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Biomechanics of Acute Subdural Hematoma

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Acute subdural hematoma (ASDH) due to ruptured bridging veins occurs under acceleration conditions associated with high rates of acceleration onset. That this is due to the strain-rate sensitivity of these veins was confirmed in an experimental model of ASDH. The results of this model were consistent with the clinical causes of ASDH, where 72% are due to high-strain falls and assaults and 24% are due to lower strain-rate vehicular injuries. A mathematical model embodying the known mechanical properties of subdural veins was used to develop tolerance criteria for the occurrence of ASDH. This tolerance curve was consistent with the clinical and experimental data but differed from tolerances previously proposed for head injury.

Acute subdural hematoma (ASDH) is the most important cause of death in severely head injured patients. This is due to three factors: high incidence (30%), high mortality (60%), and head injury severity (two thirds have Glasgow Coma Scores of 3, 4, or 5) (3). The ASDH arises from one of three sources. Direct laceration of cortical arteries and veins occurs with penetrating injuries that also lacerate the brain. Closed injuries resulting in large contusions ('pulped' temporal or frontal lobes) cause similar bleeding into the adjacent subdural space. The most common type of ASDH results from tearing of veins that bridge the subdural space as they travel from the brain's surface to the various dural sinuses. The present study confines itself to this latter type of ASDH; it defines the conditions under which these ASDH occur and the biomechanical mechanisms which cause ASDH.

The present study was designed to investigate whether high strain-rate acceleration of the head is the cause of ASDH. Clinical cases were analyzed, and experimental ASDH was produced in other primates to assess the effects of high strain-rate acceleration. A mathematical model of the brain and subdural veins was constructed.

METHODS

Clinical. Clinical data were obtained from the University of Pennsylvania Head Injury Center Data Bank. This data bank includes over 750 variables for each of 434 head-injured patients who were admitted to hospital for a non-missile head injury and who had a CT scan performed. Only several variables were utilized for this study. The variables selected for analysis include accident circumstance information, injury severity, outcome, and type of lesion detected on CT scan.

All patients in this study had an ASDH as the principal intracranial lesion verified by CT scan, surgery, or postmortem examination.

Experimental. Head injuries were produced in laboratory primates (rhesus monkeys) by a method previously described (1). In brief, the head of an animal was securely fitted into a helmet that was attached to a pneumatic actuator and linkage system. The actuator was programmed to deliver a single acceleration-deceleration pulse to the head by rotating it through a 60° arc in times varying from 5 to 25 milliseconds. The center of rotation was consistently in the lower cervical area.

The acceleration-time history was recorded by an Endevco piezoelectric uniaxial accelerometer mounted on the helmet with its sensitive axis tangential to the movement. This system is designed to minimize focal loading of the head by distributing the acceleration input diffusively over the skull. Therefore, the system provides nonimpact-distributed inertial loading conditions so that the effects of acceleration are studied in isolation.

Post-acceleration neurologic and physiologic observations were made and the animals were sacrificed at varying times for neuropathologic examination by perfusion fixation with formalin-acetic acid-methanol. Animals dying within hours of injury often had inadequate perfusion fixation and in these cases immersion fixation was utilized. Upon opening the dura before dissection, the presence and location of subdural hematomas were noted. Although minute localized films of blood were present in the subdural space of some animals, for this report, ASDH was considered to be present only if sufficient blood was present in the subdural space to cause death. This was judged to be the case if the ASDH

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caused persisting elevation of the intracranial pressure to the level of the mean arterial pressure.

RESULTS AND ANALYSIS

Clinical. Although present to some degree in 15% of all patients in the Head Injury Center Data Bank, ASDH due to bridging via rupture was judged to be the *primary* pathologic process in 38 patients. This was verified by surgery or autopsy in all cases. As expected, this ASDH group had a high mortality (64%) due in large part to the large number of patients in whom the ASDH was responsible for severely impaired neurologic status at the time of hospital admission. Eighty-two per cent of the patients had an initial Glasgow Coma Score of 8 or less and thus fell into the category of severe head injuries.

Of prime interest in this report, the cause of the injury was by fall or assault in 72% of the ASDH group while only 24% of ASDH were due to motor vehicle-related injuries. This was in marked contrast to the patients without mass lesions unconscious for more than 24 hours (diffuse brain injury) where 89% were motor vehicle-related and only 10% were due to falls and assaults (Table I). This difference in the cause of ASDH and diffuse brain injury is highly significant by Chi square analysis ($p < 0.0001$).

Experimental. Over the past several years experimental head injury has been reproduced in laboratory primates by varying the biomechanical input to the head. The acceleration-deceleration time history was always produced so as to deliver an angular acceleration in a situation where head motions are controlled and the effects of impact or contact phenomena are minimized. Since the injury apparatus considers acceleration, time duration of the application of that acceleration, and the rise time to peak acceleration to be independent parameters, these were varied widely over the course of the experiments. Acceleration was varied from 100 to 3,000 g, the impulse duration varied from 2 to 12 milliseconds of a single phase of acceleration-deceleration history, and the rise-time varied from 0.1 to 6 milliseconds. It was found that when using short rise times, as acceleration was increased with pulse duration held constant, that increasing the acceleration magnitude led to increasing injury severity (3).

At first injuries that were subconcussive but produced transient physiologic abnormalities occurred. As acceleration was increased, concussion either of mild or more severe degree could be produced. However, as acceleration was increased further, ASDH occurred. It was then found that, by keeping the magnitude of acceleration constant and increasing the pulse duration and the rise time, that ASDH could be avoided. However, at these longer pulse durations ASDH could still be produced if the acceleration magnitude was increased and the rise time shortened.

Angular acceleration produced ASDH of sufficient size to cause the animal's death in 37 of 128 cases. In all instances of ASDH, the sagittal movement of the head imparted by the acceleration caused demonstrable rupture of parasagittal bridging veins. The ASDH was always overlying the ruptured veins and its size was related to the number of disrupted veins. Although usually bilateral, the ASDH was most often much larger on one side; invariably it was frontally predominant and frequently extended into the interhemispheric fissure. Death was due to massively increased intracranial pressure and resultant marked decrease in cerebral perfusion.

Analysis of the acceleration input that caused ASDH is shown in Figure 1. Angular acceleration and its relation to the duration of acceleration (time) allows a distinction between the three injury types depicted in Figure 1. Below 5 milliseconds and below 1.75×10^5 rad/sec^2 there was an admixture of ASDH with cerebral concussion (loss of consciousness less than 30 minutes). This area on the acceleration-duration graph thus represents a transition region between concussion and ASDH. As accel-

TABLE I
Causes of head injury

	ASDH	Diffuse Brain Injury	All Patients
N	38	72	434
Fall/assault	72%	10%	48%
Vehicular	24%	89%	47%
Other	4%	1%	5%

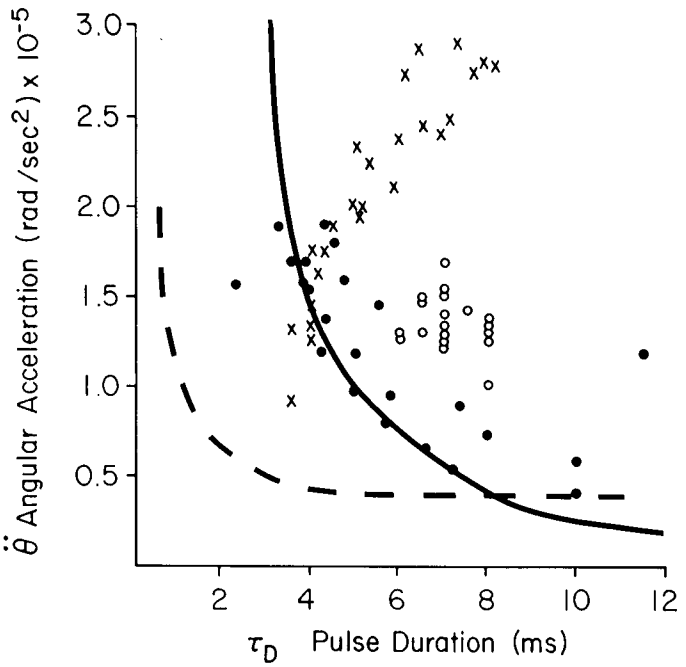

FIG. 1. Angular acceleration and acceleration duration from induced head injury in the laboratory primate are shown. Here and in Figures 3 and 4 closed circles represent cerebral concussion, open circles are diffuse brain injury (unconsciousness for hours to days), and crosses (×) are ASDH. The dashed curve is the tolerance threshold of Bycroft for 'head injury' and the solid curve is the tolerance curve of Hayashi for concussion. Our data for concussion fall near the Hayashi curve but those of ASDH and diffuse injury do not.

eration was held constant but the acceleration duration increased beyond 5 milliseconds, ASDH did not occur. Rather, diffuse brain injury occurred in which there was prolonged traumatic coma (for hours or days) without mass lesions, contusions, or raised intracranial pressure. If, however, at these longer acceleration durations (greater than 5 milliseconds) the magnitude of acceleration was increased beyond 1.75×10^5 rad/sec^2, then ASDH was produced.

In the past both analytical and experimental techniques have been directed toward establishing rational criteria for threshold levels of head injury in terms of measurable mechanical parameters (2, 4–6, 9). Examples of the results of such studies are also shown in Figure 1. The dashed line represents the solution to an analytical model of the head subjected to an angular acceleration as proposed by Bycroft (2) while the solid line is a tolerance level suggested by an analytical model by Hayashi (4) fitted to experimental data from primate studies by Ommaya (9). In both cases the basic injury modeled was cerebral concussion. Since head injury is not a unitary phenomenon, as previously discussed, it is reasonable that specific lesions such as ASDH may be described as a subset of data when viewed in this manner. The models of Bycroft and Hayashi can be characterized as monotonic functions in the acceleration/time domain and are reasonable estimates of the concussion threshold. However, these models do not examine discretely the failure of vascular elements such as the parasagittal bridging veins.

Figure 1 demonstrates that, in order to produce ASDH, the magnitude of acceleration must be increased as the acceleration duration is increased. To explain this relationship it is necessary to understand the mechanical behavior of the tissues involved and their response to dynamic loading conditions.

Analytical. Angular accelerations of the head resulting from impact or impulsive loading conditions produce deformations of the brain relative to the skull. These relative displacements transmit an approximately uniaxial extension to the parasagittal bridging veins, which, in essence, tether the brain surface to the dura and skull. This condition is depicted in Figure 2A. As the skull moves through an angle θ relative to the brain, the vessels are elongated from their resting length l_o to some stretched state l_f as shown in Figure 2B. Under this condition they experience a strain which is defined as

$$\epsilon = \frac{l_f - l_o}{l_o} \tag{1}$$

which is nondimensional and in this case may be related to the cosine θ.

In the model described by Hayashi the brain and skull were considered as elastically coupled, concentric, right circular cylinders. Hayashi solved the equations of motion for this system subjected to a constant torque applied to the outer cylinder for some pulse duration τ_D. The resulting expression for the deformation of a shear

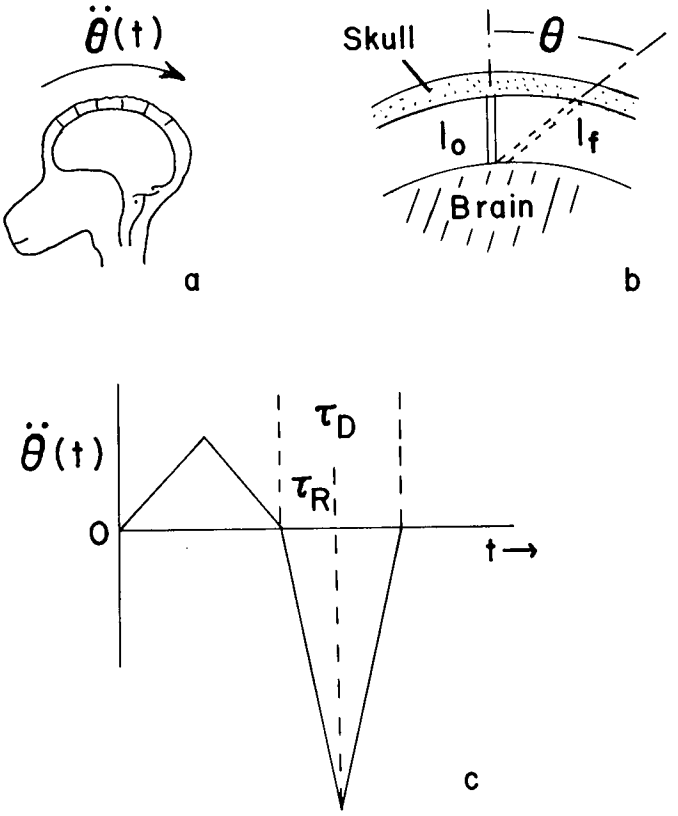

FIG. 2. a) Parasagittal bridging veins, being the sole connection between the skull and the brain, are subjected to tensile strain (stretching) when the skull and brain move differentially when subjected to an angular acceleration $\ddot{\theta}$. b) During acceleration (from left to right in this figure), the skull moves relative to the brain by amount determined by the angular displacement of the head (θ). This causes elongation of the bridging vein as it moves from its original position (parallel solid lines) to its final position (parallel dashed lines). c) An idealized acceleration ($\ddot{\theta}$) time history typical of a fall or auto crash head injury. A lower amplitude acceleration (up going) phase is followed by a higher magnitude deceleration.

member located in an annulus between the two cylinders can be written as

$$\ddot{\theta}\tau_D{}^2 = 2\pi^2 \frac{h}{R_o}\left[\frac{I_s}{I_o}\right]^2 \cdot \gamma^* \tag{2}$$

where γ^* is the critical shear strain necessary to produce cerebral concussion, and h, R_o, I_s, I_o are skull configuration quantities. By fitting this function to the experimental data obtained by Ommaya using monkeys the value of γ^* was found to be 1.13 radians or approximately 65°. As mentioned previously, this angular deformation would also result in a tensile strain if one considered that these cylinders were coupled by vascular elements in the same region. The solid curve in Figure 1 represents the function

$$\ddot{\theta}\tau_D{}^2 = 2.52 \tag{3}$$

which suggests that there exists a single value for the ultimate strain or the strain at which the tissue will fail. This strain in the elastic case is independent of the rate of strain $\dot{\epsilon}$, which is defined as

$$\dot{\epsilon} = \frac{V}{l_o} \tag{4}$$

where V is the velocity and again l_o is the resting length. Figure 2C represents an idealized acceleration waveform where the deceleration phase is larger in amplitude with a pulse duration τ_D and a rise time of τ_R where

$$\tau_R = \frac{\tau_D}{2}. \quad (5)$$

The rise time can be considered inversely proportional to the strain rate.

It has been shown by Löwenhielm (7, 8) that the parasagittal bridging veins exhibit a strongly viscoelastic mechanical behavior. That is to say that the mechanical response is strongly dependent upon the rate at which the vessel is strained and that the ultimate strain to failure decreases with increasing strain rate. Therefore, in order to examine the tolerance threshold for ASDH in terms of the measurable accelerations experienced by the head, it is critical that the shape of the acceleration/time waveform be considered.

Allowing for this viscoelastic behavior Löwenhielm showed that the maximum deformation of the vessels could be written as

$$Z(t)_{max} \sim \frac{a_{max}\tau_D}{\omega} \quad (6)$$

for pulse durations which are short compared to the natural period of the system. Advani (5) estimates this period to be approximately 10 milliseconds for the monkey.

Therefore, since the maximum elongation is proportional to the strain we may write

$$\epsilon_u \sim a_{max}\tau_D. \quad (7)$$

Further, the data obtained by Löwenhielm from autopsy specimens (8) indicate that the strain may be related to the strain rate as

$$\epsilon_u \sim \frac{1}{\dot{\epsilon}^r} \quad (8)$$

where ϵ_u is the ultimate strain and the exponent r is approximately 1.5 for the range of interest. Since $\dot{\epsilon}$ is inversely related to the pulse duration for our idealized waveform we may conclude

$$a_{max} \sim K\tau_D^{0.5} \quad (9)$$

where K is to be determined experimentally.

Figure 3 shows the experimental data plotted as a function of the maximum tangential acceleration and the reciprocal of the acceleration rise time. The function derived from Löwenhielm's results exhibits a negative slope in the region from 0 to approximately 700 sec^{-1}. Löwenhielm's data suggest that beyond 700 sec^{-1} the ultimate strain is constant. The value for K is estimated by considering the static case, i.e., very low strain-rate conditions (10 sec^{-1}) and using Hayashi's model to predict the value for a_{max}.

The results are shown in Figure 4 where the solid line represents an estimate of the tolerance threshold for

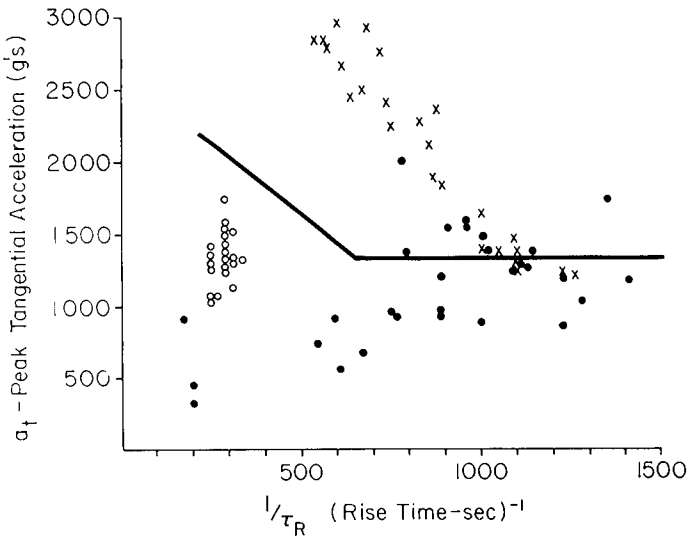

FIG. 3. The reciprocal of the deceleration rise time is plotted versus peak tangential acceleration for the experimental animals. The solid curve is the output of the mathematical model and represents a predicted threshold for ASDH. As predicted most ASDH (×) fall above this threshold.

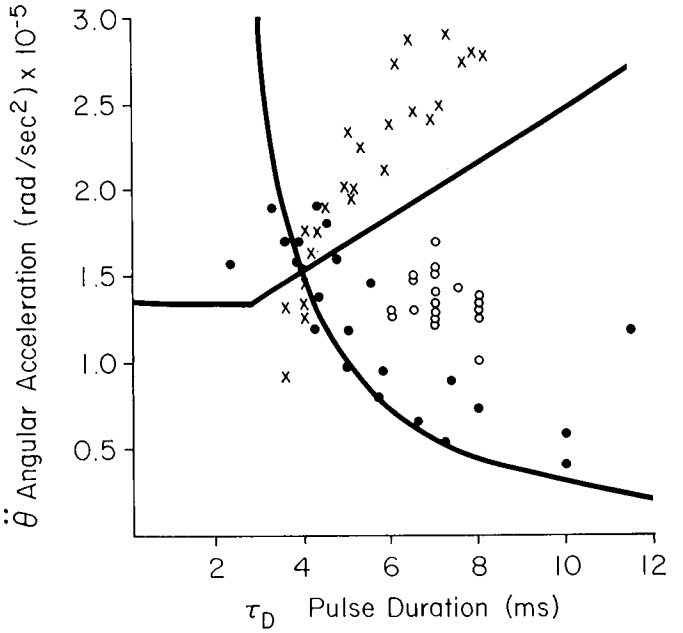

FIG. 4. Pulse duration and angular acceleration are shown as in Figure 1. Here the Hayashi curve for concussion and our math model threshold for ASDH are shown. The ASDH results fall into the area predicted by these thresholds.

ASDH. The approach provides for the critical dependence upon strain rate of the ultimate strain value such that as pulse duration increases, i.e., strain rate decreases, larger values of acceleration are required to cause failure of the bridging veins. In the limit the tolerance levels in both directions will become constant so that the curve will plateau to the right, approaching the static case.

This analysis is not intended to provide a vigorous solution for the field parameters, but rather to incorporate the concept that the viscoelastic behavior of the bridging veins precludes a single ultimate strain or ultimate stress criterion for failure.

Since the ultimate strain to failure of the bridging veins, and perhaps other intracranial tissue components, is inversely related to the strain rate, it follows that a tolerance criterion should reflect an increased threshold for injury as the strain rate is reduced (approaching the static case in the limit).

Detailed analysis of this problem, dynamic testing of fresh vein specimens, and physical model experiments using surrogate tissue material will ultimately establish the precise location and shape of such a tolerance curve for this discrete injury mechanism.

DISCUSSION

This study demonstrates that ASDH due to ruptured bridging veins occurs because of head acceleration that is associated with rapid rates of acceleration onset (i.e., high strain rate). These specific loading conditions help to account for numerous phenomena noted in human ASDH. First, it is apparent that nothing need strike the head in order for ASDH to occur. It is sufficient that the head undergo the appropriate acceleration strain-rate conditions, since in this animal model nothing strikes the head. Thus those mechanical events that result from an object contacting the head (contact phenomena [10]) are not necessary for ASDH. Although impact to the head is certainly the most common cause of clinical ASDH, it is the acceleration induced by the impact and not the head contact per se that causes ASDH. This is so because impact conditions are associated with high strain-rate conditions. All ASDH are not associated with an object striking the head, however, and examples of this point can be provided. Boxers receiving a blow to the chin, face, or ear, can develop ASDH with little or no impact to the skull (12). Similarly, ASDH can be produced in football players whose heads are set in motion by violent impacts to the torso in which no head impact occurs. Football players and motorcycle riders can develop ASDH when impact occurs to their helmets. In these instances, the helmet diffuses the focal loading of the impact much as in our experimental model but does little to diminish the head acceleration resulting from the impact. Probably the most pure form of ASDH without head impact results from violent shaking of infants. Again, nothing strikes the head but the rapid movement of the head is sufficient to exceed the bridging vein tolerance.

The acceleration-rate (strain-rate) sensitivity of the subdural bridging veins explains the predominance of ASDH in falls and assaults. Here, the head impacts or is impacted by a firm structure which itself deforms very little. Thus the acceleration (or deceleration) occurs very quickly and the rate of acceleration is very high, exactly the conditions under which the acceleration-rate sensitive veins rupture. The low occurrence of ASDH in automotive-related injuries is because of the much lower deceleration rate due to energy-absorbing mechanisms in autos. Thus, dashboard padding, deformable steering wheels, energy-absorbing steering assemblies, and laminated windshields slow down or prolong the deceleration of the head and result in lower rates of deceleration. An example of these two conditions is as follows:

A person who falls 25 feet and strikes his head on concrete (stopping distance 0.1 cm) experiences approximately 200 g of deceleration with a pulse duration of 3.5 msec. An equivalent deceleration occurs to a vehicle occupant who strikes the dashboard if the auto crashes into a rigid barrier at 40 mph. Here the dashboard deforms 10 cm, resulting in a pulse duration of 35 msec. In the first case but not the second the acceleration strain-rate conditions are appropriate to rupture the bridging veins and cause ASDH.

The importance of the principal finding of this study, that ASDH is caused by acceleration with high acceleration rate, relates to the feasibility of developing strategies to protect against ASDH. It seems, however, that ASDH will continue to be a frequent type of head injury since it is impossible to provide head protection in falls and assaults, the most common causes of ASDH. Here, since prevention is not feasible, better trauma delivery systems, early diagnosis, and rapid, aggressive treatment must be employed in order to decrease morbidity and mortality from ASDH. A recent study has demonstrated the importance of this approach (10). Such is not the case for automotive injuries and possibly also for injuries occurring to helmeted heads (football and motorcycle). Here further injury protection is still possible. A word of caution is required, however. Since the rate of ASDH already seems low in automotive crashes, it may at first appear that adequate protection is already present. In some respects this is true because the energy-absorbing devices currently in existence do work to decrease the conditions that lead to ASDH. These devices may, however, still be inadequate in that, although protecting against ASDH, the lower acceleration rates and longer pulse durations which result from these devices may place the patient in jeopardy of developing diffuse brain injury. The diffuse brain injury, in its severe form, is just as bad (with regard to mortality and morbidity) as is the ASDH (3). Thus it is possible for use of well-meaning protective devices to allow one bad injury instead of another.

The present report identifies the three factors crucial for the rupture of bridging veins and thus the production of ASDH. The two factors of prime importance are the magnitude of acceleration and the rate of acceleration onset (strain rate) but since, for equivalent acceleration-time waveshapes, the acceleration rate is highly dependent on the acceleration duration, this third factor assumes importance as well. Using these factors, tolerance curves have been established to define the circumstances by which ASDH occurs. The shapes of these tolerance curves are dependent on the mechanical properties of the subdural veins and differ markedly in shape and in

direction from other proposed tolerance curves of head injury. We conclude that tolerance criteria for each of the many types of head injury should be established, rather than continued reliance upon previously held concepts of head injury based on traditional biomechanical theories.

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DISCUSSION

DR. DONALD PAUL BECKER (Medical College of Virginia, Richmond 23298): This is an important study for two main reasons. The first is that up until this time it has been almost impossible to produce acute subdural hematomas in experimental animals. Experimental subdural hematomas have been created by injecting blood into the subdural space, and Doctor Gennarelli's model can now produce a clinically similar subdural hematoma.

Second, it is important because for the first time it helps us to understand why acute subdural hematomas are seen so much more often in patients who have a head injury from a blow or fall, and seen so relatively infrequently in individuals who have severe brain injuries in automobile accidents.

[Slide] This slide, from a paper we published recently in the *New England Journal of Medicine* (11), shows that patients with acute subdural hematomas suffered motor vehicular accidents significantly less frequently than patients who did not have acute subdural hematomas, which is in this category here.

I want to point out from this slide that the patients with acute subdural hematomas were much older. They averaged 41 years in this particular study, and the patients without subdural hematomas averaged 26 years of age. That age difference has been previously reported in the literature. It had been assumed that the people who developed acute subdural hematomas did so because their brains were lax from older age and perhaps alcoholism. I would like to ask Doctor Gennarelli if this still must not be taken into consideration—the fact that older patients do have more lax brains, or is it just because older patients fall more often than younger patients?

Finally, I would like to comment that Doctor Gennarelli's last statement, that perhaps padded dashboards and padded interiors of automobiles are more likely to convert acute subdural hematoma injuries to more diffuse brain injuries because the duration of impact is longer. It is true that the longer the duration of the impact the greater the degree of brain injury. But he has failed to comment on the fact that the padded dashboard is going to reduce the pulse pressure or the degree of force that goes through the brain. Would this reduction in force ultimately be more beneficial and therefore negate the potential danger of increased impact duration seen with a padded dash?

DR. WILLIAM F. BOUZARTH (Medical College of Pennsylvania, Philadelphia 19129): I would like to congratulate Doctor Gennarelli on an excellent presentation. Years ago I created subdural hematomas in dogs by putting pads on their heads and then setting off blasting caps. I did this to find out what our treatment should be in terms of forming subdural hematomas. Ten per cent of the control animals with no treatment had subdural hematomas. If I gave heparin after the injury, all dogs developed hematomas. The various treatments we are still using for subdurals and for brain injury also have a tendency to increase the per cent of subdural hematomas. So, there are a lot of factors that go into creating this potentially lethal injury.

DR. FRANKLIN C. WAGNER (Yale University School of Medicine, New Haven, Connecticut 06510): Doctor Gennarelli, did you have any way of determining what percentage of your animals that were subjected to increased acceleration with mixed injury had evidence of both an an acute subdural hematoma as well as a cerebral concussion?

DR. THOMAS A. GENNARELLI (Closing): To answer Doctor Wagner's question first, virtually all of the animals that had acute subdural hematomas had cerebral concussions, although it is suggested by our tolerance curve for subdural hematomas that that may not necessarily be the case. That is, you might have a situation with very high strain rates, that is, very short duration pulses, where the acceleration magnitude is still below that of concussion but is sufficient to rupture the subdural vein. So it is theoretically possible to have an acute subdural hematoma with no initial concussion. I think that does happen clinically, but probably most common is to have a concussion with a subdural hematoma. Sometimes there is a lucid interval and sometimes not, depending on how quickly the subdural hematoma occurs.

We are working more now in the diffuse injury category, and we are noticing that as we go to the longer pulse durations, if we increase the acceleration magnitude we can get an admixture of animals with diffuse injury and small subdural hematomas. I think we would find the same thing in human cases if neuropathologists would not simply look at the brain and say, "There is a huge subdural here," and not do very detailed microscopic analysis. I think if very detailed microscopic analysis were done one would find a subset of patients who have acute subdurals and who have pathological evidence of diffuse brain injuries. A few of these patients have been reported in papers by Peerless, Nevin, and others, but the focus has not been on the acute subdural in those patients.

Doctor Bouzarth is certainly correct, in that there are many other systemic and perhaps treatment factors that can make one more prone to either develop a subdural hematoma or to increase it once it begins, but we feel the primary injury is biomechanical in nature and does reflect the biomechanics of the veins in the head.

Doctor Becker's comments are most appropriate. We began

an analysis of age in our own patients and certainly found the same thing he presented to you—that is, that older people do get more subdural hematomas. In our own patients it is true that proportionately more older people fall and fewer are involved in car accidents than younger patients. The percentage of people who get subdural hematomas from falls is still much higher in the older patients than in the younger patients, and the figure I remember is that roughly 45% of the patients in the 50- to 60-year age group who fell developed subdural hematomas, where in the 20- to 30-year age group this was about 1%. So falls are more common in older people. However, older people still get more subdural hematomas from falls than do younger people.

The reason for this is severalfold. One, the mechanical properties of the veins probably change with age, just as the mechanical properties of the arteries do as one gets some degree of arteriosclerosis. There is stiffening of the vessels, in the mechanical sense of the term; that is, they become more brittle, if you will, and are more prone to fracture or rupture at lower acceleration levels than less stiff younger veins.

Another factor Doctor Becker mentioned is certainly crucial, that is, as some degree of atrophy occurs with age the subdural veins are being tethered before the injury occurs. That is, there is pre-stretch or pre-strain on the veins, so they start from a different mechanical condition when injury occurs. They already start with some degree of strain on them, so it is going to take less strain to get them to the tolerance level.

Finally, the other effect of age with a slightly smaller brain is that for the same degree of angulation—let's say 60° of head movement—the brain is liable to move farther because it has more room to move in the older patient than in the younger patient where there is less space. So, all of these factors combined will allow us to continue to see more older people with subdural hematomas.

Doctor Becker's concern for protective measures within automobiles is also shared by me. There is no question that except for seatbelts, head injuries can best be minimized by adequate crash protection in the interior of the vehicle. The installation of interior padding, deformable windshields, and energy-absorbing steering assemblies have been efforts in this direction. Our data from this study do suggest, however, that further protection is necessary. Current protection slows down the deceleration of the occupant, thereby decreasing its magnitude and lengthening the deceleration duration. This is exactly what is necessary to decrease the occurrence of subdural hematoma. Our clinical data reflect the success of this approach since subdural hematomas are now relatively infrequent in car crashes. However, current occupant protective measures are not preventing all serious head injuries. That is, they are only decreasing deceleration magnitude and increasing duration to the point below the threshold for subdural hematoma but not below the threshold for severe diffuse brain injury. Therefore, further design modification must be made to decrease these injury factors so that the diffuse brain injury threshold is not exceeded, otherwise we will only have traded one bad injury (subdural hematoma) for another equally bad injury (diffuse brain injury).