Staff Meeting Bulletin
Hospitals of the » » »
University of Minnesota

Ocular Changes In Head Injuries

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STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
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INDEX

Published for the General Staff Meeting each week during the school year, October to June, inclusive.

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William A. O'Brien, M.D.

I. LAST WEEK

Date: January 30, 1942

Place: Recreation Room,
Powell Hall

Time: 12:15 to 1:20 P.M.

Program: "Treatment of Staphylococcal Infections"
Wesley W. Spink
John R. Haserick

Discussion
Wesley W. Spink
C. J. Watson
O. H. Wangensteen
Clarence Dennis
A. D. Hirschfelder
Arild Hansen

Present: 140

Gertrude Gunn,
Record Librarian.

II. ANNOUNCEMENTS

1. HOBBY SHOW

The Minnesota Union Board of Governors is again sponsoring a Hobby Show. Any faculty member, student, or employee of the University is eligible to enter. The show will be open to the public from Feb. 16 - 22 inclusive. The displays must be checked in at the recreation room of the Union between 8 A.M. and 5 P.M. before Feb. 6. The following classifications will be included: creative art, collections, group activities, general handicrafts, household crafts, natural history, photography, technical.

Application blanks may be secured in Coffman Memorial Union headquarters office.

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2. DINNER

All Members of the University Staff
And Their Wives or Husbands

Are invited to attend an

INFORMAL RECOGNITION DINNER

in honor of
U.S. General Hospital Number 26
The University of Minnesota Unit

Tuesday, Evening, Feb. 10th, at 6:30.
In the Ballroom of the Coffman Memorial Union
Tickets $1.00
Tickets may be secured from
Superintendent Amberg's Office.

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3. MEETING

Seminar on the Structure and Behavior of Proteins. Dr. W. Heller.

"Double Refraction in Proteins"

Room 15, Medical Sciences, 8:00 P.M.

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4. WANTED

Physicians for group practice. See Dr. O'Brien for details.

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5. IMPORTANT

Occupational diseases must now be reported to the Minnesota Department of Health. Blanks for the purpose may be obtained from the Division of Industrial Hygiene, Campus.

Studies on industrial hazards will be made if brought to the attention of the director.

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III. OCULAR CHANGES IN HEAD INJURIES

Donald E. Otten
Frank E. Burch

This survey of ocular changes associated with head injury indicates the importance of close collaboration between the ophthalmologist and neurosurgeon. Such cooperation aids diagnosis in the prevention and correction of a considerable number of the unfortunate sequelae of head injuries. The purpose of this paper is not to present all of the possible ocular changes that may result from head injury but merely to give the experiences encountered after one year as ophthalmological consultant to the neuro-surgical staff of the University of Minnesota Hospitals.

Seventeen cases of head injuries were seen during this period. Of the 17, 12 presented various ocular changes. One case, seen at another hospital, is included because of the interesting features presented.

The cases included in this series present, with few exceptions, the late sequelae of head injuries. Seven of the 11 cases were seen several weeks to months after the initial injury. Very few of the early, acute signs of head injuries, viz., ecchymosis, pupillary changes, leakage of cerebrospinal fluid, etc. were encountered. For this reason the percentage of ocular findings in this series is lower than if the cases had been seen earlier, when acute signs were present.

An anatomical basis for the common occurrence of ocular changes in head injuries was laid down as early as 1879 by Berlin[16] and later substantiated by Rawling[17] and Vance[18]. These authors showed that in nearly all cases of skull fractures the base was involved. The body of the sphenoid acts as a hub for fractures resulting from forces directed at the anterior and middle fossae. These 2 fossae are bounded by spokes of thickened bones, i.e., the squamous portion of the temporal bone, the lesser wings of the sphenoid, and the crista galli of the ethmoid bone. These spokes of more resistant bone tend to confine basal fractures to the middle and anterior fossae and direct the fracture line through the body of the sphenoid. Rawling[17] gives the following valuable summary of symptoms and signs associated with fractures of the base of the skull.

Anterior Fossa	Middle Fossa	Posterior Fossa
Hemorrhages	**Hemorrhages**	**Hemorrhages**
Subconjunctival	Into temporal region	Into nuchal region
Palpebral	From the mouth	Into occipital region
Peripalpebral	From the nose	Into post-auricular region
Orbital	From the ear	
Retinal		
From the nose		
From the mouth		
Cerebro-spinal fluid	**Cerebro-spinal fluid**	**Cerebro-spinal fluid**
From the nose	From the nose	None
From the mouth	From the mouth	
	From the ear	
Brain-Matter	**Brain-matter**	**Brain-matter**
From the nose	From the ear	None

Anterior Fossa	Middle Fossa	Posterior Fossa
Air-escape	**Air-escape**	**Air-escape**
From the frontal sinus	From the mastoid antrum	From the mastoid antrum
From ethmoidal cells		
Nerve-involvement	**Nerve-involvement**	**Nerve-involvement**
Olfactory	Fifth (2nd & 3rd divisions)	Seventh
Optic	Sixth	Eighth
Third	Seventh	Ninth
Fourth	Eighth	Tenth
Fifth (1st division)		Eleventh
Sixth		Twelfth (?)

- - - -

Damage to various cranial nerves is by far the commonest type of lesion in these cases. In 11 cases with ocular disturbances, 7 had cranial nerve lesions. According to the nerves involved analysis shows: 6th nerve, 4 cases; 3rd nerve, 2 cases; 4th nerve and trochlea, 2 cases; 7th nerve, 2 cases; 5th nerve, 2 cases. The distribution of muscle involvement is comparable to the findings of other investigators[11,12]. An exception to this is the appearance of 2 cases of trochlear muscle disturbance.

From the literature[11,12,13] the appearance of trochlear muscle disturbance is rare. Wilbrand and Saenger[21] (1921) found only 2 such cases in an extensive review of the literature. Of the 2 cases seen here, one was found in conjunction with an almost complete bilateral ophthalmoplegia. In this case, there was demonstrable an extensive fracture extending well into the base of the skull. The other case resulted from a severe blow in the face which caused a displacement of the trochlear process. This abnormality is clearly substantiated by x-ray studies.

These 2 cases do not detract from the statistical evidence that isolated 4th nerve lesions arising from head trauma are uncommon, inasmuch as neither were strictly speaking cases of isolated 4th nerve involvement. Involvement of this nerve may occur more frequently than reported in conjunction with other nerve involvements, and, also where bony changes are pronounced.

The 6th nerve was the most commonly affected in the present series, a fact that is consistently found in reports by other authors.[12,21,22,23] This phenomenon may be explained on an anatomical basis. The 6th nerve has the longest intracranial course of any cranial nerve, and in its course passes abruptly over the apex of the petrous portion of the temporal bone. This relationship makes the nerve vulnerable to changes occurring in this region due to fractures. The 6th nerve is fixed both at its origin in the pons, and in the cavernous sinus. With trauma to the vault of the skull even though a basal fracture is not present, the hind brain is displaced downward into the foramen magnum. This causes a stretching of the 6th nerve which presses it against the sharp ridge of the petrous bone. A similar displacement of the brain occurs with increased intracranial pressure.[22] Cushing demonstrated that if the anterior inferior cerebellar artery ran in a course vertical to the 6th nerve, with the presence of increased intracranial pressure, the nerve is grooved by it so as to interfere with its function.[20] The 6th nerve is the weakling of the cranial nerves and is so commonly affected that it has very little localizing value.[22] In 2 cases with 6th nerve lesions, no fracture could be demonstrated. Negative roentgenographic evidence makes it difficult to decide

whether nerve damage is due to a basal fracture or some other cause, such as indirect trauma, increased intracranial pressure. Two other cases with 6th nerve involvement presented a definite fracture into the base. In both, the fracture ran in a transverse direction, through the middle fossae.

Third nerve involvement was encountered in 2 cases. One represents an isolated unilateral and complete 3rd nerve palsy. In this case, along with the 3rd nerve disturbance, there was evidence of bilateral upper motor neuron disturbance. A basal fracture was demonstrated in this case. Dr. J. C. McKinley was of the opinion that the 3rd nerve lesion was on the basis of extracerebral disturbance (fracture) and that the upper motor neuron disturbance was on the basis of multiple intracerebral hemorrhages. The 3rd nerve lesion in one case resulted in a bilateral almost complete ophthalmoplegia. A definite fracture line in the base of the skull could be demonstrated. With such extensive cranial nerve damage involving the 3rd, 4th, and 6th nerves, the fracture must have extended into the superior orbital fissure. Although not demonstrated by x-ray, it is probable that a branch of the main fracture did extend into this region as suggested by Wilbrand and Saenger.[21]

Nystagmus has been infrequently reported following head injury. Blakeslee[12] reported only 13 such cases in a series of 610 fractures of the skull. This phenomenon may at times be elicited by vestibular tests when not present spontaneously.[6] The significance of this is questionable.

The two cases of 7th nerve lesions were both associated with 8th nerve changes. These probably arose as a result of fractures into the squamous portion of the temporal bone. In one case such a fracture was demonstrable with x-rays. When the 7th nerve is involved in head trauma, the 8th nerve is also almost invariably involved. This is because of the close association of these two nerves in their passage through the temporal bone.

Neither of the 2 cases with 5th nerve lesions showed complete involvement. In one case only the ophthalmic and maxillary branches were involved. In another the right maxillary branch was affected. This latter case had in addition a palsy of the right 6th nerve. No fracture line could be found in either case. The diagnosis was necessarily made on the history and physical findings.

From the cases in the literature, it appears that the occurrence of choked discs is relatively uncommon (Holding 10%, Battle 8%, Graf 4%).[10] Cohen[11], in a series of 75 cases, stated that he had never seen a case with true papilledema following head trauma. Further reports[12] emanating from the same hospital at a later date merely say that the occurrence of papilledema is rare. This view is not born out in this series since 17% of the cases present this phenomenon.

Papilledema when found strongly suggests the possibility of a subdural hematoma (or other expanding lesion, or meningitis). This fact is well emphasized by the cases in the literature,[10] and by the cases presented in this series. Three cases presented ophthalmoscopic changes. Two of these cases demonstrated papilledema of from 3 to 4 diopters while the 3rd presented in addition to choked discs, a retinosis that has been described by Purtscher[3] as being pathognomonic of trauma to the head (so called Angiopathia, Retinae Traumatica of Purtscher). All 3 cases with papilledema were associated with subdural hematoma. In one, the case of a three months' old child in which the history of head trauma was not certain, the presence of a subdural hematoma was first suspected by ophthalmoscopic examination. All 3 cases presented bilateral equally choked discs. In rare instances the papilledema may be unilateral.[10]

The occurrence of a unilateral papilledema or the presence of more edema in one eye than in the other, may, in some instances, indicate the side on which the hematoma exists, i.e., on the side

of the most edema. A unilateral dilated pupil may also indicate the side of the lesion. In such a case the pupillary dilatation is on the same side as the hematoma.[27,5] In Blakeslee's series of 610 skull fractures, 378 patients showed pupillary change. The mortality in the group with such changes was 47.5%.[12]

The onset of papilledema may vary from hours to twelve days.[10] The exact onset in the three cases reported here could not be determined except to say that it was present at 3 days after injury in one case and after $4\frac{1}{2}$ months in another. Eagleton[28] stressed the importance of daily fundus examination in those cases where an increased intracranial pressure seemed most likely to develop. He believed that "injuries confined to the anterior part of the frontal lobe do not produce papilledema..... injuries of the posterior fossa with occlusion of the iter produce a high degree of papilledema.....injuries of the cerebral hemispheres with secondary edema produce moderate papilledema. This may last for months or may rapidly disappear." The extreme variation in the onset and the time of onset renders desirable repeated ocular examinations.

One case presented, in addition to the papilledema, a great number of white round exudates and retinal hemorrhages of various sizes and shapes. Some of the hemorrhages were punctate, others were as large as 3 to 4 disc diameters in size and lay beneath the subhyaloid membrane. Apparently this type of retinosis is quite rare or goes unnoticed, as is evidenced by the lack of reports of this condition throughout the literature (only 10 cases up to 1932).[7] Purtscher[3] believed the retinal changes due to head trauma were the result of a sudden rise in intracranial pressure. It was his theory that the sudden rise in pressure is brought about by the longitudinal compression of the spinal column that occurs in head injury. This pressure is transmitted into the subarachnoid space of the optic nerve sheath, through the optic nerve head and into the perivascular lymph spaces of the blood vessels. Rupture of the lymph sheaths of the blood vessels causes extravasation of the lymph elements into the superficial layers of the retina which accounts for the appearance of the white patches. He believed the hemorrhages to be due to a damage to the vessel walls by the same process.

The outcome of these hemorrhages may take one of three courses: complete absorption, production of a massive retinal fibrosis, or production of congenital amblyopia.[1,9] The latter is believed to be due to a large macular hemorrhage with separation of the retinal layers.

In the case cited here there was a complete absorption of the hemorrhages in about 2 weeks. It is difficult in this case to prognosticate as to the ultimate visual acuity in view of the patient's extreme youth. Vision may be impaired later due to one of the unfortunate sequelae of retinal hemorrhages.

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Visual field changes following head trauma were seen in 5 cases. In 2 cases the changes were attributed to disturbances about the optic foramen, in 1 case as a result of trauma to the chiasm, in 1 as result of the existence of long standing choked discs, and in 1 the exact explanation is doubtful. Visual field changes that occur in association with disturbances of the optic nerve as it passes through the optic foramen are not uncommon. They are usually not discovered until convalescence is established or until long afterward as in the Ancker Hospital case.

Berlin[16] was the first to give anatomical explanation for the common occurrence of this condition. In analyzing 126 autopsies of skull fracture cases, he brought out that in 80 cases, or 64% of the total cases investigated, the fractures extended into the optic foramen. Rawling[17] stressed the common occurrence of fractures passing through the region of the optic foramen and involving the anterior clinoids. Based on postmortem findings, he gave evidence that this region was the most likely place for fractures resulting from blows directed against the anterior and middle fossae.

Vance,[18] from autopsy studies on 152 cases, verified the findings of these earlier workers.

The onset of complaints in cases with fractures through the optic foramen is usually immediately following the injury. In 2 cases of this series the vision has remained impaired and has not shown any evidence of improvement as usually occurs.

The typical field change is either a peripheral field constriction or a sector defect. The latter more often than not involves the fixation point and gives rise to a great loss of vision. In one case the visual field loss amounted to a marked peripheral constriction almost to the 5 degree circle with associated depression of the central vision. In one case there was no light perception in the affected eye.

The pathological changes in the optic nerve that give rise to the visual field loss can be attributed in certain cases to fracture of the optic foramen, or the anterior clinoid process, with or without hemorrhage into the optic nerve sheath. They produce either compression of the nerve in its bony canal, rupture of the blood vessels passing from the sheath into the nerve and may deprive it of its blood supply.[15] This latter explanation affords a better basis for the occurrence of the sector type of field defect.

Field changes that occur from damage about the optic foramen may be duplicated in some cases of fracture of the anterior clinoid processes. The changes in this instance arise from a direct compression of the nerve by the displaced fragment.[17] It has been reported that there may be an immediate improvement following the initial loss, or, after the initial improvement there may remain a permanent field defect.[14] The recommendation is made[14,20] that when the diagnosis of this condition is made, a decompression of the optic foramen be carried out as early in the course of the disease as is practical. In two cases, advance field defects were present which it was felt precluded the possibility of improvement from operation. One case presented field changes that were not entirely related to trauma of the head. The field changes consisted of a bilateral central scotomata that came on following a head injury but in conjunction with which there was a sudden and severe blood loss. The patient was in acute shock when finally brought to the hospital after a delay of several hours. No evidence could be found in this case of any other possible reason for an optic neuritis. It was assumed that the central scotomata were on the basis of an acute blood loss not directly related to the head injuries. The occurrence of this type of field defect from acute blood loss must be considered when severe hemorrhage accompanies trauma to the skull.

Lillie and Adson[4] have reported 2 cases of delayed optic atrophy following fractures extending through the optic foramen. This late complication was proved to be due to callus formation with pressure on the optic nerve causing central and annular scotomata. Decompression in 1 case did not relieve the condition because undertaken too late. The presence of increased intracranial pressure makes more manifest the changes in the optic nerve by forcing fluid and cells into the perivascular spaces of the optic nerve.[19]

Roentgenological studies are not conclusive in fractures of the optic foramen.[14,20] Most frequently the diagnosis must be made from the history, field changes and the absence of any other ocular pathological changes.

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An arteriovenous aneurysm produced eye changes in one case. This was one of the 2 cases in which the fracture extended through the optic foramen causing complete monocular loss of vision. The injury had occurred over a year before the patient was seen and the visual damage was of too long standing to warrant improvement after an operative procedure. The internal carotid artery was ligated in an attempt to close the aneurysm. Postoperatively the condition was improved but 4 months later, when last seen, the audible murmur had returned.

One case was seen at Anchor Hospital

and is presented in view of its rarity and interest. Drs. Hedemark and Grove had contributed the case history. Following an auto accident in which the patient was knocked unconscious there developed subsequently a bitemporal hemianopsia. This is a very uncommon condition[15,24] and is rarely referred to in the literature. This case is unusual in that a fracture through the body of the sphenoid could be demonstrated by roentgen rays. The first studies of the visual field were made 3 months after the accident and showed amaurosis of the right eye and loss of the temporal field of the left eye. Later some vision returned in the right eye and studies of the fields revealed a bitemporal hemianopsia. The probable explanation is that extensive hemorrhage and edema occurred in the right side of the optic chiasm. As the subsiding of the edema and hemorrhage took place some function returned. As shown by Osterberg[25] experimentally when a normal optic chiasm is stretched minute tears occur in the crossing bundles, whereas the uncrossed fibers are unaffected. This probably accounts for the resultant permanent bitemporal hemianopsia and the sparing of the uncrossed fibers. A controversy may arise as to whether it is possible for an actual tearing of the fibers of the chiasm to occur.

In this case the function of the uncrossed fibers returned, while the function of the crossed fibers did not. It is difficult to understand the mechanism of this unless an actual tearing of the crossed fibers did occur. Otherwise, with the absorption of the hemorrhage and edema, the function of the crossed fibers would also have returned.

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Examination of the visual apparatus in cases of head injury should be made by the ophthalmologist periodically from the time of the injury until several months to a year thereafter. These examinations should include tests of the visual acuity and the status of the ocular muscles, an ophthalmoscopic examination of the fundi, and most important, a careful study of the fields of vision.

From a therapeutic standpoint, examination of the fundus and visual fields offers the most pertinent information. One of the 3 cases of choked discs presented a marked constriction of the visual fields. There is no doubt that relief of the increased intracranial pressure by a decompression operation seemed to prevent complete loss of vision in this case. Such decompression should be carried out in all cases of prolonged papilledema when there are signs and symptoms of increasing visual impairment.[13] This point cannot be emphasized too strongly. As a protection against insidious loss of visual function the writer follows the plan of doing daily manifest refraction, and peripheral field examination on these patients. This plan adds diagnosis and affords the patient ample protection against visual damage.

One must insist upon a daily manifest refraction, if at all possible. It may be demonstrated that in the presence of papilledema the spherical and cylindrical refraction may vary markedly even over a 24 hour period. If the proper correction is not applied false visual acuity readings will be obtained. For example, in a case of a 19 year old girl with otitic hydrocephalus the following corrections were obtained on consecutive days:

7-31-40	R.E. + 2.00 sph.	20/20
	L.E. + 1.50 "	20/20
8-1-40	R.E. + 2.00 " + .50 cyl. x 83°	= 20/20
	L.E. + 2.50 " + .75 cyl. x 95°	= 20/20
8-2-40	R.E. + 2.00 " +1.00 cyl. x 83°	= 20/20
	L.E. + 2.50 " + .75 cyl. x 95°	= 20/20
8-3-40	R.E. + 1.75 " + .75 cyl. x 93°	= 20/20
	L.E. + 1.75 " +1.00 cyl. x 99°	= 20/20

In 2 cases there was evidence of fracture through the optic foramen. When this occurs it is recommended that the optic foramen be decompressed as early as possible in order to preserve vision. Considering all 12 cases in this series, 6 cases or 50%, were at some time during their course in a position to be helped by surgical means. This high percentage emphasizes the necessity for close observation of the eyes in head injury cases to prevent ocular damage.

Ocular changes were consistently present in the majority of cases and offer diagnostic evidence of the extent of the damage caused by fractures and their sequelae. X-ray evidences of fractures were found in only 4 of the 12 cases. Most of the signs and symptoms caused by fractures of the base of the skull arise not so much from the actual fracture as from hemorrhages, edema compressing the nerves in their bony canals, and disturbances of the blood[14,15,18] supply to the various structures, all of which can occur without demonstrable evidence of basal skull fracture by roentgenological means.

In short, the x-rays are totally unreliable, except as to bony changes, whereas, in the large percentage of cases (12 of the 17 cases) eye findings are present to indicate the effect of trauma to the skull. The ocular changes are a more reliable diagnostic aid than the x-rays in determining the extent of damage, the necessity of surgical treatment, and the ultimate prognosis.

A favorable prognosis should be guarded as evidenced in the cases presented. The examination should be extended over a long period of time and at sufficiently frequent intervals to discover signs of expanding or other brain lesions and to prevent loss of vision from such insidious processes.

In conclusion:

1. Seventeen cases of head injury were seen. Twelve of the 17 presented ocular change. Eight of these 12 cases with ocular changes were seen late and for this reason none of the acute signs of ocular changes in head injuries were encountered. Probably because the cases were seen after the acute stages, no fatalities occurred.

2. Two cases of trochlear muscle disturbance were found in this series. This finding is not in keeping with the findings of other authors who report this involvement to be very rare.

3. The 6th nerve was the most commonly cranial nerve involved. This is in agreement with other writers.

4. Seventeen per cent of the cases in this series presented papilledema. A much higher figure than this is usually given. When a choked disc occurs in a case of head injury the presence of a subdural hematoma must be suspected. This conclusion is substantiated by the presence of a subdural hematoma in each of the 3 cases with papilledema.

5. When papilledema is present, a daily refraction and study of the peripheral field must be made to protect the patient from visual damage. As soon as the first signs of loss of vision occur, a cranial decompression is imperatively indicated.

7-31-40	R.E. + 2.00 sph.		20/20
	L.E. + 1.50 "		20/20
8-1-40	R.E. + 2.00 "	+ .50 cyl. x 83°	= 20/20
	L.E. + 2.50 "	+ .75 cyl. x 95°	= 20/20
8-2-40	R.E. + 2.00 "	+1.00 cyl. x 83°	= 20/20
	L.E. + 2.50 "	+ .75 cyl. x 95°	= 20/20
8-3-40	R.E. + 1.75 "	+ .75 cyl. x 93°	= 20/20
	L.E. + 1.75 "	+1.00 cyl. x 99°	= 20/20

In 2 cases there was evidence of fracture through the optic foramen. When this occurs it is recommended that the optic foramen be decompressed as early as possible in order to preserve vision. Considering all 12 cases in this series, 6 cases or 50%, were at some time during their course in a position to be helped by surgical means. This high percentage emphasizes the necessity for close observation of the eyes in head injury cases to present ocular damage.

Ocular changes were consistently present in the majority of cases and offer diagnostic evidence of the extent of the damage caused by fractures and their sequelae. X-ray evidences of fractures were found in only 4 of the 12 cases. Most of the signs and symptoms caused by fractures of the base of the skull arise not so much from the actual fracture as from hemorrhages, edema compressing the nerves in their bony canals, and disturbances of the blood[14,15,18] supply to the various structures, all of which can occur without demonstrable evidence of basal skull fracture by roentgenological means.

In short, the x-rays are totally unreliable, except as to bony changes, whereas, in the large percentage of cases (12 of the 17 cases) eye findings are present to indicate the effect of trauma to the skull. The ocular changes are a more reliable diagnostic aid than the x-rays in determining the extent of damage, the necessity of surgical treatment, and the ultimate prognosis.

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5. When papilledema is present, a daily refraction and study of the peripheral field must be made to protect the patient from visual damage. As soon as the first signs of loss of vision occur, a cranial decompression is imperatively indicated.

6. X-ray studies were significant in only 4 of the 17 cases. In general, X-rays are neither conclusive as to the presence of the fracture nor indicative as to the amount of damage present. Ocular changes would seem to be a much more reliable sign. In the majority of cases, 12 in the present series, various ocular changes were present. Damage to the intracranial structures in cases of head injury is well reflected by ocular abnormalities. These signs are so reliable that, if absent, significant damage is doubtful; and if present, on the other hand, they may be used first, as a means of diagnosing the extent of the existing condition; second, as a guide to what therapeutic procedures are necessary; third, as an aid in prognosticating the ultimate outcome.

7. Seventh nerve damage due to fractures of the temporal bone were associated with disturbances of the 8th nerve in all cases.

8. It is necessary to examine the patient periodically over a long period of time to preclude overlooking any ocular signs of delayed onset.

9. Fifty per cent of the patients in this series were benefited by the neurosurgeon. Ocular changes in most cases furnish the best indication for neurosurgical intervention. For this reason the closest collaboration of the ophthalmologist and the neurosurgeon is advised.

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DISCUSSION

OCULAR MANIFESTATIONS OF HEAD TRAUMAS

Frank E. Burch

Developments of mechanization, war injuries and automobile accidents have been chiefly responsible for increased interest in this subject. Ocular manifestations, which have resulted from the many types of head injuries, including skull fractures, concussions, cerebral hemorrhages and lacerations of the brain, warrant the effort to review their diagnostic value and prognostic significance. Claims for damages, compensation, and pensions arising from visual impairment are constantly increasing. These demand careful examination of every patient suffering a head injury to determine the cause and extent of visual impairment and to exclude malingering.

Eye signs are often a fair index of the extent and prognosis of brain damage following various traumas. As a general rule a patient with fractured skull or brain concussion with early eye symptoms or changes has less chance of eventual recovery than one whose eyes play no part in the clinical picture. Late development of eye changes demand careful diagnostic evaluation especially from a forensic viewpoint.

Study of eye changes incident to trauma was made by Blakeslee[1] of 610 cases of skull fractures. 416 of those showed eye manifestations (78%) in the form of:

1. Ecchymosis and hemorrhage in the lids, conjunctiva, or orbit.
2. Paralysis of extraocular muscles including ptosis.
3. Nystagmus.
4. Pupillary phenomena.
5. Changes in the visual fields.
6. Fundus and optic nerve changes.

1. Emphysema is by no means an uncommon result of head injury but is most often found after direct trauma to the anterior part of the skull. Fuchs[2] attributes it to fracture of the lamina

papyracea through which air penetrates to the areolar tissues of the lids anterior to the orbital fascia. It may involve the orbit, however, through a break in any of the adjacent accessory sinuses. A fracture through the floor of the orbit or its roof, permitting extravasation of air from the antrum of Highmore or the frontal sinus, in basal skull fracture, may result in proptosis of alarming degree. Hemorrhage and ecchymosis may accompany these fractures. Infection very rarely follows. The diagnostic sign is crepitation or "egg shell crackling" on palpation -- especially noticed in the early stages. When emphysema follows ethmoidal fracture it soon subsides under compress bandaging, avoidance of coughing and blowing the nose, and the prognosis is favorable.

Blakeslee observed ecchymosis and hemorrhage in the lids and/or conjunctiva in 106 (17%) of 610 patients; 88 (14.5%) showed lid or subconjunctival hemorrhage-- eighteen of these showing no other sign. Both eyes were involved in 37, one eye only in 69. Subconjunctival ecchymosis without involvement of other lid tissues was present in both eyes in 5 cases; 35 patients showed this sign in one eye alone. Hemorrhage sometimes occurred immediately, in others not until after several hours and rarely it developed only several days following injury. It is probably caused by an extravasation along the nerve sheath, bursting into Tenon's capsule, or directly into the orbital tissues from the line of fracture through the roof of the orbit. It is rarely noted in apoplexy due to hypertension or arteriosclerosis; thus one may infer that fractures through the base of the skull are more prone to produce it -- and even in the absence of corroborative evidence in radiographs, one should suspect basal fracture when it occurs.

2. Extrinsic muscle paralysis generally develops soon after injury and the patient complains of diplopia. Paralysis of the orbicularis is very commonly found with basal fractures through the petrous portion of the temporal bone, the seventh nerve being so very frequently involved in basal fractures. When it occurs late after injury it suggests meningitis. Glaser and Shafer[3] observed ocular muscle involvement in only 7% of basal fractures. One third of these cleared up within 18 months. Upon two occasions within recent years 2 patients have consulted the writer requesting surgical correction of paralytic strabismus following head injury. In each instance the patient was advised to defer operation for a time. One recovered completely within 6 months; the other had a residual esophoria after a considerable longer period and secured comfort with a prism correction.

Naturally the abducens, because of its long straight course is most frequently affected. If it develops paresis late after injury, one should suspect its involvement by inflammatory exudate, by callus, by meningitis or, rarely, by aneurysm. Fracture involving the sphenoidal fissure almost invariably produces paresis of all the extraocular muscles. The 3rd nerve is more frequently involved without its intrinsic branch; inclusion of the latter implies a more serious lesion. The 4th nerve is only rarely involved singly. One should hesitate in placing too much emphasis on the localizing value of extraocular palsies with cerebral or skull injuries. They serve only occasionally to confirm other diagnostic signs. Paralysis of convergence and of associated movements are rare. In Rawling's Hunterian Lectures[13] he proferred the opinion that basal fractures were produced both by direct and indirect violence and that fractures by contrecoup are less common than those produced by direct violence or by Arans' theory of irradiation, viz., that "fractures of the base result as extensions from fractures of the vault, the force following the shortest anatomical route of the base." From the ophthalmological standpoint it is much more important to consider the eye signs following head traumas than theories as to how they are produced.

3. Nystagmus was observed early in only 13 of Blakeslee's series and 4 of these died. Its occurrence in lesions of the posterior fossa is by no means uncommon. It has little diagnostic value.

4. Pupillary changes are the most constant sign observed and were noted in

378 of the 610 basal skull fractures reported by Blakeslee. He found that they frequently became normal in a few days or a few weeks. They were widely dilated on admission in 55 patients, 52 of whom died (94.5%). He did not find that a unilateral dilated, fixed pupil was always apt to be on the side of an epidural hemorrhage, altho Cairns[4] believed great reliance could be placed on the dilation and fixity of one pupil as an indication of the side on which the major cerebral lesion would be found. Cohen[5] believed inequality of the pupils, combined with absence of light reflex, had extremely significant prognostic value. In the 75 fracture cases reported by him the pupils were normal in 35 of the non-fatal cases, but they were normal in only 6 of the 24 fatal cases. One must conclude from the evidence that persistent pupillary fixation, whether dilated or contracted, carries a poor prognosis. When it is due to simple fracture and rupture of the middle meningeal vessels -- it may be merely transitory but should be examined for at frequent intervals in all cases of head injuries. Without associated hemiplegia, it is not of itself an indication for operation in cases of head injury.

One should remember moreover that spastic dilated pupil may follow a direct blow on the eye or from direct injury to the ocular nerves with a basal skull injury -- as well as by a rupture of the middle meningeal artery associated with hemiplegia - or otherwise. Also one should consider whether morphine has been, or should be administered -- and avoid mydriatics in making fundus examination.

5. Visual field examinations soon after head injuries frequently cannot be done and then only by confrontation tests in most cases. It is only after convalescence is advanced, or long after injury that suitable tests can be undertaken. Thus one cannot place very much reliance on reports of examinations made soon after cerebral traumas. Subjective complaint demands investigation, however, as soon as this can be made. Glaser and Shafer found involvement of the optic nerve and defects of the visual field in only 3.5% of their cases. Visual field defects present in their 9 cases were not, as a rule, in cases with fracture. Grant[6] reported a case of a patient aged 51 injured in October 1930 -- who was unconscious for 3 or 4 days, had been confined to hospital and to bed for nearly 7 months, who suffered a broken nose, many contusions of the body, bleeding from the nose, mouth and ears, but who retained good central vision after 4 years. When examined he showed a definite concentric contraction of the fields to approximately 10 degrees, with tubular vision. The optic nerves were dirty gray, with slightly blurred margins, which he attributed to a previous papilloedema. Such a condition could develop either from a cerebral concussion, from increased intracranial pressure due to hemorrhage or from a direct hemorrhage into the sheath of the nerve. These delayed evidences of basal fractures involving the foramen may be explained in still another way. Lillie and Adson[7] reported 2 cases in which late atrophy followed the formation of callus from fracture extending into the optic canal. In each case there was no ophthalmoscopic evidence whatsoever, and the x-rays were negative. There was good vision for a time after the injury. Later there developed visual symptoms and x-ray evidence of pressure upon the nerve in one case due to formation of callus. The examination of the fields of vision showed unilateral central and annular scotoma which might be interpreted as a form of retrobulbar neuritis. In one of these cases decompression of the optic canal did not relieve the condition because it was done after the nerve injury had become permanent. Coppez[8] offers still another explanation of a certain type of field change -- applicable to bilateral cases, or with chiasmal syndromes. He suggests that widening of the distance between the optic foramen, to which the optic nerve sheaths are adherent, may result in the tearing of the sheaths or even splitting of the chiasm in its middle when trauma produces an anteroposterior compression of the skull.

The association of field changes with trauma, aside from those which involve the chiasm, is always difficult. Trauma producing a hemorrhage in the

posterior visual pathways, in the visual cortex of the occipital lobe, may produce visual hallucinations, diplopia, and visual field abnormalities. The most common evidence we can elicit is a contralateral homonymous quadrantic or hemianopic defect frequently associated with nystagmus. Injuries involving the cerebellum alone produce no visual field defects, but, on the other hand, nystagmus is quite constant and asynergia of the various muscle groups is by no means uncommon. It is quite possible, after a cerebral concussion as Grant has shown, for one to find contracted fields, but these instances may also be distinctly attributable to a neurogenic or a psychogenic cause. This is especially true if the question of compensation or monetary recovery for damages is involved.

Not infrequently a patient complains of visual disturbance where a retinal lesion actually complicates a cerebral injury and the fundus examination may reveal evidence of direct retinal damage. Still there is difficulty in the explanation of certain types, such as that reported by McCullouth[9]. In this case after a trivial injury to the head. After the injury the patient was dizzy but resumed work after 10 or 15 minutes. A physician was not consulted until 4 or 5 days later. Complete blindness appeared suddenly, with difficulty in speech, vomiting, and some mental confusion. This lasted 10 days with gradual improvement. Examination two months later showed vision of 5/200 in each eye. There was field contraction down to the 5-degree isopter and the discs were pale. Ten months later vision was 15/50 in each eye but the fields were very slightly smaller. There was no definite picture of atrophy of the optic nerves. McCullough attributed the condition to an occlusion of the vascular supply to the occipital lobes by thrombosis or embolism. He suggested that there was the possibility of a preexisting homonymous hemianopia prior to the accident with subsequent loss of the remaining fields, sparing the maculae, and since the blood pressure was 200/110, this seems plausible. It evidently was not a case of tubular fields.

In the case of a pugilist, aged 22, who came under the writer's observation, following a "punch-drunk" incident -- without fracture of the skull, a homonymous hemianopia was found which indicated definite involvement by hemorrhage of the optic radiations. The fields remained unchanged two years later.

6. The importance of early fundus changes in connection with intracranial injuries is probably overrated. Papilloedema and optic atrophies are by no means early signs. Both of these require time to develop. Both may be produced by subarachnoid hemorrhage along the sheath of the nerve. Optic atrophy may be produced by direct compression independent of hemorrhage. Papilloedema may be produced by direct compression independent of hemorrhage. Papilloedema may develop from continued long pressure. In the 18 cases in which papilloedema was recorded in Blakeslee's series, 7 died.

Cohen[5] did not find a single case of "choked disc" in 5 years' experience averaging 75 annual admissions for fracture of the skull. This statement is peculiar, in view of the fact that papilloedema usually develops from increased intracranial pressure, hemorrhage into the sheath of the nerve, or meningitis. He did, however, find neuroretinitis in 3 and mild papillitis in 5 of the 24 fatal cases and a subsequent unilateral primary optic nerve atrophy in 3 and mild papillitis in 6 of the 51 non-fatal cases.

Regarding the presence or absence of papilloedema, Cairns[4] states that he saw only 7 cases of definite papilloedema in a series of 80 head injuries. Two of these were in children following intracranial hemorrhage, 3 were associated with subdural hematoma, and 2 with cerebral abscess after compound fracture. The earliest observation was 5 days after the accident. Liebriecht did not believe that these could be explained on the basis of hemorrhage into the optic nerve sheath, because the papilloedema did not occur within a few hours of or soon after injury. Cairns thought that

absence of papilloedema soon after injury in no wise excluded the possibility of a large intracranial hemorrhage, removal of which might save life; when it was present he thought the brain should be explored and decompressed. Goulden, discussing Cairn's paper, believed that papilloedema was caused by distension of the vaginal sheaths, but he did not believe that it was the sold determinant factor. Certainly the presence of a papilloedema with intraocular hemorrhage strongly points to a subarachnoid hemorrhage of traumatic origin. The possibility of its association with a small basal aneurysm should be considered. In a case observed[13] there was an unusual involvement of the eyes, associated with a basal fracture of the skull.

Dr. [redacted]., physician, 49 years old, gave a history that on December 7, 1932 after leaving his office one evening he drove home and began to shovel snow off the driveway. About 45 minutes later he was found unconscious outside his garage and was taken to the hospital.

Examination made at this time showed patient to be completely unconscious with sterterous breathing, some spasticity of the left arm, which soon disappeared, and slight cervical rigidity. The pupils were active and equal and reacted to light and accommodation. No ophthalmoscopic examination was made on admission. The general physical examination was negative. Blood pressure was 120/80. No external bruises were found anywhere on the body. Laboratory findings were essentially negative including very complete studies of the blood. Four hours after the accident he regained consciousness, vomited several times, was very restless, and complained of severe pain in the neck. Late that night he complained of loss of vision. The following day vision was slightly better but not entirely normal. A spinal tap was made on the 2nd and 3rd days after admission, at which time bloody fluid was obtained. Eleven days after the accident the record showed that the right pupil was slightly larger than the left. He remained in essentially the same condition until 5 days later when there was considerable pain in the eyeballs with complaint of cloudy vision and cervical stiffness. Four days later (20 days after the injury) when seen in consultation by me, vision in the right eye was nil and fundus details could not be made out on account of an extensive vitreous hemorrhage. Vision in the left eye was also impaired but the exact degree owing to the patient's mental confusion, could not be determined. There was a hemorrhage surrounding the left disc. X-rays which had been taken in the meanwhile showed widening of the right temporal zygomatic suture line, interpreted as indicating a basillar fracture. The ophthalmic diagnosis was "subarachnoid hemorrhage." Apparently, the patient suffered extensive intracranial hemorrhage which extended along the sheath of the optic nerve, breaking through the hyaloid membrane in each eye, producing extensive vitreous hemorrhages bilaterally and a retinal separation in the right eye.

May 31, 1933, 6 months after his accident, there was an extensive detachment of the retina in the right eye extending from the 8 to 2 o'clock meridians with residual vitreous capacity and vision was reduced to hand movements. Vision in the left eye was 20/20, with some vitreous opacities. On Sept. 8, 1933 the detachment in the right eye was unchanged and vision was limited to hand movements. Vision in the left eye was 20/15. There were a few residual opacities in each eye. This patient died of cerebral hemorrhage in Sept. 1939, 6½ years after injury.

Many cases of cerebral apoplexy associated with arteriosclerosis have been examined for internists and surgeons without any very definite evidence of changes in the nervehead or retina other than evidence of arteriosclerosis. Why does one not see more frequent evidence in the nervehead at some stage following intracranial accidents? It would seem logical that one should find much more frequent evidence of intracranial pressure after an intracranial hemorrhage whether following trauma or ordinary apoplexy. The probability is that there are too many "single" examinations and reports and not sufficient follow-up of

these cases. Eagleton[11] emphasized the fact that there should be daily, repeated ophthalmoscopic examinations by an expert ophthalmologist. In the early diagnosis of compression from intracranial injuries, although examination seldom shows papilloedema in the early stages following cerebral trauma, he contends that if the cerebral shock is great, the vessels of the nervehead are very small and similar to those seen with anemia of the nerve and retina; when papilloedema appears it is more often present in cases of secondary edema of the brain, but it depends upon the location and direction of increase in the intracranial pressure by the edema. Eagleton further contended that "injuries confined to the anterior part of the frontal lobe do not produce papilloedema. Injuries of the posterior fossa with occlusion of the iter (foramen magnum) may produce a high degree of papilloedema and that injuries of the cerebral hemispheres with secondary edema produce moderate papilloedema which may disappear quickly or may last for months."

Optic atrophy, -- altho it constitutes only a relatively small proportion of the sequelae of head traumas, is the most frequent cause of serious visual impairment. Most of them are unilateral and fall under the classification of "descending atrophies." The atrophy may not be evident for a considerable period. A case was observed by the writer, of a man who had fallen down an elevator shaft and complained of complete monolateral blindness upon recovering consciousness. There was no immediate fundus evidence but a gradually increasing pallor developed 6 weeks later. The only immediate ophthalmic evidence of basal fracture was a subconjunctival ecchymosis. The x-ray revealed no evidence whatsoever of a basal fracture. Davidson[12] states that even after 3 months one may have a slowly developing atrophy from late hemorrhages or arachnoiditis (independent of a papilloedema).

It is a fact, substantiated by the mechanics of basal fractures and autopsy findings, that ordinarily only one optic nerve is involved by basal fracture. Cantonnet analyzed 225 optic nerve lesions definitely associated with basal fractures and found only 17 (7.5%) with bilateral involvement. A bilateral involvement, therefore, assumes the presence of basal hemorrhage, fracture of the sella or a fracture of both optic foramina. In bilateral involvement it is important to rule out syphilitic atrophy and pre-existing amblyopia. Syphilis is the commonest cause of bilateral atrophies and since Cantonnet found bilateral involvement in only 7½ per cent of cases -- one should be careful to exclude syphilis when both nerves are atrophic, even though trauma may be an inducing factor.

In Glaser's and Shafer's analysis of 225 cases impairment of vision following brain trauma was most frequently attributed to fracture of the bony canal, causing hemorrhage into the nerve sheath or laceration of the nerve or both. They quote the Barkan's report of loss of vision from fracture of the anterior clinoids. It was the Drs. Barkan opinion that cases showing temporal or nasal field changes may be due to a contrecoup contusion of the nerve against the boundaries of the foramen. Eight of Glaser's and Shafer's series showed primary atrophy, only one showed a secondary atrophy. None of these 9 cases developed any improvement at any time. Also they observed that depressed fractures were less frequently accompanied by eye symptoms than in trauma without fractures. It would seem important to enquire carefully into the interval between the injury and the development of optic atrophy.

It is a significant fact that x-ray evidence of basal fractures involving the orbit is exceedingly negative. While the base of the skull is in many respects the weakest part of the skull, fractures show a very definite predilection for this area. It is of still greater significance that the orbital roofs and the optic canals are so very frequently involved. Davidson[12] states that, in basal fractures of the skull, van Hoelder found 90 per cent of orbital roofs and 73 per cent of optic canals involved by fractures. These figures are confirmed by those of La Grange.

Eagleton takes an unusual view regarding fractures, viz., that fractures of the cranium never of themselves kill the patient; that, on the contrary, it very often saves the patient's life. He contends that an injury to the vessels inside the dura which do not permit the blood or oedema fluid to escape is exceedingly more damaging and dangerous than when fracture is present; also that cases of concussion with hemorrhage are much more serious than from fractures themselves. An injury to the head may occur without unconsciousness until some hours later and death may occur from an extravasating intracranial hemorrhage which may occur between the dura and the brain before any eye symptoms have developed. Again a blow on the head may produce headache and vomiting with no x-ray evidence, with papilloedema appearing only after some weeks. An operation may reveal a small subdural hemorrhage with a fracture of the skull, or without the latter in certain old arteriosclerotic patients. He cited such a case which developed double vision and a mild papilloedema. A spinal puncture was followed by coma. When the skull was opened there was a large extradural clot over the middle meningeal artery. Eagleton says it is the patient with delayed, gradual bleeding in which papilloedema may be a late manifestation. He thinks that cases of blindness with retained light perception following a fracture through the orbital roof should be operated upon to release the pressure by removal of bone and control or drainage of the hemorrhage. Anyway, nearly everyone has agreed that papilloedema when present is a comparatively late manifestation after head injuries and that its absence in the early stages does not exclude the possibility of a large intracranial hemorrhage, the removal of which would save life. When a papilloedema is discovered by the ophthalmologist at any stage it is fairly safe to recommend operative exploration or decompression, even though the patient has few other symptoms, for he may be permanently disabled by later developments.

There is still another ocular manifestation of cranial injury which should not be forgotten, viz., the possibility of traumatic intracranial arteriovenous or other aneurysm. Most frequently the mechanism of this lesion is a basal skull fracture. The resulting clinical picture is so characteristic as to be unmistakable. One finds a unilateral pulsating proptosis accompanied by a loud systolic murmur over the temporal region. The bruit is audible to the patient. It can be abolished by pressure over the ipsolateral carotid artery. In addition, one may expect to encounter oedema of the lids and conjunctiva, venous stasis of all ocular structures, complete or partial ophthalmoplegia and in some instances a choked disc. Ligation of the carotid artery is sometimes beneficial in selected cases, especially in patients under forty. The following case was observed by the writer.

In June 1932, Mr. . sustained a blow to the right temporal region from a falling timber. He was knocked to the ground and remained unconscious for about 5 minutes. With the return of consciousness he was able to proceed to his home. The next day there was marked ecchymosis around the orbit, but this slowly disappeared. About three or four days after the accident he noticed a marked convergent squint of the right eye. This remained about one week and disappeared. Some time after this he noticed a swishing noise in the right side of his head. Synchronous with this noise he noticed a feeling of pressure behind the right eye synchronous with each heart beat. Five months after the accident he began to notice proptosis of the right eye. When this first appeared it would partly recede at times. When standing before a mirror he could see pulsating movements of the right eyeball. This also was synchronous with the noise in his head. After he developed proptosis he has also noted pain in the right eye. About April 3, 1933 the protrusion of the right eyeball became progressively worse, and was accompanied by increasing pain in the right eye, nausea and vomiting. He was admitted to the [illegible]rsity Hospitals April 6th.

Examination at the time of admission

to the University Hospitals showed the left eye to be normal. There was a marked proptosis, the reading with the Hertel exophthalmometer being 25 mm. on the right and 13 mm. on the left. The exophthalmos pulsated with each heart beat. There was definite limitation of motion in the right eye, edema of the bulbar conjunctiva so extensive that the lids could not cover the cornea. A bruit was heard over the right frontal region. Vision in both eyes was apparently good. The fundus examination was normal except for dilatation of the veins.

For a week preceding operation by Dr. William Peyton the carotid artery was compressed several times daily, gradually increasing the time until he would stand compression for thirty minutes. On May 16, 1933, under cervical block anesthesia, the common carotid artery was exposed below its bifurcation together with the first portion of the internal and the external carotid. Bulldog artery clamps were then placed on the vessels to control the flow of blood, and the artery was opened at its bifurcation into the internal and external carotid. A strip of muscle, about 6 cm. in length and about 3 or 4 mm. in diameter, was removed from the sternocleidomastoid. A silver brain clip was put on the end introduced into the internal carotid artery and it was pushed up as far as possible with bayonet forceps. The wall in the artery was then temporarily closed. The blood stream was allowed to flow through the internal carotid artery. After this flowed for a short time, perhaps 30 seconds to a minute, the clamps were reapplied and a second strip of muscle from the sternocleidomastoid with a silver clip attached, was inserted and finally a third and fourth strip were also inserted to completely fill the internal carotid artery down to its bifurcation. The internal, the external and the common carotid artery were in turn then ligated.

There was no postoperative reaction until about the 7th postoperative day when a right-sided headache began to develop. This continued approximately 4 weeks, then gradually diminished. The interpretation of the cause of this headache was the reaction and absorption of this muscle tissue with the subsequent extension of the inflammatory reaction to the surrounding dura. X-rays showed the uppermost of the brain clips were at the side of the sella turcica; therefore apparently in the region of the cavernous sinus. He obtained relief from all of his symptoms except for residual paralysis of the right 6th nerve which was present at the time of operation. After 4 weeks the exophthalmometer showed 19.5 mm. on the right eye and 13 mm. on the left. A letter from his local doctor Dec. 21, 1934 stated that the double vision had gradually disappeared and protrusion of the right eye was too slight for him to measure.

There is still another group of cases which deserve mention, viz., birth injuries. Richman[14] has conclusively shown that head trauma during labor is directly responsible for many fundus changes, some of which disappear quickly -- others leave permanent fundus changes and in some cases with visual damage.

There are many phases of head injury to be considered other than eye symptoms but the ophthalmologist should avail himself of every opportunity to examine the eyes following trauma. This should be done over a longer period than is ordinarily afforded -- because of the possibility of delayed signs which may be helpful in diagnosis, prognosis and treatment.

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IV. GOSSIP

Enroute to Grinnell College to spend the day as visiting faculty member in Health Education.

The Rockets and the Zephyrs and what have you, have revolutionized travel. Crowds of people everywhere, getting on and off trains, seeing visitors off, and meeting them. There are many in tears as fine looking young men depart.

Des Moines, on Sunday evening. The streets seem cold and deserted. A stroll through the business district reveals that there are probably more sparrows to the square inch in this town than any other I have visited. As it is just about sundown the racket increases. Evidence that they are not recent visitors can be found everywhere. Perhaps it is the absence of traffic noise which makes their presence so obvious. After sundown the noise abates.

Soft speech in many places. I am told that migrations from the south into Southern Iowa account for it. Students of population trends insist that eventually the entire midwest will be infiltrated by our soft spoken neighbors from Missouri, Tennessee, and Kentucky.

Looking for a place to eat, a favorite occupation. All seem to be full, but here is a cafeteria with a nationally known name. The gentleman carving the meat is always more impressive than the rest. I once knew a student who was a meat carver and he said he was looked up to by the rest of the employees as a man of importance. Most fathers carve night after night without gaining this distinction.

Pleasant young women who carry your tray always make me feel helpless. Although a sign at the entrance of the line warned us that the eye was probably greater than the appetite, I have fallen again. I have always imagined that roast beef is better in Iowa. I should order pork because I have my animals mixed. Interesting people eat in cafeterias. Here is a young couple who silently advance behind two young ladies bearing their trays. She is tall, thin, and neatly dressed. He is shorter, and of the ruddy type. Neither speak nor look to the right or left. Eventually, their food is placed before them and silently they look at it. She approaches her plate by delicately arching her back. He sits looking off into space meticulously conveying the food to his mouth. They seem to be on the best of terms, but they eat their meal in silence.

A local train loaded with railroad men and college boys and girls. Part of the students are going to Grinnell and the others to the University of Iowa. Everytime the train stops one or more frantically rush off for a coke. Two girls purchase ham sandwiches and then decide not to eat them after taking a few bites. There is much walking up and down the aisles and visiting back and forth. The train stops at every station.

Grinnell, Iowa. A cold, damp wind is blowing. The cheery light of the hotel is just across the street from the station. Nothing is quite so warm as a hotel room in the evening awaiting the traveling salesmen. I suppose they come in tired and hungry and like the warmth.

The next morning and my day at the college starts when the director of physical education calls to give me the lowdown. At 9:00 o'clock I speak to the class in personal health. The boys and girls are together for the first time. A young woman walks up and down the aisle anxiously peering at the numbers of vacant seats. Suddenly one of the men puts his foot straight up in the air. In spite of their size (feet) male college students must find that putting their feet up in the air rests them. The girls relax their tension by politely craning at the clock to call my attention to its place on the wall. The attention is good and the crowd seems interested. They use Diehl's Textbook of Healthful Living so I feel at home.

To the Dean's office, where we discuss the question of the need of a students' health service. The president

joins us, also two members of the psychology department, and there is much chatter now about student health.

Noon luncheon at the faculty club. How many times I have gone to these affairs and wondered what the visiting fireman thought. The table is large enough to accommodate all within hearing distance. An opinionated faculty member refuses to eat his greens which reminds me of the statement that the world is full of children who eat candy bars and drink cokes at every chance, women who do not drink milk, men who do not eat vegetables, and oldsters who do not eat meat.

2:00 P.M. I speak to a section of the class in abnormal psychology. The students vary in size and shape, but all appear to be interested in the subject of mental hygiene. Following this class I take a walk. The town is small so that you are quickly detected as a stranger. Some people speak to you, others stare at you. I am to return to the college at 4:30 to address the faculty who are very tired and want to go home. They come (late) because they have been to other meetings. I discuss the importance of health in education in the curriculum.

6:00 P.M. I am entertained at dinner in the Quadrangle. As usual the guests are late and we must file past tables of young ladies waiting to sit down and eat. A bell rings; in some places this is a signal for silent grace. In Grinnell everyone sings the college song. As usual the food is good. Students of the nutritional problems of college students have learned that if they eat at dormitories they will be well fed.

8:00 P.M. We sit after dinner in the lounge and the students go to their rooms. My good friends, the Ravitts, come over from Montezuma and we spend the rest of the evening together. Late at night my train for Des Moines and then Minneapolis.

Grinnell College has an excellent faculty and a good reputation in educational circles. I asked one student if she came there because Harry Hopkins came from there. She said "No. It was because Gary Cooper came from there." I saw Dr. and Mrs. Mussey's daughter from Rochester who is on the faculty. Everyone was most courteous and the day passed pleasantly. I believe that small colleges are more defense minded than larger ones. Their program seems to be more detailed and ambitious, every effort being made to contribute well trained young men and women to national defense.