PRIMARY HEMORRHAGIC GLAUCOMA, WITH PROBABLE SYMPATHETIC IN- FLAMMATION

A CLINICAL AND MICROSCOPIC STUDY OF A CASE *

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Pathologic investigations on hemorrhagic glaucoma have been reported in considerable number, but, owing to the peculiar combination of conditions, this case is of interest and we can, I think, make some useful deductions from it. It points out that a broad iridectomy freeing the filtration angle is not always a preventive of glaucoma, although iridectomy is the best method of treating acute glaucoma. It further indicates that diminished depth of the anterior chamber and narrowing or closure of the filtration angle are not absolutely essential for the production of glaucomatous symptoms, but that, perhaps, hypersecretion and changes in the character of the fluids of the eye impeding proper filtration are sometimes concerned in the onset of glaucoma.

History.—On the evening of Jan. 14, 1908, I was called to see M. R., a man aged 31, of Jewish extraction. The day before the patient went to Milwaukee on a business trip feeling as well as usual, never having had any severe illnesses or any previous eye trouble. Twenty-four hours prior to coming under observation he suffered with a sudden severe pain in the left eye and within two hours the globe was intensely injected and vision lowered so that he could barely make out his hand. After a long anxious night a well-known Milwaukee ophthalmologist was consulted in the morning, who told the patient to get back to Chicago as quickly as possible.

Examination.—The left eye presented all the cardinal symptoms of acute glaucoma. The globe was brick-red, the scleral vessels full and the cornea steamy with diminished sensibility. The anterior chamber was equal in depth to its fellow and contained about two minims of dark-colored blood. The pupil was fully dilated and immobile, with the characteristic glaucoma reflex. The tension was plus 3. An ophthalmoscopic examination was impossible, owing to the corneal haziness and cloudiness of the vitreous. The vision was reduced to counting fingers at two feet. To the temporal side of the globe there was a large subconjunctival hemorrhage extending to the limbus and at places in the vicinity the cornea appeared to be studded with minute hemorrhages. The field of vision was about normal. The pain in and about the eye was very severe. The fellow eye was absolutely quiet and normal in every detail; vision equaled 6/6 plus. A careful inquiry into the patient's previous history failed to ascertain any cause for the attack. His habits were the best; he drank and smoked only very moderately. There was no venereal history. It was evident that one had to deal with a case of primary glaucoma of the hemorrhagic type. As iridectomy was not advisable, the usual medical remedies, eserine (physostigmin), one grain to the ounce, combined with cocaine, dionin (ethyl-morphin hydrochlorid) 5 per cent., leeches and hot fomentations had to be relied on.

January 15, 8:30 a. m.: The patient had a good night and was now free from pain; T + 3; cornea hazy; anterior chamber of normal depth; pupil widely dilated, but hyphema less marked.

January 15, 8:30 p. m.: Condition was the same, except that the blood in the anterior chamber has absorbed, and cornea clearing and sensitive. T + 3; vision equaled fingers at six feet. On careful repeated chemical analysis and microscopic examination the urine proved normal.

January 16, 8:30 a. m.: The pupil was still dilated, but tension slightly lower than the day before. Cornea was bright and vision equaled 3/60. Ophthalmoscopy was now attempted, but owing to the cloudiness of the vitreous, the details of the fundus were not sharply defined, although the disc could be seen and appeared hyperemic, swollen and the vessels full. The retina could not be studied.

January 16, 8:30 p. m.: Since noon the patient had had some pain; the cornea was again steamy and vision reduced to fingers at two feet. The field was good. Paracentesis of the cornea was advised and concurred in by Drs. Casey Wood and Thomas Faith. On account of the previous hyphema and the still present subconjunctival hemorrhage, iridectomy was advised against.

January 17, 8:30 a. m.: The patient's condition was the same. Under 10 per cent. cocaine anesthesia, the anterior chamber was slowly emptied with a paracentesis needle, and immediately on the escape of the aqueous small rivulets of blood made their appearance on the iris. Tension was slightly lower than normal, and iris somewhat contracted. Physostigmin was instilled in both eyes and the eye which had been operated on was bandaged.

January 18, 8:30 a. m.: The bandage was removed. Anterior chamber reformed; cornea clear; pupil dilated ad maximum. T + 3, and vision equaled fingers at one foot. Hemorrhages were absorbed. A posterior sclerotomy was now made between the superior and external recti muscles under 10 per cent. cocaine, and about 10 minims of a clear, straw-colored fluid escaped. The eye was very soft. Calcium chlorid in fifteen-grain doses was given every three hours, with a view of increasing the coagulability of the blood. In addition, nitroglycerin was given, 1/100 grain, four times a day.

January 19, 8:30 a. m.: T + 3. The eye was free from pain; the pupil fully dilated; the field good. The vision equaled fingers at one foot. Small hyphema present and the cornea was slightly hazy.

January 19, 2:30 p. m.: Hemorrhage disappeared. Dr. W. H. Wilder in consultation agreed with me in still holding out against an iridectomy. A combined paracentesis and posterior sclerotomy between the inferior and external recti muscles was made, under 10 per cent. cocaine. Directly after the sclerotomy, bright red blood flowed into the lower part of the anterior chamber, appearing to come from the region of the iris root. The eye was very soft.

January 20, 8:30 a. m.: The bandage was removed. The cornea was clear; hemorrhage absorbed; pupil widely dilated; no pain.

January 21, 9 a. m.: The patient's condition was unchanged. The use of physostigmin, 3 grains to the ounce, was begun.

January 21, 2 p. m.: T + 3. The pupil was slightly contracted; now about 4 mm. in diameter; was still free from pain excepting after instillation of physostigmin. Blood pressure and coagulation time were taken by Dr. Adolph Gehrmann of the Columbus Laboratories, who found blood pressure 95 mm., pulse 80, coagulation time two to three minutes. The blood pressure was taken every day following for a week, and never went higher than 115 mm.

January 22, 8:30 a. m.: Condition was as noted yesterday afternoon.

January 23: The pupil about the same; tension slightly lower. Physostigmin was increased to 4 grains to the ounce.

January 24: Pupil was 4 mm., but there was a perceptible diminution in tension; cornea clearing.

January 25: The condition was unchanged.

January 26: The cornea was bright. The patient counted fingers at three feet; there was no change in tension from that last noted. Owing to the cloudiness of the vitreous, a view of the fundus could not be obtained.

January 29: The pupil was 2 mm.; tension + 1.

January 30: The tension was normal; field was taken (Fig. 1) and the eye transilluminated for a possible tumor, but found negative. The patient counted fingers at one foot.

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February 4: The eye was nearly white; the tension normal; vision, 6/60; the pupil 3 mm. in diameter. This condition remained until February 11, 1908.

February 12, 8:30 a. m.: At about 4:30 p. m., February 11, there was some pain in the eye, which continued during the night, but the patient attributed it to the use of physostigmin. Examination now showed a new attack. The cornea was steamy; tension +2; pupil about 4 mm. in diameter; the anterior chamber of same depth as its fellow. Iridectomy was now advised as a last resort, and the patient made aware of the possible danger. Operation was done under 10 per cent. cocain anesthesia, and a broad iridectomy performed with a Landolt keratome without a mishap.

February 13, 2 p. m.: The patient had been very comfortable; was free from pain. The bandage was removed; the cornea was clear; the anterior chamber reformed. Tension was slightly subnormal. The pupil was 4 mm.

February 16: Tension was normal; the eye quiet; physostigmin was reduced to 1 grain to the ounce.

February 17: The condition was the same. With the ophthalmoscope numerous large clots were seen floating in the

lounge, numerous fine dots were seen on Descemet's membrane, which stained deeply with fluorescein. Right eye vision equaled 6/5 minus. Accommodation was impaired. The disc was slightly hyperemic and the vitreous hazy. The left eye tension was normal. The vision equaled fingers at four feet. With these conditions, enucleation of the left eye was immediately advised and done under ether anesthesia.

March 14: Treatment was given, consisting of mercurial inunctions, pilocarpin sweats, sodium salicylate, dionin 5 per cent. in right eye.

March 28: Exudate was absorbing on Descemet's membrane; there were large masses of exudate in vitreous. The disc was slightly hyperemic. Tension was normal. Vision equaled 6/7. There was a large subconjunctival hemorrhage in stump of enucleated eye.

April 18, 1908: Right eye vision was 6/6 plus; a few very fine spots of exudate were still visible on Descemet's membrane, with the loupe. The vitreous opacities were smaller; eye accepted + 0.75 D sphere; vision equaled 6/6.

February 23, 1909: The external eye was normal; the vision equaled 6/5 —. Some vitreous opacities were still present and a few very fine dust-like opacities were still remaining on Descemet's membrane.

MICROSCOPIC REPORT

The eye was given to Dr. E. V. L. Brown for anatomic study, and he has submitted the following report:

"The specimen (Lab. No. 264) was fixed in formalin followed by Zenker's fluid, and was cut in an anterior and posterior half, the anterior half sectioned throughout the area of the coloboma and the posterior sectioned horizontally. Some 400 serial sections of each half were made and every fifth or tenth stained.

Fig. 1.—Field of vision for white (left eye), taken Jan. 30, 1908. Bright morning. Pupil 3 mm. in diameter. Disc, 1 cm.

vitreous, but nothing further could be distinguished; the patient was given syrup of hydriodic acid.

February 27: The condition was the same. The vitreous had cleared so that the location of the disc with vessels could be made out. The vision equaled 6/60.

March 1: I did not see the patient from February 27 until this day. Tension was normal, but there was a broken ring of pigment on the anterior capsule, and one corner of the nasal pillar of the coloboma adherent to capsule. Physostigmin was stopped.

March 11: There was no change in the eye until this day, when a dust-like exudate was found on Descemet's membrane. Tension was slightly above normal; cornea clear.

March 12: The patient had pain during the night. The cornea was steamy; tension +2. The use of pilocarpin was begun.

March 13: Tension was normal. The cornea was clear; keratitis punctata well marked.

March 14: Up to this date the right eye had been absolutely free from any discomfort, but this day there was a fine ciliary injection, some lachrymation, and a feeling of discomfort. Tension was normal. On examination with a

Fig. 2.—Anterior half of left eye as seen from behind (exact size).

"Seen from behind, the anterior half presented the appearance sketched in Figure 2; the lens is clear, the pars plicata and corona ciliaris are *in situ*, the processes and plicæ apparently normal and regular in arrangement. An accurate sketch of the position and extent of the operative coloboma area was made, because, clinically, a perfect and entirely effective iridectomy had been performed. No portion of the root of the iris could be seen behind the opaque corneal limbus, either before or after the eye was taken out; yet, as shown in the sketch, the gross specimen shows that a good 0.5 mm. of the root of the iris had not been removed by the operation.

"*Microscopical Study.*—In a section through the center (section 205; see photomicrograph, Fig. 3) the curvature of the cornea and sclera are seen to be about the same. The cornea shows a tendency to collapse, and the center is slightly thinned to 0.5 mm. The epithelial layer is narrowed and elevated into empty vesicles in a number of sections; some of these (as section 110) have ruptured and left Bowman's membrane exposed. The outer cells are compressed and keratinized; the inner cell nuclei are thrown slightly forward or 'stilted'; many leucocytes are seen among the cells, especially in the upper portion of the cornea. Bowman's membrane is everywhere a trifle wider than normal and shows numerous delicate nerve canals as cross-striations. The body of the cornea is normal, although the very center takes a lighter eosin stain than usual. A narrow densely nucleated scar, corresponding to the site of the corneal section for the iridectomy, lies at the upper limbus; it commences just above the end of Bowman's membrane and runs obliquely from without, inward and downward at an angle of 45 degrees from the end of Bowman's membrane to a point 2.3 mm. central to the upper end of Descemet's membrane. Here at its inner

end the cut layers of the cornea gape slightly, so that the anterior chamber lays forward into the back part of the cornea. The defect has the form of a narrow wedge, and both lips are bordered by the curled-up ends of Descemet's membrane; the surface of the triangle is covered by a single layer of new endothelial cells directly continuous with those lining the anterior chamber. The anterior end of the scar grades off into a mass of proliferating endothelial and stroma cells just beneath the limbus epithelium. A closely knit matrix of new stroma cells has definitely healed together the anterior three-fourths of the wound. In more temporal sections (see section 185) the defect produced by the operation extends forward as a narrow sinus one-half the way to the surface of the cornea. But the lips of the wound anterior to the sinus are so firmly knit together that no 'filtration cicatrix' could have been present. In sections still more temporal (such as section 110) several well-filled vessels of small size are seen running through the scar and are surrounded by considerable numbers of round cells. In all sections through the scar the surface epithelium is normally reformed, but nowhere dips down into the wound tract; the final repair has been effected entirely by proliferation of the fixed corneal cells. The lower one-half of Descemet's endothelium is thinned and the cells in part disintegrated; many clumps of small round cells and pigment detritus are found on the posterior surface of the cornea;

above that the iridectomy clinically was a very peripheric one: yet accurate measurement of the shortest portions of the stump shows that over 1 mm. of iris, measured from the circulus major to the cut end, is still present (section 143). As stated before, the angle does not show any accumulation of round cells or plastic material in any section; a heavy eosin stain does not bring out more fibrin or albuminous material than would be expected in aqueous reformed after evacuation. What fibrin is present is almost entirely in the lower half of the chamber and disposed along its walls.

"The sinus angle above is wider than that below; the cut end of the iris stump does not reach the equator of the lens anywhere and consequently the iris above has been displaced backward into a position that is even more nearly at right angles to the optic axis than in a normal eye. The iris below is thrown forward so as all but to touch the base of the cornea in many sections; indeed, some anterior root synechia to the back portion of the trabeculum corneosclerale has taken place in the sections which go through the angles just a little temporal to the iridectomy area; sections still more temporal are again perfectly free from any evidence of union between the periphery of the iris and the back part of the trabeculum corneosclerale. Both surfaces are perfectly smooth and sharply defined in all except a few peripheric sections (as, for instance, section 49) which show good-sized pigment masses.

Fig. 3.—Section of left eye (photomicrograph, magnified 17 times). The upper limbus with the corneal incision and iridectomized iris lie to the right. One notes that: (1) The cornea is thinned; (2) the lens is thrown forward; (3) the chamber is shallow; (4) the sinus angles are open; (5) the trabeculum corneosclerale is infiltrated on each side; (6) the canals of Schlemm are open; (7) pigment epithelium is detached from the iris pupil zone and adherent to the lens capsule; (8) the iris stump above is 1 mm. long.

some sections (Cf. section 110) showing as many as six clumps in the lower angle of the anterior chamber.

"The conformation of the anterior chamber and the sinus angles deserve special description. The entire lens system and iris body have been displaced forward, narrowing the chamber as a whole; indeed, the anterior pole of the lens rounds forward through the pupil to such an extent that the space between the anterior capsule and the cornea is reduced to two-thirds of a millimeter. The plane of the iris below is correspondingly thrown forward, and makes an angle of 25 degrees with the transverse axis of the eye in comparison with a normal angle of about 15 degrees. Yet the sinus angles are patent in all sections both above and below, and the fluids of the chamber have free access to the surface of the trabeculum corneosclerale; the angle above is reduced to a narrow slit but is free from cells. The iris below measures 3.23 mm. from the pupillary margin to the circulus major, whereas the iris above has been in a large part cut away by the iridectomy; the iridectomy extends from section 49 temporal to section 213 nasal; the columns are nearly parallel and nowhere in contact with the wound. The stump lies entirely behind and peripheric to the corneal portion of the corneoscleral limbus, corroborating the statement made

"The anterior half of the iris, trabeculum corneosclerale and canals of Schlemm are infiltrated with round cells; those in the iris are almost exclusively of the plasma cell type and only moderate in amount; the posterior layers of the iris do not show any considerable amount of cell increase; the stroma pigment cells are considerably disturbed toward the iris root both above and below. In most sections the infiltration about the canals of Schlemm is so dense or the cells so changed that only round heavy-staining nuclei are seen and the type of cell can not be made out. Despite this, the lumina of the canals are free and lined by a well-preserved endothelium in most sections and filled with blood in many; the position of the canals can always be readily made out. Further evidence of plastic inflammation of the iris is furnished by deposits of pigment detritus, clumps and short strips of pigmented cells on the anterior surface of the lens capsule. Their exact location and the relations to the pupil border of the iris are of great importance in any attempt to establish an order of pathogenesis in the findings.

"In the sections which go through the temporal (sections 60-120) or nasal (sections 144-205) portion of the lens capsule the pigmented cells lie in about the center of the portion of the capsule shown and these (vertical) sections go through two

areas, each about 5 mm. wide, in the horizontal plane. The central sections (120-144) show no deposits on the center of the part of the lens capsule concerned, but do show deposits more toward the equator of the lens; these central sections go through a pupil area of about two-thirds of a millimeter in diameter. These evidences of posterior synechia, therefore, extend through an area 5 mm. in width and are absent through a zone of two-thirds of a millimeter corresponding to the size of a normal pupil. Furthermore, there was no actual posterior synechia present at the time the eye was removed, because no sections show adhesion between the iris and lens; the slight expanse of the iris below (only $3\frac{1}{3}$ mm. overlap the lower equator of the lens for a millimeter in section 133) means that the pupil was well dilated at the last. One may conclude, therefore, that there was first formed a central posterior synechia in a zone corresponding to the position of the sphincter at a time when the pupil was small, and that this was later broken up by dilatation of the pupil and did not reform. These facts are in keeping with the clinical history.

"The ciliary body is markedly atrophic; the processes are all directed forward and much flattened down against the muscular portion of the body. The muscle has lost its triangular form and instead of a prominent angle on the inner anterior face, it presents a very flat curve extending forward from the region of the equator of the lens forward to the canal of Schlemm. No cell increase is found in any part of the ciliary body, and the stroma does not show any increase of connective tissue; a few of the processes are matted together at their bases; the apices are all free and there are no cells, fibrin or new tissue in the zonula ciliaris or anterior vitreous.

"The chorioidea is *in situ* and absolutely without any evidence of active or past inflammation, except where one of the sclerotomies was performed. Here the scleral wound is well healed externally, though gaping slightly internally; the cut ends of the chorioidea and retina are closely in apposition or slightly prolapsed in to the inner angle of the wound; the chorioidea shows round-cell infiltration for one-half to three-fourths of a millimeter only on each side of and in the wound and in a few sections only (section 289 and section 320); here the sclera is slightly ectatic. The chorioidea is not at all thickened by the process; the infiltration is never more than eight or ten cells in width from within outward; no epithelioid cells can be found; and the whole process apparently represents only normal wound healing after posterior sclerotomy. The retina in this area is atrophic and fused with the chorioidea into a dense atrophic scar permeated by broken-down pigment epithelium. Several *Drusen* bodies are found in the anterior part of one section, 380.

"Elsewhere the retina was detached in preparation and shows no evidence of inflammation aside from mantles of round cells about some vessels; it is markedly thinned. The vitreous is slightly shrunken in bulk, but otherwise quite normal aside from a considerable amount of fresh blood near the ora serrata below.

"One hundred and ninety serial sections through the portion of the optic nerve immediately behind the disc and including the entire course of the central vessels within the nerve show no evidence of inflammatory or atrophic changes in the nerve substance, and no changes in the blood vessels.

ANATOMIC SUMMARY

"We have therefore an active chronic iridocyclitis, healed operative corneal and scleral wounds, and an operative coloboma of the iris above. The eye shows effects of increased intraocular pressure in the displacement of the iris and lens system forward, in the flattening of the ciliary body and in the marked atrophy of the retina. No anatomic or histologic evidence of sympathetic inflammation is present. The presence of the chronic iridocyclitis throws doubt on the clinical diagnosis of primary glaucoma, and it is impossible to say from the anatomic findings that an anterior uveitis without clinical signs had not preceded and caused the glaucoma."

The conclusions deduced from the microscopic examination deserve careful consideration on account of

their differences from the clinical findings. First, as to the non-existence of anatomic or histologic evidence of sympathetic inflammation: Although the changes in the iris, ciliary body and chorioid, as described by Fuchs, were absent, nevertheless the clinical symptoms were those of a sympathetic inflammation. Whether this was due to some agent entering the exciting eye as the result of the iridectomy, paracentesis or sclerotomy, I am unprepared to say. Until the pathologic anatomy of infiltrative-proliferative uveitis as described by Fuchs is found in all cases, and more firmly established as a pathognomonic finding, our diagnosis must be based on the clinical manifestations of the disease. The mere fact that the anatomic findings in this eye show a chronic iridocyclitis does not prove that it preceded the glaucoma and made it a secondary glaucoma, since the clinical precipitates on the posterior layer of the cornea and posterior synechia were not present until eight weeks after the onset of the primary glaucoma; but they did follow just exactly twenty-eight days after the iridectomy, and may very well have caused it. The patency of the sinus angles in all sections, and the lumina of the canals of Schlemm, free and lined by a well-preserved endothelium in nearly all sections, is striking and deserves careful consideration. That a gross obstruction of the filtration angle at any point in the filtration zone accounted for the increased intraocular pressure is not at all probable, and the fact that another attack followed iridectomy seems to be both pertinent and conclusive. It is more reasonable to assume, according to Priestley Smith, Bulson and Troncoso, inasmuch as primary glaucoma in a certain group of cases is dependent on disease of the blood or blood vessels causing an excess of fluid in the vitreous chamber, the exit of which is prevented by the swollen ciliary processes, or on alteration in the constitution of the intraocular fluids with failure in drainage, that the acute glaucoma here was dependent primarily on some vascular disturbance. As a result of this vascular disturbance, possibly due to some form of toxemia, there is diminution in the circulation of the blood favoring the occurrence of hemorrhages which result rather from transudation than from ruptured vessel walls.

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ABSTRACT OF DISCUSSION

DR. S. D. RISLEY, Philadelphia: I have no adverse criticism to express concerning the treatment pursued, but I think that there is opportunity for another opinion than that given in the paper, as to the nature of the case. This man was free from arteriosclerosis. He was a healthy man, 31 years of age. The attack came on suddenly, without clinically recognized cause, and throughout the subsequent history of the case the different manifestations are, to me, unique. It is only when we come to the laboratory study of the enucleated eye that we get any light on its essential nature. The hemorrhages, which occurred, it should be borne in mind, only after the increase in tension came on, throw some light on the case. Whether the intraocular hemorrhage shall occur, as has been suggested here, during the period of low tension, or whether it shall occur with very high tension depends altogether on which set of vessels furnish the hemorrhage—the arteries or the veins. If we let up the tension very suddenly in a glaucomatous eye with high tension, by iridectomy or otherwise, it is natural that we should expect hemorrhage from a rupture of the retinal arteries or arterioles, whereas the higher the tension the greater the engorgement of the venous system of the eye becomes and therefore venous hemorrhage is more and more imminent. This man had no arterial disease, but the pathologic study of the ball shows that there was unques-

tionably a chronic iridocyclitis, *e. g.*, there was a deposit on the anterior capsule of the lens, left undoubtedly by delicate synechiae which were torn loose with the dilation of the pupil when the high tension came on. It was at this point in the clinical history that the blood leaked through the pupil into the anterior chamber, pressed forward by the increase of tension, the hemorrhage being a leakage from the venous system of the eye. I think, therefore, that we are justified in regarding this case, not as one of hemorrhagic glaucoma, associated with sclerosis of the retinal arteries, but glaucoma with hemorrhages from the venous system of the eye. It is therefore separated as wide as the poles from hemorrhagic glaucoma with arterio-sclerosis. It is important that we distinguish between them. The diagnosis should be changed to glaucoma with hemorrhages.

DR. WALTER L. PYLE, Philadelphia: Through the courtesy of Dr. Frank, a year ago I had an opportunity to examine this patient. From the clinical history and study of the physical signs at that time, I believed the case to be one of sympathetic affection, and despite the absence of the characteristic microscopic findings described by Fuchs, I am still of that opinion.

DR. H. GIFFORD, Omaha: It seems to me that it is altogether premature for us to lay down a microscopic diagram of what we consider to be the findings of sympathetic ophthalmia and say that if the clinical picture, no matter what it is, does not give microscopic findings corresponding with the diagram it is not sympathetic ophthalmia. Remember we do not know what sympathetic ophthalmia is. The idea is wrong. Here is an eye that had abundance of chance to get infected. Several openings had been made. We find the evidence of iridochoroiditis. Whether it was of a kind to produce sympathetic ophthalmia we do not know. We have to diagnose sympathetic ophthalmia from the clinical picture and not entirely from the microscopic findings at the present time.

DR. E. V. L. BROWN, Chicago: I rise to defend my anatomic findings and their significance in this case. First it is true, as Dr. Gifford says, that we do not know the organism which causes sympathetic inflammation, but this does not lessen the value of anatomic facts. The same is true of sarcoma, yet we know when sarcoma is present and when it is not, and from the anatomic facts alone. We must accept Fuchs' statement as to whether the anatomic findings in sympathetic ophthalmia are constant or not. His statement is that in 181 eyes he made a clinical diagnosis either of sympathetic inflammation or of sympathetic irritation and from the anatomic study of these cases made up his mind as to a characteristic anatomic finding. Then, without reference to the clinical histories, he divided the specimens into two groups and, so to say, said of the first group: "Each of these 25 eyes caused inflammation of the fellow eye." And of the second group: "None of these 157 eyes caused inflammation of the fellow eye." Referring then to the clinical records, he found that 24 of the 25 eyes had caused sympathetic inflammation and that none of the 157 had done so. Now, it is true, this is not a constant finding, but in my opinion one must either doubt Fuchs' word or admit he has made a most remarkable contribution. These findings have been corroborated by others and in a recent number of the *Archives of Ophthalmology* Fuchs reiterates his conclusions.

I have shown the specimens of this case to Professor Fuchs as well as to Professors Salzmann and Wintersteiner and to Mr. Treacher Collins and two well-known members of this Section, and my report is made in keeping with the remarks of these men. I would not have had the temerity to present simply my own opinion of the case. Yet I have studied the facts with an open mind. I saw the patient before the eye was removed, and my clinical diagnosis was sympathetic inflammation. The eye had had many openings made into it: there was ciliary injection and there were also precipitates on the back of the cornea of the fellow eye. But many traumatized eyes do not cause sympathetic inflammation, and no one has yet described a picture of the inflammation in the second eye so typical that we may cover the primary eye and diagnose sympathetic inflammation from the appearance of the second eye alone. A perforating wound and precipitates on the back of the other cornea do not make a diagnosis of sympathetic inflammation, and unless we go back to the dark ages we must

change a clinical diagnosis when reliable anatomic facts refute it. I hope Dr. Frank will state, in summing up, why he still considers this case one of sympathetic inflammation.

DR. G. H. PRICE, Nashville: Dr. Risley called attention to a very significant point in this particular case, when he said it was glaucoma with hemorrhage. It is a well-known physical fact that in a locomotive boiler which has been carrying a pressure of 200 pounds to the square inch and in which the pressure has run down to 30 pounds or less, there may be an explosion due to some defect in the mechanism by which this pressure is suddenly released. Or if in a boiler that is carrying a temperature of several hundred degrees, and a steam pressure of 200 pounds, a valve is suddenly opened which liberates the pressure instantaneously, the water remaining in the boiler, which is above the steaming point, is immediately converted into steam and the result is an explosion of the boiler. This is the same principle of the high-tension hemorrhage in glaucoma when we do an iridectomy; the tension being immediately released, a hemorrhage occurs because the high blood pressure suddenly overfills the vessels, causing the walls to give way, since the restraining influence of the intraocular pressure is lost. There is another very simple physical experiment that perhaps bears some relation to the condition we find present in some of these cases. If we take a flask and fill it half full of water and bring it to the boiling point and allow it to boil a few minutes, and while boiling remove from the flame and cork it, the ebullition ceases. If we then apply cold to the flask the supernatant steam is condensed, the tension lowered and the water boils again. There may be hemorrhages, depending on these two physical principles. In the case reported, it was a low-tension hemorrhage, a venous hemorrhage, and as Dr. Risley has suggested, this in all probability was due to some autoinfection or autointoxication somewhere, and the condition set up in the other eye, for which the enucleation was made, was probably brought about by just such a condition. Now if we had reached the point where we could determine whether we had a glaucoma which was due primarily to a marked increase in tension *per se*, then we would be in a position to determine what was the best procedure. In the case reported by Dr. Bull, of exceedingly high tension, if he had made an iridectomy in that case the chances are that he would have had considerable hemorrhage, due to the sudden release of the restraining influence on the retinal vessels, and so the thing to do was to gradually decrease the tension by paracentesis. But in Dr. Frank's case it was due to an entirely different etiologic factor and the relief of pressure, unless it was total, had no effect on it.

DR. F. H. VERHOEFF, Boston: I have examined many cases of sympathetic ophthalmia and before Fuchs published his work had come to the conclusion that there was a perfectly characteristic lesion. There is one point, however, connected with the histology of it that to me is not absolutely clear. The essential lesions consist of focal collections of lymphoid cells in the centers of which epithelioid and occasionally giant cells appear. Now in the early stage it is a question whether epithelioid cells are present, or if so, whether they could be distinguished from the lymphoid cells. Thus, going by Fuchs' description, when examined in this stage we must say the case is not one of sympathetic inflammation, although strictly speaking we would be wrong. However, I have never seen a case of sympathetic ophthalmitis develop after an eye showing this histologic picture was enucleated, although I have examined many such. At this early stage, therefore, if such it is, it is safe to say that the other eye will not become affected. In regard to Dr. Frank's case, I would like to suggest that the most obvious explanation is that it was a case of serous iritis complicated by glaucoma. Probably the condition had been present sometime without being noticed by the patient. Afterward, as is usual, the other eye became affected.

DR. MORTIMER FRANK, Chicago: I accept Dr. Risley's criticism of the title of my paper, but until Fuchs' findings are universally accepted as pathognomonic of sympathetic inflammation I still consider my case one of sympathetic ophthalmia according to the clinical symptoms described in the text-books, and I am willing that the pathologists should fight out the diagnosis by microscopic section.