2006

A new biomechanical head injury criterion

Charles F. Babbs
Purdue University, babbs@purdue.edu

A NEW BIOMECHANICAL HEAD INJURY CRITERION

CHARLS F. BABBS, MD, PhD

Department of Basic Medical Sciences, 1246 Lynn Hall,
Purdue University, West Lafayette, IN 47907-1246 USA
babbs@purdue.edu

Abstract

This paper presents a new analysis of the physics of closed head injury caused by intense acceleration of the head. At rest a 1 cm gap filled with cerebrospinal fluid (CSF) separates the human brain from the skull. During impact whole head acceleration induces artificial gravity within the skull. Because its density differs slightly from that of CSF, the brain accelerates, strikes the inner aspect of the rigid skull, and undergoes viscoelastic deformation. Analytical methods for a lumped parameter model of the brain predict internal brain motions that correlate well with published high-speed photographic studies. The same methods predict a truncated hyperbolic strength-duration curve for impacts that produce a given critical compressive strain. A family of such curves exists for different critical strains. Each truncated hyperbolic curve defines a head injury criterion (HIC) or concussive threshold, which is little changed by small offsetting corrections for curvature of the brain and for viscous damping. Such curves predict results of experimental studies of closed head injury, known limits for safe versus dangerous falls, and the relative resistance of smaller versus larger animals to acceleration of the head. The underlying theory provides improved understanding of closed head injury and better guidance to designers of protective equipment and to those extrapolating research results from animals to man.

Keywords: acceleration, biomechanics, brain, concussion, deformation, diffuse axonal injury, HIC, impact, shear, strain, threshold, tolerance

1. Introduction

Head injuries are common consequences of auto accidents, falls, direct blows to the head, bicycle accidents, fights, and participation in contact sports[1, 2]. Nearly two million persons with head injury are medically attended and over 300,000 are hospitalized annually in the USA [2]. One third of these injuries are severe, leading to prolonged coma, permanent neurological impairment, or death[3-6]. The majority of head injuries in man[2, 6, 7] and in experimental animal models[8, 9] are mild or moderate, and are produced by acceleration of the head only—without skull fracture[9]. Such non-penetrating head injuries result in transient loss of consciousness and usually no permanent neurological deficits. However, repeated mild head injuries (concussions) can and do lead to lasting decrements in problem solving ability, memory, and personality, as well as to anatomical lesions in the brain[10]. This paper addresses the question of how to specify the threshold between safe and dangerous levels of head acceleration in situations such as contact sports, vehicle crashes, and falls. This question arises for designers protective headgear, seat restraints, car and aircraft interiors, and the like, who must rely on head injury criteria[11-16] that specify physical parameters of harmful blows, such as critical energy, impulse (force x time)[11, 17] or acceleration[16, 18]. Such criteria help designers create equipment, rules, and operating procedures that keep the intensity of impacts to the head below the concussive threshold.

It is clear that there must be a tradeoff between the amplitude and the duration of acceleration required to produce closed head injury. Very brief intense accelerations produce the same change in head velocity, as do more moderate, prolonged accelerations. Thus one needs to generate a strength-duration curve describing harmful levels of acceleration as a function of impact duration (Figure 1). Points above the curve are considered dangerous; points below the curve are considered tolerable, if not completely safe. The farther a point is from the curve on either side, the safer or more dangerous the corresponding impact.

The classical head injury criterion of this type was generated by fitting a power function to impact tolerance data from animals and a limited number of humans for whom measured accelerations were available in the year 1971[16]. It is widely referenced and has become known as "the" head injury criterion or HIC, although there are alternative versions[11-16]. The formula for the classical Versace HIC[16] is $a^{2.5}\,\tau = 1000$, where the variable, a, is time averaged external acceleration of the head in units of Gs, the variable, τ, is the duration of head acceleration in seconds, and value 1000 is a constant in units of $G^{2.5}$-sec that defines the concussive threshold. This particular head injury criterion specifies a strength-duration curve of the form,

$$a = \frac{16}{t^{0.4}},$$

which is plotted in Figure 1.

The fractional exponent 0.4 ~ 1/2.5 derives from a log-log curve fitting procedure based upon limited data. There is no theoretical meaning to the power, 2.5. It could have just as well turned out to be 2.13 or some other number. It would be intellectually satisfying to find a head injury criterion that is directly related to the underlying physics and the actual mechanisms that cause deformation of the brain. Such an HIC could be extended with validity to impact durations not explicitly tested, to animal models of varying size, and in some cases to rotational as well as to linear accelerations.

Although we generally intuit that the farther a point is below the HIC curve, the less damaging to the brain would be the corresponding impact, purists would argue that only points on the curve itself have meaning, indicating a line of separation between measurably harmful blows, using the methods of the particular studies used to create the curve, and safer blows that did not show damage using these same methods. If a revised HIC curve were theoretically related to the degree of deformation of the brain, then one could generate a family of parallel curves representing different degrees of deformation. In this case it would be more justifiable to interpret any point in acceleration-duration space as being associated with a particular degree of brain distortion and hence potential injury.

Accordingly, the objectives of the present study were to determine the true physical relationship between whole head acceleration on the one hand and the maximal distortion within brain on the other hand, and from such an understanding to specify a strength-duration curve for any chosen maximal distortion. To do this one needs a theory describing quantitatively the maximal strain within the brain as a function of whole head acceleration. In this paper we show that such a theory exists, that it can be extended to arbitrarily long durations of acceleration, that it holds to good approximation for the curved geometry of the brain and skull, and that the predicted deformations are not substantially influenced by viscous forces within the brain. Here we shall focus on closed head injury not resulting in skull fracture, that is, injury to the brain occurring when the skull is regarded as a hollow rigid body.

2. Theory and Methods

2.0 Approach

Harmful blunt impacts can last for a wide range of durations from about 2 to 2000 msec[16]. Subsequent motion of the brain inside the skull lasts several seconds and is rarely seen. Only a few studies, using high-speed photography through a transparent plastic calvarium or high speed fluoroscopy of implanted radiodense pellets [8, 19-21], have examined the actual motion of the brain during closed head injury in animals. Fortunately mathematical analysis and modeling of the skull and brain in response to known pulses of head acceleration allow one to study a wide variety of conditions that are difficult, impossible, or unethical to reproduce in animals or humans[22]. This mathematical approach is ideal for developing an improved head injury criterion.

A person standing in a rising elevator feels a tug of artifical gravity as the elevator accelerates upward. Within the internal frame of reference of the elevator, the passenger experiences acceleration toward the floor of the elevator that is equal and opposite to the upward acceleration of the elevator in the shaft[23]. Acceleration of this local frame of reference produces artificial gravity, which by all possible experiments conducted within the elevator is indistinguishable from ordinary gravity. The effect of whole head acceleration on the brain is similar to the effect of acceleration of a rising elevator on its occupants. In this case the skull is analogous to the walls of the elevator, and the brain is analogous to a passenger in the elevator. However, since the brain is surrounded by and suspended in clear aqueous cerebrospinal fluid (CSF), the acceleration felt by the brain is attenuated by about 95 percent, since the brain weighs only about 5% as much in water as it does in air[22]. Nevertheless, considerable isovolumic deformation of the brain (which is quite soft) can take place, as the whole head is accelerated.

The nature of brain deformation during closed head injury has been revealed using analytical methods and computer models[22], coupled with remarkable experiments conducted years ago in animals fitted with transparent skull caps and studied with high speed photography[19, 21]. During brief, intense impacts the brain's velocity toward the skull increases almost instantaneously (within 1/50's of a second or less) to a velocity of tens of centimeters per second along the axis of the blow. Within another 10 msec or so the brain traverses the CSF gap and crashes into the rigid skull. The impact initiates a strain wave that traverses the brain substance at a much higher velocity, near 300 cm/sec, causing compression in one dimension and elongation in the other two perpendicular dimensions. A body of literature[24-26] suggests indirectly that it is probably the lateral expansion rather than the compression itself that damages brain substance, causing lysis of axons and damage to capillary blood vessels with micro-hemorrhaging or bruising. These are the classical lesions of closed head injury[3, 4, 9, 27, 28]. Here we describe analytically the motion of a soft, viscoelastic body such as the brain, as it is accelerates toward a rigid wall such as the skull, deforms, and then rebounds. In turn, we define the intensity and duration of acceleration associated with a given degree of maximal strain.

2.1 Brain models

The present analysis is done with strict adherence to Newton's Laws of Motion, together with published values for the material properties of the brain. In addition we adopt simplified representations of the geometry of the skull, subarachnoid space, and cranial cavity. We regard the brain as a uniform viscoelastic mass, suspended in water density cerebrospinal fluid (CSF) and residing in an internal frame of reference within the rigid skull. Definitions of symbols used in the analysis are provided in Table 1.

For simplicity, we shall consider the brain to be of homogenous density and elasticity, in keeping with experimental observations [29, 30]. We also assume that the brain is isovolumic (i.e. Poisson's ratio = 0.5), so that neither brain volume or CSF volume changes during or immediately after impact. Viscoelastic properties of brain have been rather well studied over the past 30 years, and consensus values for Young's modulus of elasticity and for an analogously defined energy loss modulus can be gleaned from the literature (Table 2). Despite rather large variability in published values, these data provide a basis for a standard model of a typical brain that is sufficient for present purposes. Brain density data are more consistent. They are summarized in Table 3. For the following theoretical work we take a typical value of brain stiffness as 10000 Pa and a typical value of brain density as 1046 kg/m^3. Water density is 1000 kg/m^3. The maximal internal fronto-occipital diameter of the human skull is taken as 18 cm.

2.2 Acceleration in internal vs. external frames of reference

In this problem it is helpful to distinguish clearly two frames of reference for motion. The first is the internal frame of reference within the skull. This frame of reference is important because it is the relative motion of brain with respect to the skull that is responsible for closed head injury. The second is the external frame of reference in which the head, neck, and body move. This frame of reference is important because an external blow or fall causes movement of the whole head in this frame. Acceleration of the rigid skull by an amount a(t) produces a corresponding artificial gravity within the skull of -a(t).

Because the brain is suspended in aqueous CSF, its buoyancy must be accounted. If ρ_1 is CSF density, ρ_2 is brain density, and V_2 is brain volume, then the force of gravity on the brain is $\rho_2 V_2[-a(t)]$, and the buoyancy of the brain is $-\rho_1 V_2[-a(t)]$. Applying Newton's second law of motion (Force = mass x acceleration) to the whole brain and ignoring drag[#],

$$(\rho_2 - \rho_1)V_2[-a(t)] = \rho_2 V_2 g(t), \qquad (1)$$

[#] It is reasonable to ignore the effects of viscous drag on the brain from CSF water, because the distance traveled by the brain through CSF is short and brain speeds relative to the skull are limited over this distance.

where g(t) is the acceleration of the brain with respect to the skull in the direction parallel to whole head acceleration. In turn,

$$g(t) = -\frac{(\rho_2 - \rho_1)}{\rho_2} a(t) \qquad (2)$$

in the internal frame of reference of the skull. This acceleration is substantially less than the acceleration, a(t), of the head as a whole in magnitude and opposite in direction. If brain and CSF density were perfectly matched, the acceleration of the brain with respect to the skull would be zero. In reality, brain density is approximately 1.05 and CSF density is approximately 1.00[29, 31, 32] (Table 3), so internal brain acceleration is about 5 percent of external head acceleration.

In cases of rotational accelerationof the skull (Figure 2) we have for internal coordinates x and y and neck radius r_{neck} , defined in Figure 2 the local components of internal acceleration

$$g_x(x,y,t) = g(t)\left(\frac{r_{neck} + y}{r_{neck}}\right) \quad \text{and} \quad g_y(x,y,t) = g(t)\left(\frac{x}{r_{neck}}\right) \qquad (3)$$

This two-dimensional acceleration is considered in detail in reference 22, which shows that the major component of brain motion is in the x direction. Here we consider a simplified one dimension case of linear brain acceleration along the axis of the blow, when x, y, and the distance moved by the head during the actual blow are small with respect to r_{neck}.

2.3 Three ranges of acceleration duration

To further reduce the problem, it is helpful to consider three ranges of duration for whole head acceleration. These can be termed short, intermediate, and long duration impacts. Short duration accelerations terminate while the brain is moving toward the skull and before it has hit the inner wall of the skull. Intermediate duration accelerations terminate after the leading edge of the brain has reached the skull but before the induced strain wave has traveled one full brain diameter along the axis of acceleration. During this time the brain becomes increasingly compressed against the side of contact. Long duration accelerations last even longer than this time of maximal compression. They pin the brain against the skull and, as long as acceleration persists, prevent recoil of the brain toward the opposite side of the skull. Recoil can occur after termination of long duration accelerations.

2.4 Short duration impacts

2.4.1 Motion before brain-skull contact

Computation of brain motion with respect to the skull is straightforward for external head acceleration of magnitude a(t) and duration τ, as long as τ is less than the critical duration, $\hat{\tau}$, when the leading edge of the brain hits the skull. Here the critical duration $\hat{\tau} = \sqrt{2s/g}$, ignoring viscous drag from the CSF, where s is the width of the normal CSF gap between the brain and the inner aspect of the skull and

$$g = \frac{(\rho_1 - \rho_2)}{\rho_2} \cdot \frac{1}{\tau} \int_0^{\tau} a(t)dt, \qquad (4)$$

is the mean internal acceleration of the brain with respect to the skull. Use of the mean acceleration over time to represent blows to the head is well precedented in the literature[16] and is sufficient for present purposes. Here CSF width, s, is a given anatomic parameter of the system, which is approximately equal to 1 cm in adult humans. For short duration blows the instantaneous velocity of the brain is g t for times, t, less than τ, after which brain velocity remains constant. The final velocity of the whole brain toward the skull in the internal frame of reference is then simply $v_0 = g\tau$, where the subscript 0 refers to the instant of brain-skull contact (Figure 3, top).

2.4.2 Motion after brain-skull contact

After brain-skull contact there is propagation of a compressive strain wave through the whole brain, accompanied by lateral expansion in dimensions orthogonal to the compression, according to Poisson's ratio. Note that strain waves discussed herein are not the same as sound waves or pressure waves propagating through tissue water. Strain waves are waves of deformation within a soft elastic material. These waves propagate much slower than do sound waves in water. In their 1994 textbook, ***The Mechanisms of Continua and Wave Dynamics***, Brekhovskikh and Gancharov[33] have described the exact nature of the compressive strain wave in a column of elastic material impacting a rigid wall at constant velocity, v_0. They show that for a column of density, ρ, having uniform stiffness (Young's modulus of elasticity), E, and hitting a rigid wall with initial velocity, v_0, a wave of compressive strain is propagated through the column in a particular last-in/first-out pattern.

Figure 3 illustrates Brekhovskikh—Gancharov (or B-G) compression of a uniform one-dimensional model of the brain. At time t = 0 an elastic column of length, L, hits a rigid wall with initial velocity v_0. The wall acts on the column with a force that initiates a strain wave, which propagates along the column with velocity, $c = \sqrt{E/\rho}$. For this idealized one-dimensional case with no viscous damping or energy loss, the compression is of uniform degree in the compressed region and is zero elsewhere. A rectangular wave of

compression and lateral expansion travels from the wall toward the free end of the column. At time t = L/c the entire column is uniformly compressed. The amount of compressive strain is simply v_0/c. Because we can compute $v_0 = g\tau$, as just described, it is a simple matter to find the value of compressive strain, v_0/c. Moreover, if the brain material is isovolumic during deformation (Poisson's ratio = 0.5), then the expansive strain perpendicular to v_0 is equal to $0.5v_0/c$.

Thereafter, for times, t, approximately in the range $L/c < t < 2L/c$ there is recoil, in reverse order, beginning with the free end. For times $t > 2L/c$ the entire column recoils with velocity v_0. In this idealized case, our brain model, having suffered a "coup" would drift at velocity $-v_0$ toward the other side of the skull where it would undergo a mirror image deformation or "contrecoup". Without energy loss due to damping, the cycle would be repeated indefinitely.** This one-dimensional analytical model captures several essential aspects of brain motion in closed head injury.

A recent paper utilizing two dimensional finite element models of the brain confirms the essential correctness of the Brekhovskikh—Gancharov analysis of short duration blows to ellipsoidal head and brain models[22]. Compressive strain waves propagate through the entire brain, just as outlined in Figure 3. This predicted motion also is remarkably similar to that observed directly through clear plastic (Lexan) calvaria in rare experimental studies of monkeys subjected to controlled head injury[19, 21]. In Sections 2.5 through 2.9 we extend this analysis to longer duration impacts, to viscoelastic brain material, and to non-rectangular geometries. It is helpful first to specify the strength duration curve for a simple B-G elastic body in response to short-duration impacts.

** In the absence of damping it is easy to show conservation of energy at maximum compression, when axial brain length is diminished by amount ΔL. The stored energy at maximum compression is $U = \frac{1}{2}k(\Delta L)^2$, where $k = \frac{EA}{L}$ is the spring constant for the elastic material of length L and cross section A, and $\Delta L = \frac{v_0}{c}L = v_0 L\sqrt{\frac{\rho}{E}}$ at maximum compression. Hence, $U = \frac{1}{2}\frac{EA}{L}\frac{v_0^2 L^2 \rho}{E} = \frac{1}{2}mv_0^2$, which is the kinetic energy of the original bar of total mass, m, moving at v_0 toward the wall. A similar approach shows conservation of energy at all times during a cycle of impact, compression, and recoil.

2.4.3 Strength-duration curve for a one-dimensional brain

Suppose there is some critical level of strain ε^* that produces clinically significant concussion and that ε^* is a constant of normal brain anatomy and physiology. In this case we can characterize accelerations as harmful or not according to whether they can be expected to produce strain of ε^* or greater. For short duration pulses, which last less than the time it takes the brain to traverse the CSF gap, it is easy to find the strength-duration curve for combinations of acceleration amplitude and acceleration duration that produce ε^* and are therefore harmful. In particular,

$$\varepsilon^* = \frac{v_0}{c} = \frac{g\tau}{\sqrt{E/\rho}} = g\tau\sqrt{\frac{\rho}{E}} \quad \text{or} \tag{5a}$$

$$g = \left(\varepsilon^* \sqrt{\frac{E}{\rho}}\right)\frac{1}{\tau}, \tag{5b}$$

which is a simple hyperbola, relating mean acceleration, g, to impact duration, τ. Expression (5b) specifies combinations of intensity and duration that will produce strain ε^* in brains having material properties E and ρ. This hyperbola in acceleration-duration space represents the theoretical head injury criterion for short duration impacts.

Here we assume that large compressive strains and the associated lateral expansions are the injurious mechanical events during closed head injury. (For curved brain models strain concentration at the site of impact also happens, as explained in Section 2.8.) However, we also know that some brain acceleration occurs during normal daily activities such as running and jumping, which is evidently harmless, and also that axons of peripheral nerves tolerate a certain degree of bending and stretching attendant to daily life. Accordingly, there must be a threshold for truly injurious strain, ε^*. The narrowness of the width of the CSF surrounding the brain limits ε^* to about 40 percent compression/20 percent expansion. That is, since the skull is rigid, the brain can only be compressed and expand so much. Perhaps for this reason single concussions suffered without damage to the skull are usually not lethal. However, we do know that some non-penetrating blows to the head can be dangerous and symptomatic, suggesting that there is a pathological value of ε^* that is less than the anatomic limit.

2.5 Intermediate duration impacts

A slightly different pattern of compression can happen with longer duration blows, when the external head acceleration persists beyond the time required for the brain to make contact with the inner aspect of the skull. In this case there can be relatively more distortion of the side of the brain striking the skull.

2.5.1 Stress and strain

When the duration of acceleration extends beyond first contact, one can use the principle of superposition for strain waves to find the net stress and strain at the contact point[33]. The net strain is the sum of the strain produced by an elastic rod hitting the wall at v_0 with no gravity (case 1, as in Figure 3) and the strain produced by the added force built up as a result of gravity (case 2) after the brain contacts the skull. One can visualize case 2 by imagining a person who gently places a cube of gelatin on a table and then lets go, allowing it to sag toward an equilibrium shape under 1G. From Brekhovskikh—Gancharov analysis we know that, in response to any force on the bottom surface of the cube, a strain wave will travel through the gelatin with velocity $c = \sqrt{E/\rho}$. Now consider the one dimensional brain model. If the strain wave of distortion travels distance, $x = c(t - \hat{\tau})$, out from the wall, then the weight of the collapsed brain tissue on the wall, that is the extra force for artificial gravity g > 0, is ρAxg. In turn, the total contact stress at post contact time, $t - \hat{\tau}$, is that caused by the initial velocity, as in case 1 for short duration impacts, plus the extra stress caused by gravity persisting after time $\hat{\tau}$, or

$$\sigma_c = E\frac{v_0}{c} + \frac{\rho A x g}{A} = E\frac{v_0}{c} + \rho c(t - \hat{\tau})g \qquad (6a)$$

for post contact times $t - \hat{\tau} < L/(c + v_0 + g(t - \hat{\tau}))$. Noting that $E/\rho = c^2$, we have

$$\sigma_c = E\frac{v_0}{c} + \frac{E}{c}(t - \hat{\tau})g. \qquad (6b)$$

The strain at the point of contact, which is the maximal strain in this weighted system during propagation of the outbound strain wave across brain length, L, is given by contact stress divided by Young' modulus, E, or

$$\varepsilon_{max} = \frac{v_0}{c} + \frac{g}{c}(\tau - \hat{\tau}) \qquad (7)$$

for accelerations of duration τ where $\hat{\tau} < \tau < \hat{\tau} + L/(c + v_0 + g(\tau - \hat{\tau}))$. This is the maximum strain in the model during propagation of the outbound strain wave under g. To specify the time frame for (7) in terms of given system parameters, we note for CSF width, s, we have $\hat{\tau} \approx \sqrt{2s/g}$ and $v_0 \approx g\hat{\tau}$. So the contact strain in (7) for intermediate duration impacts happens for approximate durations $\sqrt{2s/g} < \tau < \sqrt{2s/g} + L/(c + g\tau)$.

2.5.2 the extended strength duration curve

During the outbound strain wave for a critical harmful strain, ε^* , we have from Equation (7) $c\varepsilon^* = v_0 + g\tau - g\hat{\tau}$. But $v_0 \approx g\hat{\tau}$ under the prevailing artificial gravity, g , and so the strength-duration curve for a critical harmful strain, ε^* , can be written to good approximation by $c\varepsilon^* = g\tau$, or

$$g = \frac{c\varepsilon^*}{\tau}, \text{ for } \underline{\text{all}} \quad \tau < \hat{\tau} + L/(c + g\tau). \tag{8}$$

Therefore, the strength-duration curve for the concussive threshold remains hyperbolic for both short and intermediate duration impacts!

It is helpful to specify the upper bound of impulse duration in (8)

$$\tilde{\tau} = \hat{\tau} + L/(c + \tilde{g}\,\tilde{\tau}) \tag{9a}$$

in terms of the dimensions and material properties of the brain. To do this we note that for any particular threshold, ε^*, we have

$$c\varepsilon^* = \tilde{g}\,\tilde{\tau}\,. \tag{9b}$$

For the no-drag case the CSF thickness

$$s = \frac{1}{2}\tilde{g}\,\tilde{\tau}^2\,. \tag{9c}$$

Combining (9a), (9b), and (9c) to eliminate the variables $\tilde{g}$ and $\hat{\tau}$, we have

$$\tilde{\tau} = \frac{1}{c}\left(\frac{L}{1+\varepsilon^*} + \frac{2s}{\varepsilon^*}\right) = \sqrt{\frac{\rho}{E}}\left(\frac{L}{1+\varepsilon^*} + \frac{2s}{\varepsilon^*}\right). \tag{10}$$

Expression (10) gives the extent of the hyperbolic region of the strength duration curve in terms of fundamental geometric and mechanical properties of the brain model.

Thus the strength-duration curve during outbound propagation of the strain wave ($\hat{\tau} < \tau < \tilde{\tau}$) is an extension of the hyperbolic strength-duration curve for short impact durations $\tau < \hat{\tau}$. This is a helpful, simplifying result. However, for intermediate duration impacts the degree of compressive strain is not uniform across the length, L, of the brain. Because of the added effect of persistent acceleration, there is instead a gradient of compressive strain, which is greatest at the wall and diminishes with distance from the wall. For the purpose of defining harmful impacts, we are interested in the maximal distortion, ε^*, anywhere in the brain. Accordingly, to generate a head injury criterion it is

sufficient to use the maximal strain at the point of contact as a measure of undesirable distortion.

2.6 Long duration impacts

For durations of acceleration greater than $\tilde{\tau}$, as defined in (10), the brain remains pinned against the rigid skull under the force of g. There are small oscillations in strain associated with internal reflection of the strain wave. The maximal strain experienced by the brain at any time remains the same as that given by (7) for t = $\tilde{\tau}$, even for indefinitely long internal acceleration. Hence we can sketch the entire strength-duration curve for times from near zero to infinity by combining expressions (8) and (10), that is,

$$g = \frac{c\varepsilon^*}{\tau} \text{ for } 0 \le \tau \le \tilde{\tau},$$

$$g = \frac{c\varepsilon^*}{\tilde{\tau}} \text{ for } \tau > \tilde{\tau}. \qquad (11a)$$

Recall that lower case g represents time-averaged acceleration of the brain surrounded by CSF within the skull. If we use the variable $a(\tau)$ to represent mean acceleration of the head with respect to an external frame of reference over impulse duration, τ, then using Equation (2) we have $g = a(\tau)(\rho_1 - \rho_2)/\rho_2$. In terms of external acceleration of the head, which is the usual way of specifying an HIC,

$$a(\tau) = \frac{\rho_2}{\rho_1 - \rho_2}\frac{c\varepsilon^*}{\tau} \approx 20\frac{c\varepsilon^*}{\tau} \text{ for } 0 \le \tau \le \tilde{\tau},$$

and

$$a(\tau) = \frac{\rho_2}{\rho_1 - \rho_2}\frac{c\varepsilon^*}{\tilde{\tau}} \approx 20\frac{c\varepsilon^*}{\tilde{\tau}} \text{ for } \tau > \tilde{\tau} \qquad (11b)$$

2.7 Estimating the effect of viscous damping

Brekhovskikh—Gancharov theory assumes that the elastic rod impacting a rigid surface in Figure 3 is purely elastic. Now let us regard the elastic rod as being composed of parallel spring and damper elements connected in series (a Voight/Maxwell body). Assume that the various dampers in series act as an equivalent damper with viscous loss modulus, D, cross sectional area A, and length L. D is a material property of brain tissue, which is defined analogously to Young's modulus of elasticity, with a value on the order of 200 kg/m/s[22]. The total work required to compress the equivalent damper may be estimated as average force exerted by the equivalent damper, multiplied by the distance moved. Since the velocity of tissue diminishes from v_0 to zero during compression, let us estimate

average force as one half v_0 multiplied by the damping constant, $\mu = DA/L$. Then for short duration impacts the product of average force and distance, namely $\frac{1}{2} v_0 \frac{DA}{L} \cdot L\varepsilon$, equals the energy absorbed by the damper. Now suppose that the strain at the time of maximum compression is uniform along length, L. (Finite element models show that it diminishes slightly farther from the wall, because of damper induced slowing of rod velocity. Still, uniform strain at maximal crunch is a useful approximation.) Then, from conservation of energy we must have that the original kinetic energy minus the energy absorbed by the damper equals total energy stored in the springs at maximal compression, or

$$\frac{1}{2}\rho A L v_0^2 - \frac{1}{2} v_0 \frac{DA}{L} L\varepsilon_{max} = \frac{1}{2}\frac{EA}{L}(\varepsilon_{max} L)^2 . \qquad (12)$$

This is a quadratic equation in ε, which we can solve to determine how a small amount of damping, D, influences maximal strain ε_{max}.

In particular,

$$\varepsilon_{max}^2 + \frac{D v_0}{EL}\varepsilon_{max} = \frac{\rho v_0^2}{E} = \frac{v_0^2}{c^2} \qquad (13)$$

For D = 0, we have $\varepsilon_{max} = \frac{v_0}{c}$, as before. For nonzero damping moduli, D, strain at maximal crunch depends on the ratio $\frac{D}{E} \cdot \frac{v_0}{L}$. For brain, experimental data[22] suggest D/E ~ 200/10000 = 0.02. For threshold concussive blows in humans the ratio $v_0/L \sim 4$; that is, the brain is traveling at about 4 brain diameters, about 70 cm, or less each second when it hits the skull. For small D/E the quadratic (13) can be well approximated as

$$\varepsilon_{max} = \sqrt{\frac{\rho v_0^2}{E}} - \frac{1}{2} \cdot \frac{D}{E} \cdot \frac{v_0}{L} \approx \frac{v_0}{c} - 0.04 \qquad (14)$$

Thus strain is slightly less with realistic viscous damping. The purely elastic B-G model of brain, for which $\varepsilon = v_0/c$, gives a modest overestimate of maximal strain.

2.8 Curved, higher-dimensional geometry

Another difference between the simple B-G model and real brains is the presence of curved geometry. In the present impact problem the essence of the difference is that the leading edge of the brain is not flat, but has a rounded nose. It is this nose that strikes the skull first. The contact area of the nose is smaller than the mid-level cross section of the brain. This situation is depicted for a spheroid impacting a hard flat surface in Figure 4(a). In this

case the spheroid can be modeled as cylindrical core, surrounded by a collar, as shown in Figure 4(b). The core cylinder has volume V_{core} , and the peripheral collar, which never makes contact with the impacting surface, but which carries momentum and kinetic energy, has volume, V_{collar}.

Consider the time of maximum compression of the core against the wall. Ignoring the shear energy in the collar, which can be shown to be quite small, we can analyze this system at maximal compression using the relationship

Initial kinetic energy = strain energy per unit volume x core volume,

or

$$\frac{1}{2}\left(V_{core}+V_{collar}\right)\rho v_0^2=\frac{1}{2}E\varepsilon_{max}^2\cdot V_{core}\,. \tag{15}$$

In (15) we assume that at maximal compression there is uniform strain, ε_{max} , throughout the core only, as suggested in Figure 3, that the collar does not cushion the blow, and that collar volume is substantially smaller than core volume. Solving for maximal strain using $E/\rho=c^2$, we have

$$\varepsilon_{max}=\frac{v_0}{c}\sqrt{1+\frac{V_{collar}}{V_{core}}}\approx\frac{v_0}{c}\left(1+\frac{1}{2}\frac{V_{collar}}{V_{core}}\right)\,. \tag{16}$$

The maximal strain for a short duration impact is increased by a modest factor equal to about half the ratio of collar to core volumes.

2.9 A complete, discontinuous HIC and strength-duration curve

Combining the above considerations we notice that the effect of damping causes the strain to be slightly overestimated by the elastic B-G expression $\varepsilon_{max} = v_0/c$, while the effect of curvature causes the strain to be slightly underestimated by $\varepsilon_{max} = v_0/c$. That is, if we add viscous damping there should be a little less maximal compression at the point of contact, but if we add curvature there should be a little more. Hence we have offsetting errors, and after consideration of the complexities, the simple one-dimensional B-G model holds up rather well. To a good approximation the strength duration curve defining a head injury criterion for any selected critical maximal strain ε^* is still given by Equations (11a) and (11b) for a simple elastic brain model. The strength duration curve is hyperbolic up to a duration of $\tilde{\tau}$, after which it is constant.

3. Comparison with biological data

3.0 Critical strain

To compare the proposed hyperbolic head injury criterion with experimental data, it is necessary to specify a critical level of strain, ε, that is presumed to be injurious. For example, suppose that the critical compressive strain $\varepsilon^* \approx 0.3$. This value implies that a 30% intracranial compression, accompanied by 15 percent orthogonal stretch for Poisson's ratio = 0.5, occurs during blows at the concussive threshold. The critical positive strain for stretch injury has been relatively well established by experiments carried out in isolated nerves. For example, functional or morphological impairment occurs in squid axons after 12 percent stretch [25], in guinea pig optic nerves after 15 percent stretch [34], and in sciatic nerves of frogs after 18 percent stretch [26]. Using Poisson's ratio = 0.5 these experimental values of positive stretch would correspond to compressive strains in whole brain of 0.24, 0.30, and 0.36, with a mean value of 0.30. Hence, as a working value, it is indeed reasonable to suppose that $\varepsilon^* \approx 0.3$.

3.1 Comparison with experimental strength-duration curves

Using this value for critical strain one can compare the proposed truncated hyperbolic HIC with clinical and experimental data describing the concussion threshold in humans and animals. Figure 5 shows the summary clinical data, redrawn from Versace[16], as open triangles in acceleration-duration space. Also shown as open circles are data of Yarnell[35] from experimental whiplash injury in a primate model. The curved line represents the proposed truncated hyperbolic HIC as given in Equation (11b). The truncated hyperbolic function fits both short and long duration data. It is also is based upon the underlying physics of the problem, rather than being purely descriptive.

A separate analysis of concussion threshold data in monkeys was done by Ommaya[12], who found that any short duration blow in monkeys producing an angular head velocity of greater than 250 radians/sec produces physiological concussion. Taking the neck radius for a monkey as about 7 cm and applying Equation (3), the linear external head acceleration associated with 250 radians/sec head and neck rotational velocity is

$$\bar{a} \cdot \Delta t = v_{head} = 0.07 \frac{m}{radian} \text{ x } 250 \frac{radian}{sec} = 17.5 \frac{m}{sec}$$

at threshold external head velocity. Since internal acceleration of the brain with respect to the skull is about 5 percent of external head acceleration, given the weight of the brain in cerebrospinal fluid, then

$$v_{brain} \approx 0.05 \cdot 17.5 \frac{m}{sec} = 0.875 \frac{m}{sec}$$

would be the velocity of the brain approaching the skull. According to B-G theory, this internal brain velocity would produce a critical compressive strain of

$$\varepsilon^* = \frac{v_{brain}}{c} = \frac{0.875\,\mathrm{m/sec}}{3.1\,\mathrm{m/sec}} = 0.28,$$

with a corresponding stretch of 0.14 in orthogonal dimensions. This value is very close to the mean value of $\varepsilon^* \approx 0.3$ obtained from animal experiments in isolated nerves.

3.2. Comparison with known injurious and safe accelerations in humans

Estimation of the range of safe head accelerations can provide another point of validation. Adams [27] noted that severe diffuse axonal injury in humans, happening as a result of falls, occurs only after falls from substantially greater than a person's own height—for example, from a ladder, bridge, elevator shaft, or even a mountain! As before, we expect from theory that a given acceleration-time product, creating a given change in whole head velocity Δv, will produce a given maximal strain in the brain. For falls from a particular height, we can estimate Δv as the velocity of a falling body at the Earth's surface from a particular height, h, which is $\Delta v = \sqrt{2gh}$. For a grown man standing approximately 2 meters high this works out to be about 6 m/sec. Then, accounting for the weight of brain floating in CSF, such a fall would produce about 10% compressive strain or 5% elongation strain, and would be relatively safe from the point of view of experimental studies of stretched neurons[26, 34, 36].

In another study of safe accelerations, reviewed by Margulies and Thibault[18], peak rotational acceleration and angular velocity following sub-concussive blows to the heads of volunteer boxers were recorded with specially instrumented helmets. These blows produced changes in rotational velocity of the head of 25 rad/sec. For an effective radius of the neck of about 0.2 meters/radian in humans, the linear Δv is 0.2 x 25 = 5 m/sec, essentially the same value as for safe falls above.

3.3 Comparison with direct observations of brain motion

Although rare and difficult to reproduce today, direct observations of the motion of the brain within the skull during closed head injury in primates have been made after surgical replacement of the calvarium with a transparent plastic material[19, 21]. Gosch and coworkers were able to take high speed photographs through a Lexan calvarium of a strain wave passing through the brain of an anesthetized Rhesus monkey subjected to blunt impact[21]. They found that "The cerebral mass maintains momentum of acceleration in relation to the skull, which results in concomitant compression of the intracranial contents". A maximal compressive strain of 20 percent is directly observable from published photographs of the brain surface[21]. The pattern of compression is similar to that shown in Figure 3 for compressive strain waves. Moreover, from the images taken 1/200 sec apart one can

estimate the speed of travel of the compression wave at approximately 20 mm/5 msec = 4 m/sec. From theory just presented and published values of Young's modulus of brain, one would expect the compression wave velocity to be about $\sqrt{E/\rho} = \sqrt{10000/1000} = 3.1$ m/sec, which is in reasonable agreement with observation, given the large variability in measured values of Young's modulus, E, for brain (Table 2). Thus the simple B-G model and the associated family of truncated hyperbolas (Figure 6) are able to predict and synthesize much diverse and hard-to-obtain experimental data in the field of head injury.

4. Discussion

The whole head acceleration required to produce a given maximal strain within the brain can be can be described by a truncated hyperbola when plotted as a function of duration. There is a separate hyperbola for each level maximal strain or brain deformation. The resulting family of curves (Figure 6) represents a reasonable, approximate solution to an open problem in biomedical engineering: how to predict deformation of the brain caused by a given blow to the head.

This problem has been hard to study in the laboratory because brain deformation happens over a short period of time in a moving frame of reference and is concealed from view. Studies of actual brain motion in instrumented human volunteers are ethically and practically impossible. Pathological studies of threshold or mild concussions are impossible, because the patients recover. Human autopsy data are available only for lethal concussions, which rarely occur under conditions in which the acceleration and duration can be known. (Impulses can be estimated, however, for falls, as discussed in Section 4.) Fortunately, analysis of the physics involved can produce theoretical insights that can be validated with available published data.

The exact shape of the strength-duration curve for threshold head injury matters in the making of policy decisions and in the design of protective gear and equipment. For example, if one assumes the traditional HIC ($a^{2.5}\ \tau = 1000$) as a guide, shortening a long duration impact from 200 msec to 100 msec would make the impact substantially safer. However, the discontinuous SD curve of Figure 5 is absolutely flat for durations greater than about 90 msec. Another example relates to the safety of heading a soccer ball. By changing the inflation pressure of the ball it is possible, for example, to convert a 4 G, 10 msec ball-head impact into a 2 G, 20 msec impact[37]. Under the traditional HIC concept, in which mean acceleration is raised to the 2.5^{th} power and duration is raised only to the first power, halving intensity and simultaneously doubling duration would make an impact only about 35 percent as dangerous as the original one. However, if the strength-duration curve is actually hyperbolic for the short durations of head-ball contact, then there is no advantage in safety with regard to acceleration injury of the brain.

A head injury criterion based upon an underlying theory is more reassuring than one based solely on curve fitting from limited animal experiments. Theory also suggests that there is a family of tolerance criteria (nested hyperbolas in Figure 6), which are related to different levels of maximal strain and different levels of brain damage. This more biophysical

approach allows extrapolation to situations quite different from those in which the original data for a curve fit were collected. Examples include brains with greatly different dimensions and different CSF thicknesses, as well as blows with substantial rotational as well as linear acceleration.

For example, Ommaya and coworkers[12] have suggested that the acceleration required to produce concussion, i.e. the tolerance level, should vary as the physical scale of the animal. They suggest that larger animals are more vulnerable to head injury than are smaller ones. The existence of such a relationship is important in extrapolating results from animal models to humans. In its simplest embodiment B-G theory predicts that the critical strain for a given head acceleration is independent of brain size and is determined by two scale-independent material properties of brain, namely stiffness and density, so that $\varepsilon_{max} = v_0\sqrt{E/\rho}$. However, the scale of the animal, in particular brain length, L, does factor into the HIC in a subtle way. The point of truncation of the hyperbola is different for smaller animals for two reasons. First, the time for the brain to traverse a smaller CSF gap, s, is less for smaller animals. Second, the time required for an outbound strain wave to traverse the brain under persistent acceleration is also less.

Thus the critical duration, $\tilde{\tau}$, (Equation 10) is itself dependent on body size, because it is related to CSF width and to brain width. For example, if CSF width in a mouse is one tenth that in a human, and the brain width is $1/18^{th}$ that of a human, then the SD curve for a mouse is greatly altered, as shown in Figure 7. Appreciation of such subtleties may be important in interpreting results from small animal models of head injury, which are most commonly studied today. Results in small animal models might underestimate the risk of concussion if extrapolated to humans. The present analysis and the associated family of biomechanical head injury criteria provide a means of predicting the maximal local deformation of the brain for any particular blow to the head, specified in terms of its mean acceleration and duration, the stiffness and density of brain tissue, and also indirectly, in terms of the size of the head.

Figures and Legends

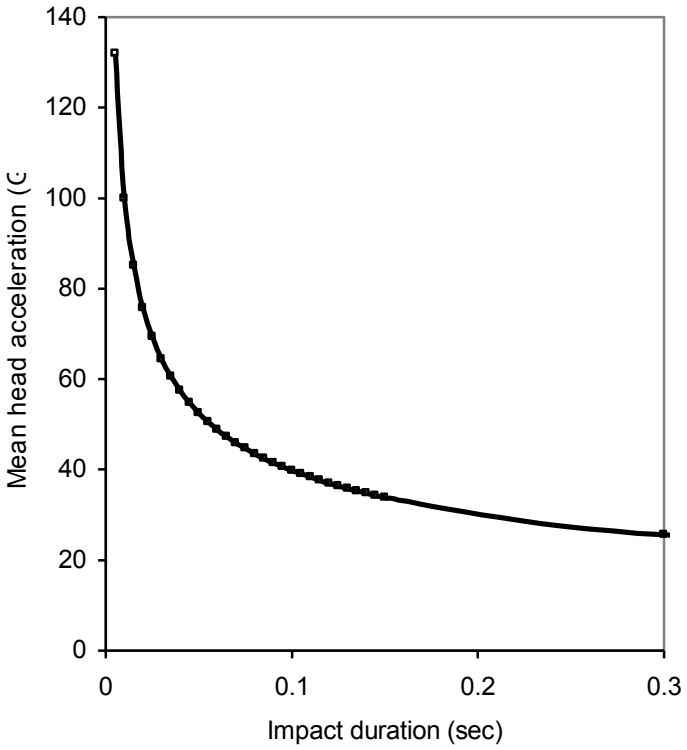

Figure 1: Typical strength-duration curve defining a concussive threshold.

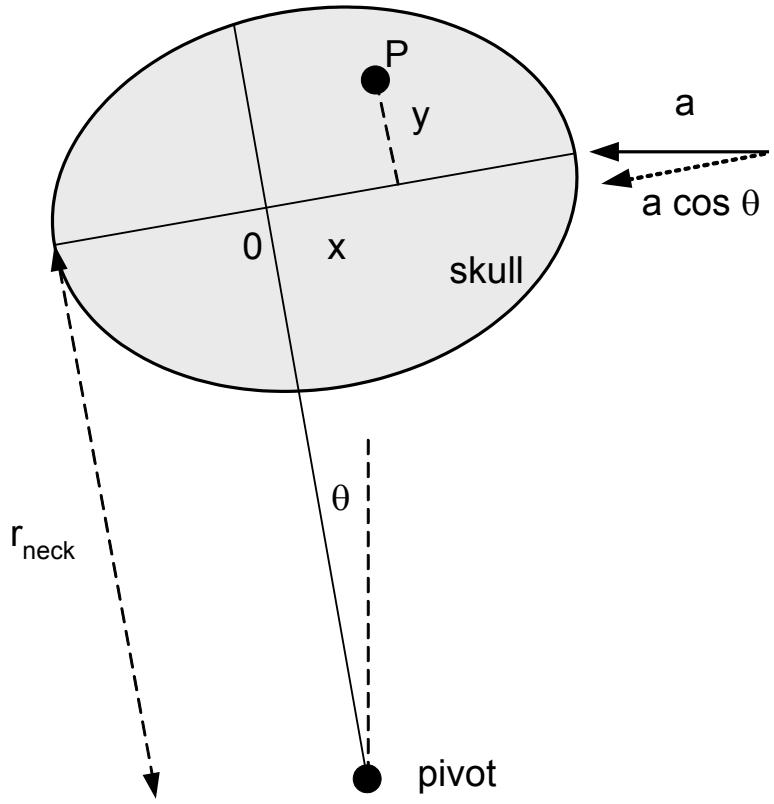

Figure 2. Internal frame of reference of the skull. The forehead is accelerated by a blow from the right with horizontal linear acceleration a. In response the head rotates backward through angle θ, which remains small during the brief time of impact. As long as $\cos\theta \approx 1$ during impact, a mass at point, P, feels acceleration toward the boundary of the skull having component magnitudes

$$\ddot{x} = a\frac{r_{neck} + y}{r_{neck}}\left(1 - \frac{\rho_{CSF}}{\rho_{brain}}\right) \text{ and } \ddot{y} = -a\frac{x}{r_{neck}}\left(1 - \frac{\rho_{CSF}}{\rho_{brain}}\right).$$

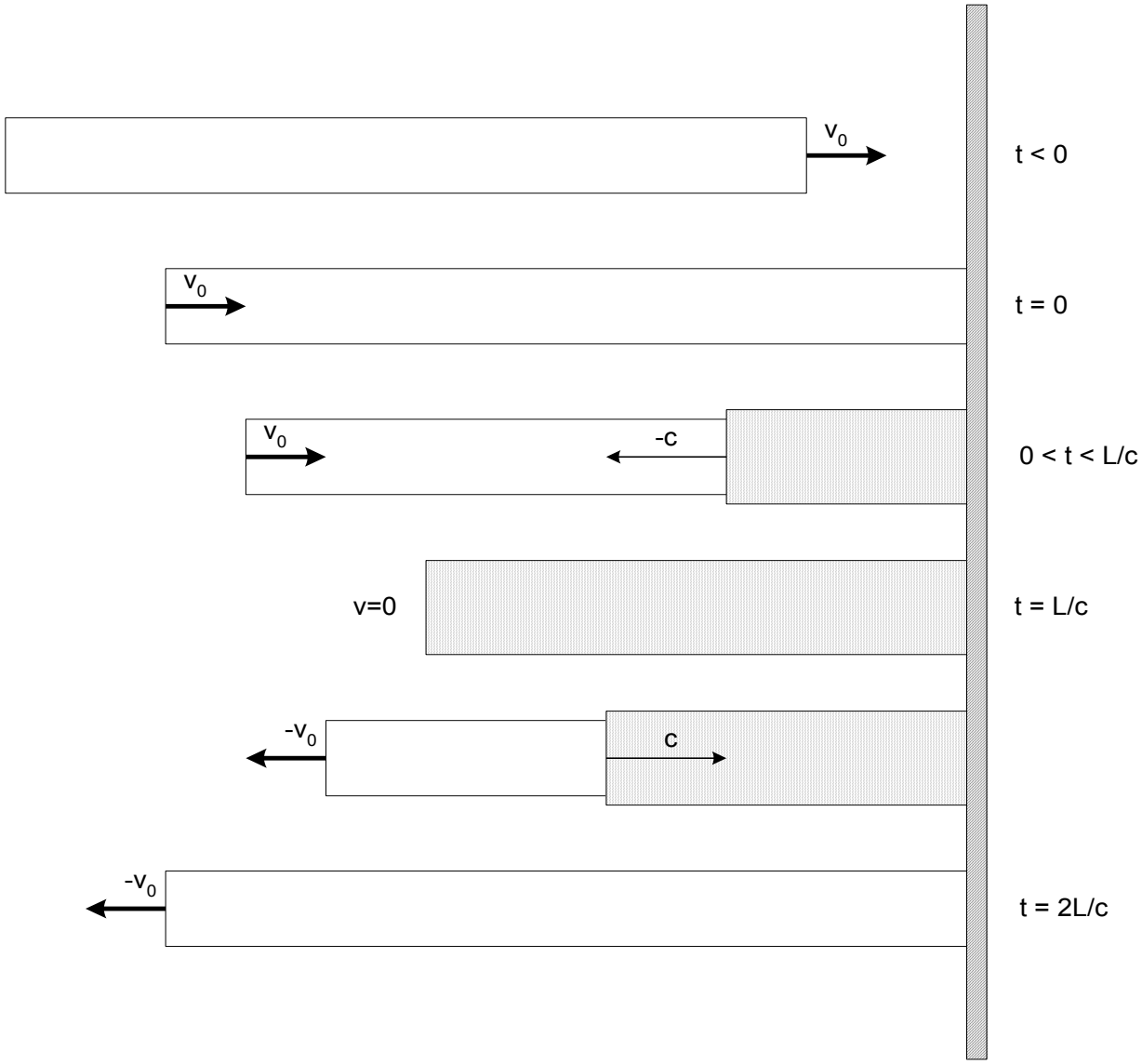

Figure 3. Phases of motion of an elastic bar hitting a solid wall with initial velocity, v_0. Propagation of a compressive strain wave is shown at successive times after impact. The initial length of the bar is L. The strain wave velocity is $c = \sqrt{E/\rho}$. Typically $c >> v_0$.

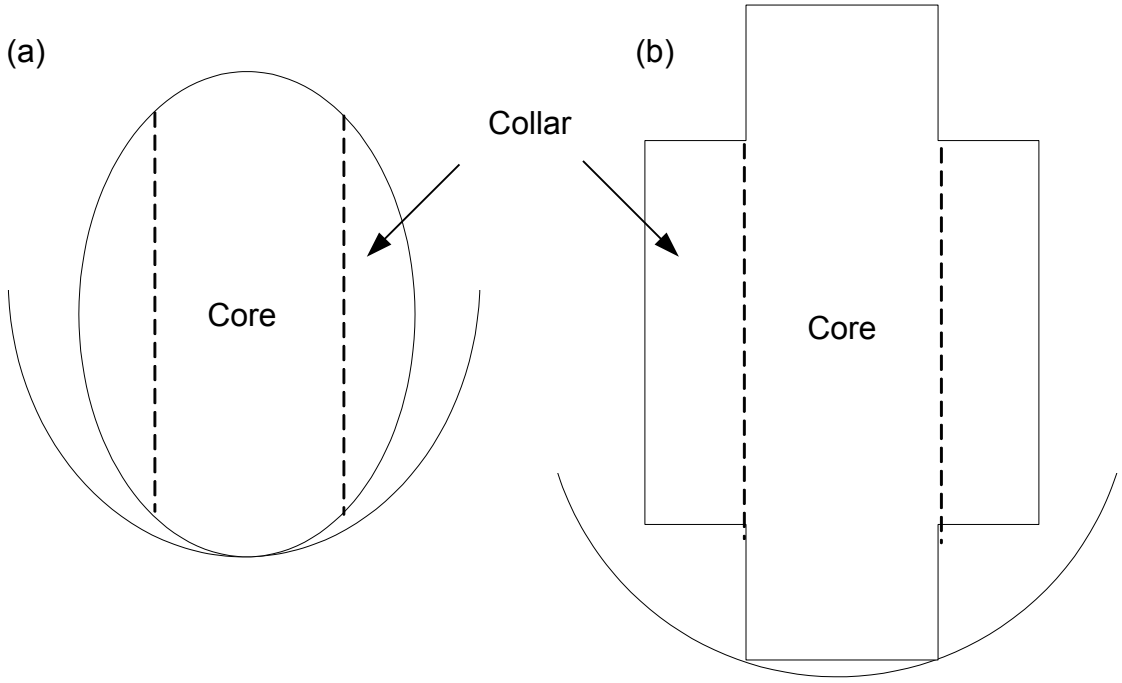

Figure 4: Curved brain impacting a concave surface of somewhat greater radius of curvature. (a) smooth ellipsoidal model (b) cylindrical core and collar model. Acceleration is from the bottom, as in a rising elevator.

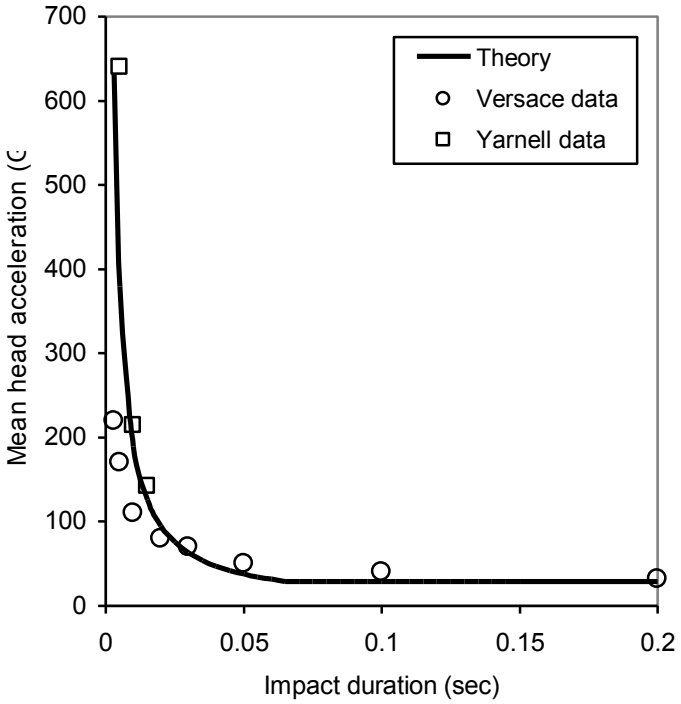

Figure 5: Comparison of present theory with experimental data in humans[16] and in monkeys[35] describing the threshold for concussive injury. The neck length of monkeys (radius for angular acceleration) is taken as 7 cm. Theoretical curve is a plot of Equation (11b) for the concussive threshold in the form of a truncated hyperbola for an adult human model ($\varepsilon^* = 0.30$, s = 1 cm, c = 3.1 m/sec, L = 18 cm).

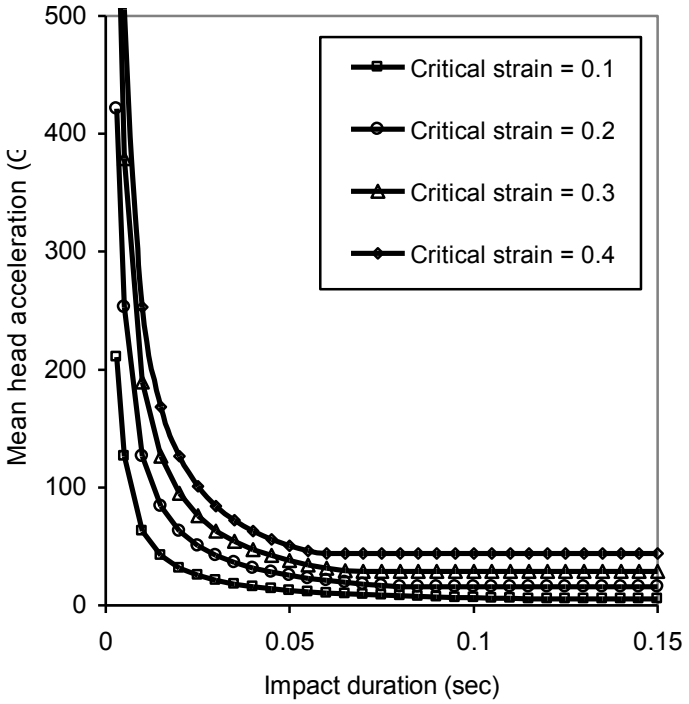

Figure 6: A family of truncated hyperbolic strength-duration curves for four presumed levels of critical injurious compressive strain.

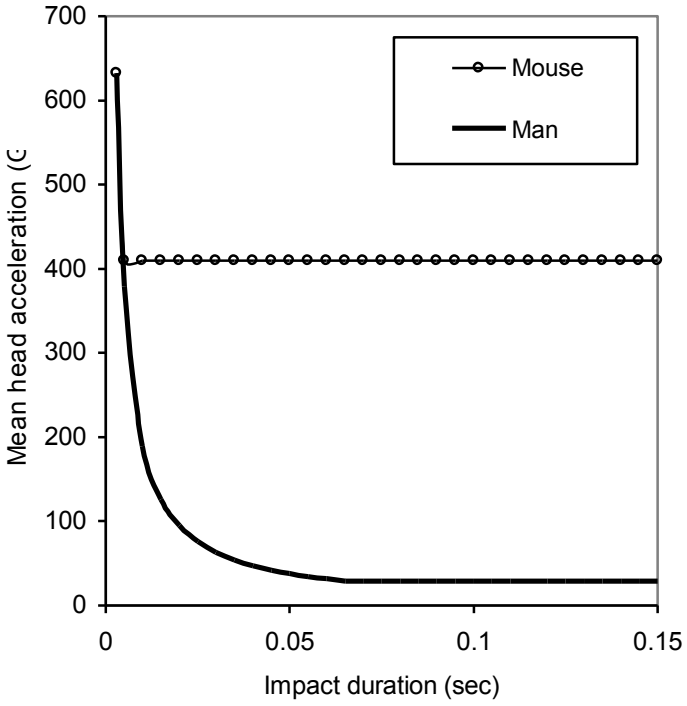

Figure 7: Head injury criteria of mice and men. Differing acceleration tolerances of large and small animals are explained at longer duration pulses by a difference in scale. For mouse brain length L = 1 cm and CSF width s = 0.1 cm; for man L = 18 cm and CSF width = 1 cm.

Tables

Table 1. Nomenclature

A	cross sectional area of brain model
a(t)	brief, forceful acceleration of whole head due to external force
c	strain wave propagation velocity through brain
D	damping or loss modulus of brain tissue
E	Young's modulus of elasticity of brain tissue
ε	local strain in a model of the brain
ε^*	a threshold harmful compressive strain
g	average acceleration of brain toward skull during impact based upon weight of brain in CSF water
k	spring constant of a dx length column of elastic material, namely $k = AE/dx$
L	length of column of elastic material in brain model along the axis of linear acceleration
μ	damping constant of a dx length column of elastic material, namely $\mu = AD/dx$
ν	Poisson's ratio
ρ_{CSF}	mass density of cerebrospinal fluid
ρ	mass density of brain
R, r	neck radius
s	width of fluid filled gap between brain at rest and inner aspect of skull i.e. the distance traveled by the brain through CSF before brain-skull contact
τ	duration of acceleration impulse
$\hat{\tau}$	duration of acceleration impulse ending at the instant the brain strikes the skull
t	time

θ	angle of brain rotation about a pivot point near the base of the neck
$\dot{\theta}$	angular velocity
$\ddot{\theta}$	angular acceleration
v	velocity
v_0	velocity of brain toward skull at instant of brain-skull impact
V	volume
x	longitudinal distance along axis of initial acceleration
y	transverse distance perpendicular to the x-axis

Table 2. Viscoelastic properties of brain: measured values of Young's modulus of elasticity (E) and damping modulus (D) of brain near 1 Hz with 0 to 20% compressive strain.

E (Pa)[#]	D (Pa-sec)[#, *]	Investigator	Year
10,000	6	Ommaya[38]	1968
3,000	22	Fallenstein[39]	1969
15,000	--	Metz[40]	1970
17,000	770	Galford[41]	1970
22,000	200	Shuck[42]	1972
11,000	--	Sahay[43]	1992
8,000	180	Donnelley[44]	1997
5,000	--	Miller[30]	1997
3,000	--	Miller[45]	2000
8,000	400	Babbs[22]	2005
10,200 ± 6,200	260 ± 290	Mean ± SD	

Representative median value for each study. These studies include a wide variability of biological samples and test techniques (relaxation, pure shear, compression, various loading rates, magnitude of applied strains, temperatures, post mortem changes in samples, or the use of pre-conditioning trials to establish repeatable results).

*Converted from shear moduli when necessary using $E \approx 3G$[46, 47]. The damping modulus, D, is defined analogously to Young's modulus, namely the damping coefficient $\mu = DA/L$ for a block of viscoelastic material of cross sectional area A and length, L, parallel to the direction of compression or extension. The damping modulus, D, represents viscous losses in the material.

Table 3. Measured values of brain density (ρ)

ρ (g/cm^3)	Investigator	Year
1.044#	Shigeno[29]	1982
1.044*	Shigeno[29]	1982
1.040	Duck[32]	1990
1.044	DiResta[31]	1991
1.056	Babbs[22]	2005
1.046	Mean	

Gray matter
* White matter

References

1. Gurdjian ES. Head injury from antiquity to the present with special reference to penetrating head wounds. Springfield, IL: Charles C Thomas, 1973:24-25, 54-57, 72-75.
2. Kraus JF. Epidemiology of head injury. In: Cooper PR, ed. Head Injury. Baltimore: Williams and Wilkins, 1993:pp. 1, 4-5,14,15, and 20-25.
3. Adams JH, Graham DI, Scott G, Parker LS, Doyle D. Brain damage in fatal non-missile head injury. J Clin Pathol 1980; 33:1132-45.
4. Graham DI, Adams JH, Gennarelli TA. Pathology of brain damage in head injury. In: Cooper PR, ed. Head Injury. Balitmore: Williams & Wilkins, 1993:91-113.
5. Gennarelli TA. Cerebral Concussion and Diffuse Brain Injuries. In: Cooper PR, ed. Head Injury. Baltimore: Williams & Wilkins, 1993:137-158.
6. Strich S. Shearing of nerve fibers as a cause of brain damage due to head injury: a pathological study of twenty cases. The Lancet 1961; 2:443-448.
7. Dacey RG, Vollmer D, Dikmen S. Mild Head Injury. In: Cooper PR, ed. Head Injury. Baltimore: Williams and Wilkins, 1993:pp. 159-165 and 172-181.
8. Ommaya AK. Experimental head Injury in the monkey. In: Walker AE, ed. Head Injury. Philadelphia: J B lippincott Company, 1966:260-275.
9. Adams JH, Graham DI, Gennarelli TA. Head injury in man and experimental animals: neuropathology. Acta Neurochir Suppl (Wien) 1983; 32:15-30.
10. Barth JT, Macciocchi SN, Giordani B, Rimel R, Jane JA, Boll TJ. Neuropsychological sequelae of minor head injury. Neurosurgery 1983; 13:529-33.
11. Hirsch AE. Current problems in head protection, Head Injury Conference, University of Chicago, 1966. J B Lippincott Company.
12. Ommaya AK, Fisch F, Mahone RM, P C, F L. Comparative tolerances for cerebral concussion by head impact and whiplash injury in primates. Biomechanics 1993:265-274.
13. Lighthall JW, Melvin JW, K U. Toward a biomechanical criterion for functional brain injury. Biomechanics 1993:621-627.
14. Lockett FJ. Biomechanics justification for empirical head tolerance criteria. J Biomech 1985; 18:217-24.
15. Margulies SS, Thibault LE. An Analytical Model of Traumatic Diffuse Brain Injury. Biomechanical of Engineering 1989; 111:241-249.
16. Versace J. A review of the severity index, Proceedings of the 15th Stapp car crash conference, New York, 1971.
17. Kornhauser M, Lawton RW. Impact tolerance of mammals. In: Ely LD, ed. Proceedings of the fourth AFBMD/STL symposium advances in ballistic missile and space technology. Vol. 3. Oxford: Permagon Press, 1961:368-394.
18. Margulies SS, Thibault LE. A proposed tolerance criterion for diffuse axonal injury in man. Biomechanics 1992; 25:917-923.
19. Pudenz RH, Shelden CH. The lucite calvarium-a method for direct observation of the brain. Journal of Neurosurgery 1946; 3:487-505.
20. Gosch HH, Gooding E, Schneider RC. Distortion and displacement of the brain in experimental head injuries. Surg Forum 1969; 20:425-6.

21. Gosch HH, Gooding E, Schneider RC. The lexan calvarium for the study of cerebral responses to acute trauma. J Trauma 1970; 10:370-6.
22. Babbs CF. Brain motion and deformation during closed head injury in the presence of cerebrospinal fluid. Journal of Mechanics in Medicine and Biology 2005; 5:277-306.
23. Paris A. Subtle is the Lord -- The Science and the Life of Albert Einstein. Oxford and New York: Oxford University Press, 1882:552.
24. Shi R, Pryor JD. Pathological changes of isolated spinal cord axons in response to mechanical stretch. Neuroscience 2002; 110:765-77.
25. Galbraith JA, Thibault LE, Matteson DR. Mechanical and electrical responses of the squid giant axon to simple elongation. J Biomech Eng 1993; 115:13-22.
26. Gray JAB, Ritchie JM. Effects of stretch on single myelinated nerve fibers. J Physiol 1954:84-99.
27. Adams JH, Doyle D, Ford I, Gennarelli TA, Graham DI, McLellan DR. Diffuse axonal injury in head injury: definition, diagnosis and grading. Histopathology 1989; 15:49-59.
28. Gennarelli TA, Thibault LE, Adams JH, Graham DI, Thompson CJ, Marcincin RP. Diffuse axonal injury and traumatic coma in the primate. Ann Neurol 1982; 12:564-74.
29. Shigeno T, Brock M, Shigeno S, Fritschka E, Cervos-Navarro J. The determination of brain water content: microgravimetry versus drying-weighing method. J Neurosurg 1982; 57:99-107.
30. Miller K, Chinzei K. Constitutive modelling of brain tissue: experiment and theory. J Biomech 1997; 30:1115-21.
31. DiResta G, Lee J, Arbit E. Measurement of brain tissue specific gravity using pycnometry. Neuroscience 1991; 39:245-251.
32. Duck FA. Physical Properties of Tissue -- A Comprehensive Reference Book. London: Academic Press, 1990:226-245.
33. Brekhovskikh LM, Goncharov V. Mechanics of Continua and Wave Dynamics. Berlin: Springer-Verlag, 1994.
34. Bain AC, Meaney DF. Thresholds for mechanical injury to the in vivo white matter. 43rd Stapp car crash conference proceedings 1999:295-302.
35. Yarnell P, Ommaya AK. Experimental cerebral concussion in the rhesus monkey. Bull N Y Acad Med 1969; 45:39-45.
36. Meaney DF, Smith DH, Shreiber DI, et al. Biomechanical analysis of experimental diffuse axonal injury. J Neurotrauma 1995; 12:689-94.
37. Babbs CF. Biomechanics of heading a soccer ball: implications for player safety. ScientificWorldJournal 2001; 1:281-322.
38. Ommaya AK, Faas F, Yarnell P. Whiplash injury and brain damage: an experimental study. Jama 1968; 204:285-9.
39. Fallenstein GT, Hulce VD, Melvin JW. Dynamic mechanical properties of human brain tissue. Biomechanics 1969; 2.
40. Metz H, McElhaney J, Ommaya AK. A comparison of the elasticity of live, dead, and fixed brain tissue. Biomechanics 1970; 3:453-458.
41. Galford JE, McElhaney JH. A viscoelastic study of scalp, brain, and dura. J Biomech 1970; 3:211-21.

42. Shuck LZ, Advani SH. Rheological Response of Human Brain Tissue in Shear. Journal of basic engineering 1972; 94:905-911.
43. Sahay KB, Mehrotra R, Sachdeva U, K BA. Elastomechanical characterization of brain tissues. Biomechanics 1992; 25:319-326.
44. Donnelly BR, Medige J. Shear properties of human brain tissue. J Biomech Eng 1997; 119:423-32.
45. Miller K, Chinzei K, Orssengo G, Bednarz P. Mechanical properties of brain tissue in-vivo: experiment and computer simulation. J Biomech 2000; 33:1369-76.
46. Muvdi BB, McNabb JW. Engineering mechanics of materials. New York: Springer-Verlag, 1991.
47. Fung YC. Biomechanics : mechanical properties of living tissues. New York: Springer-Verlag, 1981:23-57.