



## Subarachnoid Trabeculae: A Comprehensive Review of Their Embryology, Histology, Morphology, and Surgical Significance

Martin M. Mortazavi<sup>1,2</sup>, Syed A. Quadri<sup>1,2</sup>, Muhammad A. Khan<sup>1,2</sup>, Aaron Gustin<sup>3</sup>, Sajid S. Suriya<sup>1,2</sup>, Tania Hassanzadeh<sup>4</sup>, Kian M. Fahimdanesh<sup>5</sup>, Farzad H. Adl<sup>1,2</sup>, Salman A. Fard<sup>1,2</sup>, M. Asif Taqi<sup>1,2</sup>, Ian Armstrong<sup>1,2</sup>, Bryn A. Martin<sup>1,6</sup>, R. Shane Tubbs<sup>1,7</sup>

### Key words

- Arachnoid matter
- Lilliequist membrane
- Microsurgical procedures
- Subarachnoid trabeculae
- Subarachnoid trabecular membrane
- Trabecular cisterns

### Abbreviations and Acronyms

- CSDH:** Chronic subdural hematoma  
**CSF:** Cerebrospinal fluid  
**DBC:** Dural border cell  
**DL:** Diencephalic leaf  
**GAG:** Glycosaminoglycan  
**LM:** Lilliequist membrane  
**ML:** Mesencephalic leaf  
**PAC:** Pia-arachnoid complex  
**PPAS:** Potential pia-arachnoid space  
**SAH:** Subarachnoid hemorrhage  
**SAS:** Subarachnoid space  
**SAT:** Subarachnoid trabeculae  
**SEM:** Scanning electron microscopy  
**TEM:** Transmission electron microscopy

From the <sup>1</sup>National Skull Base Center, Thousand Oaks, California; <sup>2</sup>California Institute of Neuroscience, Thousand Oaks, California; <sup>3</sup>Advocate BroMenn Medical Center, Normal, Illinois; <sup>4</sup>University of Arizona College of Medicine, Tucson, Arizona; <sup>5</sup>University of California Irvine Medical Center, Irvine, California; <sup>6</sup>University of Idaho, Moscow, Idaho; and <sup>7</sup>Seattle Science Foundation, Seattle, Washington, USA

To whom correspondence should be addressed:  
 Martin M. Mortazavi, M.D.  
 [E-mail: [m\\_mortazavi@hotmail.com](mailto:m_mortazavi@hotmail.com)]

Supplementary digital content available online.

Citation: *World Neurosurg.* (2018) 111:279-290.

<https://doi.org/10.1016/j.wneu.2017.12.041>

Journal homepage: [www.WORLDNEUROSURGERY.org](http://www.WORLDNEUROSURGERY.org)

Available online: [www.sciencedirect.com](http://www.sciencedirect.com)

1878-8750/\$ - see front matter © 2017 Elsevier Inc. All rights reserved.

### INTRODUCTION

In the third century B.C., Herophilus, a Greek physician and the father of anatomy, first described the brain as being enclosed within the arachnoid membrane.<sup>1</sup> In the seventeenth century, Gerardus Blasius and Andreas Ottomar Goelicke referred to the arachnoid membrane as “*tertia cerebri meninge*” or the third cerebral meninge.<sup>2-8</sup>

■ **INTRODUCTION:** Brain is suspended in cerebrospinal fluid (CSF)-filled subarachnoid space by subarachnoid trabeculae (SAT), which are collagen-reinforced columns stretching between the arachnoid and pia maters. Much neuroanatomic research has been focused on the subarachnoid cisterns and arachnoid matter but reported data on the SAT are limited. This study provides a comprehensive review of subarachnoid trabeculae, including their embryology, histology, morphologic variations, and surgical significance.

■ **METHODS:** A literature search was conducted with no date restrictions in PubMed, Medline, EMBASE, Wiley Online Library, Cochrane, and Research Gate. Terms for the search included but were not limited to subarachnoid trabeculae, subarachnoid trabecular membrane, arachnoid mater, subarachnoid trabeculae embryology, subarachnoid trabeculae histology, and morphology. Articles with a high likelihood of bias, any study published in nonpopular journals (not indexed in PubMed or MEDLINE), and studies with conflicting data were excluded.

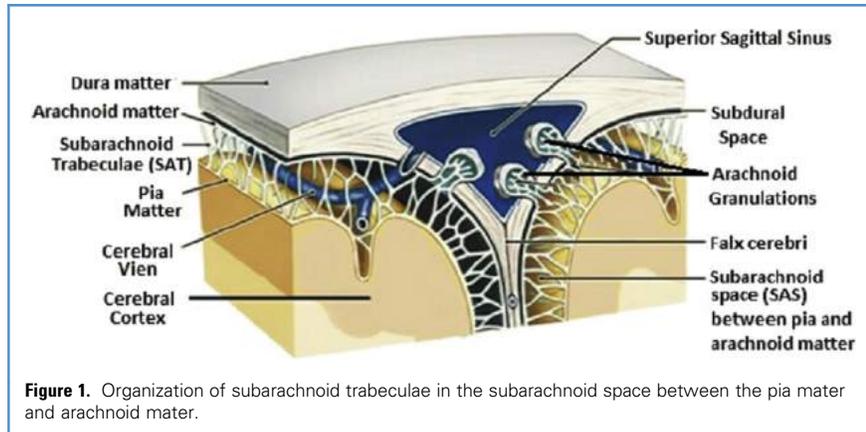
■ **RESULTS:** A total of 1113 articles were retrieved. Of these, 110 articles including 19 book chapters, 58 original articles, 31 review articles, and 2 case reports met our inclusion criteria.

■ **CONCLUSIONS:** SAT provide mechanical support to neurovascular structures through cell-to-cell interconnections and specific junctions between the pia and arachnoid maters. They vary widely in appearance and configuration among different parts of the brain. The complex network of SAT is inhomogeneous and mainly located in the vicinity of blood vessels. Microsurgical procedures should be performed with great care, and sharp rather than blunt trabecular dissection is recommended because of the close relationship to neurovascular structures. The significance of SAT for cerebrospinal fluid flow and hydrocephalus is to be determined.

The current name of arachnoid mater is attributed to Frederick Ruysch and his description of a spiderlike morphology in 1699.<sup>9</sup> It is a delicate avascular layer in direct contact with the dura and separated from the pia mater by the cerebrospinal fluid (CSF)-filled subarachnoid space, showing distinctive histology and pathology.<sup>1,10</sup>

During the late 1960s, Anderson and Hayreh along with others described subarachnoid trabeculae (SAT), which are sheets or columns of collagen-reinforced material stretching between the arachnoid and pia membranes<sup>11-13</sup> (Figure 1). The

delicate neural tissue of the brain is suspended within the CSF by buoyancy, in accordance with the Archimedes principle, and also mechanically stabilized by the SAT within the pia-arachnoid complex (PAC).<sup>14</sup> SAT constrain relative movement between the skull and the brain as proposed in the shaken baby syndrome hypothesis.<sup>15</sup> These SAT, also referred to as arachnoid trabeculae, subarachnoid space (SAS) trabeculae, or leptomeningeal trabeculae, can be seen with light microscopes but are too thin to be detected by ultrasonography or clinical magnetic resonance imaging.<sup>16</sup> Nevertheless, high-resolution magnetic



resonance imaging has been used to visualize arachnoid adhesions and tissue microstructure within the SAS.<sup>17,18</sup>

Most anatomic research has focused on the subarachnoid cisterns and arachnoid mater but few data on SAT have been reported. The aim of this study is to detail the configuration of SAT and provide information on their embryologic origin, histology, and morphologic variation. Their potential role in CSF flow and their surgical significance are also discussed.

## METHODS

A comprehensive review of the published literature was conducted in PubMed, Medline, EMBASE, Wiley Online Library, Research Gate, Science Direct, Elsevier, Cambridge journals, SAGE journals, and Oxford journals. Terms for the search were subarachnoid trabeculae, subarachnoid trabecular membrane, arachnoid mater, subarachnoid trabeculae embryology, subarachnoid trabeculae histology and morphology, trabecular cisterns, and Lilliequist membrane (LM). No date restrictions were imposed. The decision to involve or eliminate reviews, and data extraction, were completed by the authors, and any controversies and disagreements were resolved by discussion.

## RESULTS

The literature search initially yielded 1113 articles. One hundred and ten of these articles were relevant to SAT, their embryology, histology, morphology, function, and the significance of their microsurgical anatomy for the

neurovascular structures within the subarachnoid cisterns. To ensure the high standard of the review, articles with a high possibility of bias, and any study published in nonpopular journals (not indexed in PubMed MEDLINE), were excluded. Animal studies describing the embryologic development and the histology of the SAT were included. These articles included 19 book chapters, 58 original articles, 31 review articles, and 2 case reports containing reviews of the literature.

### Embryologic Development of SAT

In 1975, McLone and Bondareff<sup>19</sup> reported a detailed electron microscopic study of the embryonic development of SAT in the mouse, which is similar to that in humans.<sup>20</sup> Many of the data available on SAT embryology are based on his work. The pattern of trabecular structure is set during the first 17 postconceptual days in mice. Embryologically, the trabeculae are the remnants of the common precursor that forms both the meningeal arachnoid and pia layers.

### FORMATION OF THE POTENTIAL PIA-ARACHNOID SPACE

During embryogenesis, the initial development of the SAS takes place in 2 phases.

**Phase 1: The Development of a Space-Holding Mesenchymal Layer.** Shortly after closure of the neural tube, a mesenchymal layer moves forward from the future neck region of the developing spine to invade between

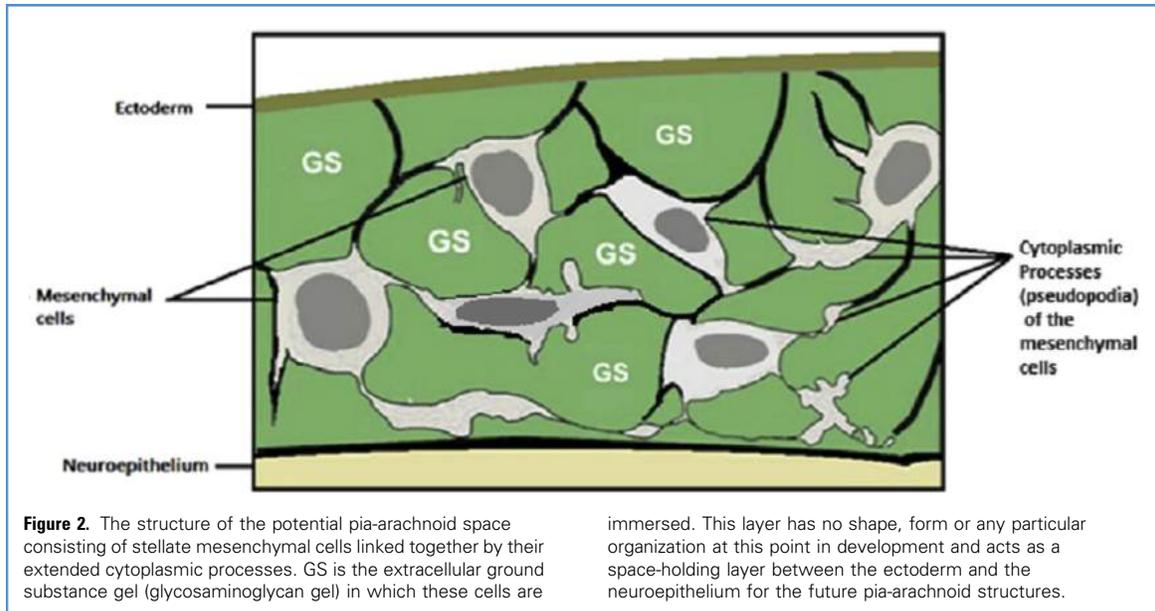
the embryonic epithelium (ectoderm) and the developing neuroepithelium of the telencephalon.<sup>16,19,21</sup> At this stage, there are no arachnoid or pia membranes in the potential pia-arachnoid space (PPAS). This formless layer is composed of a gel-filled mesenchymal network as ground substance and acts as a space-holding layer for the future pia-arachnoid structures (Figure 2).

The space-holding mesenchymal layer is made up of widely spaced, stellate mesenchymal cells linked to each other through long extended interconnecting cytoplasmic processes called pseudopodia.<sup>16,19,21</sup> The extensive extracellular space is filled with glycosaminoglycan (GAG) gel through which gases move by diffusion, but there is no bulky movement.<sup>22</sup> This stage is referred to as meninx primitive, or primitive SAS, by Osaka et al.<sup>23</sup>

**Phase 2: Origination and Expansion of Fluid-Filled Cavities (Lacunae) Causing Compaction of the Mesenchyme and Fibrous Material.** The trabecular structure originates from the localized withdrawal of this GAG gel, which occurs at days 10–13 post-conception from arbitrarily positioned centers that start to appear in the gel, resulting in randomly spaced and sized fluid-filled holes. As the cavities enlarge, the remaining mesenchymal elements consisting of cells and fibers are forced to assemble in the tissue that remains in the cavities. As the cavities meet, the mesenchymal material lining the cavities resists further advancement, leaving thin walls of mesenchyme in random directions, which become the origin of the SAT. The loss of GAG gel on the upper and lower surfaces of the PPAS during days 13–16 allows the mesenchyme to compact to form membranes (Figure 3).

The upper and lower surfaces of the pluripotential placeholder mesenchymal cells start to specialize, becoming fibrocytes, blood cells, vessels, and other tissues, and reinforce these new membranes. These surfaces give rise to the arachnoid and pial structures/membranes to which the trabecular structure remains attached.<sup>24</sup> This description is generalized from rat fetal tissue. Few data on the embryology of the spinal cord SAT are available.

Concentrations of fibrous material also appear, lining the expanding liquid-filled lacunae. Bundles of microfibrils and collagen are commonly associated with



lacunae in the outer pia-arachnoid layer and can serve as struts to maintain an open sub-arachnoid pathway. As the resulting lacunae approach each other closely, the remaining mesenchymal cells and fibers become pressed into curtains stretched across the SAS. These curtains have holes in them through which CSF can flow (Figure 3).

These remaining walls are the trabeculae. Osaka et al.<sup>23</sup> described the resultant fluid space as essentially the cleared-out connective tissue space that is formed late in embryonic life. The framework of the SAS, consisting of the outer arachnoidal membrane, the trabeculae, and the inner pial layer is established by the seventeenth post-conception day.

#### Trabecular Attachments

Collagen fibers from the trabeculae are attached to the arachnoid mater, which forms the top surface of the PPAS, reinforcing it with collagen so it can withstand relatively powerful forces.<sup>16</sup> Below the PPAS, the trabecular collagen passes through the pia mater, across the subpial space, and attaches to the basement membrane, beneath which it is embedded in a layer of astrocytes and oligodendrocytes.<sup>25</sup>

#### Histology and Morphology of SAT

To understand the histology of SAT in greater detail, the histology of the arachnoid membrane is crucial.

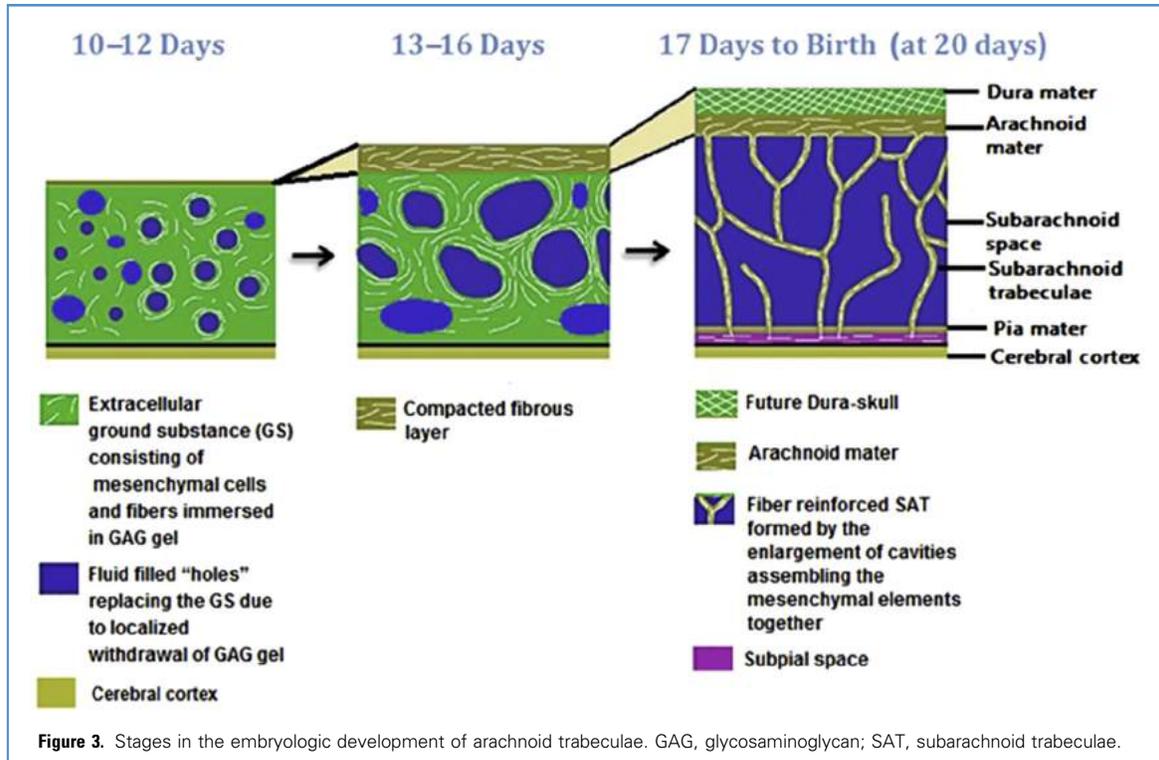
**Arachnoid Mater.** The most superficial layer of the arachnoid, referred to as the subdural mesothelium, subdural neurothelium, or dural border layer, comprises layers of thin, densely arranged cells that abut the dura mater and is considered by Schachenmayr and Friede to be a portion of the dura<sup>16,18,26-29</sup> (Figure 4). Adjacent to this dural border layer is the arachnoid barrier layer, which consists of tightly packed polygonal cells, round nuclei coupled with pale cytoplasm, and a basement lamina that distinguishes it from the rest of the arachnoid. These cells are conjoined by characteristic tight junctions, absent in the dural border and desmosomes that form an impermeable barrier to CSF.<sup>30-34</sup> Nabeshima et al.<sup>35</sup> described this portion of the arachnoid in humans as similar to, but significantly thicker than, the barrier layer of other mammals. Both the dural border and the arachnoid barrier layers are distinguishable by the lack of collagen fibrils that can be found in the pia and arachnoid maters. Deeper to the arachnoid barrier layer, the arachnoid becomes more loosely packed and is intermittently interlaced with collagen fiber bundles. The innermost portion of the arachnoid consists of a narrow layer of leptomeningeal cells that are interconnected by desmosomes and gap junctions.

**SAT.** It seems that previous literature oversimplified the morphology of the SAS

and the SAT. The current concept of trabecular columns connecting the arachnoid to the pia mater is more diverse and complex than previously believed.<sup>30,36-38</sup> The collagen fibers of SAT are enveloped by leptomeningeal cells that are connected through desmosomes and gap junctions, without tight junctions<sup>29</sup> (Figure 5).

Anderson<sup>11</sup> and Hayreh<sup>12,13</sup> acknowledged the presence of SAT in the SAS of the human optic nerve without mentioning types or distribution. Killer et al.<sup>40,41</sup> acknowledged differences in the structure and distribution of SAT among the different segments of the optic nerve. Several investigators have used different terminologies when describing the morphology of SAT. Parkinson<sup>42</sup> used terms such as arachnoid septae, trabeculae, and rough strands to describe spinal SAS structures. Delmaset et al.<sup>43</sup> used terms such as stout, columnar, and sheetlike to describe cranial SAT. Alcolado et al.<sup>29</sup> also used the term sheetlike, along with filiform and chordae. Killer et al.<sup>41</sup> chose to use the terms trabeculae, pillar, and septae to describe SAS morphology of the human optic nerve.

**SAT Variations.** Variations in SAT Along the Optic Nerve. The arachnoid mater and SAS along with the SAT surrounds the nerve throughout its course to the orbital cavity, where they fuse with the sclera.<sup>44</sup> Killer et al. found the trabeculae to be distributed among the bulbar and intra-canalicular



portions, but contrary to the observations by Hayreh<sup>42,43</sup> and Liu and Kahn,<sup>45</sup> the trabeculae were most dense in the bulbar segment of the optic nerve. Septae were located in the midorbital portion, and pillars were found in the intracanalicular and midorbital portions of the optic nerve SAS.

**Bulbar Segment of the Orbital Optic Nerve.** Scanning electron microscopy (SEM) of the bulbar segment showed that the SAS were widest at this segment and contained numerous round SAT without broadening at the arachnoid and pia layers.<sup>40,41</sup> (Figure 6). They were found to have branches that formed a complex and delicate network. The measured width of the trabeculae ranged from 5 to 7  $\mu\text{m}$ .<sup>40,41</sup> They were enveloped in a sheath of flat, smooth leptomeningeal cells that on occasion contained fenestrations 0.2–1.0  $\mu\text{m}$  wide, probably because of the transmission electron microscopy (TEM) preparation and perhaps were not real holes. TEM showed these leptomeningeal cells forming single or multiple layers; the multiple layers were attached by desmosomes.<sup>41,45</sup> It also showed that an

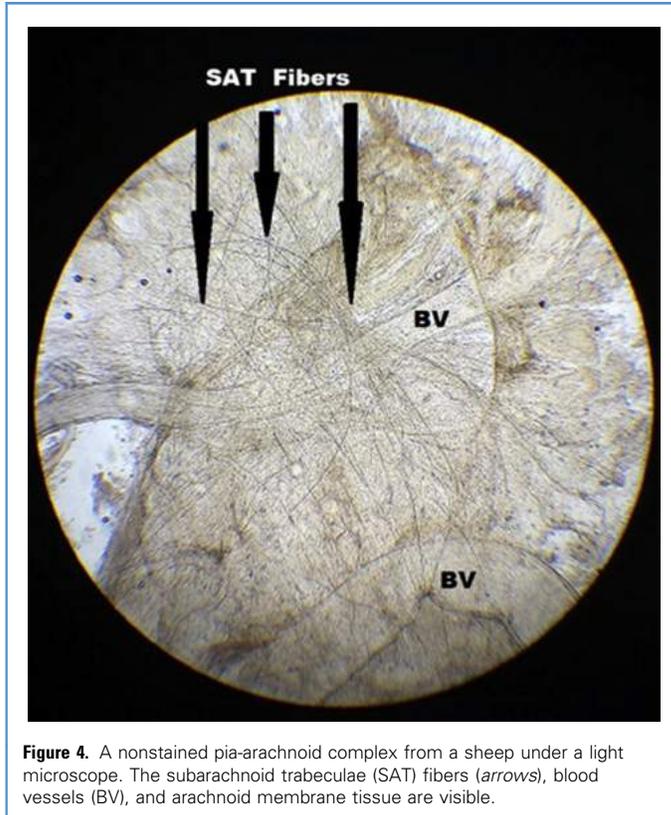
extracellular matrix supported these cells. The center of the trabeculae consisted of densely packed collagen fibrils organized into small bundles. Slim cytoplasmic bridges were seen connecting one trabecula to an adjacent one. Occasionally, the trabecular networks were noted to contain a blood vessel or 2, as also reported by Alcolado et al.<sup>29</sup>

**Midorbital Segment of the Orbital Optic Nerve Portion.** The SAS of the midorbital segment was smaller than that of the bulbar segment and consisted of an abundance of broad septae and round pillars but contained no trabeculae.<sup>40,41,45</sup> Measurements were not provided for the septae, which were described as dividing the SAS into chambers and containing large perforations that connected adjacent chambers. However, the pillars were measured at a diameter ranging from 10 to 30  $\mu\text{m}$  and possessed broadened ends at both terminations.<sup>40,41,45</sup> The larger diameter and broadened ends of the pillars differentiated them from the trabeculae, which were smaller in diameter and lacked broadened ends. However, the histology of the leptomeningeal cells and

the central components of the septa and pillars was comparable to that of the trabeculae.

**Intracanalicular Portion of the Optic Nerve.** The SAS of the intracanalicular segment was extremely narrow and consisted of pillars and trabeculae as previously described. The center of the canal established 1 or 2 large pillars approximately 0.5 mm in size and encompassing 1 or 2 blood vessels.<sup>41,45</sup> The other parts of the intracanalicular segment showed either delicate round and slightly curved trabeculae of approximately 5  $\mu\text{m}$  diameter or single pillars with a diameter of approximately 25  $\mu\text{m}$ , which expanded at the dural and pial attachment of the arachnoid layer.<sup>45</sup> At the orbital opening of the canal, the trabeculae were more abundant, running in parallel and bridging the SAS obliquely.

**Variations in SAT Along Blood Vessels and Nerves.** SAT enclose the small blood vessels and adhere to the surface of larger blood vessels in the SAS and cisterns.<sup>1</sup> Furthermore, fine capillaries have been found in the trabeculae of rats.<sup>46</sup> At the sites of attachment, the trabeculae cells



**Figure 4.** A nonstained pia-arachnoid complex from a sheep under a light microscope. The subarachnoid trabeculae (SAT) fibers (arrows), blood vessels (BV), and arachnoid membrane tissue are visible.

become continuous with the cells on the surface of the pia or the blood vessels.<sup>1</sup> According to Yaşargil,<sup>46</sup> SAT also adhere to the nerves within the SAS. SAT tend to be thicker where the arteries and nerves pass through the trabeculated wall from one cisternal compartment to another. In most individuals, the 3 cisterns in which the arachnoid trabeculae and membranes are condensed and present the greatest impediments during operations are the interpeduncular cistern, the quadrigeminal cistern, and the cisterna magna.<sup>47,48</sup> Neural elements including nerve endings in the arachnoid and SAT, mainly in the cisterna magna, have been described; they could contribute to conveying information about CSF pressure gradients and also in cerebral vasospasm.<sup>46</sup>

In 2015, Saboori and Sadegh<sup>20</sup> used Sprague-Dawley rats to investigate the histology and morphology of SAT more fully since it had been shown that the trabeculae in rats and humans were morphologically similar.<sup>49-51</sup> SEM showed tree-shaped SAT that consisted of branches from the arachnoid mater converging into a single trunk attached to the pia mater. Other

shapes of trabeculae were also mentioned and described as “plates,” “veillike,” “pillars,” and “rods.”<sup>20</sup> The veillike morphology was reported in regions where there was a greater density of SAT, usually associated with the closeness of blood vessels. SEM also established that there are holes in the trabeculae ranging in size from approximately 0.5 to 3.0  $\mu\text{m}$ , making them permeable.

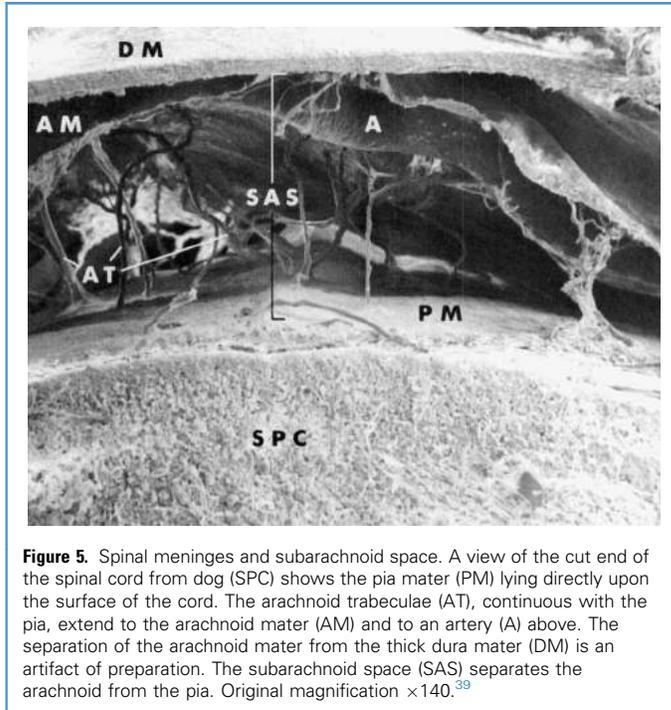
TEM was performed and the general appearance of the collagen fibril bundles, the thickness of the individual collagen fibers, and the periodicity seen with alternating light and dark periods confirmed what Kierszenbaum and van der Rest and Garrone<sup>52</sup> had claimed: that the SAT were composed of type I collagen.<sup>53</sup> TEM also showed the collagen fibril groups to have a lateral and a transverse orientation, which confirmed the rodlike morphology of the SAT seen on SEM. Fibroblastic cells surrounded the collagen fibril groups that make up the SAT. Also, the SAT were surrounded by 50–200 nm of extracellular matrix that consisted not only of collagen fibrils but also of fibronectins, laminins, and proteoglycans. The collagen fibril

groups had fluctuating densities of fibrils; some were very densely packed, whereas others were less dense. Fluid was also found in deeper layers of the arachnoid between cells, which indicated that the arachnoid layer must have some degree of permeability.

LM: An Anatomic Variant. According to Froelich et al.,<sup>54</sup> the LM is a complex and variable arachnoidal structure that is either a single-layered, 2-layered, or 3-layered membrane. It is formed by a group of anatomically distinct arachnoid sheets: a diencephalic leaf (DL), a mesencephalic leaf (ML), and a pair of diencephalic–mesencephalic leaves.<sup>55</sup> According to Wang et al.,<sup>47</sup> it consists of 3 layers: mesencephalic, diencephalic, and a pair of hypothalamic membranes.

Spinal SAT. The literature on spinal SAT is sparse and knowledge about them is limited. Nauta et al.<sup>56</sup> were the first to review the anatomy of the spinal subarachnoid with reference to the cadaver dissection work of Key and Retzius and based on their own operative experiences.<sup>57</sup> Nauta et al.<sup>56</sup> found that despite some variations, there were consistent features in the spinal arachnoid anatomy. To resolve the confusion in the standard texts and literature from a diversity of names, descriptions, and drawings of the human spinal subarachnoid septa and trabeculae, Parkinson in 1991<sup>42</sup> carried out a study by examining 62 complete human cords under the dissecting microscope. He found that anteriorly there were essentially no connecting septa or trabeculae between the cord and the arachnoid membrane. Posteriorly, there is a scanty series of connecting fibers and fenestrated sheets 1 or 2 mm on either side of the midline (dorsolateral septa) in the upper cervical region. These fibers become increasingly more widespread in the lower cervical region and remain extensive in the lumbar enlargement, beyond which they progressively dwindle to end abruptly at the filum terminale origin.

According to Rickenbacher et al.,<sup>58</sup> only in the upper cervical region are there few trabeculae in the midline both ventrally and dorsally. In the lower cervical region, only the dorsal trabeculae fibers show membranous expansion, first forming an incomplete and then caudally a complete membrane called the dorsal subarachnoid septum (septum



**Figure 5.** Spinal meninges and subarachnoid space. A view of the cut end of the spinal cord from dog (SPC) shows the pia mater (PM) lying directly upon the surface of the cord. The arachnoid trabeculae (AT), continuous with the pia, extend to the arachnoid mater (AM) and to an artery (A) above. The separation of the arachnoid mater from the thick dura mater (DM) is an artifact of preparation. The subarachnoid space (SAS) separates the arachnoid from the pia. Original magnification  $\times 140$ .<sup>39</sup>

posticum).<sup>58</sup> This septum extends as far down as the conus medullaris dividing the dorsal SAS into left and right compartments. Throughout the length, there are many unexplained, redundant, nonbranching, beaded, thicker rogue strands. All of these strands differ in character from the right-angle fiber arrangement of the denticulate ligament, the 2 leaves of which are often separated to form segmental longitudinal tunnels.<sup>42</sup> Intermittently, they become tangentially adherent to the arachnoid membrane. In the thoracic region, these trabeculae form relatively fenestrated membranes running obliquely anteroinferiorly in correspondence to the nerve roots, forming slanting compartments.<sup>58</sup> In the lumbar region, these root septa divide into trabeculae that become progressively scant toward the cauda equina and along its course. Throughout the cauda equina, strands are haphazardly arranged connecting the roots and supporting blood vessels.<sup>42,58</sup> Parkinson<sup>42</sup> found no evidence of change in the number or type of connection with age.

The spinal SAT give shape to tubular arachnoid sheaths for each nerve root and for the spinal cord.<sup>59</sup> Fila radicularia, the nerve rootlets for each dermatome, are

joined to each other by strands and webs. The trabecular arachnoid of the spine restricts nerve root movement to a definite extent, holding each root in its position within the dural sac and in relation to other nerve roots.<sup>59</sup> As the ventral roots of the spinal nerves traverse the ventral part of the SAS, their filaments are hinged to each other and to the ligamentum denticulatum by the delicate trabecular connective tissue.<sup>58</sup>

#### Functions of SAT

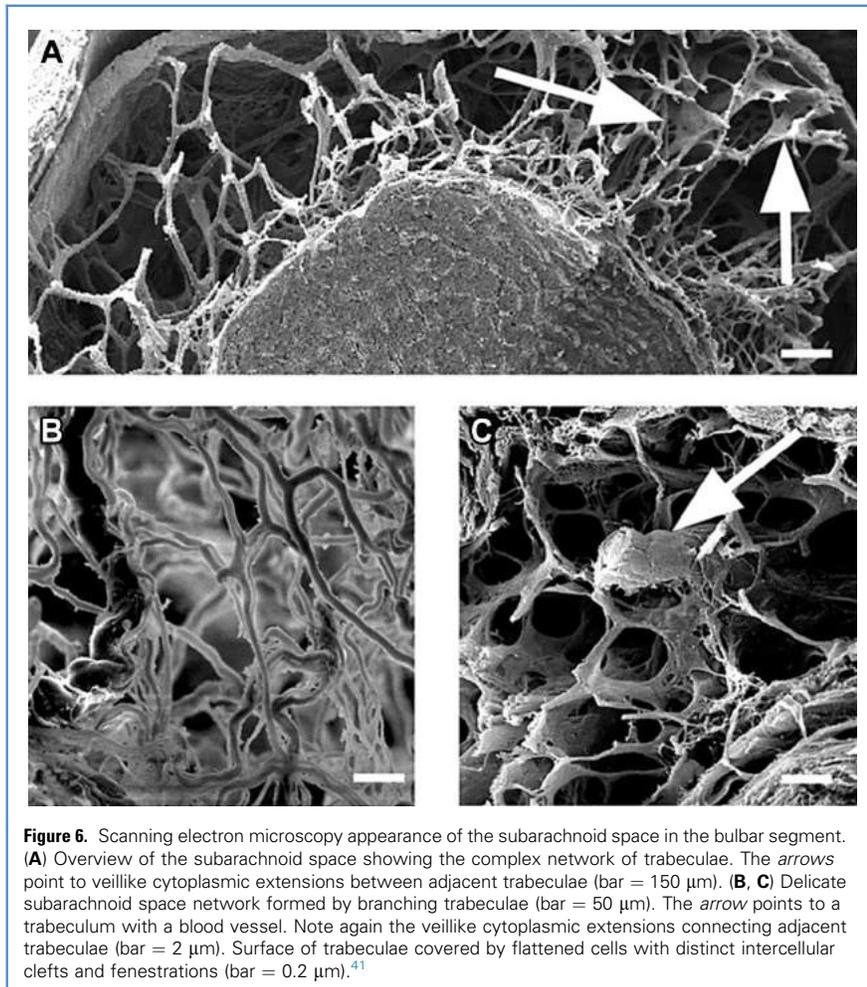
**Mechanical Properties.** According to Killer et al.,<sup>40</sup> SAT could play more of a filler role rather than a support role. Other reports<sup>40,60-64</sup> state that SAT seem to be important as mechanical pillars between the pia and arachnoid membranes, damping and constraining the movement of the brain relative to the skull, and thereby possibly affecting traumatic brain injuries. The random three-dimensional redundant structure of the walls is resilient and can lose a few elements without failure under severe conditions such as trauma. In such conditions, the stress is redistributed among the remaining elements of its structure.<sup>15,60</sup>

A model described by Scott et al.<sup>64</sup> suggests that the PAC, which includes the

arachnoid membrane, arachnoid trabeculae, subarachnoid vasculature, and pia membrane, has a significant effect on brain biomechanics and increases the local variability of stress along the brain. This study, carried out using complex finite element models of the immature piglet brain to identify changes in cortical stress distribution, showed that incorporating the regional variability of PAC substructures substantially altered the distribution of the main stress on the cortical surface of the brain. This finding shows that despite the small volumetric contribution of the PAC to the intracranial space, the microstructural variability has a considerable effect on brain mechanics during head rotation, thereby contributing significantly to brain deformation. The data suggested that this regional variability of PAC substructures could also influence localized predictions of intracranial hemorrhage.<sup>64</sup>

**Role in CSF Flow.** The curtainlike structure of the SAT stretched across the SAS has holes through which CSF flows (Figure 7). This feature could play a part in the CSF dynamics between the SAS of the optic nerve and the chiasmal cistern and contribute to understanding of the pathophysiology of asymmetric and unilateral papilledema.<sup>40,41</sup> Changes in their structure after trauma or hemorrhage could in principle contribute to posttraumatic and posthemorrhagic hydrocephalus. Alterations in CSF flow velocities have been noted in the spinal SAS near arachnoid adhesions.<sup>18,65</sup> Several computational fluid dynamics–based studies have indicated the possible importance of SAT in CSF solute transport and pressures. Stockman<sup>66</sup> found that SAS microanatomy increased CSF flow mixing but had a relatively small effect on overall CSF velocity profiles. Tangen et al.<sup>67</sup> found that SAT drastically affected solute transport within the SAS. Gupta et al.<sup>68,69</sup> implemented a computational fluid dynamics model that included anisotropic permeability within the cortical SAS caused by pillar-shaped SAT.

**Role After Hemorrhage.** In subarachnoid hemorrhage (SAH), SAT as collagen bundles in the SAS are considered to have a role in activating the Hageman factor in the coagulation system.<sup>70,71</sup>



**Figure 6.** Scanning electron microscopy appearance of the subarachnoid space in the bulbar segment. (A) Overview of the subarachnoid space showing the complex network of trabeculae. The arrows point to veil-like cytoplasmic extensions between adjacent trabeculae (bar = 150 µm). (B, C) Delicate subarachnoid space network formed by branching trabeculae (bar = 50 µm). The arrow points to a trabeculum with a blood vessel. Note again the veil-like cytoplasmic extensions connecting adjacent trabeculae (bar = 2 µm). Surface of trabeculae covered by flattened cells with distinct intercellular clefts and fenestrations (bar = 0.2 µm).<sup>41</sup>

Platelets are also believed to be activated by trabeculae, as well as by thrombin during SAH.<sup>70,71</sup>

**Role in Formation of Chronic Subdural Hematomas.** Chronic subdural hematomas (CSDHs) usually occur after a head trauma in children, and without trauma in the alcoholic population and in the elderly (>65 years) as a result of cerebral atrophy and degeneration when there is adequate subdural space.<sup>72,73</sup> SAT may contribute to posttraumatic CSDH or hygromas possibly by tears in minor vessels supplying the arachnoid, as well as tears in the arachnoid caused by traction of the trabeculae.<sup>74,77</sup> The trabeculae in the SAS are more condensed compared with the subdural space, contributing more to the delicate nature of bridging veins. In trauma, the tinny fragile walls of these bridging veins, the

organization of collagen fibers, and the absence of strengthening trabeculae lead to separation of the dural border cell (DBC) layer and tearing of bridging veins, resulting in bleeding and hematoma formation.<sup>74,78</sup>

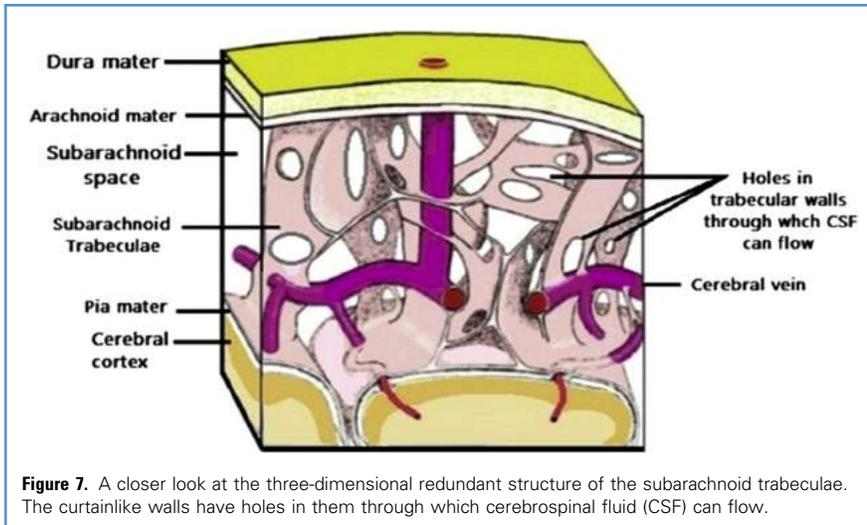
In elderly patients with age-adequate cerebral atrophy, a negative pressure is produced within the cranium as well, as the distance from the skull to the cerebral cortex becomes longer.<sup>74</sup> The bridging veins are stretched on the atrophied hemisphere and may become torn even by a minor unnoticed nontraumatic acceleration-deceleration injury.<sup>74,75</sup> If the distance surpasses the length of SAT, it causes the separation of the DBC layer, forming a hypothetical subdural space in which the blood from the bleeding bridging veins is accumulated.<sup>75</sup> Cranial morphology and the degree of cerebral atrophy are the 2 actors that determine the force pulling the SAT

that leads to the separation of the DBC layer.<sup>75</sup> Intracranial hypotension caused by CSF leakage and coagulopathies can also lead to CSDH in the same manner.<sup>74,75,79</sup>

### Surgical Significance of SAT

Arachnoid membranes, including LM, are of paramount surgical importance and are key landmarks in microsurgical procedures.<sup>80-82</sup> These structures help to delineate the contour of the lesions, hence protecting nearby brain structures. Nevertheless, neurosurgeons should always pay particular attention to the topography of the cisterns and associated arachnoidal adhesions and trabeculae in microsurgical approaches to preclude injury to the surrounding neurovascular structures. Furthermore, the trabecular membranes are structured in a compartmental form that can limit the spread of blood (e.g., from a ruptured aneurysm) to other cisterns by allowing the injury to remain localized by observing the blood-filled cistern.<sup>80,83-85</sup>

Although Yaşargil<sup>46,86,87</sup> provided a detailed description of the subarachnoid cistern, the compartmental trabecular membranes remain to be described. Vinas et al.<sup>88-90</sup> were the first to describe the microsurgical anatomy of the compartmental SAT and their surgical significance in detail. These investigators noted that the SAS is lined by trabecular membranes, extending from the arachnoid mater to the pia mater.<sup>88-90</sup> They also noticed that in certain areas, trabecular membranes form dense networks that resemble an authentic true membrane.<sup>88-90</sup> Yaşargil (1984) and Vinas (1994) also reported that these SAT divide the SAS into compartments called cisterns.<sup>46,89</sup> According to Yaşargil et al.,<sup>46,86,87</sup> these SAT networks hold a significant microsurgical importance because they provide physical support to the arteries, veins, and nerves that pass within and through them. However, the walls of the cisterns direct the flow of CSF through openings of various sizes.<sup>88-90</sup> These membranes help to protect the entire SAS from collapse during rupture or a surgical approach to a cistern with the consequent loss of CSF.<sup>85,87,89</sup> However, in certain situations such as SAH or bacterial meningitis, the flow of fluid through various cisterns can be retarded or prevented.<sup>88-90</sup>



**Figure 7.** A closer look at the three-dimensional redundant structure of the subarachnoid trabeculae. The curtainlike walls have holes in them through which cerebrospinal fluid (CSF) can flow.

Microsurgical procedures should be performed with great care and the trabeculae pulled as lightly as possible because of their relationship to neurovascular structures<sup>88-90</sup> (see [Video 1](#)). Vinas et al.<sup>88-90</sup> examined the relationship between SAT found in supratentorial and infratentorial levels and at the levels of tentorium to their corresponding blood vessels and cranial nerves. According to the data available in the literature, the variations of the subarachnoid trabecular membrane and subarachnoid cisterns are summarized in [Tables 1](#) and [2](#).<sup>88-94</sup>

LM is an important anatomic landmark in the approach to the parasellar and premesencephalic and prepontine areas.<sup>80,88-90,95-97</sup> Understanding of trabecular membranes, including the LM and its relation to the surrounding structures, which can be determined by preoperative imaging, can help surgeons to plan the route of access, improve exposure, and minimize injuries.

The DL of this membrane has significant importance in surgical approaches to the sellar and parasellar region and divides the cisterns of the skull base into pre-Liliequist and post-Liliequist groups.<sup>54,55,96,98</sup> The ML has less surgical importance than does the DL.<sup>54,95</sup> The ML separates the supratentorial from infratentorial cisterns.<sup>54,87,97</sup> In perimesencephalic lesions, such as diaphragm sellae meningiomas, and trigeminal neuromas, the ML can be preserved by displacing upward and can

provide a safe and clear surgical plane for operating on these tumors, if it is followed clearly.<sup>54,55,96,98</sup>

## DISCUSSION

Previous studies have sought to describe the meninges and their roles in brain and spinal cord function and stability. Comparatively little attention has been given to the SAT and their role. Yaşargil made ground-breaking efforts in describing the topographic anatomy of subarachnoid cisterns and their importance in cerebrovascular and skull base surgery.<sup>46,86,89</sup> SAT were also mentioned as part of these subarachnoid cisterns but their structure and role have not been further delineated. In recent years, researchers have described SAT anatomy and variations in the central nervous system.<sup>11-13,20,29-38,40-81,99</sup>

SAT have mostly been seen as part of the PAC. In recent years, they have attracted increasing interest. Their role has evolved from being a filler of the SAS into supporting pillars. This role immediately puts them at the center for balancing intracranial and intraspinal biomechanics, stabilizing neurovascular structures such as cranial nerves, arteries, and veins in the arachnoid cisterns and also affecting CSF flow, thereby giving them surgical significance.

### The Role of SAT as Supporting Structures and Interaction with Intracranial Pressure

During microsurgery, SAT maintain the SAS as a firm open CSF-filled

compartment. Dissection within the subarachnoid cisterns depends on sectioning of the SAT. An interesting model developed by Scott et al.<sup>64</sup> implied that changes in the density of SAT could affect the local biomechanical properties of the brain, making specific regions prone to traumatic bleeding. Studies are under way measuring the biomechanical properties of the SAT and their potential alterations during different CNS diseases.

Although the SAT and arachnoid membrane act as a support structure, they are also held in place by intracranial pressure within the CSF system. Alterations in intracranial CSF pressure can affect the SAT, arachnoid membranes, and CSF space geometries. For example, post mortem, the arachnoid membrane quickly delaminates from the dura and collapses on top of the cortical surface as a result of loss of intracranial pressure concomitant with CSF absorption into the dural venous sinuses as venous pressure decreases. Thus, postmortem visualization of SAT is difficult because the layers of tissue and fibers become relatively tightly cupped around the brain. The cortical SAS changes so dramatically that the cortical CSF normally enveloping the brain is nearly nonexistent. This situation can make anatomic dissection and study of the SAT and related structures challenging and drastically alters study of the normal CSF compartmental volumes and geometry. Similarly, during surgical opening of the arachnoid membrane, CSF pressures are altered and can thereby also affect the normal CSF space geometry and volumes. Delamination of the arachnoid membrane from the dura and leakage of CSF into this region can result in formation of arachnoid cysts and other cavities. The interaction of CSF flow and pressures can lead to alterations in arachnoid cyst volume.

### The Relationship Between the Number of SAT and Propensity for Cerebral Trauma

As Scott et al. suggested in their numeric modeling studies discussed in detail earlier, more numerous SAT could increase the likelihood of injury to the brain. Increased SAT can increase surface stresses on the brain, leading to a greater propensity for injury to the delicate brain tissue and cortical draining veins. Here, there is a huge gap of knowledge that needs to be addressed in future research.



Video available at  
[WORLDNEUROSURGERY.org](http://WORLDNEUROSURGERY.org)

**Table 1.** Paired and Unpaired Trabecular Membranes

Subarachnoid Trabecular Membrane	Supratentorial Level	Infratentorial Level	At the Level of Tentorium
Paired	Anterior cerebral membrane	Superior cerebellar membrane	Cerebellar precentral
	Posterior communicating membrane	Basilar membrane	Superior cerebellar
	Anterior choroidal membrane	Anterior inferior cerebral membrane	Lateral oculomotor
	Lateral oculomotor membrane	Posterior inferior cerebral membrane	Caudal oculomotor
	Caudal oculomotor membrane		Perforated membrane
	Olfactory membrane		
	Carotid membrane		
Unpaired	Liliequist membrane	Liliequist membrane	Liliequist membrane
	Top basilar	Chiasmatic	Top basilar

### Potential Role of SAT in Hydrocephalus and Related Disorders

Understanding their anatomy, along with further understanding of CSF dynamics, sheds new light on potential functions of the SAT. It is not unrealistic to assume that thickening of the SAT after trauma or especially SAH could be a cofactor in the development of communicating hydrocephalus. For example, a local increase in SAT distribution could alter the pulse-damping characteristics of the intracranial cavity and/or flow dynamics,<sup>21,24,100</sup> thereby affecting interstitial fluid transport within the CNS tissues.<sup>69,101-103</sup> If this process could be shown then the surgical literature would need to reform its terminology of communicating hydrocephalus to micro-obstructive hydrocephalus if hypertrophied SAH proved to be the cause. Other recent work has indicated a possible

role of CSF pulse-timing along the spine in syringomyelia.<sup>104-107</sup> The elongated SAT structure and integral fluid-solid connection to the delicate tissue surface could also contribute via the cellular mechanosensitivity of CNS tissues.<sup>108-110</sup>

### Surgical Importance of SAT

The role of SAT in maintaining the neurovascular structures within the SAS has been extensively outlined in this study. Retractorless microneurosurgery has emerged as an important aspect of modern safe neurosurgery. Cautious sectioning of the SAT within the SAS during surgery allows the neurosurgeon not only to mobilize the cranial nerves, arteries, and veins within the subarachnoid cisterns and to protect them but also to mobilize the brain easily with no need for retraction. Hence, access to areas that previously

needed retraction would be facilitated by careful sharp dissection of the SAT, and without retraction. The emergence of retractionless microneurosurgery, and the importance of neurovascular structures within the subarachnoid cisterns and the potential role of SAT, are all reasons enough to extend much-needed basic and clinical research to these previously overlooked but important structures.

### CONCLUSIONS

SAT are delicate thin mesenchymal columns of collagen-reinforced material stretching between the arachnoid and pia membranes and appear to be significant for the stability of the SAS, the protection of the cranial nerves, arteries and vessels within the subarachnoid cisterns, the stability of the brain within the SAS, and potentially

**Table 2.** Paired and Unpaired Subarachnoid Cisterns

Subarachnoid Cisterns	Supratentorial Level	Infratentorial Level	At the Level of Tentorium
Paired	Olfactory cistern	Pontocerebellar cistern	Ambient cistern
	Carotid cistern	Cerebellomedullar cistern	
	Sylvius cistern	Ambient cistern	
	Crural cistern		
	Posterior communicating cistern		
	Oculomotor cistern		
Unpaired	Chiasmatic cistern	Basilar cistern	Interpeduncular cistern
	Lamina terminalis cistern	Cisterna magna	Quadrigeminal cistern
	Pericallosal cistern	Supra-cerebellar cistern	Superior cerebellar cistern
			Velum interpositum cistern

CSF flow, because of their redundant three-dimensional structure. Understanding their anatomy and function could be crucial for establishing a new understanding of the evolution of hydrocephalus. Careful dissection seems to be a crucial aspect of modern surgical techniques for safe retractorless neurosurgery. Specific biomechanical studies are needed to understand their physiologic significance and their potential causative or reactive role in different conditions of the brain and spinal cord.

## REFERENCES

- Adeeb N, Deep A, Griessenauer CJ, Mortazavi MM, Watanabe K, Loukas M, et al. The intracranial arachnoid mater: a comprehensive review of its history, anatomy, imaging, and pathology. *Childs Nerv Syst*. 2013;29:17-33.
- Bakay L. Discovery of the arachnoid membrane. *Surg Neurol*. 1991;36:63-68.
- Sanan A, van Loveren HR. The arachnoid and the myth of Arachne. *Neurosurgery*. 1999;45:152-155 [discussion 155-157].
- Casseri I (1627) *Tabulae anatomicae LXXIIX*. Venice.
- Varolius C (1573) *De Nervis Opticis nonnullisque aliis praeter communem opinionem in Humano capite observatis*.
- Goelicke AO, Ruysch F (1697) *Epistola anatomica, problematica nona*. Apud J. Wolters.
- Blasius G, Vesling J (1666) *Syntagma Anatomicum*.
- Olry R, Haines DE. *NEUROwords 11. Arachnophobia: spiders and spider's webs in the head*. *J Hist Neurosci*. 2001;10:198-200.
- Ruysch F, Etmüller ME. *Epistola, Anatomica, Problematica, Duodecima*. Amsterdam: Joannem Wolter; 1699.
- Tahir MZ, Quadri SA, Farooqui M, Bari ME, Di X. Tension arachnoid cyst causing uncal herniation in a 60 year old: a rare presentation. *CNS Neurol Disord Drug Targets*. 2012;11:127-131.
- Anderson DR. Ultrastructure of meningeal sheaths: normal human and monkey optic nerves. *Arch Ophthalmol*. 1969;82:659-674.
- Hayreh SS. Pathogenesis of oedema of the optic disc. *Doc Ophthalmol*. 1968;24:289-411.
- Hayreh SS. The sheath of the optic nerve. *Ophthalmologica*. 1984;189:54-63.
- Moore KL, Dalley AF. *Head. Clinically Oriented Anatomy*. 5th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2006:885-1043.
- Guthkelch AN. Infantile subdural haematoma and its relationship to whiplash injuries. *BMJ*. 1971;2:430-431.
- Talbert DG. The embryological development of the form of the trabeculae bridging the sub-aracnoid space. *J Trauma Treat*. 2014;3:198.
- Sigmund EE, Suero GA, Hu C, McGorty K, Sodickson DK, Wiggins GC, et al. High-resolution human cervical spinal cord imaging at 7 T. *NMR Biomed*. 2012;25:891-899.
- Gottschalk A, Schmitz B, Mauer UM, Bornstedt A, Steinhoff S, Danz B, et al. Dynamic visualization of arachnoid adhesions in a patient with idiopathic syringomyelia using high-resolution cine magnetic resonance imaging at 3T. *J Magn Reson Imaging*. 2010;32:218-222.
- McLone DG, Bondareff W. Developmental morphology of the subarachnoid space and contiguous structures in the mouse. *Am J Anat*. 1975;142:273-293.
- Saboori P, Sadegh A. Histology and morphology of the brain subarachnoid trabeculae. *Anat Res Int*. 2015;2015:279814.
- Kaplan S, Bolender D. Embryology. In: Polin RA, Fox WW, eds. *Fetal and Neonatal Physiology*. 1st ed. Philadelphia, PA: WB Saunders; 1992:19-36.
- Cormack DH. Loose connective tissue and adipose tissue. In: Cormack DH, ed. *Ham's Histology*. Philadelphia: Lippincott; 1987:155-187.
- Osaka K, Handa H, Matsumoto S, Yasuda M. Development of the cerebrospinal fluid pathway in the normal and abnormal human embryos. *Childs Brain*. 1980;6:26-38.
- Bondareff W, McLone DG, Decker SJ. Ultrastructure of gliothelium in the brain of mice and man. *Anat Rec*. 1973;175:487.
- Braak E. On the fine structure of the external glial layer in the isocortex of man. *Cell Tissue Res*. 1975;157:367-390.
- Andres KH. [On the fine structure of the arachnoid villi in mammals]. *Z Zellforsch Mikrosk Anat*. 1967;82:92-109 [in German].
- Andres KH. [On the fine structure of the arachnoid and dura mater of mammals]. *Z Zellforsch Mikrosk Anat*. 1967;79:272-295 [in German].
- Pease DC, Schultz RL. Electron microscopy of rat cranial meninges. *Am J Anat*. 1958;102:301-321.
- Alcolado R, Weller RO, Parrish EP, Garrod D. The cranial arachnoid and pia mater in man: anatomical and ultrastructural observations. *Neuropathol Appl Neurobiol*. 1988;14:1-17.
- Schachenmayr W, Friede RL. The origin of subdural neomembranes. I. Fine structure of the dura-arachnoid interface in man. *Am J Pathol*. 1978;92:53-68.
- Reese TS, Karnovsky MJ. Fine structural localization of a blood-brain barrier to exogenous peroxidase. *J Cell Biol*. 1967;34:207-217.
- Brightman MW, Reese TS. Junctions between intimately apposed cell membranes in the vertebrate brain. *J Cell Biol*. 1969;40:648-677.
- Stahelin LA. Structure and function of intercellular junctions. *Int Rev Cytol*. 1974;39:191-283.
- Wade JB, Karnovsky MJ. The structure of the zonula occludens. A single fibril model based on freeze-fracture. *J Cell Biol*. 1974;60:168-180.
- Nabeshima S, Reese TS, Landis DM, Brightman MW. Junctions in the meninges and marginal glia. *J Comp Neurol*. 1975;164:127-169.
- Jin X, Lee JB, Leung LY, Zhang L, Yang KH, King AI. Biomechanical response of the bovine pia-arachnoid complex to tensile loading at varying strain-rates. *Stapp Car Crash J*. 2006;50:637-649.
- Guo P, Weinstein AM, Weinbaum S. A hydrodynamic mechanosensory hypothesis for brush border microvilli. *Am J Physiol Renal Physiol*. 2000;279:F698-F712.
- Rao V, Lyketsos C. Neuropsychiatric sequelae of traumatic brain injury. *Psychosomatics*. 2000;41:95-103.
- Cloyd MW, Low FN. Scanning electron microscopy of the subarachnoid space in the dog. I. Spinal cord levels. *J Comp Neurol*. 1974;153:325-368.
- Killer HE, Laeng HR, Groscurth P. Lymphatic capillaries in the meninges of the human optic nerve. *J Neuroophthalmol*. 1999;19:222-228.
- Killer HE, Laeng HR, Flammer J, Groscurth P. Architecture of arachnoid trabeculae, pillars, and septa in the subarachnoid space of the human optic nerve: anatomy and clinical considerations. *Br J Ophthalmol*. 2003;87:777-781.
- Parkinson D. Human spinal arachnoid septa, trabeculae, and "rogue strands". *Am J Anat*. 1991;192:498-509.
- Allen DJ, Low FN. Scanning electron microscopy of the subarachnoid space in the dog. III. Cranial levels. *J Comp Neurol*. 1975;161:515-539.
- Snell RS. *Clinical Neuroanatomy*. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2010.
- Liu D, Kahn M. Measurement and relationship of subarachnoid pressure of the optic nerve to intracranial pressures in fresh cadavers. *Am J Ophthalmol*. 1993;116:548-556.
- Yasargil MG. Subarachnoid cisterns. In: Yasargil MG, ed. *Microneurosurgery*. Vol. 1. New York: Thieme Verlag; 1984:5-53.
- Wang SS, Zheng HP, Zhang FH, Wang RM. The microanatomical structure of the cistern of the lamina terminalis. *J Clin Neurosci*. 2011;18:253-259.
- Matsuno H, Rhoton AL Jr, Peace D. Microsurgical anatomy of the posterior fossa cisterns. *Neurosurgery*. 1988;23:58-80.
- Alcolado JC, Moore IE, Weller RO. Calcification in the human choroid plexus, meningiomas and pineal gland. *Neuropathol Appl Neurobiol*. 1986;12:235-250.

50. Frederickson RG. The subdural space interpreted as a cellular layer of meninges. *Anat Rec*. 1991;230:38-51.
51. Scott G, Coats B. Micro-scale finite element modeling and optical coherence tomography imaging of the pia arachnoid complex. In: *Proceedings of the 17th U.S. National Congress on Theoretical and Applied Mechanics, East Lansing, Michigan, USA, 2014*.
52. van der Rest M, Garrone R. Collagen family of proteins. *FASEB J*. 1991;5:2814-2823.
53. Kierszenbaum AL. *Histology and Cell Biology: An Introduction to Pathology*. Philadelphia, PA: Mosby Elsevier; 2007.
54. Froelich SC, Abdel Aziz KM, Cohen PD, van Loveren HR, Keller JT. Microsurgical and endoscopic anatomy of Lilliequist's membrane: a complex and variable structure of the basal cisterns. *Neurosurgery*. 2008;63:ONS1-ONS8 [discussion ONS8-ONS9].
55. Lü J, Zhu XL. Cranial arachnoid membranes: some aspects of microsurgical anatomy. *Clin Anat*. 2007;20:502-511.
56. Nauta HJ, Dolan E, Yasargil MG. Microsurgical anatomy of spinal subarachnoid space. *Surg Neurol*. 1983;19:431-437.
57. Key A, Retzius MG. *Studien in der Anatomie des Nervensystems und des Bindegewebes*. Stockholm: Samson & Wallin, P.A. Norstedt & Söner; 1875-76 [in German].
58. Rickenbacher J, Landolt AM, Theiler K. *Applied Anatomy of the Back*. Berlin, Germany: Springer Science & Business Media; 2013:240.
59. Reina MA, De Andrés JA, Hadzic A, Prats-Galino A, Sala-Blanch X, Van Zundert AA. *Atlas of Functional Anatomy for Regional Anesthesia and Pain Medicine: Human Structure, Ultrastructure and 3D Reconstruction Images*. Switzerland: Springer International Publishing; 2014:479-497.
60. Zoghi-Moghadam M, Sadegh AM. Equivalent fluid model for CSF and SAS trabeculae using head/brain damping. *Int J Biomed Eng Technology*. 2010;4:195-210.
61. Talbert DG. It's a stitch-up: the function of subarachnoid trabeculae. *J Trauma Treat*. 2016;5:318.
62. Ramakrishnan V, Dahlin R, Hariri O, Quadri SA, Farr S, Miulli D, et al. Anti-epileptic prophylaxis in traumatic brain injury: a retrospective analysis of patients undergoing craniotomy versus decompressive craniectomy. *Surg Neurol Int*. 2015; 20:6-8.
63. Tahir MZ, Quadri SA, Hanif S, Javed G. Traumatic retroclival epidural hematoma in pediatric patient—case report and review of literature. *Surg Neurol Int*. 2011;2:78.
64. Scott GG, Margulies SS, Coats B. Utilizing multiple scale models to improve predictions of extra-axial hemorrhage in the immature piglet. *Biomech Model Mechanobiol*. 2016;15:1101-1119.
65. Chang HS, Nagai A, Oya S, Matsui T. Dorsal spinal arachnoid web diagnosed with the quantitative measurement of cerebrospinal fluid flow on magnetic resonance imaging. *J Neurosurg Spine*. 2014;20:227-233.
66. Stockman HW. Effect of anatomical fine structure on the flow of cerebrospinal fluid in the spinal subarachnoid space. *J Biomech Eng*. 2006; 128:106-114.
67. Tangen KM, Hsu Y, Zhu DC, Linninger AA. CNS wide simulation of flow resistance and drug transport due to spinal microanatomy. *J Biomech*. 2015;48:2144-2154.
68. Gupta S, Soellinger M, Grzybowski DM, Boesiger P, Biddiscombe J, Poulikakos D, et al. Cerebrospinal fluid dynamics in the human cranial subarachnoid space: an overlooked mediator of cerebral disease. I. Computational model. *J R Soc Interf*. 2010;7:1195-1204.
69. Gupta S, Soellinger M, Boesiger P, Poulikakos D, Kurtcuoglu V. Three-dimensional computational modeling of subject-specific cerebrospinal fluid flow in the subarachnoid space. *J Biomech Eng*. 2009;131:021010.
70. Kasuya H, Shimizu T, Okada T, Takahashi K, Summerville T, Kitamura K. Activation of the coagulation system in the subarachnoid space after subarachnoid haemorrhage: serial measurement of fibrinopeptide a and bradykinin of cerebrospinal fluid and plasma in patients with subarachnoid haemorrhage. *Acta Neurochir (Wien)*. 1988;91:120-125.
71. Kasuya H, Shimizu T, Okada T, Takahashi K, Summerville T, Kitamura K. Significance of trabeculae in subarachnoid hemorrhage. Measurement of bradykinin, fibrinopeptide A, and thromboxane B<sub>2</sub> in cerebrospinal fluid. *Neurol Med Chir (Tokyo)*. 1988;28:880-885.
72. De Bonis P, Trevisi G, de Waure C, Sferrazza A, Volpe M, Pompucci A, et al. Antiplatelet/anticoagulant agents and chronic subdural hematoma in the elderly. *PLoS One*. 2013;8:e68732.
73. Sim YW, Min KS, Lee MS, Kim YG, Kim DH. Recent changes in risk factors of chronic subdural hematoma. *J Korean Neurosurg Soc*. 2012;52: 234-239.
74. Yadav YR, Parihar V, Namdev H, Bajaj J. Chronic subdural hematoma. *Asian J Neurosurg*. 2016;11: 330-342.
75. Lee K-S. Chronic subdural hematoma in the aged, trauma or degeneration? *J Korean Neurosurg Soc*. 2016;59:1-5.
76. Matsumoto S, Tamaki N. Subdural hydromas. In: McLaurin RL, ed. *Extracerebral Collections. Advances in Neurotraumatology*. Vol. 1. Vienna: Springer; 1986. Published under the Auspices of the Neurotraumatology Committee of the World Federation of Neurosurgical Societies.
77. Aarabi B, Chesler D, Maulucci C, Blacklock T, Alexander M. Dynamics of subdural hygroma following decompressive craniectomy: a comparative study. *Neurosurg Focus*. 2009;26:E8.
78. Beck J, Gralla J, Fung C, Ulrich CT, Schucht P, Fichtner J, et al. Spinal cerebrospinal fluid leak as the cause of chronic subdural hematomas in nongeriatric patients. *J Neurosurg*. 2014;121: 1380-1387.
79. Bosche B, Molcanyi M, Noll T, Kochanek M, Kraus B, Rieger B, et al. Occurrence and recurrence of spontaneous chronic subdural haematoma is associated with a factor XIII deficiency. *Clin Neurol Neurosurg*. 2013;115:13-18.
80. Lu J, Zhu XL. Characteristics of distribution and configuration of intracranial arachnoid membranes. *Surg Radiol Anat*. 2005;27:472-481.
81. Locke CE, Naffziger HC, Galen B. Vieussens, Ruysch, quoted by H. Hinman. The cerebral sybarachnoid system. *Arch Neurol Psychiatry*. 1924; 12:411.
82. Greenberg RW, Lane EL, Cinnamon J, Farmer P, Hyman RA. The cranial meninges: anatomic considerations. *Semin Ultrasound CT MR*. 1994;15: 454-465.
83. Scott G, Coats B. Microstructural characterization of the pia-arachnoid complex using optical coherence tomography. *IEEE Trans Med Imaging*. 2015;34:1452-1459.
84. Mancall EL, Brock DG. *Gray's Clinical Neuroanatomy*. Philadelphia, PA: Elsevier Health Sciences; 2011.
85. Lee JH. *Meningiomas: Diagnosis, Treatment, and Outcome*. London: Springer; 2008.
86. Yasargil MG, Kasdaglis K, Jain KK, Weber HP. Anatomical observation of the subarachnoid cisterns of the brain during surgery. *J Neurosurg*. 1976;44:298-302.
87. Yasargil MG, Antic J, Laciga R, Jain KK, Hodosh RM, Smith RD. Microsurgical pterional approach to aneurysms of the basilar bifurcation. *Surg Neurol*. 1976;6:83-91.
88. Vinas FC, Dujovny M, Fandino R, Chavez V. Microsurgical anatomy of the arachnoid trabecular membranes and cisterns at the level of the tentorium. *Neurol Res*. 1996;18:305-311.
89. Vinas FC, Fandino R, Dujovny M, Chavez V. Microsurgical anatomy of the supratentorial arachnoid trabecular membranes and cisterns. *Neurol Res*. 1994;16:417-424.
90. Vinas FC, Dujovny M, Fandino R, Chavez V. Microsurgical anatomy of the infratentorial trabecular membranes and sub-arachnoid cisterns. *Neurol Res*. 1996;18:117-125.
91. Patestas MA, Gartner LP. *A Textbook of Neuroanatomy*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2016.
92. Tubbs RS, Shoja MM, Loukas M. *Bergman's Comprehensive Encyclopedia of Human Anatomic Variation*. Hoboken, NJ: Wiley; 2016.
93. Krstic RV. *Human Microscopic Anatomy: An Atlas for Students of Medicine and Biology*. Berlin, Heidelberg: Springer; 2013.

94. Standring S, ed. *Gray's Anatomy: The Anatomical Basis of Clinical Practice*. Edinburgh: Churchill Livingstone/Elsevier; 2008.
95. Liliequist B. The anatomy of the subarachnoid cisterns. *Acta Radiol*. 1956;46:61-71.
96. Mortazavi MM, Rizq F, Harmon O, Adeeb N, Gorjian M, Hose N, et al. Anatomical variations and neurosurgical significance of Liliequist's membrane. *Childs Nerv Syst*. 2015;31:15-28.
97. Tandon PN, Ramamurthi R. *Textbook of Neurosurgery*. 3rd ed. New Delhi, India: Jaypee Brothers; 2012.
98. Zhang XA, Qi ST, Huang GL, Long H, Fan J, Peng JX. Anatomical and histological study of Liliequist's membrane: with emphasis on its nature and lateral attachments. *Childs Nerv Syst*. 2012;28:65-72.
99. Weller RO. Microscopic morphology and histology of the human meninges. *Morphologie*. 2005;89:22-34.
100. McLone DG. The subarachnoid space: a review. *Childs Brain*. 1980;6:113-130.
101. Park EH, Eide PK, Zurakowski D, Madsen JR. Impaired pulsation absorber mechanism in idiopathic normal pressure hydrocephalus: laboratory investigation. *J Neurosurg*. 2012;117:1189-1196.
102. Park EH, Dombrowski S, Luciano M, Zurakowski D, Madsen JR. Alterations of pulsation absorber characteristics in experimental hydrocephalus. *J Neurosurg Pediatr*. 2010;6:159-170.
103. Simon MJ, Iliff JJ. Regulation of cerebrospinal fluid (CSF) flow in neurodegenerative, neurovascular and neuroinflammatory disease. *Biochim Biophys Acta*. 2016;1862:442-451.
104. Yeo J, Cheng S, Hemley S, Lee BB, Stoodley M, Bilston L. Characteristics of CSF velocity-time profile in posttraumatic syringomyelia. *AJNR Am J Neuroradiol*. 2017;38:1839-1844.
105. Bertram CD, Heil M. A poroelastic fluid/structure-interaction model of cerebrospinal fluid dynamics in the cord with syringomyelia and adjacent subarachnoid-space stenosis. *J Biomech Eng*. 2017;139. <https://doi.org/10.1115/1.4034657>.
106. Elliott NSJ, Bertram CD, Martin BA, Brodbelt AR. Syringomyelia: a review of the biomechanics. *J Fluids Structures*. 2013;40:1-24.
107. Martin BA, Labuda R, Royston TJ, Oshinski JN, Iskandar B, Loth F. Spinal subarachnoid space pressure measurements in an in vitro spinal stenosis model: implications on syringomyelia theories. *J Biomech Eng*. 2010;132:111007.
108. Franze K, Janmey PA, Guck J. Mechanics in neuronal development and repair. *Annu Rev Biomed Eng*. 2013;15:227-251.
109. Goriely A, Geers MG, Holzapfel GA, Jayamohan J, Jerusalem A, Sivaloganathan S, et al. Mechanics of the brain: perspectives, challenges, and opportunities. *Biomech Model Mechanobiol*. 2015;14:931-965.
110. Lee L. Riding the wave of ependymal cilia: Genetic susceptibility to hydrocephalus in primary ciliary dyskinesia. *J Neurosci Res*. 2013;91:1117-1132.

*Conflict of interest statement:* M.M.M.: Haag-Streit consultant, Depuy Synthes, American Surgical Company; S.A.F.: SBMT-Haag-Streit Skull Base fellowship grant; M.A.T.: Stryker Neurovascular consultant; B.A.M.: Research Funding from Alcyone Lifesciences and Voyager Therapeutics.

Received 6 October 2017; accepted 8 December 2017

Citation: *World Neurosurg*. (2018) 111:279-290.  
<https://doi.org/10.1016/j.wneu.2017.12.041>

Journal homepage: [www.WORLDNEUROSURGERY.org](http://www.WORLDNEUROSURGERY.org)

Available online: [www.sciencedirect.com](http://www.sciencedirect.com)

1878-8750/\$ - see front matter © 2017 Elsevier Inc. All rights reserved.