Anatomy & Physiology: Current Research
ISSN: 2161-0940

Talbert, Anat Physiol 2016, 6:1
DOI: 10.4172/2161-0940.1000188

Research Article Open Access

Brain Motion under Sub-Traumatic Impact

David Talbert*

Imperial College, London, United Kingdom

***Corresponding author:** David Talbert, Imperial College, Institute of Reproductive and Developmental Biology, Imperial College School of Medicine, Queen Charlotte's Hospital, London W12 ONN,U.K.; E-mail: d.talbert@imperial.ac.uk

Rec date: Nov 18, 2015; **Acc date:** Dec 14, 2015; **Pub date:** Dec 21, 2015

Abstract

Aim: It has commonly been assumed that the brain "floats" in cerebrospinal fluid, restrained only by the bridging veins. However, modern textbooks show that the brain is actually suspended in CSF by an ultrasonically invisible "cobweb" (arachnoid) of collagen reinforced tissue. This would appear to prevent the cortex-skull relative movement necessary for the shearing action currently thought to cause the injuries diagnostic of Shaken Baby Syndrome. The aim of this study was to investigate brain behavior in everyday (sub-traumatic) conditions.

Method: A study into brain motion in vehicle accidents was found, in which radio-opaque markers were injected into fresh cadaver heads to enable movement of brain tissue during sub-traumatic impact to be recorded. Factors relevant to infants were extracted.

Results: Markers in various parts of the brain moved on different trajectories, indicating that fresh brain tissue is floppy. Markers closest to the meninges followed skull movement closest, indicating that the cortex did not slide under the skull linings. Any lagging movement of the deep brain was accommodated by deformation of the brain.

Discussion: Collagen bundles within trabeculae appear to be continuous with those in the inner aspect of the arachnoid and to those in the subpial space thus "stitching" the arachnoid and pia-maters together. However there are no collagen fibres in the brain, only astrocytes bonding the brain to the basement membrane. Anchor failure will occur first at this (pial) end as the pial mat is lifted off the cortex dragging minor surface vessels with them, forming cortical contusions.

Conclusion: Contrary to current assumptions that the brain is semi-rigid and trabeculae are weak, fresh brain is softly resilient and the Arachnoid and Pia Maters are firmly stitched together with collagen fibres and so cannot slide. The brain is floppy enough to accommodate local movements by deforming.

Keywords: Brain; Cadaver; Cerebrum; Contusions; Shaken baby; Subarachnoid trabeclae

Introduction

It has commonly been assumed that the brain "floats" in cerebrospinal fluid restrained only by the bridging veins connecting the cerebral cortex to the dural lining of the skull [1]. If this were so, any sudden rotation of the skull might strain the bridging veins to breaking point, causing bleeding and hematomas. But, as modern textbooks show [2], the brain is actually suspended in CSF by ultrasonically invisible trabeculae, a "cobweb" of collagen reinforced tissue from which the arachnoid membrane gets its name (Gr Arachnoid = Spiderlike) [3]. If the trabeculae prevent the brain sliding it is obviously desirable to determine how the brain does behave. There are very few experimental studies of the movement of the brain during and following an impact, but one carried out in the course of a study of vehicle collisions [4] demonstrates features relevant to investigation of imposed trauma on infants. Relating findings from the automobile industry to aspects of the pediatric "Shaking + Impact" hypothesis [5] is the subject of this paper.

Basic Concept of the Study by King, Yang and Hardy

King et al. used purpose built experimental equipment devised at Wayne State University, Detroit [4] which included a high speed (1000 frames per second) X-ray digital video camera system. They mounted fresh cadaver heads in a frame that enabled them to translate and rotate freely in a single plane. The head was mounted inverted to facilitate removal of any gas accumulating in the skull. Provision was made for low-severity testing with a calibrated impact set such that the brain subsequently returned to its initial configuration under its own elastic forces, showing it had not been damaged.

To monitor brain tissue movements, they implanted a series of X-ray opaque pellets of tin. Tin is, of course, much denser than brain tissue and by itself might cut through tissue during fast tissue movement during impact. To avoid this, the tin pellet was surrounded by polystyrene so as to produce a structure of similar mean density to brain tissue. These Radio-opaque Neutral Density Targets (NDT) were fabricated using 1.9 mm tin granules inserted in thin walled polystyrene cylinders 5 mm long and 2.5 mm diameter to produce a mean density of around 1.5 mg/ml. Holes were drilled through the skull to allow insertion of a 3 mm diameter cannula through which the NDTs were placed in columns about 1 cm apart (Figure 1).

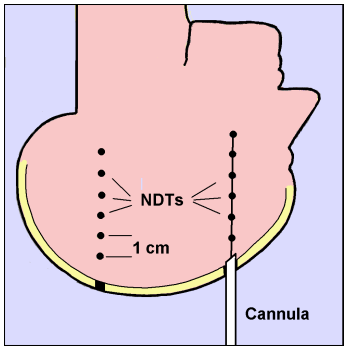

Figure 1: King et al.'s marker system as set up for anterior and posterior columns.

Relative movement within the brain

Using this system it was possible to track NDTs within 0.1 mm accuracy in 3 dimensions at a rate of 1000 frames per second.

Figure 2 shows tracings of movements of six of these markers when a head was swung into a fixed block (a deceleration test). These are tracks of NDT movements relative to the skull as seen in the sagittal plane. It is clear that movements differ in the various regions tagged by the NDTs, that is, the brain does not move as a solid structure, it is more like the traditional "Wibble-Wobble Jelly on a Plate". This may seem surprising to those who have handled formaldehyde preserved brain. Aldehydes are a widely used class of preservatives used to "Fix" tissue by chemical action. They work by attaching themselves to sites on two different molecules, a process known as "cross-linking". This disables both the macromolecules and any microorganisms that may be present. However, this also produces a great increase in mechanical strength by multiple links throughout and between cells, cross-linking and linking them to cytoskeletons. This is highly desirable for subsequent microscopic examination, one can't prepare 100 um sections from jelly, but it destroys the normal mechanical properties of the tissue. The familiar brain taken from formaldehyde storage is considerably more rigid than the fresh cadaver brains in Figure 2.

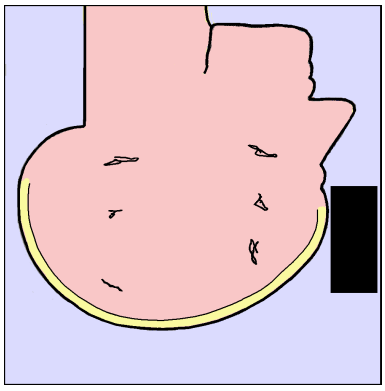

Figure 2: Locus in the sagittal plane of six of the NDTs following forehead impacting a fixed block.

Extrapolation of these Findings to Infant Heads

The cadaveric heads were of average age 77 years, and so their brains were encased in rigid skulls. In infants of a few months old the skull is thin, and made up of plates held together by somewhat elastic sutures. Thus infant skulls are capable of three dimensional movements that could not have occurred in the specimens obtained by King et al. Nevertheless, qualitatively, the character of the movements gives valuable insight into what may be the expected behavior of the infant brain.

A theoretical model of an infant brain is shown in Figure 3. Bridging veins collect blood from the cortex (pink); carry it across the subarachnoid space, through the arachnoid and dural (yellow) membranes and into the superior sagital sinus. The arachnoid and pia membranes are "stitched" together by threads or sheets of collagen impregnated tissue known as trabeculae. Scanning electron microscopy [6], Figure 5, shows they have an apparently random distribution and orientation. (That is because they appear early in embryological life by a subtraction process [7], they are not formed per se, they are the remnants of this subtraction process). In normal life such stitching will prevent the dangerous sliding of the brain relative to the skull that is assumed to occur in the current "Shaken Baby Syndrome" hypothesis [1]. It is postulated that three co-planar columns of NDTs are inserted into this brain model in the manner described by King et al., Figure 3.

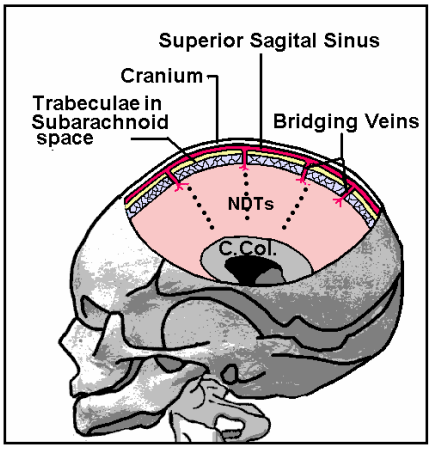

Figure 3: The hypothetical model of an infant brain with three columns of radio-opaque markers implanted.

Inertia Effects, as Described by King et al., Relevant to an Infant Skull Model

(1) *"A key observation is that the displacements of the brain seem to lag the motion of the skull. The inertia of the brain tends to keep it in place as the skull undergoes changes in motion".* This is known as Newton's Law: Force = Mass × Acceleration. The force required to start something moving depends on its mass and how quickly we want it to start moving (accelerate). The meninges are relatively strong and so will move the cortex surface with the skull. However, the force to start the bulk of the cerebrum moving reaches it through the soft brain tissue itself, and so is relatively weak. The acceleration will be less, and so deep parts of the brain will take longer to catch up with the skull.

King et al.'s results indicate that the brain deforms to accommodate such differential movement.

(2) With regard to relative movement of brain tissue and skull they observe that *"large motions near the center of the brain due to angular rotation of the head are seen while this motion is less near the skull."* This gradient of movement implies that the cerebral cortex surface normally follows movements of the skull, presumably dragged by the trabecular "cobweb" network.

(3) Another momentum effect is seen when the deep brain catches up with the skull rotation. Although it has reached its normal position relative to the skull it is travelling at some speed, so it continues to travel, overtaking the skull. *"As the head begins to rotate, local brain tissue tends to keep its position and shape with respect to the inertial frame, creating relative brain displacement and deformation. As the head rotation slows, reaches steady state or changes direction, the brain motion exceeds that of the skull."* They note that the brain subsequently reaches its normal position relative to the skull in an oscillatory manner.

(4) *"Another important observation is that after impact the brain seems to return to its initial configuration."* That is to say for impacts at everyday (non-damaging) levels the brain behaves as an elastic body, i.e. it is not significantly rigid.

Application to the Infant Head

Figure 4 illustrates the sequence of events these observations predict in infant heads. Experimentally, in King's low severity experiments, such impacts did not cause structural damage, as evidenced by recovery to initial configuration. Such recovery depends on the integrity of the trabecular bonding of the arachnoid membrane to the pial/brain surface across the subarachnoid space, Figure 3. Figure 5(a) is drawn from figures in a scanning electron microscope study of trabeculae surrounding the optic nerve by Killer et al. [6]. The optic nerve is not a true nerve; it is an extension of the brain reaching out to an eye [8] it carries the axons of over 1 million retinal nerves [9]. This makes it a convenient site to sample cerebral structure and behavior. It can be seen that trabeculae are not neat straight strings stretched across the subarachnoid space as they appear in the simplified diagrams in text books. They are sheets of random size and orientation, a structure related to their embryonic formation [7]. Such structures are described in engineering terms as "redundant" They have more components than necessary and are able to resist forces applied in any direction. However they are only as reliable as their anchor points. Electron microscopy has shown collagen fibers within the trabeculae to be continuous with collagen layers in the inner aspect of the arachnoid membrane [10]. The tensile strength of collagen in catgut is similar to that of mild steel [11] making trabecular anchoring to the arachnoid pretty secure. Bonding at the other (pial) end of trabeculae is to the subpial collagen network. This in turn is connected through the basement membrane to the glial astrocytes in the brain cortex itself. There are no collagen reinforcing fibers within the brain so ultimately trabecular anchoring at their brain end is to astrocytes and neurons in the brain itself. If the jolt were sufficiently increased, eventually the basement membrane would detach from the astrocyte feet allowing the cortex surface to be lifted off, a contusion, Figure 5(b),

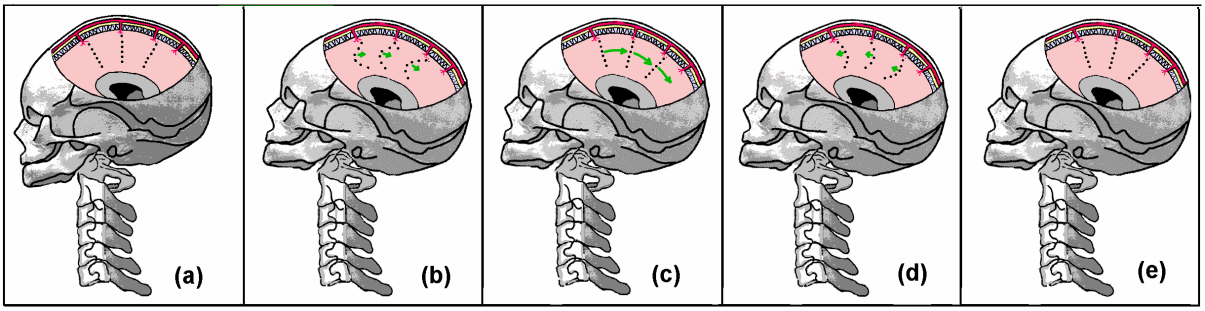

Figure 4: Hypothetical sequences of events following impact on forehead of infant. Panel (a) represents the starting position with radio-opaque, neutral density markers inserted. Shortly after an impact on the forehead (b) the skull has been abruptly tilted back. The cortical surface has moved with it, pulled by the trabecular cobweb network (cross-hatched). The markers show that the deep brain has been left behind and is in the process of catching up (short green arrows.) In (c) the deep brain has caught up with the skull, but it is still moving (long green arrows). In (d) The deep brain has "overtaken" the skull but its internal forces are pulling it back.(short green arrows) After some oscillation (corresponding to Figure 2) it settles down in its natural position relative to the skull in the skull's new position, (e).

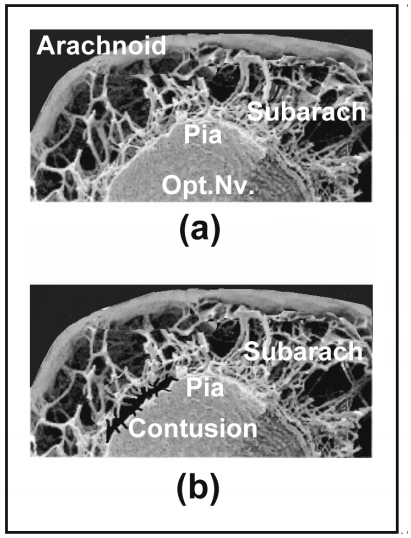

Figure 5: Bonding failure at the Pia-brain-cortex junctions (a) leads to tearing at the brain surface (contusions), (b).

This is precisely what Ommaya et al. [12] found in their experimental shaking of monkeys. When the jolt they used was adjusted so that half of the subjects were brain injured and half were not, the commonest injuries were contusions of the brain surface.

Conclusions

The study by King et al. shows that fresh brain does not act as a solid, it is soft (Mushy [13]) and floppy. Under impact the intact brain cannot slide around under the skull. The cortex surface remains firmly attached to the meningeal layers lining the skull. The fresh brain being softly elastic normally accommodates any movements by deforming, not by sliding. Applying these findings to the case of infants being shaken, such a model implies that in shaking sufficiently violent to cause brain damage, injury to the brain surface (contusions) should be common. This agrees with the experimental findings of Ommaya, [12] but not with current definitions of SBS [1] which says that contusions are "unusual". This further supports the contention that the current model of SBS is invalid.

References

1. American Academy of Pediatrics (2001) Shaken Baby Syndrome:Rotational Cranial Injuries-Technical Report. Pediatrics 108(1): 206-10.
2. Moore KL, Dalley AF (2006) Clinically Oriented Anatomy. (5thedtn). Philadelphia: Lippincott Williams and Wilkins. pp. 885-1043.
3. Sanan A, Loveren HR (1999) The Arachnoid and the Myth of Arachne. Neurosurgery 45: 152-7.
4. King AI, Yang KH, Hardy WN (2011) Recent Firsts in Cadeveric Impact Biomechanics Research. Clinical Anatomy 24: 294-308.
5. Duhaime AC, Christian CW, Rorke LB, Zimmerman RA (1998) Nonaccidental head injury in infants-The Shaken-Baby Syndrome. N England J Med 338: 1822-9.
6. Killer H, Laeng H, Flammer J, Groscurth P (2003) Architecture of arachnoid trabeculae, pillars, and septa in the subarachnoid space in the human optic nerve: anatomy and clinical considerations. Br J Opthalmol 87: 777-81.
7. Talbert DG (2014) The Embyological Development of the form of the Trabeculae Bridging the Subarachnoid Space. J Trauma Treatment.
8. Moore KL, Dalley AF (2006) Summary of cranial nerves. Clinically Oriented Anatomy. 5 ed. Philadelphia: Lippincott Williams & Wilkins: 1123-55.
9. Warwick R, Williams PL (1973) The Cranial Nerves. In: Warwick R, Williams PL, editors. Gray's Anatomy. 35 ed. Longmans: 996-1030.
10. Alcolado R, Weller R, Parrish E, Garrod D (1988) The Cranial Arachnoid and Pia Mater in Man: Anatomical and Ultrastructural Observations. Neuropathol Appl Neurobiol 14: 1-17.
11. Kaye G, Laby T (1982) General Physics. Tables of Physical and Chemical Constants. (14thedtn). London: Longman, 34.
12. Ommaya AK, Faas F, Yarnell P (1968) Whiplash injury and brain damage. JAMA 204: 285-9.
13. Cormack DH (1987) Nervous tissue and the nervous system. Ham's Histology. (9thedtn). Philadelphia: Lippincott: 339-87.