

It's a Stitch-Up: The Function of Subarachnoid Trabeculae

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Abstract

The Shaken Baby Hypothesis assumes the brain can slide in the skull, but trabeculae (thin strips of collagen reinforced tissue that link across the subarachnoid space) appear to prevent this. They are so thin that they are undetectable by ultrasound or MRI imaging systems. Pediatricians in the 1970s were unaware of their existence. The Shaken Baby Syndrome hypothesis ignores them. The purpose of this study was to investigate whether omitting the trabecular structures from the SBS hypothesis would have made any difference to the legal validity of cases based on it.

When the Shaken Baby Syndrome concept was created in the 1970s it was believed that there was a layer of fluid between the Dura and Arachnoid Maters (a "Subdural Space") which allowed the brain cortex to slide. Electron microscopy has since shown that the Subdural Space does not exist.

Electron microscopy has shown a "Cob-Web" of tissue (Trabeculae) linking the Arachnoid and Pia Maters. There is no subarachnoid space in the "Shaken Baby" model so no subarachnoid trabeculae are considered. Collagen fibres within these subarachnoid trabeculae are continuous with bundles of collagen in the inner aspect of the arachnoid and with collagen bundles in the subpial space, effectively "stitching" the pia and arachnoid membranes together.

By implanting radio-opaque markers in cadaver heads and using high speed X-ray recorders vehicle accident researchers established that fresh brain is softly elastic. Under impact various regions move over different loci at different speeds. This trabecular stitching causes the cerebral cortex surface to follow skull movement closely. The cortex cannot slide, it reversibly deforms to take up movements. Therefore the bridging veins cannot be strained as was assumed in the 1970s and the Shaken Baby Syndrome hypothesis is invalid.

Keywords: Cortex; Macrocephaly; Shaken Baby; Subarachnoid; Subdural; Venous hypertension

Introduction

Present theories of Shaken Baby syndrome assume the brain can slide around under the skull. This article examines whether this assumption is justified. Injuries attributed to this relative movement are known as the triad, subdural bleeding, retinal bleeding, and altered mental function. If present, carers are considered guilty of causing injury or death of an infant by shaking. The forensic examination concentrates on establishing who did the shaking and was it the result of stress on the carer. Many have queried the degree of shaking required to produce the injuries and whether an impact would be needed to produce sufficient brain/skull movement to tear the bridging veins [1,2]. This article examines the fundamental factors governing whether such relative movement can occur in the first place.

The Subdural Space: Subdural or Subarachnoid?

The Arachnoid membrane is fairly tough but very thin. During dissection surface tension causes it to cling tightly to the brain surface. Before the extensive study of the meninges with the electron

microscope it was assumed that it was actually attached to the cortex (Figure 1a). It was also believed that between the dura and the arachnoid there was a fluid filled space, the subdural space. In 1991, Haines carried out a literature search of opinions of the nature of this subdural space [3]. Out of 42 articles the majority (36/42) of textbooks and atlases in gross anatomy, histology, and neuroscience, stated categorically that a subdural space was present between the arachnoid and dura. In a minority of this sample (6/42) the probable existence of a subdural space was clearly qualified or specifically denied. Haines quotes Romanes that "*The arachnoid is separated from the dura mater by a bursa-like, capillary space (the subdural space) containing a film of fluid. This forms a sliding plane where movement is possible*". Clearly in 1991 opinion of a very large majority of neurologists in various fields was that there was fluid space between the dura and the arachnoid mater. Today this concept still forms the basis of the "Shaken Baby Syndrome" (Figure 1b) (Drawn from Understanding *Shaken Baby Syndrome*) [4] (Figure 2).

However, Electron Micrograph studies revealed that there was no subdural space, and therefore no opportunity for sliding at this position (Figure 1b). There is a fluid filled space, but that is between the arachnoid and pia maters, the subarachnoid space.

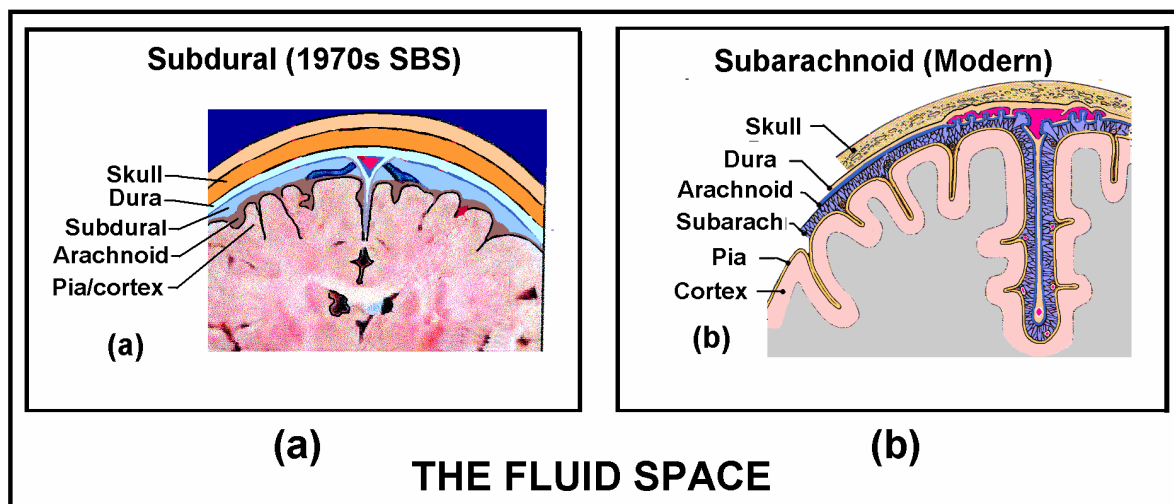


Figure 1: The fluid spaces. In the primitive model (a) (current when the SBS concept was created and in modern SBS literature), lies between Dura and Arachnoid. In the current model (b) the fluid space lies between arachnoid and pia maters.

Whereas fluid in the subdural space was believed to be static (Bursa-like), cerebrospinal fluid (CSF) is known to drain through the subarachnoid space. This is significant, because in the embryo the Arachnoid and Pia maters develop from a common layer which effectively partially splits, leaving tissue (trabeculae) connecting them [5]. In contrast the dura and the arachnoid develop individually, only the bridging veins connecting them. There is no mention of the subarachnoid trabeculae in the SBS defining documents.

With the development of electron microscopic techniques it became clear that a subdural space (between the dura and arachnoid) did not exist. In 1993, Haines summarized the situation, as “Fibroblasts specialized to form the arachnoid trabeculae attach to the inner surface of the arachnoid layer, bridge the subarachnoid space, and surround vessels in the subarachnoid spaces as well as attach to pia on the surface of the brain [6]. Under normal conditions, there is no evidence of a naturally occurring space being extant at the dura-arachnoid junction. A space may appear at this point subsequent to pathological/traumatic processes that result in tissue damage with a cleaving opening of the structurally weakest plane in the meninges-through the dural border cell layer. Furthermore, when a space does appear, it is not “subdural” in location but rather within a morphologically distinct cell layer”

Trabeculae, being undetectable by ultrasound or MRI imaging systems, were unknown to pediatricians in the 1970s [7,8]. The present brain damage component of the Shaken Baby Syndrome hypothesis is based on this 1970s concept with no mention of trabeculae. However, pathologists have long been familiar with trabeculae. Examples are, Gray [9] in 1973, Haines [6] in 1993, Alcolado [10] in 1988, Killer [11] in 2003, Weller [12] in 2005, Agesti [13] in 2010, Moore [14] in 2006. Unfortunately, neither pediatricians or pathologists were aware of the significance of their knowledge to the other. The purpose of this study was to investigate whether omitting the trabecular structures from the SBS hypothesis would have made any difference to the legal validity of cases based on it [15].

Trabecular Studies

Although in medical textbooks the trabeculae are typically represented by straight lines the structure is more like a cob web [16]. In fact the arachnoid was given its name, from the Greek for spider, by the pathologist Gerardus Blasius in the 17th century [17]. Scanning electron microscopy (SEM) revealed that the cobweb was made up of sheets and filaments of random orientation in Figure 2.

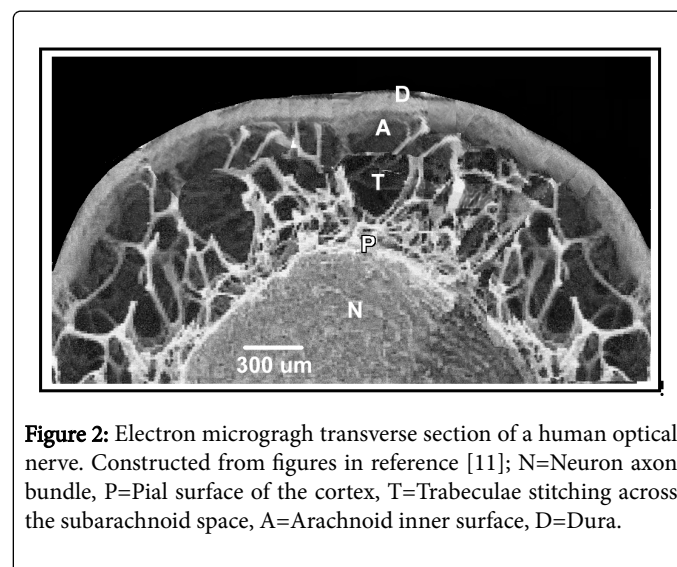


Figure 2: Electron micrograph transverse section of a human optical nerve. Constructed from figures in reference [11]; N=Neuron axon bundle, P=Pial surface of the cortex, T=Trabeculae stitching across the subarachnoid space, A=Arachnoid inner surface, D=Dura.

Optic nerves are not really single nerves, they are outgrowths of the brain, one for each eye containing up to 1.7 million axons of retinal ganglion cells [18]. They are surrounded by meninges like the rest of the brain. Alcolado found that the collagen fibres running in trabeculae were continuous with those of the inner aspect of the arachnoid and with those in the subpial space [10]. Thus the subarachnoid trabeculae effectively “stitch” the pia and arachnoid membranes together. They also locate and support blood vessels passing through them (Figure 3).

Trabecular Structure, Collagen Connections, Lengths etc

The question then arises, “are these trabeculae strong enough to materially influence brain behaviour under shaking stress?”

As can be seen in Figure 2, trabeculae are not simple ties across the subarachnoid space. They are a honeycomb-like in structure, so individual wall lengths have little meaning. However Frankel et al. obtained an equivalent length [19]. They measured the distance from the inner table of the calvarium to the subadjacent cortical gyrus in newborn infants. They found the range of widths was from 0-3.3 mm (1.6 ± 0.8 mm). Thus the equivalent trabecular length might be considered to be 1.6 mm. Hence the maximum movement the trabeculae could allow would not exceed about ± 1.6 mm.

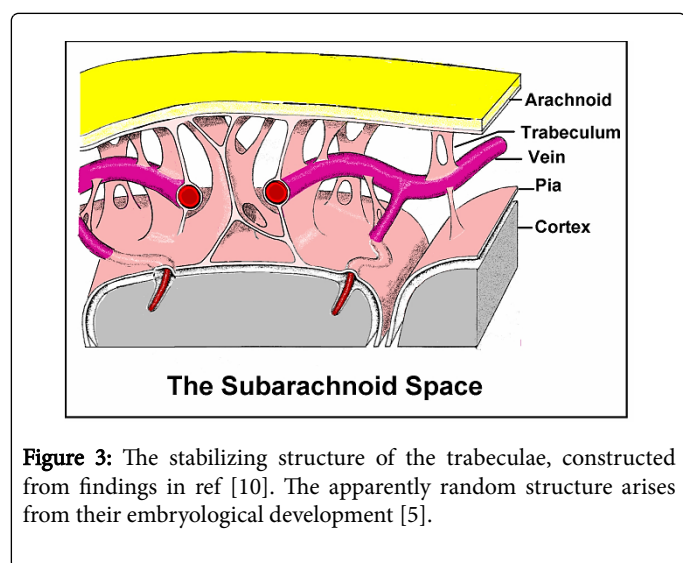


Figure 3: The stabilizing structure of the trabeculae, constructed from findings in ref [10]. The apparently random structure arises from their embryological development [5].

Individual trabeculae are weak. They are typically only a few tens of microns thick but they are very numerous [11]. The situation is similar to that of the clothing attachments made of “Velcro”. There, the individual hooks are feeble but when they all are attached their combined effect is powerful. To undo them one does not attempt to pull them all off at once, one peels them off sequentially. Individually trabeculae are weak, but combined in the intact brain, they are potentially strong. However during dissection they easily get peeled away and for a long time no-one realised they were there.

Brain Elastic Properties

In discussions of SBS the brain is often spoken of in terms of the “preserved” version seen in lectures or dissections. The preservatives used considerably stiffen the brain, the in vivo brain is considerably softer. Cormack describes it as “mushy”, “The CNS is virtually unsupported by connective tissue [20]. Instead the neuronal cell bodies and processes obtain support from other kinds of cells that are similarly derived from neuroectodermal cells of the neural tube. These non-neuronal constituents of the CNS are called neuroglia or glial cells. The cell bodies of neurons and their various associated glial cells are the principle components of gray matter. The cell bodies of neurons lie surrounded by tangled masses of fibres that represent the beginnings and endings of nerve fibres. Due to the matted appearance in the light microscope this component of gray matter is called neuropil (Gr. Pilos=felt)”. So, unlike most body tissues there is no

supportive tissue. Astrocytes cross-link neuron axons and fine blood vessels to provide some stability but the structure is more like cooked spaghetti than a sponge.

Brain Movement under Sub-Traumatic Impact

King et al. studied brain motion resulting from blunt sub-traumatic impact in cadaver heads using a high speed X-ray system developed at the Herrick-Davis Motion Analysis Lab of the Henry Ford Hospital in Detroit. It used image intensifiers and digital video cameras to be able to track neutral density target markers inserted into the brain, to within 0.1 mm accuracy in three dimensions. They used small, neutral density (1.5 gm/ml), radio-opaque markers implanted through a 3 mm diameter cannula. In their experiments the cameras were set to produce 512 by 512 pixel images at 1,000 frames per second.

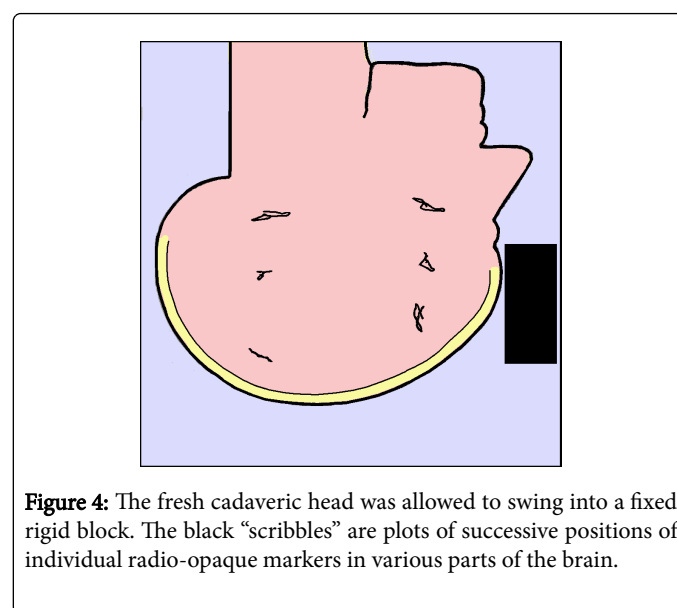


Figure 4: The fresh cadaveric head was allowed to swing into a fixed rigid block. The black “scribbles” are plots of successive positions of individual radio-opaque markers in various parts of the brain.

Fresh adult cadaver heads were suspended in a freely movable monitoring frame and subjected to sub-traumatic impacts [21]. Simultaneous position of the markers were recorded using a custom built X-Ray video recording system, at 1000 frames per second. Figure 4 illustrates one of their findings. Clearly, different parts of the brain traced different loci, i.e. the brain did not move as a whole (as a Sponge), more like a jelly. Importantly, they observed that “large motions near the centre of the brain due to angular rotation of the head are seen while this motion is less near the skull”. That means that the brain did not freely float, the cortex surface was restrained to move with the skull where it was stitched to the dura, while the rest of the brain deformed to accommodate movements.

Discussion

The consequences of including the trabeculae in the 1970s subarachnoid model are illustrated in Figure 4. The subarachnoid space is represented in blue. The dimensions of the subarachnoid space have been grossly exaggerated for clarity (If drawn to scale it would be barely one line thick). Panels (a) and (c) illustrate the normal conditions. Panels (b) and (d) represent the situation when impact from behind has forced the skull forward.

As declared in the SBS hypothesis, a freely floating brain [a] would get “left behind”, Figure 5. This would stretch the cortical bridging

veins, possibly causing them to tear before they had pulled the brain up to its normal position relative to the skull. With the contemporary arachnoid model [22], (c) the trabeculae stitch the arachnoid and pia layers together, so that they move as one. The lagging displacement is taken up in the brain itself, shown stippled in [d]. King et al. noted that in their impact experiments the largest brain-skull displacement occurred between the "middle" of the brain and the skull [21]. The transient displacement got progressively less when measured closer to the skull. Thus In the contemporary model there can be no stretching of the bridging veins, since the arachnoid and brain surface move together [d].

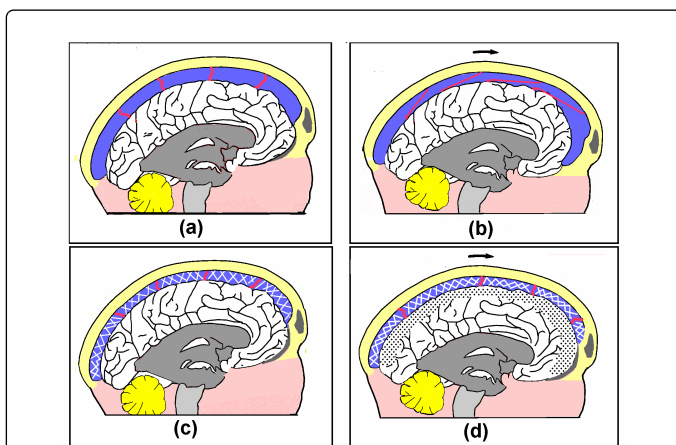


Figure 5: The effect of trabecular "stitching". Panels (a) and (c) illustrate the normal conditions, without (a) and with (c) trabeculae. The subarachnoid space is represented in blue, bridging veins in red. The dimensions of the subarachnoid space have been grossly exaggerated for clarity (If drawn to scale it would be barely one line thick). Panels (b) and (d) represent the situation when impact from behind has forced the skull forward. Without trabeculae, (b), the brain would be left behind, stretching the bridging veins. With trabeculae stitching the arachnoid and pia together (d) the brain deforms temporarily to take up the relative movement.

King's impacts were deliberately sub-traumatic, but Ommaya et al. studied injuries at threshold values of whiplash stress [22]. They subjected Rhesus monkeys to traumatic whiplash stress sufficient to cause 50% of them to suffer concussion. *Fifteen of 19 considered to have been concussed were found to have visual evidence for macroscopic damage as marked by surface haemorrhages.* Collagen fibres running in trabeculae are continuous with those of the inner aspect of the arachnoid and with those in the subpial space [6,10]. The sub-pial network is ultimately weakly bonded to the cortex surface. Excessive tension in the trabeculae would therefore be expected to pull the sub-pial network off the brain surface producing contusions. Thus contusions are to be expected to be the dominant form of injury at the threshold of shaking injury. However, the AAP definition of SBS says "*Visible cerebral contusions are unusual*". That is to say, in SBS, intracranial haemorrhages usually occur in the absence of contusions [23]. This indicates that Shaken Baby injuries result from a mechanism other than shaking.

There is little doubt that a syndrome exists whose properties result in the injuries currently referred to as Shaken Baby Syndrome. If the explanation of shaking now appears impossible, as above, how else

could these symptoms appear? A clue was given by the Dr Caffey, referred to in the original definition of shaken baby syndrome [24]. Dr Caffey's list of phenomena that he had found associated with the "whiplash shaken infant syndrome", preceding an event was wider than the Triad. It included: unexplained convulsions, hyperirritability, bulging fontanel, paralyses, and forceful vomiting. Bulging fontanel suggests raised intra cranial pressure, and forceful vomiting suggests pyloric stenosis [7,16,24]. These pressure symptoms arise naturally from the forceful vomiting in pyloric stenosis, when the large abdominal blood content is violently compressed, driving venous blood into the head [25].

Further support for a hypertensive mechanism comes from the association of macrocephaly with SBS [26]. Raised intra cranial pressure causes the dural tissue underlying sutures to signal for growth at the cranial sutures [27,28]. The stretch does not have to be continuous, skull growth messenger mRNA released during transient intracranial pressure continues to act afterwards, producing Benign Hydrocephalus of Infancy [29]. More subtle factors are produced by cerebral venous hypertension, such as arise from inadequate cerebral venous development in the embryo [30]. Developing macrocephaly in late pregnancy may give warning of the need for neonatal intracranial drainage.

Whereas the occurrence of SBS is currently considered unpredictable, an alternative cerebral venous hypertension model using the same data, offers possible preventative or palliative measures. In cases presenting with violent vomiting, check for pyloric stenosis. If present, consider Ramstedt's procedure [26,31]. In cases presenting with seizure and a large head consider surgical drainage of excess CSF to normalise CSF pressure [32,33]. This fluid may contain new or old blood from previous minor episodes.

Conclusion

This study set out to discover whether ignoring the trabeculae stitching the arachnoid mater to the pia mater would have made any difference to the legal validity of SBS cases. It is concluded trabeculae make a profound difference. The brain is not free to slide inside the skull restrained only by the bridging veins, as was assumed in the 1970s and accepted ever since. Ignoring the presence of trabeculae renders the SBS hypothesis invalid [15].

However, interpreting the features of "SBS" as of hydro-mechanical origin yields a model that is consistent with Caffey's observations and suggests possible preventative and palliative measures. The presence of the trabeculae cannot be ignored.

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