PROTOTYPES OF HEAD INJURIES: APPLICATION TO ANIMAL EXPERIMENTS

To provide some order in the discussion of brain injuries the classification of injuries as "open" or "closed" is used. The basic criterion is the involvement of the dura. If it is intact, the injury is a "closed" one even though the skull may be fractured. If the dura is penetrated it is an "open" injury. Only the mechanics of "closed" brain injuries will be discussed here.

Closed brain injuries usually result from a blow delivered by a blunt non-penetrating object. The head may be moving when it strikes a hard unyielding surface, or the head may be stationary when it is struck by a moving surface. In the first case the head is decelerated to a stop and in the second case it is accelerated to some resultant velocity. There is no essential difference so far as the injury potential is concerned. Experiments have shown that if the head is fixed so it can not be accelerated and a blow is delivered to it injury is much less likely to occur than it is for the same blow delivered to a head free to move (LE COUNT and APFELBACH 1920). Thus it is apparent that the acceleration produced by a blow is a prime element in the injury mechanism.

Accelerations of the head may be translational , or angular in nature. Translational acceleration is produced in a body if the resultant of the applied forces passes through the center of gravity of the body. If the applied force system is a couple, or a pure moment angular acceleration alone is produced. In the usual situation the resultant force does not pass through the center of gravity and the force system is not a couple. Consequently both translational and angular accelerations are produced. For the head, attached as it is, to the neck any prolonged translational acceleration would lead eventually also to angular acceleration.

In addition to these accelerations which may be thought of as steady-state whole-body effects the blow also produces waves of compression which propagate through the skull and brain. These waves reflect wherever changes in acoustical impedances are encountered and eventually develop a highly transient but very complicated stress pattern. This process is further complicated by differences in the propagation characteristics of the skull and the brain. If any local stresses in the developed stress pattern exceed the level of tolerance of the tissue at that point presumably lesions will be produced, or some form of tissue damage will occur.

To simplify the consideration of the different prototypes of head injuries in more detail the head can be treated as a fluid filled sphere.

1. Translational acceleration. If a rigid spherical shell filled with an incompressible fluid is subjected to a force which passes through the center of the sphere as shown in Fig. 1 a pressure gradient is developed across the liquid. This gradient develops because the force necessary to accelerate all of the liquid to the right of any given plane such as B-B must be supplied by the pressure in the fluid at that plane. If the sphere is not completely filled with fluid a cavity

develops at the right side of the sphere and no pressure change will occur at that point. Thus the pressure increases linearly from the ambient pressure at that point to a maximum at the left side where the force producing the acceleration is applied. At any given point the pressure is hydrostatic, i.e., the same in all directions.

If the sphere is completely filled with fluid the pressure gradient produced by the acceleration is the same as in the previous case but the pressure at the right side of the sphere drops below ambient and the zero pressure (ambient) in the gradient is found at the center of the sphere. If the drop of the pressure below ambient is enough to make the local pressure less than the vapor pressure of the fluid, cavitation will occur. In this process bubbles form in the fluid and then when the pressure gradient disappears these bubbles collapse. If such a process takes place in the brain, lesions could be produced when the bubbles collapse. Some researchers have hypothesized that rupture of blood vessels may occur if the pressure differential between the inside of these vessels, and the tissue outside is increased significantly either by the pressure gradient across the brain, or by the cavitation process.

If the assumptions of the rigid sphere and the incompressible fluid are relaxed the basic mechanism is unchanged but the levels of pressure for a given acceleration are changed and the point of zero pressure is shifted.

The original idea of the contre-coup brain injury was that the brain was initially moved in the direction of the point of load application, and probably elastically compressed. Then when the acceleration decreased or vanished the brain sprang back toward its original position and in the process bumped into the skull and thus was injured. This idea is no longer considered valid since the stresses that would be developed by this secondary impact would be of the same nature as those produced on the other side of the brain by the primary impact but would be smaller in magnitude. Therefore if any injuries are produced they should be observed at the impact side instead of vice-versa.

In addition to the possibility of injury by the cavitation process there is also a possibility that the development of pressure in the brain may cause or tend to cause extrusion of the brain out through the foramen magnum. If such an extrusion occurred it would no doubt be accompanied by high shear stresses which would produce lesions in the part of the brain affected.

2. Rotational Acceleration. If the spherical shell is subjected to a pure moment an angular acceleration will result. The magnitude of this acceleration will depend on the magnitude of the applied moment and the mass moment of inertia of the rotation system. Angular acceleration of the shell does not necessarily result in an angular acceleration of the fluid within it. The moment necessary to accelerate the fluid must be communicated to the fluid through the shell and the only way this can be done is through friction developed between the fluid and the containing spherical shell. However, if an inviscid fluid is assumed, no friction (or viscosity) will be available to accelerate the fluid. Thus the shell accelerates

in rotation but the fluid remains at rest. Although the skull is not spherical and the brain is not an inviscid fluid the same tendency for the brain to remain stationary while the skull rotates would be present if the skull were given an angular acceleration. Relative motion between the skull and the brain can not occur however, except to a very limited extent, without tearing of connective tissues, and blood vessels, such as the bridging veins. Also this relative motion can cause overstretching and lesions in the brain stem area.

Except for trauma produced in the brain stem, injuries caused by angular acceleration of the head should appear near or at the surface of the brain. As a consequence of these considerations HOLBOURN (1943/1945) has hypothesized that skull rotation rather than skull translation is the basic mechanism by which brain injuries are produced.

3. Translational and rotational accelerations. Since ordinarily a blow to the head will produce both translational and rotational accelerations brain injuries will usually consist of both lesions and hemorrhages within the brain caused by the translational acceleration, and lesions and hemorrhages at or near the surface caused by the rotational accelerations. The latter will usually predominate.

4. Wave Phenomena. If the rise time and the duration of the force applied to the fluid filled sphere are long in comparison to the period of the lowest mode of vibration the sphere will behave essentially as it would have under static application of the same load. Vibrations of the sphere can be neglected under those conditions (RAYLEIGH 1906). In this case the actions described in (1) and (2) would occur. If the duration of the applied force is not long the ensuing wave motion must be considered. In general the duration of forces applied to the head are long and the rise times are long because of the cushioning effect provided by the scalp. The usual duration is of the order of 1 to 2 milliseconds (GOLDSMITH 1972) and this is about one order of magnitude greater than the transit time through the brain and skull. The fundamental period referred to by RAYLEIGH would be twice this transit time.

In addition to the wave propagation effects the contact phenomena (or local deformations) must also be considered.

The mechanisms involved and the pressures developed under certain conditions have been investigated by a number of researchers (GÖTTINGER 1950, ENGIN 1968, BENEDICT 1969, BENEDICT et al. 1970, KOPECKY and RIPPERGER 1969). To illustrate the possible order of magnitude of these effects BENEDICT et al. considered an elastic spherical shell filled with a compressible fluid loaded axis symmetrically. The mathematical expression (GOLDSMITH quoted by BENEDICT) used for the variation of the applied load with time and the distribution of that load is

$$P(\varphi, t) = Be^{-b\left(\frac{t}{T}\right)^2} \sin\left(\beta \frac{t}{T}\right) \cos(\gamma \varphi)$$

where B, β and γ are constants and T is the duration of the loading. This expression indicates a load that varies sinusoidally with time while the peak ampli-

udes of the sinusoid decay exponentially. The force begins at zero and has a rise time to the first peak which is approximately 1/4 of the period of the sinusoid. The angle φ is a polar angle which varies from zero at the point of application of the load to 180 degrees at the contre-coup point. A time of T = 0.001 sec. was chosen for the duration of the loading. The constant B determines the peak value of the force. A value of 2500 lb/in^2 was selected for this constant because this value results in stresses which are in the range of the elastic limit of skull material. Values of the other constants are given by the authors but are not repeated here. Calculations of the pressure levels in the fluid at various times indicate that relatively high negative pressures are developed at both the impact pole and the counterpole, but not at the same time. No indications of what might be considered a focusing effect is seen in these computed results, and the pressure near the center of sphere remains near zero. The authors suggest that the high negative pressures at the two poles result from an outward displacement of the shell which occurs at the counterpole when the stress waves set up in the shell by the impact reach that point. These same stress waves are reflected back to the impact pole and produce there also an outward deflection. As the shell deflects in the outward direction it attempts to pull away from the fluid and thus creates a tension (negative pressure) in the fluid.

These computed results indicate that a negative pressure is developed in the vicinity of the antipole whether the impact is treated as a quasi-static loading, or a transient loading. Thus the basic conditions necessary for cavitation to occur would appear to be present in a head that has been subjected to a blow. It is, of course, not established conclusively that lesions which have been observed in the interiors of injured brains have resulted from cavitation.

An inward bending of the shell occurs initially at the point of load application. Without regard to the waves which may be propagating in the shell it is obvious that this inbending will eventually be recovered if the elastic limit of the shell has not been exceeded. When the shell does recover it "overshoots" like a stretched spring that has suddenly been released, and as a consequence it attempts to pull the fluid with it, thus generating in the fluid a tension (or negative pressure). Hence cavitation related trauma may be observed on both the coup and contre-coup sides of the head.

Since the skull configuration and strength vary from one point to another the contact phenomena and the level of effects produced by a blow both at the point of contact, and within the brain will vary as the point of contact or load application is moved.

The validity of these observations regarding prototypes of brain injury can only be confirmed or denied by experimental studies, in which head injuries are produced by the application of forces which produce either translational or rotational type accelerations. The nature of, and the locations of the resulting injuries must then be determined by histological examination. Some results of this type are presented in the following section.

APPLICATION TO ANIMAL EXPERIMENTS.

Translational acceleration was administered from above (impact direction V according to SPATZ 1950) using the concussion gun described by FOLTZ et al. (1953). A single impact of subcommotio strength, at a speed of 7.1 m/sec, resulting in a peak acceleration of 205 g, imparted to the freely movable head of a cat, caused neither behavioral nor histologic changes in the CNS; whereas repeated impacts of the same intensity, without causing primary traumatic lesions, did produce secondary traumatic alterations due to circulatory disturbances. Lesions in the cerebellum included scattered loss of Purkinje cells (especially at the summits of the lobuli of the vermis), proliferation of Bergmann's glia, thinning of the granular cell layer with glial reaction, and glial proliferation in the striae medullares and white substance. Alterations in the cerebrum were less severe; they consisted of disseminated ischemic nerve cells and a moderate glial proliferation in the white substance.

Impacts of concussive (commotio) strength, i.e., producing the clinical symptoms of cerebral concussion in cats, namely unresponsiveness, have a velocity of 8.3-9.4 m/sec, resulting in peak accelerations of 280-400 g. After one such impact, the histologic alterations prove to be traceless with the methods of investigation used today. We found, in particular, no evidence for glial cell proliferation. However, after repeated impacts of equal intensity and at intervals of one to two days, the cerebral cortex showed, in addition to scattered ischemic nerve cells, extensive focal and pseudolaminary necroses of the parenchyma and loss of nerve cells in various parts of the Ammon's horn formation. Tissue alterations in the cerebellum, although less intense, corresponded in quality to those caused by successive impacts of subcommotio strength.

It follows that blunt impacts of intensities that do not cause noticeable tissue alterations when applied singly may elicit secondary alterations due to circulatory disturbances when applied successively in repeated experiments. A sustained permanent brain injury can therefore result from secondary lesions alone, with no primary traumatic alterations present at all. The time interval between impacts has a distinctive influence on the nature of the morphologic alterations.

Considerable primary traumatic lesions are produced by impacts with a velocity of 10.5 m/sec or more, which produce peak accelerations of 400 g or more. In all instances there were subarachnoid and subdural hemorrhages, so-called cortical contusions at the impact pole and the counterpole, single intracerebral hemorrhages, and traumatic necroses. Speeds of the impacting instruments of 17.2 and 18.3 m/sec are fatal to a cat. Accelerations produced by these impacts were not measured because fractures occurred (UNTERHARNSCHEIDT 1963, 1970, 1972, UNTERHARNSCHEIDT and RIPPERGER 1970, UNTERHARNSCHEIDT and SELLIER 1966). See Table 1.

Rotational Acceleration. In experiments concerning the effects of rotational acceleration on the CNS, controlled non-deforming rotational acceleration was directed through a known path near C7-Th1 of 24 squirrel monkeys (Saimiri

sciureus). The equipment used in these studies was designed by HIGGINS and SCHMALL (1967) and HIGGINS et al. (1967), (Fig. 2). The monkeys were subjected to rotational accelerations ranging from 101000 to 386000 rad/sec^2. The result was a continuum of clinical effects from no observable signs through concussion to death (UNTERHARNSCHEIDT and HIGGINS 1969).

The lowest rotational accelerations employed (101 000 - 150 000 rad/sec^2) caused apparently no primary or secondary alterations in the cerebrum. However, the next higher accelerations, up to 197 000 rad/sec^2, produced in 10 of 13 animals subarachnoid hemorrhages, combined in one instance with primary traumatic hemorrhages in the oculomotor nerve, and tears and avulsions, mainly of veins and capillaries, in superficial cortical layers in 8 animals. Accelerations of more than 200 000 rad/sec^2 caused severe primary traumatic hemorrhages in the cortex and white substance. Rotational acceleration of more than 300 000 rad/sec^2 were not survived. The monkeys subjected to these extremely high accelerations were the only animals to show additional hemorrhages in more central regions of the brain, i.e., very close to the central pivot.

Nearly all the animals tested showed small rhectic hemorrhages in various segments of the spinal cord. Capillary and venous hemorrhages were more frequently found disseminated in the gray substance and were caused by longitudinal and transverse stretching of ascending and descending vessel branches. They were seen in all segments of the cord. These lesions were not fatal and produced no clinical signs in the animals. In two instances a subdural hemorrhage was found in the cauda equina.

It must be pointed out that the primary traumatic lesions found in the cortex are venorhectic, and occasionally arterio- or capillary rhectic hemorrhages of the more superficial cortcial layers, as evidenced by torn vessel walls. Also, these hemorrhages are always associated with vessel systems running at right angles to the cortical surface. See Table 2.

In Summary, not only does a qualitative difference exist between the primary traumatic cortical hemorrhages produced by rotational acceleration and the so-called cortical contusions found in translational injuries, but there are also different patterns of distribution for the primary traumatic lesions encountered in both types of acceleration, inasmuch as these lesions are arranged in a cylindrically symmetric pattern after translational acceleration, as compared to a radially symmetric pattern located close to the midline after rotational acceleration.

Except for the question of location, these considerations seem to be valid also for the interpretation of findings in the spinal cord, although correlations are not as patently manifest here as they are in the brain. Nevertheless, the relation between severity of primary traumatic lesions and magnitude of acceleration is evident throughout the entire CNS.

CONCLUSIONS: If the injury producing force of a blow to the head can be

classified as one of the protoype injury mechanisms described here, the quality and location of the resulting tissue alterations can be predicted with a reasonable certainty. Tolerance levels for linear and rotational accelerations for rabbits, cats and squirrel monkeys have been established.

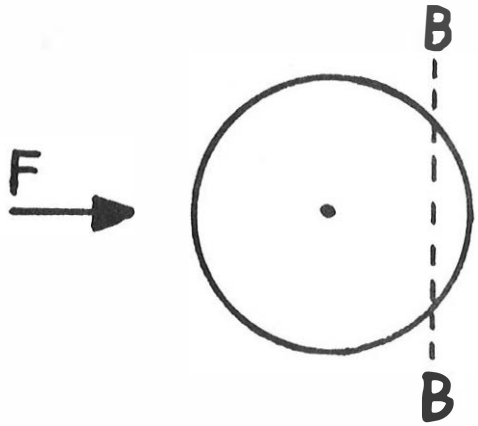
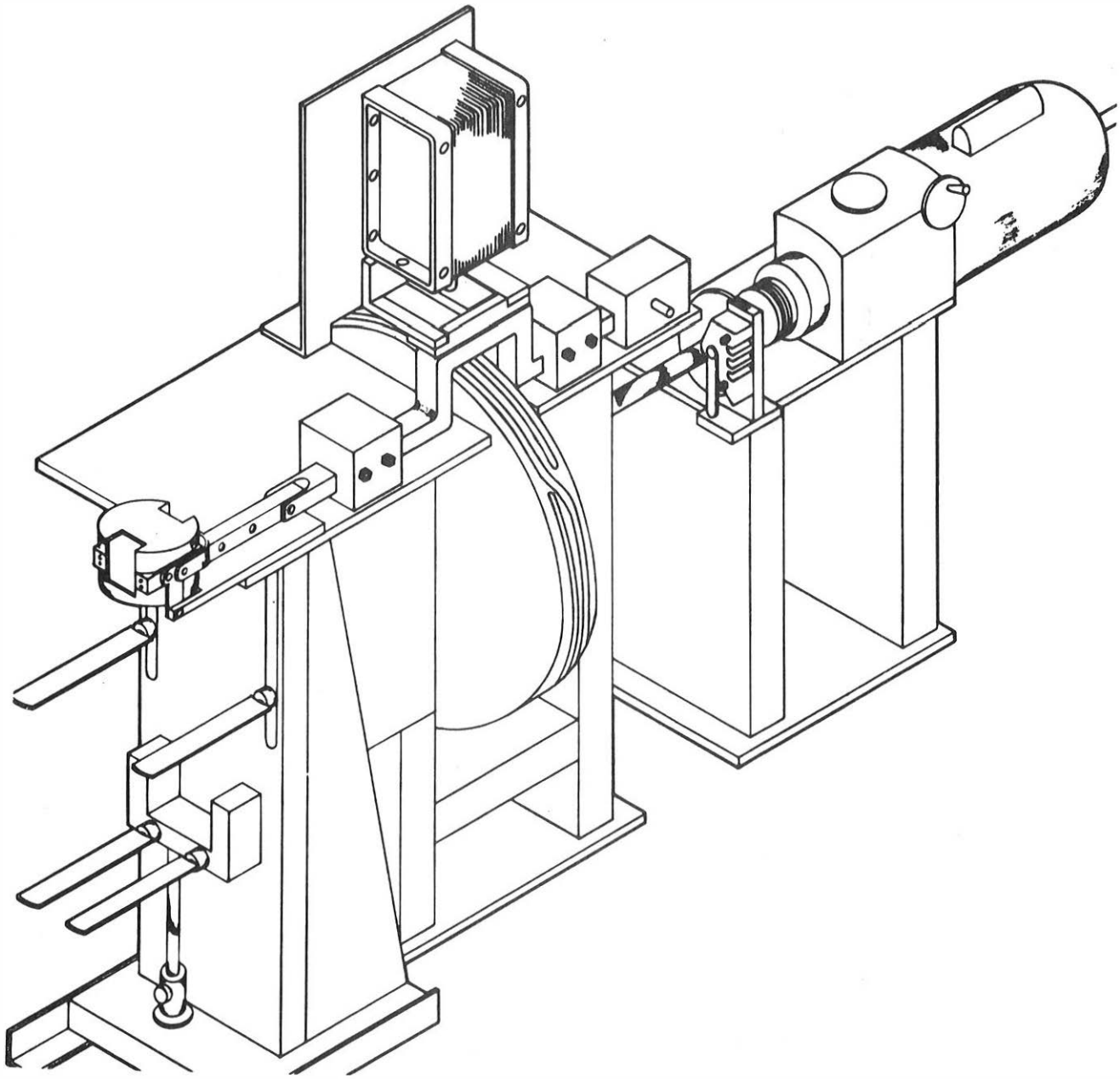


Fig. 1

Fig. 2 Perspective view of HAD II



Animal experiments—translational (linear) acceleration

Diagnosis	Number of impacts	Intensity of impact: in m/sec	Intensity of impact: in km/h	Acceleration	Behavior	Unconsciousness	Primary traumatic alterations	Secondary traumatic alterations
	Subconcussive strength. Subcommotio cerebri.							
	Single blow	7,1	25,0	Cat ≈ 205 g Rabbit ≈ 190 g	Unremarkable	None	None	None
	5 successive blows	7,1	25,0	Cat ≈ 205 g Rabbit ≈ 190 g	Reversible parapareses of front legs	None	None	None
Subcommotio cerebri	10 successive blows	7,1	25,0	Cat ≈ 205 g Rabbit ≈ 190 g	Largely reversible parapareses of front legs	None	None	Secondary traumatic lesions in cerebellum and cerebrum.
Subconcussive strength	10 successive blows	7,5	27,0	Cat > 205 g Rabbit ≈ 260 g	Irreversible parapareses of front legs	None	None	Secondary traumatic lesions in cerebrum and cerebellum.
	15 successive blows		30,0	Cat > 205 g Rabbit ≈ 280 g	Tetrapareses, irreversible in front legs, reversible in hind legs except for weakness	Occurs in part of experiments after summation. "Summation type."	None	Secondary traumatic lesions. Cerebellum, severe Cerebrum, moderate.
Commotio cerebri	Single blow	8,3–9,4	30,0–34,0	Cat ≈ 315 g Rabbit ≈ 400 g	Unremarkable after regaining consciousness	General unconsciousness, lasting from several seconds to minutes.	None	None
Cerebral concussion Concussive strength	Single blow repeated at daily and weekly intervals, resp.	8,3–9,4	30,0–34,0	Cat ≈ 315 g Rabbit ≈ 400 g	Unremarkable after regaining consciousness	General unconsciousness. Its duration is reduced as the number of blows is increased. "Adaptation type."	None	Cerebrum: Disseminated ischemic alterations. Electivo necroses of parenchyma. Loss of neurons and glia reaction in Ammonshorn. Cerebellum: Loss of Purkinje cells and granular cells.
Primary traumatic tissue alterations (Contusio cerebri)	Single blow	10,5	37,0	Cat ≈ 360 g Rabbit ≈ 450 g	.....	Rather long unconsciousness.	Epidural, subdural, subarachnoidal hemorrhages. So-called cortical contusion et impact and antipole. Rupture of extra and intracerebral vessels. Central hemorrhages.	Partial and total necroses, hemorrhagic necroses, edematous lesions.
	Single blow	13,6	49,0	Cat ≈ 525 g	.....	Long unconsciousness.	Most severe primary traumatic alterations of same quality as above.	Most severe secondary traumatic alterations of same quality as above.
	Single blow	16,1	58,0	Cat	.....	Severe primary traumatic alterations and lacerations which the cats survive in rare instances.		
Lethal intensities	Single blow	17,2–18,3	62,0–66,0	Cat	.....	Most severe lesions and disruptions, involving bones and dura (open injuries). Tissue destructions and lacerations which are not survived by the cats.		

(Unterharnscheidt, 1965)

Table 1: Relations between impacting force and animal behavior, and resulting tissue alterations (input-output model). A continuum of findings is noted, ranging from normal after single subcommotional impact to secondary traumatic tissue lesions when the impact is repeated, to a concussion syndrome that produces no tissue lesions after a single impact, but causes secondary traumatic brain damage with repeated doses, to primary traumatic lesions and, finally, fatal outcome with the application of the highest intensities. The two uppermost columns that are framed in heavy lines show the intensities capable of producing permanent brain damage only after repetitive application of force (secondary traumatic lesions). The third column shows the intensities producing primary traumatic tissue lesions. The last column notes the lethal impact intensities.

Rotational acceleration (squirrel monkeys; samiri sciureus)

Rotational acceleration in rad/sec²	Morphological findings
1.01–1.50 × 10^5 rad/sec²	No subdural, no subarachnoid hemorrhages. No lesions in cerebrum, cerebellum, midbrain, and pons.
1.53–2.81 × 10^5 rad/sec²	Subdural and subarachnoid hemorrhages. Rhectic hemorrhages in cranial nerves. Primary traumatic hemorrhages in superficial layers of the cerebral cortex, near the midline. Single rhectic hemorrhages in the medulla oblongata.
3.27–3.86 × 10^5 rad/sec²	Massive subdural and subarachnoid hemorrhages. Severe primary traumatic hemorrhages in the cerebral cortex, extending into the central white matter. Small rhectic hemorrhages in the hippocampus formation. Rhectic hemorrhages in cerebellum, midbrain, pons, and medulla.

With the exception of a few animals, the spinal cord showed small rhectic hemorrhages in various segments. The severity of these lesions, too, was related to the magnitude of angular acceleration and increased with intensity. The correspondence was not as obvious as it was with the cerebrum, cerebellum, midbrain, pons, and medulla. These spinal-cord findings were not fatal, and the test animals showed, indeed, no clinical signs.

(Unterharnscheidt and Higgins, 1969)

Table 2

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E. A. RIPPERGER, PH. D.
Aerospace Engineering and Engineering Mechanics
The University of Texas at Austin, Austin, Texas 78712

and

Friedrich J. UNTERHARNSCHEIDT, M. D.
Naval Aerospace Medical Research Laboratory
New Orleans, Louisiana 70189