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A Theoretical Model of Benign External Hydrocephalus That Predicts a Predisposition towards Extra-Axial Hemorrhage after Minor Head Trauma

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Key Words

Subdural hematoma · Head trauma · External hydrocephalus · Mathematical model

Abstract

Introduction: There is controversy over whether there exists a predisposition towards bleeding into the subdural space in infants with benign external hydrocephalus (BEH) or other enlargement of the extra-axial space (e.g. subdural hygroma). The presumed etiology implicates shear forces in over-stretching the extra-axial blood vessels. We have created a model of the intracranial space that approximates certain aspects of BEH. Using this model, we predict situations where children with BEH will bleed into the extra-axial space when normal infants will not. ***Methods:*** The cranial model consists of two spheres representing the brain and the skull. The distance between them represents the width of the extra-axial space. The spheres are concentric (with interspheric distance equal to N) in the normal condition and nonconcentric in BEH. In BEH, the distance between the two spheres varies from N to Q ($0 < N \leq Q$) over a 90° arc. By Euclidean geometry, if the brain is translated relative to the skull, such as by external trauma, by a distance M ($N \leq M \leq Q$, with the translation orthogonal to the vector of N and parallel to that of M or vice versa), then the final length V_f of a vein V is $V_f = (N^2 + M)^{1/2}$, and the stretch ratio V_f/V_i is: $V_f/V_i = (N^2 + M^2)^{1/2}/N \cdot S_i$, with S_i the slack factor, where i represents either n for normal or b for BEH and M = N in the normal condition. ***Results:*** Given an equivalent capacity of veins to resist stretch injury (based on the proportion of change in length), for brain translations after a low-impact head injury, stretch ratios for BEH veins range from 1.677 to 3.436, whereas in the normal condition they range from 1.061 to 1.179. Therefore, for an increase in subarachnoid space from 3 (normal) to 6 mm (BEH), brain translocation in BEH will stretch veins beyond an average breaking point when the translation for the normal condition will not. ***Conclusions:*** Mathematical modeling of the cranial vault produces a relationship between venous stretch and the width of the extra-axial space. These equations predict an increased frequency of venous stretch injury in the situation of widened extra-axial space. Such venous injury is consistent with forces generated by minor trauma. This relationship, as predicted by our model, could underlie a predisposition towards extra-axial bleeding after minor head trauma in infants with BEH.

Introduction

Benign external hydrocephalus (BEH) is an age-related self-limiting condition in which there is widening of the extra-axial fluid spaces, in particular the subarachnoid

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(SAS) and subdural spaces. It occurs in infants and usually resolves spontaneously by about 3 years of age [1–3]. The natural history is 'benign' i.e. children attain normal developmental milestones and show no clinical sequelae [4–9]. Therefore, neurosurgical treatment is generally not undertaken for uncomplicated BEH. The etiology of BEH remains poorly understood, but children are often macrocephalic, which may be a familial condition [5, 10].

Benign external hydrocephalus has, however, been associated with subdural hematoma (SDH) [11, 12]. The incidence of SDH in children with BEH is suspected to be much higher than that seen in the general population, as in one group of children with BEH where the incidence of SDH was 3/20 [1, 13]. Thus, children with BEH seem to be predisposed to developing acute SDH either spontaneously or following minor head trauma [1, 10, 11, 13]. The etiology of SDH in the setting of BEH is also poorly understood but presumably derives from anatomical and geometric stresses on blood vessels in the enlarged SAS/subdural space [12, 14, 15].

SDH has various causes, the commonest of which is stretch failure of veins that bridge the subdural space [15–17]. Biomechanical models of acute SDH due to bridging vein failure have been developed based on the tensile properties of veins and their anatomical orientation in the subdural space [16, 18–20]. These models focus on overstretch of the bridging veins with ultimate strain failure as the mechanism of SDH. Strain rate, strain duration and relative stretch all seem to have a role in determining the ultimate falure rate of veins, thereby causing hemorrhage. In a finite element analysis, Huang et al. [18] examined the individual contributions of linear and rotational acceleration. They found that the rotational component far outweighs the linear and that the worst injuries result from occipital impacts.

The present study was designed to examine whether, when undergoing anteroposterior trauma, the anatomical conditions of BEH may explain a tendency towards developing SDH. Using Euclidean geometry, we constructed a mathematical model of the brain and skull representing the anatomical differences between children with BEH and normal children. We compared the tendency towards bridging vein failure after minor trauma in the two conditions.

Methods

Let us model the normal skull and brain as two concentric hollow spheres with centers of gravity at the same point. Clearly the distance between these spheres is constant regardless of direction. Similarly, we can model the skull and brain in BEH as a system of two nonconcentric hollow spheres, with centers of gravity along a craniocaudal axis in the sagittal plane. The bridging veins must span the distance between the brain and skull in either case, and they are presumed to be slack and at approximately 90° angles to the spheres [15]. We can calculate the percent stretch, or stretch ratio, in a bridging vein when the system of two spheres impacts an immovable object (e.g. the head hits the ground after the child falls from a low height) given either the normal condition or BEH (fig. 1).

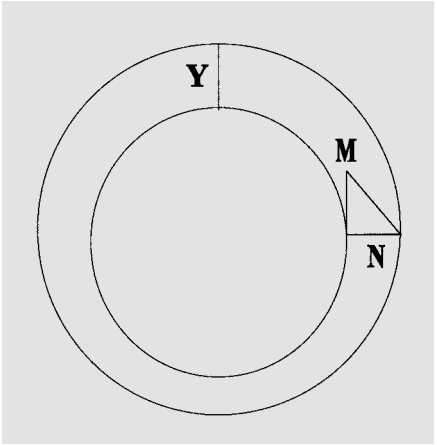

Fig. 1. Cross section through a model of the brain and skull.

Let N = the distance between the two spheres (brain and skull) in the normal condition at rest (a constant); Y = the intersphere (brain to skull) distance variable in BEH at rest; Q = the maximum intersphere distance in BEH at rest; M = the maximum distance of translation of the inner sphere with respect to the outer sphere in either condition (normal or BEH) at impact; V = length of a bridging vein (V_i = initial length, V_f = final length), and R_s = the stretch ratio = V_f / V_i.

Our model assumes the following:

(1) Any bridging vein has the tensile properties of a string, that is, it ruptures (and therefore hemorrhages) at an ultimate stretch.

(2) Corresponding veins in the normal condition and in BEH have the same length regardless of the original distance between the brain and skull.

(3) The veins in either condition (normal or BEH) at rest are oriented like spokes on a wheel (orthogonal to both the brain and skull).

(4) Veins in either condition at rest are 'slack', without tension.

The slack in a vein is the nontension vein length that exceeds the original distance between the two spheres (brain and skull). The slack factor, then, is the percent excess vein length, given by the vein length at rest divided by the intersphere distance at rest. We represent this excess by the slack factor S_i, $i \in \{n, b\}$, which becomes S_n in the normal condition and S_b in BEH. Then the initial lengths of veins are: $V_i(\text{nor}) = N \cdot S_n$ and $V_i(\text{BEH}) = Y \cdot S_b$ with $Y \in [N, Q]$ for normal and BEH, respectively.

Note that by assumption 2 above: $N \cdot S_n = Y \cdot S_b$ and $S_b \leq S_n$.

(5) Drag (friction) forces of CSF within the system are equal regardless of the original width of the extra-axial space (i.e. the dis-

Table 1. Estimated stretch ratios for subdural bridging veins in children with BEH and in normal children

S_n	S_b	N, mm	M, mm	R_s(nor)	R_s(BEH)
1.2	1.2	2.5	2.5	$\sqrt{2} = 1.414/S_n = 1.179$	$\sqrt{2} = 1.414/S_b$
			5.0		1.863
			7.5		2.635
			10.0		3.436
		3.0	3.0		$\sqrt{2} = 1.414/S_b$
			6.0		1.863
			9.0		2.635
1.33	1.33	2.5	2.5	$\sqrt{2} = 1.414/S_n = 1.061$	$\sqrt{2} = 1.414/S_b$
			5.0		1.677
			7.5		2.372
			10.0		3.092
		3.0	3.0		$\sqrt{2} = 1.414/S_b$
			6.0		1.677
			9.0		2.372

tance between the spheres). Furthermore, we will take these forces to be negligible.

(6) The interspheric relationships remain unchanged until impact, i.e. drag (friction) forces of air are negligible.

(7) The collision is partially inelastic such that the system's outer sphere (skull) and the immovable object (ground) remain in contact at all times after initial impact. However, they do not deform.

(8) The force (and therefore velocity) of impact is such that the inner sphere (brain) translocates to contact the outer sphere (skull) but does not deform. This implies a low impact force or 'minor head injury' that will not cause brain injury (contusion). The inner sphere may, however, bounce back off the outer sphere and thus oscillate.

Given the above assumptions, the outer sphere will decelerate instantaneously at impact, while the inner sphere will continue to travel with constant acceleration until it impacts the skull (by assumption 4). It follows that, at impact, the net translation (M) of the inner sphere within the outer sphere is the initial distance between the spheres. Thus, M = N in the normal, and $M \in [N, Q]$ in BEH depending on vein position and impact direction.

Recalling that corresponding veins in normal and BEH children have the same length (and no tension) at rest, we see that veins in BEH will have more room to stretch until the brain collides with the skull, and therefore a greater number of veins will stretch further in BEH.

In our first test of the model, we will limit our calculations in this presentation to anterior/posterior force vectors. Venous stretch parallel to the force vector is trivial to calculate: the BEH scenario allows increased translational stretch by linear combination. This increase is $\Delta V_{(BEH - norm)} = M - N$. Analyzing each condition individually, we find that $V_f(nor) = N + N = 2N$ and $R_s(nor) = V_f / V_i = 2N/(N \cdot S_n) = 2/S_n$. Similarly, $V_f(BEH) = M + N$ and therefore $R_s(BEH) = M + N/N \cdot S_n$. Since $M > N$ in this impact situation, we see that for each value of N, $R_s(BEH)$ exceeds $R_s(nor)$, which is the intuitively expected outcome.

Calculating the final length for bridging veins orthogonal to a fronto-occipital impact along the plane containing the central axis in either condition is more complicated. For lateral orthogonal veins, it follows by the Pythagorean Theorem and the geometry of isosceles right triangles that the final length in the normal condition is $V_f(nor) = N \cdot (2)^{1/2}$.

Therefore, for the normal condition, with $V_i(nor) = N \cdot S_n$ and $V_f(nor) = N \cdot (2)^{1/2}$, the stretch ratio R_s is:

$R_s(nor) = V_f(nor)/V_i(nor) = [N(2)^{1/2}]/N \cdot S_n = (2)^{1/2}/S_n$;

and for BEH, with $V_i(BEH) = Y \cdot S_b = N \cdot S_n$, the final length V_f is:

$V_f(BEH) = (N^2 + M^2)^{1/2}$,

and the stretch ratio is:

$R_s(BEH) = V_f(BEH)/V_i(BEH) = (N^2 + M^2)^{1/2}/N \cdot S_n$.

Note that in the special case of M = N, this reduces to the normal condition.

Results

Utilizing the equations derived above, we can now test the hypothesis that BEH anatomy will be more susceptible to minor trauma causing a SDH. We will assume that the subarachnoid enlargement in BEH is >5 mm distance from brain to skull [10, 21] and will define 2.5 as the mean between 0 and 5 for normal. We will presume a BEH extra-axial space range between 5 and 10 mm (mean = 7.5 mm). Utilizing these estimates, stretch ratios for minor head injury in children with or without BEH are calculated in table 1.

Using typical values from the distance ranges defined above and a conservative estimate of $S_n = S_b$, we calculated the ultimate stretch ratios as listed in table 1. We chose values for M to be multiples of N for clarity in intuitive interpretation and for ease of calculation. A slack factor of 1.2 corresponds to 20% slack in the vessels and 1.33

to 33% slack in the vessels. For lateral orthogonal vessels, the symmetry of the normal condition gives the constant stretch ratios of 1.179 for vessels with 20% slack and 1.061 for vessels with 33% slack. For BEH, the stretch ratio increases significantly with increasing distance. The stretch ratios in BEH for M:N distance ratios of 2, 3 and 4 are 1.863, 2.635 and 3.436, respectively, for 20% slack, and 1.677, 2.372 and 3.092, respectively, for 33% slack.

Previous estimates of ultimate subdural stretch (stretch required for venous rupture) predict that an average stretch ratio of 1.5 will result in vein breakage [19]. Therefore, according to our calculations, venous failure occurs with much greater frequency in the case of BEH than in the normal condition, based on the increased stretch ratios as calculated (>1.5 for either 20 or 33% slack factor at even a two-fold increase in SAS width).

Discussion

SDH is a major cause of morbidity and mortality in severely head-injured patients. This is particularly true in infants and children under 2 years of age, in whom this entity is a much commoner occurrence than in older children or adults. It is generally accepted that acute SDH develops secondary to disruption of bridging veins [16]. Thus over-stretch of the bridging veins with ultimate strain failure has been implicated and has been the focus of numerous models for the mechanism of SDH [16, 18–20]. Variables studied include acceleration magnitude and type (i.e. rotational versus translational), acceleration duration, acceleration strain rate and direction of venous flow. Huang et al. [18] have examined vein failure in the context of acceleration-deceleration injuries, looking in particular at the individual contributions of rotational and translational acceleration.

By a Euclidean geometric analysis of the anatomical differences between the subdural veins of children with BEH and normal children, we have developed a mathematical model that predicts a higher failure rate for BEH veins compared to normal veins after an anteroposterior impact causing brain translocation relative to the skull. We infer, as do Huang et al. [18], that this higher venous failure implies that infants with BEH are predisposed to developing SDH after minor head trauma. Specifically, in our model, after a low-impact head injury, stretch ratios for BEH veins range from 1.677 to 3.436. In the normal case, venous stretch ratios range from 1.061 to 1.179.

That a vein ruptures at an ultimate stretch was shown by Lowenhielm [22] and again by Lee and Haut [19]. In examining bridging veins from 8 unembalmed cadavers, Lee and Haut found the ultimate stretch ratios (i.e. ratios at vein failure) to be 1.51 ± 0.24 ($n = 29$) and 1.55 ± 0.15 ($n = 34$) for low and high strain rates, respectively. Thus, our values for BEH venous stretch are well above the reported means for vein failure, whereas those for normal are below. If we consider the 95% confidence intervals for their data, the ranges are 1.03–1.99 and 1.25– 1.85 for low and high strain rate injuries, respectively. It is clear that bridging veins in the normal child may be damaged with minor head injury; however, if our model is correct, then at any given strain rate in a minor head impact, a greater percentage of bridging veins is likely to hemorrhage in a child with BEH than in a normal child. Moreover, Laubscher et al. [10] report a SAS range from 6 to 23 mm for children with BEH. Thus, our SAS space range is a conservative sample, and R_s(BEH) ratios for individual children may be much greater than our estimates, yielding even greater vein failures rates.

Though impacts to the skull cannot be generalized to impacts on the brain [23], previous studies modeling head injury and SDH have found that impacts involving rotational forces result in greater stretch of bridging veins than impacts resulting in purely translational forces [18]. Huang et al. [18] found that linear stretch is 2.4% strain, rotational is 10.5% and angular (linear plus rotational) is 14.5%. As purely translational impacts are rare [23], according to these biomechanical models of SDH, our model predicts the minimum stretch difference between BEH and normal, since the presence of a rotational component would result in even greater vein strain/stretch. Thus, though stretch ratios would increase for both conditions, R_s(BEH) would still be greater than R_s(nor), indicating a relatively increased tendency towards vein failure in BEH.

Previous models have utilized some of the same assumptions and approaches as we have in examining head trauma and SDH. Ours were chosen as a first attempt to model the BEH situation; however, we can examine the consequences of relaxing each assumption individually.

Wilkins [23] discusses that contact of the skull with surfaces of different texture/elasticity (e.g. concrete versus carpet) does not affect the outcome. Therefore, we can infer that not only will the BEH and normal skulls be equally deformable, but moreover, the deformations are negligible.

For ease of mathematical manipulations, we assumed a low impact velocity, such that the brain will not deform. Let us allow the velocity to increase moderately such that the brain deforms but does not become injured (i.e. no cerebral contusion). Then, the actual distance traveled by

a point of attachment where the vein contacts the brain may be even greater than N or M (for normal or BEH, respectively), resulting in even greater stretch ratios for both. Though R_s(nor) will approach R_s(BEH) as the deformation increases, this scenario is not particularly relevant to the present study, as there is no controversy about high-impact head injury tending to cause brain injury in most children [12].

The brain/skull system is not a double-hulled sphere as we assume, although spherical models have been used previously [20, 24]. The brain and skull are also not concentric convex geometric objects, sphere or otherwise. However, the exact shape may be irrelevant, as the only geometric distances used in the calculation are M, N and Q. The model can be adequately generalized to any convex shape as long as the distances M, N and Q can be estimated. In this case, the calculations would not change from those above.

As veins attach to the brain and skull from all directions and at various angles (but generally very close to 90°) [14, 15, 19], our assumption that the veins are orthogonal to both the brain and the skull is for clarity of mathematical calculations. Certainly, in both BEH and normal children, there exist numerous veins that are nearly orthogonal [15]. For these veins, our model offers a good estimate which is $R_s(BEH) > R_s(nor)$. For those veins that are farther from orthogonal, we can conceptually examine the veins in the normal condition and in BEH that are at corresponding angles. Here we can use the Cosine Law for obtuse triangles $V_f^2 = V_i^2 + T^2 - 2V_i \cdot T \cdot \cos\Theta$, where Θ is the obtuse angle between the sides V_i and T, T = the distance of translation in either condition). Since $R_s = V_f/V_i$, for equal angles and original CSF space width, we see intuitively that $R_s(BEH) > R_s(nor)$ (calculations not shown).

Radiologic studies of children with BEH show that the brain is a movable mass within the cranial vault. Bianco et al. [25] documented that a change in head position results in CSF redistribution and brain relocation within the skull in BEH. Averaging all movements and positions, then, one may presume that the brain may be more centrally located in BEH with CSF equally distributed throughout the SAS over the cerebrum. In this case, it might be more accurate to represent the BEH brain and skull as two concentric spheres, but with a greater SAS width than in the normal condition. It is clear, however, that the brain is necessarily somewhat tethered posteriorly by the falx and tentorium, and our experience with BEH is a tendency towards frontal/parietal SDH. The dural anatomy effectively creates CSF pockets: 2 spaces anteriorly and 3 spaces posteriorly (focus at falx/cerebellum), preventing completely uniform CSF distribution. We are presently refining our model to account for these anatomic constraints. These adjustments will also lend themselves to incorporating rotational forces, which Huang et al. [18] have indicated contribute more to bridging vein rupture than do translational forces. Anatomic limitations notwithstanding, our current model is an intuitive representation of the geometric differences between BEH and the normal condition based on the anatomy observed in common neuroimaging.

We have assumed that corresponding veins in BEH and normal children are of equal length. If we allow the lengths to be different, then either BEH veins are shorter than normal veins or BEH veins are longer. The former case is trivial, as the stretch ratio $R_s = V_f/V_i$ and, consequently, the vein failure rate will increase in BEH relative to the normal, confirming our findings.

When BEH veins are longer, one way to look at the relative likelihood of rupture in BEH versus normal veins is to consider a vein length for the normal V_i(nor) such that the resulting stretch ratio R_s(nor) is equal to the lesser of 1.5 or R_s(BEH), since the calculated stretch ratios for BEH are all greater than 1.5. Again by the geometry of the normal condition, using 1.5 as a conservative estimate and the equation $R_s(nor) = (2)^{1/2}/S_n$, we have $S_n = (2)^{1/2}/1.5 = 0.943$. That this number is less than 1 means that in order for normal veins to reach the ultimate stretch break point (1.5), they would need to be under tension at rest [they are at (0.943–1)·100% slack = –5.7% slack]. This seems unlikely, as constant venous stretch would tend to result in vein growth and laxity.

If, however, we allow the BEH veins to lengthen, then R_s(BEH) may fall below 1.5. According to our model, the determining factors for orthogonal vein overstretch ($R_s \geq 1.5$) and subsequent rupture seem to be the ratio of original distance translated and, of course, the slack factor. With reasonably generous estimates of 20% slack and 10 mm BEH translation, a stretch ratio of 1.5 requires an original BEH distance of 6.7 mm; or, restated, an N:M ratio of 2/3 (and 20% slack) is sufficient to allow risk of vein overstretch and rupture. Though it is not feasible to estimate the slack in individual veins in vivo, it is reasonable that in a rapidly growing space (such as the SAS of a child with benign external hydrocephalus), venous elasticity may be compromised if vein growth lags behind SAS growth. These veins with low slack factors may be more susceptible to overstretch.

Therefore, even if the vein lengths in BEH are greater than the vein lengths in the normal condition, for each

strain rate, R_s(BEH) will exceed R_s(nor). Inferring that greater stretch leads to greater vein failure rates, as do Huang et al. [18], this suggests a greater probability of SDH forming after minor head injury for children with BEH compared to normal children.

Clearly, vein strain/stretch varies not only between veins within an individual, but also between individuals, depending on the elasticity of a given vein. This study does not attempt to define a certain degree of impact that will cause a hematoma in all children with BEH but, rather, predicts a predisposition towards SDH in children with BEH relative to normal children. Moreover, because our model is based on geometry rather than physiology or biophysics, our model may apply not only to BEH, but also to other situations where the SAS/subdural space is enlarged and the surrounding fluid may redistribute about the brain, such as subdural hygroma [10]. Further examination of the geometric and anatomic conditions of children with widened extra-axial spaces in the context of impacts with rotational acceleration, variable impact directions and specific calculations for vein orientation will allow more conclusions about general trauma to the BEH head.

The prediction that a relatively minor impact that would not cause a SDH in a normal child may cause a SDH in a child with BEH or widened extra-axial spaces brings to focus an important issue regarding the nature of accidental versus nonaccidental pediatric head injury. A history of low-impact head injury has been felt to be consistent with CT findings of SDH in the absence of other signs of abuse [23, 26–28]. We would add that such a scenario is particularly possible in the presence of BEH or other causes of increased SAS. Practical implications of our model include a need for heightened awareness for the possibility of serious consequences of minor trauma in infants with BEH and, therefore, the possible need for precautions to prevent such injury.

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