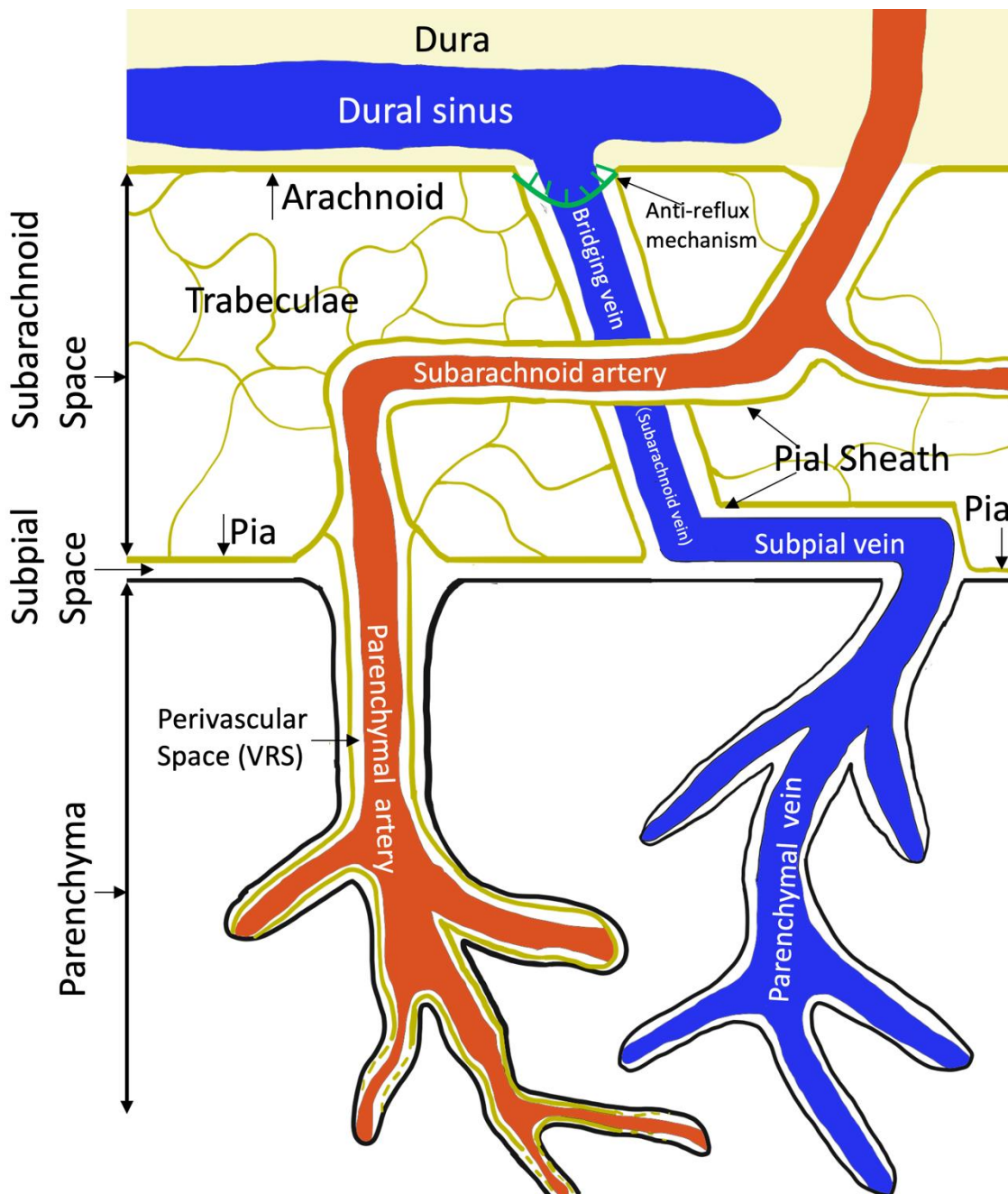


Figure 2. Microanatomy of intracranial veins.

The intracranial veins are classified into four: parenchymal, subpial, subarachnoid veins, and dural sinuses, according to their anatomical dispositions. Both subpial and subarachnoid veins are covered with the pial membrane or pial sheath. The anti-reflux mechanism (outflow cuff/sphincter) borders the two venous compartments: systemic and cerebral venous compartments.