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Cranial Venous Blood Cavitation: A Possible Mechanism of Traumatic Brain Injury Associated with Blast Wave exposure and Head Impacts

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Abstract:	Background: The commonly accepted theorized mechanisms for traumatic brain injury (TBI) are Shear/Strain and Cavitation theories: the former is purported to occur with 20-150 × g head accelerations in the sporting arena, while the latter is the predominant etiology of TBI in the setting of blast waves from high order explosives.1,2 Goeller emphasizes the research and present day theories surrounding cavitation in blast-induced Traumatic Brain Injury (bTBI), specifically implying that skull deformation is a significant factor causing cavitation.2 Prior authors contend that the stretching of axons (with resultant injury) is the likely pathophysiology of bTBI.3 The purpose of this mathematical exploration of slosh-based cavitation was to determine if it is a viable alternative to Shear/Strain theory, even at lower impacts levels.5 Specifically, we aimed to model slosh energy absorption, which is theorized to generate cavitation, in the setting of non-Newtonian fluid characteristics (i.e. blood, which has been typically modeled as Newtonian in TBI experimentation) to examine slosh as a proposed, or even preferred, mechanism for TBI. Our working hypothesis was that venous blood is more prone to cavitation (as opposed to the previously modelled water or CSF), as the compliance (expandability and collapsibility) of a lower pressure vascular system and valve-less cranial veins allow blood to move, unimpeded, in and out of the cranial-

	spinal space. Methods: We aimed to evaluate the forum of cavitation promoters and thresholds, thus evaluating the current understanding of cavitation as a potential mechanism for TBI in the military and tactical exposure as well as other forms of lower impact force TBI in general. The effects of vessel compliance, surface tension of the liquid, and non-Newtonian viscosity of the liquid (as a function of shear rate) were considered pertaining to the cavitation equation to determine the threshold impact forces that would incite cavitation in such cranial venous conditions. Findings: The resultant models lend credence to including slosh hemodynamics and the blood's Non-Newtonian properties in the modeling of TBI in human brains, and concluded that blood, given the right conditions, should cavitate in the cranial veins at impact levels of less than 85 × g force. In fact, current areas of study even include the pathophysiologic responses of blood, itself, to blast waves which may further elucidate the underlying mechanisms of blast-induced TBI (bTBI).7 Discussion: Prior authors have questioned the conventional cavitation metrics (cavitation number) and the thresholds that induce this phenomenon.6 Recent investigators have proposed a hydrodynamic physics term, slosh as a biological mechanism for cavitation-induced TBI. Specifically, this work is concerned with the effect of acceleration or imparted pressure waves on cranial blood and how the "room to slosh within a moving container" (or compliance of the vessel) can promote cavitation and injury. Sport induced TBI (sTBI) can lead to neurological dysfunction even at sub-concussive exposures,8,9 therefore investigating the potential that cavitation can occur at levels of force impartation commonly encountered during sport may also be critical to furthering our scientific understanding of TBI.
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Introduction

The pathological changes seen with traumatic brain injury (TBI), as led by Shear/Strain Theory,[10,11] have been plagued by the question of how the physics of a concussive wave can result in the chemical findings identified within the brain space following head impact exposure.[12] Shear/Strain theory alone may be unable to account for the strong chemical bond changes seen post TBI, especially at low impact force levels.[10,11] On the other hand, *cavitation* implosion—the rapid formation and collapse of vapor pockets in a fluid in response to pressure variation—has been studied extensively in nature, and can give rise to extreme temperatures and energy release.[13-16] In fact, it has been estimated that cavitation increases chemical reactivity by one million fold.[17]

Historically, the modeling of cavitation for generalizing to human biology has often simplified the fluid dynamics by modeling blood, a non-Newtonian fluid, as a Newtonian fluid such as water.[2,18-23] Compared to water, the density of blood in-vivo is not uniform. Instead, it is dependent on a multitude of host factors: the level of an individual's fitness, the presence of alcohol and fatigue, the organ of interest surrounding the blood, the biological containers housing it, and a person's age.[24] More importantly, water maintains a constant viscosity despite flow conditions, and this is termed "Newtonian" behavior. Conversely, the viscosity of blood is not constant and depends on "non-Newtonian" flow conditions.[25] This is an important distinction when considering the fluid dynamic outcomes in response to cranial impacts and the resulting head accelerations. Modeling non-Newtonian blood as a Newtonian fluid is likely a flawed approach, as the large number of blood cells and macromolecules in blood make it a polymeric solution, meaning the particles within the whole blood interact with each other, altering the rheological properties of the solution as a whole (Figure 1). For example, blood cells have negatively charged sialic acid molecules concentrated on their membranes which react with

blood proteins, such as fibrinogen and albumin. This underlies the increased and more variable viscosity of blood, in contrast to water. The unique cohesive properties of blood are well understood and utilized in other fields, such as forensics[26] yet seem to be neglected in the context of TBI.

Fig. 1.

One reason that this has implications for understanding mechanisms of TBI is that blood viscosity decreases as shear rate increases (i.e., blood is described as a "shear-thinning" non-Newtonian fluid). Shear is an observation of the rate at which two parallel planes, whether solid or fluid, move past each other, equaling relative velocity divided by distance between two moving planes ([m/s]/[m], units simplify to reciprocal seconds [s^{-1}]). Shear rate is not to be confused with shear stress, which equals force over unit area acting parallel to some surface element [N/m2].[27,28] In the context of fluid dynamics, shear is present between adjacent fluid layers, particularly if a force impacted on the fluid induces flow. In addition to blood, common shear-thinning fluids include ketchup, peanut butter, paint, and shampoo.[29]

Experimentally, blood is often assumed as Newtonian due to the Fåhræus–Lindqvist effect[30]—the decrease in apparent blood viscosity in smaller diameter blood vessels due to the formation of a plasma "erythrocyte-free" layer, which directs cells to the center of flow. The resulting lower viscosity (compared to whole blood) of the plasma layer at the blood-endothelium interface gives a reduced apparent blood viscosity. Literature supports that Newtonian modelling of blood globally underestimates wall shear stress within the vasculature, and non-Newtonian models further show increased sensitivity to changes in velocity metrics with increased vascular complexity – both conducive to cavitation.[5] It is further conceivable that interactions among erythrocytes and other blood constituents are still present in the flow located

centrally within the vessel,[31] and trauma/blast induced disruption of these interactions, may contribute to the likelihood of cavitation occurrence.[32]

Another reason blood is assumed to behave in a Newtonian manner is that above 100 s^{-1}, the viscosity appears to plateau (Figure 2).[33-35] This plateau region is termed *infinite-shear viscosity*. Blood *in vivo* experiences an average shear rate of 200 s^{-1}, reaching up to 1000 s^{-1} in the smallest vessels, both values clearly falling in the infinite-shear viscosity spectrum.[36] In the current work, we have identified two mathematical non-Newtonian models of blood viscosity: the Power Law viscosity model and the Carreau viscosity model.[37] Both characterize the viscosity of blood as nonlinear at the 100 s^{-1} shear rate region (Figure 2). However even if blood is operating at a seemingly "Newtonian" viscosity, it is again conceivable other, potentially not yet elucidated intermolecular relationships may be interrupted upon a dramatic pressure wave passing through the cranium, thus allowing blood to become less viscous, especially in regions of low shear rate (e.g., next to blood vessel walls). Such characteristics would promote blood-based cavitation at a more sensitive threshold than the Newtonian model for blood, as viscosity and shear-thinning properties intensify bubble behavior in cavitation.[33,38]

Studies that have previously explored cavitation as a mechanism of TBI have placed (or modeled) water in fully filled containers in an effort to mimic the skull and contents, and these studies do not account for the compliance of the neuro-vascular system and are, therefore, unable to account for the inherent cavitation "sloshing" that is now argued to occur.[18,22] In vivo, cranial venous vessels, with their valve-less, elastic, floppy walls, are 30 times more compliant than arterial vessels.[39] Thus, they would more readily allow fluids to separate into vapor cavities. Unfortunately, numerical evaluation for this compliance parameter cannot be easily considered, especially as it affects cavitation, and is further complicated by non-Newtonian implications that

remain mathematically problematic. In the context of venous conditions optimizing cavitation potential, the literature indicates that increasing pressures on a fluid medium reduces cavitation propensity, thus higher arterial pressures may allow less cavitation than lower pressured, highly compliant veins (average venous pressure is 2-6 mmHg compared to the average arterial pressure of 100 mmHg).[40-42] Specifically, pressure suppresses cavitation in a linear manner.[43]

The critical pressure for the onset of cavitation is commonly regarded as equivalent to the vapor pressure of the liquid, but it is somewhat less than, and a function of, surface tension and the radius of the microbubble nuclei.[44] As the surface tension of the liquid decreases, the cavitation bubbles become less stable and limited in size, while the energy of the implosion is reduced.[45] Notably, the surface tension of blood at 22 C is around 55 mN/m.[46] This is lower than water, which is about 72 mN/m at the same temperature,[47] and commonly used in modeling TBI.[18] This indicates that cavitation in blood is more likely (relative to water) due to the increased ability for fluid planes to slide along each other from shear-thinning viscosity. It is possible that water-driven models are more catastrophic (more energy released after bubble implosion) than those modeled with blood because of the lower surface tension; however, the energy released from blood cavitation would still be at a magnitude more likely to cause damage to surrounding neural tissue, resulting in TBI, as has been supported by recent investigators.[48]

If cavitation is considered a potential mechanism for TBI at low, humanly imparted impact levels, then a better understanding as to the mechanisms underlying cavitation in blood (and related biological fluids including cerebrospinal, interstitial and intercellular fluid)[48] is required. In light of this, the current report offers both the theoretical constructs and the resultant mathematical model for cavitation as a mechanism for TBI alterations. This is demonstrated through the simulation of a blood-like fluid inside a moving, compliant “sloshable” vessel, which

the authors purport to be a far better representation of the biological system in question. The current work proposes that the shear-thinning properties of blood will affect accuracy of predictions made for cavitation within blood by overestimating the impact force necessary to cause cavitation: cavitation in the non-Newtonian, shear-thinning model of blood will occur more readily than in the Newtonian model of blood. This is scientifically feasible, given: (1) blood in larger cranial vessels, such as those in the human skull, may be experiencing less than $100s^{-1}$ immediately prior to impact, (2) if the blood flow instantaneously changes direction as a result of an impact force to the skull, the resulting wide range of shear rates experienced by the fluid (blood) may allow its shear-thinning properties to significantly affect the potential for cavitation, and (3) interactions within blood are likely further disturbed as a result of high-impact forces and the infinite-shear viscosity of blood is not as accurate as a more specific non-Newtonian model for blood (which does not currently exist). We contend that upon initiating an impact leading to an acceleration of 100 g's, commonly experienced in football impacts leading to clinically indicated concussion,[49] flow (and thus shear) changes over a continuum and may exhibit all three regions of shear.

In the examples above, a shear-rate region of 1 s^{-1} to 100 s^{-1} would promote cavitation by facilitating the separation of fluid molecules, creating a vapor cavity. Further, as one considers the velocity of a pressure wave travelling through a non-Newtonian blood medium, the pressure wave should be affected by the associated fluid's viscous properties[50] and may, therefore, contribute to an increased likelihood of cavitation occurring in cranial blood vessels. This would again indicate that a Newtonian model of blood would wrongly overestimate the conditions leading to cavitation, and thus TBI.

Materials and Methods

We aim to theorize how the currently unavailable mathematical implications of (a) non-Newtonian blood viscosity and (b) quantified vessel compliance would produce more accurate estimations of the likelihood of cavitation occurring in cranial blood. Thus, in general, Newtonian assumptions were made for this analysis as the mathematical relationship of shear-thinning fluids such as blood and cavitation are not well understood.[51] In our modeling of an imparted TBI, the cranial vein and the blood it contains becomes "the system", and the pressures defined within the system must include the internal venous blood pressure (caused by the cardiac output and systemic vascular resistance) and a passing pressure wave due to an imparted cranial impact. Variables considered were: body/blood temperature, blood density, hematocrit, shear rate experienced by blood in the vein, blood and plasma viscosity, and impact force (*g*'s). We defined a "medium" vessel, for approximation purposes, as one that has a radius much larger than the radius of component cells; in otherwise smaller vessels the non-Newtonian rheology of the cells and cell constituents must be considered.[1,52]

For observation, two non-Newtonian models were used to estimate viscosity in addition to the Newtonian model for comparison. In the first, we created a "viscosity" MATLAB (Mathworks, Natick, MA) function to determine Newtonian, Carreau (non-Newtonian), and Power Law (non-Newtonian) viscosities for blood. These models have previously been used in blood viscosity studies.[35,36] For Newtonian:

$$\eta_{plasma} = 0.0013\ [\frac{Ns}{m^2}]$$

$$T_{body} = 310.15\ [K]$$

$$\eta_{Newtonian} = \eta_{plasma} \times \frac{1}{1-\alpha\varphi}\ [{}^{Ns}/_{m^2}], \qquad (1)$$

where

$$\alpha = 0.076 \cdot e^{[(2.49\varphi)+(\frac{1107}{T} \cdot e^{-1.69\varphi})]} \quad 17$$

$$\varphi = 0.5 \quad .$$

For Carreau:

$$\eta_{Carreau} = \eta_\infty + (\eta_0 - \eta_\infty) \cdot [1 + (\Lambda\dot{\gamma})^2]^{\frac{N-1}{2}} , \qquad (2)$$

where

$$\dot{\gamma} = shear\ rate\ [s^{-1}]$$

$$\eta_0 = 0.056\ [Pas]$$

$$\eta_\infty = 0.00345\ [Pas]$$

$$\Lambda = 3.313\ [s]$$

$$N = 0.3568 \quad .$$

For Power Law:

$$\eta_{Power\ Law} = \eta_0 \times \dot{\gamma}^{N-1}[Pa * s] , \qquad (3)$$

where

$$\dot{\gamma} = shear\ rate = 0 - 1000[s^{-1}]$$

$$\eta_0 = 0.035\ [Pas]$$

$$N = 0.6 \quad .$$

Fig. 2.

Viscosity was calculated as a function of shear rate experienced by blood under biological conditions, not during impact. This is partially because the shear rate upon impact is unknown.

However, it is logical that shear rate, upon impact, would be higher than resting shear rate conditions. Therefore, Figure 2 above provides insight to the following question: in normal biological conditions, what is the non-Newtonian viscosity of blood at different shear rates (i.e., different locations along the venous portion of the cardiovascular system)?

The "vessel" function written in MATLAB was used to determine the parameters of the vessel of interest, and had two outputs: (1) *bv* (for venous "blood vessel") was a matrix that contained components of density (for blood, the model could easily be expanded to include cerebral spinal fluid or interstitial fluid if modeled in a tube), radius of vein, average length for respective size vein, blood pressure, and initial velocity of blood, and (2) a velocity matrix that contained a range of final velocities based on initial blood flow velocity and impact force, analyzed from zero to one hundred *g*'s. Values used for the initial velocity of blood are comparable to results produced by anatomically correct, geometrically complicated venous models.[4] The function then produces the *bv* and *v_out* matrices based on the information in Table 1.[38]

Table 1.

Where radius = diameter/2, the following output matrix is produced: bv=[ρ,r,l,bp,v_in] where rho is density, r is radius, l is length, bp is blood pressure, and v_in is initial blood velocity. The acceleration due to gravity is 9.81 m/s^2 and duration of impact is assumed to be 0.015s.[50] The second output matrix produced is v_out, where the first row is 0-100 to indicate g-force, and the second row is the corresponding velocity after impact at that force which is later used in the cavitation equation.

To calculate cavitation number, the following were used:

$$P_{ambient} = P_{atm} + P_{BP}\ [Pa] \quad (4)$$

$$P_{vap} = e^{20.386-\frac{5132}{T}} \cdot \frac{101325}{760} + (5332 + 6666)[Pa], \quad (5)$$

where ambient pressure is the absolute blood pressure (note that a typical blood pressure reading is relative to the atmosphere), pO2 in venous blood was assumed 5332 Pa and pCO2 in venous blood was assumed 6666 Pa and these values were added to account for the effect of dissolved gases on vapor pressure of the solvent (blood), and

$$T_{body} = 310\ [K]$$

$$P_{atm} = 101325\ [Pa]$$

$$g = 9.81\ [\frac{m}{s^2}] \quad .$$

The cavitation equation:

$$\sigma = \frac{2 \cdot (P_{ambient} - P_{vap})}{\rho \cdot {v_{fin}}^2} \quad . \quad (6)$$

Results

Based on this present equation, for a medium-sized cranial vein, venous blood accelerated away from an erythrocyte or vessel wall, and when the change in velocity imparted by impact increased over 0.015 seconds at 85 *g*'s, up to a velocity of at least 12.5 m/s, cavitation occurred as the cavitation number dropped below 1 (Figure 3).

Fig. 3

These results evidence that a pressure wave induced by a cranial acceleration of 85 g's in a Newtonian fluid will result in cavitation; a non-Newtonian model would indicate the cavitation threshold being reached at a pressure wave which corresponds to an acceleration lower than 85 g's.

Discussion

The British scientific response to the World War II effort led to numerous medical advances, but few were as significant as the work performed on traumatic brain injury, with pioneers such as Hugh Cairns and Hylas Holbourn. They established that damage to the human brain "is a consequence of movements, forces, and deformations, interdependent, and strictly follow Newton's Laws of Motion".[53] The use of physical models, and the elucidation of the physics of the brain's response to injurious force vectors, established the stress and strain basis for the effective acceleration and duration of head impact. Many subsequent pioneers contributed to our understanding of TBI physics, including such researchers and clinicians as Gurdjian, Langfitt, Gennerelli, among others.[53-55] Gross, in 1958 first proposed another, different TBI mechanism in describing cavitation as the creation of local vacuum cavities in the brain and their ability to release energy upon spontaneous collapse.[56] While Hardy and others subsequently have agreed that cavitation effects may be operant, in many cases of TBI there has not been a general acceptance of the theory, in large part because cavitation cavities have never been visualized in vivo.[57]

In direct opposition to Holbourn and Cairnes' Newtonian view of TBI, blood within the cranium is non-Newtonian, has a higher dissolved gas content, and does not have constant viscosity as kinetic energy is imparted. Furthermore, Newtonian models for blood underestimate wall shear stress.[5] Based on these rheological characteristics, the current model indicates that blood inside a vessel, such as a vein, should cavitate more readily, or at lower accelerations, compared to a Newtonian model of blood. In addition, we propose that intracranial venous blood is not fully enclosed (no valves)[58] and thus can slosh into and out of the cranial space. This allows aliquots, or even molecules, of liquids to move away from each other, promoting vapor

cavities and resultant cavitation. We acknowledge this proposition will likely be difficult to model mathematically.

We assumed that the cavitation threshold for blood is 1 (equal to water); however, in reality the threshold would need to be higher in blood due to its lower surface tension. If we model water in vein/cranial conditions, it will cavitate at 85 *g*'s. Therefore, the important finding of the current effort is that blood can cavitate in the crania at some acceleration (impact) less than 85 *g*'s: interestingly, a force commonly seen in many sports-related collisions. The cavitation equation also needs to be expanded to include vessel compliance characteristic of veins, surface tension of the liquid, and viscosity of the liquid (as a function of shear rate) to accurately determine impact force that would cause cavitation in cranial venous conditions.

Overall vessel compliance is an important consideration for two reasons (in the neurosurgical literature, it is even given a classification called the "Compensatory Reserve Volume or Elastance").[59,60] First, it facilitates planes of fluid separating during impact at the nidus of bubble formation, leading to cavitation (Figure 2). Second, compliance of the vessel becomes relevant when discussing the specific mechanism of brain injury as a result of cavitation. Chen shows that when a cavitation bubble implodes on or very near to a compliant vessel surface, the surface can either distend or invaginate,[35] up to, but not necessarily rupturing, as energy from the bubble implosion is redirected as a result of the boundary's elasticity. This may explain why vessel rupture is not more commonly seen in concussion, especially if cavitation is a viable mechanism for neural tissue injury. Specifically, if vessels distend due to their compliance during cavitation implosion, it is possible that this transient physical expansion of the vascular tissue disrupts surrounding neural tissue, leading to concussion with intact vasculature and, furthermore, allows this phenomenon to be ultimately survivable. As

compliance increases, slosh energy may be transferred to the nearby neurons or other cells of the brain, causing damage to the brain tissue as a result of cavitation in the blood and despite the vessel walls remaining intact. Based on these properties of energy transfer, and the updated modeling of blood fluid dynamics in a vein-like vessel, we speculate that cavitation may underlie the structural, and especially, the chemical brain alterations associated with head impacts at levels much lower than previously thought. Based on our proposed model of blood cavitation, the cascade of brain alterations in response to head impact accelerations can occur at approximately 85 x *g* forces, and likely occur at accelerative forces of significantly less magnitude than previously considered. Cavitation Theory, not Shear Strain Theory, can ultimately provide clarity for understanding chemical cascades seen at relatively lower physical blast or concussive impacts.[12]

Conclusions

Concussions have been observed from sports-related impacts at approximately 60-160 g's. Our work shows that it is possible for cavitation to occur at 85 g's given historically attested Newtonian conditions, and we theorize the true non-Newtonian nature of blood contributes to "lowering the threshold" thus allowing cavitation to occur well below 85 g's (and consequent concussions to occur at these same accelerations/decelerations). These forces are exceeded by orders of magnitude during explosive blasts, therefore warrant consideration as a mechanism of not just blast-induced TBI but also impact related (such as sport induced sTBI) as well.

Future work to protect against TBI must include improvements to cranial study models in laboratory settings that take into account the above corrected influences of dissolved gases, non-Newtonian characteristics of fluids, movement of the modelled container (skull) and compliance. Despite the relationship between pressure and cavitation, it is likely that compliance, more so

than pressure, has the greatest effect on cavitation, as the change in blood pressure is negligible compared to $P_{ambient}$ and P_{vap} values. A novel jugular compression collar shows promise in affecting these compliance parameters to lower incidence of both sports-imparted and blast-induced TBI.[7] The mathematical relationship of cavitation in a shear-thinning fluid and compliance must be determined experimentally rather than theoretically. Regardless, decreasing the compliance of a vessel absolutely has effects on cavitation risks, and even though reflecting it mathematically remains problematic, we contend experimentation should be able to account for this variable going forward. We expect these corrected future experiments, taking these properties into account, will ultimately demonstrate cavitation at considerably lower than 85 × g forces and bring cavitation theories back into light even at concussive levels commonly evidenced in daily exposures and sport.

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Figure legends

Fig. 1. Blood flow in a vein. Depicts a general velocity profile typically observed in a moving fluid. A microscopic air bubble will serve as a nidus for cavitation upon a pressure wave due to an impact force causing acceleration of the head or an explosive blast wave. The initial blood flow velocity is assumed negligible compared to (likely turbulent) flow velocity after impact, therefore initial flow speed and direction with respect to vessel walls is not depicted.

Fig. 2. Three models of Blood Viscosity. Above 100s-1, blood is generally regarded as Newtonian, however the non-Newtonian Power Law model, and non-Newtonian Carreau model, show that there is some variability in viscosity modelling for blood. Blood vessels have shear range of 0-1000s-1 with the average being around 200s-1.

Fig. 3. Cavitation as a function of Velocity B (top). Cavitation as a function of Acceleration due to Impact Force (bottom). Acceleration denoted in g's.

Figure

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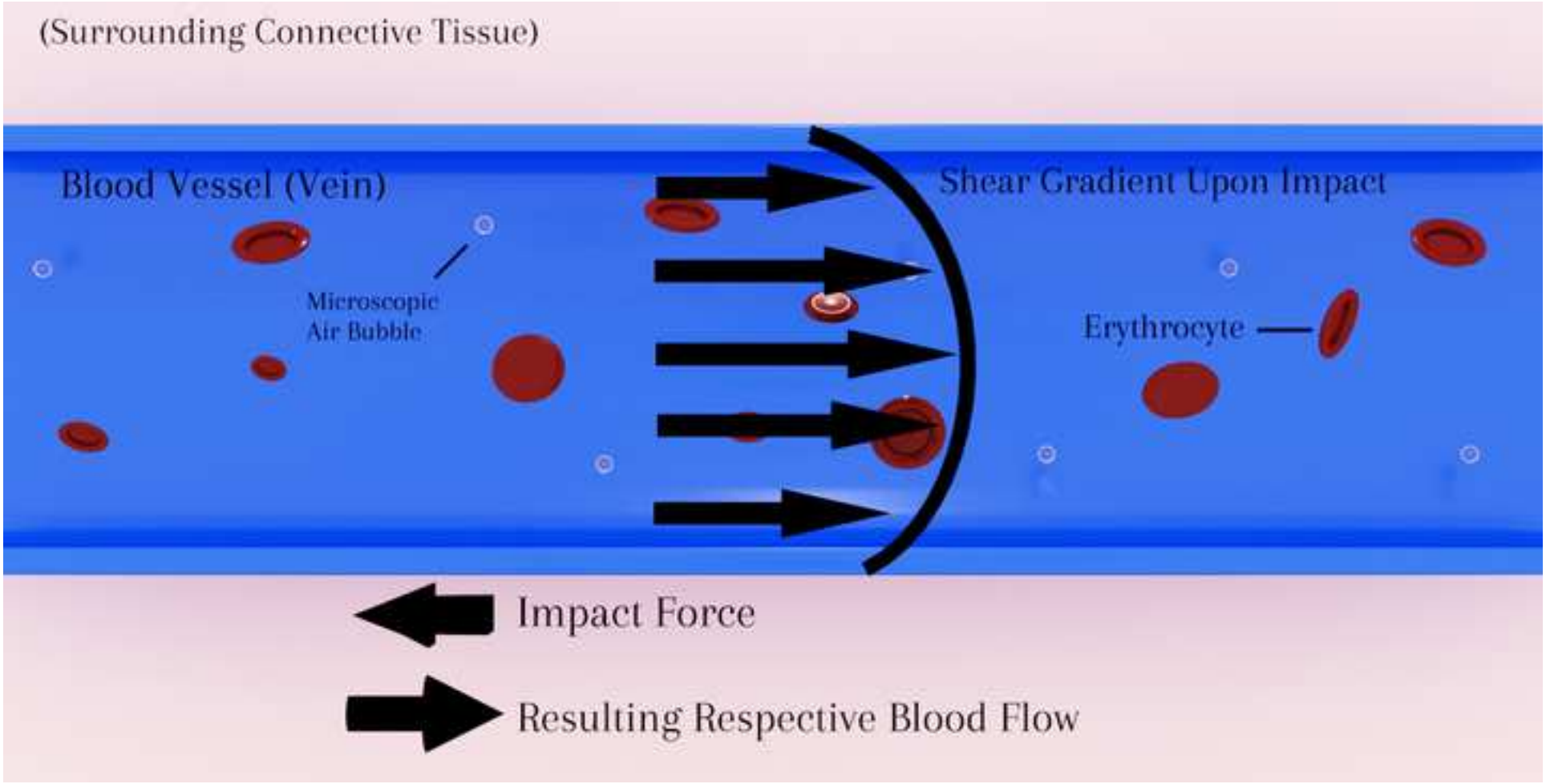

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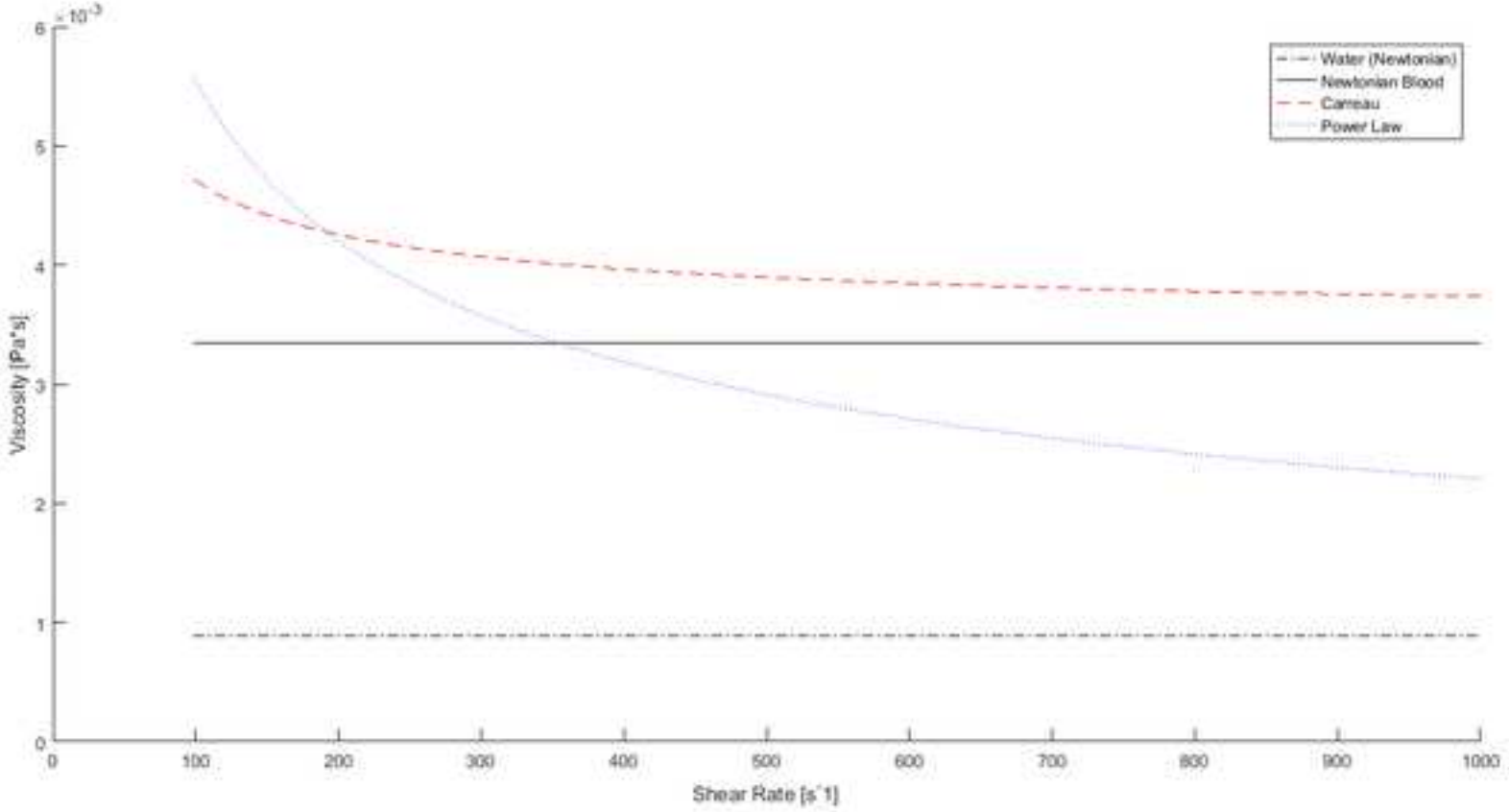

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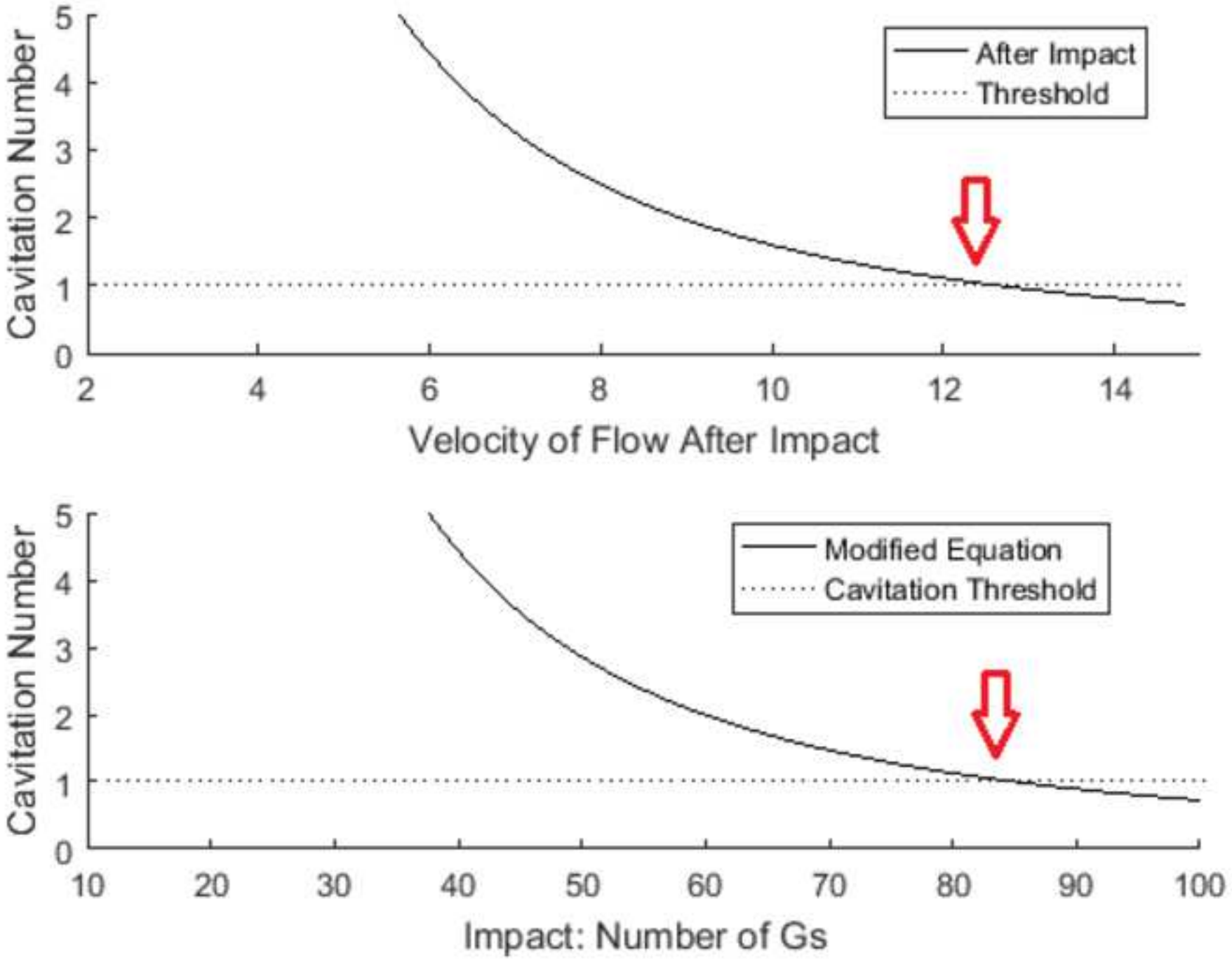

Table 1. Blood Vessel Parameters.

Size of Vessel	Density [kg/m^3]	Diameter [m]	Length [m]	Blood Pressure [Pa]	Initial Blood Velocity [m/s]
Small (capillary)	1060	9.00E-06	1.00E-03	3990	0.05
Medium (Venule)	1060	2.00E-05	7.00E-04	2261	0.07
Large (Vein)	1060	3.70E-03	1.00E-02	1330	0.11
Other (CSF in tube)	1069	3.70E-03	1.00E-02	-	negligible

Cover Letter

Monday, September 25, 2017

Military Medicine-AMSUS

Dear Editorial Board of *AMSUS*,

Please find enclosed the manuscript titled **"Cranial Venous Blood Cavitation: A Possible Mechanism of Traumatic Brain Injury Associated with Blast Wave exposure and Head Impacts."** We would like the manuscript to be considered for publication in ***Military Medicine-AMSUS***. We feel that the manuscript is suitable for consideration for publication in your journal. We hope you agree.

This manuscript represents original unpublished material and is not under consideration for publication elsewhere. This manuscript is the original work of the authors and all the authors have approved its submission. There is no potential conflict of interest. Specifically, there are no financial relationships with any manufacturers, including, but not limited to grants, honoraria, consulting fees, royalty fees, ownership, or support in preparation of the manuscript.

Thank you for your time and consideration.

Gregory D. Myer, PhD, FACSM, CSCS
Director of Research
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Abstract

Background: The commonly accepted theorized mechanisms for traumatic brain injury (TBI) are Shear/Strain and Cavitation theories: the former is purported to occur with 20-150 × g head accelerations in the sporting arena, while the latter is the predominant etiology of TBI in the setting of blast waves from high order explosives.[1,2] Goeller emphasizes the research and present day theories surrounding cavitation in blast-induced Traumatic Brain Injury (bTBI), specifically implying that skull deformation is a significant factor causing cavitation.[2] Prior authors contend that the stretching of axons (with resultant injury) is the likely pathophysiology of bTBI.[3] The purpose of this mathematical exploration of slosh-based cavitation was to determine if it is a viable alternative to Shear/Strain theory, even at lower impacts levels.[5] Specifically, we aimed to model slosh energy absorption, which is theorized to generate cavitation, in the setting of non-Newtonian fluid characteristics (i.e. blood, which has been typically modeled as Newtonian in TBI experimentation) to examine slosh as a proposed, or even preferred, mechanism for TBI. Our working hypothesis was that venous blood is more prone to cavitation (as opposed to the previously modelled water or CSF), as the compliance (expandability and collapsibility) of a lower pressure vascular system and valve-less cranial veins allow blood to move, unimpeded, in and out of the cranial-spinal space.

Methods: We aimed to evaluate the forum of cavitation promoters and thresholds, thus evaluating the current understanding of cavitation as a potential mechanism for TBI in the military and tactical exposure as well as other forms of lower impact force TBI in general. The effects of vessel compliance, surface tension of the liquid, and non-Newtonian viscosity of the liquid (as a function of shear rate) were considered pertaining to the cavitation equation to determine the threshold impact forces that would incite cavitation in such cranial venous conditions.

Findings: The resultant models lend credence to including slosh hemodynamics and the blood's Non-Newtonian properties in the modeling of TBI in human brains, and concluded that blood, given the right conditions, should cavitate in the cranial veins at impact levels of less than 85 × g force. In fact, current areas of study even include the pathophysiologic responses of blood, itself, to blast waves which may further elucidate the underlying mechanisms of blast-induced TBI (bTBI).[7]

Discussion: Prior authors have questioned the conventional cavitation metrics (cavitation number) and the thresholds that induce this phenomenon.[6] Recent investigators have proposed a hydrodynamic physics term, slosh as a biological mechanism for cavitation-induced TBI. Specifically, this work is concerned with the effect of acceleration or imparted pressure waves on cranial blood and how the "room to slosh within a moving container" (or compliance of the vessel) can promote cavitation and injury. Sport induced TBI (sTBI) can lead to neurological dysfunction even at sub-concussive exposures,[8,9] therefore investigating the potential that cavitation can occur at levels of force impartation commonly encountered during sport may also be critical to furthering our scientific understanding of TBI.