REVIEW

The central role of bridging veins in the pathogenesis of chronic subdural hematoma

El rol central de las venas puente en la patogénesis del hematoma subdural crónico

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ABSTRACT

Background: The prevalence of chronic subdural hematoma continues to increase and is expected to become the most common neurosurgical condition by 2030. Its origin has been associated with the rupture of bridging veins into the subdural space. **Method:** This review aims to systematise knowledge about the bridging veins to the superior sagittal sinus through a narrative literature review of articles in English and Spanish from 2012 to 2025. **Results:** The bridging veins to the superior sagittal sinus exhibit great anatomical and morphological variability. Three types of anatomical configurations are identified; long and tortuous veins rupture more easily, especially when they enter at a right angle. **Conclusion:** Physiological aging causes brain atrophy and loss of vein elasticity, predisposing to rupture due to multifactorial causes. They have a diverse angioarchitecture and play a central role in the origin of spontaneous and traumatic chronic subdural hematomas.

Keywords: Bridging veins; Venous sinus; Superior sagittal sinus; Chronic subdural hematoma

RESUMEN

Introducción: La prevalencia del hematoma subdural crónico continua en aumento y se convertirá en la condición neuroquirúrgica más frecuente para el 2030. El origen se ha relacionado con la ruptura de las venas puente al espacio subdural. **Método:** Esta revisión tiene el objetivo de sistematizar los conocimientos sobre las venas puente al seno sagital superior. A través de una revisión bibliográfica narrativa de artículos en idioma inglés y español del 2012 al 2025. **Resultados:** Las venas puente al seno sagital superior muestran una gran variabilidad anatómica y morfológica. Se identifican tres tipos de configuraciones anatómicas, las venas largas y tortuosas rompen con más facilidad, sobre todo cuando entran en ángulo recto. **Conclusiones:** El envejecimiento fisiológico causa atrofia cerebral y pérdida de la elasticidad de las venas, lo que predispone a la ruptura de causa multifactorial. Tienen una angioarquitectura diversa y juegan el rol protagónico en el origen del hematoma subdural crónico espontáneo y traumático.

Palabras clave: Venas puente; Senos venosos; Seno sagital superior; Hematoma subdural crónico

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INTRODUCTION

Chronic subdural hematoma (CSDH) is a collection of venous blood between the dura mater and the arachnoid, covering the surface of one or both hemispheres.[1] The prevalence of CSDH continues to rise and is estimated to become the most common neurosurgical condition by 2030. Its origin has historically been associated with mild head trauma, which causes rupture of the bridging veins (BVs) into the subdural space. Older adults are more susceptible to this type of hematoma due to cerebral atrophy, changes in inflammatory response, and angiogenesis associated with physiological aging.[2-4]

Wei *et al.*[5] characterize it as a prevalent neurosurgical condition in the geriatric population, with an annual incidence of 1 to 13 per 100,000 inhabitants. An increase in overall frequency is documented due to the use of antithrombotic therapies and trauma mechanisms related to aging.[6-8]

To date, the pathophysiology involved in CSDH is controversial, and several theories have been proposed. Originally, it was assumed that initial traumatic bleeding was caused by tears in the intracranial bridging veins, which led to additional fluid absorption in the subdural space through osmosis and thus to liquefaction and progression in size of the hematoma. However, current studies show a much more complicated pathogenesis.[9] It is characterized by a delayed presentation that often occurs weeks to months historically linked to a minor, sometimes unnoticed, head injury, with growing evidence suggesting that ongoing inflammation, cerebral atrophy, and microvascular fragility are central pathophysiological drivers.[10]

In line with the above, Estrella López *et al.*[11] report an incidence of 58 per 100,000 inhabitants in people over 70 years old, with mortality rates of 4% and morbidity of 11%. These authors summarize its origin following a mild cranial trauma that causes the rupture of the bridging veins with a slow, low-pressure bleed into the subdural space.[12,13] This, in turn, triggers a local inflammatory response characterized by: proliferation of granulation tissue, development of fibrous membranes to form the hematoma, and angiogenesis with the formation of immature vessels. These neovessels are prone to rupture, which explains recurrent bleeding that perpetuates the inflammatory cascade.[14]

The elements provided justify the organization of the clot and the cavity limited by the external and internal membranes. The first contains many sinusoidal vessels that are fragile macrocapillaries (neovascularization) that bleed easily in a multifocal manner. These repeated bleedings, originating from the outer membrane, are considered the most important factor in the progressive increase in hematoma, which can occur after minor trauma.[15] However, the liquefaction of the clot is attributed to the internal membrane, which has variable architecture, which can be homogeneous or heterogeneous, which in turn can be multiseptate, calcified, multilobulated or have multiple layers, to form the organized or mature HSC. It is considered an inflammatory and angiogenic condition, with complex management for general medicine and neurosurgery; Therefore, its timely diagnosis and treatment improve the health status of patients.[16-19]

CSDH can be atraumatic, thus it has been documented as a late complication of intracranial hypotension, which causes spontaneous rupture of bridging veins. However, some evidence has also shown the formation in patients aged 69 years or older, as a consequence of spontaneous intracranial hypotension. Furthermore, it may arise from a prolonged interaction between the dural layers and CSF leakage into the pachymeninges, leading to hemorrhagic transformation of a hygroma or slow organization of subdural blood.[20]

HSDC is one of the most common neurological disorders and manifests as a spectrum of neurological symptoms ranging from progressive motor deficit and cognitive decline to deterioration of consciousness.[21,22] The diagnosis is made using computed axial tomography. However, radiomics emerges as a field that integrates artificial intelligence with imaging to improve diagnostic accuracy, prognostic prediction, including hematoma expansion and recurrences.[23] Although there are different treatment options with good outcomes, there is no consensus or established standards. The usual treatment is surgical evacuation through trepanation. [24-26]

The **objective** of this review is to systematize the knowledge about VBs to the superior sagittal sinus (SSS) due to its importance in the origin of CSDH.

METHODS

A narrative literature review was conducted of various articles on BVs and CSDH, with an emphasis on those published in the last decade, in order to descriptively synthesize and analyze anatomical, physiological, and biomechanical concepts that link the spontaneous or traumatic injury of the BVs to the SSS with the development of CSDH.

Search period

Articles in English and Spanish from 2012 to 2025 were included. The search was conducted in search engines and databases such as PubMed, Scopus, SciELO, Web of Science, and Google Scholar. The main descriptors in Spanish related to the topic were used: bridging veins, venous drainage system of the brain, biomechanics of the BVs, chronic subdural hematoma, cerebral atrophy. The English terms employed were: bridging veins, superior sagittal sinus, venous drainage of brain, chronic subdural hematoma, cortical atrophy, biomechanical properties.

Article selection criteria

Studies that presented meta-analyses, systematic reviews, explicit methodological description, updated information, images from anatomical studies, cadaver dissections, experimental research, and comparisons of results from different investigations were prioritized.

Types of articles

Thirty-three articles were selected. Of these, 15 were review articles (including three meta-analyses); these were evaluated using the SANRA scale (Scale for the Assessment of Narrative Review Articles), and four of the reviews received the highest score while the rest were of acceptable quality. Additionally, 13 original articles, four case reports, and one monograph were accepted.

The literature review made it possible to identify the morphophysiological variants of the VP, the effects that physiological aging has on them and the brain, and the particularities of rupture biomechanics. These topics allowed for discussing the importance of the BVs in the SSS, in the formation of the CSDH, which have been documented individually in the articles published on the subject.

RESULTS

Conceptualization and background of bridging veins to the SSS

BVs were first mentioned in the scientific literature due to their association with subdural hematomas (SDHs). Trotter, cited by Mortazavi *et al.*[27] was the pioneer in attributing BV rupture as a possible cause of SDH. These authors also point out that in 1934, John Leary concluded that most subdural hemorrhages are the result of the rupture of BVs crossing the subdural space or of veins on the arachnoid surface, a statement that was supported by Cabot in 1940.

The BVs are connecting vessels that drain venous blood flow from the surface of the brain around the venous sinuses, mainly from the SSS and the transverse sinus.[28] During intrauterine life, the anatomy of the cerebral venous system changes in response to morphometric and hemodynamic adaptations of the growing organism.[29,30] Around 12 weeks of intrauterine life, 10 to 18 BVs can already be found. Their walls are thin and comprise two portions: the subdural portion measuring 10 to 600 microns in length and the subarachnoid portion measuring 50 to 200 microns.[31]

Studies of cerebral angiographies have shown the variable angioarchitecture of the venous and dural system. Accessory or tributary venous sinuses have been found, located in the dural convexity, with a flattened morphology. They can be very long or short, and the BVs may apparently terminate in them, outside the sinus. In addition, BVs almost entirely covered by dura mater were observed, and BVs with an intradural course were appreciated.[32]

Regarding the subdural course, Migueiss *et al.*[33] emphasize that the passage through the subdural space provides no structural support to the BVs, making them predisposed to rupture, especially from longitudinal stress or rotational acceleration caused by the relative movement of the brain within the skull. The authors identify that age, sex, and medical history influence the highlighted variability of the BVs. These aspects will be discussed below.

Morphofunctional variability of the BVs

Compared to arteries, the veins of the brain exhibit greater individual morphofunctional variability during development, and most PAs (45%) originate from the cerebral hemispheres. Many anatomical variants of the superior cortical venous system are described in terms of number, size, symmetry between hemispheres, and drainage patterns.[34]

Variability in number

The average number of BVs at the SSS ranges from 11 to 45, with a predominance in the anterior frontal region, and the confluence angles vary around 50 degrees, with significant changes in diameter (decrease) related to age.[35,36]

Variability in topography

According to their location, three types of corticofrontal anterior, temporal, and cerebellar BVs have been studied. The frontal ones are associated with CSDH and have three drainage areas: lateral convexity, medial surface, and lateral surface.[27]

Morphological variability

Most superior sagittal sinus veins drain distal to the coronal suture, and three types of anatomical configurations are described: Type I, which is a single drainage trunk to the SSS, represents about 68% with a diameter of 1.9 mm and a length of 30.5 mm. Type II, in which two or more contiguous veins join at the same point (candelabra-shaped), account for a little over 32% with an average diameter of 3.2 mm and a length of 32.5 mm. Type III, in which several BVs join (arboriform or plexiform shape) to drain two or more gyri. Anterior to the coronal suture, the most frequent configuration is Type I in 57% of hemispheres, according to Karatas *et al.*[37]

Between the coronal suture and the postcentral sulcus, they tend to be longer and more numerous; most BVs first drain into a venous lake, with Type II and III predominating (Fig. 1).

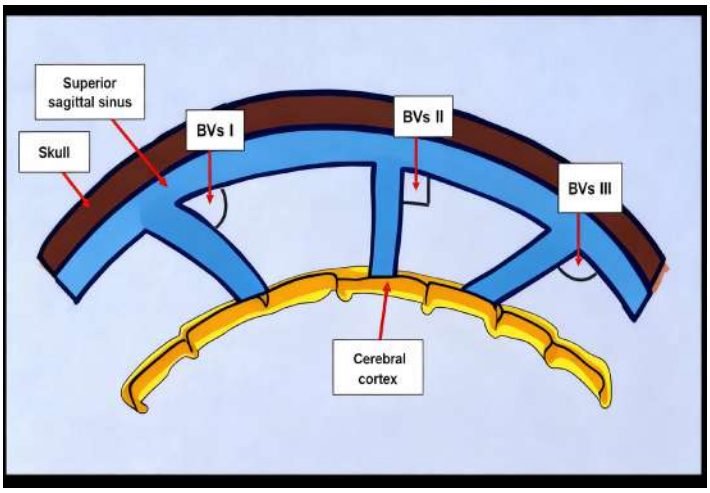

Figure 1. Anatomical configuration of the BVs entrance into the SSS. Karakatas *et al.*[37] and Baltasavias *et al.*[32]
Source: Author's personal drawing.

The BVs wall consists of an outer sheet or layer of collagen fibers with a lumen lined by endothelial cells and a variable number of fine elastic fibers within the vein wall. In addition, a complete elastic layer has been observed near the endothelial layer, which has been associated with the potential for stretching and recovery after deformation.[38]

Variability of the BVs lumen at its opening into the SSS: Another classification proposed by Ye *et al.*[39] relates morphology to anatomy, taking into account that the orientation of collagen fibers changes at the level of the venous opening, and the lumen diameter becomes narrower, identifying three groups (Table 1, Fig. 2).

The development of brain tissue, venous sinuses, and the skull determines variations in the configuration of the BVs, in the drainage routes, and in the entry to the SSS (Fig. 2). Shapiro *et al.*[40] note that there is an abrupt transition between the rounded lumen of the cortical vein and the biconcave morphology of the dural sinus. They cite an investigation, who described 11 BVs draining into the SSS on each side, highlighting that venous lakes do not receive BVs directly; however, some drain into meningeal veins before entering the venous sinus. They also describe that the diameter of the BVs increases just before opening into the venous sinus. At this point, the collagen fibers have a helical orientation, and the myoendothelial cells are organized like a sphincter in the longitudinal direction.[32,41] This point has been referred to as the outflow cuff with an average diameter of 2.04 mm and an area under normal pressure of 3.3 mm^2.

Variability in the length of the BVs: Variations in the length and outlet of the BVs have been associated with the risk of rupture. In this regard, Famey N *et al.*[42] describe three types of BVs, which are shown in Table 2 and Fig. 3.

Table 1. Anatomical and morphological characterization of the BVs at their opening into the venous sinuses.

Type	Anatomical characteristics	Morphological characteristics
A	Direct connection: the bridging vein enters the SSS directly.	The venous wall is connected to the dura mater due to the loss of connective tissue. However, there is dense connective tissue attached to the SSS.
B	The BVs travels a certain distance along the dural wall. The opening of the vein is trabecular.	The wall of the BVs has a strong connection with the SSS, and collagen fibers are arranged in different layers.
C	The BVs runs a certain distance along the dural wall. Arachnoid granulations are present at the opening of the vein.	Endothelial cells and collagen fibers around the venous wall.

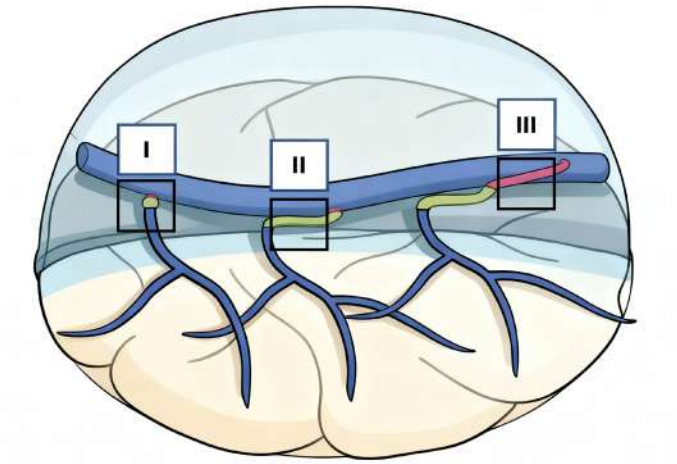

Figure 2. Graphical representation of the opening morphology of the BVs to the dural sinus.
Source: Taken from Ye *et al.*[39] modified by the author.

Table 2. Classification of BVs according to length, shape, and risk of rupture.

Type of BVs	Length and shape	Risk of rupture
I	Short and straight (30 %)	Lower risk
II	Long and winding (50 %)	High risk in brain injurie
III	Redundant or in S (20 %)	Associated with arteriovenous malformations
IV	Plexiform	

Source: Famey *et al.*[42]

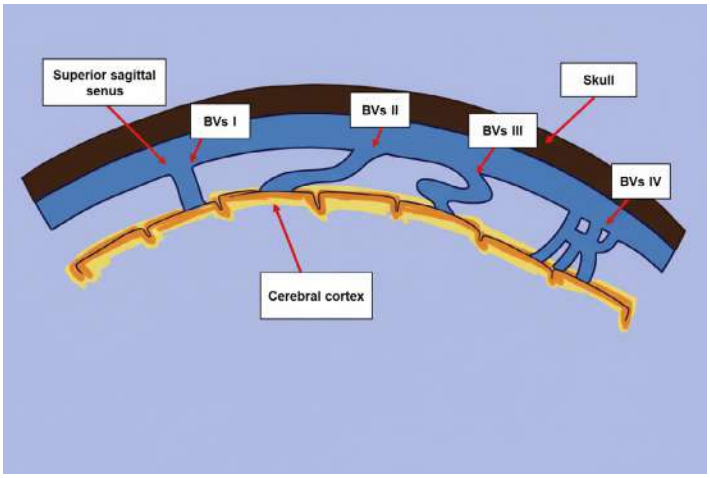

Figure 3. Morphology of BVs entry into the SSS according to Famey *et al.*[42]
Source: Author's own drawing.

Neuroimaging techniques and cadaveric dissection have facilitated the study of the anatomical location, size, and number of BVs in adults. During anatomical dissections of these structures, the continuity between the arachnoid and the dura near the SSS has been described as a kind of arachnoid sheet or meningothelial structure; the presence of supporting fibrous tissue around the vein has also been detailed. The latter has not been identified in children, which increases susceptibility to rupture, potentially causing acute SDH secondary to cranial trauma in shaken baby syndrome.[43] Likewise, variations in the diameter with dilation of the bridging veins have been identified in diseases such as multiple sclerosis.[41]

Variability of the angle of BVs drainage into the SSS: The importance of the angle at which the BVs drains is also addressed and described. Famey *et al.*[42] and Zhu[43] demonstrated that angles greater than 80° independently increase the risk, regardless of length (Table 3, Fig. 4).

In general, veins that are more tortuous, long, and thin, entering at a right angle, are more fragile and susceptible to rupture from traction mechanisms, especially if they are located in the frontal region.[44, 45]

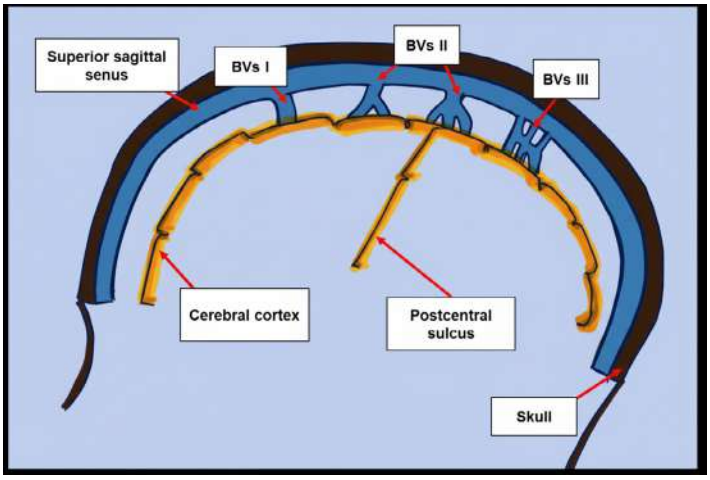

Figure 4. Angle of entry of the BVs into the SSS according to Famey N, *et al.*[42]
Source: Author's own drawing.

Table 3. Relationship of the BVs insertion angle to the SSS and the risk of rupture.

Outlet into the dural sinuses	Frequency	Insertion angle	Fracture mechanics	Risk
Superior sagittal sinus	70-80 % of the BVs	Perpendicular (90 degrees)	Greater tension due to axial traction stretches the vein in the direction opposite to its fixed insertion in the SSS	High risk
Transverse sinus	15- 20 % of the cases	Oblique (45 - 60 degrees)	Shear stress combined with torsion. Lower direct stress but greater tortuosity in its path	Moderate risk
Right sinus	Less than 10 %	Acute (less than 30 degrees)	Distributed forces. Lower mechanical stress. Less prone to traumatic ruptures, but associated with spontaneous SDH in patients with intracranial hypertension.	Low risk

Source: Famey N, *et al*[42] and Zhu Y[43]

Zhu *et al.*[43] studied 137 BVs and divided them into two groups:

- Anterior group: 62 BVs, with approximate diameters of 2 mm +/- 9 mm and an entry angle of 93 ± 34 degrees.
- Posterior group: 75 BVs with average diameters of 3 mm +/- 1.1 mm and an entry angle of 43 ± 25 degrees. These were the largest in diameter and therefore had a smaller entry angle.

These authors highlighted that the wall shear stress acts on vascular endothelial cells, is parallel to the longitudinal axis of the vessel, and is formed by the friction between cerebral blood flow (CBF) and the fixed vascular wall, remaining relatively stable. Folgueira and Acuña[44] specify that the veins draining into the anterior part of the SSS do so in the same direction as the CBF; in contrast, those that open into the posterior part do so against the flow.

Predisposing factors for BVs rupture

Information on the influence of age on BVs is limited. It is considered to have a significant negative effect on mechanical properties such as strength, elasticity, and distensibility. A deterioration of the veins' mechanisms for responding to stretching and distortion is described, along with a weakening of their mechanical response capacity. With physiological aging, there is a degeneration of elastin fibers and an increase in deposits of rigid collagen in the venous wall (phlebosclerosis), which reduces their distension capacity. It is also accompanied by sclerosis of the lumen, which may partially obstruct flow, and atrophy of the endothelial layers with a decrease in structural proteins such as type IV collagen, increasing the risk of spontaneous rupture.[45]

Cerebral atrophy and the increase in subdural space are secondary to progressive aging. The decrease in brain volume is accompanied, compensatorily, by an increase in the amount of subdural cerebrospinal fluid, maximum stretching of the BVs, and an increase in the range of brain movement within the cranial cavity, which is referred to as cortical movement (CM). The relative maximum CM has been estimated in the posterior frontal and precentral regions, ahead of the site where BVs most frequently rupture. This discrepancy between the two areas may be explained by their anatomical variability. The BVs in the anterior parietal region are shorter and more aligned with the distribution of loads. In general, subdural hematomas are more frequent in the frontal and parietal regions, corresponding to the site of maximum relative CM.[45,46]

According to Familiari *et al.*[47] different theories explain how cerebral atrophy promotes the spontaneous or traumatic formation of CSDH. In the first theory, atrophy causes alterations in fluid dynamics and pressure balance between the subdural and subarachnoid spaces, which are not entirely clarified. These hemodynamic changes lead to the redistribution of cerebrospinal fluid with dilation of the overall ventricular system and subarachnoid space. The pressure within the latter can facilitate communication with the subdural space and give rise to the hygroma, considered the first stage of CSDH, which initiates the local inflammatory cascade (Fig. 5).

These same authors describe the second theory in which atrophy causes mechanical distension of the venous plexuses (VP), which acts as the mechanical stimulus that initiates a chemical response. The elongation of the VP promotes the release of nitric oxide and other vasodilators by the endothelial cells of the venous wall, causes the relaxation of the smooth muscle cells arranged within the vessel wall, thereby increasing its lumen and causing transendothelial cellular filtration. In this way, the inflammatory cascade begins with angiogenesis for the formation of HSDC.[47]

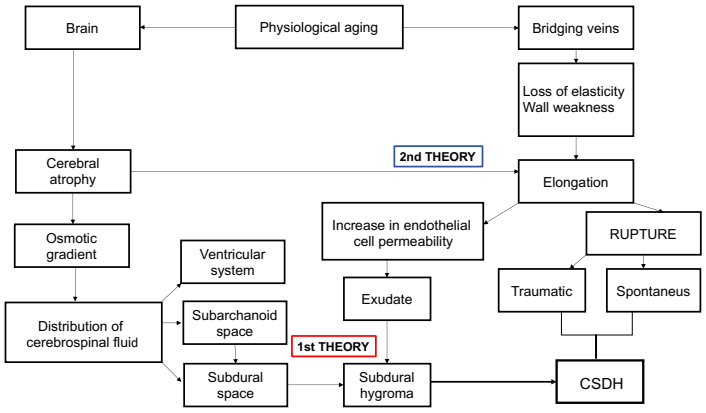

Figure 5. Theories related to the *pathophysiology* of cerebral atrophy in the formation of HSDC.
Source: Elaborated by the author based on Familiari *et al.*[47]

Shapiro *et al.*[40] describe that in 1965, Rowbotham and Little identified two types of CSDH. One is secondary to traumatic rupture of the BVs as they cross the subdural space to reach the superior sagittal sinus, which they attributed a good prognosis to after surgical evacuation. The second occurs without a history of trauma, due to a minimal and recurrent exudation or bleeding from the internal dural plexus, observed in degenerative changes within brain tissue. The latter have a poorer response to surgical drainage but a better prognosis after embolization.

Imaging studies show that the parietal region, especially at the level of the parietal eminence, is the most frequent location of HSDC, followed by the frontal region. This is related to the increase in the curvature of the cranial vault and the decrease in its radius. The dilation of the cortical arachnoid cisterns due to atrophy has a particular effect of low surface tension in this area, which facilitates the passage of fluids from the subarachnoid space to the subdural space.47,48

Biomechanics and BVs rupture

When a subject traveling at a certain speed is abruptly stopped and the head collides with a solid structure, the skull stops but the brain, due to its elasticity and the inertia this condition imparts, continues moving at the previous speed, striking the inside of the cranial vault. Since most of these accidents occur in a frontal position, the anterior poles of the frontal and temporal lobes are the most affected by the contusion. Another effect that sudden deceleration can cause by the same mechanism is the rupture of BVs, especially those connected to the SSS. This trauma mechanism produces intracranial pressure gradients that subject the brain to traction and shear forces, resulting in two typical injuries: subdural hematoma and diffuse axonal damage. Then the nature of the injuries will depend on the type and magnitude of the acceleration, the direction of the head movement, and the duration of the acceleration change, which can be linear, rotational, or angular.[49]

Gavrila *et al*[49] point out that the vulnerability of the BVs is related to the mechanical properties of the vessel and depends on the structure and geometry of its wall. They highlight the great biological variability of the histological, mechanical, and morphological properties of the BVs. In their study, they found a total of 832 BVs in the right hemispheres and 749 in the left, with no difference in the average diameter. There was a predominance in the parietal lobes, with 977 BVs, frontal lobes with 547 BVs, and occipital lobes accounting 57. The average per hemisphere was 11.59 (1-30) BVs and per brain 23.18 (2-48) BVs.

The authors[49] noted that the regional distribution across the lobes presented an interesting finding. In the parietal lobe there were one to two BV with greater visibility in the Rolandic and Prerolandic areas, followed by the frontal and occipital lobes with one BV per region. The diameter ranged from 0.37 to 3.24 mm, up to 5.8 mm, with no significant differences between the hemispheres but differences between the lobes. Thus, in the parietal lobe, the diameter measured near the SSS was 1.42 mm, in the frontal lobe it was 1.28 mm, and in the occipital lobe it was 1.2 mm. They concluded that the parietal lobe has more BVs and with a larger caliber, especially near the frontal lobe, which is a critical anatomical area for BV rupture.

Monea G *et al.*[50] indicate that in the genesis of SDH, the fundamental cause is the sudden longitudinal stretching of the subdural part of the BVs. Most BVs in the SSS have a frontal course before ending in the sinus, making them more vulnerable to occipital trauma. These authors highlight the contribution made by Delye *et al.* in 2001, who suggested that the junction of the BVs with the SSS played an important role in the biomechanical response at low strain ranges. Similarly, they considered the subdural segment of the BVs as a zone of weakness.

CONCLUSIONS

The BVs to the SSS exhibit great anatomical variability. The diameter, length, and characteristics of the subdural course, as well as the shape and entry angle to the SSS, are determining factors in biomechanics. It explains the susceptibility to rupture from minimal trauma secondary to linear and angular acceleration mechanisms. Physiological aging causes brain atrophy and loss of elasticity of the BVs; both factors combined, predispose to the formation of spontaneous and traumatic CSDH. In both cases, the common factor is the cascade of local inflammatory response, which leads to fibrinolysis and angiogenesis responsible for the formation of membranes and the perpetuation of CSDH. These elements help document the key role of BVs in the origin of CSDH, regardless of the cause.

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Conflict of interest

None

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