

Whiplash Injury and Brain Damage

An Experimental Study

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Experimental whiplash injury in rhesus monkeys has demonstrated that experimental cerebral concussion, as well as gross hemorrhages and contusions over the surface of the brain and upper cervical cord, can be produced by rotational displacement of the head on the neck alone, without significant direct head impact. These experimental observations have been studied in the light of published reports of cerebral concussion and other evidence for central nervous system involvement after whiplash injury in man.

Considerable controversy surrounds the term "whiplash injury," typically used to describe the violent flinging of the head on the neck produced by a rear-end automobile collision. This confusion concerning what is a very commonly made diagnosis, reflects the extreme scarcity of experimentally demonstrable facts which could clarify the medical as well as medicolegal issue. A few workers have, in the recent past, attempted to remedy this situation, but their efforts have been mainly directed to elucidating the injurious effect of whiplash mechanisms on the musculoskeletal system,¹⁻³ primarily in the neck structures. Our approach to this subject began indirectly in a study of experimental head injury in subhuman primates (primarily the rhesus monkey) over four years ago.^{4,5} Initially, we were concerned with establishing valid, reproducible experimental models of head injury with the intention of extrapolating data of preventive, diagnostic, and therapeutic significance to man along the continuum of the pri-

mate species.⁶⁻⁹ We had succeeded in defining, for the first time, various statistically significant levels of energy for the production of experimental cerebral concussion (ECC) in the monkey, in terms of the impulse of impact and resultant linear acceleration of the unrestrained head (Fig 1).^{5,10} During these experiments we discovered that a cervical collar, which reduced the rotational displacement of the head on the neck but did not prevent stretching of the neck or reduce linear acceleration of the head, succeeded in raising the previously established threshold for experimental cerebral concussion by head impact in 50% of the monkeys. In order to explain these observations it was imperative to determine why the presence of a cervical collar allowed the experimental subject to sustain head impact at impulse significantly greater than was possible if no collar was worn. The most significant variable affected by the cervical collar appeared to be the amount of rotational displacement of the head on the neck (expressed as either rotational velocity or rotational acceleration). Because such displacements are readily produced without head impact by a whiplash injury, we predicted that whiplash injury alone should produce cerebral concussion if rotational displacement of head was indeed a significant physical mechanism in head injury by impact. This prediction was supported by our experimental demonstration that levels of rotational acceleration of the monkey head exceeding 40,000 radians/sec² could produce experimental cerebral concussion, if the duration of such accelerations is greater than 10 msec.⁷ Our recent work has extended and supported these findings and significantly has revealed that in addition to reproducing the entity of experimental cerebral concussion *without direct impact to the head*, experimental whiplash injury can produce consistent brain damage in the monkey as evidenced by subarachnoid and subdural hemorrhage, cerebral contusions, and

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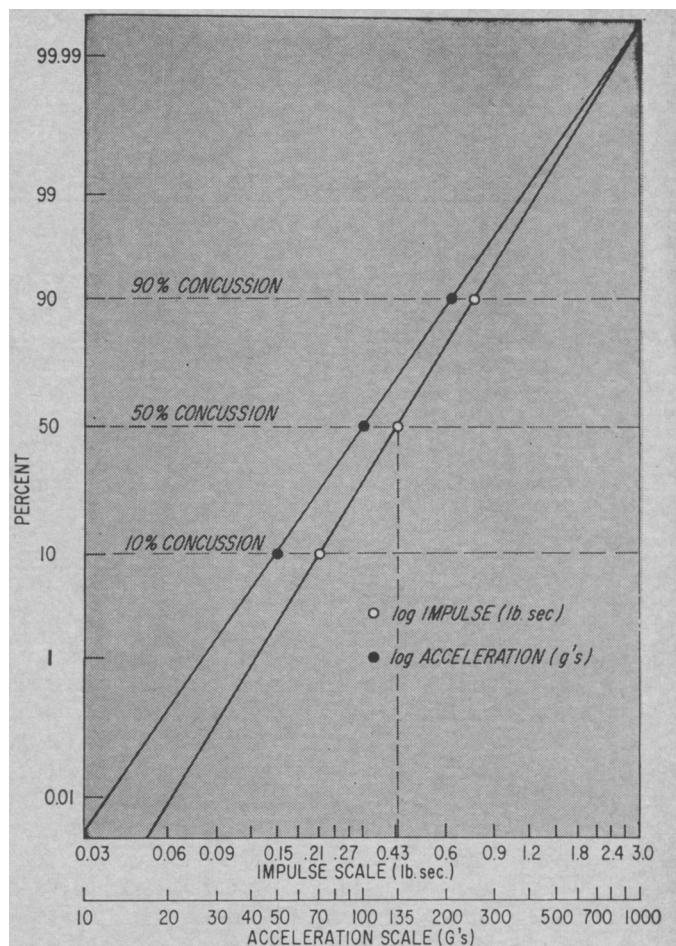
breakdown of the blood-brain barrier to Evans blue.

Method

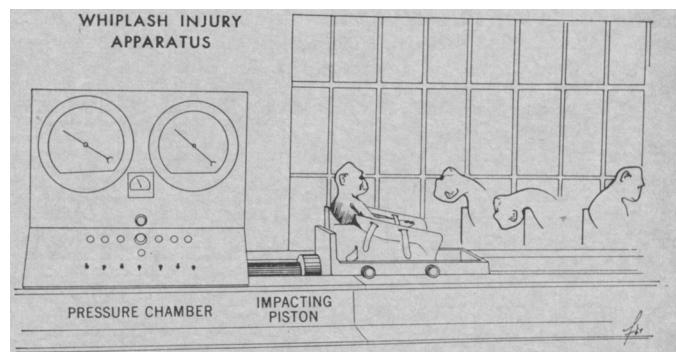
Rhesus monkeys were given barbiturate anesthesia and secured in a contoured fiberglass chair mounted on a rigid carriage. This apparatus could move freely on roller-skate wheels over a 20-foot long track. An air-compression device was attached at one end so that its piston could deliver impacts at various force levels to the rigid support of the monkey carriage. A braking system was constructed so that gradual deceleration of the carriage was achieved. Thus, the sudden impact could produce high accelerations of the carriage and forward-facing monkey so as to mimic a rear-end collision (Fig 2). The resultant whiplash of the head on the neck and shoulders of each animal was recorded by a high-speed, 16-mm movie camera at 1,000 frames/sec. Detailed analysis of such movies allowed measurement of the rotational or angular displacement of the monkey's head which could be related to elapsed time. From such data the rotational velocity and acceleration could easily be calculated. These movies also served to confirm that although the back of the head was stopped by the back of the chair, no significant direct head impact was sustained. This was confirmed by additional experiments which the back of the head was stopped by crushable materials of known strength. The levels of impact sustained were less than that required for concussion in 10% of the animals (Fig 1). Normal speed 16-mm movies at 24 frames/sec were also taken of the animals' responses and behavior before, during, and after the whiplash injury. Before each whiplash experiment, 10 cc of 0.5% Evans blue was given intravenously. The animal was then allowed to attain a light plane of anesthesia so that aversive responses to stimuli were consistently present. The loss of such behavioral responses, as well as abolition of superficial reflexes (eg, the corneal and palpebral reflexes) was our criterion for the production of experimental cerebral concussion. After sustaining the whiplash injury the animals were observed for varying intervals (from one hour to one week after trauma). After the administration of pentobarbital (Nembutal) anesthesia the animals were killed by perfusing the arterial system with isotonic saline and 10% formaldehyde-saline. The entire brain and proximal spinal cord were then removed as part of a routine autopsy. This report is concerned primarily with the *macroscopic* findings in the brain and cervical cord at 24 hours after whiplash injury in monkeys who had apparently completely recovered from the cerebral concussion induced by the whiplash trauma. Detailed pathological results, including microscopic alterations at various intervals after trauma, will be given in later reports.

Results

Over 50 monkeys have received whiplash injury in our experiments to date. Of this number, 19



1. Physical indices for cerebral concussion in 10%, 50%, and 90% of a group of rhesus monkeys. Acceleration values directly recorded by piezoelectric accelerometers; impulse values calculated from directly recorded force of impact curve.



2. The impacting piston delivers blows of controlled force to the reinforced rear of the monkey chair. Diagrams of the monkey's head and shoulders show the type of head displacements that occur during the whiplash injury.

animals were considered to have been concussed according to the criteria described in the section on methodology. Fifteen of these 19 monkeys were found to have visual evidence for macroscopic damage as marked by surface hemorrhages which were primarily subdural in nature. In the nonconcussed group, there were 22 in whom adequate postmor-

Effects of Experimental Whiplash Injury

Monkey No.	Duration of Experimental Cerebral Concussion	Distribution of Visible Hemorrhages and Contusions
W-16	2 min, 10 sec	None
W-28	5 min	Right parietal convexity, left lateral aspect of medulla and both temporal poles; minimally over anterior aspect pons and medulla bilaterally
W-29	2 min	Left frontoparietal convexity; bilateral parasagittal parietal region contusions along falx; subtemporal (R>L) and frontal (L>R) contusions
W-31	2 min, 36 sec	Diffuse right hemisphere hemorrhage and contusions; maximal in parietal zone, lesions on anterior midbrain and pons and right subtemporo-occipital surface
W-32	20 min	Bilateral hematoma, R>L; posterior aspect medulla and cervical cord, both frontal and temporal poles and left occipital lobe contusions; hemorrhage and contusion in left brachium pontis extending into adjacent cerebellum
W-33	32 sec	Bilateral parietal parasagittal zones
W-34	32 sec	Similar to W-33 plus bilateral subtemporo-occipital contusions
W-35	3 min	Left frontal parietal convexity contusion and clots; subtemporo-occipital surface small prepontine contusions
W-36	2 min, 30 sec	Left frontoparietal convexity, right subtemporo-occipital zone
W-37	2 min, 12 sec	Bilateral hematoma entire hemisphere; diffuse subarachnoid hemorrhage, including cervical cord
W-38	3 min, 15 sec	Left midparietal convexity
W-39	4 min	Bilateral parasagittal contusions along falx; small subarachnoid hemorrhage
W-40	30 sec	Bilateral parasagittal contusions and hematoma, L>R; maximal in parietal zone; contusions also at frontal poles, orbital cortex, and temporal poles; small midpontine contusion
W-42	25 sec	None
W-43	6 min	Minimal parasagittal contusions along falx; lower cervical cord contusion and hemorrhage
W-44	3 min	Subdural hematoma over posterior aspect upper cervical cord
W-48	30 sec	None
W-50	1 min, 30 sec	Left parietal convexity and left lateral aspect of cervical cord subdural effusions
W-51	25 sec	None

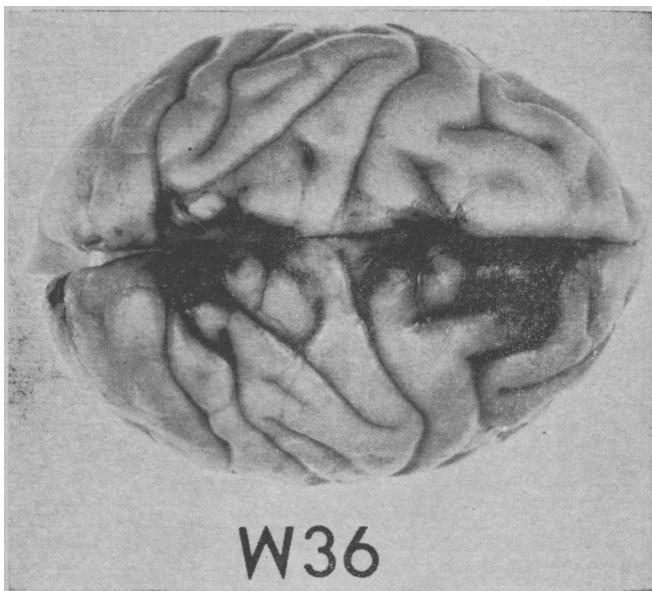
tem studies have been completed. Not a single animal in the nonconcussed group showed any macroscopic evidence of brain damage; in particular, there were no surface hemorrhages. The distribution of the surface hemorrhages noted in the concussed series of animals is summarized in the Table. It will be noted that the parietal parasagittal zones, the medial supra callosal surfaces of the two cerebral hemispheres, the tips of the frontal and temporal lobes, the inferior orbitotemporal and temporo-occipital (particularly the latter) cerebral surfaces, and the brain stem, including the upper cervical cord, were primarily involved. Representative photographs of the cerebral hemisphere lesions seen are shown in Fig 3 and 4. The brain stem and cervical cord showed surface hemorrhages in eight of this series of animals, and in two the hemorrhage was almost entirely confined to the upper cervical cord. Only one animal had a visible lesion in a deeper structure of the brain (a hemorrhage extending into the cerebellum from the brachium pontis). All the other animals with gross surface hemorrhages showed no macroscopically visible lesions in the deeper structures.

The values for rotational acceleration of the monkey head for 40 whiplash injury experiments are plotted against the *durations* of such acceleration (Fig 5). Nineteen concussive and 21 nonconcussive points are shown, and the resultant curve is drawn so as to separate most of the concussive from the nonconcussive data. The validity of this curve has been confirmed by our current experiments which suggest that in the time regime studied, the threshold of cerebral concussion is velocity rather than acceleration dependent (A. E. Hirsh and A. K. Ommaya, unpublished data). These observations have certain important implications for the mechanism of cerebral concussion and also implications for other effects of brain trauma.

Comment

The curve in Fig 5 is a technique of data presentation based on the method of showing gravity-tolerance data as a *sensitivity curve* first published by Kornhauser and Gold in 1954.¹¹ It is an excellent technique to show how *time duration* affects the peak acceleration necessary to induce a given level of response (ie, cerebral concussion in this case). Thus, the shorter the duration, the greater the amplitude of angular acceleration required for the same response (concussion). One important application of such a curve is to obtain the critical amplitudes and durations of such velocities and accelerations at which concussion and brain damage will first begin to occur in a certain percentage of animals. Similar data from a series of subhuman primates of gradually increasing brain weight (eg, squirrel monkey, rhesus monkey, and chimpanzee) will then allow a more valid extrapolation of such experimental data to the injury threshold for whiplash injury in man in terms of the rotational velocity or acceleration of the head. We are currently developing this scaling method theoretically and experimentally on the basis of an assumption that the level of injurious rotational acceleration is inversely proportional to the two-thirds power of the mass of the brain.*

Although a considerable body of medical literature on whiplash injury is to be found, there has been a tendency to decry the organic reality of the complaints expressed by patients sustaining such injuries unless evidence of neurological deficit on clinical examination is found. Apart from the excellent papers of Torres and Shapiro¹² and a few other workers¹³⁻¹⁵ the possibility of significant, albeit not totally disabling, brain damage following whiplash injury has not been given serious consideration. We do not contend that our experiments prove that whiplash injury in man can produce lesions similar

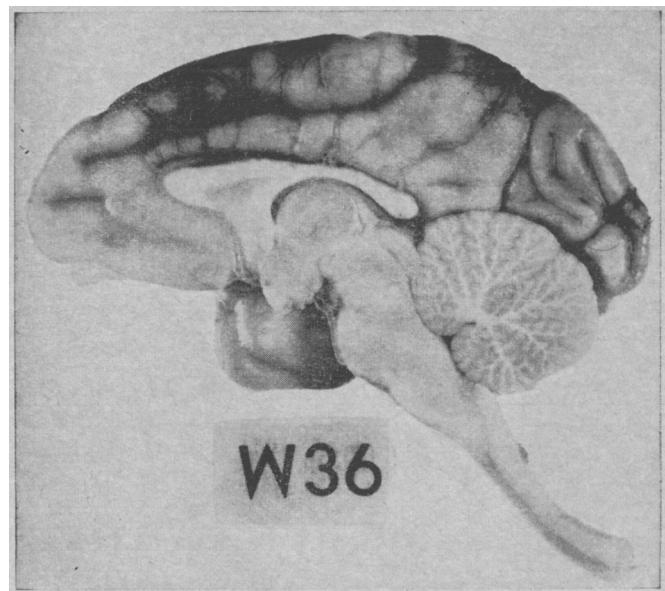


3. Cerebral hemispheres of monkey W-36 viewed from above. Note the significant parasagittal frontoparietal contusions. Subclinical hematomas overlay these contusions bilaterally.

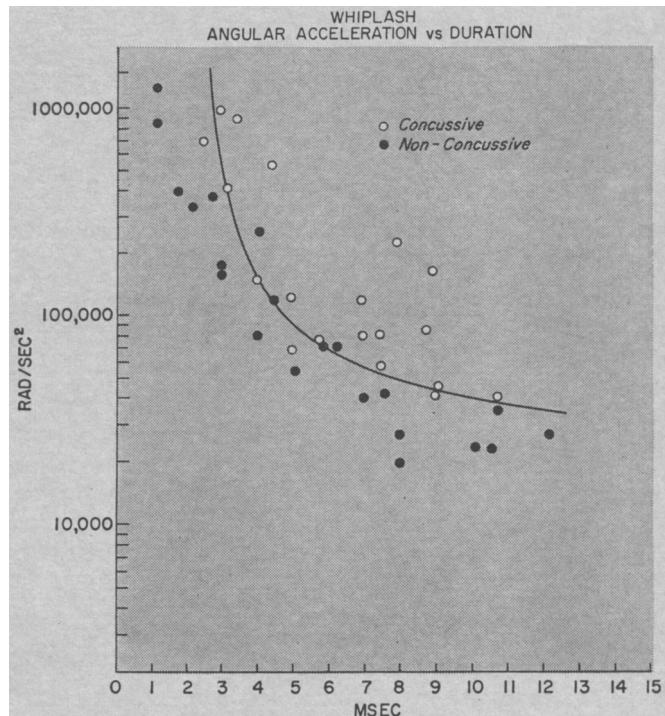
to what we have demonstrated in monkeys. This contention can only be made if two conditions are fulfilled: (1) the quantitative demonstration of the actual angular accelerations that can be sustained by the human head under various conditions producing a whiplash, eg, rear-end automobile collisions, at various speeds; and (2) the quantitative demonstration of a valid scaling method based on sound theoretical and experimental evidence showing that the extrapolation of such injury threshold data to the human head indicate levels of angular acceleration which actually fall within the range found in the demonstration required in the first proposition.⁸

Neither of these conditions have been satisfied to date and one is left with two facts. First, that experimental cerebral concussion and brain injury, as indicated by surface hemorrhages, can be produced by whiplash injury in a fairly reproducible manner in the monkey and that such injury is compatible with survival of the animal without obvious neurological deficit. This does *not* mean that significant acute or delayed neurological and behavioral defects will not be found if more precise investigations are made. Second, because it is easier to injure the much larger brain of man as compared to that of the monkey, it is suggested that the levels of angular acceleration required to produce cerebral concussion and brain injury in man will be much lower than those shown in Fig 5, perhaps in the order of 6,000 to 7,000 radians/sec².⁸

The demonstration of such brain hemorrhages produced by severe rotational acceleration of the head supports the theory of Holbourn on the mechanics of head injury.¹⁶ Based on experiments with gelatin models of head cross-sections and two-di-



4. Medial aspect of sagittal section of brain seen in Fig. 3. Note the paramedian blood staining of the brain.



5. The relation of amplitude to duration of rotational accelerations of the head recorded in experimental whiplash injury. Time (abscissa) is measured in milliseconds and acceleration (ordinate) in radians/sec² (radian = 57.29 degrees).

mensional photoelastic-stress analysis techniques, Holbourn predicted the areas of maximum shear strain that would be produced in the brain after a head injury. Among such areas the parasagittal zones and fronto-orbito-temporal surfaces were most prominent. The cortical draining veins enter the sagittal sinus in the parasagittal zone and thus are particularly liable to rupture. We found that subdural bleeding in these areas was by far the

commonest visible lesion. Although there is no direct evidence, it is also reasonable to assume that cerebral concussion is also produced by such shear strains generated by the rotational acceleration of the head and its contents.

In a survey by Gay and Abbott it was found that in 62% of cases of whiplash injury in man, there is historical and symptomatic evidence of a cerebral concussion.¹³ Frankel reported an incidence of 22% to 30% of cerebral concussion in another series of whiplash injuries.¹⁴ Our experimental findings are consistent with such clinical experience while serving also to illuminate the mechanics of such injuries to man.

The clinical implications of such investigations are twofold. First, patients sustaining whiplash injury severe enough to produce significant complaints, particularly if they persist for 24 hours, should receive at least the type of treatment given patients with simple closed-head injuries producing cerebral concussion. A history of disturbance of consciousness of even an apparently mild nature, such as a short period of retrograde or posttraumatic amnesia alone, should be sought for diligently. Electroencephalographic¹² and electrocardiographic¹⁰ tests may well prove of significant prognostic value if obtained on at least two occasions in the early postwhiplash-trauma period. One should also bear in mind that a small amount of subarachnoid hemorrhage can produce very marked headaches and a stiff neck, two common complaints of patients after whiplash injury which are usually attributed to injury of musculoskeletal structures

and the cervical roots and their branches. Second, a more rational therapy of such injuries needs to be devised. However, it is only after further investigations to satisfy the two conditions outlined above are completed that such therapeutic aspects will be capable of solution.

Until such time, however, it is a matter of crucial importance that we investigate and manage the clinical problems of whiplash injuries in our patients not only with regard to the musculoskeletal and peripheral nervous systems, but also with greater attention to the finer details of behavioral and neurological deficits and to the results of our special investigations. Furthermore, we should search for points of clinical-pathological resemblance between patients with head injury and others with whiplash injury. This recommendation is based on our experimental evidence which suggests that the whiplash type of injury mechanism may be of significant importance in producing the effects of closed-head injuries under conditions when the head is free to move.⁷

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Generic and Trade Names of Drug

Pentobarbital—Nembutal.

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