

**THE PATHOLOGY AND MECHANICS
OF EXPERIMENTAL CEREBRAL CONCUSSION**

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FOREWORD

This report is the second on experimentally produced concussion. WADC Technical Report 58-193, "Biophysics of Concussion," reports the initial studies. The second part of WADC TR 58-193 was written by the author* of this report and includes his early findings regarding the neuropathology of experimental cerebral concussion. The work reported herein was performed in the Bionics and Neurophysiology Section, Bioacoustics Branch, Biomedical Laboratory, Aerospace Medical Laboratory, and in the Laboratory of Neuropathology, Medical Center, University of Michigan. The investigation is in support of Project No. 7231, "Acoustic Energy Control," Task No. 71786, "Biological Aspects of Vibratory and Acoustic Energy." Captain R. G. Hansen served as task engineer. The technical assistance of Robert Horne is gratefully acknowledged. This study is now being printed in the A. M. A. Archives of Neurology.

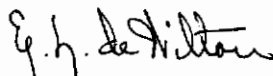
The animal experimentation reported herein was performed in accordance with the "Rules for Animal Care" established by the American Medical Association.

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ABSTRACT

A blow to the cat's head produces an abrupt displacement at the craniocervical junction. This results in a fiber lesion at the ventral circumference of the spinal cord at C-1, opposite the prominence of the odontoid process. Thick fibers are affected more severely than thin fibers. Axonal reaction is found in the nucleus gigantocellularis of the reticular formation, the nucleus vestibularis lateralis, the red nucleus, and others. All these nuclei send their descending fibers through the damaged region. Changes are consistent. Intensity is related to the severity of concussion so that the duration of concussion can be estimated histologically without knowledge of the experimental data. The mechanical forces used to produce concussion are the same type described by other investigators. An experimental analysis of the mechanical factors involved in the production of damage reveals stretch and flexion to be most important.

PUBLICATION REVIEW



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Contrails

THE PATHOLOGY AND MECHANICS OF EXPERIMENTAL CEREBRAL CONCUSSION

INTRODUCTION

Concussion is a transient alteration of the central nervous system resulting from an impact to the head. It is defined physiologically by a temporary abrogation of certain reflex functions and pathologically by the absence of gross destruction of brain substance but presence of finer cellular changes. Both physiological and pathological evidences indicate a particular affection of the brain stem.

Denny-Brown and Russell (ref. 5) distinguished an acceleration concussion of the freely movable head from a compression concussion of the rigidly fixed head. The present investigation is limited to the neuropathology and mechanics of experimental acceleration concussion in cats. The relationship of acceleration concussion to compression concussion is discussed on the basis of these findings.

Two experimental series are reported in this article. The first describes the histopathology of cats subjected to concussion* as compared to the symptoms and the mechanical forces involved. The second describes several experiments analyzing the mechanics of concussion.

MATERIALS AND METHODS

Sixty-nine cats were used in the various experiments reported in this article. Eighteen were used for the experiments reported in the first part studying the effects of a blow to the head by means of a pneumatic hammer. These cats received 0.1 cc/pound Dial. before the experiment, reducing the motor activity and facilitating handling of the cats but not rendering them unconscious. The loss of consciousness upon concussion, therefore, could be observed.

* Hereafter called "concussed cats"

The cats were struck at the region of the sagittal suture by a pneumatic hammer driven by compressed nitrogen. The pressure in the driving device could be varied ad libitum and was set routinely at 250 pounds per square inch. The striking device was so arranged that the plunger traveled 1-1/4 inches prior to making contact with the head and an equal distance after making contact. High-speed cinematographic recordings of the plunger showed a linear increase of velocity reaching 39 feet per second within 9.1 milliseconds. The acceleration at the start of the movement was about 30 feet per second².

If the contact surface of the plunger was small, impression fracture invariably resulted. To protect the cats from fracture, a small acrylic helmet, reinforced with a metal disk, was molded to get the contour of the skull. The cat's head, with the helmet attached but not firmly fastened, was positioned to a resting bar which facilitated delivery of the blow in the same manner each run. Upon contact with the plunger, the head was allowed to give way and the helmet flew free from the skull.

An effort was made to obtain three groups of cats with reflex abrogation (concussion) from 0 to 30 seconds, from 30 seconds to 1 minute, and exceeding 1 minute. Animals with a loss of corneal reflex or respiration in excess of 1 minute had a high mortality. To obtain a sufficient number of cats in this group multiple blows summing several concussions of shorter duration were struck. More than four blows were never applied. All were applied on the same day with about 1-hour intervals. Fatal runs were not included in the experimental material.

High-speed cinematographic recordings were taken from 11 experiments at a speed of 3,000 frames per second. Velocity and acceleration were computed for the cat's nose and the frontal bone. Third and fourth order equations were fitted by the least squares method to the scatter graph of displacement versus time from contact time.

The cats were killed under pentothal anesthesia after a survival time of 8 days, the optimal period to produce maximal chromatolytic changes. The brains were perfused according to the prescriptions of Koenig, Groat, and Windle (ref. 24). A complete series of paraffin sections, 20 microns thick, was cut from the medulla oblongata and midbrain of several cats. Most others were cut to incomplete series providing about 120 sections per animal. In addition, representative samples were cut from the cerebral hemispheres, the diencephalon, and any region not appearing normal macroscopically. The portion of the cervical cord from the caudal part of the fourth ventricle to the second cervical segment was cut median sagittally. One half was embedded in paraffin while the other was cut serially into frozen sections, 17 microns thick.

The paraffin material was stained with Einarson's chromalum galloxyanin and a small part also with hematoxylin eosin. The frozen sections were stained with the unreduced variant of Hortega's silver carbonate technique (ref. 31) and with sudan black.

FIRST SERIES

Symptoms of Concussion

The typical symptoms of experimental concussion (refs. 5, 16, 29, and 35) are: loss of consciousness (failure to react to stimuli and loss of postural tonus); temporary abrogation of corneal reflex, pinna reflex, and respiration; a steep rise of blood pressure; an initial jerk followed by postural tone and flaccidity; and irregularities of the heart rate. This symptomatology is consistent among various species such as monkeys, dogs, cats, guinea pigs, rabbits, and even frogs (ref. 24). These symptoms were observed in the present experiments.

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The duration of loss of consciousness always exceeded the reflex abrogation. Consciousness returned gradually, the animals passing through a period of drowsiness, sluggishness, and poor coordination. Normal behavior returned within a few hours of the experiment.

Abrogation of corneal reflex and of respiration are useful criteria to determine the severity of concussion (ref. 5). The respiratory failure usually lasted from 10 to 50 seconds, but longer failures were observed occasionally. The durations of abrogation of corneal reflex and respiration were usually in the same order, but the respiratory failure exceeded the abrogation of corneal reflex in the majority of the cats. Cats with respiratory failures exceeding 1 minute would sometimes show a temporary reappearance of the corneal reflex before respiration returned. A temporary reappearance of the corneal reflex was also observed in a few fatal blows after which respiration did not recover.

The severe concussions showed an initial jerk with general rigidity, extension of limbs, and retraction of the head (stretch response), followed by general flaccidity. Clonic movements of the limbs or loss of urine and defecation were observed only in lethal blows. Vomiting was observed only once.

The arterial pressure was not recorded. Bradycardia was observed in about 80 percent of the experiments. The rest showed tachycardia. Bradycardia frequently followed subconcussive blows while tachycardia accompanied severe concussions. However, no definite correlation could be established. The irregularities of heart rate disappeared quickly within seconds or a few minutes. Bradycardia was considered a subthreshold effect by Denny-Brown and Russel (ref. 5); but Gurdjian and Webster (ref. 16) observed bradycardia also in moderately and severely concussed cats; and Walker, Kollros, and Case (ref. 35) list bradycardia among the typical general symptoms of concussion.

Physical Analysis of the Blow

Denny-Brown and Russell (ref. 5) obtained concussion if the head was struck by a pendulum moving at approximately 29 feet per second. In the present experiments, maximal velocities of 28 to 42 feet per second were recorded at a pad on the frontal bone of the cats. They were reached in an average of 0.004 second (range from 0.001 to 0.008 second). Figure 1 shows the velocity plotted against the respiratory failure: there seems a rather narrow range of concussive velocity since one cat was not concussed at 34 feet per second while two fatalities occurred at 41 feet per second. Two cats with impression fractures were not included in the table because of widely discordant results.

In addition to the velocity of the pad at the frontal bone, also the velocity of the cat's nose was computed. Comparison of the data revealed that the velocity of the nose was about 1.5 times greater than that of the frontal bone (range, from 1.2 to 1.9) as shown in figure 2. The different velocities obviously resulted from the fixation of the head at the neck, while the nose could move freely. The craniocervical junction, therefore, absorbed energy resulting in a considerable strain.

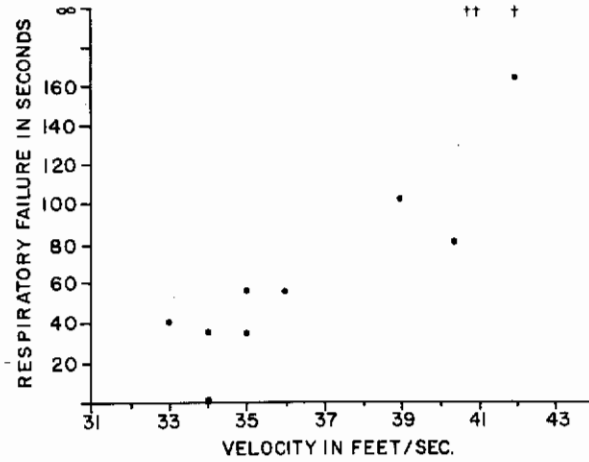


Figure 1. Duration of Respiratory Failure Plotted Against Velocity of the Head (Recorded at the Frontal Bone)

The three crosses indicate fatal runs.

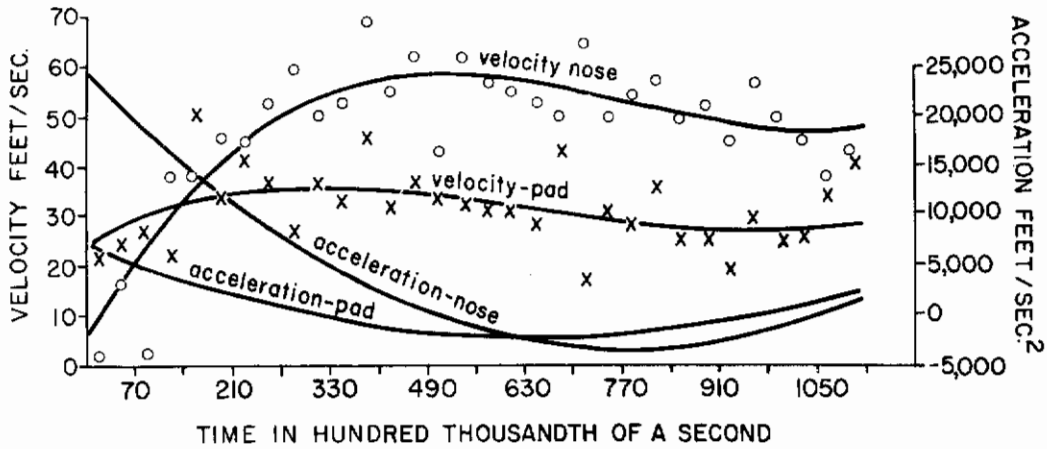


Figure 2. Velocity and Acceleration Recorded at the Cat's Nose as Compared to That of a Pad at the Frontal Bone

All measurements were taken from the same film strip. There are considerable differences of velocity and acceleration between parts of the skull. The crosses and circles mark the individual measurements.

The different velocities of parts of the skull are critical for the comparison of the data of various investigators. Denny-Brown and Russell used a pendulum striking at the occipital bone at 29 feet per second. The velocity of the cat's nose, thus, was certainly greater as evidenced in the present experiment where a maximal velocity of 68 feet per second was recorded at the nose although the plunger yielded only 36 to 40 feet per second. Our cats were struck parietally but the cats of Denny-Brown and Russell were struck occipitally. Our higher concussive velocities, therefore, reflect only the different site of application. A head struck at a velocity of 29 feet per second at the occipital bone probably obtains a velocity of 35 to 40 feet per second at the frontal region and up to 60 feet per second at the nose. Govons (ref. 12) reported a speed of 64 feet per second for the head of dogs concussed by the explosion of blasting caps. The site of recording was not disclosed (high-speed movies), but coincides with that of the cats' noses in the present experiment. The data of Govons and Denny-Brown and Russell, therefore, are probably not discrepant, contrary to Govons' interpretation.

The accelerations computed from the velocity curves showed a considerable variation ranging from 5,000 to 16,000 feet per second² (about 150 to 500 g) with an average of 9,800 feet per second² (300 g). The quantitative relationship of concussion and acceleration was not as clear-cut as that of velocity and concussion. This agrees to some extent with the findings of Gurdjian et al. (ref. 17) who recorded accelerations in the same range. The voluntary human tolerances reported by Lombard et al. (ref. 27) are much smaller (38 g). The importance of intracranial pressure changes, emphasized by Gurdjian et al., is discussed later.

Neuropathological Findings

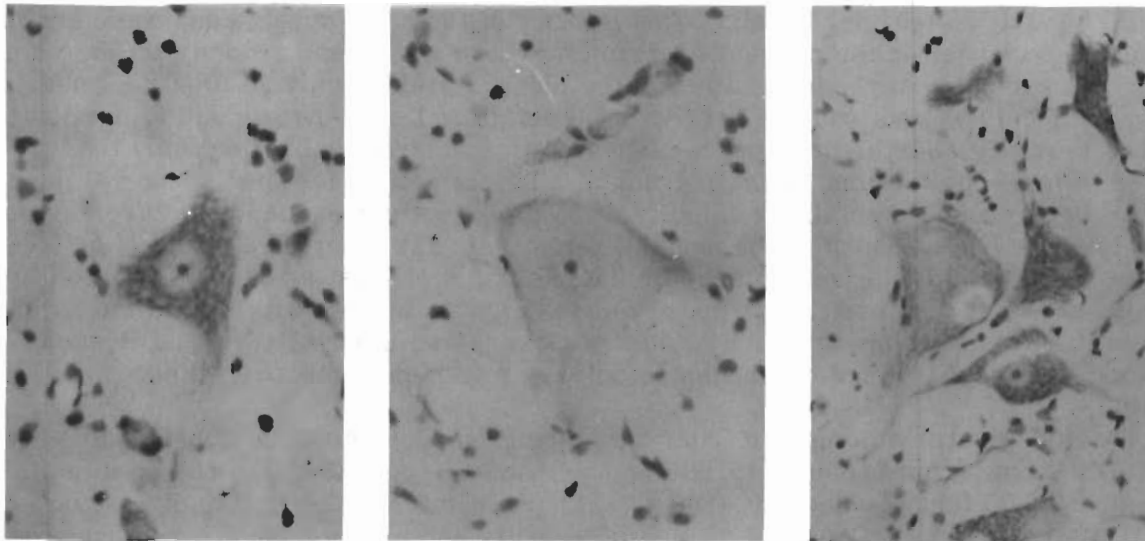
Various histological findings have been described following experimental concussion but a consistent, typical pathology was reported first by Windle et al. (refs. 39, 40, 41). These authors observed chromatolysis and loss of nerve cells in the reticular formation, the lateral vestibular nucleus, the red nucleus, and less consistently in some other nuclei of the medulla oblongata. The motor nuclei of cranial nerves were not damaged. Identical cell changes were found in the present experiment. In addition, however, there was a typical fiber lesion of the ventral circumference of the first cervical segment and this lesion proved the cause of cell changes. Fiber damage was also observed by Jakob (ref. 23) and Windle, Groat, and Magoun (ref. 39), but a relationship of fiber damage and chromatolysis was denied by the latter.

Cell Changes. — All showed chromatolysis in the reticular substance of the medullae oblongatae and the lateral vestibular nucleus. The extent of damage was related to the severity of the concussion.

The affected neurons were swollen and showed a central chromatolysis with dustlike disintegration of Nissl bodies. Eccentric nuclei were found in the lateral vestibular nucleus, but were not so frequent in the reticular formation. The nucleoli were vacuolated. More advanced changes showed a complete chromatolysis of nerve cells (ghost cells) with shrunken and deformed nuclei (figure 3). Finally the cells disintegrated.

The reticular formation of the medulla oblongata was maximally affected in its nucleus gigantocellularis. * Less affected were the caudally adjacent nucleus reticularis medulla oblongata and the cranially adjacent nucleus reticularis pontis. In all these nuclei the medial, large-celled part was more affected than the lateral, small-celled part.

*The nomenclature of the nuclei of the reticular formation refers to Brodal (ref. 2).



A. Normal Nerve Cell in the Reticular Formation B. Complete Chromatolysis of a Nerve Cell of the Reticular Formation C. Chromatolytic Neuron in the Red Nucleus

Figure 3. Neuron Chromatolysis Produced by Experimental Concussion (using chromalum gallocyanin)

The large Deiter's cells in the nucleus vestibularis lateralis showed central chromatolysis like the nucleus reticularis gigantocellularis. The magnocellular part of the red nucleus also showed central chromatolysis but was affected only in the severe concussions. The parvocellular part was not damaged. Alterations of large pyramidal cells in the cerebral cortex (swelling and apical dislocation of the nucleus) were observed occasionally in severe concussions. A few chromatolytic cells were found in the nucleus of the descending trigeminal tract, the lateral nuclei of the medulla oblongata, and some other nuclei; but changes in these nuclei were not a typical sequel of concussion.

All the motor nuclei of the cranial nerves and most of the sensory nuclei of cranial nerves were not damaged. The protection of these nuclei was particularly conspicuous at levels where damaged nuclei were located in their immediate neighborhood.

Fiber Damage. —A circumscribed fiber lesion at the ventral circumference of the first cervical segment was found in all cats. To demonstrate this lesion, the brain and spinal cord must be removed without transection and the region between the caudal end of the fourth ventricle and the second cervical segment should be cut serially in longitudinal sections.

Fibers were damaged in a roughly triangular area symmetrical to the ventral median fissure. Longitudinal sections (figure 4) showed maximal damage of the spinal cord at C-1, grading off rapidly toward the medulla oblongata. The fibers caudal to C-1 showed a typical Wallerian degeneration. The damaged area thus corresponded with the level of the odontoid process.

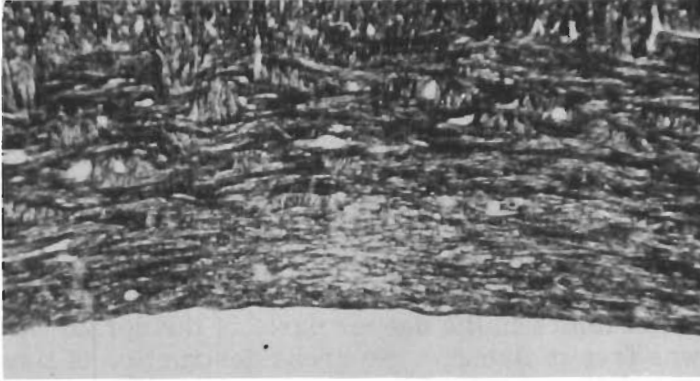


Figure 4. Longitudinal Section of the Ventral Tracts at C-1, Showing a Circumscribed Lesion at the Level of the Odontoid Process (Stained with unreduced variant of Hortega's silver carbonate technique)

After a standard survival time of 8 days, the lesion showed an interruption of the thick fibers with typical terminal swellings and clubs (figure 5). Fibers of medium thickness and thin fibers were occasionally affected in severe lesions, but the majority of thin fibers was not damaged. Most of the terminal fiber swellings were found at the level of C-1, scattered over a region of several millimeters length. They were found neither farther caudally beyond C-2, nor cranially beyond the pyramidal decussation.

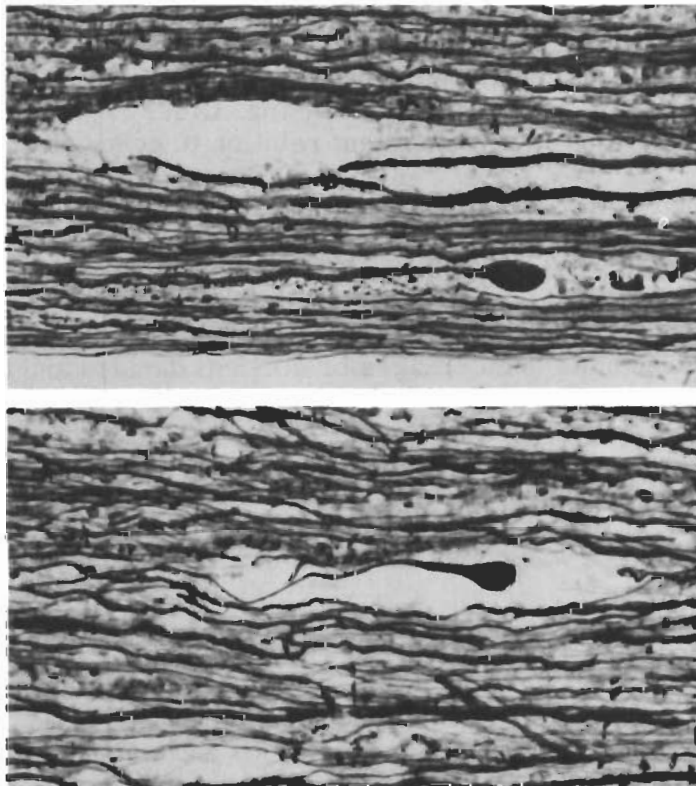


Figure 5. Typical Terminal Swellings of Interrupted Thick Fibers. Thin Fibers are not Damaged (stained with unreduced variant of Hortega's silver carbonate technique).

The myelin sheaths of the thick fibers caudal to C-1 showed an irregular structure or a complete disintegration and there was incorporation of myelin particles into macrophages. The sheaths of medium-sized fibers were less affected, showing only slight irregularities. The sheaths of thin fibers were not damaged even if immediately adjacent to disintegrated thick fibers. No fat was found after a survival time of 8 days, but after 6 weeks massive deposits of fat demarcated the degenerating tracts. Little fat was found proximal to the lesion.

The size of the damaged area varied with the severity of the injury. In severe injuries, the damage extended into the lateral portions of the ventral fasciculi. The lateral and dorsal fasciculi, however, were not damaged in most of the animals. Only a few very severe injuries showed some damaged fibers in the deeper parts of the dorsal tracts. The dorsal circumference of the cord was free of damage. No gross destruction of tissue occurred in the lesion. On the contrary, the persistence of the thin fibers tended to conceal the true extent of damage.

Small hemorrhages about the canalis centralis were occasionally found following severe concussion. The level at which these hemorrhages occurred coincided with that of the fiber lesion, but the latter was independent from the hemorrhages. Jakob (ref. 23), Denny-Brown and Russell (ref. 5), and Peters (ref. 28) described these hemorrhages as being a frequent, but not consistent, complication of experimental concussion.

The chromatolysis in the nuclei of the medulla oblongata obviously represented an axonal reaction resulting from the fiber damage at C-1. The specific affection of certain nuclei was explained by the path of their descending fibers which all passed the damaged region at C-1. These anatomical relations are discussed later.

Inconsistent Complications. —The findings described above accompanied concussion consistently. Their intensity was related to the severity of concussion. Other regions were occasionally damaged but the damage showed no consistent relation to concussion.

Cortical lesions were rare and none of the cats showed a typical cortical contusion with complete tissue necrosis. This protection from cortical contusions, in spite of a blow capable of producing impression fracture, was attributed to the use of the helmet. Subarachnoid hemorrhage was found in several cats, but in only two of them was it accompanied by a focal but incomplete loss of nerve cells in the cortex with proliferation of microglia. Correlation of either subarachnoid hemorrhages or cortical damage and the symptoms of concussion were evident.

Other complications of concussion were inconsistent chromatolytic changes in the motor neurons of the anterior column of the upper segments of the cervical cord. This chromatolysis probably resulted from an occasional insult to the anterior roots, but neither localization nor intensity of the damage was consistent or showed relations to the symptomatology.

Correlation of Severity of Concussion and Extent of Histological Damage

The preceding text referred several times to correlations between the extent of histological damage and the severity of concussion. These observations were substantiated by evaluating the histology without knowledge of the experimental data.

The intensity of the fiber damage at C-1 was described in terms of weak, mild, or severe, on the basis of the number of interrupted and swollen fibers. Such a quantitative evaluation of the series was facilitated by the exceptional reliability of Hortega's silver carbonate technique which permitted staining all sections at equal intensity without a single failure. The experimental protocols were not disclosed before the histological investigation was completed. Table I shows the results of this experiment.

TABLE I
CORRELATION OF CONCUSSION AND HISTOLOGICAL DAMAGE

Cat No.	Intensity of Fiber Damage	Respiratory Arrest Each Blow
1493	weak	0
1470	weak	0
1504	weak	0, 0, 0, 0
1453	weak	10"
1495	weak	24"
1503	weak	10", 0, 0, 12"
1467	weak	32"
1462	weak	40"
1484*	weak	1' 30"
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1458	weak-mild	46"
1457	mild	35"
1466	mild	30", 1' 30"
1490	mild	3'
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1492	severe	35", 43"
1439	severe	40", 20", 30"
1508	severe	40", 30", 25", 30"
1485	severe	2' 45", 3'
1512*	very severe	0, 0, 23", 0

*These two cats showed an obvious discrepancy of pathological and experimental data.

The table discloses that the histological findings permit a reasonably accurate estimate of the severity of concussion. Only two of the cats showed an obvious discrepancy of pathological and experimental data. Cat No. 1512 showed very severe damage, but had only one short respiratory arrest in 4 blows. However, a closer examination of the protocols showed the remark: "cat seems in worse shape than indicated by reflex abrogation." In this cat, there was an exceptionally large complicating central hemorrhage at C-1 which probably aggravated the symptoms.

The intensity of the fiber damage was paralleled by the extent of cell changes in the medulla oblongata. The frequency of chromatolytic cells increased with the severity of the concussion. The red nucleus, in particular, was involved only in severe injuries. Concussions below 30 seconds showed only a few chromatolytic cells in the reticular formation and the lateral vestibular nucleus so that an ample material of serial sections was required for diagnosis. Severe concussions showed chromatolytic cells in almost any section of the proper regions.

SECOND SERIES

The first series has shown that the symptomatology of concussion is related to a lesion at the ventral circumference of the first cervical segment and an axonal reaction in the nuclei which send their fibers through this region. This lesion is directly opposite to the odontoid process. It is likely, therefore, that the odontoid process plays a causal role in the mechanics of concussion. The following mechanical factors were considered to interact at the craniocervical junction: (a) stretch, (b) subluxation of the odontoid process, (c) herniation of the medulla into the foramen occipitale (pressure gradient), (d) rotation, and (e) flexion or extension. These factors are analyzed individually in the second part of the investigation.

Cervical Stretch

Because of the particular site of the lesion at C-1, both symptomatology and pathology of concussion could be reproduced by other means than by applying a blow to the head: cervical stretch has been found effective in this regard. Cervical stretch as a pathogenic factor in concussion was suggested first by Wright (ref. 43) and investigated later by Hollister, Jolley, and Horne (ref. 22). The neuropathology of cervical stretch has been described previously (ref. 10). The following paragraphs provide a synopsis of these findings.

Twenty-four cats were dropped about 30 inches with their heads fixed in a restraining collar. The sudden deceleration of the head stretched the neck abruptly. The symptoms accompanying this injury are indiscernible from a true concussion produced by a blow to the head (ref. 22). The neuropathology of these cats showed the same type of fiber lesion at C-1, and the same distribution of chromatolytic cells in the medulla oblongata as was characteristic for animals receiving a blow to the head (ref. 10). There was also a correlation between the duration of symptoms and the extent of histological damage. The only histological difference between animals receiving a blow to the head and those subjected to cervical stretch was the complete absence of any complicating cortical injuries in the latter. This could be expected, since the cortical damage was a direct result of the blow, not related to concussion.

Cervical stretch thus serves as a crucial experiment showing that all the typical signs of experimental concussion can be reproduced without applying a blow to the head. That cervical stretch actually exists in acceleration concussion was shown previously (page 4) on the basis of the different velocities of parts of the head.

Subluxation of the Odontoid Process

The spatial coincidence of the cervical fiber lesion with the level of the odontoid process could be interpreted: (a) since the odontoid process is the most prominent structure in the ventral wall of the craniocervical junction, any straining of the cord would show maximal damage at this point; (b) a subluxation of the odontoid process could contribute actively to the mechanics of damage. The following experiments were designed to determine the pathogenic importance of a subluxation of the odontoid process.

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The parietal region of 15 cats was trephined shortly after death and the brain and the upper segments of the spinal cord were removed. The cranial vault and the spinal channel then were filled as quickly and as completely as possible with acrylic resin or with Kerr dental impression medium. The trephine hole was closed and sufficient time was permitted to lapse to yield a permanent impression in the medium as controlled by previous tests and by concurrent samples. At the optional phase of hardening, 7 of these preparations received the same type of blow as was used to concuss cats. After complete hardening, the casts were removed from the body. Eight control preparations did not receive blows.

The deepest impressions of the odontoid process were found in cats receiving blows (figure 6). Systematic measurements of the depth of the impression, however, showed only a slight difference (not statistically significant) between the two groups. The dorsal circumference of the foramen occipitale sometimes left a deep impression indicating that the skull had been dislocated ventrally in relation to the cervical column.

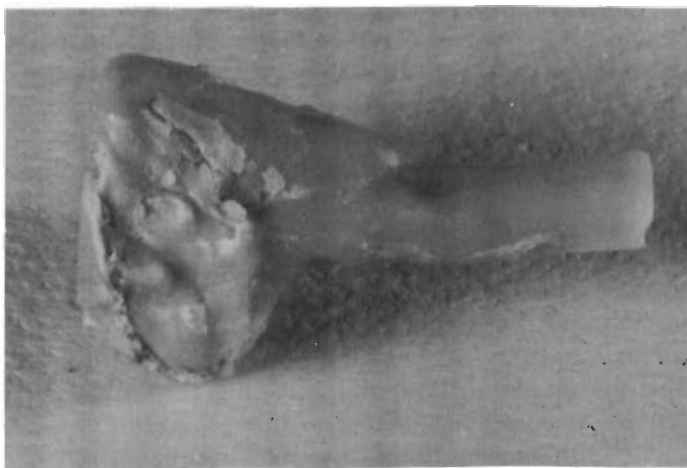


Figure 6. Polyacryl Cast of the Craniocervical Junction, Exhibiting the Deep Impression Left by the Odontoid Process after a Blow to the Head

Summarizing the findings, a subluxation of the odontoid process possibly contributes to aggravate the damage, but so far there is no evidence for a critical importance of this mechanism.

Herniation of the Medulla Oblongata into the Foramen Occipitale

Shearing forces at the foramen occipitale due to pressure gradients at this region were discussed by Gurdjian, Webster, and Lissner (ref. 18). Such a herniation of the medulla oblongata into the foramen occipitale could occur in the dropping experiment used to produce cervical stretch. The controversy of herniation versus stretch was investigated in cats dropped with supported body. In this experiment, the inertia of the brain and its tendency to herniate into the foramen was unchanged but cervical stretch was prevented. Cats could not be concussed under these conditions as already observed earlier by Wright (ref. 3). A device was also available which permitted stretching the craniocervical junction at various rates. First tentative experiments indicated that the inertia of the brain was irrelevant as compared to cervical stretch.

Gurdjian et al. (ref. 17) observed that a sudden pressure pulse within the fixed cranial vault causes concussion, whereby higher pressures require a shorter duration to become effective (ref. 18). Shearing forces in the brain stem, due to a pressure gradient between cranial vault and cervical channel, were considered responsible for concussion. In acceleration concussion, the impression of the skull by the blow allegedly would cause a similar pathogenic mechanism. This theory had to identify concussion with a severe bone deformation since a blow accelerating the head without bone impression would produce a negative pressure gradient opposite to the impact (refs. 14, 15, 17). Earlier studies by the author of this report showed much smaller pressure gradients in the basal cistern (ref. 7) than at the hemispheres (ref. 8).

Using a protective helmet in the present investigation raises skepticism as to the significance of bone impression. The comparison of the effects of supported and unsupported drop show that the hydromechanics of the cranial vault is irrelevant if compared with cervical displacement under the present experimental conditions. The histopathology of the compression concussion produced by Chason et al. (ref. 4) resembles that of acceleration concussion described by Windle, Groat, and Fox (ref. 40). If shearing forces were active, one should expect the distribution of damage to be related to the bony structures of the foramen magnum, as analyzed in cortical herniations by Holbourn (refs. 20, 21). It is difficult to conceive how herniation or shearing forces could produce a selected damage to the reticular formation and the lateral vestibular nucleus.

Rotation

Rotation of the brain within the cranial vault during head injuries was demonstrated by Sheldon et al. (refs. 30, 32). The significance of rotation for experimental concussion was tentatively investigated in this laboratory.* The heads of six cats were twisted by a device which permitted a twist of 180° in about 160 milliseconds with a terminal velocity of 3 degrees/millisecond. The rotation stopped within 1.5 msec. Rapid twisting of the necks of the cats did not produce concussion. Neuropathological investigation showed no damage in the brain or cervical cord. Although these experiments were not exhaustive, rotation was considered unlikely as a method for producing concussion.

Flexion and Extension of the Neck

Molds were made of the cervical channel of cats with their necks in extreme flexion or extension. Such molds demonstrated the critical importance of the tip of the odontoid process around which the maximal bending and straining of the cord occurs (figure 7).

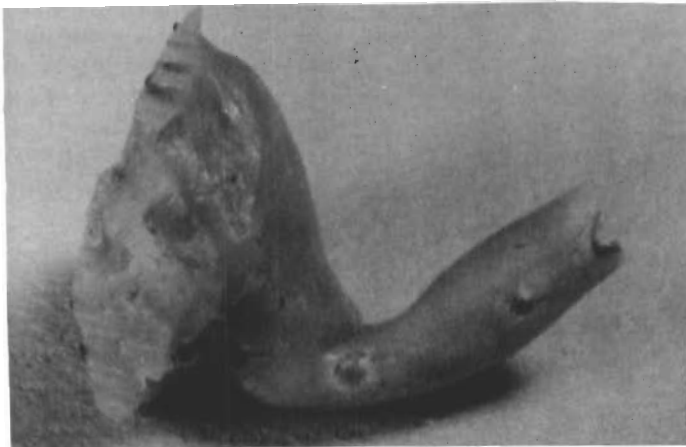
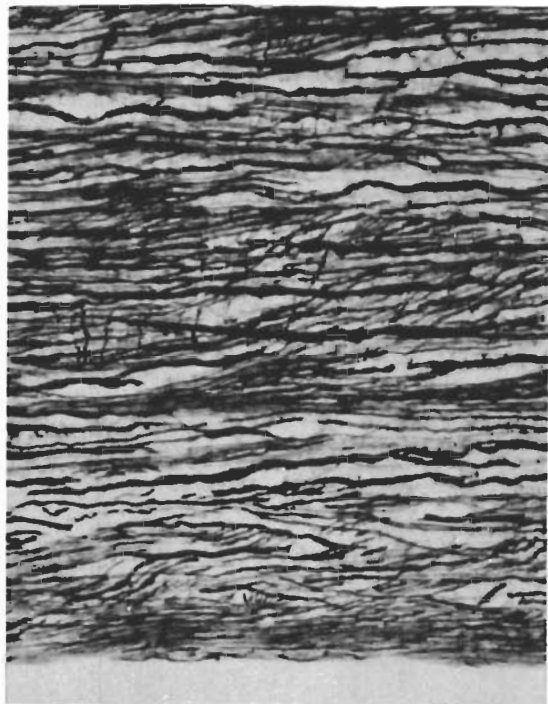


Figure 7. Polyacryl Cast of the Craniocervical Junction in Extreme Flexion of the Head

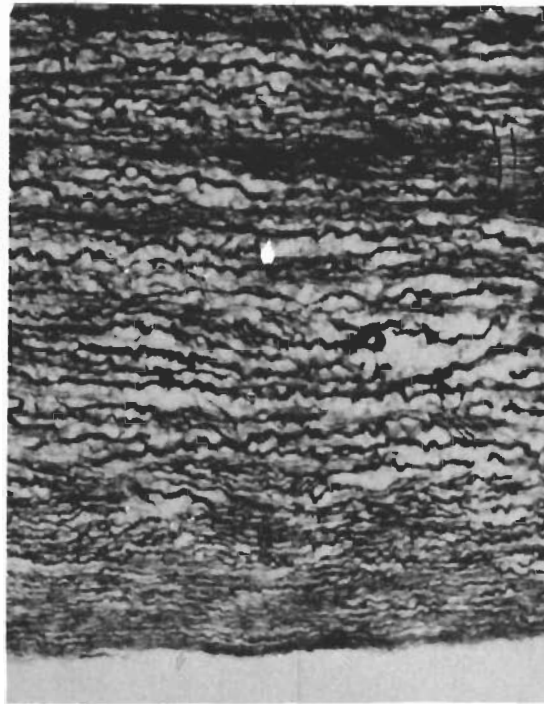
Note the sharp indentation of the tip of the odontoid process.

*Hessler, D.C., Unpublished Data

If the brain and cord were fixed by perfusion while the cat's head was held in extreme flexion a considerable longitudinal compression of fibers could be seen in the ventral circumference of the cord (figure 8). The effects of compression were particularly evident in the thick fibers. Such a compression or a rapid stretching could likely damage the fibers, if occurring during less than 10 milliseconds.



A. Represents Fixation of the Cord by Perfusion with the Head Held in Extreme Flexion



B. Shows the Same Region Fixed at a Normal Position of the Head

Figure 8. Longitudinal Section of the Ventral Fiber Tracts (stained with unreduced variant of Hortega's silver carbonate technique)

The analysis of the movements of the head reported in the first series suggests that flexion and stretch will interact under natural conditions, and both probably contribute to the damage. Histologically both result in a longitudinal compression or straining of fibers. One may question if it is feasible to distinguish stretch, or flexion and extension, as separate factors producing concussion.

DISCUSSION

If the blow accelerates the head, energy is absorbed at the craniocervical junction. An abrupt displacement (stretch, bending) at this region produces a lesion at the ventral circumference of C-1 opposite the odontoid process which is the most prominent bone structure in this region. This lesion results in axonal reaction in the nuclei of the brain stem which send their fibers through the medioventral part of the cervical cord.

Brodal and Torvik (ref. 3) showed that the majority of the descending fibers of the reticular formation originate in the nucleus reticularis gigantocellularis and descent in the medioventral funiculus. Fewer fibers originate in the caudal nucleus reticularis medullae oblongatae and the cranial nucleus reticularis pontis. Brodal and Torvik's diagram of the distribution of chromatolysis in the reticular formation after lesion of the cervical cord is an accurate representation of the findings in concussed cats, except that the lateral, parvocellular part is not damaged so much following concussion. The greater vulnerability of the thicker fibers probably accounts for this difference, but thinner fibers are damaged as well in severe or multiple blows so that the difference is only of quantitative nature.

The fibers from the lateral vestibular nucleus also follow the ventral tracts while the rubrospinal fibers descent more laterally and are involved only in larger lesions, explaining the lesser affection of the red nucleus.

Windle et al. attributed the chromatolysis to a direct insult to the cells. However, there is no satisfying explanation for the limitation of damage to a few nuclei while others remain protected. The central type of chromatolysis and its time course, showing maximal alterations after about 8 days, are compatible with axonal reaction. Early changes of the chromophil substance were emphasized by Windle et al., but do not necessarily reject axonal reaction. Also, transportation of oxidative enzymes from the damaged nuclei along their descending fibers to the side of the fiber lesion has been observed following cervical stretch (ref. 9), so that the functional correlation of all the changes described above was demonstrated histochemically. These profound changes in the chemical composition of cells and tracts during axonal reaction might be related to the detection of nucleic acid compounds (ref. 34) and acetylcholine (ref. 1) in the cerebrospinal fluid following concussion. There is no evidence to support the view that disturbances of circulation are responsible for the changes described above.

Krems, Schoepfle, and Erlanger (ref. 25) showed that a single blow to a nerve by means of an air gun causes the nerve to discharge once or for a short period, followed by paralysis. Such a response from the reticulo- and vestibulo-spinal fibers would be compatible with the initial jerk and stretch response, followed by flaccidity which was observed in experimental concussion (page 2). The sudden discharge upon the blow was recorded by Walker, Kollros, and Case (ref. 35) but was interpreted as a sudden breakdown of cell membranes. Spiegel et al. (ref. 33) recorded the abolition of electrical activity directly in the red nucleus and other nuclei.

As to the abrogation of corneal reflex and respiration two acting factors are suggested: (a) More than half of the giant cells of the nucleus reticularis gigantocellularis project to the spinal cord and the rest have ascending fibers. Many nerve cells, however, seem to have dichotomic axons branching in both directions (ref. 3). The discharge of the fibers upon a sudden interruption of the descending branches could upset the homeostasis of the reticular system causing loss of consciousness, respiration, and corneal reflex. A similar mechanism of spinal inhibition via vestibular or brachial plexus stimulation has been shown recently by Gernandt and Gilman (ref. 11) in support of earlier clinical observations (ref. 26). Electroencephalographic changes following concussion (refs. 6, 37, 38) may be induced in the same way, since Hollister, Jolley, and Horne (ref. 22) could produce symmetrical slow wave activity by cervical stretch. (b) Another factor to be considered is that the functional impairment would affect a much larger group of fibers than the morphological destruction and degeneration. Although the neuropathology mainly shows thick fibers undergoing degeneration, the function of thin fibers as well as a much larger area will certainly be temporarily impaired.

Both factors are compatible with the fact that concussion can be obtained in the decerebrate preparation (ref. 5).

The lesion at C-1 could be interpreted as a complicating cord injury or whip-lash injury rather than cerebral concussion. However, this lesion is evidently responsible for the cellular changes in the medulla oblongata attributed to concussion. There is also a definite relationship between histological findings and the generally accepted symptomatology of concussion. The threshold forces required to produce this damage are in the same order as those reported by other authors for concussion. Since histological damage and symptoms changed at the same rate, no discrete symptomatology was left to distinguish a "truly cerebral" concussion from the syndrome described herein. There is, thus, no reason to believe that this article describes a cervical syndrome different in any way from the "concussion" of the majority of physiological experimenters. Several of the individual findings, such as the hemorrhages in the cord at C-1, have been observed before by other investigators (refs. 5, 23, 28).

It is proposed to reserve the term, "experimental acceleration concussion," for the specific pathogenic syndrome described herein. This syndrome should be distinguished from other types of head injuries. The importance of such different factors as rotation of the brain (refs. 30, 32), shearing forces (refs. 20, 21), cavitation (refs. 14, 15, 36), breakdown of cell membranes (ref. 35), and changes of colloid properties (ref. 19) for the genesis of brain injuries is far from being settled. However, these factors should be distinguished from the clear-cut syndrome of "acceleration concussion."

Another question concerns the relationship of experimental acceleration concussion and clinical "commotio cerebri." This question can be answered only by human neuropathology and it is difficult to answer, indeed, since commotio cerebri is not a fatal condition. Routine pathological material is of little value, since the customary dissection of brain and spinal cord invariably destroys the critical region. Groat, Windle, and Magoun (ref. 13) have shown that "experimental acceleration concussion" is readily produced in monkeys. There is little doubt, therefore, that this syndrome will be found as well in man.

The mechanics of the whip-lash injuries seem similar to those of experimental acceleration concussion. However, a direct application of force to the head is usually necessary to absorb sufficient energy at the craniocervical junction to damage C-1. Whip-lash injuries do not always limit the conveyance of energy to the head; the absorption of energy is not primarily limited to the craniocervical junction but is spread over the entire cervical and thoracic vertebral column. Whip-lash injuries, thus, may be complicated by concussion if of sufficient strength to cause cervical displacement.

SUMMARY

A blow accelerating the head produces an abrupt displacement at the craniocervical junction. Such a displacement results in a characteristic fiber lesion at the ventral circumference of the first segment of the cervical spinal cord opposite the odontoid process. Thick fibers are more severely affected than thin fibers. The distal parts of the fibers undergo Wallerian degeneration. Axonal reaction is found in the nuclei which send their descending fibers through the damaged region: the reticular formation of the medulla oblongata, particularly its nucleus gigantocellularis, the lateral vestibular nucleus, to a smaller extent the red nucleus, and, inconsistently, other nuclei. This

pathology is consistently found in cats which received a blow to the head. It shows a definite relationship to the severity of symptoms so that a reasonable estimate of the duration of reflex abrogation can be made without knowledge of the experimental data. The symptomatology and the mechanical forces recorded are identical to those generally accepted for the definition of concussion.

Because of this pathogenic mechanism, both the typical symptomatology and the pathology of acceleration concussion can be reproduced by other means than by applying a blow to the head: for example, cervical stretch. An experimental analysis of the various mechanical factors involved reveals that stretch and flexion of the craniocervical junction are most important for the mechanics of concussion. Dislocation of the odontoid process, rotation, and herniation of the medulla into the foramen occipitale are not of critical significance.

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