CONTUSION OF THE BRAIN[1]

By John H. W. Rhein, M.D.

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM AT THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE; NEUROLOGIST TO THE HOWARD HOSPITAL

Concussion is looked upon by many writers as a molecular vibration in the brain substance. On the other hand Kocher,[1] Keen[2] and others believed that there is an actual injury to the brain substance. In support of the former contention, the experimental work of Koch[3] and Filehne and that of Witkowski[4] on dogs shows that fatal concussion may be caused without more than evidence of hyperemia of the brain and it coverings.

Recently I have been called upon to examine a number of patients who had received injuries to the head and in whom symptoms of so-called concussion have lasted several days, also one patient with localizing symptoms who recovered, and one with localizing symptoms who died. I have been able to add to these a case kindly furnished me by Dr. W. G. Spiller. As a result of this experience I was led to make a study of the subject of contusion of the brain from a microscopic standpoint, as well as, in part, clinically. The number of cases is scarcely large enough to permit of any positive deductions, but some interesting features of the subject have been developed as a result of this study, the results of which may be looked upon as a preliminary report.

I found in the first place that the study of contusion of the brain is largely neglected by neurologists and on the other hand that comparatively little has been written about the subject even by surgeons. At first after reading Phelps's[5] paper on the subject in which he so ably and exhaustively deals with contusion and laceration of the brain I was tempted to drop the subject entirely. His paper however treats of the pathology of the subject from a macroscopic standpoint largely.

Little has been written since his paper published in 1893 and

[1] Read by title at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914. From the laboratory of Neuropathology, University of Pennsylvania, and the Philadelphia Polyclinic and College for graduates in Medicine.

this brief paper on the subject is warranted, I believe, by the fact that sufficient attention has not been given since to the subject by neurologists, whose assistance is or should be sought in each case before operation is decided upon.

Phelps analyzed 124 cases of brain injury occurring in his own experience. As a result of this study he stated that laceration and contusion were first in frequency and importance among all the injuries to the head. Contusion he stated may occur without lacerations and vice versa but they are usually associated.

FIG. 1. Case 1. Showing subdural and subpial hemorrhage.

There were 28 cases of laceration without noticeable contusion in 58 cases, and but 10 cases of contusion without lacerations. By laceration he means a tearing of the tissue, the wound containing a coagulum beneath which is found a granular detritus of the brain structure. He described subcortical laceration as a simple extravasation of blood into the deeper tissues. In contusion he claims there is no solution of the continuity of the brain fibers but there are minute hemorrhages in the brain substance.

In Phelps's experiences the lacerations occurred more especially on the basal surface of the temporal and frontal convolutions but all parts of the brain may be affected, including the pons, cerebellum, the corpora striata and the gyrus fornicatus. Lesions may

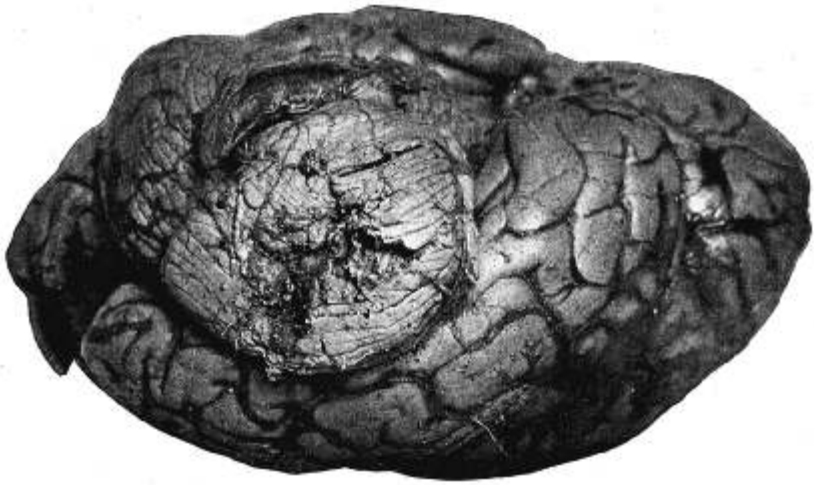

FIG. 2. Case 1. Showing area of softening in cerebellum.

be superficial or deep, or may implicate the interior of the brain without reaching the surface. The membranes may or may not be involved and the pial hemorrhage may extend over large areas. The hemorrhage may be the only lesion in some places.

Kocher (in 1901) has given the best description of the subject since Phelps's paper. He quotes six cases, five of them fracture of the bones of the skull and one, a case of marked contusion aggravated by excephalitis. Contusion of the temporal lobes, of the left frontal lobe and of the lower surface of the frontal lobe was observed in these cases caused by contre-coup and there was associated, epidural and sub-dural extravasation.

Page[6] in 1902 in discussing the subject of concussion of the brain in some of its surgical aspects described bruising of the brain tissue, not only at the seat of injury but also at a point opposite, due to contre-coup or the tearing of the walls of the brain from sudden displacement of the cerebro-spinal fluid from the lateral ventricle through the Sylvian aqueduct and fourth ventricle.

In the cases briefly to be described the chief points of interest

are as follows: Extensive pial hemorrhage may exist without any injury to the cortex or cortical cells. Contused areas showed fairly sharply defined infiltration of blood with little demonstrable effect upon the adjoining cortical tissue. An inflammatory process was not found in the brain tissue. No evidence of a proliferative process was observed in that portion of the pia which was the seat of the hemorrhage. Injury to the brain appeared in some places to be due to hemorrhage extending from the pia. The cortical cells adjacent to the hemorrhagic infiltration of the cortex did not appear to show any evidence of degeneration.

The question arises, is it not advisable to drain the hemorrhagic area in the brain by an incision and thus relieve the pres-

FIG. 3. Case I. Showing subpial hemorrhage which extruded into the superficial cortical layers.

sure and ultimate destruction of the brain tissue which would ensue if the hemorrhage were allowed to remain? If the bruised areas were drained would not the tissues have less of a tendency

to undergo softening and would not the relief of the pressure add to the chances of saving life?

CASE 1. E.McQ., aged 61, was admitted to the Philadelphia General Hospital, April 28, 1906, and died three days later. There is no history of injury but there is no doubt from the autopsy findings that she must have had some serious blow or injury before admission. The examination by Dr. Spiller was as follows: She had recurrent convulsive attacks involving the left side of the face in the upper as well as the lower distribution. The eyeballs were jerked to the left clonically and there was a slight jerking of the right eyelid in addition to the violent spasms of the left. The right arm was flexed at the elbow and the left

FIG. 4. Case 2. Showing areas of contusion—and hemorrhage into cortex.

arm and both legs were rigid at first. This was followed by clonic spasms in the left arm and both legs but not in the right arm. She was slightly stuporous between the convulsive attacks but replied to questions although unreliably. These convulsions occurred about every 10 or 15 minutes.

The left side of the face was distinctly paretic in both the upper and lower distributions of the facial nerve. She closed the eyelids of the left side but with less force than on the right. The tongue was protruded straight. There was left hemianopsia. Sensation to touch and pain was diminished on the whole left side of the body, including the face. The left arm and leg seemed to be completely paralyzed. The triceps and biceps tendon reflexes were very prompt on both sides but more so on the left.

The left arm and leg were flaccid. The patellar reflex was prompt on each side and a little more so on the left. The Achilles jerk was not obtained on either side. There was no ankle clonus and the Babinski phenomenon was uncertain on the left side and probably absent on the right. At a previous examination it was present on the left side.

Two days after admission the patient was operated upon, a diagnosis of brain tumor having been made in the absence of any history of injury. The skull was opened on the right side over the motor area. No tumor was found but when the dura was opened large quantities of fluid somewhat blood tinged escaped. The hemorrhage was readily controlled and the dura was allowed to fall into place without suturing. The patient died the following day.

In the brain there was found on the right side in the region of the frontal lobe and especially over the face center an extensive sub-pial hemorrrage which extended down to the basal surface. There was also an area of hemorrhagic infiltration in the region of the arm center. A hemorrhagic area in the temporal lobe on the basal surface was observed. In addition to this a sub-dural hemorrhage in the region of the parietal lobe on the right side was seen and in the posterior part of the left cerebellar hemisphere there was also an area showing softening from bruising or contusion.

Microscopically in the precentral region there was seen a hemorrhage which at first sight appeared to be a hemorrhagic area in the brain cortex but which proved to be an accumulation of blood beneath the pia which pushed the brain matter down beneath it. Just beneath the pial hemorrhage the cortical tissue showed very little damage. The main lesion consisted of a hemorrhage into the cortex at the site of the contusion. In the tissue adjoining the area of contusion there was some rarification of the tissue and space formation. In the frontal region there was a hemorrhage between the pia and the arachnoid which pressed down on the cortex. In most places the cortex was perfectly normal but in one place the hemorrhage had extended into the cortex. The pia, at the seat of the hemorrhage was vascular, and the walls of the vessel were thicker and the lumen larger than normal. The pia itself was thickened and stained amorphously. There was not found any round cell infiltration.

Adjoining the hemorrhage into the cortex were smaller hemorrhages and space formation. In one section an enormous pial hemorrhage did not materially injure the cortex and in only a small area was there seen any disintegration of the cortex from the hemorrhage. There was in the pia very extensive hemorrhagic extravasation which occurred independent of any implication of the cortex.

FIG. 5. Case 2. Showing areas of contusion.

In the areas of contusion the brain substance was infiltrated with red blood cells to such an extent as to entirely mask any of the normal appearance of the brain. Adjoining this before reaching normal brain tissue were seen many microscopic hemorrhages. Here the ganglion cells could be seen well stained and intact.

There was no evidence of any inflammatory process or reaction. The sections stained with thionin showed no degeneration of the nerve cells in the cortex.

CASE 2. F. S., aged 53, was admitted to the Philadelphia Polyclinic Hospital on January 24, 1913. He was brought into the accident ward in an unconscious condition having been knocked down in a fight, his head as he fell striking the cement floor of an abattoir where he worked. There was no external evidence of injury with the exception of some contusion over the left eye

and in the occipital regions. He was not entirely comatose upon admission but did not make replies to any questions. He was restless and red faced. The pupils were contracted and reacted to light. As far as could be ascertained there did not seem to be any paralysis upon admission.

On January 26 two days later he was still unconscious and there was incontinence of urine and feces. Both knee jerks were increased and there was a Babinski phenomenon on the left. On this date two days after the acident there was observed twitching of the right side of the face which became almost constant and continued until the patient died five days later. On the twenty-eighth two days after the convulsions of the face began, the right arm became the seat of clonic convulsions and on the following day the right leg also became involved. He died seven days after accident without regaining consciousness.

At the autopsy a fracture of the base of the skull extending from the occipital region to the frontal region on the left side was found. The whole brain was very badly bruised. In the left precentral region involving the second and third frontal convolution in their posterior parts there was an area of hemorrhagic softening measuring 6 cm. by 3 cm. Extensive sub-pial hemorrhage surrounded this area and extended forward and downward to the basal surface. There was a sub-dural hemorrhage in this region also. On the basal surface of the brain could be found areas of softening or contusion of the brain tissue occupying the region of the anterior temporal lobe and the anterior portion of the frontal lobe. Surrounding these areas was found extensive hemorrhage into the pia to a greater or less extent.

Microscopically studies were made of the contused areas. The pia arachnoid was in places alone involved, the hemorrhage separating the pia from the arachnoid. Beneath these places the cortex was intact, showing nothing abnormal with the nuclear stain and no destruction or alteration of the cells when stained with thionin.

In the areas of contusion the brain substance was infiltrated with blood and here there could be seen no traces of the normal brain tissue. Adjoining these areas minute hemorrhages were seen in the cortical layer for a short distance in transition to healthy brain tissue. The cells in these localities showed no change though perhaps they took the stain less intensely than the normal. Just adjoining the extensive hemorrhagic infiltration of the cortex in one place the cells stained poorly by the thionin stain and a few very poorly or not at all. In the pia there was no evidence of proliferation or exudation of round cells, nor in the cortex at the seat of contusion was there any round cell infiltration.

CASE 3. W. B.. aged 46, was admitted to the Philadelphia Polyclinic Hospital on December 16, 1913. He was in a some-

what dazed condition upon admission and complained of pain in the head, having received a contused wound in the right parieto-occipital region. Twenty-four hours later he had a general convulsion. Upon examination two hours after the convulsion his condition was as follows. He was confused and had difficulty in expressing himself. Further examination revealed the fact that he was aphasic. He was unable to understand spoken language or writing and was unable also to express himself, although there was no anarthria. There was a tendency to divergence of the right eye. The pupils were very small but reacted promptly and the rotation of the eyeballs was good. There was possibly a slight twitching of the right external rectus. When examined by Dr. Peter the next day, the eye grounds were practically negative.

On the following day an examination showed in addition to the aphasia some apraxia. For example repeatedly when asked to close his eyes he would show his teeth or he would make a fist.

An X-ray examination showed a possible fracture in the right occipital region. There was a gradual improvement in his condition, including disappearance of the aphasia and apraxia which persisted for about a week. His chief complaint then consisted of pain in the occipital region and over both scapulas. Mentally he was very slow and answered questions in monosyllables.

At the end of five weeks he was discharged cured. (A week later he committed suicide by inhalation of illuminating gas.)

This case was one in my opinion of contusion or perhaps laceration of the temporal region on the left side causing apraxia and aphasia. The hemorrhage into the cortex must surely have been absorbed allowing thus the function of the brain tissue to be restored, the pressure being relieved by absorption of the hemorrhage.

It would seem to me in studying the cases of so-called concussion, in which the symptoms persist for several days and in which even weeks pass by before the patient regains the normal state that the condition is best explained on the basis of a contusion or actual injury to the brain substance itself. The brain becomes bruised as it is thrown against the bony structure of the cranium. The difference between concussion and laceration is one largely of degree, there being no sharp line between concussion and contusion on the one hand or contusion and laceration on the other.

LITERATURE

1. Kocher. Nothnagel's System, Wien, 1901.
2. Keen. In DaCosta's Modern Surgery, 1914.
3. Koch and Filehne. In Cushing. Keen's System of Surgery, Vol. 3, p. 186.
4. Witkowski. In Cushing.
5. Phelps. New York Med. Jour., 1893, p. 29.
6. Page. Brain, 1902, Vol. 25, p. 1.