





Cerebral venous anatomy: implications for the neurointerventionalist

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Dedicated to the memory of Professor Yun Peng Huang.

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ABSTRACT

Meaningful contributions to neurointerventional practice may be possible by considering the dynamic aspects of angiography in addition to fixed morphologic information. The functional approach to venous anatomy requires integration of the traditional static anatomic features of the system—deep, superficial, posterior fossa, medullary veins, venous sinuses, and outflow routes into an overall appreciation of how a classic model of drainage is altered, embryologically, or pathologically, depending on patterns of flow—visualization made possible by angiography. In this review, emphasis is placed on balance between alternative venous networks and their redundancy, and the problems which arise when these systems are lacking. The role of veins in major neurovascular diseases, such as dural arteriovenous fistulae, arteriovenous malformations, pulsatile tinnitus, and intracranial hypertension, is highlighted, and deficiencies in knowledge emphasized.

INTRODUCTION

Until the 20th century, the study of vascular anatomy was limited to looking at *ex vivo* channels. Insight into the implied flow required additional imagination. Angiography provided critical information into physiologic anatomy that grounded our understanding of vascular diseases—particularly lesions characterized by arteriovenous shunting and venous congestion. Much of “functional anatomy” is about reanimating the static anatomical notions cemented within medical school curricula into the flowing dynamism of a living subject. In the venous system especially, it is impossible to understand variability, redundancy, and function without flow-based methods.

KEY SOURCES

Our embryological perspective on the venous anatomy derives principally from early work of Dorcas Padget.¹ Key contributions were made by Yun Peng Huang,^{2,3} Raybaud, Lasjaunias, Berenstein, and Ter Brugge.⁴ From a surgical perspective, the works of Rhoton are essential and freely available online.⁵ Marked discrepancy in nomenclature among different sources can be a source of both frustration and misunderstanding (online supplemental material). Many additional sources are listed below.

We will proceed from “inside out”—from the deep venous system and brainstem to superficial veins, sinuses, dura, and drainage pathways, globally illustrated in [figure 1](#).

Deep venous system

Tributaries of the internal cerebral vein classically are subdivided into medial and lateral groups (with respect to the lateral ventricle).² Their angiographic locations are much easier to understand when correlated with cross-sectional imaging—particularly post-contrast volumetric T1 and susceptibility-weighted sequences. The idea of concentric, linked vascular rings provides an excellent basis for understanding variations⁴ ([figure 2A–D](#)). For angiographic visualization, late venous phase images of full-contrast angiograms are best, as subependymal veins continue to opacify after surface veins have cleared.

Basal vein of Rosenthal

Conceptualizing the basal vein as a conduit linking the cavernous sinus, lateral mesencephalic system, and the Galen is key. Variations can be viewed as whole or partial discontinuities between the three main segments (first/anterior/striate/telencephalic, second/middle/peduncular/diencephalic, and third/posterior/mesencephalic)³ ([figures 1 and 2E–H](#)). This broadly anastomotic nature and unique embryology defy outright assignment to either superficial or deep venous systems, highlighting some artificiality in this distinction. One clinical implication is that slowly progressive straight sinus occlusions, or conditions with competing Galenic/straight sinus venous inflow (falcatentorial arteriovenous fistulas), are tolerated better if the internal cerebral veins can drain via the basal vein anastomoses. In addition, the basal vein is also in balance with the convexity, its size often inversely related to that of the superficial middle cerebral (superficial sylvian) veins.

Posterior fossa

Huang’s angiographic work on posterior fossa veins is recommended^{6,7} as well as that of Naidich, Duvernoy and colleagues.⁸ While 2D angiography may be confusing, mental superimposition of brainstem structures on angiographic images is helpful ([figure 2I,J](#)) for vein localization, and may be facilitated by correlation with volumetric post-contrast MRI or cone-beam CT. Veins classically have been subdivided into anterior

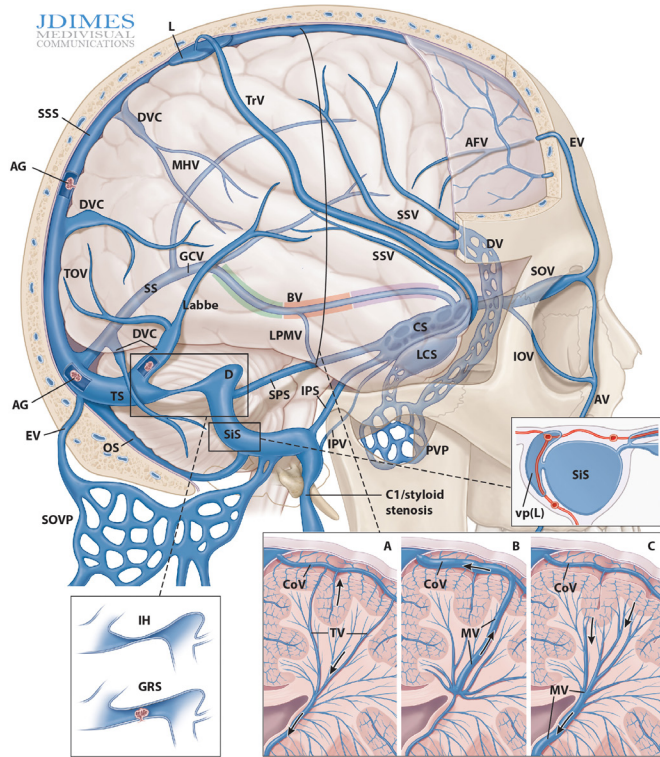


Figure 1 Schematic of cranial and upper cervical venous systems, emphasizing hemodynamic balance, interconnectedness or isolation of available drainage pathways, and specific venous pathologies. AFV, anterior frontal vein; AG, arachnoid granulation; AV, angular vein; BV, basal vein, with its three functional segments (telencephalic=purple; mesencephalic=red; galenic=green). CoV, cortical vein; CS, cavernous sinus; D, diverticulum; DV, diploic vein; DVC, dural venous channel; EV, emissary vein; GCV, Galen; GRS, granulation-related stenosis; IH, intracranial hypertension-related stenosis; IOV, inferior ophthalmic vein; iPS, inferior petrosal sinus; IPV, inferior petroclival vein; L, venous lake; LCS, laterocavernous sinus; LPMV, lateral (anastomotic) pontomesencephalic vein; MHV, medial hemispheric vein; MV, medullary veins; OS, occipital sinus; PVP, pterygopalatine venous plexus; SiS, sigmoid sinus; SOV, superior ophthalmic vein; SOVP, suboccipital venous plexus; SPS, superior petrosal sinus; SS, straight sinus; SSS, superior sagittal sinus; SSV, superficial sylvian (superficial middle cerebral) veins; TOV, temporo-occipital veins; TrV, Trolard vein; TS, transverse sinus; TV, transmedullary (transcerebral) veins; vp(L), venous pouch (Lake). Inserts A-C: A, normal anatomy; B, superficially-draining developmental venous anomaly (DVA); C, deep-draining DVA.

(petrosal), superior (galenic), and posterior (torcular) groups draining the cerebellum and the brainstem group.⁹ Dominance of one group is usually associated with hypoplasia of others. The system is highly redundant and cerebellar venous infarcts are rare. However, the cervicomedullary junction veins are less well collateralized, with congestion pathophysiology of foramen magnum or petrous apex dural arteriovenous fistulas sometimes resembling that of spinal dural arteriovenous fistula homologs (figure 2K,L)

Deep/superficial venous balance and medullary venous system

There is an embryologically based balance between predominant white matter drainage into the subependymal tributaries of the deep venous system, and the subcortical veins draining superficially.¹⁰⁻¹² The superficial and deep systems are linked by vestigial transmedullary (transcerebral) veins (figures 1 and 2A).¹⁰⁻¹² Little is known

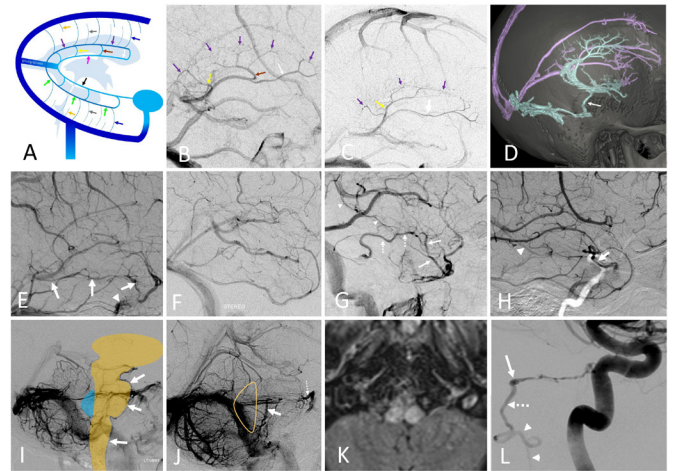


Figure 2 (A–L) Internal cerebral, basal, and deep venous systems (A–L), highlighting venous drainage balance. (A) The idea of “concentric rings” or grid-like arrangement can be very helpful in conceptualizing the deep system and its variations internal cerebral vein – pink; basal – black; thalamostriate – brown; septal – white; direct lateral – yellow; longitudinal caudate – purple; inferior ventricular – green; medullary – grey; subcortical – orange; transmedullary – blue). (B) Typical internal cerebral vein anatomy. The thalamostriate vein collects most of longitudinal caudate drainage, with remainder draining via the direct lateral vein. (C) Longitudinal caudate veins collect posteriorly into the direct lateral vein, often misidentified as “caudal opening of thalamostriate vein” (same colors). (D) The lateral group (green, caudate, inferior ventricular) veins drain via the lateral mesencephalic vein (arrow), while medial group drains “normally” into the Galen. (E) Full extent of basal vein (arrows) from the Galen to the cavernous sinus (arrowhead). (F) No functional cavernous connection. (G) Disconnected basal vein segments. anterior (arrows) drains into cavernous sinus; middle (dashed arrows) into the lateral mesencephalic vein; posterior (arrowheads) into Galen; (H) Hypoplasia of mid-segment (same arrows as G). (I) Superimposing brainstem/fourth ventricle on venous images can be very helpful. Note how well the anterior pontine/mesencephalic/medullary veins (arrows) outline the brainstem. (J) The same group is hypoplastic, with some brainstem draining into vein of the lateral recess (arrow), and into a small bridging opening into the cavernous sinus. (K, L) Medullary venous congestion due to a small petrous apex fistula (arrow) with restricted outflow into the lower brainstem venous system (arrowheads) via the vein of lateral recess (dashed arrow).

about medullary and transmedullary hemodynamic behavior or the extent of individual variation, though superb in vivo resolution is now possible (figure 3A–C). In some “normal” cases, increased prominence of deep veins is associated with reduced caliber of the superior sagittal sinus (SSS), possibly reflecting the global shift in the balance of subcortical drainage into the deep venous system (figure 3D,E). Under extreme pathologic congestion of the vein of Galen, such as falcotentorial junction fistulas, transmedullary veins provide an important alternative pathway for drainage of the deep venous system (figure 3J–L). (figure 3)

The developmental venous anomaly (DVA) can be seen as a consequence of disturbance in the balance of medullary drainage between superficial and deep systems.^{2,13-18} In deep draining DVAs, the normal superficial drainage of the pial system is deficient, and instead routes subcortical drainage via medullary veins into the subependymal system. The opposite deficiency of medullary drainage into the deep venous system may be compensated by subependymal venous drainage towards the convexity via superficially draining DVAs. (figures 1 and 3F) The same applies in cerebellum,

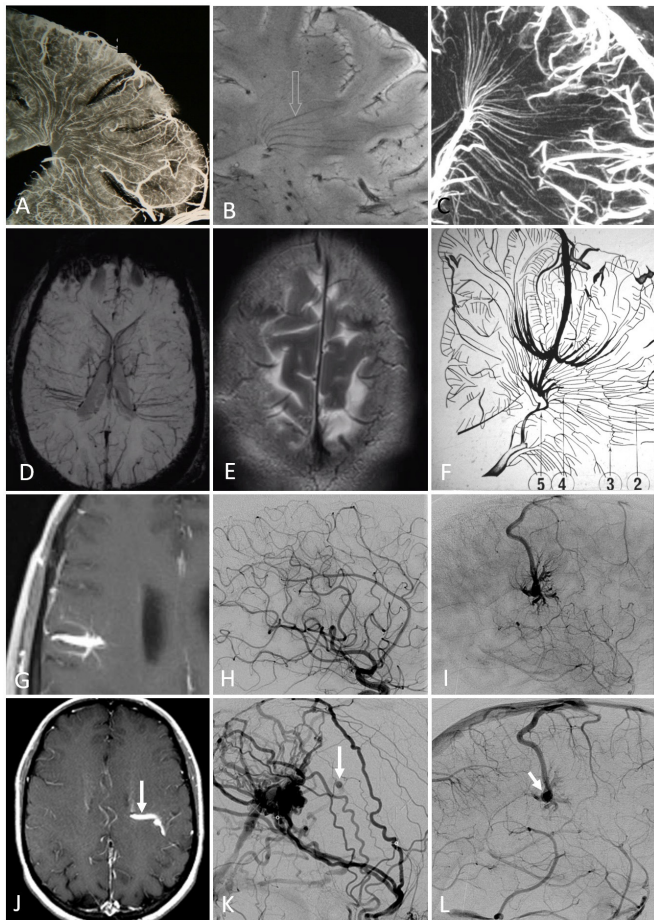


Figure 3 (A–L) Medullary venous system. A–C: Medullary veins ex vivo (A), 7 Tesla T2 (B), Dyna CT maximum intensity projection (C) reconstructions. (D, E) Prominent medullary veins (susceptibility weighted MRI) in a setting of superior sagittal sinus hypoplasia; (F) Yun Peng Huang collection drawing of medullary zones of convergence and relationship with developmental venous anomaly (DVA); (G–I) Rare DVA-appearing lesion is opacified in early rather than late angiographic phase—terms such as “microfistulous” or “arterialized” DVA have been used. Understanding remains limited; diagnosis without flow-based imaging is currently impossible. (J–L) Falcotentorial junction fistula, with prominent transmedullary vein (arrow) on T1 post MRI (J) The same vein is seen in the venous phase of fistula drainage (K), as well as in the venous phase of the brain (L) as sole drainage of the left internal cerebral venous system, attesting to severe congestion.

where the “deep” system is exemplified by the vein of the lateral recess. In all cases, the stem of a DVA is an enlarged medullary vein. The root cause and timing of primary drainage deficiency (lack of development, thrombosis, other) remain unclear.

The above view implies that a territory subserved by a DVA is less able to compensate for additional insults such as developing stenosis or thrombosis—a possible explanation for coexistence of DVAs and cavernous angiomas. The full story is certainly more complicated, and the most remarkable thing about DVAs is how rarely they can be definitively implicated as the root cause of any dysfunction.^{14 15} The very rare “arterialized”, “microfistulous”, or “transitional” DVAs (with premature rather than late venous opacification on angiography or time-resolved cross-sectional imaging) are poorly understood (figure 3G–I).

Another implication of deep-superficial balance concerns angioarchitecture of brain arteriovenous malformations

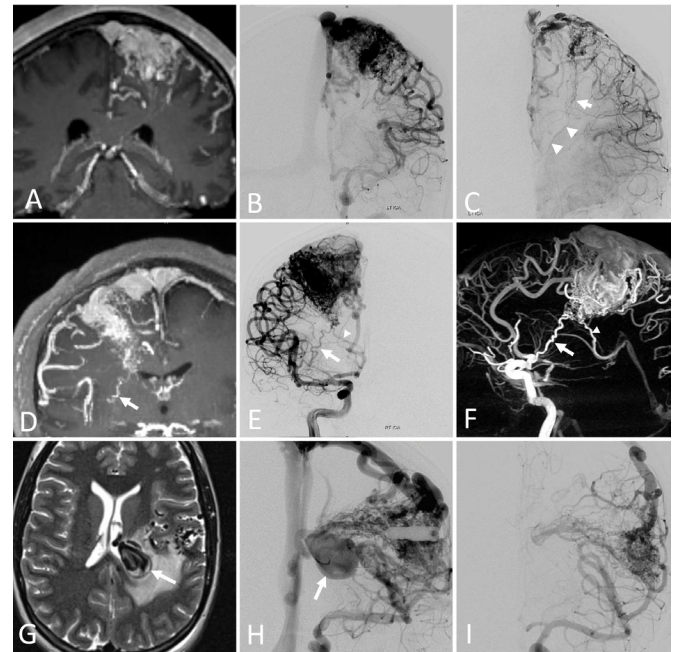


Figure 4 (A–I) Variable drainage of arteriovenous malformations involving deep white matter. (A–C) Despite relatively superficial appearance of a gyral-type nidus (A,B), transmedullary drainage (arrow) into the subependymal system (arrowheads) is present (C). (D–F) The nidus extends to the subependymal surface (D), and has lateral lenticulostriate (arrows) supply (E,F). However, the amount of deep venous drainage (arrowheads) is relatively minor. (G–I) This nidus also extends to the subependymal surface (G), and has massive deep drainage with a symptomatic varix (arrows) in the thalamostriate vein. (I) Post targeted n-butyl cyanoacrylate embolization of deep arterial supply.

(figure 4). Among the wedge-shape/sulcal types extending deep into the white matter, there is marked variability in deep and superficial venous drainage. In most cases there is a single, presumably enlarged, medullary vein central to the nidus. Its drainage direction might be determined by developmental balance in each individual and by subsequent pathophysiological changes. The single draining vein morphology, superficial or deep, implies a deficiency in alternative outflow and has been shown to be a risk factor for increased hemorrhagic potential.¹⁹

Superficial venous system

The countless morphologic variations in surface venous anatomy can be conceptualized as balance between the four main collectors:

1. Trolard/Rolando/superior anastomotic
2. Labbé/inferior anastomotic
3. Superficial middle cerebral/superficial sylvian
4. Basal

Progressive dominance of one is usually associated with corresponding hypoplasia of others (figure 5A–F). The balancing factor is often dictated by the degree of interconnectedness, or lack thereof, between these systems (figure 5A–C). This can be helpful in deciding which veins may be surgically sacrificed or preserved. Preoperative identification of uncommon drainage patterns, such as large frontal veins draining into the anterior-most segment of the superior

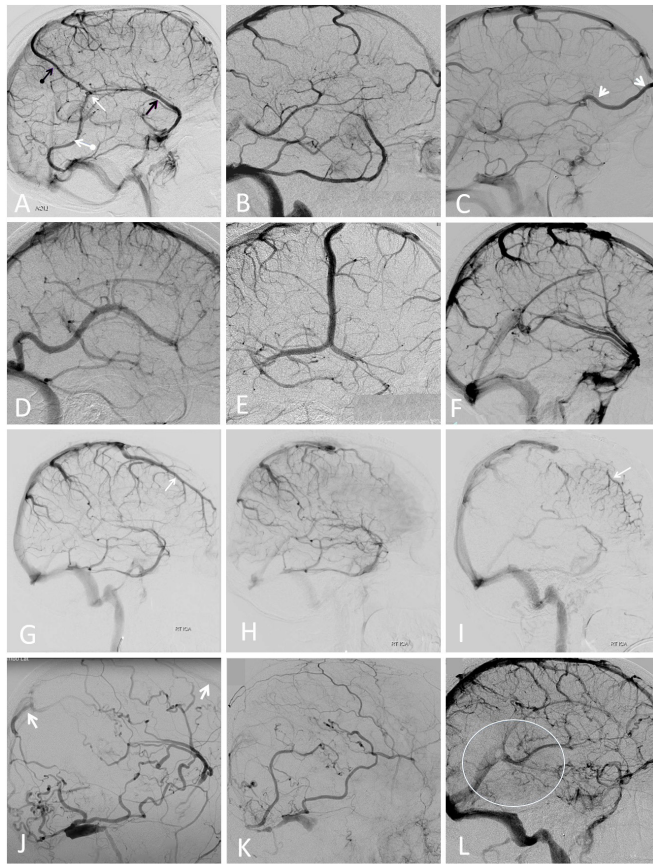


Figure 5 (A–L) Spectrum of interconnectivity and dominance in superficial venous system: (A) fully well-developed and interconnected superficial sylvian (black arrow), Labbé (white ball arrow), and superior anastomotic/Trolard (black ball arrow) systems; white arrow – confluence. (B) Well-developed but disconnected convexity veins. (C) Prominent and isolated (no interconnections) anterior frontal vein can be important in surgical planning. (D–F) Developmental dominance of Labbé (D), Rolando/Trolard system (E), superficial middle cerebral (also known as superficial sylvian) (F) veins is associated with hypoplasia of other systems. (G–I) Prominent parasagittal convexity isolated vein (arrows) before (G) and after inadvertent intraoperative occlusion (H,I), with marked venous congestion. (J) Left external carotid injection – sigmoid dural fistula with “trapped sinus” and extensive cortical reflux, but no hemorrhage or edema due to the presence of Labbé collaterals with sylvian and convexity veins emptying into the superior sagittal sinus. This is contrasted with extensive venous infarct (K) in another trapped sinus sigmoid fistula, where left external injection (K) shows Labbé congestion with no collaterals. Venous phase internal carotid artery injection in the same patient (L) shows congestion of the temporal lobe (oval) and lack Labbé visualization.

sagittal sinus (figure 5C,G–I) is important in assessing the need for sinus-sparing procedures.

The goal of evaluation often relies on a gestalt impression: imaging must demonstrate the presence of venous drainage for all major portions of the hemisphere—lack thereof should raise suspicion of either occlusion (isolated cortical or venous sinus thrombosis) (figure 6) or venous congestion by another source of inflow (as in dural fistula).

Dural fistulas are best seen as venous diseases. The arterial component is mainly relevant during treatment, while venous pathophysiology is the principal driver of clinical presentation and disease evolution, as reflected in main fistula classification

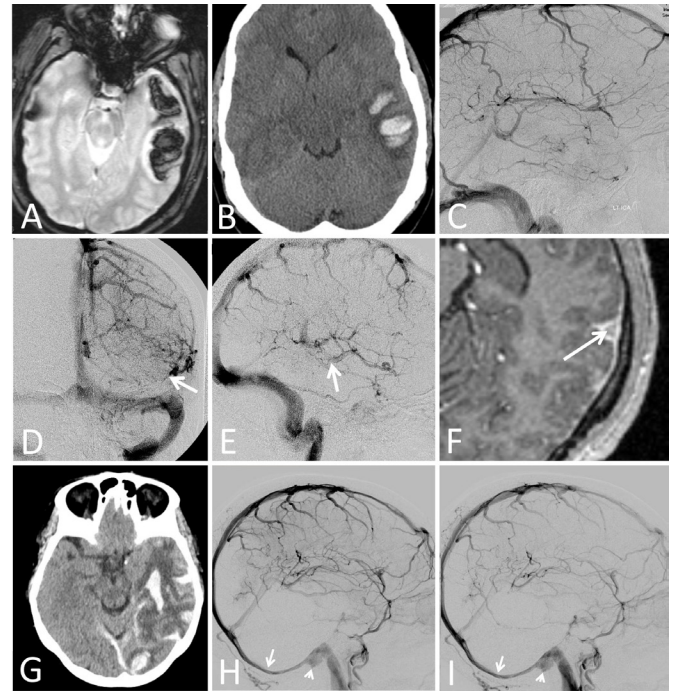


Figure 6 (A–I) spectrum of venous thrombosis. A–C: “Cryptogenic” left temporal hematoma (A, B) is angiographically shown to result from isolated cortical venous thrombosis; note lack of temporal lobe venous drainage (C) and patent sigmoid sinus. (D–F) Another isolated cortical thrombosis of the Labbé. Stump with filling defect (D,E arrows), and on subsequent MRI (F, arrow) (G–I) Extensive venous infarction in setting of left transverse/sigmoid sinus thrombosis. The size of infarction is due to developmental dominance of the now occluded Labbé – the other convexity veins are small. The right transverse sinus is hypoplastic. The superior sagittal sinus drains into the patent left jugular bulb via the occipital sinus (arrows). Note thrombus-related stump of the proximal sigmoid sinus (arrowheads).

systems.^{20 21} More difficult to adjudicate, but very important, are patient-specific differences in the extent of venous collateralization as determinants of congestion and venous infarct: extensive cortical reflux can be tolerated with efficient venous collaterals (figure 5J), whereas major hemorrhagic infarcts result from small fistulas draining into veins with no effective anastomoses (Figure 5K,L).

Dural sinuses

The entire dura is a kind of “tabula rasa”—a sheet of tissue with tremendous arterial and venous developmental capacity.²² The dural arterial network is highly redundant²³ and capable of massive proliferation, particularly in dural shunts. Likewise, any part of the dura can support a sinus, which is one of the hallmarks of “meninx primitiva”—the early embryonic tissue from which dura will develop.²² Major sinuses can be fenestrated, multichanneled, or deviated, thus coursing away from dural reflections (figure 7A–F)—with implications for surgical planning, especially for posterior fossa approaches. Sufficient information is often available on cross-sectional studies.

With respect to dural fistulas, the seat of disease is almost always a (probably venous) channel or pouch within the sinus wall, on which the arterial tributaries converge. Identification and superselective transvenous embolization of these pouches

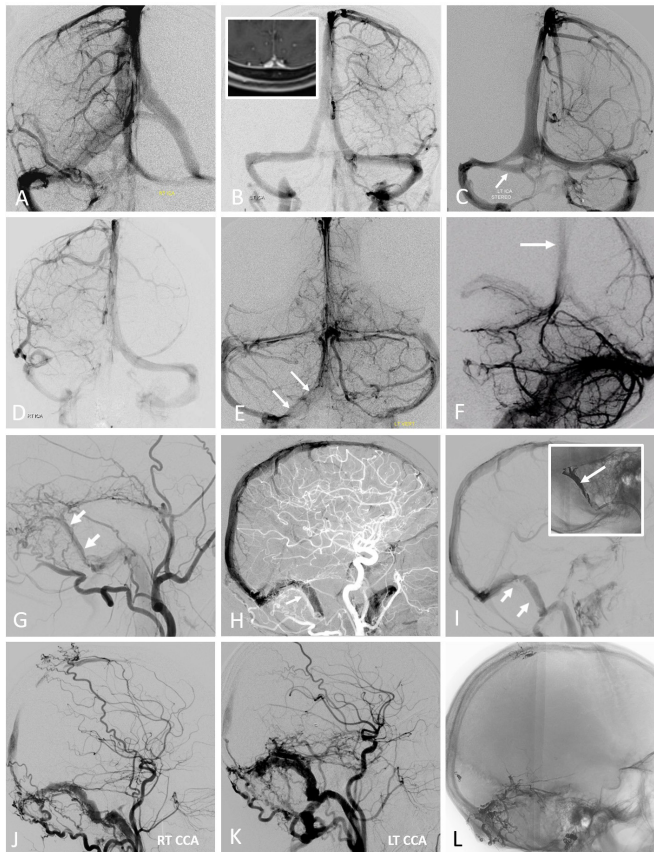


Figure 7 (A–L) Variable architecture of dural venous sinuses. (A–C) Mutichanneled (A) and fenestrated (B) superior sagittal sinus (SSS). (C) Fenestration of transverse sinus. (D) Classic transverse sinus hypoplasia—the sinus sigmoid is well developed as it receives the large Labbé. (E) Occipital sinus (arrow) usually drains into the torcular, not marginal sinus. Note balanced hypoplasia of both transverse sinuses. (F) Incidental falcine sinus with no pathology. (G–I) Superselective transvenous dural fistula embolization. (G–I) Grade I sigmoid fistula with multiple arterial pedicles converging on a single common channel (arrows) in the inferior wall of the sigmoid sinus (G), well seen on an arterial mask image of venous phase DSA (H). A small amount of liquid embolic (I) injected via transvenous route produces cure with minimal risk and sinus preservation. (J–L) Highly complex case of multiple bilateral transverse/sigmoid/condylar and SSS dural fistulas (J,K) cured by superselective transvenous embolizations of all but one fistulous pouches within sinus walls with sinus preservation and minimal amount of strategically placed embolic material (L). Only one site required left occipital transarterial closure.

is often technically feasible, with sinus preservation and less risk than transarterial routes (figure 7G–L).^{24–26}

Dural venous channels

Focal physiologic narrowing is often seen at the “bridging” segment of a cortical vein entering the sinus. However, it is quite common, especially for larger caliber cortical veins, to join the dura some distance away from a major sinus—emptying into a “dural channel” which then joins the larger named sinus (figure 1 and figure 8). This can be recognized by the abrupt transition between the round cross-section of a cortical vein and the biconcave morphology of the dural sinus. Particularly prevalent posteriorly, receiving the terminal drainage of the Labbé and occipital veins, they sometimes contain an arachnoid granulation

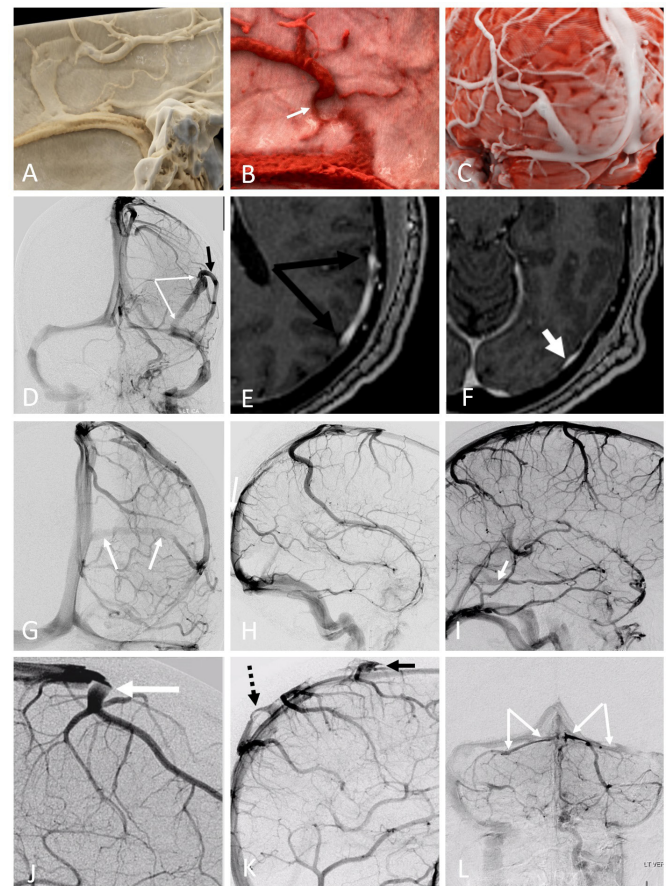


Figure 8 (A–L) Spectrum of dural venous channels. (A–C) Cinematic rendering in three cases—round cortical veins join a flattened dural channel, which then empties into the named sinus. A prominent arachnoid granulation (arrow) is seen in (B). (D–F) Angiographic and MRI appearance of the same patient with a large Labbé-like vein (black arrows) emptying into a long dural sinus (white arrows). (G, H) Long occipital channel (arrows), easily overlooked on a lateral view. (I) Tentorial sinus channel draining the basal vein. (J) Frontal parasagittal convexity vein, with transition point (arrow) into dural channel. (K) Convexity junctions with “venous lakes,” projecting above the superior sagittal sinus. (L) Tentorial channels are extremely common, seen on frontal views as a thin line (arrows) along the tentorium, receiving posterior/torcular group of cerebellar surface veins.

(figures 1 and 8B). These under-recognized channels are quite common (~25%) with obvious clinical implication to avoid damaging them during surgical exposure.

Much better known is the frequent presence (over 85%) of analogous sinus channels in the tentorium cerebelli,^{27,28} typically receiving the torcular group veins (figure 8L). An occasional basal vein, with hypoplasia of its posterior segment, will drain into a tentorial channel (figure 8I). The interhemispheric cortical veins may enter a dural channel within the falx. Also well known are “dural venous lakes” receiving large superior convexity veins²⁹—another expression of dural venous potential (figure 8J,K).

An unproved clinical implication is the possible relationship between dural channels and “tentorial”/convexity dural fistulas^{30,31} (figure 9). These are characterized by seemingly direct communication between dural arterial networks and a cortical vein, away from a major sinus. We believe that the anatomical location for this connection is the dural venous channel, which prior to establishment of the shunt served as drainage outlet

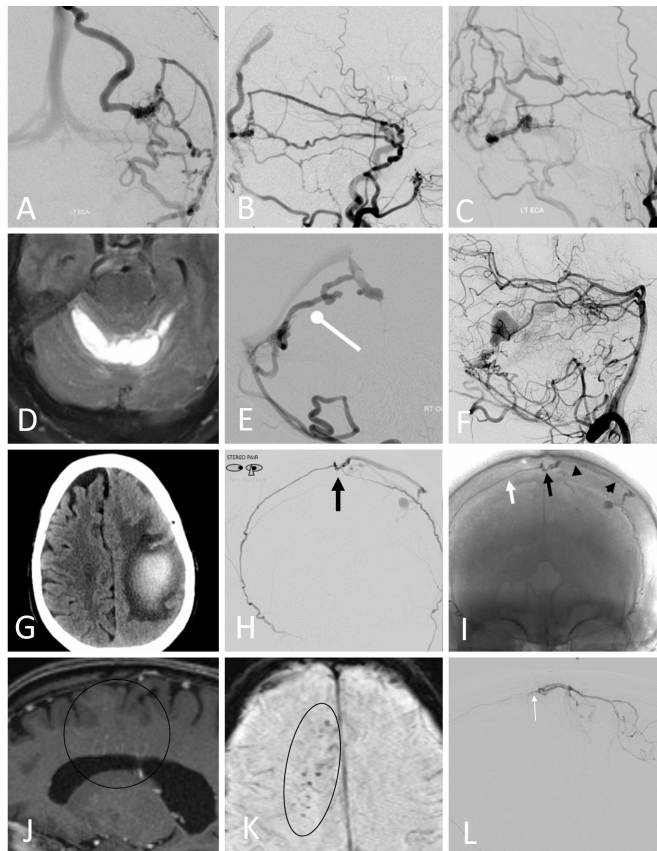


Figure 9 (A–L) Spectrum of dural fistulas draining into an isolated vein. The fistula site may be located within a dural venous channel. (A, B) Temporo-occipital and (C) inferior temporal fistulas. (D, E) Hemorrhage (D) related to tentorial fistula (E) emptying into ectatic superior vermician vein (ball arrow). (F) Venous varix in another tentorial fistula. (G–I) Small fistulas can result in large venous infarcts (G) when there is no functional venous outflow. A convexity fistula (black arrows), with n-butyl cyanoacrylate cast in the right middle meningeal artery (white arrow) and recipient cortical vein (arrowheads). (J–L) Another convexity fistula. Note enlargement of transmedullary veins (ovals) which help drain the convexity and avoid the kind of large infarct seen in G–I. The microcatheter (arrows) is nearly at the fistula point (L).

of the index cortical vein. The usual sites of these uncommon fistulas mirror those of dural venous channels—tentorium, temporo-occipital convexity, and parasagittal vertex.

Venous sinus stenosis

Stenoses at transverse/sigmoid sinus junctions have been definitively linked to intracranial hypertension³² and pulsatile tinnitus^{33–36} (figure 10). The latter can be present as a sole symptom, without intracranial hypertension, or in a setting of mildly increased cerebrospinal fluid pressures, with no visual or other findings. The typical clinical history is unilateral pulsatile tinnitus on the side of the narrowed sinus, completely or nearly completely abolished by ipsilateral jugular compression. In this setting, venous stenting has been consistently effective in abolishing pulsatile tinnitus. The jugular vein compression maneuver is also key for other suspected ipsilateral venous causes, including diverticula, jugular wall dehiscence, and C1 jugular vein stenosis in our experience. Lack of aural symptom change with ipsilateral jugular compression reduces the outcome success of any venous treatment, including C1-styloid process region surgeries.

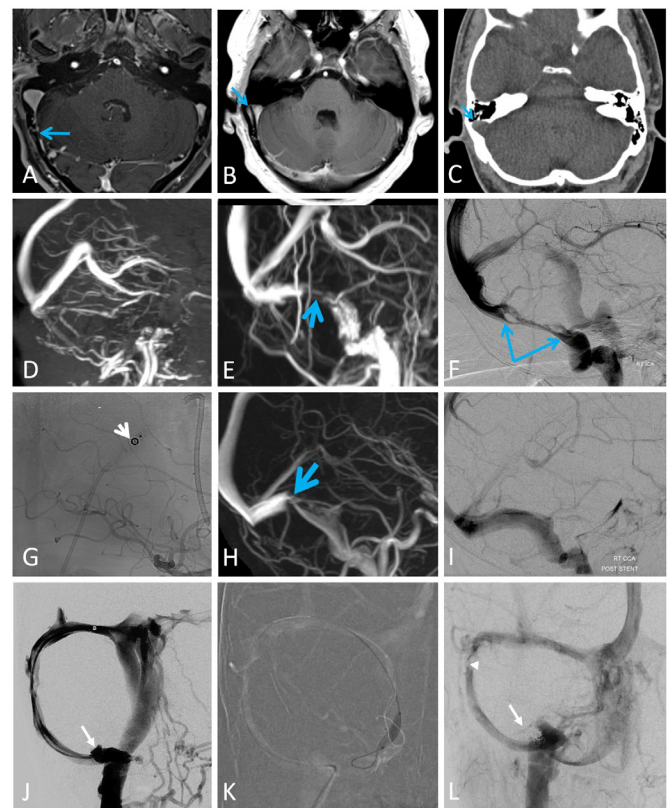


Figure 10 (A–L) Spectrum of sinus stenosis. (A–C) High-grade stenosis (A, arrow), fat-suppressed volumetric T1-post. A jet flow artifact, sometimes seen especially well on a 2D T1 post (B, arrow), highlights the secondary nature of many sigmoid sinus diverticula, which are caused by flow-related temporal bone remodeling (C, arrow). Note mirror image stenosis on the left. This patient presented with standalone pulsatile tinnitus, which resolved after stenting. (D–F) Substantial variability in visualization of sinus hampers numerical quantification of stenosis. Typical over-estimation on time of flight (D), compared with contrast MR venography (E) and angiography (F) — note contralateral stenosis in (F). (G–I) Persistent stenosis (H, blue arrow) despite presence of working programmable shunt (G, white arrow). Shunting abolished all intracranial hypertension symptoms except for severe pulsatile tinnitus, which resolved after stenting (I). (J–L) Unusual case of right pulsatile tinnitus in patient with small sigmoid stenosis (arrowhead) and large occipital sinus, with a flow-related diverticulum (J, arrow) at its junction with the torcular. (K) Balloon inflation within occipital sinus in awake patient abolished the sound. Coiling of the diverticulum (L, arrow) abolished pulsatile tinnitus while preserving the large occipital sinus.

Most, though not all, sinus diverticula in our experience are secondary phenomena, developing in response to stenosis-related flow jet remodeling of the temporal bone (figures 1A–C and 10). Stent dilation of the stenosis with coverage of the diverticulum predictably has been successful in alleviating symptoms in our experience, although primary occlusion of the diverticulum may work as well—with stenting reserved if the pulsatile tinnitus persists or returns.

In the setting of medically refractory intracranial hypertension, sinus stenting continues to supplant ventriculoperitoneal shunting.^{30–32} Highly favorable initial results, however, are followed by recurrence in 10–20% of patients, often with imaging findings of sinus narrowing outside the stent construct.^{32–37} In our opinion, the cause–effect relationship

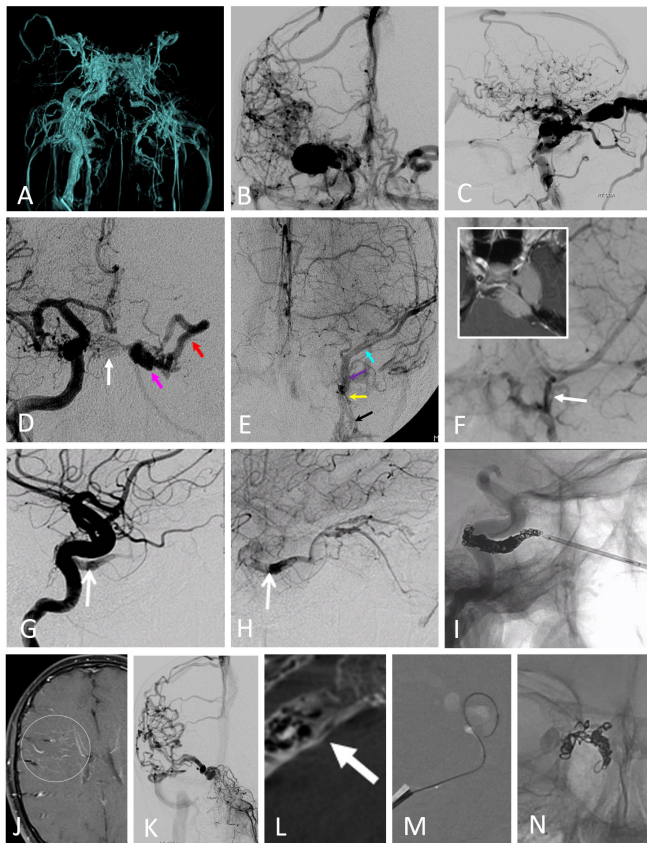


Figure 11 (A–N):Cavernous and laterocavernous sinuses. (A–D) The cavernous sinus as junction of extracranial and intracranial venous systems is well demonstrated by massive congestion in direct carotid cavernous fistulas: (A) patient 1, volume rendered Dyna; (C, D) patient 2 with proptosis and status epilepticus. (D–E) Functional independence of laterocavernous sinus. (D) Left cavernous sinus (pink arrow) dural fistula, supplied in part via posterior clival branches of the right meningohypophyseal trunk (white arrow), congests the left ophthalmic vein (red arrow). (E) However, dominant left superficial sylvian veins (blue) continue to drain normally into the hemodynamically separate laterocavernous sinus (purple), which empties via foramen ovale (yellow) into the pterygopalatine venous plexus (black). (F) Meningioma (inset) fills the cavernous sinus. Nevertheless, large sylvian drainage into the patent laterocavernous sinus (arrow) is unaffected, and needs to be preserved. (G–I) Small posterior cavernous dural fistula producing severe visual acuity impairment due to lack of effective orbital venous outflow—as in brain and spine, lack of outflow is more important than fistula size. (I) Cure by direct orbital puncture. (J) Medullary venous congestion. (K) Large posterior cavernous dural fistula with exclusive cortical drainage. (L) Barely seen proximally (arrow) and occluded distally inferior petrosal sinus, which could nevertheless be catheterized and traversed (M) to allow for superselective coil occlusion of the fistulous pouch (N).

between sinus stenosis and intracranial hypertension remains unclear at this time.

Outflow – cavernous sinus

Key topics related to the venous disposition of the cavernous sinus are its bidirectional linkage between intracranial and extracranial venous systems, and the compartmentalized confluence of cerebral venous drainage into the cavernous sinus (figure 11).^{38–40} The frequently present laterocavernous sinus—a separate compartment lateral to the outer wall of the cavernous

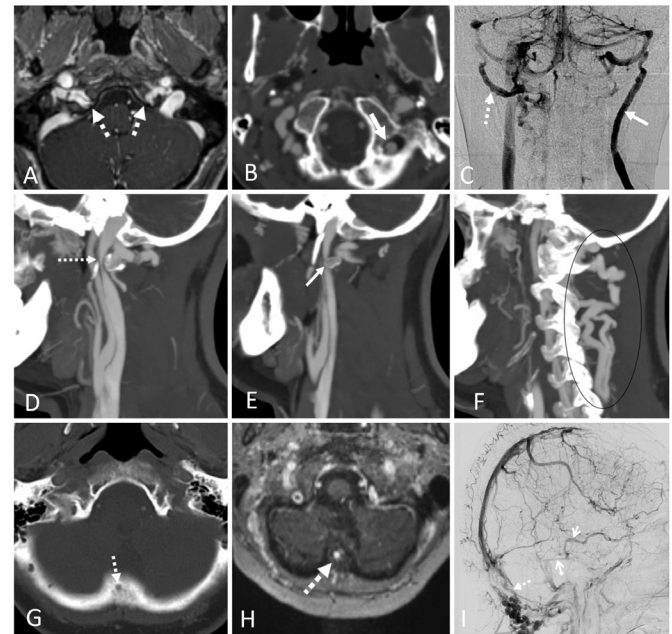


Figure 12 (A–L) Condylar/emissary veins. The jugular, condylar/suboccipital, and emissary/diploic outflow systems are in balance. (A) Anterior condylar veins (dashed arrows) are the same as the hypoglossal vein, traversing the canal. (B) Large posterior condylar vein (arrow). (C) The left posterior condylar vein (arrow) drains almost the entire sigmoid sinus into the external jugular vein, in the setting of ipsilateral jugular C1 stenosis. Right jugular stenosis is less severe, and a smaller posterior condylar vein (dashed arrows) drains into the vertebral venous plexus. (D–F) Supine position bilateral prominence of the suboccipital veins (F) associated with extrinsic jugular narrowing between the lateral mass of C1 and the styloid process (D, left side, dashed arrow, E, right side, solid arrow). (G–I) Extensive thrombosis of bilateral transverse, sigmoid, straight sinuses, and anterior superior sagittal sinus. The bulk of both hemispheres drains via an unassuming but life-sustaining torcular emissary vein (dashed arrows). A critical flow channel in the otherwise occluded right sigmoid sinus drains the basal vein (arrowhead), which empties via the lateral mesencephalic vein into patient superior petrosal sinus (ball arrow). There was no venous infarct and the patient made a full recovery.

sinus, often receives drainage from the sphenoparietal sinus or directly from superficial middle cerebral veins.³⁸ It may remain functional despite congestion or occlusion of the cavernous sinus proper by dural arteriovenous fistulas, carotid cavernous fistulas, or masses (figure 11D–F).

Most cavernous dural fistulas form in the posterior portion of the sinus, and typically are treated by transvenous routes, given the transarterial hazards posed by extensive extracranial–intracranial anastomoses and potential for cranial nerve injuries.⁴¹ The myriad venous connections of the sinus allow for multiple potential approaches, including via the inferior or superior petrosal sinuses (figure 11J–N),^{42–43} the superior ophthalmic vein, or direct puncture through the orbit or foramen ovale (figure 11G–I).^{44–47}

Outflow – jugular, condylar, diploic, emissary veins

The many pathways for venous blood to exit the skull may be considered as balanced and complementary, with deficiency of one usually compensated by prominence of alternatives. Progressive lack of redundancy is associated with correspondingly increased likelihood of dysfunction if primary outflow

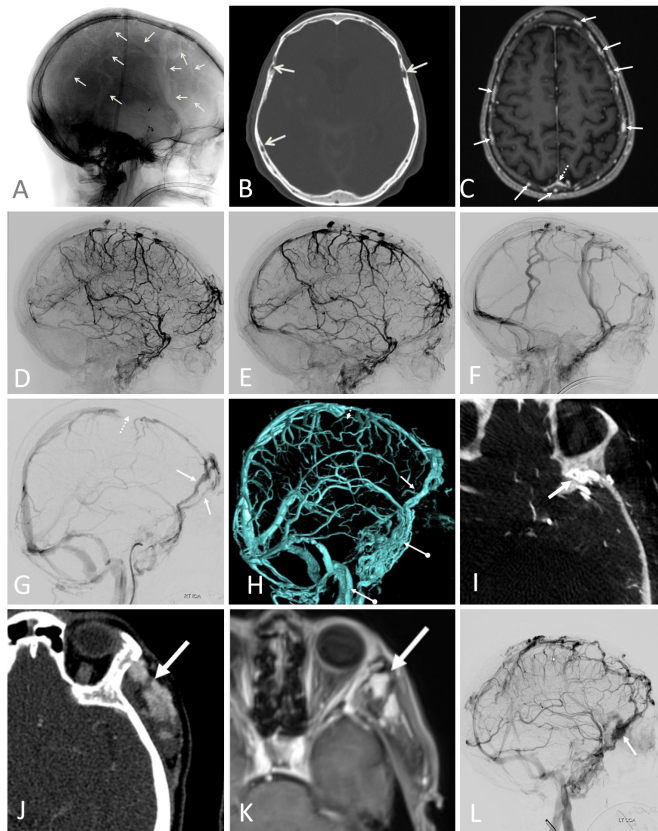


Figure 13 (A–L) Diploic and emissary veins. Typical appearance of diploic venous skull lucencies on X-ray (A) and CT (B). (C) Prominent diploic channels (arrows) compensating for hypoplasia of the superior sagittal sinus (SSS, dashed arrow). (D–F) Same patient, early (D), late (E), and very late (F) venous phases. It is often difficult to appreciate how much flow diploic/emissary veins carry in individual cases without an angiographic study. They usually appear very late in the venous phase (F), and are easy to miss otherwise. (G–I) Prominent bifrontal diploic veins (arrows) draining anterior frontal convexity in the setting of focal SSS (dashed arrows). The diploic nature is best seen on axial Dyna image (I). (J–L) Sclerotherapy referral for position-dependent left temporalis region swelling, presumed venous malformation (J,K). Angiography (L) shows another case of SSS hypoplasia, with diploic drainage opening into the same venous pouch. No sclerotherapy was performed. Similar compensatory drainage can be seen in sinus pericranii.

constraint develops. For example, unilateral venous sinus thrombosis is much better tolerated if the contralateral transverse/sigmoid system is well-developed.⁴⁸

The conceptual overemphasis on “normal” internal jugular drainage impedes understanding of cerebral venous disorders. Angiographic data are gathered overwhelmingly in a non-physiologic, fully supine position (most of us do not even sleep this flat). Outflow differs substantially between recumbent and standing positions,^{49–51} and much more study is needed.

Incidental narrowing of the internal jugular vein between the lateral mass of C1 and the styloid process is common.^{52–53} Notwithstanding the demise of multiple sclerosis misassociation, it continues to surface as a target for remedy of “styloidogenic headache”/venous Eagle syndrome, etc. The paucity of systematic studies invites future thoughtful investigation.⁵⁴

The condylar system is a major source of physiologic outflow, especially in non-supine positions [figure 1](#) and [figure 12A–F](#).^{51–55} Its prominence varies, and is usually greater with C1 level jugular narrowing ([figure 12D–F](#)). There is currently no clear indication for closure of condylar or other outflow veins in the hopes of alleviating pulsatile tinnitus, as it is likely to worsen rather than improve global outflow dynamics, even if pulsatile tinnitus improves due to overall flow reduction.

Substantial venous outflow also may occur through diploic and emissary veins ([figures 1 and 3](#)), in balance with other outflow routes ([figure 12G–I](#)). Anterior superior sagittal sinus (SSS) hypoplasia may be compensated by frontal diploic or emissary drainage ([figure 13G–I](#)), while rare global SSS hypoplasia may be associated with adaptive drainage into superficial middle cerebral, Labbé, or internal cerebral systems, as well as diploic/emissary veins ([figure 13A–F](#)). Sinus pericranii is the extreme form of emissary venous drainage—its main defining feature being superficially visible to the eye. Its widespread definition as a venous malformation illustrates the conundrum of inadequate understanding of cerebral venous balance, and is better viewed (like the developmental venous anomaly) as an adaptive drainage route. Carefully planned closure of small sinus pericranii can be well-tolerated.⁵⁶ Occlusion of larger ones, especially without preoperative angiographic evaluation, risks disastrous venous infarction, while correction of frequently associated craniosynostosis can improve cerebral outflow.⁵⁷

Limitations/future directions

Our quantitative understanding of both cerebral and cranial outflow dynamics remains basic, as does the relationship between venous sinus morphology, venous pressures, cerebrospinal fluid pressure, flow, and absorption, especially in non-supine positions. Arterial test occlusions, although imperfect and less often necessary in the age of flow diversion, remain highly useful, especially in complex surgical cases. However, we currently have no way of performing meaningful venous test occlusions, since parenchymal injury in the setting of outflow restriction is often delayed and difficult to access prospectively. To date there are no practical methods for cerebral venous repair, or for improving venous outflow by creation of new routes—hence our strong emphasis on preserving existing ones.

The subject of glymphatics is important, with increasing animal and human evidence of their existence and potentially important roles in fluid balance, immunity, waste product clearance, and sleep, among others.^{58–59} Imaging normal lymphatic channels anywhere in the body is challenging. A whole new dimension in cerebrovascular organization will continue to be a topic of intense research.

We can expect continued improvement in imaging capabilities, which already rival *ex vivo* methods. Tremendous amounts of anatomical and physiologic data are being accumulated and will probably yield progress in many areas. Likewise, evolution in endovascular devices is likely to facilitate safer and more advanced venous catheterizations, which may open new therapeutic frontiers.

CONCLUSIONS

Modern imaging capabilities allow for superb correlation between cross-sectional (usually static) and angiographic (dynamic) sources, while advanced cross-sectional tools provide stunning *in vivo* resolution that will continue to

improve, promising advances in our venous understanding, which still has many limitations.

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