tricular hemorrhage. I am glad that Dr. Allen has brought out another point clearly, viz., that when the motor tract is completely cut across by hemorrhage, it is not probable that convulsions will occur. A case of this kind was presented by Dr. Boeck. A man had hemiplegia from cerebral hemorrhage, and convulsions occurred on the side on which the hemorrhage was, but not on the other. My explanation was that the motor fibers were cut on the side of the hemorrhage, and therefore convulsions occurred on the same side, but not on the opposite side, from general irritation of the brain.

DR. A. R. ALLEN, Philadelphia: In answer to Dr. Spiller's question as to the preservation of the motor tract in the cases that had convulsion: One case suffered considerable damage to the entire posterior limb of the internal capsule, which damage I think was late in the hemiplegic attack. I take it for granted that at the time the patient had the convulsions and rigidity, the hemorrhage had not been as extensive as it was found at the autopsy, that being indicated by the clinical history.

ZONULAR OPACITY OF THE CORNEA.*

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Zonular opacity of the cornea is described very briefly in the majority of text-books. Some do not mention it at all, a number dismiss it with a short paragraph, while a very few give it a page.

Nettleship contributed an article of about 25 pages on this subject to the *Archives of Ophthalmology*, in 1879, giving in detail 22 cases seen by various observers. Bock, Graefe, Usher, Best, Leber, and a few others have also investigated the subject quite extensively. The first description was given by Dixon in 1848, followed by Bowman, who went more deeply into the matter. Dixon called it calcareous film of the cornea, finding calcium carbonate and phosphate in the parts removed. Graefe named it band opacity, while Fuchs employs the term given at the heading of this paper. It has been more frequently designated as ribbon-like opacity, or keratitis, and calcareous keratitis.

Zonular opacity occurs in two forms, primary and secondary; the former in eyes otherwise normal, the latter in those blind from glaucoma, iridocyclitis or other intraocular affections. While neither form is common, the primary is much rarer than the secondary form.

It has been described as having the appearance of minute dots crowded together, "punctiform opacities," a gray stripe stretching across the cornea, most marked a little below the center, on the part exposed when the eye is open. The opacity usually begins about the same time at two points near the inner and outer corneal margins, meeting later in the middle line. Some cases have begun centrally. There is always a narrow, transparent zone about the margin.

The progress of the disease is extremely slow, usually continuing for years, although Wells saw one case develop in a few months. Sooner or later both eyes are almost certainly involved, but one may be affected quite a number of years before the other. In some cases there are spells of severe pain, while in others (probably the majority) there is no pain at all. There is no indication of ulceration, the corneal epithelium being smooth and unbroken, in the primary form.

This disease occurs principally in men and after middle age. In 14 cases tabulated by Nettleship, all but 4 patients were over 45 and only one patient was under 30.

The pathology of this affection consists in the deposit of hyaline masses and lime in the upper layers of the cornea, or, as it has been otherwise expressed, "hyaline degeneration of corneal cells that later become calcareous." Bowman's membrane may be the part first affected, and there may be also proliferation of connective tissue.

The etiology is obscure. The location of the trouble in the exposed portions of the cornea has led to belief in a local cause. But if this were true, why is it found in so few persons? Must there not be some predisposing tendency? In gouty subjects, or those with uric-acid diathesis, the nutrient fluid of the cornea, according to Leber, is supposed to be richer in lime salts and these may become deposited by evaporation. Swanzy says that in primary cases the cause of the degeneration is simply loss of vital energy in the cornea from vascular changes, attributed by Nettleship to gout, renal disorders and cardiac affections. Fuchs saw a case in a physician who had blown calomel into his eyes for years, and Toplanski met the disease in hatmakers whose eyes were irritated by flying pieces of hair. Graefe believed that, as the secondary form of this disease followed glaucoma, so the primary form was akin to the glaucomatous process or would eventually go over into glaucoma. As would be expected, these cases are made worse by atropin.

Sight has been restored or improved in some cases by scraping the cornea, the lime deposits coming away in flakes. In other cases iridectomy has proved of great advantage. May advises the use of carbonate of soda after scraping. Sallerbeck advocated the hourly use of 5 per cent. hydrochloric acid, while Nettleship tried nitric acid in one case without effect. Noyes stated that these cases rarely are benefited by any method, and Fox, in his text-book, in 1904, says treatment is of no avail. Guillery, of Cologne, has recently tried ammonium chlorid, beginning with a 2 per cent. solution and gradually increasing to 20 per cent. While his article¹ is headed "Calcareous Opacity of the Cornea," it is evident that he means opacity from lime burns, a very different thing from the subject of this paper.

The following case from my records, the only case of primary zonular or calcareous opacity of the cornea seen in 17 years of eye-work, will serve to illustrate the points of this disease. It is also worth recording on account of its rarity and the gratifying result of operative treatment. Indeed, all cases of such a rare disease, about which there is so little definite knowledge, should be reported fully.

Mr. W. W., born in England; first seen by me Feb. 23, 1897, when he was 34 years old. There was, then, a slight haziness of left cornea to the outer side, and he suffered considerable pain. He was a bricklayer and engaged at this time in the construction of sewers. On the theory that the keratitis might be malarial, he was given quinin, followed by arsenic. (De Schweinitz² described a peripheral annular parenchymatous infiltration of the cornea, of malarial origin, separated from the margin by a zone of clear tissue.) Our diagnosis seemed to be confirmed by the fact that the pain soon yielded to the antimalarial treatment. He was seen only a few times in 1897 and 1898, and then passed from observation for a number of

* Read in the Section on Ophthalmology of the American Medical Association, at the Fifty-ninth Annual Session, held at Chicago, June, 1908.

1. Arch. Opth., Sept., 1907.

2. Philadelphia Polyclinic, 1895.

years. The opacity evidently increased very slowly in extent and density.

September 1, 1904, he returned, complaining of pain in the right eye, the vision of which was normal, but there was a faint, almost imperceptible, haze near the outer margin and rather low down. The opacity of the left eye was now very dense and covered most of the cornea, being surrounded by a zone or rim of clear tissue, very narrow, and nearly the same width in all parts of the circumference, except the upper margin, where it was a little wider. Vision in this eye was reduced to light perception only.

The opacity in the right eye progressed very slowly. Attacks of severe pain occurred at irregular intervals without a particle of redness of the ball. Tension was at all times normal. The pupil was round and responsive to light. He was given quinin and arsenic again, without any apparent effect. Analysis of urine showed no albumin or sugar. He had the appearance of being plethoric, was rather fleshy, with red cheeks. He was not a user of alcohol in any form, but was a great meat eater.

His internal treatment for a long time consisted in the alternate use of salicylate of sodium and iodid of potassium. Pilocarpin and holocain locally, with the addition of dionin a little later, seemed to modify the pain, and after a few months the attacks occurred at longer intervals and then ceased altogether. The dionin had no effect on the opacity. The cessation of pain and the long preservation of good vision led us to believe in the efficacy of the treatment. But it is more than likely that it was simply the naturally slow progress usually seen in such cases. A very weak solution of atropin was tried at the suggestion of one of my colleagues, who, however, advised its discontinuance at the next visit, noting some loss of vision. This accords with Graefe's view that atropin is contraindicated in zonular opacity. (In Nettleship's article mention is made of the use of atropin in several cases with no statement as to its effect further than that the pupils dilated readily.)

My patient was sent to Dr. James M. Ball of St. Louis, with a note asking if this were not a case of zonular or ribbon-shaped opacity, pictured and described on page 335 of his textbook. He replied that it was a typical case, and suggested an iridectomy downward and inward on the left eye. I could not see the advantage of this, as the rim of clear tissue in that direction was so very narrow. It looked to me that an upward iridectomy would be more promising, if any were done, as the rim was wider here than at any other part. But the patient was averse to any operation just then, the vision being still good in the other eye.

The faint opacity seen first in the right eye gradually extended upward and toward the center of the cornea, another band starting a few months later from near the inner margin and extending also toward the center. For a number of months the space directly in front of the pupil was not encroached on. Finally, a very faint haze appeared there, reducing distant vision from 20/20 to 20/40, where it remained for some time, falling to 20/70 for a while and then returning to 20/40. For nearly two years vision was good enough for his regular work as foreman in one of the departments of the Malleable Castings Company. But finally the opacity over the pupillary region deepened and extended a little higher, so that work had to be given up. He could not see enough to go about by himself.

Early in October, 1906, assisted by my colleague, Dr. F. A. Morrison, I proceeded to scrape off a space directly in front of the right pupil, and two weeks later made another scraping, this time assisted by Dr. W. N. Sharp, who examined some of the scrapings under the microscope, finding degenerated cells, probably hyaline, but no lime crystals. This did not flake out, as some have described it. Probably the other eye, in which the disease was more advanced, would have shown this. I regret that we did not try the effect of acids.

The scraping was followed by the use of weak carbonate of soda solution, as suggested by May, but, although we had scraped as much as we dared, there was no improvement of vision. In fact, it was temporarily made worse.

After the inflammatory reaction had subsided, he was sent

to the Indianapolis City Hospital, where I did an upward iridectomy, resulting in 15/40 vision with +6. sph. = +2. cyl., axis 180. This enabled him to return to work and earn as much as ever. This vision has been maintained now for more than a year.

About eight months ago he was helping a fellow workman, a sufferer from trachoma, drop medicine into his eye, and accidentally infected his own left eye, fortunately not the one I had operated on. Prompt treatment with strong solutions of silver nitrate at the office, and 1 to 4,000 bichlorid solution at home, controlled this well, and the good eye has never become infected.

It is interesting to note that there has been some improvement in the unoperated eye. At one time light perception alone existed, and in November, 1906, when the other eye was operated on, there was only faint shadowy perception of large objects, not enough to enable him to get about without being led, but the sight of this eye now is 10/200. Is this due to the internal use of salicylates and iodids? Has the local treatment for the trachoma or the irritation of that disease had any tendency to clear it up? Does the operation on one eye have any effect on the other? These questions are easy to ask, but very difficult to answer. I can give no satisfactory explanation. The opacity certainly looks less dense than formerly, especially directly in front of the pupil. In one of the cases given by Nettleship, Mr. Fairlie Clarke, under whose care the patient was, thought the central clear space in front of the pupil due to spontaneous chipping away of the film.

In view of the good result in this case, and in some of the cases reported by Bowman, Walton, Graefe, Nettleship and others, the statements of Noyes and Fox about the uselessness of treatment sound strange. Possibly they had in mind only the secondary form, occurring in eyes already blind, aside from the opacity, and therefore not amenable to treatment.

In conclusion, I would emphasize the interesting features of this case, the comparatively early age at which the affection began, the rather unusual pain in the first stages and the remedies therefor, the improvement in the vision of the unoperated eye, and the gratifying result of the iridectomy in the other. These circumstances, together with the rarity of this form of the disease and the paucity of the literature, seem to justify the detailed report of this interesting case.

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DISCUSSION.

DR. E. V. L. BROWN, Chicago: So far as I know, no cases of primary zonular opacity have come to anatomic study and therefore the etiology is not clear. The secondary cases are explained on the basis of the hyalin and calcareous degeneration of the anterior part of the cornea and Bowman's membrane; many of these cases have been studied. I have sections from four or five cases and have looked them over in connection with this discussion. The main point, it seems to me, is the clearing up of the left eye. There are two possibilities. First, the gradual clearing of the infiltrate about the areas of calcareous degeneration may have taken place from the absorption of the fatty degenerated infiltration, in the manner contended for by Wossius, who has recorded the findings of fatty substance in the anterior part of the cornea; yet there was no inflammation of this eye at any time. Gradual clearing might occur without any vascularization of the cornea, but any marked change would be accompanied by some so-called inflammatory phenomena. The other cause I have thought of is also accompanied by vascular changes attendant on the trachoma, which appeared in the eleventh year of the case. There is just a possibility that these calcareous infiltrations in the anterior part of the cornea were sequestered and thrown off just as any foreign body would be, and that enough of this took place in

the central area of the cornea to allow clearing. The case was followed up for some time after improvement of vision and there has been no decrease which would seem to make this an impossible explanation. When there has been such a defect, essentially an ulcer, and the epithelium grows over it again, there is invariably the development of connective tissue between the stroma and the depressed epithelium. The exhibition of fluorescein at this time would have determined if there was an ulcer and I would like to ask if it was used at any time for the determination of a defect in the epithelium. I only suggest this as a possible means of the clearing.

DR. L. H. TAYLOR, Wilkes-Barre: I have seen only one case of this kind and that not in my own practice. In this there were two bands, one in each eye; one at an axis of 45 and the other of 135 degrees. It was perfectly symmetrical, all the way of the same width, and the tissue above and below perfectly clear.

DR. EDWARD JACKSON, Denver: I have seen three cases of primary opacity of this kind at comparatively long intervals. The first was seen very soon after I entered practice. I saw this patient for some years and there was no change in the opacity up to death. The second one I saw once or twice at the Wills Eye Hospital and the third I have seen within the last two years. The opacity in all three was characteristic, involving the part of the cornea exposed in the palpebral opening, but showing a transparent position between the margin of the opacity and the margin of the cornea. There was no history of previous disease and no evidence of any other disease.

The last patient had noticed failing vision for seven or eight years; she had not attempted to do any reading or sewing for five years. When she came to me the best obtainable vision was 4/45. She had lived in Kansas and had been much exposed to dust for many years. The film was gray, slightly brownish and consisted of minute dots. The cornea reflex was absolutely perfect and the epithelium perfectly smooth. In all these cases I advised scraping the cornea. The first two did not submit. This one did and under holocain the film was scraped away. The central densest portion scraped away easily; it came away in flakes. Where the opacity was less dense it did not flake that way, and was more difficult to remove. The operation was done under local anesthesia and without pain, but was followed by severe pain, such as we have from abrasion of the cornea, which lasted some eight or ten hours, and gradually passed away. It was relieved a good deal by instillations of holocain. At the end of a week vision had fallen from 4/45 to 4/100. At the end of the second week, however, it began to improve, and at the end of three months the vision in one eye was 4/9 and in the other 4/12. The opacity appeared to be chiefly on the under surface of Bowman's membrane and consisted of isolated dots. I could not get any certain evidence that there was any calcium carbonate in the flakes.

DR. F. C. HEATH in answer to questions: Fluorescein was not used because the corneal epithelium was smooth, and I saw no indication for its use. There was no pannus following the trachoma. The film was grayish in color. I observed the same regularity of which Dr. Taylor speaks and the film was transverse.

Treatment of Hydrophobia in Ancient Times.—The *Tribune Médicale*, May 30, describes the old practice of immersing persons in the sea as a preventive and cure of hydrophobia, citing the legend that Euripides was saved by this means after having been bitten by a rabid dog. A number of cases are quoted from the historical records of the eighteenth century in which persons sought the sea as a preventive after having been bitten. One old chronicle comments on the death of an English lord from hydrophobia notwithstanding that he had been duly immersed in the sea: "This proves that there is no remedy for the imagination, and that people are cured less by the sea than by their imagination that they are cured." We learn from the case of a page of M. de Nevers, in the days of Henri IV., that when the thalassotherapy failed it was the practice to put a bullet through the head of the victim when the hydrophobia had declared itself. The page in question "died in peace just as they were on the point of firing a harquebus at his head."

Clinical Note

A TYPICAL CREST-SHAPED SEPTAL DEFLECTION,

WITH PRESENTATION OF SOME NASAL INSTRUMENTS.*

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In a deflected septum removed from a cadaver, the deflection was of the variety I have classified as crest-shaped (Fig. 1), because the apex of its angle of convexity consisted of a sharp, cartilaginous ridge ascending upward and backward parallel to the anterior border of the vomer, but lying about three-eighths of an inch below it. This variety of deflection I regard as the fundamental deformity in nearly all nontraumatic deviations, the anatomic condition consisting of a curving over of the incisor and superior maxillary crests and the anterior portion of the vomer into the naris of the convexity, associated with an overriding of these bony parts

Fig. 1.—Lateral view of the nasal septum showing typical crest-shaped deflection with sharp cartilaginous crest, d; l, perpendicular plate of the ethmoid bone; v, vomer presenting convex surface of bony deflection towards observer; c, cartilage of the septum overriding the vomer downwards and backwards. The dotted line indicates the position of the anterior border of the vomer covered by the overlying cartilage. Drawn from specimen.

by an extension of the septal cartilage downward and backward, the lower limit of this cartilaginous extension forming the sharp crest referred to. It is this crest with a strip of the underlying bone which is so often sawed off, with but partial freeing of the patient's naris, while the more important obstruction due to the body of the deflection, of which the crest merely forms the sharp angle, is ignored. The cartilaginous strip mentioned is always found roughened on its inner surface where it is attached to the side of the bony septum, is present in most deviations of the nasal septum and has to be lifted off from the underlying bone before the latter can be found and resected. I regard the deviation in the bony septum as the main feature in the majority of non-

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