SPECIFIC CORD DAMAGE AT THE ATLAS LEVEL AS A PATHOGENIC MECHANISM IN CEREBRAL CONCUSSION*

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Significant changes in the neurons of some of the brain stem nuclei were found following experimentally induced brain concussion (Windle, Groat, and Fox, (5), Windle, Groat, and Magoun, (6)). A chromatolysis was found in the neurons of the reticular formation of the medulla oblongata, in the lateral vestibular nucleus (Deiter's nucleus), the red nucleus, the nucleus of the spinal trigeminal tract, and certain other nuclei. The motor nuclei of the cranial nerves remained unchanged. The number of damaged cells in the affected nuclei was in relation to the severity of the trauma. A reduction of the cell population of the affected nuclei was found in longer follow-ups (7, 2).

The present report describes the neuropathology of cats in which an abrupt stretch of the cervical vertebral column was produced by dropping the anaesthetized cats with the heads fixed in a restraining collar (Hollister, Jolly, and Horne (3)). This experimental technique resulted in the same type of cell degeneration in the brain stem as described by Windle et al. in animals which had received a blow to the skull, or true head injury. In addition and in contradiction to prior studies, a specific fiber damage was resolved confined to the first segments of the spinal cord. The thick fibers were specifically affected. The damage was localized and required a special investigation of the cervical cord; it was apparently produced by the intimate cord-bone relationship at the atlas level. Fiber damage and chromatolysis in the axons were related phenomena. In controls, exactly the same type of injury was found also in cats which had received true head injuries by a blow on the head.

MATERIAL AND METHODS

The brains and spinal cords of 32 cats were studied, 24 of which were used for the drop experiment. The injury caused by the dropping procedure induced temporary loss of respiration and corneal reflex, indiscernible from the results of an experimental concussion by head injury. Such an experimentally induced impairment of brain stem reflexes is termed hereafter “concussion”; the definition has to be emphasized, since the relationship of the experimental syndrome to the clinical picture of concussion still has to be defined. A description of the experimental arrangement and findings is reported separately by Hollister, Jolly and Horne (3).

It was the author’s intention to determine if a relationship existed between neurohistopathological findings and duration of “concussion”. Therefore, it was decided to group the cats according to the total length of time in which they were “concussed”: those “concussed” more than 1 minute, less than 1 minute, and those subjected to a similar experimental run, but not “concussed”. Since it was not possible to predict the length of time

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In this article are reported the neuropathological findings of an experimental investigation which is the subject of the joint WADC Technical Report 58-193, with Hollister, Jolly, and Horne.
of a "concussion" (loss of corneal reflex), it was necessary to "concuss" some animals 1 to 3 times, and the total concussion time of over 1 minute was attained as a result of several "concussions" within a few days. Seven cats were "concussed" 65 to 226 seconds, 10 cats were "concussed" 5 to 55 seconds, and 7 cats were not "concussed". Of this latter group, 5 received a muscle tetanizing current in the cervical region. It was assumed that the current may protect the cat from "concussion" as described by Hollister, Jolly and Horne.

The maximal appearance of chromatolytic neurons is known to occur on the sixth to eighth day following concussion. For this reason 6 cats from each of the above groups were sacrificed within a period from the sixth to fourteenth day after the experimental drop. The remainder of the cats was sacrificed 23 to 52 days after the drop. None of the animals used in these groups exhibited gross findings such as bleedings, softenings, or edema. To avoid the possibility of a misleading interpretation, only severe alterations of the neurons, as evidenced by heavy or complete chromatolysis, were used (figs. 2-4). Since the main emphasis was placed on an evaluation of the quantitative differences between the three groups, only 2 normal control cats were used.

The fixation of the brain and cervical spinal cord was performed with 10% formalin or isotonic saline-formalin respectively, supravitally perfused from the aorta under deep pentothal anaesthesia. The brain and cervical spinal cord were removed in toto, stored in 10% formalin, and imbedded in paraffin. The entire medulla oblongata and midbrain, including parts of the cerebellum were cut serially and alternate ten and twenty micra thick sections were taken at 100 micra intervals. Random samples were also taken from the cerebral cortex, the basal ganglia, and the diencephalon as well as random frozen sections from the brain stem. The junction of the medulla oblongata and spinal cord and the cervical spinal cord was cut transversally and longitudinally; in a longitudinal cutting the spinal cord was halved median-saggitally, using one half for frozen sections and the other one for paraffin sections. By alternatively longitudinal and transverse sections, the complete area from the fourth ventricle to the lower cervical segments could be investigated. Staining was performed by Einarson's chromalum-gallocyanine, hematoxylin-eosin, sudan black for myelin sheaths, oil red O, sudan orange, and Landau's silver technic for neurofibrils.

An additional group of 5 cats received blows to their heads from a pneumatic hammer. These brains were treated in a manner similar to that described above, but not all of the brains were fixed by perfusion. One of these 5 cats showed a small cerebral softening and another one a subarachnoidal bleeding. Another group of 3 cats which were killed by the dropping experiment was investigated. Two of the cats were killed by the first drop and the third one by the last drop of a series of 6 subsequent drops.

Ten cats (6 dropped cats and 4 cats which received blows) with traumatic subarachnoidal bleedings were also studied. In these cats, which had been used for other experiments, only the distribution of the bleeding was studied and mapped by drawing. A superposition of the individual drawings established a schema of the typical localization of bleedings.

Studies on the topographical relationship of the spinal cord and the vertebrae were performed by an anatomical preparation of the cat's head and neck in which the left half of the cranial bone and parts of the vertebral arcs were removed, but all the joints were carefully left intact. A thin metal foil was attached to the ventral surface of the brain and spinal cord. X-ray pictures of this preparation revealed bone-cord relationships in different positions. The mandible was removed to obtain better X-ray pictures.

RESULTS

A. Cell Changes in the Brain: The cytological findings described hereafter were the only indication of brain damage observed by the author. No indication was found of gross damage, or microscopical bleedings, edema etc. A chromatolysis in large neurons of the reticular formation of medulla oblongata and pons and in the lateral vestibular nucleus (Deiter's nucleus) was present in most of the
Fig. 1. Normal neuron of the reticular formation, chromalum-gallocyanin, 200 X.

Fig. 2. Complete chromatolysis in a neuron of the reticular formation, chromalum-gallocyanin, 200 X.

Fig. 3. Chromatolytic neuron with eccentric nucleus and chromophilic neuron in immediate vicinity, Deiter's nucleus, 200 X.

Fig. 4. Eccentric nucleus in a chromatolytic neuron of the reticular formation, chromalum-gallocyanin, 200 X.

Fig. 5. Incomplete chromatolysis, Deiter's nucleus, chromalum-gallocyanin, 200 X.

Fig. 6. Chromophilic neuron in the reticular formation, chromalum-gallocyanin, 200 X.

Fig. 7. Normal neuron and two chromophilic neurons in the trigeminal motor nucleus, chromalum-gallocyanin, 200 X.

Fig. 8. Interrupted axon with club-like swelling, hematoxylin-eosin stain, 200 X.

Fig. 9. Cystic vacuolation of a swollen and interrupted axon, hematoxylin-eosin stain, 200 X.
animals. Since the quantity of damaged cells was about proportional to the abrogation of the corneal reflex, the findings for the 3 groups of animals are summarized. As an indication of the quantity of damaged cells, the following terms were defined: few, if a small number of cells was found in a series of about 20 sections of the appropriate region; moderate, if an average of one or two chromatolytic cells was found in a single section; frequent, if many damaged cells were found in a single section.

Animals whose clinical symptoms ranged below one minute, showed a heavy or complete chromatolysis in a moderate number of the large neurons of the reticular formation and Deiter's nucleus 8 days after the experiment. In the reticular formation, the nucleus reticularis gigantocellularis* exhibited the greatest number of chromatolytic neurons; fewer of them were found in the nucleus reticularis pontis, and they were rare in the caudally situated nucleus reticularis medullae oblongatae†, even in its large neurons. The small neurons of the laterally situated parvocellular part of the reticular formation were not affected. In Deiter's nucleus, the subnuclei α† and γ† were affected principally, and less damage was found in the subnuclei β† and δ†. In most of the animals, the damage in the reticular formation was somewhat more pronounced than that in Deiter's nucleus.

The cell bodies and the dendrites of the chromatolytic neurons were enlarged, containing in the earlier stages (about up to the sixth day) a dust-like disintegrated chromatin (fig. 5); later a central or complete chromatolysis was found (figs. 2 to 4). The central type of chromatolysis was frequent in Deiter's nucleus (fig. 3). No dislocation of the cell nucleus was usually found in the reticular formation (fig. 2), but excentric nuclei were frequent in Deiter's nucleus (figs. 3, 5).

At the stage of a complete chromatolysis (ghost cell), the nucleus shrank and disintegrated. The nucleolus was enlarged during the period of dust-like disintegration of the chromatin and exhibited heavy vacuolation; in ghost cells the nucleolus appeared hyperchromic and shrunken.

Contrary to the pronounced chromatolysis in the reticular formation and Deiter's nucleus, no chromatolysis was found in other nuclei of the brain stem, particularly in the motor nuclei of the cranial nerves (fig. 7). The protection of the large neurons of these nuclei was in striking contrast to the findings in the closely adjacent reticular formation.

Chromophilic neurons, on the other hand, were either moderate or frequent not only in the affected nuclei (figs. 3, 5, 6), but also in many other nuclei of the brain stem (fig. 7). The distribution of the chromophilic neurons was irregular and somewhat different in all or nearly all of the animals. Although the chromophilic neurons were irregularly distributed and were not confined to specific nuclei, their frequent appearance in the experimental material made them a typical feature of the findings. Artificial shrinkage simulating chromophilic neurons was excluded by the conditions of fixation.

In animals with post-experimental reflex impairment which ranged above 1

† The terminology used identifies the nuclei with those in the atlas of the rabbit's rhombencephalon by Meessen and Olszewski (4).
minute, the chromatolytic neurons were frequent, but their distribution was not changed. In the reticular formation, the chromatolytic neurons were found distributed further into the nucleus reticularis pontis\dagger and medullae oblongatae\dagger and even neurons of smaller size were involved. The damage to Deiter’s nucleus was more pronounced. Some chromatolytic neurons also appeared in other nuclei, such as the nucleus lateralis oralis\dagger and lateralis caudalis\dagger medullae oblongatae, nucleus prepositus hypoglossi\dagger, subnucleus Deiter’s δ\dagger, dentate nucleus, and the nucleus of the descending trigeminal root. These findings agree with the description by Windle, Groat, and Fox (5) with exception of the cochlear nucleus in which no degeneration was found in the present investigation. The motor nuclei of the cranial nerves showed no chromatolysis. The most pronounced difference between the moderately and the heavily damaged group was the constant involvement of the red nucleus in the latter. A central chromatolysis, eccentric nuclei, vacuolated and enlarged nucleoli, swelling of the cell body and its dendrites, and finally, development of the typical ghost cells were found in the neurons of the magnocellular portion of the red nucleus; the parvocellular portion was protected. The cerebral cortex was also slightly involved in the most heavily damaged animals; an incomplete chromatolysis was seen in some of the large pyramidal cells associated with a pronounced dislocation of the nucleus towards the apical dendrite. Chromophilic neurons were found in about every nucleus, but without a fixed distribution.

The animals of the third group showed no reflex impairment following the experiment; no evident damage was found in 3 of these 7 cats. In the other 4 cats a few chromatolytic neurons were found in the reticular formation and Deiter’s nucleus, but these neurons were definitely less frequent than in the other groups. The red nucleus was not involved. Chromophilic neurons, on the other hand, were exceedingly frequent in some of the cats of this group, showing the highest incidence of chromophilic neurons of all the material.

With minor exceptions, the findings in the neurons are identical with those reported by Windle, Groat and Fox (5), Windle and Groat (7), and Groat and Simmonds (2).

B. Findings in the Cervical Spinal Cord: An insignificant swelling of nerve fibers in the lower medulla and upper cervical cord was reported by Windle, Groat, and Fox. The damage reported was confined to the deeper part of the ventral and lateral funiculi; the peripheral zone and the dorsal funiculi were spared.

The damage to the fibers was significant in the author’s material and definitely more pronounced than that described by Windle, Groat, and Fox. The different results may be explained by the complete serial study of the cervical cord in the present investigation. A pronounced damage was revealed at levels of C 1 and increasing towards C 2. Thus, sections from the area of about C 1–2 may show different intensity of damage according to the exact level from which they were taken. Removal of the brain and cord in toto, and longitudinal sectioning of the critical region with interspaced transverse sectioning are recommended procedures for investigating the damage.
All of the cats which showed chromatolysis in the neurons also exhibited damage to the fibers. This damage was confined to the thick fibers in the medioventral portions of the cervical spinal cord. The intensity of damage was proportional to the diameter of the fibers; the medium-sized fibers showed less damage, and the thin fibers were not damaged. The maximal onset of the damage was found in the first two segments of the spinal cord in contrast to the sparse findings above the first segment.

In longitudinal paraffin sections, the axons of the thick fibers exhibited a diffuse swelling and dilatation of the space around the fiber. The axon diameter increased caudally, ending in a club-shaped swelling (figs. 8, 9) where the axon was interrupted. The end-club was surrounded by a heavily dilated periaxonal space. Large cysts may appear within the club, and nuclei of migrating cells could be found attached to the club. Such club-shaped axon interruptions appeared most frequent at the level of the first, second, and third segment.

The changes of the myelin sheath started with an irregular staining of the myelin by sudan black. This led to a varicose swelling of the sheath, and, finally, to a globular breakdown marking the course of the fiber by deposition of irregularly-shaped myelin deposits (figs. 12, 13). These myelin fragments were then incorporated by macrophages (figs. 10, 12). Small myelin particles could be found attached to the surface of swollen axons, but otherwise the heavily dilated periaxonal space appeared empty.

Thin fibers exhibited no recognizable damage, even in the neighborhood of disintegrated thick fibers or immediately below the surface of the spinal cord. Medium thick fibers exhibited a moderate irregularity only of structure and stainability of the myelin, but did not show a breakdown of the sheath (fig. 12).

In transverse sections, the irregular staining of the sheaths of the thick fibers could be recognized and in the progressed stages large, empty tissue lacunae which sometimes contained macrophages were found (fig. 10). The loss of thick fibers was most pronounced at the medioventral surface of the cervical spinal cord and extended into the ventral median fissure. Laterally, the damaged fibers gradually disappeared. The lateral tracts showed fewer damaged fibers (fig. 11), and these only in severe injuries. Damaged fibers in the dorsal fasciculi were extremely rare. The distribution of the damage was symmetrical, and decreased toward the central portion of the cord. In only one cat was the damage further extended laterally on one side than on the other. The damage was most pronounced in the peripheral thick fibers; the peripheral thin fibers showed no damage as was the case with all thin fibers. The lack of damage to peripheral thin fibers may explain Windle, Groat, and Fox’s reference to a deep damage.

The degree of damage, expressed by the number of damaged fibers, and the size of the damaged area, was proportional to the amount of chromatolysis in the neurons and to the clinical severity of the injuries.

The gray matter of the cervical spinal cord exhibited no typical damage, although chromophilic neurons were seen frequently and chromatolytic neurons were found occasionally.

The damage to the thick fibers gradually reached maximal severity in the
Fig. 10. Severe damage in the ventral fiber tracts; irregular staining, swelling, and disappearance of sheaths, myelin incorporated by macrophages. Transverse section, C 3, sudan black, 200 X.

Fig. 11. Lateral fiber tracts in the same section as in Figure 10: no or very slight damage, 200 X.

Fig. 12. Ventral fiber tracts below the surface of the spinal cord. Disintegration of the myelin sheaths of the thick fibers, irregular structure of the myelin of medium fibers and complete protection of the thin fibers. Myelin particles incorporated by macrophages. Longitudinal section, C 3, sudan black, 200 X.
first 2 segments of the spinal cord, and only a small amount of damage was noted in the medulla. For this reason, damaged fibers may be difficult to find in sections from the lower medulla, even though they are abundant in the cervical spinal cord. This condition is attributed more to the off-grading at the higher levels than to the diffuse course of the fibers in the medulla. This will be demonstrated later by the distribution of fat droplets. Since there was no gross tissue lesion present, the change in the intensity of fiber damage had to be used to localize the damaging mechanism.

The change in the intensity of the fiber damage was well demonstrated by the distribution of fat droplets which outlined the damaged area. This phenomenon was a late sequence of the injury, since it was found exclusively in 6 animals which had been used in the dropping experiments 36 to 126 days prior to the sacrifice. At the level of C1 and C2, fat droplets were found lining the medioventral surface of the cord and were attached to damaged fibers (fig. 14). The amount of fat deposits increased markedly within the first segments, and grew less severe at lower levels of the cervical cord. In both transverse and longitudinal sections, the damaged area was clearly outlined by fat droplets. Almost no fat droplets were found in these animals at the level of the medulla oblongata and at the junction of the medulla and the cervical spinal cord. At no time were these fat droplets found in experimental animals sacrificed prior to 26 days.

C. Findings in Cats Killed by the Dropping Experiment: To obtain additional information on the mechanism which produced the fiber damage, the brains of 3 cats which died following the dropping experiment were studied. One of these cats was killed by the last drop in a series of 6 drops. The evidence of damage in this cat was of the same nature, but more pronounced than in the other cats. Two of the 3 cats showed subarachnoidal bleedings.

No chromatolytic cells were seen in the brain stem of these cats, but a slight and indistinct disarrangement of the chromatoin pattern associated with vacuolation of the nucleolus was found. Chromophilic neurons were extremely rare.

In the upper cervical cord, damage to the sheaths was found in the same typical localization as described above. Such myelin sheaths stained irregularly, showing darker and lighter areas, or fragmentation. The surface of the sheaths was slightly varicose, but the space around the sheaths was not dilated. No complete breakdown of the sheaths was seen. Medium and thin fibers of the ventral tracts as well as the dorsal and lateral tracts showed no damage, or only a slight vacuolization of the sheaths.

Since these 3 animals died following the experiment, the damage to the sheaths was due to a direct mechanical insult which affected particularly the thick myelin sheaths.

Both the early appearance of the features of the damage and the increasing intensity of the damage in the first segments of the spinal cord preclude the possibility of a secondary fiber degeneration. Thus, the chromatolytic pattern in the medulla obviously results from axon damage in the spinal cord. The medioventral location of this damage explains the specific involvement of some
Fig. 13. Disappearance of thick fibers, protection of thin fibers, and some macrophages at the ventral surface of the spinal cord; longitudinal section, sudan black, 200 X.

Fig. 14. Fat droplets outlining the damaged area, equivalent section to Fig. 13. Oil red O, 200 X.

Fig. 15. Superposition of the cord in relation to the odontoid process reveals flexion and possible straining of the cord around the process.

Fig. 16. Metal foil attached to the ventral surface of brain and spinal cord reveals maximal flexion occurring in the region in which the maximal damage is found.
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nuclei sending their fibers through this region. The exclusive damage to the thick myelin sheaths explains why some other nuclei, such as the nucleus fastigii, sending thinner fibers through the same region, remain protected. The type of chromatolysis in Deiter’s nucleus and the red nucleus is similar to that following axon interruption. The different feature of the chromatolysis found in the neurons of the reticular formation seems a specific characteristic of this nucleus rather than a specific type of damage. The time lapse between the injury and the completion of the chromatolysis is adequate for the appearance of a retrograde degeneration.

D. Relationship of Drop Experiment and Head Injury: The findings in the nuclei of the brain stem were similar to those reported by Windle, Groat, and Magoun. These investigators used a pendulum for applying the blow to the head versus Hollister’s arrangement of vertical drop with fixed head. It seems likely that the dropping procedure would result in a greater cord damage than the pendulum type of blow, but a specific association between vertical drop and spinal cord damage had to be proven. It is possible that both experimental methods might result in a damage to the medioventral part of the spinal cord, although this damage has not been described previously.

To answer this question, a group of 5 cats was studied which received blows on the head by a pneumatic hammer. Three animals exhibited no gross findings, and the fourth animal showed a subarachnoidal bleeding. The fifth animal, displayed a small cerebral softening.

The findings in this series were not discernible from those obtained in the drop series. Not only the distribution of the chromatolytic neurons, and the irregular appearance of the chromophilic neurons, but also the cervical spinal cord damage, and the relationship between the symptoms and the amount of histological damage were exactly the same in both series. Thus, by no means was a difference found between the dropping procedure and true head injuries; the damage of the cervical spinal cord is not specifically related to the dropping procedure.

E. Investigations of the Mechanism of the Spinal Cord Damage: The confinement of most of the histological damage to the medioventral surface of the cervical spinal cord indicates a specific mechanism acting in both head injuries and the dropping experiment. This specific mechanism can be revealed by X-ray pictures of anatomical preparations in which the ventral surface of the brain and spinal cord was marked by an attached metal foil (figs. 15, 16).

If X-ray pictures of different positions of the head are superimposed, it becomes evident that the atlanto-occipital junction is the only region where a sharp flection of central nervous tissue is anatomically possible. The first vertebra and the odontoid process form the point of intersection around which the flection of the spinal cord occurs. A forced, rapid position change of the head strains the spinal cord around this point because of the intimate cord-bone relationship. This point of intersection is directly opposite to those portions of the ventral spinal cord where heavy fiber damage and fat deposition have been noted. The present material does not allow any conclusion as to how much a
subluxation of the odontoid process during a forced position change might contribute to aggravate the effect, but it is felt that such subluxation may be a highly important factor.

A subluxation of the odontoid process might produce a heavier damage than that described here, since this is the critical region for death by hanging which is similar to the dropping procedure. This supposition agrees with the distribution of bleedings found in 10 other cats (6 from dropping experiments, 4 with blows). A summarized mapping of these bleedings reveals the highest incidence (9 out of 10 cats) at the critical region, at the level of the odontoid process. (fig. 17).

DISCUSSION

In the present investigation, the neuropathology of an experimental procedure is reported which is accomplished by the dropping of cats with the heads
fixed in a holder (Hollister, Jolly, and Horne). Although the result of the procedure cannot be called a head injury, the dropping procedure produced a temporary loss of the corneal reflex and the respiration indiscernible from an experimental concussion. The dropping also produced the chromatolysis in certain nuclei of the brain stem which was described as the morphological substrate of concussion. This chromatolysis appeared in the large neurons of the reticular formation and Deiter's nucleus after about 8 days. In more severe insults, these changes also appeared in the red nucleus and other nuclei. A quantitative relationship was found between chromatolytic neurons and clinical symptoms.

In contrast to previous reports, considerable damage was found in the thick fibers at the ventral surface of the upper segments of the cervical spinal cord. Early changes in the structure of the myelin were seen in cats killed by the dropping method. The later stages exhibited a breakdown of the sheath, axon swelling and interruption, and later deposits of fat droplets. These changes decreased considerably towards the medulla oblongata and showed maximal damage caudally from the first spinal segment. This typical distribution implies a confined damage at the level of the atlas. The intensity of this fiber damage was proportional to the amount of chromatolysis in neurons of the brain stem.

The cervical fiber damage was not specifically related to the dropping experiment, since identical findings were also revealed in cats struck on the head with a pneumatic hammer. Therefore, the chromatolysis of the neurons in both the dropping experiment and head injury, or true concussion, is most probably a retrograde degeneration resulting from the fiber damage in the spinal cord. This explanation is more reasonable than a specific vulnerability of single nuclei by the insult. The central type of chromatolysis which was frequently found in the present material in Deiter’s nucleus and the red nucleus is typical for axon interruption. The atypical feature of the reticular formation (rarely excentric nuclei or a clear central type of chromatolysis) seem specific characteristics of these neurons. The time lapse towards the maximal chromatolysis (about 8 days) can be considered adequate for a retrograde degeneration. The appearance of the first changes in the chromatin pattern after 24 hours (Windle, Groat, and Fox) does not reject a retrograde chromatolysis, since it is known that such alterations occur as early as 24 hours following experimental axon interruption. The insignificant fiber damage found in previous investigations is probably due to the specific distribution of degenerating fibers; the increase of damage below the first segment can be demonstrated only if this area is specifically investigated. In a routine removal of the brain, the critical area is frequently cut or damaged. The medioventral confinement of the damage is responsible for the typical distribution of chromatolytic neurons which send their fibers through the damaged area. Since only thick fibers were damaged, other nuclei such as the nucleus fastigii were protected, although they sent thin fibers through the same area. The exclusive effect on thick fibers is explained by a higher vulnerability of the thick myelin sheaths as demonstrated in the cats killed by dropping. X-ray investigations revealed flexion or strain of the cervical cord around the odontoid process. This flexion acts in forced changes of position of the head and may operate as a damaging mechanism. It is suggested that a
subluxation of the odontoid process might enhance this mechanism. No similar mechanism can act at the dorsal surface of the spinal cord.

The problem arises as to whether the present findings, or also those previously reported in the literature following experimental concussion, are in fact the anatomical substrate of concussion. This is suggested by the quantitative relationship of symptoms and morphological damage. If the author's findings and conclusions are correct, it follows that the substrate of concussion, the acute symptoms of loss of the corneal reflex and respiration, seem to be due to a shock to the thin, undamaged fibers which recover within a short time. The more lasting or permanent damage to the disintegrating thick fibers could explain the post-concussion symptoms. This hypothesis is in agreement with the function of the involved fibers, since the loss of corneal reflex or respiration is not evidently related to the rubrospinal, reticulospinal, or vestibulospinal fibers, neither is it directly related to the damage of the neurons of Deiter's nucleus and the red nucleus from which the descending fiber tracts originate. The described mechanism would explain why a heavy brain laceration can occur without concussion if no position change of the head is involved. It also would agree with the findings of Denny-Brown and Russel (1) who obtained concussion even in the decerebrate animal. The term "brain" concussion would definitely be wrong if the above assumptions are correct.

The assumptions do not prove the association of the described substratum with clinical concussion. However, the question probably should be formulated inversely: since the described type of damage indicates a clear-cut pathogenic mechanism at the atlas level which evidently works in head injuries, it should be asked how frequently this syndrome is, in fact, responsible for the rather indirectly defined clinical picture of concussion. It seems more than reasonable to assume a specific cord injury at the atlas level behind at least many instances of so called "brain" concussion.

SUMMARY

The central nervous system of cats was investigated in which an abrupt stretch of the cervical vertebral column had been performed. The same type of brain stem damage was found as has been described following experimental concussion: a chromatolysis appeared in the large neurons of the reticular formation and Deiter's nucleus, in severe injuries also in the red nucleus and some other nuclei. The motor nuclei of the cranial nerves were not involved.

A significant fiber damage was found in serial studies of the cervical spinal cord. Thick fibers in the medioventral portions of the cervical spinal cord were affected exclusively. This damage was maximal at the atlas level but sparse above this level.

The locally defined fiber damage corresponds to the distribution of chromatolytic neurons sending their fibers through the damaged area; both findings were quantitatively related. Changes in the fiber structure were found also in cats killed by the experiment.

X-ray investigations revealed an intimate spinal cord-bone relationship at
the level of the damaged area of the spinal cord. In particular, a straining of the spinal cord around the odontoid process occurred in forced position changes of the head and could be enhanced by a subluxation of the odontoid process.

Identical neurohistopathology was found in dropped cats subjected to an abrupt stretch of the cervical spinal cord and in control cats receiving a blow to the head. The chromatolysis in the neurons in both the stretching experiment and in cats receiving a blow to the head, is most probably a retrograde degeneration resulting from the fiber damage in the spinal cord. Therefore, a specific mechanism of cord injury at the atlas level seems responsible for many instances of so called "brain" concussion.

REFERENCES