Vascular malformations involving the brain are divided into subgroups, including arteriovenous malformations (AVM), developmental venous anomalies (DVA), cavernous malformations and capillary telangiectasias. These lesions are further categorized into those that demonstrate shunting from arterial to venous systems (i.e. the AVM), and those that do not have shunting (DVA, cavernous malformation, and capillary telangiectasia). In addition, mixed malformations (i.e. lesions composed of more than one malformation) occasionally occur. The most common lesions seen within a mixed malformation are the DVA in combination with a capillary telangiectasia. Understanding the associated imaging findings and potential complications of these lesions assists in determining the appropriate treatment options.

Arteriovenous Malformation

Arteriovenous malformations are high flow shunts between the arterial and venous systems without an intervening capillary bed. These lesions are subdivided into the classic arteriovenous malformation and the arteriovenous fistulas.

Classic Arteriovenous Malformation

The classic AVM (also known as pial AVM) results from an abnormal connection between the arteries that normally supply the brain parenchyma and the veins that would normally drain this region. Imaging and pathology reveal an enlarged feeding artery, a nidus consisting of numerous arteriovenous shunts and dysplastic vessels, and an enlarged draining vein. The nidus can be characterized as glomerular (compact and the intervening brain is not normal), or diffuse (normal brain is interspersed between the abnormal vessels). (Fig. 1) The glomerular form is the most commonly encountered, whereas the diffuse form is rare, but is the type found in cerebrofacial arteriovenous metameric syndrome (CAMS).\(^2\) CAMS is a rare, non-hereditary disorder consisting of brain-retino-facial angiomatosis resulting multiple AVMs involving the face, eye and brain. (Fig. 2) AVMs occur anywhere in the brain or spinal cord, but the majority are supratentorial. Ninety-eight percent of these lesions are solitary. If multiple AVMs are seen, syndromes such as hereditary hemorrhagic telangiectasia (HHT) or CAMS need to be considered.\(^2\)

Figure 1: AVM with glomerular nidus. Photograph of a gross specimen reveals a compact nidus adjacent to the left lateral ventricle with no intervening normal brain. Notice that the left hemisphere is smaller than the right. This finding results from chronic ischemia due to vascular steal.

Figure 2: CAMS. Axial FLAIR (A) and sagittal T1 (B) reveal multiple flow voids consistent with an extensive, diffuse AVM involving the left hemisphere, as well as the soft tissues of the face and scalp.
The peak age for presentation is in the 20 to 40 year old age group (mean age approximately 30 years).\(^3\) Fifty percent of these patients present with hemorrhage, and the risk for hemorrhage is 2-4% per year.\(^4,5\) Once a hemorrhage occurs, there is a 6-18% risk of rehemorrhage within the first year.\(^6\) This rate gradually returns to 2-4% per year. The mortality rate with the first hemorrhage is approximately 10%. Each successive hemorrhage has an increasing mortality rate, and by the time the patient has their 3\(^{rd}\) hemorrhage the mortality rate reaches 20%.\(^6\) Twenty-four to 30% of patients present with seizure unrelated to hemorrhage.\(^3,7\) Other non-hemorrhage presentations include headache and focal neurologic deficit.

The underlying etiology of AVMs remains unknown, but various hypotheses suggest they are congenital in origin and result from either retention of early embryonic vascular connections, alteration of these vascular connections, or alterations of the embryonic vascular remodeling process in combination with angiogenesis or dysgenesis of the capillary system.\(^8,9\) More recent hypotheses challenge the congenital origin and suggest that these lesions may result from angiogenic and inflammatory responses to a postnatal event, and several case reports describe de novo development of AVMs.\(^10,11\) AVMs demonstrate a dysregulated angiogenesis and undergo continued vascular remodeling. Vascular endothelial growth factor (VEGF), which mediates endothelial proliferation, has been found to be elevated in these patients.\(^12\)

In the glomerular form, neural tissue is typically found between the dysplastic vessels of the nidus. (Fig. 3) This tissue is frequently atrophic and gliotic and calcification may be seen. The surrounding brain may develop gliosis resulting from vascular steal related to the AVM.

Imaging findings of AVMs vary depending on the size of the lesion, and presence of hemorrhage or calcification. On CT calcification may be seen in up to 30% of cases.\(^13\) CT and MR angiography can be utilized to demonstrate the feeding arteries and draining veins. Smaller lesions may be difficult to visualize on non-contrast CT since they are filled with flowing blood that is isoattenuating to slightly hyperattenuating to normal brain. On MRI, flow voids are seen within the lesion, giving the classic “bag of worms” appearance. T2* GRE may reveal hypointense blooming if hemorrhage or calcification is present. In some cases, T2 hyperintensity is identified in the adjacent brain parenchyma consistent with areas of gliosis. (Fig. 4) Conventional angiography is still considered the “gold standard” for demonstrating the internal angioarchitecture. The internal carotid, external carotid and vertebral circulation must all be evaluated, since approximately 27% of AVMs have a dual arterial supply.\(^14\) Conventional angiography is considered to be the best method to detect intranidal aneurysms.

There are characteristics of AVMs that may be associated with a greater risk of hemorrhage. One of the greatest risk factors for hemorrhage is evidence of prior hemorrhage.\(^15\) Utilization of gradient echo imaging can assist in detecting evidence of prior hemorrhage. The location of the lesion plays a role. AVMs in periventricular regions, in the basal ganglia, or involving the thalamus are at greater risk. These regions are supplied by small perforating arteries that are not designed to handle
Venous drainage is also associated with higher risk. Veins are more prone to developing stenosis. Single results in increased pressure. The central draining factor is obstruction of venous outflow, which of hemorrhage. (Fig. 5) Another important risk factor is obstruction of venous outflow, which results in increased pressure. The central draining veins are more prone to developing stenosis. Single venous drainage is also associated with higher risk. Other risks from AVMs include nonhemorrhagic neurologic deficits. Imaging findings suggesting a higher risk of neurologic deficit include evidence of arterial steal, high flow shunt, mass effect or hydrocephalus, venous congestion or outflow obstruction, and long pial course of the draining vein.

AVMS can be treated with surgery, endovascular embolization, radiosurgery, or a combination of these methods. Certain characteristics of the AVM help predict the risk of surgical morbidity and mortality. The Spetzler Martin grading scale is a method of scoring AVMs to predict surgical morbidity and mortality. The size, location, and venous drainage of the lesion is assessed and graded on a point scale (Table 1). If the score is one or two, the risk of surgical mortality is less than 1% and the surgical morbidity is less than 10%.

<table>
<thead>
<tr>
<th>Size</th>
<th>Small (&lt;3cm) = 1</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Medium (3-6 cm) = 2</td>
</tr>
<tr>
<td></td>
<td>Large (&gt;6 cm) = 3</td>
</tr>
<tr>
<td>Location</td>
<td>Noneloquent = 0</td>
</tr>
<tr>
<td></td>
<td>Eloquent = 1</td>
</tr>
<tr>
<td>Venous drainage</td>
<td>Superficial = 0</td>
</tr>
<tr>
<td></td>
<td>Deep = 1</td>
</tr>
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</table>

Table 1. Spetzler Martin Grading Scale for AVMs

Arteriovenous Fistulas
Arteriovenous fistulas are distinguished from AVMs by the presence of a direct, high flow fistula between artery and vein. There is no intervening nidus. These include the dural arteriovenous fistula (dAVF), the cavernous carotid fistula (CCF) and the vein of Galen malformation (VOG).

Dural Arteriovenous Malformation
The dural arteriovenous malformation (dAVF) consists of arteriovenous shunts within the dura. Arterial supply is from meningeal branches and drainage is via either dural sinuses or meningeal or subarachnoid veins. They comprise 10-15% of intracranial vascular malformations.
There are 2 types of dAVF – adult and infantile.\(^\text{18}\) The infantile form is a rare congenital condition in which multiple high-flow arteriovenous shunts occur involving several thrombosed dural sinuses. Fetal US reveals a hypoechoic heterogeneous lesion centered on the torcula. Fetal MR confirms the dural location of the mass. T1-weighted images reveal heterogeneous signal with areas of hyperintensity; hypointensity is typically seen on the T2-weighted images. (Fig. 6) In one study in which pathologic evaluation was performed, a large dural hematoma was noted in the posterior fossa, corresponding to thrombus in the enlarged cerebral veins.\(^\text{18}\) The etiology of the infantile form is postulated to be a response to venous obstruction in the fetus.\(^\text{19}\)

**Figure 6.** Fetal dural sinus arteriovenous fistula. (A)SSFSE T2 weighted image of a 22 week fetus demonstrates a large, hypointense mass in the dural space centered on the torcula. (B) Coronal T1 post contrast performed on day of life 1 demonstrates an enlarged sagittal sinus with thrombosis.

The adult form typically presents in middle-aged to older patients and is thought to be secondary to dural sinus thrombosis resulting from trauma or inflammation, but the etiology is not completely understood.\(^\text{20}\) One of the proposed etiologies involves 3 steps: 1) initial thrombosis of a dural sinus leading to impaired venous drainage and increased sinus pressure; 2) secondary dilatation of physiologic shunts between the thrombosed sinus and arterial structures (typically extracranial); 3) recanalization of the thrombosed sinus allowing for direct arterial shunting into the sinus.\(^\text{21}\) These lesions consist of tiny vessels in the wall of a thrombosed dural venous sinus. The most common locations involve the transverse-sigmoid sinuses and cavernous sinus.\(^\text{21}\) The clinical features vary from mild complaints, such as headache, vertigo, and tinnitus to neurologic deficits and hemorrhage. In general, symptoms are related to the drainage pattern and location.\(^\text{21}\)

Different classification systems exist for the dAVF, one of which is the Cognard classification. (Table 2) The more cortical drainage present, the greater the risk of hemorrhage. Davies, et al. categorized them as benign or aggressive lesions depending on the presence or absence of retrograde leptomeningeal venous drainage (RLVD).\(^\text{22}\) In type IV dAVF by the Cognard classification, there is direct cortical drainage and venous ectasia; two-thirds of these cases will have hemorrhage.\(^\text{23}\) The presence of RLVD on angiography indicates a clinically aggressive lesion and is an indication for active treatment, which may consist of endovascular embolization, surgical resection, or radiosurgery.

<table>
<thead>
<tr>
<th>Type</th>
<th>Venous drainage</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Antegrade sinus drainage</td>
</tr>
<tr>
<td>II</td>
<td>Insufficient sinus drainage</td>
</tr>
<tr>
<td>IIa</td>
<td>Retrograde sinus drainage</td>
</tr>
<tr>
<td>IIb</td>
<td>Retrograde CVR</td>
</tr>
<tr>
<td>IIa+IIb</td>
<td>Retrograde sinus drainage and CVR</td>
</tr>
<tr>
<td>III</td>
<td>CVR only without venous ectasia</td>
</tr>
<tr>
<td>IV</td>
<td>CVR only with venous ectasia</td>
</tr>
<tr>
<td>V</td>
<td>Spinal venous drainage</td>
</tr>
</tbody>
</table>

CVR = cortical venous reflux


**Table 2.** Cognard Classification of dAVF

Non-contrast CT may be normal. CT angiogram potentially reveals tortuous dural feeders and an enlarged dural sinus. In aggressive cases, enlarged cortical draining veins can be seen. (Fig. 7) On MRI, flow voids may be seen around the involved dural sinus, and the sinus may be thrombosed. T2 weighted images reveal heterogenous signal with areas of hyperintensity; hypointensity is typically seen on the T2-weighted images.
hyperintensity may be seen in the adjacent brain due to vascular steal. (Fig. 8) White matter edema suggests venous congestive encephalopathy. The MRA may be negative, but an occluded sinus with collateral flow may be seen on MRV. Conventional angiography reveals multiple arterial feeders, most commonly from dural or transosseous branches of the external carotid artery, but tentorial or dural branches from the internal carotid or vertebral arteries can also be involved. The involved dural sinus is often thrombosed. Flow reversal may be seen in the dural sinus and the cortical veins. In clinically aggressive cases, tortuous engorged pial veins are seen. This is referred to as the pseudophlebitic pattern. (Fig. 9)

**Carotid Cavernous Fistula**

The carotid cavernous fistula (CCF) consists of an abnormal communication between the internal carotid artery (ICA) and cavernous sinus resulting in enlargement of the cavernous sinus. The superior ophthalmic vein (SOV) will typically be enlarged; however, in some cases the dilated SOV will be contralateral to the CCF. The Barrow system classifies CCF by arterial supply and venous drainage. (Table 3) These are either direct and high flow due to a direct communication between the ICA and the cavernous sinus, or indirect and lower flow due to a connection between the cavernous sinus and dural ICA or external carotid artery (ECA) branches. A direct CCF is usually due to trauma or intracavernous rupture of an ICA aneurysm. The indirect form typically occurs spontaneously and is only rarely secondary to trauma.\(^{24}\) Patients typically present with ocular symptoms – pulsating exophthalmos, orbital bruit, chemosis – resulting from anterior venous drainage. More aggressive symptoms such as intracranial hemorrhage are rare.\(^{25}\)

Imaging findings reveal orbital proptosis, enlarged extraocular muscles, dilated superior ophthalmic vein, and enlargement of the cavernous
sinus on the involved side. Normally, the walls of the cavernous sinus should always be straight or concave. MRI imaging reveals an increase in the number of flow voids within the involved sinus. (Fig. 10) Angiography reveals prompt filling of the superior ophthalmic vein in the arterial phase. (Fig. 11) Conventional angiography may help elucidate the etiology and be utilized for embolization with coils or glue.

<table>
<thead>
<tr>
<th>Type</th>
<th>Arterial supply and venous drainage</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Direct ICA-cavernous sinus high-flow shunt</td>
</tr>
<tr>
<td>B</td>
<td>Dural ICA branches-cavernous shunt</td>
</tr>
<tr>
<td>C</td>
<td>Dural ECA branches-cavernous shunt</td>
</tr>
<tr>
<td>D</td>
<td>ECA/ICA dural branches shunt to cavernous sinus</td>
</tr>
</tbody>
</table>

ICA = internal carotid artery, ECA = external carotid artery

Table 3. Barrow’s Classification of Carotid Cavernous Fistulas

Figure 10. Carotid cavernous fistula. Axial T2-weighted image reveals multiple flow voids within the left cavernous sinus (arrows). The wall of the cavernous sinus is convex on the left. Left orbital proptosis is present and the extraocular muscles are enlarged and edematous. There is inflammation of the periorbital and intraconal fat.

Figure 11. Carotid cavernous fistula. Maximum intensity projection from a 3D time of flight MRA demonstrates increased signal in the right cavernous sinus, and the right superior ophthalmic vein is visible (arrow).

but the arteriovenous shunts with the MPV are thought to occur during 6 to 11 weeks gestation. The resultant increased flow through the MPV prevents the transient fetal venous drainage pattern from involuting. In 50% of these patients, the straight sinus does not form and the vein drains via a falcine sinus. (Fig. 12) These lesions most commonly present in the neonatal period and are the most common cause of non-cardiac congestive heart failure in the newborn period. Patients may develop ischemic damage involving the brain parenchyma due to a vascular steal. Occasionally, they first present later in infancy and rarely, they can present in adulthood. Older infants usually present with milder cardiac symptoms, most often coming to medical attention due to hydrocephalus from compression of the aqueduct or posterior 3rd ventricle. Seizures may also occur. Children and adults may present with headaches or subarachnoid hemorrhage. Patients presenting in the neonatal period typically have a worse prognosis than those presenting later. They are more common in males than in females with a ratio of 2:1.

VOGM are classified as either choroidal or mural. In the choroidal form, there are multiple feeders from the pericallosal, choroidal, and thalmo/perforating arteries resulting in an extensive arterial network between the arterial feeders and

Vein of Galen Malformation

The vein of Galen malformation (VOGM) is an arteriovenous fistula resulting in dilatation of the median prosencephalic vein (MPV), which is the embryonic precursor to the vein of Galen. The exact etiology of the malformation is not known,
the dilated venous pouch. (Fig. 13) In the mural form, there are few feeders from collicular or posterior choroidal arteries that end directly within the wall of the MPV. Over 90% of these lesions are choroidal, and the choroidal form typically presents in neonates. The mural form usually presents in infancy.

Figure 12. Vein of Galen malformation. Sagittal T1-weighted image reveals abnormal flow voids communicating with an enlarged falcine sinus (arrow).

Figure 13. Choroidal vein of Galen malformation. DSA lateral view after internal carotid injection shows multiple feeds predominantly from pericallosal and choroidal arteries. Early filling of the enlarged venous pouch is demonstrated.

CT imaging reveals a dilated venous pouch. Mass effect from the dilated vein may result in hydrocephalus due to compression of the aqueduct of Sylvius. Parenchymal atrophy may be seen due to vascular steal, and calcifications may be seen secondary to ischemic brain damage. (Fig. 14) Rarely, intraventricular hemorrhage can occur. CT angiography can be utilized to map the arterial and venous structures. However, catheter angiography is still the gold standard for the evaluation of VOGM angioarchitecture. On MR, T1 hyperintensity may be seen in the pouch if a thrombus is present. T1 hyperintensity can also be seen in the brain parenchyma in the setting of ischemia or calcification. Reduced diffusion may be seen in the setting of acute infarction. In severe cases, diffuse brain destruction, referred to as melting brain, may be seen.

Figure 14. Vein of Galen malformation. (A) Axial post contrast CT demonstrates enhancement of the enlarged venous pouch. Encephalomalacia with ex vacuo dilatation of the left lateral ventricle is noted. (B) Photograph of gross specimen demonstrates marked cerebral atrophy due to the vascular steal along with the enlarged venous pouch.

The timing of endovascular treatment is determined by the patient’s clinical presentation. If congestive heart failure is refractory to medical therapy in the newborn period, emergent embolization is necessary. If the patient is not in heart failure, then embolization can be delayed until 5-6 months, reducing the risk of affecting brain maturation. Embolization may be achieved through the arterial or venous route. The arterial method is preferred since occlusion of the venous aneurysm may interfere with deep venous drainage or result in perforation of the venous aneurysm.
Prior to the development of endovascular embolization, the mortality rate for neonates with VOGM was 100%. In a recent study by Lasjaunias, et al., the mortality rate for neonates was 52%. The treatment of associated hydrocephalus is controversial. Shunt placement may alter venous drainage which can exacerbate brain damage, and is usually reserved for refractory hydrocephalus after transcatheter embolization performed.

Cavernous malformation

Cavernous malformations (CM) are slow flow lesions, which in the past resulted in them being referred to as “occult” or “cryptic” malformations, since they did not show up on catheter angiography. They occur in about 0.4 – 0.8% of the population, and account for 10-15% of all CNS vascular lesions, making them the second most common CNS vascular malformation after DVAs. These lesions may be inherited in an autosomal dominant pattern in up to 20% of cases, in which case there are multiple lesions in over 50% of cases. The remainder are sporadic, and in these cases only 10-20% will have multiple lesions. Patients most commonly present in the 3rd or 4th decades of life, but one-quarter of cases present in infancy or childhood. Patients may present with seizure, focal neurologic deficits, or acute intracranial hemorrhage; however, most are asymptomatic at the time of diagnosis. The location of the lesions plays a role in the symptomatology. Those that are infratentorial tend to more commonly present with hemorrhage, whereas supratentorial lesion are more likely to present with seizure.

CMs are vascular hamartomas comprised of variable sized intercapillary vascular spaces, sinusoids, and larger cavernous spaces without intervening brain tissue. These lesions lack smooth muscle support and are filled with blood or thrombus. CM are associated with hemosiderin, whether or not they have overtly hemorrhaged, which may be related to absent or diminished tight junctions seen on ultrastructural studies. This implies a localized loss of the blood-brain barrier and diminished vascular stability.

CMs may occur in combination with DVAs, and there is evidence to suggest a causative link between the two. Dan Millan Ruiz, et al. reported the presence of hemosiderin laden macrophages in the region of the DVA. Suggested etiologies for this finding include blood diapedesis through the walls of the venous radicals of the DVA or rupture of a venous radical. It is hypothesized that repeated microhemorrhages activate angiogenic growth factors that over time result in the formation of a CM.

On imaging, these lesions have little or no mass effect unless they are complicated by hemorrhage. The risk of hemorrhage is 0.25-0.6% per year. It has been reported that CMs associated with DVAs tend to hemorrhage more frequently than isolated malformations. Once hemorrhage occurs, the annual risk of recurrent hemorrhage goes up to 4.5%. However, some have reported that the increased risk is only elevated for the first 2-3 years after the initial hemorrhage, and then drops back to baseline.

CT imaging is frequently negative, especially if the lesion is less than 1 cm, or in the absence of
calcification or hemorrhage. Calcification may be seen in up to 60%. On T2-weighted imaging, a peripheral hemosiderin rim may result in a black “halo” around the lesion. (Fig. 16) The adjacent brain parenchyma is normal, and surrounding vasogenic edema is not present unless the lesion is complicated by hemorrhage. (Fig. 17) These lesions may have internal areas of thrombosis or hemorrhage, and these blood products are of varying ages. The presence of methemoglobin results in hyperintensity on T1 images. Findings on post-contrast imaging are variable, with enhancement ranging from none to moderate. It is important to look for a DVA on post contrast imaging, as the location of the DVA will be important if surgical treatment is planned in order to avoid inadvertent damage to venous drainage.

![Figure 16. Cavernous malformation. Axial T2-weighted image reveals a cavernous malformation within the right temporal lobe. The low signal rim corresponds to hemosiderin deposition.](image)

Treatment for these lesions includes observation in cases where the lesions are asymptomatic or surgical resection. In patients who present with seizures, surgical treatment should be utilized if their seizures increase in severity or do not respond to antiepileptic drugs. In patients with hemorrhage, a conservative approach is usually initially taken, but if the hemorrhage causes severe neurological symptoms or recurs, then surgery should be considered. Radiosurgery may be considered as an alternative for those lesions that are progressively symptomatic but surgically inaccessible.

![Figure 17. Hemorrhagic cavernous malformation. (A) Axial T2-weighted image reveals a cavernous malformation with mild mass effect and surrounding vasogenic edema secondary to hemorrhage. Fluid-fluid levels are seen within the lesion, and a partial hemosiderin rim is present along the medial aspect. (B) Axial T1-weighted image without contrast reveals a “popcorn ball” lesion with areas of hyperintensity within the CM corresponding to methemoglobin.](image)

**Developmental Venous Anomaly**

Developmental venous anomalies (DVA) are the most commonly encountered cerebral vascular malformations. In a series of 4,069 brain autopsies, they were encountered in 2.6% of cases. They are thought to represent anatomic variants that arise from maldevelopment of fetal cortical venous drainage, most likely resulting from recruitment of parenchymal veins to compensate for loss or absence of a portion of the cerebral venous system. DVAs are comprised of enlarged medullary veins that drain into a venous trunk that flows into a dural sinus or deep ependymal vein, resulting in a “palm tree” or caput medusa appearance on imaging. (Fig. 18) These lesions are usually solitary, although two or more draining separate regions of the brain have been reported in 1.2-16% of cases.

Patients are usually asymptomatic with the DVA representing an incidental finding on imaging, but acute thrombosis of the collecting vein may result in hemorrhage or infarction. Associated cavernous malformations, which have been reported to be...
associated with DVAs in 13-40% of cases, may also be an etiology of hemorrhage or seizure.\textsuperscript{49,50}

![Image of developmental venous anomaly](image)

**Figure 18.** Developmental venous anomaly. (A) Axial T1 post contrast reveals a “palm tree” appearance of a left frontal developmental venous anomaly that drains into a deep ependymal vein. (B) Axial T1 post contrast demonstrates an associated cavernous malformation (arrow).

The classic imaging finding of the *caput medusa* allows for ease of diagnosis on both contrast enhanced CT or MRI. On non-contrast CT, the draining vein will typically appear isoattenuating to slightly hyperattenuating to the cortex, but if acutely thrombosed, then a markedly hyperattenuating vein may be seen. MR imaging may reveal flow voids in the region of the medullary veins and draining vein depending on size. Occasionally on MR imaging, T2 hyperintensity may be seen in the region of a DVA. This may occur in completely asymptomatic patients, but can also reflect acute edema from thrombosis, or gliosis from chronic outflow obstruction. In a study of 84 DVAs by San Millan Ruiz, et al. utilizing CT and MRI imaging, brain parenchymal abnormalities were noted in close to two-thirds of cases. These findings included locoregional brain atrophy in 29.7%, white matter lesions in 28.3%, cavernous malformations in 13.3%, and dystrophic calcifications in 9.6%. They also reported intraparenchymal hemorrhage possibly related to the DVA in 2.4% and stenosis involving the collecting vein in 13.1%.\textsuperscript{50} Catheter angiography can be utilized to evaluate the hemodynamic behavior of developmental venous anomalies in patients presenting with hemorrhagic or ischemic complications in the drainage territory of a DVA.\textsuperscript{42}

DVAs are “leave alone” lesions in that resection will result in venous infarction of the area drained by the DVA.

**Capillary Telangiectasia**

Capillary telangiectasias represent localized collections of dilated capillary-like vessels interspersed within normal brain. (Fig. 19) These lesions comprise 4-12% of vascular malformations, and are usually small, asymptomatic, incidental findings, although there are rare reports of symptoms including hemorrhage, seizure, vertigo, tinnitus, and cranial nerve dysfunction.\textsuperscript{51} There is one reported case of a malignant fatal progression.\textsuperscript{52} The etiology of these lesions is unclear, but it is hypothesized that they are acquired lesions secondary to underlying venous anomalies.\textsuperscript{53} Most are located in the pons, but other common locations include the temporal lobe, medulla, or caudate. They are usually small, but in a small number of cases, they may exceed 1 cm in size.\textsuperscript{51} The majority are solitary lesions, but they can be multiple in syndromes such as Osler-Weber Rendu, ataxia telangiectasia, or Sturge Weber. Unlike other vascular malformations of the brain, there is no reported calcification, gliosis or hemosiderin laden macrophages in the adjacent brain on microscopic exam.\textsuperscript{51}

Capillary telangiectasias are not visible on CT imaging. MR imaging may reveal a focal area of T2 hyperintensity that on post-contrast imaging reveals ill-defined enhancement described as having a “stippled” or “brush stroke” appearance. An enlarged vessel is seen adjacent to these lesions in approximately two-thirds of cases, thought to represent a draining vein.\textsuperscript{54} T2\* imaging reveals associated low signal thought to reflect the presence of deoxyhemoglobin due to sluggish blood flow through the region.\textsuperscript{55} (Fig. 20) There is no
associated mass effect. These lesions are occult on angiography, which may reflect sluggish flow, hemorrhage within the lesion, or abnormal vessels of small size.51

Unfortunately, these lesions may be mistaken for a more aggressive process, such as glial neoplasm. Location, low signal on T2*, enhancement, and lack of mass effect help support the diagnosis of capillary telangiectasia. A study by Finkenzeller, et al. found that these lesions also have a low signal on DWI, and they suggest utilizing this finding to help increase diagnostic accuracy.55 Most are small and are monitored over time. No treatment is required.

Summary
There are a variety of vascular malformations of the CNS. Some of these are aggressive, high flow lesions and have a risk of hemorrhage or other complications; others behave in a more benign manner. Knowledge of the imaging findings of these lesions, along with the findings that might indicate which ones may hemorrhage or have other associated poor outcomes, aids in making treatment decisions.

The views expressed in this material are those of the author, and do not reflect the official policy or position of the U.S. Government, the Department of Defense, or the Department of the Air Force.

References