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THEORETICAL CONSIDERATIONS

Anatomy and development of the meninges: implications for subdural collections and CSF circulation

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Abstract The dura is traditionally viewed as a supportive fibrous covering of the brain containing the dural venous sinuses but otherwise devoid of vessels and lacking any specific function. However, review of the embryology and anatomy reveals the dura to be a complex, vascularized and innervated structure, not a simple fibrous covering. The dura contains an inner vascular plexus that is larger in the infant than in the adult, and this plexus likely plays a role in CSF absorption. This role could be particularly important in the infant whose arachnoid granulations are not completely developed. Although subdural hemorrhage is frequently traumatic, there are nontraumatic conditions associated with subdural hemorrhage, and the inner dural plexus is a likely source of bleeding in these nontraumatic circumstances. This review outlines the development and age-specific vascularity of the dura and offers an alternative perspective on the role of the dura in homeostasis of the central nervous system.

Keywords Subdural hemorrhage · Bridging veins · Embryology · Anatomy

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Introduction

Infantile subdural hemorrhage (SDH) is often traumatic, and ruptured bridging veins are thought to be the source of subdural blood in traumatic conditions. However, infantile SDH occurs in a number of conditions in which there is no evidence of mechanical trauma. SDH without trauma has been reported in benign extracerebral fluid collections, a condition characterized by macrocephaly and increased cerebrospinal fluid (CSF) in the subarachnoid space as well as in glutaric aciduria type 1, a metabolic disease associated with macrocephaly and large extraaxial CSF collections [1–7]. Nontraumatic subdural collections are also reported in states of decreased CSF volume, most notably in spontaneous intracranial hypotension with CSF leak, and following CSF shunting procedures [8, 9]. In addition, nontraumatic subdural collections are reported with hyponatremic dehydration, congenital and vascular malformations, coagulation disorders, dural venous thrombosis, meningitis, and other conditions [2, 10–12]. The diverse presentations of nontraumatic subdural collections challenge the hypothesis that tearing of the bridging veins is the primary cause of such collections. This review outlines the embryology, anatomy and function of the intradural venous plexus and offers an alternative explanation for subdural bleeding in the infant.

Embryology and anatomy of the dura–arachnoid interface

The dura and arachnoid are easily separated, as recognized by surgeons and pathologists who regularly lift the dura from the arachnoid with great ease. With mechanical separation of the two layers, an apparent space is created.

However, this space is an artefact of cleavage through a tissue plane [13–19]. The embryology of the dura makes clear that no such preformed space exists. Meningeal mesenchyme derived from neural crest cells initially forms a cellular network between the brain and the skin that is without discriminating features [20–22]. This pluripotent precursor is traditionally referred to as the meninx primitiva. The meninx primitiva subdivides into two layers, the inner endomeninx (which differentiates into the pia-arachnoid), and an outer ectomeninx (which forms the dura and bones of the neurocranium) [22]. The dura and neurocranium remain in close apposition and share a physiologic relationship that persists in postnatal life [20, 23]. The outer layer of the dura forms the inner periosteum of the skull and the inner layer forms the dural folds (falx and tentorium) and contains the dural sinuses.

At all times during development, the dura and arachnoid remain attached: there is no anatomic subdural space comparable to other embryonic cavities, such as the pleural and peritoneal cavities. But although there is no preformed space, there is a distinct soft-tissue layer at the dura–arachnoid interface that is easily disrupted. This morphologically distinct junctional soft-tissue layer is characterized by flattened fibroblasts with sparse intercellular junctions, no extracellular collagen, and prominent extracellular spaces [13–19]. The cells in this interface layer have been variably termed subdural mesothelium, neurothelium, inner dural cell layer and subdural cells. However, the most frequently used descriptive term is "dural border cell layer," coined by Nabeshima et al. [15]. The accumulation of blood in this unique layer of cells confers the typically crescentic appearance of SDH on imaging examination and is responsible for the neurosurgical appearance of a clot beneath the fibrous dura but superficial to the glistening surface of the external limiting arachnoid membrane. The precise anatomic description of a subdural collection is an accumulation of fluid or blood within the disrupted layers of the dural border cell layer (Fig. 1).

In summary, the dura and arachnoid arise from a common origin and through differentiation and condensation the final structure is one of continuity. In an attempt to avoid futile arguments over language, Orlin et al. [19] suggested modification of the term "subdural space" to "subdural compartment" to reflect the presence of a morphologically distinct layer of cells at the dura-arachnoid interface.

Embryology and anatomy of the bridging veins

As the meninx primitiva differentiates into contiguous but distinct layers, the venous drainage is also differentiating into discrete systems. Separation of the venous drainage of the brain from the dural venous drainage gives rise to the specific anatomy of the bridging veins. Initially, there is a primitive endothelial plexus of venous connections between

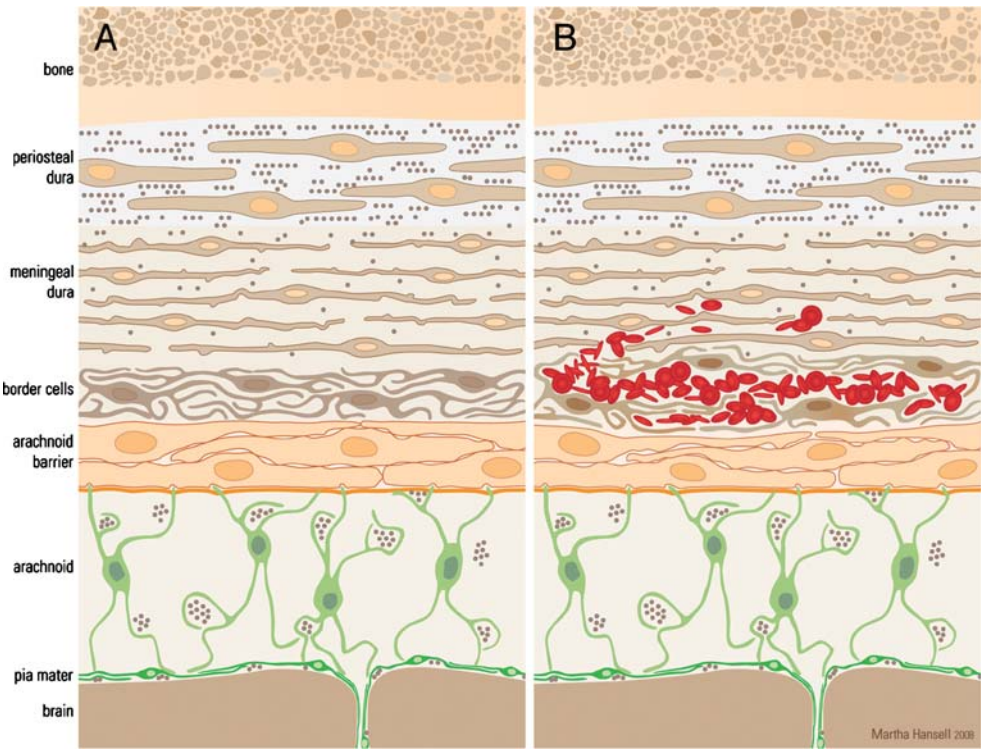

Fig. 1 Subdural collection. **a** Dural border cell layer is composed of loosely adherent cells, no extracellular collagen, and enlarged extracellular spaces. **b** SDH represents a collection of blood disrupting the dural border cell layer

the dural and pial layers. However, early in the first trimester, the continuous net of primitive endothelial channels draining the brain and those draining the dura undergo a venous cleavage, such that most of the numerous primary venous anastomoses between the pia and dura resorb [24, 25]. The few remaining venous anastomoses enlarge and become the definitive bridging veins. The veins draining the majority of the dorsal convexity of each cerebral hemisphere collect into the superior cerebral veins. These superficial veins coalesce to form between 10 and 18 large bridging veins that eventually penetrate the dura–arachnoid interface layer and travel for a variable distance within the dura before entering the superior sagittal sinus [26–28].

Embryology and anatomy of the dural veins

Although the bridging veins form in the first trimester through the process of venous cleavage, the dural venous structures to which the bridging veins drain undergo modifications throughout gestation. The major dural venous sinuses do not attain their adult configuration until some months after birth. The early fetal dural venous connections are plexiform and are in a constant state of change. The adjustment in this primitive dural plexus is necessary because of the dramatic intrauterine growth of the cerebral hemispheres. A large part of this adjustment is made possible by the spontaneous migration of the principle dural veins, and for this a venous plexus is essential [24]. Padget [25] eloquently conveys the inevitability of such a sequence of embryonic events: "such alterations are not foreordained by the adult configuration but are determined by adjacent structures. The embryonic channels should not be thought of as busily engaged in building mature vessels, but as carrying on their functional activity in the best manner possible for the moment with regard to the available space and amount of work to be done."

One of the principle elements of the embryology of the intracranial venous system is that the veins intrinsic to the brain and those intrinsic to the dura separate early in development. The early cleavage of these two venous territories is an essential component of the establishment of the blood–brain barrier. The process of venous cleavage reduces the number of pial-to-dural connections, thereby reducing the points at which veins intrinsic to the brain communicate with the systemic circulation.

As large-caliber conduits from the brain to the systemic circulation, the bridging veins are neither small nor fragile. Biomechanical data confirm that these veins rupture only under considerable force [29, 30]. The bridging veins are therefore improbable sources for SDH occurring without trauma.

Alternatives to bridging vein rupture

A more thorough understanding of the vascularity of the dura reveals a potential alternative source of bleeding in nontraumatic conditions. Although not routinely discussed in the SDH literature, dural vascularization is more abundant and complex than is generally presumed [31–36]. The dural vascularity is well described in the historical literature. Key and Retzius [35] in 1875 carefully noted the presence of both a superficial capillary network close to the bone and a fine capillary network located along the inner portion of the dura. As early as 1936, Hannah [36] speculated that it was the inner dural capillary layer that ruptured to produce SDH.

More recently, Kerber and Newton [32] used injection techniques, radiography, and microdissection to provide a complete description of the dural vascularity. They also describe a few peculiarities that are important when the function of the dura is considered [32]. The main blood supply to the dura comes from the meningeal arteries. These arise in the primitive ectomeninx of the embryo specifically to nourish the developing skull [25]. Hence, the meningeal artery and accompanying meningeal veins are superficially located in the outer aspect of the dura. The main meningeal arteries give rise to a rich anastomotic layer of vessels that are also superficial in location. The primary anastomotic vessels give rise to four smaller arterial structures: (1) the arteries to the skull, (2) the secondary anastomotic arteries, (3) the arteriovenous shunts, and (4) the penetrating arteries. It is the penetrating vessels that are the most relevant to the discussion of SDH. The penetrating vessels leave the superficial dural surface and extend to within 5 to 15 μm of the dural border cell layer. The penetrating arterioles end "in an unexpected and extremely rich capillary network. The capillaries are present throughout the dura, including the falx and tentorium, and are extremely rich parasagittally, where they may form several layers" [32]. It is this unexpectedly rich vascular plexus that borders the subdural compartment (Fig. 2).

The dural venules, unlike vessels in the brain, are permeable and contain pores visible by electron microscopy [37–39]. Moreover, the dural vasculature is well innervated by branches of the trigeminal nerve (Fig. 3), and postcapillary venules leak with neurogenic or chemical stimulation [38–41]. These "leaky" vessels that lie within the dura near the dura–arachnoid interface are strong candidates for the source of bleeding in nontraumatic conditions (Fig. 4).

Dural vascularity in the infant

The dural vascularity in the adult is more abundant than is generally assumed. In the infant, the vascularity of the dura

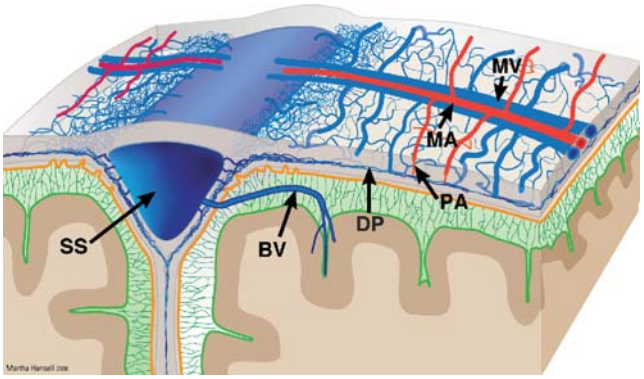

Fig. 2 The meningeal arteries and veins are superficially located, while the dural venous plexus (*DP*) is located within the inner portion of the dura. The dural plexus is particularly dense parasagittally, where it connects to the parasagittal sinus independently of the cortical bridging veins (*BV* bridging vein, *MA* meningeal artery, *MV* meningeal vein, *PA* penetrating arteriole extending to inner dural plexus, *SS* superior sagittal sinus)

is even more pronounced, largely because of the plexiform nature of the dural venous connections. Padget [25] indicates that the plexiform nature of the early dural venous channels is required for intrauterine brain growth. If a dural venous plexus is needed for growth of the brain, then it is not surprising that the venous plexus is larger in infants than in adults. Streeter [24] and Padget [25] both report that at birth the venous dural connections differ from the adult configuration. Streeter [24], in particular, writes of the prolonged adjustment of the intradural venous connections of the falx and tentorium. The structure of the torcula (confluens sinuum), frequently web-like even in adults, is a remnant of the plexiform nature of the fetal dural veins. Kaplan et al. [42] note that the veins of the dorsal aspect of the falx and the venous lakes of the tentorium are all readily visualized in late-term fetuses and infants. Gooding et al. [43] report that opacification of the tentorial intradural venous structures after cerebral angiography is present in 20% of children and only 1% of adults. Browder et al. [44] specifically evaluated the venous lakes in the suboccipital dura mater and falx cerebelli in infants and found that the intradural venous lakes are larger in infants, and gradually diminish in adolescents and adults.

Although the larger intradural venous plexus in the infant might be a remnant of the tremendous intrauterine growth that has occurred, the function cannot be limited to the facilitation of growth, as the venous plexus is a persistent feature of the adult dura. Le Gros Clark [45] in 1920 emphasized that the lateral lacunae should not be viewed as simply diverticula of the sagittal sinus but as a complicated parasagittal "meshwork of veins." As noted by Kerber and Newton [32], the parasagittal venous plexus is particularly dense. Rowbotham and Little [33] describe the parasagittal plexus as so profuse as to give it a "primitive air." The authors go on to suggest that the plexus is so dense and so very far in excess of the metabolic needs of the fibrous and elastic tissue that it must have some other functional significance.

Understanding the physiologic role of this plexus could provide insight into the nontraumatic conditions that result in bleeding from the plexus. Although the function of the plexus is not fully understood, it is highly likely that it plays a role in the movement of CSF from the subarachnoid compartment into the venous and lymphatic system [31, 37, 46]. CSF movement across the arachnoid barrier layer is traditionally thought to occur via arachnoid granulations. Therefore, it is appropriate to review the development of the arachnoid granulations in the infant.

The arachnoid granulations and their relationship to the intradural venous plexus

The arachnoid granulations are traditionally considered to be one of the mechanisms by which CSF moves from the subarachnoid space into the systemic circulation. While they may play a dominant role in the adult, arachnoid granulations are not fully formed in the infant. Le Gros Clark [45] describes the evolution in the appearance of the arachnoid villi from microscopic structures at birth to the visible protrusions of arachnoid granulations: "At birth they

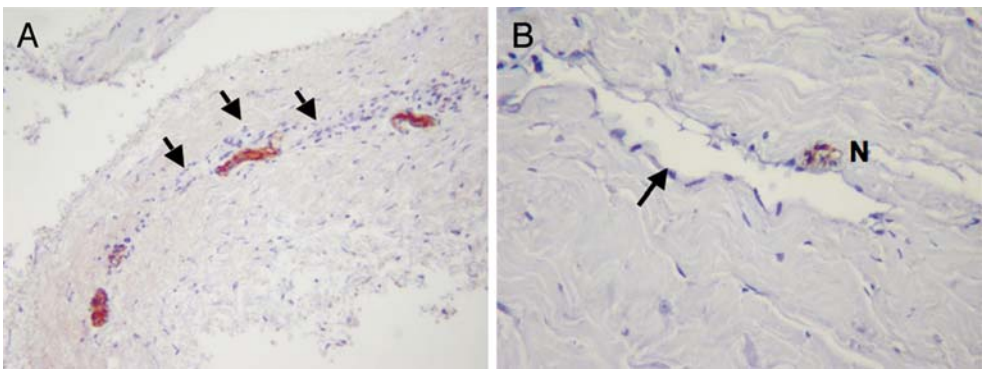

Fig. 3 Dural vasculature. **a** Nerve fibers (*stained brown*) among vessels of the inner dural plexus (*arrows*). **b** High-power view demonstrates nerve fibers extending to the endothelium of a vessel of the dural plexus (*N* nerve fiber)

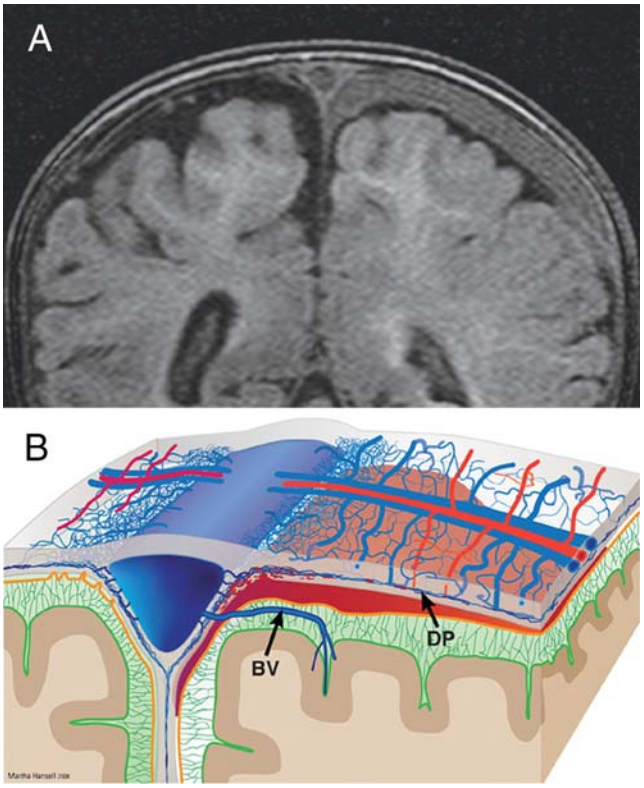

Fig. 4 Left subdural compartment hemorrhage. **a** T1-W coronal MR image shows left subdural compartment hemorrhage. **b** Graphic representation of hemorrhage originating from the inner dural plexus (*BV* bridging vein, *DP* dural plexus)

are imperceptible even on examination with a hand lens. At the age of six months they are still invisible, but by 18 months they are quite obvious on close inspection. They appear in the first instance in the regions where the parieto-occipital and central veins open into the sagittal sinus. From there they spread forwards and backwards along the superior margin of the cerebral hemispheres, and at the age of three they are disseminated over a considerable area." A recent study quantifying the area and distribution of the arachnoid granulations confirms the observations of Le Gros Clark: the arachnoid granulations occur with greatest frequency in the region of the parasagittal venous plexus [46].

The intimate relationship of the arachnoid granulations and the parasagittal plexus has been demonstrated by Fox et al. [37] and more recently by Han et al. [28]. Using a resin injection technique, these authors carefully delineated the relationship between the parasagittal network of venous channels, the arachnoid villi, the superior sagittal sinus, and the bridging veins. The arachnoid villi are interdigitated between the trabeculae of the dura that occur at the site of the intradural venous plexus (Fig. 5). The bridging veins do not directly connect to the intradural venous plexus [28, 37]. Instead (as expected from the embryology of venous cleavage), the bridging veins independently enter the sinus beneath the parasagittal venous plexus (Fig. 6).

Review of the anatomy demonstrates that the arachnoid villi and the intradural venous plexus are intimately related, both anatomically and developmentally, and the two must be studied in conjunction [45]. Their relationship might have particular relevance given the known association of SDH in states of both increased CSF volume (benign extracerebral collections of infancy) and decreased CSF volume (spontaneous intracranial hypotension).

CSF transport and the anatomy of the dura

By its anatomic proximity and intimate association with the arachnoid granulations, the parasagittal venous plexus presumably plays a role in CSF absorption. How the CSF moves from the subarachnoid space through the arachnoid barrier cell layer and into the interstitium of the dura is not clear, particularly in the absence of arachnoid granulations. However, multiple experiments indicate that movement along nerve roots and exiting vessels plays a role [47–50]. Research in neonatal animals shows that tracers injected into the subarachnoid space are transported into the dura, filling the interstitium of the dura in a parasagittal location [49]. Interestingly, fluid injected into the interstitium of the dura has been shown to collect in the parasagittal venous

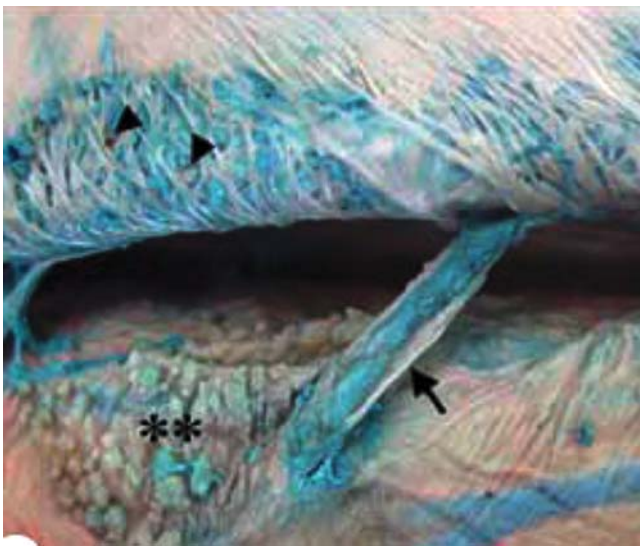

Fig. 5 With the dura reflected off the brain, the relationship of the arachnoid granulations (*asterisks*) and the venous plexus (filled with blue latex, *arrowheads*) is well demonstrated. Note bridging vein, which has been reflected back from the dura (*arrow*) (reprinted with permission from Springer Science + Business Media: Han H, Tao W, Zhang M. The dural entrance of cerebral bridging veins into the superior sagittal sinus: an anatomical comparison between cadavers and digital subtraction angiography. Neuroradiology 2007;49:169–175, Fig. 2C)

Fig. 6 Vascular resin cast of fetal cadaver demonstrates the parasagittal plexus in communication with the superior sagittal sinus. The endothelial channels of the plexus communicate with the sinus independently of the bridging veins. **a** Superior view shows the intricate parasagittal plexus and its relationship to the sagittal sinus. **b** Inferior view shows bridging veins communicating with the sinus beneath the parasagittal plexus (*BV* bridging vein, *DP* parasagittal dural plexus) (used with permission from Ming Zhang, University of Otago, New Zealand)

plexus and to subsequently fill the venous sinuses. This forward filling of the parasagittal venous plexus indicates that the interstitium of the dura and the intradural venous plexus communicate with each other [31, 35, 37]. How they communicate is not known, as the human dura is thought to lack lymphatics. However, a closer inspection of the dura reveals a system of interstitial channels that are intriguing candidates for transport of CSF.

Careful examination of the dura shows that though many of the intradural channels are lined by endothelium, some channels within the dura are not lined by endothelium. They appear microscopically as rounded spaces between the fibers of the dura, in some areas intimately associated with the endothelial lined channels that drain into the dural sinuses (Fig. 7). Early authors referred to these unlined channels as "juice channels" [31], indicating a role in fluid transport and/or resorption. If the interstitium of the dura is part of the pathway from the subarachnoid space to the dural venous system, then it is not unreasonable to speculate that the amorphous material filling the extracellular spaces of the dural border cell layer represents CSF. Such a hypothesis is supported by ultrastructural CSF tracer studies in which tracer injected into the subarachnoid space is followed into the dura. In these studies, tracer is found within highly vesiculated dural border cells and within the widened extracellular spaces surrounding the dural border cells [47]. Thus, some of the CSF transported across the arachnoid barrier layer might collect in the dural border layer, and from there move into the deeper layers of the interstitium and into the unlined channels. From the unlined channels of the interstitium of the dura, the CSF could then be transported or absorbed into the endothelial lined parasagittal plexus.

CSF and subdural collections

A recent study of subdural collections has shown that CSF-specific protein is present in 100% of subdural hygromas and 93% of patients with chronic SDH [51]. CSF tracer studies performed in infants with subdural collections show that radiopharmaceutical injected into the subarachnoid space moves into the subdural compartment [52]. CSF accumulation within subdural collections is hypothesized to

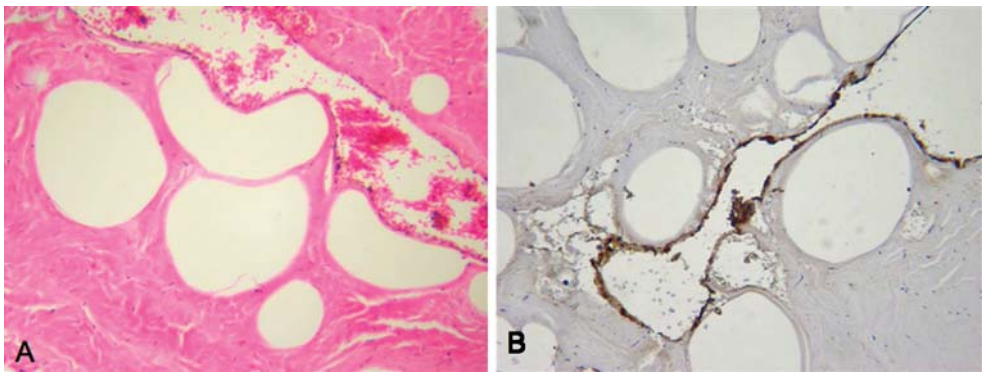

Fig. 7 Pathologic section of dura. **a** Sections through meningeal dura show well-circumscribed, non-endothelial-lined channels that are intimately associated with the thin-walled meningeal venules and venous lakes. **b** The endothelium of the venous plexus stained with factor VIII, surrounded by unlined channels

be the result of a tear in the arachnoid membrane, although other mechanisms have also been put forward [51]. It is difficult to invoke a tear in the arachnoid as a primary event in nontraumatic subdural collections, much as it is difficult to envision how a primary tear would result in fluid preferentially dissecting from the subarachnoid space into a soft-tissue compartment. A ball-valve mechanism has been proposed as an explanation [51], though there is no proof that such occurs. If hemorrhage is the primary event that produces a subdural collection, a secondary tear in the arachnoid membrane would result in disruption of the CSF–blood barrier and allow flow of blood and plasma from the subdural compartment into the subarachnoid space. Such a breakdown of the CSF–blood barrier is not supported by the imaging findings in which there is no evidence of subarachnoid blood (nor evidence of changes in MRI signal intensity in the subarachnoid compartment to suggest mixing of plasma).

An alternative explanation is that CSF is present in small amounts at all times in the dura. Any alteration of the CSF absorption pathway from the dura into the dural sinuses that produces an accumulation of CSF within the dura could produce imaging findings of a nonhemorrhagic subdural collection (subdural hygroma). In cases of hemorrhage, bleeding into the layers of the dura could disrupt the normal mechanisms by which CSF is transported and result in accumulation of CSF within the subdural compartment. The variability of the amount of CSF within subdural collections could be dependent on a multitude of factors, including the age of the patient, the size of the hemorrhage, and the location of the hemorrhage with regard to the developing mechanisms of CSF transport.

Transport of CSF into the dura and into the parasagittal plexus would result in mixing of CSF with blood in the parasagittal plexus. Such a phenomenon would provide an explanation for the pathologic observation that the parasagittal venous plexus is always devoid of clot, even when there is extensive clot in the adjacent superior sagittal sinus [27, 37]. The presence of a CSF transport pathway within the infant dura would not only explain the common finding of CSF within subdural collections, but it would also explain why the imaging finding of a fluid-fluid level within a collection is not always predictive of the age of the hemorrhage [53]. Moreover, a CSF-absorbing pathway that is different in the infant compared to the older child might help explain why infant SDH most often presents as a thin, nonclotted film, rather than the thicker, clotted SDH more typically seen in older children and adults [54–57].

In summary, CSF might move from the subarachnoid space into the interstitial spaces of the dura through a variety of mechanisms (along exiting nerves, exiting vessels, by transcellular transport and, in the older child, through arachnoid granulations). Once in the dura, CSF could move from the interstitium into the dural venous plexus and subsequently into the dural sinuses. Therefore, the frequent finding of CSF in subdural collections is not necessarily the result of a tear in the arachnoid. Instead, CSF within subdural collections could be a reflection of the physiology of the dura.

Questions not answered by the bridging vein rupture hypothesis

Traditionally, SDH has been ascribed to rupture of the bridging veins as they cross from the subarachnoid space through the dural border cell layer. However, it is peculiar that 1- to 3-mm diameter bridging veins should rupture preferentially into an 8-μm-thick dural border cell layer rather than diffusely into the subarachnoid space: “What is unusual about SDH is the frequency with which hemorrhage occurs in the potential space” [58]. It would be an evolutionary oddity if the veins responsible for draining large portions of the cerebral cortex had a focal, inherent weakness in their wall. Yet this is the most frequently cited explanation with reference made to the work of Yamashima and Friede [59]. These authors describe a variable thickness of 10 to 600 μm for the walls of the veins traversing the subdural space as compared to the relatively uniform thickness between 50 and 200 μm for the walls of the veins in the subarachnoid space. They conclude that the subdural portions of the bridging veins are more fragile and more prone to rupture. However, their patient population was small (four adults; ages 53, 77, 77, and 85 years), and only frontal bridging veins were sampled. Three of the four patients died of diseases that are associated with increased central venous pressures. The chronic increased central venous pressure might have affected the morphology of the veins in their patients, as suggested by the authors’ comment: “Extracellular debris was common, being embedded in the thickened zones of basement membrane, presumably indicating degeneration and disintegration of smooth muscle cells” [59]. The small sample size in their study, along with the lack of significant differences in wall thickness between the intradural and subarachnoid portions of the veins, calls into question the conclusion that preferential rupture is a common event. Perhaps of more importance is that the findings in these elderly patients cannot be assumed to be representative of the infant.

Although bridging vein rupture has long been considered the source of SDH, rarely are torn bridging veins identified at autopsy [60, 61]. Craig [62] reported a 50% incidence of SDH in 126 neonates at post-mortem examination, but in only three of these could he confirm torn bridging veins. Cushing [63] noted: “In two of the cases I have examined I

have satisfied myself that such ruptures were present. A positive statement, however, cannot be given even for these cases, since the dissection and exposure, difficult enough under any circumstances … are the more so when they are obscured by extravasated blood." The preceding observation may be old, but no new method has replaced detailed observation and meticulous descriptions of findings at surgery and autopsy.

It is even more difficult for the pathologist because the bridging veins are readily torn by removal of the brain. Post-mortem injection of contrast medium into the sagittal sinus prior to brain removal has been advocated by some authors as a way to identify bridging vein rupture [64–66]. However, the technique suffers from artificially produced retrograde pressure to fill veins that could already contain post-mortem clot. In addition, post-mortem injection experiments must take into account the presence of the venous plexus. Retrograde injection will fill the valveless parasagittal plexus and produce imaging findings of contrast agent outside the dural sinuses and bridging veins. With enough retrograde pressure, extravasation of contrast agent from the plexus could occur and produce a subdural collection on cadaveric imaging studies.

All of the above arguments are not to say that bleeding from the bridging veins does not occur, but the site of hemorrhage might not be confined to the 8-μm-thick dural border cell layer. Instead, the site of hemorrhage could be from the portion of the vein traversing the fibrous dura, dissecting subsequently into the subdural compartment. In a recent morphologic study of the bridging veins, Vignes et al. [67] found a unique cuff of circular collagen fibers to be present surrounding the intradural bridging veins at their entrance into the sagittal sinus. A similar innervated sphincter has been described at the junction of the vein of Galen with the straight sinus [68]. The change in orientation of fibers could be responsible for the terminal dilatation of the intradural portion of the bridging veins just before their entrance into the sagittal sinus [27, 69]. Whether the terminal dilatation produces a structurally weaker wall prone to leaking is unknown. However, the distinctive orientation of fibers is intriguing, as the orientation likely serves a particular purpose. Vignes et al. [67] hypothesize that the helical orientation of collagen fibers and associated smooth muscle fibers act as a sphincter with a role in the regulation of intracranial venous pressure. Such a hypothesis is supported by preliminary work that demonstrates the terminal cuff is more pronounced in patients with increased intracranial pressure and produces vascular dilatation of the proximal vessel [69]. The presence of a sphincter at the junction of the bridging veins and the sagittal sinus is important information and indicates a structural and functional role in cerebral venous hemodynamics that is not fully understood.

Intradural blood versus subdural blood

The propensity for blood to collect in the subdural compartment is explained by the unique anatomy of the layers of the dura: blood in the interstitium of the dura, after it reaches enough volume, follows the path of least resistance and collects into the loosely adherent dural border cell layer. Such a mechanism has been demonstrated experimentally. Hannah [36] injected citrated blood into the outer layers of fresh dura and found that blood invariably finds its way toward the inner surface of the dura in "raised blebs." Likewise, Fox et al. [37] made superficial intradural injections with vinyl acetate and acrylic resin and not only filled the intradural venous channels but also produced a subdural resin collection. The anatomy of the dura–arachnoid interface brings forth a semantic issue that lies at the heart of this discussion: is the blood in an SDH intradural blood? Embryologically and anatomically, the answer is clear: the dural border cell layer is part of the dura. However, this answer merely addresses the location of the collection of blood. Therefore, a better question is, where did the blood originate before it collected into the avascular subdural compartment [19]? The answer is that disruption of any vessel (arteriole, capillary, venule or vein) within the substance of the fibrous dura could produce a subdural collection.

Distribution of intradural/subdural hemorrhage

Conceptualizing the subdural compartment as intrinsically part of the dura helps clarify some of the discrepancies in radiologic terminology and neuropathologic observation, particularly with regard to the location of tentorial and falcine hemorrhage. It is not uncommon to see falcine hemorrhage interpreted on imaging studies as "interhemispheric extraaxial hemorrhage" [58]. This is a visual description of the finding rather than a more rigorous account of the discrete anatomic location of the hemorrhage. Likewise, tentorial hemorrhage has been incorrectly characterized as subarachnoid on post-mortem CT scans when at pathology the blood was confined to the interstitium of the tentorium [70]. In part, this misconception arose because the falx and tentorium are generally, and incorrectly, considered avascular fibrous support membranes containing major dural sinuses but otherwise devoid of vessels. However, understanding the venous anatomy of the dural folds leads to a better understanding of the radiology. The intrafalcine venous network that drains into both the inferior and superior sagittal sinus is most pronounced posteriorly [24, 71]. Because of the unique embryology of the dural venous system, the falcine and tentorial venous channels are particularly prominent in the

infant [25, 42–44]. Hence, the radiologic finding of small falcine and tentorial hemorrhages can be readily explained as the imaging equivalent of the venous congestion and intradural hemorrhage so commonly seen in pathologic practice [61, 72] (Fig. 8).

Possible implications of the anatomy of dura

The thin-walled intradural vascular plexus is a likely source of hemorrhage in nontraumatic conditions. However, bleeding from the plexus is not limited to nontraumatic causes. Impact trauma (including nonaccidental impact) could easily disrupt the fragile venous mesh, with subsequent bleeding. Mechanical deformation of the dura at the sites of suture overlap during delivery producing disruption of the plexus would be a reasonable explanation of the common finding of SDH in otherwise asymptomatic newborns [73–76].

The relationship of CSF absorption to the dural plexus might be particularly important in the infant whose arachnoid granulations are not fully developed. CSF has been traditionally thought to represent a cushion for the brain, reducing tension on nerve roots and acting as a mechanical buffer. However, it is clear that the regulation of CSF flow is also vital to normal brain health. CSF is in communication with the interstitial compartment of the brain and plays a major role in normal brain homeostasis [77]. Normal fluid turnover is important not only for movement of nutrients and hormonal signaling but also in the elimination of catabolites and toxins [77, 78]. Infants whose brains are not fully myelinated have a proportionately greater volume of interstitial water, and they form fluid at a slower rate per mass of tissue than adults. These factors "lessen the capability of immature brain to eliminate unneeded catabolites" [78].

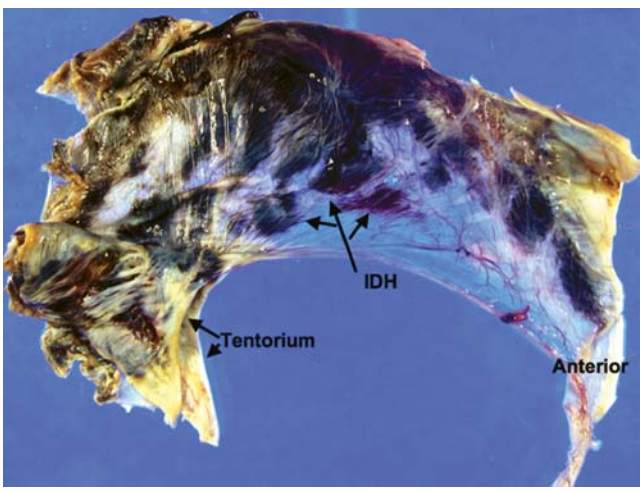

Fig. 8 Intrafalcine hemorrhage. Sagittal view of the falx and portions of the tentorium from a newborn who died 6 h after birth with severe hypoxic ischemic injury to the brain (*IDH* intradural hemorrhage) (reprinted with permission from Dr. Josephine Wyatt-Ashmead)

Bleeding from the intradural venous plexus could potentially affect normal CSF flow dynamics and significantly affect brain function.

States of decreased CSF volume and pressure are associated with considerable encephalopathy, including deep brain swelling, coma, and seizures [79–81]. Therefore any derangement of CSF balance is important to recognize and even more important to correct. The syndrome of spontaneous intracranial hypotension (spontaneous CSF leak) was largely unknown until the advent of MRI [82]. It is not a rare syndrome, and it might be under-diagnosed in adults [83, 84]. Spontaneous CSF leak has been reported in children [85–87]. The incidence of spontaneous CSF leak in infants is not known, as the primary symptom (orthostatic headache) cannot be ascertained. Additional imaging tools (gadolinium-enhanced MRI, radionuclide cisternography, myelography) have been found useful in the diagnosis of the condition in adults [82] and could potentially be used to investigate the syndrome in infants.

Conclusion

The precise anatomic definition of SDH is hemorrhage collecting into a specialized compartment of loosely arranged cells within the dura. Trauma remains a common cause of SDH, and in collaboration with others in the health-care team, a meticulous search for additional evidence of trauma should be made. At the same time, nontraumatic causes must be considered.

Although the origin of the blood could be any vessel within the dura, the extensive capillary and venous plexuses of the dura are the most reasonable candidates for the source of hemorrhage in nontraumatic conditions.

The plexus of veins within the dura undergoes considerable developmental change in the first year of life. The regression of the venous plexus parallels the development of the arachnoid granulations. Although the function of the venous plexus is not fully understood, it likely plays a role in CSF absorption and this role might be particularly important in infants with developing CSF transport pathways. The presence of an intradural CSF absorption pathway helps explain the high prevalence of CSF within both hemorrhagic and nonhemorrhagic subdural collections. Further study of the function of the plexus might yield clues to the etiology of nonhemorrhagic collections.

Review of the anatomy brings a new perspective to the function of the dura: it is clear that the dura is more than a fibrous covering for the brain. Instead, it is a complex, innervated and remarkably vascularized membrane whose

physiology is not completely understood. It is through renewed multidisciplinary collaboration with those who study the unique innervation and vascularization of the dura that we will begin to answer some of the questions surrounding its function. The answers to be gained from such collaboration might provide important insights into the etiology of infantile SDH, as well as furnish new paradigms for the treatment of subdural collections.

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