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TECHNICAL NOTE

Influence of the benign enlargement of the subarachnoid space on the bridging veins strain during a shaking event: a finite element study

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Abstract There is controversy regarding the influence of the benign enlargement of the subarachnoid space on intracranial injuries in the field of the shaken baby syndrome. In the literature, several terminologies exists to define this entity illustrating the lack of unicity on this theme, and often what is "benign" enlargement is mistaken with an old subdural bleeding or with abnormal enlargement due to brain pathology. This certainly led to mistaken conclusions. To investigate the influence of the benign enlargement of the subarachnoid space on child head injury and especially its influence on the bridging veins, we used a finite element model of a 6-month-old child head on which the size of the subarachnoid space was modified. Regarding the bridging veins strain, which is at the origin of the subdural bleeding when shaking an infant, our results show that the enlargement of the subarachnoid space has a damping effect which reduces the relative brain/skull displacement. Our numerical simulations suggest that the benign enlargement of the subarachnoid space may not be considered as a risk factor for subdural bleeding.

Keywords Finite element model · Shaken baby syndrome · Benign enlargement of the subarachnoid space · Child abuse

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Introduction

Confusion exists in the literature regarding the benign enlargement of the subarachnoid spaces (BESS) which has been addressed by different terms [1–5]. This condition is characterised by reversible enlargement of the subarachnoid spaces of infants with a large head circumference, with or without ventriculomegaly, due to accumulation of cerebrospinal fluid (CSF). Various terms have been used to describe this condition in the literature, benign expansion of subarachnoid spaces, benign external hydrocephalus, benign infantile hydrocephalus, subarachnoid space enlargement, and extra-cerebral fluid collection. These different terms have been used arbitrarily to describe similar conditions, most probably due to the lack of appropriate criteria and clinical profiles to define the entity of BESS. Early studies using CT suggested that these normally prominent extra-cerebral collections resided in the subdural space, and that a subdural collection in otherwise normal infants was therefore of little significance. But the improvement of imaging techniques has shown that these collections are located within the subarachnoid space [3–5]. Various terms like subdural fluid collection, hygroma, effusion, and chronic subdural haematomas have been interchangeably used in various reports in the literature dealing with subdural fluid collections of varying etiology. Simultaneously, BESS and subdural effusion have been labeled to be identical, while the clinical profile and causations of the two entities are described to be different. Because of several such points of confusion, a controversy exists regarding the influence of the enlargement of the subarachnoid space on the risk of developing subdural haemorrhage. It has been said that a minor trauma could lead to a subdural bleeding or even that subdural haemorrhage may occur spontaneously in this case [2, 6–11]. This

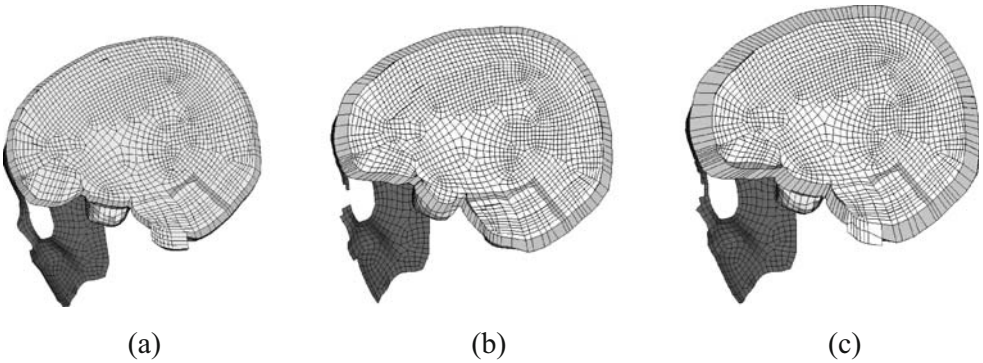

Fig. 1 Finite element model of a 6-month-old child with normal subarachnoid space (**a**) 4 mm width (**b**) and 8 mm width (**c**)

is supposed to be the consequence of a less limited motion of the brain within the skull due to the excess of CSF leading to an increased stretch on the bridging veins. But the fact that prominent subarachnoid spaces are common in infants, and subdural haemorrhage is rare, would indicate that there is no scientific basis for that supposition. In order to study the influence of the size of the subarachnoid space on the bridging veins, a finite element analysis has been preformed. Finite element models have previously been described in the field of forensic sciences and have already been used by Roth et al. to compare a shaking event and an impact using a finite element model of a 6-month-old child head [12–16].

Materials and methods

Benign enlargement of the subarachnoid space is usually diagnosed in the first year of life [2], and as there is a peak frequency of shaken baby syndrome cases around the age of 6 months [17], we have chosen to use the finite element model of a 6-month-old child which had been used and described in our previous paper. The width of the CSF of the model (2 mm corresponding to a 50th percentile), has been increased to 4 and 8 mm, corresponding respectively to a 45-cm (95th percentile) and 55-cm (above 95th percentile) head circumference. The value of 8 mm width was voluntarily taken in order to investigate extreme cases of BESS and better understand the mechanisms involved. Figure 1 illustrates the different modified models with variable width of the subarachnoid space. The weight and volume of the brain were identical in the three cases.

Material properties implemented in the model were taken from the literature and were described in our previous paper [12].

A vigorous shake was then simulated with the model. The shaking cycle was taken from Prange et al. experiments [18]. As it was done in our previous study, the relative motion of the brain and the skull were computed in different parts of the model, along the falx. This motion corresponds to the maximum strain of the bridging veins, which link the cortex to the superior sagittal sinus. The bridging vein's initial length was measured between a node on the inner surface of the skull and a node on the outer layer of the brain of the model, therefore, its length corresponds to the width of the CSF.

The strain of the bridging veins is a function of the initial and final lengths as illustrated in equation 1:

$$\varepsilon = \frac{l - l_0}{l_0} \tag{1}$$

where ε represents the strain, l and l_0 length and initial length, respectively.

Results

Results show that bridging veins strain in different areas along the falx decreases as the width of the subarachnoid space increases. Figure 2 illustrates the time history strain of a bridging vein located in the vertex area. Maximum strain reached 0.9 for a 2 mm (normal) width and reached 0.22 for an 8 mm width.

Figure 3 illustrates the time history strain of a bridging vein located in the occipital area. Maximum strain reached

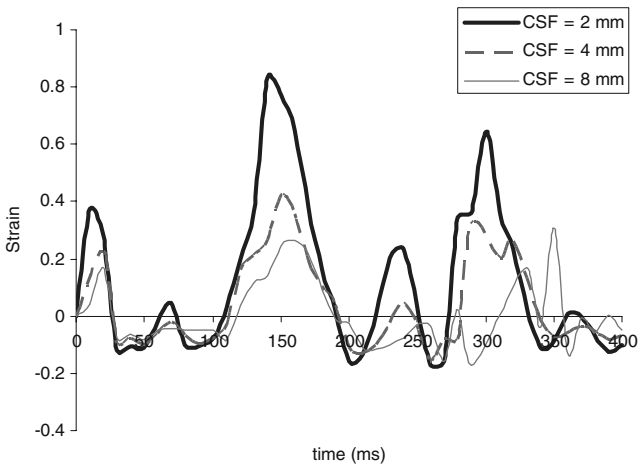

Fig. 2 Bridging vein strain along the vertex area

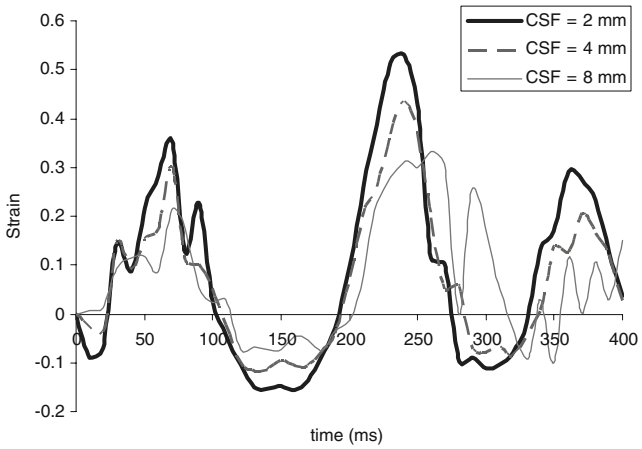

Fig. 3 Bridging vein strain in the occipital area

0.55 for a 2 mm (normal) width and reached 0.34 for an 8 mm width.

Discussion

When an infant is violently shaken, the relative motion between the brain and the skull essentially due to inertia of the brain, will lead to a stretching of the bridging veins which form a short trunk passing directly from the brain to the dura mater without any tortuosity [19]. The cranial ends of the bridging veins are firmly fixed to the rigid dura mater, while the cerebral ends are attached to the movable hemispheres. Yamashima and Friede have shown that the subdural portion of the vein is more fragile than its subarachnoid portion explaining the laceration of the vein and the subdural location of the haematoma [19].

For the past years, benign enlargement of the subarachnoid space has been widely mistaken with subdural collections and especially with what has been called subdural hygroma. Subdural hygroma, which develops after an acute subdural bleeding, is mainly asymptomatic because developing as a space-filling lesion mimicking the development of BESS. If the hygroma persists, a neomembrane will sometimes form with neovascularisation leading to spontaneous microhaemorrhage. Repeated haemorrhage will lead to a chronic subdural haematoma. Distinguishing external hydrocephalus from subdural hygroma may be very difficult, both leading to an increased head circumference and appearing as hypodense on CT-scans, but their evolution will be different, subdural hygroma leading sometimes to subdural haematoma through microhaemorrhages which may be caused by minor trauma [20]. Our model does not take into account the modification of the properties of the tissues as a consequence of subdural hygroma. The subarachnoid space has been widened in our model leading to a growth of head circumference. The tissue properties are considered as normal in case of BESS which is not the case of subdural hygroma for which our conclusions are therefore not relevant even if there is also in this latter case a growth of head circumference.

One of the argument sometimes stated by authors is that as no history of trauma or risk factors for child abuse had been identified, it could be concluded that the subdural bleeding occurring during the follow-up of a child presenting with BESS could be explained by a possible spontaneous bleeding. But this argument appears clearly insufficient to exclude a possible case of non-accidental head injury and to conclude on a scientific basis.

Benign enlargement of the subarachnoid spaces is frequently found on imaging in otherwise normal infants. These patients are often macrocephalic. Typically, the head circumference is large at birth and exceeds the 95th percentile later in the first year of life. Although the collections are usually located over the frontal and temporal convexities, prominence of the basilar cisterns and anterior inter-hemispheric fissure are commonly seen [21].

Generally, this condition is self-limiting and requires no specific intervention. It could be the consequence of insufficient resorption of the CSF through immature arachnoid villi [22]. This enlargement is generally regarded as a non-pathologic process that resolves uneventfully. Long-term observations of infants with benign, congenital widening of the subarachnoid space revealed no increased frequency of subdural haematomas over time [1, 23]. On the other hand, post-traumatic hydrocephalus can follow a significant head injury. Impaired CSF absorption at the level of the arachnoid villi due to arachnoiditis or blood within the subarachnoid space, may be the explanation of such an evolution leading to an enlarging head circumference. It is important to consider in this case that the enlarging of the head circumference due to external hydrocephalus is the consequence and not the cause of the bleeding.

Papasian and Frim have developed a theoretical model of benign external hydrocephalus that predicts predisposition towards extra-axial haemorrhage after minor trauma [24]. This model is made of two concentric spheres representing the brain and the skull. The major problem is that the model does not take into account the mechanical properties of the CSF and especially the damping effect, which seems to play a role when considering the bridging vein strain. If the CSF properties were those of the air, then their conclusions would certainly be correct. But the mechanical properties of the CSF are close to the properties of the brain and both closely interact after impact or shaking. Our simulation compares the strain of bridging veins passing without any tortuosity from brain to skull.

Authors have suggested there could exist an initial strain on the bridging veins due to the enlargement of the

subarachnoid space. Baker described the bridging veins as taking a straight course with no tortuosity to allow for the possible displacement of the brain in 1938 [25]. This initial description is still to date. The benign enlargement of the subarachnoid space is asymptomatic and slowly takes place within the skull, there is for the time being no evidence that there could exist an initial strain on the bridging veins in this case. The bridging veins still have a straight course may there be BESS or not. For this reason the bridging vein's initial length was measured between a node on the inner layer of the skull and a node on the outer layer of the brain of the model. Our finite element study supports the idea that children presenting with pure benign enlargement of the subarachnoid space are not at a statistically greater risk for subdural haemorrhage. There exists a damping effect on the relative motion between the brain and the skull due to a greater amount of CSF. In the absence of a possible medical explanation concerning a subdural bleeding (brain pathology, haemophilia), the finding of an associated benign enlargement of the subarachnoid space should not be regarded as excluding the possibility of a major head injury may it be accidental or not.

It must be outlined that our work is a numerical simulation, and that some uncertainties remain. Very few mechanical properties exist in the literature concerning pediatric tissues. We have used available constitutive laws which may be improved in future works. Also for ethical reasons, experimental tests on children are impossible, therefore, to time, no validation of the finite element model can be performed. Nevertheless, finite element methods can help understanding the biomechanics of child abuse.

Conclusion

We performed numerical simulations of a shaking event using a finite element model of a 6-month-old child head on which the subarachnoid spaces had been enlarged. Our results show that the enlargement of the subarachnoid space seems to have a damping effect which reduces the relative brain/skull displacement. Our numerical simulations suggest that the benign enlargement of the subarachnoid space may not be considered as a risk factor for subdural bleeding.

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