BILATERAL BLOOD STAINING OF THE CORNEA.

HARRY S. GRADLE, M.D.

CHICAGO, ILLINOIS.

This is the detailed report of a case in which the blood staining of the cornea was bilateral, and followed iridectomy for multiple adhesions of the iris to the lens. Read before the Eye and Ear Section of the Illinois State Medical Society, May 17, 1922.

Blood staining of the cornea has been recognized as a clinical entity for many a year, altho it has been only within the last decade that the nature of the staining elements has been identified. A fair share of the cases of this disease eventually clear, thus obviating the possibility of staining experiments and, as it is impossible to determine a priori which cases will clear, comparatively few are available for chemical analysis of the staining elements during the early stages. The case here reported is of interest from another standpoint, and as far as a search of the literature reveals, it is unique. The findings will be described in somewhat more detail than a mere case report would warrant because of the unusual condition.

August 20, 1919, M. G., female, aged 35. About 6 months ago, inflammation of the right eye followed shortly by similar inflammation of the left. No treatment. Condition gradually subsided and recurred frequently. No etiologic factor known. Gradual decrease in vision. Status Praesens—R. Faint ciliary injection. Multiple posterior precipitates on the cornea. Anterior chamber shallow. Iris somewhat atrophic and pupillary edge very serrated by multiple dense posterior adhesions, which almost entirely prevent dilatation of the pupil. Some exudate on and opacity of anterior lens capsule. Vitreous hazy, but no formed floaters. Fundus is seen hazily, but seems normal. Vision, 0.1. Tension normal.

L. Eye pale. No posterior precipitates. Iris atrophic, but not as markedly so as in the right. Pupillary edge is free only in upper quadrant and bound down elsewhere by multiple posterior adhesions. Anterior capsule clearer than right. Vitreous hazy. Fundus is hazy, but seems normal. Vision, 0.6. Tension normal.

A search for etiologic factors of the old iritis revealed no general pathology beyond a chronic constipation. The usual local treatment of atropin, dionin, and heat was ordered, and K. I. was given internally. The condition of both eyes continued for about a year with alternate subacute exacerbations of the iritis and remissions, complicated by intermittent increase in the intraocular tension. The vision continued to decrease slowly.

Operation.—July 8, 1920.—Bilateral keratome iridectomy (upward). The pupillary edge of the right iris could not be freed and consequently the sphincter was left intact. Both anterior chambers filled with blood which was immediately renewed upon removal.

July 11, 1920. R. Moderately injected. Wound well closed. Anterior chamber full of blood. L. Some edema of lid. One corner of wound blocked with a tag of iris tissue. Tension increased.

July 14, 1920. Both eyes becoming paler. Both corneae show a good window reflex, but are dull reddish brown in color and are completely opaque. Ordered dionin, heat, K. I. internally, and sweats.

July 19, 1920. No change in condition. Started subcutaneous injection of 8 mg. of fibrolysin every other day.

July 27, 1920. Color of right cornea has changed from a deep red brown to a green brown.

July 30, 1920. Color of left cornea now changed to green brown.

August 6, 1920. Beginning clearing of right cornea in extreme periphery.

Sept. 3, 1920. Clearing of right cornea advancing rapidly. Tension normal. Left eye pale, but flushes

easily. Beginning of clearing of cornea in extreme periphery. Tension minus.

Sept. 25, 1920. Nasal half of right cornea has cleared sufficiently so that iris is barely visible. Temporal half still greenish and opaque. Anterior chamber normal. Iris details not clearly visible. Pupil is filled with membrane and is drawn upward. Complete opacity of lens. The left cornea is somewhat flattened. The absorption of the greenish opacity is very slow and the cornea is becoming vascularized. A minute tag of iris is still incarcerated in the angle of the wound. The iris details are not visible. There seems to be a complete opacity of the lens. Tension minus.

Jan. 27, 1921. The right cornea is normal except for a slight area of opacity in the inferior temporal quadrant. Discission of the right lens was performed, very cautiously for fear of further hemorrhage.

The left eye is unchanged from the last record.

Feb. 25, 1921. Extensive needling of the right lens. There was not a great deal of reaction to this operation, and altho there was a slight hemorrhage into the anterior chamber, the blood was promptly absorbed.

June 21, 1921. No change in the external condition of the right or left eye. An unsuccessful endeavor was made to perform a central iridotomy with a Ziegler knife.

July 7, 1921. A broad keratome incision was made in the right cornea and an *irito-ektomie* of the Elschnig type was attempted. Altho no iris tissue was removed, the pupillary edge of the coloboma was freed and a large opening was made thru the membrane that filled the coloboma. Behind this was an irregular mass of lens cortex and capsule.

August 3, 1921. The membrane reformed, contracting the iris down and completely closing the coloboma so that the lens is not visible. The clearing of the left cornea has ceased, despite all forms of local treatment, and organization of the infiltrated cornea is becoming apparent. With a Graefe knife, a 1/3 incision was made thru the right cornea and with the tip of the knife, a puncture and counterpuncture thru the iris was made. On finishing the cut, a fair sized coloboma was produced. Thru this considerable lens cortex was removed with a Jaeger spoon. About three beads of vitreous escaped. The eye recovered from this extensive operation readily and with but little irritation.

Dec. 7, 1921. The right eye was pale. The cornea showed several peripheric areas of scar, corresponding to the extensive operative wounds. In the coloboma were some capsular remains that covered the optical axis. These were severed by a simple needling.

April 1, 1922. Right eye pale. The cornea is slightly flattened and shrunken and shows numerous peripheric scars that are the result of the various operative procedures, but there is no opacity due to the blood staining in evidence, nor can any be discovered with the slit lamp. However, the lymph system of the cornea is very irregular with marked enlargement of the lymph channels. The anterior chamber is moderately deep in the center but is shallow in the upper and outer periphery, where the iris lies near to the posterior surface of the cornea. The iris is irregularly atrophic and shows much scar tissue on its surface. There is a coloboma upward that is closed with a membrane. In the center, corresponding to the pupillary area, is a coloboma of irregular outline, which lies below the former margin of the pupil, the latter having been displaced upward. Thru this coloboma, a clear view of the fundus can be obtained. The vitreous is slightly hazy, but the retina and nerve head present no pathology. The tension of the eye is normal. + 11.00 sp. ⊃ + 3.00 cyl. ax. 40 = vision of 0.6, and with reading correction added, Jaeger 2.

The left eye is pale. The cornea is flattened, and at the upper inner edge of the former iridectomy wound is a small black subconjunctival area of former iris prolapse. The cornea is diffusely opaque, and with the slit lamp

the opacity is seen to be within the corneal stroma, but evidently not involving the anterior or posterior membranes. The individual areas of opacity are not sharply outlined, but merge into one another, with partially clear areas of corneal tissues between. The opacity is greyish white in color. There is a considerable vascularization by deep scleral vessels, four main trunks entering the cornea. There is not much ramification of the vessels. Corneal nerves can be seen at times and these appear to be thickened. The anterior chamber is shallow. Iris details cannot be seen, but the pupil appears to be dilated with a regular coloboma upward. The lens is entirely opaque. The tension is normal and the vision is light perception with good projection.

According to Maghy[9], blood staining of the cornea was first described by Baumgarten[1] in 1883, whereas in reality the first description of the condition emanated from Schmidt-Rimpler[14] in 1875, and was called by him "Apoplexy of the Cornea." The present name was given the condition by Hirschberg[8] in 1896. In all, some 43 cases have been described by the various authors, about half of them accompanied by an anatomic study of the cornea. Undoubtedly many more cases have been observed, but have not made their appearance in the literature.

Up to 1913, Kusama[11] was able to collect the reports of 21 cases, to which have been added since cases by the following authors: Wells[17]; Buchanan[3], 5 cases; Fisher[6]; Begle[2], 4 cases; Maghy[9], 3 cases; Wernicke[18]; Matsuoka[10], 2 cases; Pissarello[12], 3 cases; Elschnig[5]. In practically all of the cases studied anatomically, there was unanimity regarding the presence within the cornea of the highly refractile oval bodies so characteristic of this condition, which were first described by Treacher Collins[4] and shortly after by Vossius[15] and somewhat later by Roemer.[13] These authors all agreed that the bodies were probably derived from the blood elements that found their way into the cornea, but careful inquiry into the chemical nature of the bodies was not possible.

In 1914, Begle[2] made a spectroscopic analysis of a blood stained cornea and established the fact that "In this condition, a solution of hemoglobin is imbibed by the cornea. To the presence of this hemoglobin solution the greenish or brownish discoloration of the cornea must be ascribed." But his method of analysis failed to reveal the chemical nature of the intracorneal refractile bodies and led him merely to the suggestion that "they are elementary noniron containing split product of hemoglobin, despite the fact that positive reaction for a number of the end products of protein disintegration were not obtained."

Simultaneously there appeared an article from the Tokio Clinic by the Japanese Regimental Surgeon Kusama[11], whose investigations led him to the following conclusions: "It is very probable that the blood in the anterior chamber loses its normal characteristics, (this is the theory advanced by Gutmann[7]) and that the hemoglobin which has diffused out of the red blood cells, forces its way into the parenchyma of the cornea by diffusion. From the hemosiderin thus carried into the cornea, which eventually loses its iron, the peculiar refractile bodies arise. From their characteristics, these belong to the group called by Unna[15] "Melanosiderin." Maghy[9] agrees with this view in the main.

In many of the cases described clinically, the entire cornea was not involved in the staining process, while in those in which the entire cornea became opaque, it was very noticeable that there was a fairly clear band of normal corneal tissue at the limbus. This is ascribed by Maghy to the better lymph circulation of the cornea in this area. The staining usually followed perforating or blunt injury of the eyeball; but in some cases was subsequent to an iridocyclitis, particularly of a persistent and chronic type. The condition appeared in from three days to three months after the exciting factor set in, and was almost always dependent upon the presence of more or less blood in the anterior

chamber. In some cases, the chamber was full of blood, whereas in others, there was but two or three millimeters of hyphema. A few cases of entire clearing of the stained cornea have been reported; but in the majority the condition persisted or complications arose that necessitated removal of the eyeball. The lens became cataractous in some of the cases, while in others the nutrition of the lens did not appear to be disturbed.

The case here reported presents the following unusual features: (1) Both corneas became stained with blood at the same time, subsequent to bilateral iridectomy. This procedure admits of discussion. Many prominent ophthalmologists believe in and practice operation upon both eyes at the same time, while others condemn such practice. Many factors, the discussion of which is not pertinent here, must needs be taken into consideration, not the least among which is the economic situation of the patient. As far as a search of the literature shows, this is the only bilateral case on record.

(2) The blood staining appeared on the sixth day after the iridectomy, during which time the chambers were full of blood. The diagnosis was made tentatively on the fifth day, but was not definite until the sixth. This time element coincides with that found in the majority of the case reports.

(3) The intraocular tension, subsequent to operation, was at no time increased in the right eye, and eventually this cornea became perfectly clear.

(4) The intraocular tension in the left eye became increased immediately subsequent to the operation and in this eye the cornea did not clear. In this eye there was a small iris prolapse in one corner of the wound; and whether this was incidental to or causative of the increased tension and subsequent opacity of the cornea, or whether there was some underlying predisposition of that eye toward the unfortunate outcome cannot be told. What is the relation between intraocular tension increased subsequent to the hemorrhage and failure of the cornea to clear?

(5) In both eyes, the opacity of the cornea extended completely to the limbus and involved every particle of transparent cornea. In this fact, this case varies from all heretofore reported.

(6) The right cornea cleared completely, while the left remained slightly more than translucent. Both eyes appeared the same in the beginning, with the single exception of the small iris prolapse in the left, and both were subjected to the same treatment. Incidently, it is very questionable whether treatment had any influence upon the clearing process at all. That difference in regeneration is beyond the possibility of explanation.

(7) The right eye was subjected to six more or less violent operative procedures, involving at different times considerable hemorrhage into the anterior chamber without any further staining of the cornea. Did a localized immunity to the split products of hemoglobin, either with or without iron, come into being? Or did Descemet's membrane develop a closer mesh, that prevented the diffusion of hemoglobin into the cornea; or did the blood that appeared during the later operations in the anterior chamber retain the hemoglobin within the red blood cells, or did the blood in the anterior chamber absorb so rapidly that there was not sufficient time for the diffusion of the hemoglobin into the aqueous and subsequent diffusion into the cornea?

(8) The lenses in both eyes became completely opaque. This was probably due to the nutritive disturbance induced by the inflammatory reaction of the anterior uvea and the consequent exudate.

(9) The remarkable connective tissue forming capabilities of the right iris were evidently a manifestation of the toxic irritability of the tissue.

(10) The ultimate corrected vision of the right eye is better than the vision of that eye was before the hemorrhage and blood staining occurred.

REFERENCES

1. Baumgarten. Archiv. f. Ophthal., vol. xxix, 3, p. 117. 1883.
2. Begle. Archives of Ophthalmology, vol. 43, p. 393.
3. Buchanan. Ophthalmoscope, vol. x, p. 190.
4. Collins, Treacher. Transactions Ophthal. Soc. of U. K. 1896.
5. Elschnig. Klin. M. f. Augenheil., vol. 63, July, 1920.
6. Fisher. Jour. of Ophthal. and Oto-Laryng., vol. v, p. 384.
7. Gutmann. Cent. f. p. Augenheil., 1903, p. 43.
8. Hirschberg. Cent. f. p. Augenheil., 1896, p. 177.
9. Maghy. Amer. Journ. of Ophthal., vol. 2, 1919, p. 633.
10. Matsuoka. Amer. Journ. of Ophthal., vol. 3, 1920, p. 564.
11. Kusama. Klin. M. f. Augenheil., vol. 53, p. 99.
12. Pissarello. Archiv. di Ottal., vol. 25, 1918, p. 125.
13. Roemer. Sammlung zwanglos. Abhand. aus Aug. ii. p. 8.
14. Schmidt-Rimpler. Klin. M. f. Augenheil., 1875, p. 317.
15. Unna. Hdbuch. der Biochemie des Menschen and der Tiere, Ergänzungsband, 1913.
16. Vossius. v. Graefe's Archiv, vol. xxxv, 2.
17. Wells. Homeop. Eye, Ear, Nose and Throat Journ., 1909, p. 497.
18. Wernicke. Rev. Cubana de Oftal., vol. 1, p. 787.

THIRD NERVE REFLEXES.

J. P. WILLIAMS, M.D.

LINCOLN, NEBRASKA

Ophthalmic migrain or scintillating scotoma, disturbance of heart action, profound vertigo and disturbed sleep are here grouped as reflexes. The most important cause is exposure to bright light which disturbs the pupil reflex. Accommodative strain is next and muscular imbalance a third cause. A case is cited in which hyperphoria when uncorrected caused unconsciousness. Read before the January meeting of the Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society, see p. 654.

Some years ago, when speaking of muscle imbalances with an oculist of wide experience, he made the remark to me that he believed that if we could uncover and correct the latent hyperphorias, the lateral muscles would largely take care of themselves. This was at a time when it was quite the custom, and the proper thing, to do tenotomies on the slightest excuse, some reporting as high as twenty or thirty operations on a single individual for lateral imbalance.

In a recent conversation with a colleague of ability and experience, he remarked that he rarely corrected muscle errors mechanically or surgically, and I inferred from his remarks that he made but little effort to uncover latent muscle errors with a view to relieving the symptoms that I shall detail presently. Somewhere between the two extremes there is a proper mean. It is quite easy for any of us to fall into the habit of becoming extreme in any of our routine examinations, and I readily recognize this weakness in myself and I shall gladly accept criticism if my conclusions seem extreme.

TYPES OF REFLEXES.

The class of cases that I wish to attribute to third nerve reflexes are not well defined in our text books. Under what is known as brief scintillating amaurosis, we have one manifestation. Zigzag flickering dark lines appear to pass before the eyes, with more or less complete obliteration of the visual field. The attack lasts usually ten to fifteen minutes, followed by a headache, often severe, and associated frequently with aphasia, marked disturbances of memory and speech. There is often a distinct aura such as tingling of the fingers, or feet, prickling sensation of the chest, numb feeling of the face.

In a second type the flickering blindness is entirely absent, in fact it is in the first type alone that there are any distinct ocular manifestations, and for this reason the second and the third types are more easily overlooked or misinterpreted. The patient feels ill,