Neuroanatomy of cranial dural vessels: implications for subdural hematoma embolization

Maksim Shapiro, Melanie Walker, Kate T Carroll, Michael R Levitt, Eytan Raz, Erez Nossek, Nader Delavari, Osman Mir, Peter Kim Nelson

INTRODUCTION
General aspects of dural vascular anatomy and pathology

Based largely on gross anatomical observations drawn from cadaveric dissections, Dr Henry Gray concluded in his classic textbook that the dura mater was nearly avascular.1 Over the next century, the discovery of vessel specific stains and other visualization methods led to the demise of this misconception. Modern tools have continued to advance our knowledge of the anatomy and pathophysiology of dural based diseases.2–6

The dura has three layers, each of which is associated with a vascular network. From superficial to deep, there are periosteal, meningeal, and border zone layers. The corresponding vascular networks are named outer, transitional, and inner (figure 1). The periosteal layer of cranial dura is apposed to the periosteum, and consists of a dense capillary inner network, located in the innermost border zone layer, where cells of the same name closely adhere to the arachnoid. Vessels within this capillary sized network have a characteristic parallel appearance, measuring about 10 μm in diameter. The majority of subdural hematomas arise within the inner capillary layer, enlarging in a cycle of hemorrhage, hyperproliferation, fragility, and re-hemorrhage.7 8

No less an authority than Virchow described what we now know as subdural hematoma as pachymeningitis hemorrhagica interna, suggesting an inflammatory etiology.9 10 Later, this view was largely abandoned in favor of the torn bridging vein hypothesis. While this may be valid for major trauma, notably the non-accidental neonatal type,11 the theory has suffered from inconsistent association with demonstrable trauma, indefinite demonstration of recognizably torn bridging veins (on intraoperative or post mortem inspection), and the simple improbability of frequent ruptures in a tiny potential space. Nevertheless, ‘minor trauma’ continues to be seen as an important inciting, if not recurrent, event in hematoma pathogenesis.

Among other things, Dr Henry Gray described the dural outer capillary network as originating from the more extensive microvascular supply of the arachnoid. Indeed, there are consistent descriptions of large perivascular arachnoidal capillaries traversing into the superficial dura mater, where they connect with the outer layer of cranial dura.12 Along this line, a novel, comprehensive study by Sigalet et al. in 2010 described a network of capillary size vessels forming connections between the dural covering and the superficial arachnoid based on in vivo studies.13

Although the outer dural layer is avascular to a degree, it is intimately associated with a microvascular plexus known as the middle meningeal plexus, which arises from the middle meningeal artery (MMA) and its major branches.14 The network is a fractal-like structure covering the entire dura. Its primary anastomotic arteries range between 100 and 300 μm,15 connect the major dural branches, and are readily visible angiographically (figure 2). There is substantial variation in the size and pervasiveness of these anastomoses—no different than in all things vascular (figure 2). The secondary anastomotic arteries, still in the outer layer and measuring 20–40 μm, link the primary ones and may be visible on DYNA CT or similar high resolution volumetric imaging, especially when pathologically enlarged (figures 2 and 3). Outer network vessels also participate extensively in supply of the skull—its angiographic enhancement can be difficult to differentiate from that of dura. Finally, several ex vivo studies suggest the presence of physiologic arteriovenous anastomoses/shunts in the outer layer, perhaps as large as 50–90 μm in diameter.14

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On the other hand, in 1965, Rowbotham and Little, in a seminal paper, wrote: “Clinically there are two types of subdural hematoma, one being due to traumatic rupture of the superior cerebral veins as they cross the subdural space to enter the superior sagittal sinus. In this group the prognosis following surgical drainage is good. In the other type, however, no history is obtained of trauma and the outlook following surgical drainage is poor. We suggest here that this second group of hematoma arises from oozing from the inner dural plexus,
trick of encouraging reports, would have gained attention sooner had the prevailing idea of bridging vein rupture not been adhered to as devoutly, or had the long existing literature on true pathogenesis been better known in the endovascular community.

Meningeal venous system
While the genus specific pattern of meningeal veins on the endocranium is well known to paleontologists, dural venous anatomy receives almost no attention in the medical literature, despite Padget’s seminal work outlining many anatomical features that now can be observed angiographically. The role of the meningeal veins in the evolution of subdural hematoma remains unclear. For example, to what extent do inner layer veins contribute to the bleeds? Are the aforementioned physiological arteriovenous shunts fact or artifact? Fortunately, these questions seem to have no immediate implication on transarterial embolization.

Meningeal lymphatic system
The role of meningeal lymphatics in subdural hematoma also remains unclear. While their apparent location parallel to the dural venous sinuses and MMA places them in the proximity of the meningeal pathology, their role is yet to be explored.

Angiographic aspects of dural vascularization

Inner network
The normal inner capillary network, composed of vessels of about 10 µm diameter, is below angiographic resolution. When chronic subdural collections are present, the varied patterns or enhancement are likely those of a pathologically thickened and hyperemic inner layer (figures 3 and 4). If imaged during active surgical drainage is because the compressional factor is minimal, the dominant pathology being degenerative vascular changes within the brain tissue itself. This long quote is a beautiful example of how anatomical advances lead to informed disease insight.

The idea of chronic subdural hematoma (cSDH) as a product of recurrent bleeding within the inner layer is supported by multiple studies. Indeed, the membranes frequently seen within the hematoma, as well as on its inner surface, are composed of border zone cells and proliferating, fragile inner layer vessels—not ‘naked’ clot against the arachnoid—establishing the hematoma as, at least histologically, intradural (figure 1). This is by no means a fringe view. While we invite the reader to look deeper into the notion of classical three layered meninges, and particularly the dura–arachnoid junction, certainly for historical and practical reasons the ‘subdural’ hematoma will continue to be referred to as such.

The notion of subdural hematoma resulting from repetitive, indolent inner layer hemorrhage naturally suggests embolization as a management strategy, first reported by Mandai et al in 2000 and more recently highlighted in multiple publications and reviews. We may also wonder whether a straightforward procedure such as MMA embolization, with a two decades long historical and practical reasons the ‘subdural’ hematoma will continue to be referred to as such. While we invite the reader to look deeper into the notion of classical three layered meninges, and particularly the dura–arachnoid junction, certainly for historical and practical reasons the ‘subdural’ hematoma will continue to be referred to as such.

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microhemorrhage, characteristic pooling of contrast can be seen (figure 4A–C) on DSA. Variation in the degree of membrane enhancement may be due to differences in membrane stage, size, and proliferative activity (figure 4D–I).

**Outer network**

The primary anastomotic vessels, approximately 100–300 μm in diameter, are large enough to allow for small particles and liquid embolic agents to penetrate into adjacent vascular territories. ‘Wedged’ microcatheter position can be used to establish flow arrest conditions within the catheterized vessel distal to the microcatheter tip and is particularly useful as part of an embolization strategy (figure 5). The outer arterial network anastomoses across midline (figure 2F–H) with corresponding branches of the contralateral fractal outer network serving as both potential access to and a possible revascularization route for the contralateral convexity hematoma. Outer layer arteries also supply the skull (figure 2C,D); its enhancement may be difficult to differentiate from that of the dura.

Both ex vivo and angiographic studies point to the consistent presence of discrete arteries or arterial networks in the walls of major venous sinuses (figures 2 and 6). These channels strategically link multiple arterial territories, and play an important role in the angioarchitecture of dural fistulas. Apart from proximity to the jugular foramina, they are located well away from
The secondary network of the outer layer (below 50 µm) can be demonstrated in the falx cerebri and tentorium cerebelli on cranial nerves, and can be useful vascular routes for embolization (figure 6E–H).

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Figure 6  (A–H) Arterial network in sinus walls. (A) Schematic, used with permission from www.neuroangio.org. (B) Double mask common carotid artery injection showing occipital origin jugular branch visualizing the arterial channel in the wall of the sigmoid sinus heading towards a dural fistula (arrow). (C) Pial to dural anastomoses outlining arteries in the wall of transverse (black arrow) and sigmoid (white arrow) sinuses, also supplying a dural fistula (pink arrow). (D) Posterior meningeal supply to artery in the wall of the straight sinus (arrowheads) in a patient with falcotentorial junction fistula (arrow). (E–H) Same patient. Ethmoidal dural fistula, with prominent supply via paired arteries in the wall of the superior sagittal sinus (same as anterior meningeal arteries). This allowed for endovascular access to the fistula, followed by its closure with Onyx-18 (G, H).

Petrous and cavernous branches
The frequently discussed petrous and rarely mentioned cavernous branches of the MMA are usually very small and originate immediately after the MMA emerges from the foramen spinosum (figure 8). Sometimes, the petrosquamosal branch is mislabeled as petrous, as the former projects over the petrous bone in lateral views. Frontal projections are very helpful, as the MMA tends to turn laterally inside the skull before giving off the petrosquamosal branch, whereas the more proximal, small petrous branch projects superiorly (figures 2 and 8). On lateral DSA views, the petrous branch is frequently obscured by very dense petrous bone and the superimposed petrosquamosal branch; however, it is better seen on good quality DYNA and rotational angiographic acquisitions. Supply to the facial and petrosal nerves can also arise from the accessory meningeal artery (figure 9) or the stylomastoid branch (occipital artery or posterior auricular artery origin), or a combination thereof.

The cavernous branch of the MMA, projecting medially on frontal views, anastomoses with the posterior branch of the inferolateral trunk, thus making it both a potentially dangerous EC-IC anastomosis and a possible source of supply to Meckel’s (trigeminal) cave and its nerves (figure 8). 39

The safest policy is not to embolize from a position proximal or near the foramen spinosum, both for the sake of these branches and the subsequently discussed orbital anastomoses. If proximal embolization is necessary (and no orbital collaterals are
rarer. The AICA—meningeal artery connections vary from territory to territory over another, with extremes being progressively more common: the recurrent meningeal dural supply proportionally reduces or eliminates the territory available for non-opthalmic routes of embolization, possibly accounting for some procedural failures (figure 5). The 100–300 μm primary anastomotic arteries may be large enough to allow at least partial embolization of

Figure 9 (A–I) Importance of anatomy and technique in the evaluation of the meningeal circulation. (A) Left frontal subdural collection. (B) Global external VR view showing separate origins of the frontoparietal (anterior, white arrows) and petrosquamosal (posterior) dural branches from the internal maxillary artery. The frontal branch ‘arises’ from the accessory meningeal artery (AMA). (C) Middle meningeal artery (MMA) injection shows supply to the posterior branch. (D, E) Proximal AMA injection demonstrating supply to the frontal dura; no apparent orbital communication. (F) DYNAX CT axial reconstructions of the same proximal injection showing supply of the petrosal and facial nerve arterial arcade (white arrow). Notice the catheter in the foramen ovale (black arrow), thus a too proximal embolization risks CN V injury. (G, H) A more distal injection under ‘flow arrest’ conditions now shows previously not visualized connection with the ophthalmic artery (black arrow) via the artery of the sphenoid ridge (white arrow). (I) DYNAX CT axial reconstructions of same injection.

Variant MMA origins

We emphasize a global approach to understanding the spectrum of MMA variations, particularly as they involve the ophthalmic artery, shown in figure 10. Any two interconnected vascular territories show variable balanced, partial, or complete dominance of one territory over another, with extremes being progressively rarer. The AICA-PICA and PCOM-P1 relationships are common examples. Middle meningeal–ophthalmic relationships follow the same principle.

A relatively common variant, at ~0.5% incidence in the literature, but about 2% in our experience, is recurrent meningeal origin from the ophthalmic artery. As mentioned above, there is a spectrum: the recurrent branch most often supplies only part of the frontal dura (figure 10D–F), less commonly a larger frontoparietal portion (figure 5), or rarely the entire MMA territory (figure 10A–C). We have not observed any correlation between the presence of a recurrent meningeal variant and cases of cSDH presenting for MMA embolization. Less common origins of the MMA are also well described in the literature (figure 11).

Figure 10 (A–O) Meningo–ophthalmic relationships, emphasizing the ‘spectrum’ principle. Balanced typical adult pattern is the most common, followed by partial annexations of either ophthalmic territory by the middle meningeal artery (MMA) (recurrent meningeal frontal branch only) or vice versa (meningo-lacrimal variant), followed by the least common complete annexations (meningo-ophthalmic and complete recurrent meningeal variants). (A–D) Complete middle meningeal origin from the ophthalmic artery (white arrows, (A, B)). No MMA is seen from the external injection (C). (D–F) Ophthalmic origin supply of frontal dural branch only (arrows (D, E), best visible in parenchymal phase of brain injection), with some part of the middle meningeal supply still arising from usual IMAX location (arrows (F)). (G–I) Typical adult pattern. (J–L) Classic meningo-lacrimal variant. The lacrimal branch characteristically projects over the superolateral orbit in frontal view (oval (K)), and extends anteriorly on the lateral view (oval (L)). (M–P) Meningo-ophthalmic variant—essentially complete supply of orbit via the MMA, with no visible ophthalmic artery on ICA injection (M). If only common and external injections are done prior to MMA EMBO, it is best to look for a prominent vessel overlying the orbit, with medial extension, on frontal view (arrow (N)) and choroidal blush on the lateral view (arrow (O)).

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Figure 11 (A–H) Rare but still useful variants to know. (A–D) Pharyngostapedial variant—early embryonic origin of the middle meningeal artery (MMA) from the stapedial artery (petrous segment origin, arrows (A, B, C)) with a highly characteristic course between the two crura of the stapes and over the cochlear promontory (D). Most patients have no pulsatile tinnitus or other clinical issue. (E–H) Basilar artery origin of the MMA—in fact this is a trigeminal artery to the MMA connection, as evidenced by its course alongside the trigeminal nerve on volume rendered (G) and fused DYNAX CT and T1 axial MR images (H).
Figure 12  (A–H) Subdural collection in the setting of ischemia related autosynangiosis. (A, B) Predominantly hypodense subdural collection in a patient with remote right middle cerebral artery (MCA) infarct. (C, D) Two months after images in (A, B), the overall collection has shrunk but there is a new inferior frontal component. (E, F) Early arterial (E) and venous phase (F) images of right internal carotid artery injection, demonstrating chronic MCA occlusion, and a region of relative hypoperfusion in the right mid-frontal convexity. (G, H) Lateral and frontal projection ECA images show an autosynangiosis between the frontal branch of middle meningeal artery (MMA) and mid-frontal superior division MCA territory, matching the hypoperfusion area seen in (F). Thus non-targeted MMA embolization in this case would result in a right frontal infarct. Case courtesy of Dr David Gordon, NYULMC.

recurrent meningeal territory from adjacent vascular beds. This can be achieved by employing a ‘wedged’ microcatheter position to hydraulically push small particles or liquid embolic agents through the anastomoses.

MMA–orbital anastomoses

Our training program emphasizes identifying the ophthalmic artery in every angiogram. Whenever meningeal vessels supplying a lesion require embolization, there is potential for an ophthalmic complication, be it a subdural collection or torcular fistula.

The classic descriptions are of meningolacrimal and meningophthalmic variants. In the former, more common disposition (~5%), the MMA supply is limited to lateral orbital contents, entering the orbit via the foramen of Hyrtl (figures 10J–L and 11).\textsuperscript{42} In the latter, rarer variant (<1%), MMA supplies the entire orbit (figure 10M–O). In reality, these variants are not binary entities, but again part of a spectrum that includes a number of ophthalmic/middle meningeal anastomoses, usually linked by the sphenoid branch. This strategically positioned artery, like a railroad line, rides the sphenoid ridge, linking the frontotemporal territory, and therefore less common, is with the ophthalmic branch, forming the meningophthalmic variant. Finally, the least common disposition is the last stop on the sphenoid line, where a transfer to the tentorial arcade railroad takes the rare traveler beyond the ophthalmic anastomosis all the way to the cavernous sinus, into an area usually supplied by the inferolateral trunk (figure 8). Inadvertent embolization in this variant, as in the case of the aforementioned cavernous branch, risks both cranial nerve injury and penetration into the ICA.

Visualization of these connections is a complex function of their developmentally determined and physiologically modified size, flow direction, catheter position, injection pressure, manipulation related (and therefore dynamic) vasospasm, resistance of overall vascular bed, and its change during embolization, etc (figure 9). Visualization may be further hampered by patient movement. Pragmatically, the possibility of MMA–ophthalmic connections should always be kept in mind, even if it is not readily appreciated during non-selective angiography.

Dural–pial autosynangiosis

Autosynangiosis refers to the parasitization of the extracranial dural supply by the subjacent pial surface\textsuperscript{44} and may be observed in settings of chronic cerebral ischemia or arteriovenous shunts. The presence of a fluid collection barrier between the dura and pia might argue against the possibility of autosynangiosis, however, we did come across an example (figure 12). Appreciation of the specific vascular anatomy should be noted, and care must be taken to avoid non-target embolization in these unique cases.\textsuperscript{45}

Technical aspects of embolization

Many approaches have been described. Exposure to preoperative tumor embolization varies widely in fellowships, and MMA embolization is likely patterned after these protocols. Whether, and when, ongoing randomized studies will help establish its

### Table 1  Summary of embolic agents\textsuperscript{21 47–51}

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<tr>
<th>Embolic agent</th>
<th>Advantages</th>
<th>Disadvantages</th>
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<tr>
<td>Particles</td>
<td>Consistent penetration</td>
<td>Requires delivery and visualization medium (typically contrast and a normal saline)</td>
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<td>Multiple size options</td>
<td>Delayed recanalization may occur</td>
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<td></td>
<td>Inexpensive</td>
<td>May require increased fluoroscopy and procedure times compared with nBCA</td>
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<td></td>
<td>Injection not painful</td>
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<td></td>
<td>Aspherical type (contour PVA)</td>
<td>Possibly induces less necrosis then spherical ones</td>
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<th>Liquid agents</th>
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<tr>
<td>TRUFILL n-BCA</td>
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<tr>
<td>Strategic distal penetration is possible</td>
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<tr>
<td>Short working time can be an advantage</td>
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<td>Possible catheter retention</td>
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<th>Onyx</th>
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<tr>
<td>Permanent occlusion</td>
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<tr>
<td>Longer working times</td>
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<tr>
<td>Strategic distal penetration is possible</td>
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<tr>
<td>General excellent radiopacity</td>
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<tr>
<td>Injection may be painful</td>
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<td>Possible catheter retention</td>
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<th>Coils</th>
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<td>Pushable fibercoils</td>
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<td>Highly thrombogenic</td>
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<th>Detachable</th>
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<tr>
<td>Retrievable</td>
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<td>Compatible with 0.017 type delivery microcatheters</td>
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MMA, middle meningeal artery; n-BCA, n-butyl cyanoacrylate.
into some areas and not as much into others. While we favor small particle size (45–150 µm) to maximize penetration, many operators prefer larger sizes to minimize the probability of non-target embolization. From an anatomical perspective, anything larger than 300 µm may not effectively penetrate beyond the main trunks, and in our opinion is suboptimal. At the end, we prefer to seal the trunk with coils. Pushable fiber coils require a larger diameter (0.021 inch) delivery microcatheter but are an order of magnitude cheaper than detachable coils, and highly effective.\textsuperscript{46, 47}

When orbital connections are demonstrated, we prefer selective catheterization of the frontal branch distal to the orbital supply (figure 13A–D). Anything above the orbital roof is likely safe. If there is still flow around the microcatheter, particles can be used (minimizing reflux), followed by coil occlusion. If there is a ‘wedge’ or ‘flow control’ position, we favor liquid embolic agents. The transient plug created by this technique allows the penetration of liquid embolic with minimal or no reflux and increases the ease of microcatheter removal after injection. To further minimize reflux, a dual lumen balloon microcatheter is an excellent choice.

**CONCLUSIONS**

Anatomical considerations support the rationale for MMA embolization in cSDH. The questions of efficacy and treatment durability will hopefully be answered by randomized controlled trials in the near future. If MMA embolization is confirmed to be an effective overall strategy, we must also investigate why it sometimes fails. The optimal embolic agent(s) remain unknown, and techniques vary widely, however anatomy does guide choices in individual patients. From a cost perspective, particles with or without pushable coils are clear winners in most contexts. Imaging advances will continue to enhance our understanding of the dural vasculature, and with that future angiographic interventions.

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**Figure 13** (A–F) Strategic use of liquid embolic n-butyl cyanoacrylate (nBCA) in the setting of orbital anastomosis. (A, B) Middle meningeal artery (MMA) angiography with a large meningoacral variant (arrows, also see figure 10J–L) contributing to the supply of the superior orbit and cutaneous tissues. Note also a relatively large petrous branch (arrowheads). (C, D) DSA of frontal branch distal to the origin of the meningoacral artery using a Scepter C balloon microcatheter. The balloon is inflated to eliminate reflux. Note penetration of contrast into the arterial arcade of the superior sagittal sinus (broken arrows) and falx cerebri (arrowhead). Also present is a frequently seen dural balloon is inflated to eliminate reflux. Note penetration of contrast into the arterial arcade of the superior sagittal sinus. (A–F) Strategic use of liquid embolic n-4

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