Acta neuropath. (Berl.) 12, 200—204 (1969)

Pathomorphology of Experimental Head Injury Due To Rotational Acceleration*

FRIEDRICH UNTERHARNSCHEIDT

Division of Neuropathology and Experimental Neurosurgery
University of Texas Medical Branch, Galveston, Texas, U.S.A.

LAWRENCE S. HIGGINS

Life Sciences Division, Technology Incorporated, San Antonio, Texas, U.S.A.

Received September 10, 1968

Summary. We have shown that pure non-deforming rotational acceleration to the head is capable of producing lesions not only in the brain but through the entire length of the spinal cord, extending to the cauda equina.

In clinical circumstances in which humans are subjected to severe rotational acceleration of the head, the spinal cord, as well as the brain should be examined pathologically.

Zusammenfassung. In experimentellen Untersuchungen konnte gezeigt werden, daß reine, nicht-deformierende Rotations-Acceleration des Schädels nicht nur Läsionen des Gehirns, sondern auch des gesamten Rückenmarkes bis zur Cauda equina bewirken kann.

In Fällen, wo Menschen schwerer Rotations-Acceleration des Schädels ausgesetzt sind, sollten Gehirn *und* Rückenmark pathologisch untersucht werden.

Key-Words: Rotational Acceleration Trauma — Head Injury, Experimental — Angular Acceleration — Traumatic Necrosis — Spinal Cord Lesion.

Experimental pathomorphological studies concerning the influence of blunt violence on the free moving skull of animals have been reported by JAKOB (1913) and PETERS (1943). The investigation was further pursued along the same line by WINDLE, GROAT and FOX (1944), among others. The velocity of the mass, and the acceleration imparted to the skull were measured by UNTERHARNSCHEIDT (1962, 1963, 1968), UNTERHARNSCHEIDT and SELLIER (1965, 1966), and SELLIER and UNTERHARNSCHEIDT (1963, 1965), in experimental acceleration and deceleration traumas produced by single and repeated blows to the free moving head.

The present study concerns itself with the effects of experimental rotational acceleration on the CNS, which had not been investigated before. To study these effects alone, a specially designed device was fabricated which produces a non-deforming rotational acceleration through an axis near C-7-Th-1 through a known path. This device has been described elsewhere by HIGGINS and SCHMALL (1967).

Material and Methods

Twenty-five adult squirrel monkeys (Samiri sciureus) were subjected to this environment at acceleration levels which covered the range from no observable clinical effect to lethality within twenty minutes.

* Supported in part by Contract DA-49-193-MD-2610 with the U.S. Army Medical Research and Development Command, Washington, D.C., NB 07377-01, with USPHS-Dept. of Health, Education and Welfare and Mary K. Petersen Foundation.

The experiments were geared to yield first insights into the quality and location of primary traumatic lesions related to this type of trauma. For this reason, surviving animals were sacrificed five days after the experimental injury. Acceleration and physiological signals were acquired using transducers and signal conditioning apparatus, and recorded on a high speed optical oscillograph. The method and results of acceleration calculations, and the detailed clinical findings have been reported elsewhere (HIGGINS *et al.*, 1967).

Following fixation in 10% formalin, the brains and spinal cords were imbedded in paraffin and celloidin. They were then cut stepwise in serial sections and stained with hematoxylin and eosin, van Gieson, Masson-Goldner, and Nissl methods. The thickness of the celloidin sections was 20 μ and that of the paraffin sections was 9 μ. The histological sections of the cerebrum, midbrain, pons, medulla oblongata and cerebellum were stained with the Nissl method and with hematoxylin-eosin; those of the spinal cord were also stained with van Gieson and Masson-Goldner.

Results

Three of twenty-five animals died spontaneously after 8, 16 and 20 minutes. They had suffered the highest rotational accelerations (3.27; 3.63 and 3.86×10^5 radians/second^2). All three animals exhibited severe primary traumatic alterations; subdural, subarachnoid hemorrhages, rhectic hemorrhages at the base of the frontal and/or temporal lobes, the occipital lobes, near the midline, and in more central parts of the brain (hippocampus and around the third ventricle).

Examination of the remaining twenty-two monkeys showed an obvious increase in the degree of primary alterations with increasing angular acceleration. In addition to subdural and subarachnoid hemorrhages (Fig. 1), tearing and avulsion of veins in superficial cortical layers, leading to rhectic hemorrhages and partial and total traumatic necroses, as well as rhectic hemorrhages in cranial nerves, caused by stretching were observed. With the exception of a few animals, the spinal cords always showed small rhectic hemorrhages, largely in gray matter in various segments. They appeared even with low intensities, but increase less distinctly with greater intensity.

Discussion

In any acceleration of the head, either translational, or rotational components may predominate. Studies of rotation alone are few in number. If the impact axis runs through the center of gravity of the skull or its model, the resulting motion is translatory. If, however, the axis traverses the skull obliquely, outside the center of gravity, the result is a combined translatory and rotatory movement typically illustrated in the uppercut in boxing. The process can be compared to a sphere rotating on a stick.

In a generalized motion of the head, this motion can be described as a translation of the center of mass of the head and a rotation about this center of mass. It can be shown on kinematic grounds alone (OELKER *et al.*, 1966) that the head-brain system would be more sensitive to the rotational than to the translational acceleration component. In fact, the farther from the center of rotation the particle of interest is, the greater acceleration suffered, and thus the greater potential for injury. Thus, a point near the center of rotation is only likely to be damaged if the rotation (or translation) is severe.

In translational acceleration produced trauma (e.g. man falls backward on ice), the lesions are usually found opposite to the impact point. This has been reported by a number of authors (see SPATZ, 1950; SELLIER and UNTERHARNSCHEIDT, 1963).

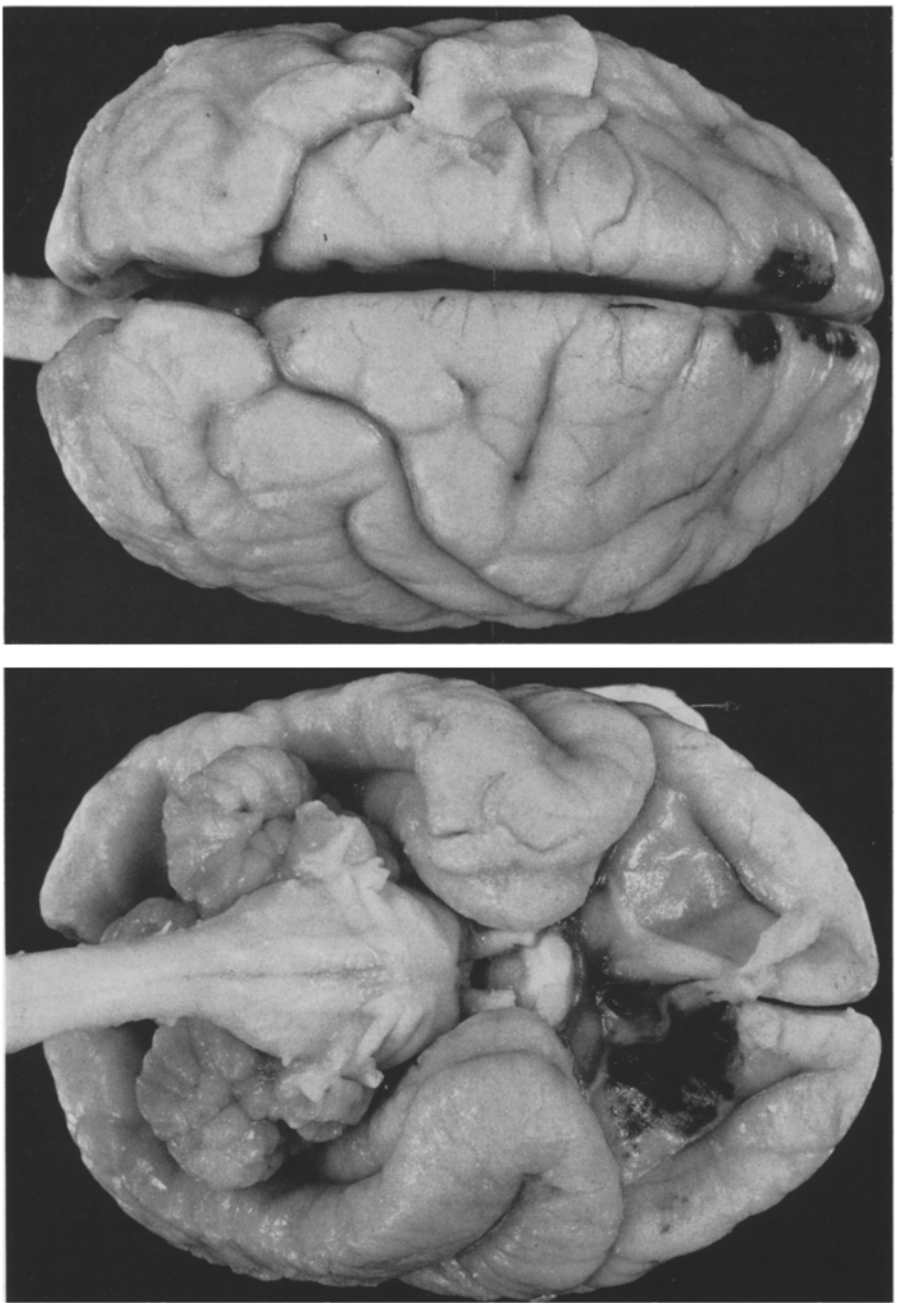

Fig. 1a and b. Squirrel monkey no. 14. Rotational acceleration of the head 1.87×10^5 rad/sec^2. a Small focal subarachnoid hemorrhages (and histologically rather superficial rhectic hemorrhages due to vessel rupture after strain) near the midline on the pallium and in central parts in the fissura interhemispherica. b Subarachnoid hemorrhages (and histologically rather superficial rhectic hemorrhages due to vessel rupture after strain) in basal portions of the frontal lobe. The left tractus olfactorius also shows hemorrhages. Macrophoto

Very few groups have studied rotational acceleration (OMMAYA *et al.*, 1968; MARTINEZ, 1968). None of these have controlled the path taken by the head, or the waveform of acceleration used. The experimental designs closest to our own are those in which the head is caused to move by inertia forces resulting from whole body acceleration. However, in our setup, the head is moved while encased in a plaster-filled helmet. Hence the primary input is to the head, and not to the whole body; yet we have demonstrated significant lesions in the spinal cord.

Rotational traumas, by contrast, involve considerable tensile forces between the accelerated head and the inert brain. It can be expected, according to the physical analysis of translational and rotational traumas, that the different lesioning mechanisms also produce different patterns of lesions. In the histological evaluation special attention was given to the effect of rotation of the head on the various parts of the spinal cord—one aspect that had been neglected so far.

As we have mentioned above, the pattern and quality of histopathological lesions was different in rotational traumas. We expected subdural hemorrhages due to tearing of bridging or connecting veins between the brain and dura; subarachnoid hemorrhages predominantly in the midline, straining and tearing of vessels in cranial nerves, in superficial layers of the cortex, and with stronger intensities, also in deeper parts of the brain. We expected vessel ruptures due to strain in the spinal cord far from the impact site, but along the principal axis of stress. That hyperflexion and hypertension can produce changes in position of the spinal cord, spinal column, roots and cauda equina, under quasi static conditions, has already been pointed out by BREIG (1960) on cadavers.

References

BREIG, A.: Biomechanics of the central nervous system. Some basic normal and pathologic phenomena. Stockholm: Almquist & Wiksell 1960.

HIGGINS, L. S., and R. A. SCHMALL: A device for the investigation of head injury effected by non-deforming head accelerations. Proc. 11th Annual Stapp Car Crash Conference, Society of Automotive Engineers, New York, Paper No. 670905 (1967).

JAKOB, A.: Experimentelle Untersuchungen über die Schädigungen des Zentralnervensystems (mit besonderer Berücksichtigung der Commotio cerebri und Commotionsneurose). NISSL-ALZHEIMER: Histologische und histopathologische Arbeiten über die Großhirnrinde, Bd. 5, S. 182—341. Jena 1913.

MARTINEZ, J. L., and D. J. GARCIA: A model for whiplash. J. Biomechanics **1**, 23—32 (1968).

OELKER, C. E., F. H. PRIMIANO, and H. C. WOODING: The investigation of the parameters of head injury related to acceleration and deceleration. Technology Incorporated Annual Report, Contract DA-49-193-MD-2610 (TI 2610-66-4) Dayton, Ohio (1966).

OMMAYA, A. K., F. FAAS and P. YARNELL: Whiplash injury and brain damage. J. Amer. Med. Ass. **204**, 285—289 (1968).

PETERS, G.: Über gedeckte Gehirnverletzungen (Rindenkontusion) im Tierversuch. Zbl. Neurochir. **8**, 172—208 (1943).

SELLIER, K., u. F. UNTERHARNSCHEIDT: Mechanik und Pathomorphologie der Hirnschäden nach stumpfer Gewalteinwirkung auf den Schädel. Hefte Unfallheilk., Heft 76. Berlin-Göttingen-Heidelberg: Springer 1963.

— — The mechanics of the impact of violence on the skull. In Proc. Internat. Congress of Neurological Surgery, Copenhagen, Aug. 28, 1965. Excerpta Medica Intern. Congr. Series **110**, 87—92 (1965).

SPATZ, H.: Brain injuries in aviation. In: German Aviation Medicine, World War II. Dept. of the Air Force, Vol. 1, pp. 616—640 (1950).

UNTERHARNSCHEIDT, F.: Experimentelle Untersuchungen über gedeckte Schäden des Gehirns nach einmaliger und wiederholter stumpfer Gewalteinwirkung auf den Schädel. Fortschr. Med. **80**, 369—378 (1962).
— Die gedeckten Schäden des Gehirns. Experimentelle Untersuchungen mit einmaliger, wiederholter und gehäufter stumpfer Gewalteinwirkung auf den Schädel. Monogr. Gesamtgeb. Neurol. Psychiat., Heft 103. Berlin-Göttingen-Heidelberg: Springer 1963.
— Mechanics and pathomorphology of closed head injuries. Proc., Impact Injury and Crash Protection, Wayne State University, Detroit, May 9 and 10, 1968.
—, and K. SELLIER: Pathomorphology of nonpenetrating brain injuries. In: Proc. III. Internat. Congress of Neurological Surgery, Copenhagen, Aug. 23—28. Excerpta Medica Internat. Congress Series **110**, 93—103 (1965).
— — Mechanics and pathomorphology of closed brain injuries. In: Conference Head Injury Planning Committee, Chicago, Febr. 7—9, 1966, Chapt. 26, pp. 321—341. Philadelphia: Lippincott 1966.
WINDLE, W. F., R. A. GROAT, and C. A. FOX: Experimental structural alterations in the brain during and after concussion. Surg. Gynec. Obstet. **79**, 561—572 (1944).

F. UNTERHARNSCHEIDT, M.D.
Division of Neuropathology and
Experimental Neurosurgery
University of Texas Medical Branch
Galveston, Texas, 77550, U.S.A.

Aktuelle Mitteilungen · News items · Renseignements actuels

Le VIème *Congrès International de Neuropathologie* se tiendra à Paris, du 31 Août au 4 Septembre 1970, sous la Présidence du Dr. JEAN GRUNER. Toutes les informations peuvent être demandées au Secrétaire Général, le Professeur JEAN LAPRESLE, à l'adresse suivante: Hôpital de la Salpêtrière, 47 Boulevard de l'Hôpital, 75-Paris 13ème.

2. Donausymposium für Neurologie

5.—9. Mai 1969, Wien

Neurologie: 5. und 6. Mai 1969

Themen: „Demyelinisierende Erkrankungen des Nervensystems mit besonderer Berücksichtigung der Multiplen Sklerose"
„Die Rehabilitation neurologischer Erkrankungen"

Neuropathologie: 7. Mai 1969

Themen: „Demyelinisierende Erkrankungen des Nervensystems mit besonderer Berücksichtigung der Leukodystrophie"
„Freie Vorträge"

Kinderneurologie: 8. und 9. Mai 1969

Themen: „Die kindliche Epilepsie mit besonderer Berücksichtigung der BNS-Krämpfe"
„Dyslexie"

Die Tagung steht unter der Ehrenpräsidentschaft von Dr. MACDONALD CRITCHLEY, Präsident der WFN.

Veranstalter der neuropathologischen Tagung: Österreichische Arbeitsgemeinschaft für Neuropathologie.

Leiter: Prof. F. SEITELBERGER

Schriftführer: Doz. Dr. K. JELLINGER

Neurologisches Institut der Universität Wien, 1090 Wien, Schwarzspanierstraße 17

Organisation: Wiener Medizinische Akademie für Ärztliche Fortbildung, 1090 Wien, Alserstraße 4 (Allgemeines Krankenhaus-Direktionsgebäude)