That the Shaken Baby Syndrome is based on a false subarachnoid model and is therefore invalid

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Abstract
Introduction
In the Shaken Baby Syndrome (SBS) concept subdural bleeding is attributed to tearing of bridging veins at the Dural/Sagital Sinus junction resulting from excessive tension during shaking of the infant by the carer. The brain is assumed to rotate within the skull, stretching these veins until they get pulled out of their attachments. The critical region where relative skull/brain movement would take place is across the subarachnoid space. This present study was to establish the mechanical factors involved.

The Hypothesis
The basis of the Shaken Baby Syndrome concept is that intracranial bleeds arise from relative movement of brain and skull during shaking. No mention is made of the “cob-web” of fine sheets and columns in the subarachnoid space stitching the arachnoid and pia maters together in a manner that would severely limit that movement. Without consideration of this factor the SBS concept is invalid.

Evaluation of Hypothesis
It was found that the classic model of the brain floating in cerebrospinal fluid, located by the bridging veins was incorrect. The apparently empty space is actually filled with a cobweb of collagen reinforced “Trabeculae” which are too thin to register on ultrasound or MRI machines. It is these trabeculae that locate the brain within the cerebrospinal fluid. The bridging veins are also supported in this cobweb. Moreover, the viscous effects of this combination of fluid and hundreds of trabeculae appears to provide a hydro-mechanical shock absorber system.

Conclusion
The present SBS hypothesis does not even mention the trabecular structure. The trabecular network in the subarachnoid space appears to have evolved to counter precisely the brain/skull movement that the SBS concept depends on. Until that effect is evaluated the SBS concept must be considered invalid.

Introduction
In the Shaken Baby Syndrome (SBS) concept subdural bleeding is attributed to tearing of bridging veins, at their Dural/Sagital Sinus junction, during shaking of the infant by the carer. The brain is assumed to rotate within the skull, stretching these veins until they are pulled out of their attachments. Bridging veins are attached to the inside of the skull (the dura mater) at one end, and to the surface of the brain (Pia Mater) at the other. The brain has been assumed to float freely in Cerebrospinal Fluid (CSF) in the subarachnoid space, restrained in position by the bridging veins. If the infant were shaken, these veins would attempt to pull the brain to follow the skull rotation. If the shaking were violent enough it is believed that the tension in these bridging veins might become sufficient to pull them away from the Dura allowing haemorrhaging and leaving torn tissue at the site. There is considerable literature trying to evaluate the degree of violence necessary to produce this.

And yet; in a modern textbook, Moore and Dalley point out that: “Although it is commonly stated that the brain ‘floats’ in CSF, the brain is suspended in the CSF-filled subarachnoid space by the arachnoid trabeculae”, bridging veins are not mentioned in this context. This study is of the implications of this update.

The Hypothesis
The basis of the Shaken Baby Syndrome concept is that intracranial bleeds arise from relative movement of brain and skull during shaking. The brain is enveloped in a sequence of membranes (see figure 1(a)) The outer, the dura, is attached to the skull. Inside that is the arachnoid with the fluid filled subarachnoid, then the Pia and finally the Glia that attaches the Pia to the brain cortex. In the SBS concept relative movement between brain and skull is said to occur across the fluid filled subarachnoid space. However, the subarachnoid space is not empty as in figure 1(a) it is filled with a network of ultrasonically invisible collagen reinforced fibres, figure 1(b)

In SBS documents no mention is made of this “cob-web” of fine sheets and columns in the subarachnoid space. They stitch the arachnoid and pia maters together in a manner that would severely impede that movement.

The message of this present hypothesis is that without consideration of this protective factor the SBS concept is invalid.

Evaluation of Hypothesis
Subarachnoid structure and the origin of Subarachnoid Trabeculae
Although the Dura Mater and Pia Mater were recognised by ancient anatomists it was not until 1664 that the arachnoid mater was officially recognised and named by the Dutch anatomist Gerardus Blasius. It is very thin and transparent and during dissection surface tension causes it to cling to the brain surface. Frederick Ruysch, also Dutch, demonstrated its existence by inserting a straw under its surface and blowing air under it (see figure 1 in ref 6). Although the subarachnoid space

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appears empty on the screens of ultrasound and MRI machines it is in fact actually filled with a cob-web of collagen reinforced fibres known as “arachnoid trabeculae”. “Arachnoid”, (derived from Greek for spider), referring to the cobweb appearance of this structure. These trabeculae have long been common knowledge among pathologists, e.g. in Grays Anatomy 1973. Histologically identical cells have been found in Pial and Arachnoid suggesting that they originate from of a single embryonic membrane. McLone, studying the foetal mouse subarachnoid space noted that at 10 foetal-days (prior to the secretion of cerebrospinal fluid,CSF) the arachnoid presents as a large extracellular space filled with mesenchyme. By day 13, CSF has begun to seep into and replace the ground substance of the mesenchyme. Trabeculae are not specifically created, they result as a process of random vacularisation of this initially uniform body of mesenchyme. The resulting lacunae get larger at the expense of the mesenchymal ground substance, until the tissue between neighbouring lacunae reaches a minimum thickness. The remaining tissue becomes a trabeculum. McLone and Bondareff comment “Bundles of microfibrils and collagen are commonly associated with lacunae in the outer pia-arachnoid layer and may serve as struts to maintain an open subarachnoid pathway.”

Actually these “struts” are under tension, “guy ropes” might be a better analogy. The result is walls in random direction in three dimensions, in engineering terms, a “redundant structure”. Redundant structures are resilient, they have no fault lines and can suffer the loss of a few elements without failure. Stress is redistributed among the remaining elements (Figure 2).

**The Mechanics of the Pia-Arachnoid**

Postnatally there are several tissue layers between the skull and the brain cortex. Figure 1 shows their relative configuration. The thicknesses are not to scale. (Orlin et al. give a photographic cross-section showing relative thicknesses from skull to brain cortex in the pig).

Normally the arachnoid membrane is attached to the Dura and hence to the skull, and the Pia is attached to the brain cortex, with the trabeculae linking them across the subarachnoid space. This is where movement could potentially occur. This region was described by McLone as the Pia-arachnoid because its linings were all derived the same pluripotential type of cell. Electron microscopy has shown that the Pia Mater itself is only one cell thick, with the cells tightly joined at the edges. The Pia Mater forms a barrier to movement of large molecules. It is not a structural element. Its associated structural components (collagen etc.) lie below, in the subpial space. Its delicate physiology suggests that if excessive tension occurs in the bridging veins, it is the Pia end that should tear away before the dural end. This has been found experimentally.

**Brain Positioning within the skull**

Electron microscopic examination of the physiology of the subarachnoid space has revealed that it is not the bridging veins that tether the brain as previously supposed. Both bridging veins and the brain are supported by the trabecular network.

“Trabeculae” means “beams”, referring to the way they appear to support the arachnoid mater above the pia mater. Actually the trabeculae are too flimsy to support anything in compression, but in vivo they are in tension. They define the shape of the subarachnoid space as the buttons define the shape of an aired. Thus the bridging veins are embedded in a “forest” of very fine trabecular fibres, traversing the subarachnoid space, over the cortex and around the spinal cord, see fig 7.7 in ref. Trabecular elements contain fibrocytes, collagen, and are wrapped around by a layer of pial cells. Figure 3(a). These elements combined to form “curtains”. Figure 3(b).

Single trabecular elements are typically 5 to 7 µm across, figure 3(a).
Such elements are combined to form cords and “curtains” attached between Arachnoid Mater and Pia Mater; figure 3(b). MRI scanners can resolve pixels of about 600um, but the trabeculae in figure 3(b) are only about 65 um thick. Moreover in figure 3(b) they occupy less than 1% of the cross section of the picture, so the averaged screen effect would only be a very dark grey, i.e. their presence would not be detectable on screen 11. Similarly, trabecular curtains are too thin to produce echoes on ultrasound. So on Ultrasound or MRI screens one can see blood vessels crossing between the Pia and Arachnoid membranes but there appears to be nothing else there.

The Subarachnoid Space

The Pia-arachnoid “Shock Absorber”

In small animal species brain inertia has little effect but it is possible that in large mammals the arachnoid trabecular system has evolved to produce a shock absorber system for heavier brains. The trabecular walls produce a degree of compartmentalisation 14. For movement to occur CSF must be forced from one compartment to another. The brain cannot move relative to the skull until the CSF moves out of the way. This occurs in many microflows, through tiny gaps between...
trabeculae. Until then, the fluid forces force the brain to move with the skull, a shock absorber mechanism.

**Discussion**

**Injury Threshold, the tear along the dotted line effect**

In 1968, Ommaya et al.\(^\text{15}\) secured monkeys on a rail-mounted trolley which could be accurately accelerated by compressed air to simulate collision by a following vehicle. They found that in monkeys, the onset of cortical damage was an “All-or-None” (threshold) phenomenon. Below an estimated 40,000 radians/seconds\(^2\) for 10 mS nothing happened. At higher impact their table shows that cortical surface contusions, subarachnoid and subdural haemorrhages appeared, in that order\(^\text{16}\).

**Contusion Confusion**

Contusions are defined as an injury without a break in the skin, from the Latin contundere to bruise or crush. Hematomas are local collections of blood, in this context occurring from the dural ends of the bridging veins. Ommaya’s results also show that if a shake were sufficiently violent, the veins and trabeculae may indeed pull out, but it is at their pia (cortex) end first, before causing subdural hematomas\(^\text{15,37}\). It requires greater violence to pull out the dural ends of bridging veins.

Ommaya’s tabulation of results\(^\text{15}\) shows that contusions of the cortex surface can occur without the presence of hematomas, but not vice versa. Subdural hematomas form part of the AAP definition of SBS which says contusions are “unusual”\(^\text{1}\), the reverse of Ommaya’s experimental findings. This implies that it is unusual for current SBS cases to be due to shaking.

**Conclusion**

The SBS hypothesis was founded on a defective model which did not represent the physical facts. It does not take into account the presence or significance of the trabecula structure. The mechanics of a “cobweb” of collagen reinforced trabeculae suggests the evolution of a quite sophisticated, stress distributing, shock absorbing system, quite different from the current concept of a sloppy suspension by a few elastic bridging veins. The present SBS hypothesis is invalid.

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**References**