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TOLERANCES FOR CEREBRAL CONCUSSION FROM HEAD IMPACT AND WHIPLASH IN PRIMATES*

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Abstract – Experimental head impact and whiplash injury experiments have been conducted in 3 sub-human primate species in order to define tolerance thresholds for onset of cerebral concussion. Preliminary analysis of our data support a hypothesis that approximately half of the potential for brain injury during impact to the unprotected movable head is related to head rotation, the remaining brain injury potential of the blow is related to the contact phenomena of the impact.

Data derived in these experiments is compared with values predicted from some scaling considerations previously developed. Predictions are made that the levels of head rotation during whiplash, in excess of 1800 rad./sec^2, will probably result in cerebral concussion in man.

INTRODUCTION

IN EARLIER reports from this laboratory, we had observed that angulation of the head on the neck was a necessary common denominator for brain injury in the absence of skull fracture during direct and indirect head impact (Ommaya *et al.*, 1966). Cerebral concussion as well as macroscopic intracranial hemorrhage could be produced by impact to the freely movable head as well as by whiplash injury (Ommaya *et al.*, 1968). These experimental studies of the role of rotational displacement of the head in injuring the brain led us to a direct experimental testing of Holbourn's hypothesis (Holbourn, 1943) for the mechanism of head injuries. According to this theory, head rotation caused by impact resulted in rotary distortion of the brain with high resultant shear stresses. Cerebral concussion, contre-coup lesions and other effects of head impact were attributed primarily to such rotational effects while the distortion of the skull by the blow (contact phenomena of impact) was thought to produce only local contusions and skull fracture. The translational component of the impact was considered to be non-injurious. Holbourn implied that the response of the brain within the skull could be represented by a single degree of freedom spring-mass system. Thus short-duration impacts would produce injury proportional to the rotational velocity induced by the blow while long duration impacts would display a dependence on the head's rotational acceleration for injury (Holbourn, 1943). We were able to confirm the rotational velocity dependence of short-duration pulses producing cerebral concussion in both direct impact head injury and whiplash trauma in Rhesus monkeys. The main facet of Holbourn's hypothesis was however *not* proven: thus if head rotation were indeed the crucial brain injury mechanism, cerebral concussion should be produced at an identical threshold for rotational velocity of the head irrespective of *how* the head rotation was induced i.e. directly or indirectly, and the local effects of impact should have no influence on the threshold for cerebral concussion. Our experimental data did not support this

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prediction and indeed showed that about twice the rotational velocity was required to produce cerebral concussion when the animal experienced indirect impact (whiplash) (Hirsch *et al.*, 1968; Ommaya and Corrao, 1970). This suggested a significant contribution to brain injury by the local effects of impact (i.e. contact phenomena).

Our current hypothesis for brain injury by mechanical trauma to the head is therefore a considerable modification of the rotational theory and is best stated as follows.

Approximately 50 per cent of the potential for brain injury during impact to the unprotected *movable* head is directly proportional to the amount of head rotation, the remaining potential for brain injury will be directly proportional to the contact phenomena of impact (e.g. skull distortion). Thus in considering *protection* of the brain from mechanical trauma to the head two factors have to be controlled; first, the amplitude of head rotational motion must be reduced, and second, skull distortional effects mitigated. Hitherto, the latter aspect has been considered of prime significance in head protection and insufficient attention has been given to the role of head rotation. In an earlier report, we had also presented our theoretical derivation of a scaling method aimed at extrapolating experimental data on brain injury thresholds from sub-human primates to man in terms of an inverse proportion between the $\frac{2}{3}$ power of relative brain weights and levels of rotational acceleration (Ommaya *et al.*, 1967). This method made it possible to compare the injurious effects of head rotations in different primate species but did not include scaling for the contact phenomena of impact.

The present report summarizes all our data on direct and indirect impact induced cerebral concussion in three sub-human primate species and compares the fit of data to theory for our scaling predictions.

METHODS

Cerebral concussion was produced in two ways; by direct impact to the occipital zone of the head and by experimental whiplash injury caused by impact to the base of a mobile chair carrying the seated animal. Both of these techniques were identical to that described in our earlier publications (Ommaya *et al.*, 1966; Ommaya *et al.*, 1967; Ommaya *et al.*, 1968; Ommaya and Corrao, 1970; Holbourn, 1943; Hirsch *et al.*, 1968; and Ommaya *et al.*, 1966). Experiments were performed in three sub-human primate species, the rhesus monkey (brain weight = 70–100 g), the squirrel monkey (20–27 g), and the chimpanzee (350–500 g). The number of each of three sub-human primate species used in these tests are indicated as follows:

Species	Head Impacts	Whiplash Injury
Squirrel monkey	35	35
Rhesus monkey	100	100
Chimpanzee	12	26

A compressed air actuated piston weighing 3·8 lb was used to impact the head of the rhesus and squirrel monkeys. The force of the impact was controlled by regulating the air pressure behind the piston. Impact force was sensed by a strain gage dynamometer attached to the striking end of the piston. The velocity of the piston upon impact was measured by recording the voltage generated as a magnet imbedded in the piston passed through a stationary coil. This technique was found to be inadequate to produce significant brain injury in the chimpanzee. A modified humane stunner described previously as well as a specially adapted Hy-G device were utilized for the chimpanzee impacts (Faas and Ommaya 1968). Details of this methodology will be published separately. Animals were usually anesthetized with Nembutal or Sernylan (phencyclidine hydrochloride) prior to preparation for testing. After restraint in the seat had been secured the level of anesthesia was allowed to diminish until the animal exhibited stable vital signs, consist-

ently present corneal and palpebral reflexes, regular respiration and responded to external stimuli with coordinated voluntary movements. The criteria for cerebral concussion onset were similar to those described earlier (Ommaya *et al.*, 1966(b)) and are summarized as follows:

1. Loss of coordinated responses to external stimuli.
2. Apnea > 3 sec, followed by irregular slow respiration.
3. Bradycardia (rate decreased by 20–30 beats/min).
4. Loss of corneal and palpebral reflexes.
5. Loss of voluntary movements.
6. Pupillary dilatation > 15 sec.

Impacts were given to the occipital zone in all species, attempting to define appropriate 50 per cent concussive levels in all direct impact tests. Whiplash injury tests were also performed so as to provide inputs at the approximate 50 per cent concussive level. Displacement of the head on the neck during direct as well as indirect impact was recorded against a background grid by high speed cinematography at 3000–5000 frames/sec. Values for rotational velocity and acceleration of the *head* were calculated from these displacement data.

RESULTS

Data from tests involving 83 Rhesus monkeys, 42 squirrel monkeys, and 11 chimpanzees have been considered to date in this study. The results of the Rhesus experiments have been extensively reported (Ommaya *et al.*, 1966a; Ommaya *et al.*, 1966b; Ommaya et al., 1967; Ommaya *et al.*, 1968; Ommaya *et al.*, 1969a; Ommaya and Corrao, 1970; and Hirsch *et al.*, 1968) and will be referred to here only to make data comparisons and indicate analytical procedures which we consider of importance.

Figures 1 and 2 are plots which indicate the maximum values of head response of 83 Rhesus monkeys subjected to whiplash and head impact. The use of the coordinates, rotational velocity and rotational acceleration is fully explained in Hirsch *et al.* (1969). In essence the assumptions in this treatment lead to the relationship (Kornhauser, 1954):

$$\theta_D = \frac{\dot{\theta}_D}{\omega} \qquad (1)$$

where $\dot{\theta}_D$ is damaging rotational velocity (rad./sec)
$\ddot{\theta}_D$ is damaging rotational acceleration (rad./sec^2) and
ω is natural frequency of brain rotation (rad./sec).

From this equation, it appears that depending on the time period of the initial maximum force input to the head an acceleration dependence or a velocity dependence of cerebral concussion can be demonstrated.

Data from 19 squirrel monkeys impacted and 23 subjected to whiplash are plotted in a similar fashion in Figs. 3 and 4. There is considerable scatter in the data although the whiplash plot indicates that the tolerance value may lie at about 300 rad./sec.

Whiplash tests were conducted on 11 chimpanzees (1), 2 eyeballs out and 9 eyeballs in (1). Data from these tests are plotted in Fig. 5. Here there is somewhat less scatter and a tolerance line could reasonably be drawn at about 70 rad./sec.

The acceleration tolerance, of course, is speculative since no duration exceeded 20 msec. If the assumptions are correct, however, and we could ascertain the natural frequency in rotation of the brain within the skull for each animal an estimate could be made of the $\ddot{\theta}_D$ by using equation (1). For example, the Rhesus monkey brain was determined (Hirsch *et al.*, 1968) to have a frequency of 5–10 Hz. The rotational velocity associated with whiplash concussion is about 300–350 rad./sec. Substitution of these values in equation (1) yields a damaging range of rotational acceleration ($\ddot{\theta}_D$) of 10,000 to 20,000 rad./sec^2. A similar treatment can be given the whiplash

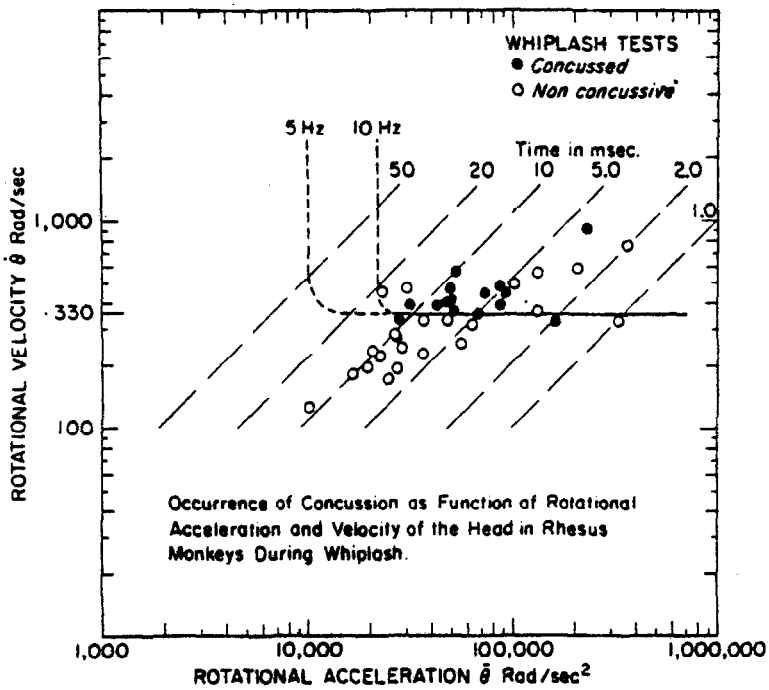

Fig. 1. Tolerance curve for Rhesus monkey depicting thresholds for 50 per cent probability of onset of cerebral concussion in terms of rotational velocity (for short duration blows) and rotational acceleration (for long duration blows) after whiplash injury (indirect impact).

data from chimpanzee and squirrel monkey experiments. Here, however, we do not have direct evidence of the brain frequency. One isolated cinefluoroscopic observation made in a patient with radiopaque silver clips attached to the brain surface after removal of a subdural hematoma indicates that man's brain has a rotational frequency of about 4–5 Hz. This is similar to the Rhesus monkey frequency of 5–10 Hz and hence it is not unreasonable to assume that the chimpanzee and squirrel monkey will not be significantly different. Using a value of 10 Hz for the squirrel monkey and 5 Hz for the chimpanzee, substitution in equation (1) gives values of 19,000 and 2200 rad./sec^2 respectively.

It is well at this point to consider the purpose in these studies—our goal is to determine the tolerance of man to whiplash and head impact. We have attempted some scaling work in a previous study (Ommaya *et al.*, 1967). Figure 6 is a plot of the scale relationship between sub-human primates and man for concussive levels of rotational acceleration. This curve is derived from Holbourn's inverse $\frac{2}{3}$ power law which states that (Ommaya *et al.*, 1967):

$$\ddot{\theta}_m = \ddot{\theta}_p \left(\frac{M_p}{M_m}\right)^{2/3}$$

where $\ddot{\theta}_p$ is the tolerance value in rad./sec^2 of the prototype animal, in this Rhesus monkey and

$\ddot{\theta}_m$ is the tolerance value in rad./sec^2 of man or any other animal whose brain mass M_m is known.

We have plotted on this curve, the experi-

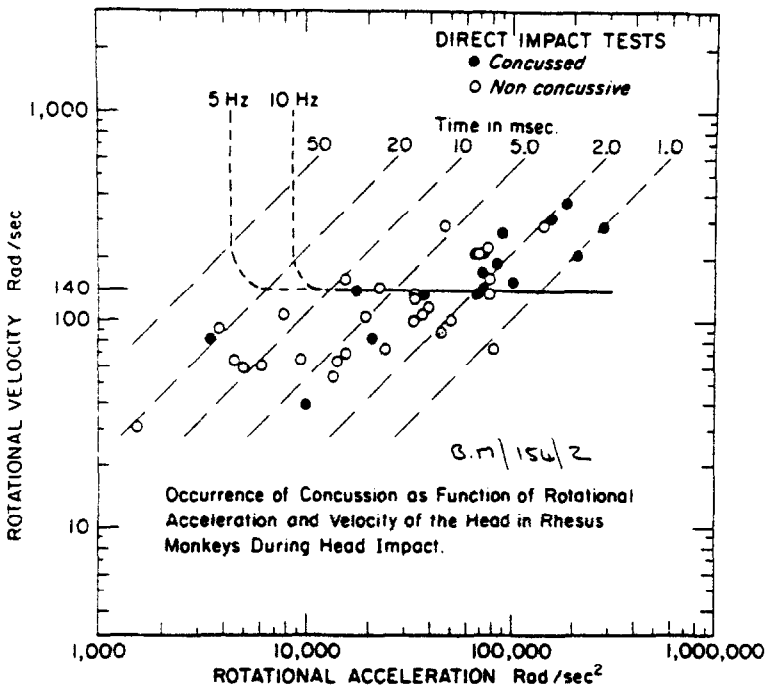

Fig. 2. Tolerance curve for Rhesus monkey depicting thresholds for 50 per cent probability of onset of cerebral concussion in terms of rotational velocity (for short duration blows) and rotational acceleration (for long duration blows) after direct head impact.

mental values derived from Rhesus monkey, squirrel monkey, and chimpanzee. It can be seen that the correspondence is good considering the crude approximation of brain frequencies and rough estimates of tolerance values made. From this curve we can predict that man may suffer a concussion in an exposure of over 1800 rad./sec². There is one case which has been studied from which some valuable data regarding mans tolerance may be deduced (Ommaya and Yarnell, 1969). Although in this patient clear-cut concussion with traumatic amnesia was not described, the production of a large subdural hematoma suggests a level of injury reasonably close to the threshold for cerebral concussion (Ommaya *et al.*, 1966b). In our animal experiments, hemorrhages over the brain surface occurred only in the concussed group.

The facts about this case are sufficiently detailed for us to try to estimate the rotational acceleration of the patient's head immediately after the collision.

When a moving vehicle strikes a stationary vehicle in a non-elastic collision both vehicles attain a common velocity. Knowing the masses of the truck and the car and their initial velocities we can apply the principle of the conservation of momentum to calculate this common velocity. In this case it is 32·7 ft/sec (about 22 mph).

Experimental work on automobile collisions suggests that the peak velocity (V_f) of the combined masses is reached about 0·16 sec (T_t) after the crash. Assuming that the time to each peak acceleration (A_f) is half this value, then:

$$A_f = \frac{(V_f/T_t)}{2} = \frac{32{\cdot}7}{0{\cdot}08} = 409 \text{ ft/sec}^2$$

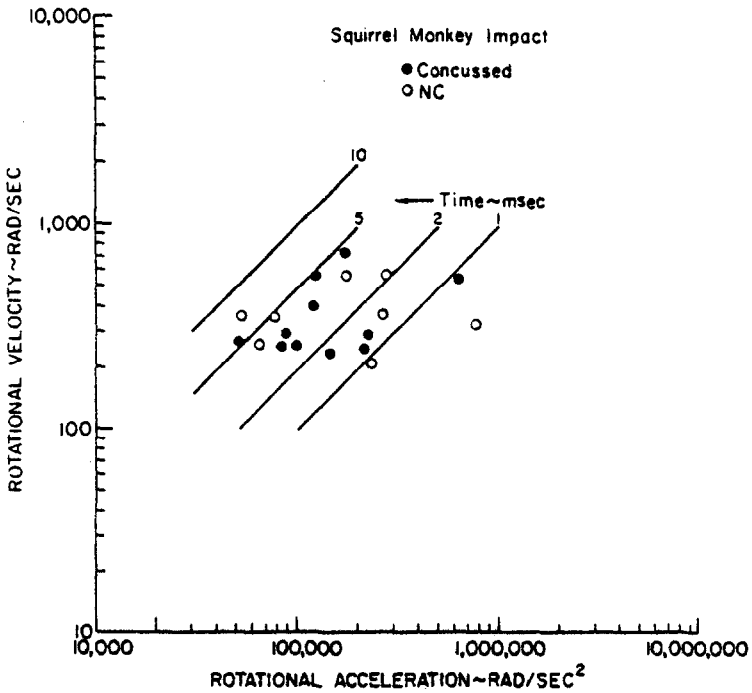

Fig. 3. Preliminary data plot of rotational velocity vs. acceleration during direct head impact in the squirrel monkey. The distribution of concussed and non-concussed data does not yet allow the drawing of tolerance curves.

Ewing (Ewing *et al.*, 1969) has suggested that the acceleration at a point on the head is at least twice that of the input acceleration to the base of a seated man. If the pivotal distance is 6 in., the rotational acceleration of the head would have been about 1636 rad./sec². In this case then, rotational acceleration of about 1600 rad./sec² caused brain injury in a human subject. This value has been included in Fig. 6. It can be seen that it falls within a reasonable area of the predicted tolerance level.

DISCUSSION AND CONCLUSION

Our experiments to date do not allow us to define the role of the contact phenomena of impact and particularly how they would scale to predict tolerance to head impact in man from data in lower primates except in as much as the associated head rotations may be consistently related to the local effects of impact. Our experiments in the chimpanzee strongly indicate that the local factors of skull and scalp thickness, ratio of brain–head weights and strength of the head–neck junction contribute greatly to the effects of impacts. Thus in Rhesus monkey and man the ratio of brain mass to head mass is usually greater than ½ whereas in the chimpanzee the relatively more massive skull and scalp reduces this fraction considerably. We have been impressed that the thick, movable and springy scalp combined with a thick strong skull in the chimpanzee serve as an excellent 'helmet' which raises the threshold for impact and has to date prevented the production of clear-cut cerebral concussion by impacts approaching 4000 lb of force applied for short durations

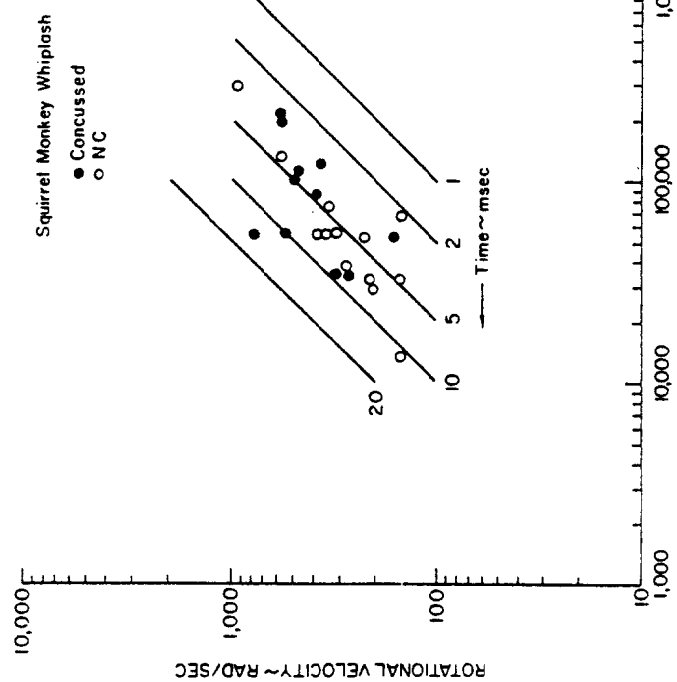

Fig. 4. Preliminary data plot of rotational velocity versus acceleration during whiplash injury in the squirrel monkey.

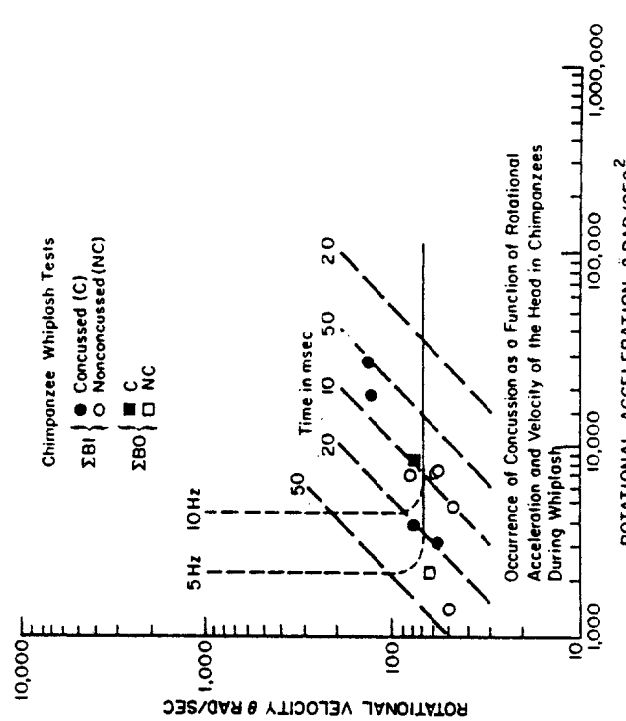

Fig. 5. Tolerance curve for onset of cerebral concussion in chimpanzee after whiplash injury. ΣBI and ΣBO at top of figure refer to 'eyeballs in' and 'eyeballs out' position of animal relative to direction of impact.

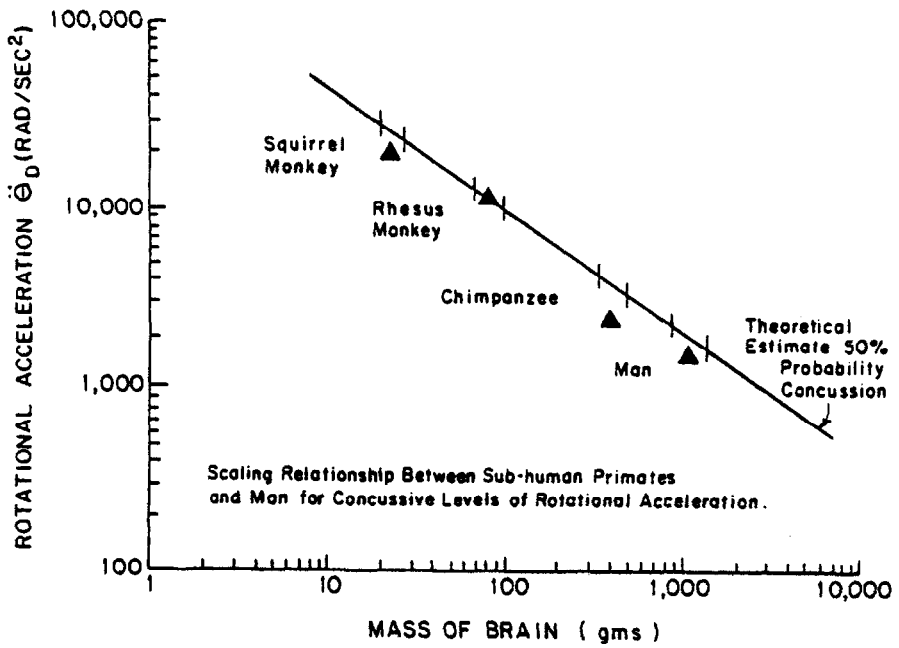

Fig. 6. Theoretical scaling of probability for onset of cerebral concussion in primates assuming that the crucial variable between species is mass of brain and that the crucial injury mechanism is severe shear strain imposed by brain rotation.

(< 5 msec, impulse < 2·5 lbsec). Contributing to this reduction of the injuriousness of the contact phenomena of impact by the head structure is the reduction of head rotation by the massive neck muscles of the chimpanzee. To date our impacts have not succeeded in producing head rotations approaching those achieved in concussive *whiplash* injury in the chimpanzee.

In conclusion, therefore, it appears that considerable work remains to be done but our current hypothesis remains a valid approach to definition of the comparative tolerances for brain injury by direct and indirect impact to the heads of primates.

We intend to develop more refined theoretical models, both discrete and continuous to further define the mechanism of brain injury as well as the tolerance of man's brain to mechanical trauma.

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