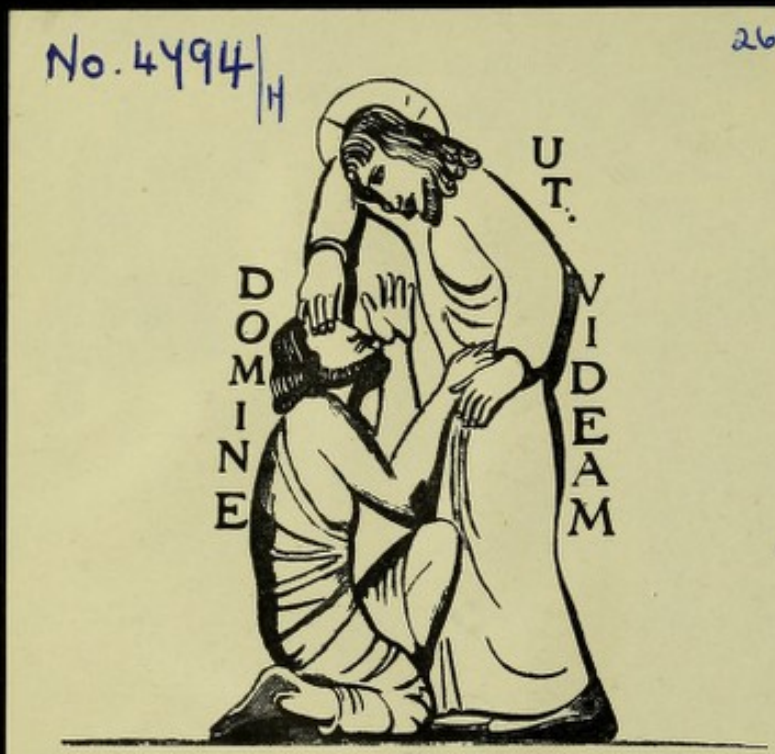
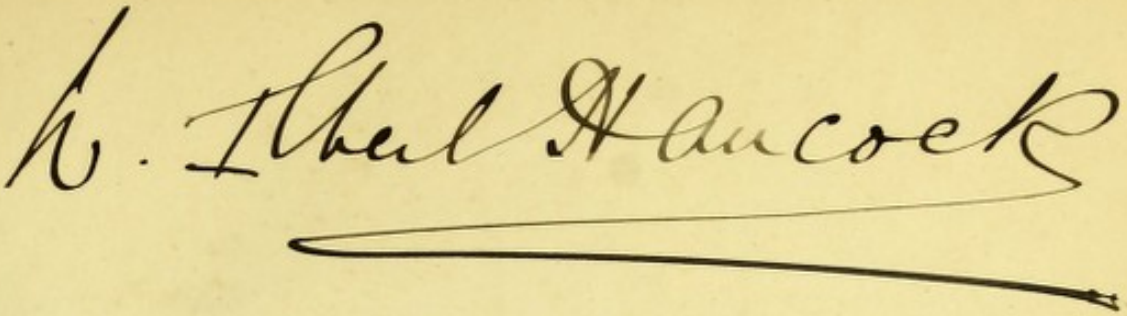
On the pathology and treatment of glaucoma : being a revised publication, with additions, of the Erasmus Wilson Lectures, delivered at the Royal College of Surgeons of England, in March, 1889 / by Priestley Smith.





THE PATHOLOGY AND TREATMENT OF GLAUCOMA.

ON

THE PATHOLOGY AND TREATMENT

OF

GLAUCOMA,

BEING A REVISED PUBLICATION, WITH ADDITIONS,
OF THE
ERASMUS WILSON LECTURES,
DELIVERED AT THE ROYAL COLLEGE OF SURGEONS OF ENGLAND,
IN MARCH, 1889.

BY

PRIESTLEY SMITH,

OPHTHALMIC SURGEON AND CLINICAL LECTURER ON DISEASES OF THE EYE,
QUEEN'S HOSPITAL, BIRMINGHAM.
AUTHOR OF THE JACKSONIAN PRIZE-ESSAY ON GLAUCOMA,
ROYAL COLLEGE OF SURGEONS, 1879.

WITH SIXTY-FOUR ILLUSTRATIONS BY THE AUTHOR,
AND TWELVE PHOTO-ZINCOGRAPHS.

LONDON
J. & A. CHURCHILL,
11 NEW BURLINGTON STREET.

1891.

PRINTED AT THE HERALD PRESS, BIRMINGHAM,
BY WRIGHT, DAIN, PEYTON, AND CO.

PREFACE.

This volume is a revised publication, with additions, of three lectures which were delivered at the Royal College of Surgeons under the Erasmus Wilson bequest, in March, 1889, and printed in the *British Medical Journal* shortly afterwards.

During the two years which have elapsed since that time several parts of the subject have been more fully worked out. The causes of glaucomatous complications after operations for cataract have been more clearly defined, mainly through the labours of Mr. Treacher Collins ; the connection between primary glaucoma and certain dimensional variations in the eye has been more positively established ; the condition of the vortex veins in glaucomatous eyes has been further investigated ; and some additional varieties of glaucoma have been examined.

An account of these matters is here given ; a short section describing the secondary changes produced by high pressure in the eye is added, and the treatment of glaucoma is dealt with more fully than before. The subject-matter, although considerably increased in amount, is still presented in the original form of three lectures.

In an Appendix at the end of the volume are placed certain tables of measurements and statistics which may be useful for reference, together with an account of the methods which were employed in preparing and examining specimens.

To the many friends who, as the following pages will show, have kindly helped me with pathological material, notes of cases, and statistics, I here repeat my hearty thanks.

PRIESTLEY SMITH.

BIRMINGHAM,
July, 1891.

TABLE OF CONTENTS.

LECTURE I.

LECTURE II.

LECTURE III.

PATHOLOGY OF GLAUCOMA.

LECTURE I.

Definition of the Subject.—Estimation of the Intraocular Pressure. The Manometer; the Finger-test; the Tonometer. Principles of Tonometry; sources of error; practical utility. Amount of the intraocular pressure as estimated by these means. Its equality in aqueous and vitreous chambers.—Secretion of the aqueous and vitreous fluids. Evidence afforded by structure and relations of the parts; by morbid anatomy; by physiological experiments. Excretion of aqueous fluid. Excretion of vitreous fluid. Experiments as to the amount of fluid which traverses the chambers in a given time, and as to the effects produced by differences of pressure in the two chambers respectively. Summary.

DEFINITION OF THE SUBJECT.

The healthy human eye presents to the educated touch of the surgeon a certain elastic resistance: its flexible walls are kept in a state of moderate tension by the fluids which traverse the chambers. Any eye which presents a decided variation from this characteristic condition, whether in the direction of hardness or of softness, is, as we know by experience, an unhealthy eye. I propose to speak in these lectures of the disorders which are associated with an increased tension of the eyeball—disorders to which we apply the name glaucoma.

With the advance of knowledge the word glaucoma has changed its meaning. In the early days of our art, it was applied without discrimination to the various diseases in which a grey or greenish-blue appearance replaces the normal blackness of the pupil. In later times, when a clear distinction had been made between the opacities which lie in the crystalline lens and

those which lie behind it, the name was reserved for some of the deeper seated and more destructive disorders, the nature of which remained unknown. Hardness of the eyeball, as a part of such disorders, received at that time only casual notice. In the year 1830, Mackenzie pointed out the frequent overfulness of the chambers in glaucoma, and made the first attempt to relieve that condition by puncturing the tunics. Twenty-five years later, von Graefe, studying the subject with the aid of the ophthalmoscope, convinced himself that this overfulness was no mere complication of the disease, but was the essential cause of the leading symptoms, and was led by this conviction to his beneficent discovery of the curative action of iridectomy. Since that time the name glaucoma, losing its original meaning, has been used to indicate a disease characterised by increased tension of the globe. Certain other characteristics—cupping of the disc, contraction of the visual field—are recognised as consequences of the pressure.[1]

Some writers, however, still deny that what we call glaucoma is necessarily connected with increased pressure. They point to certain exceptional cases in which the contracted field and the excavated disc are found in company with normal tension, and on the strength of these exceptions they maintain that glaucoma is the expression of some unknown agent which usually raises the pressure and excavates the disc at the same time, but which occasionally excavates the disc without raising the pressure. This assumption is, I think, unnecessary. The excess of pressure is sometimes slight ; it is often intermittent ; it may even be absent for long periods of time ; it is very probable, therefore, that these cases of glaucoma with normal tension are cases which have been examined only during the

[1] An admirable outline of the history of the subject may be found in Snellen's recent "Historical Essay on the Development of our present Knowledge of Glaucoma," "Ophthalmic Review," February, 1891.

intermissions of increased tension. Permit me to mention a case in illustration :—

Sir William Bowman was consulted by a lady in 1865. He noted—and he has kindly placed the notes at my disposal—increased tension and commencing excavation, diagnosed glaucoma, and spoke of iridectomy. No operation was performed. Twenty years later this lady came under my own care with deeply excavated discs, contracted fields, and impaired vision. I saw her many times ; sometimes the tension was quite normal, sometimes it was decidedly increased. Vision was still useful, but failing rather rapidly. After consultation with Sir William Bowman, iridectomy was performed on both eyes with good result.[2] Now this lady must have had for years an unmistakable glaucoma, but at times, and probably very frequently, a normal tension. Yet pressure was there at the beginning and it was there at the end. I cannot doubt that it was an essential factor in the process.

There is ample evidence that the typical excavation of the papilla—the glaucoma-cup—is a product of high pressure. It is to be found, by dissection or otherwise, in every eye which has suffered long or frequently from high tension.[3] It is not to be found after very brief periods of high tension, for the excavation involves atrophic changes which need time for their

---

[2] The case is fully recorded in the "Ophthalmic Review" for 1885, p. 261. The following notes illustrate the variable character of the tension :—

May 13th, 1885. I visited the patient at her own house. At 5 p.m. the tension of both eyes, tested more than once, was quite normal ; at 11 p.m., when she was tired and overwrought by superintending an operation on another member of the family, it was decidedly in excess, the pupils being larger than before.

The left eye, which was slightly the worse of the two, was iridectomised seven days before the right. At the time of the first operation both eyes had *plus* tension, and the iris of the operated eye prolapsed when the section was completed. At the time of the second operation the second eye had quite normal tension and no prolapse occurred. The value of rest in producing remission of tension was well marked in this case.

[3] Exceptions to this rule are occasionally to be found in cases in which the disc has been covered by a tumour or other new formation.

development, but even in such cases the microscope shows the first step of the process—a pushing back of the lamina cribrosa[4]; and even in the healthy excised eye high pressure artificially applied causes the same displacement.[5]

It is possible, of course, though not probable, that a similar excavation may arise from causes other than an excess of pressure. Should this prove to be the case, it will be well to give to such cases some name other than glaucoma.

Glaucoma, then, may be broadly defined as *an excess of pressure within the eye, plus the causes and the consequences of that excess.* To understand the morbid process in its entirety, we must study first the normal intraocular pressure and the processes by which it is maintained; secondly, the causes which lead to an excess of pressure; and, thirdly, the consequences of such excess.

ESTIMATION OF THE INTRAOCULAR PRESSURE.

It will be well, in the first place, to consider the means by which the intraocular pressure has been measured or estimated.

Manometer.—In certain of the lower animals the pressure has been accurately determined by means of the manometer, an instrument which brings the intraocular fluid into direct communication with a column of mercury or other fluid. The average normal pressure, the variations due to pulse and respiration, and those produced by ligature of arteries and veins, by section and irritation of nerves, and by the action of certain drugs, have all been ascertained. The manometer has also been applied in a few cases to the living human eye, namely, to eyes doomed to excision, the measurement being made immediately before the operation.

---

[4] Brailey, R. L. O. H. Reports, vol. ix., p. 208.

[5] Laker, "Klin. Monatsbl. f. Augenheilk.," May, 1886, p. 187; and "Ophthalmic Review," 1886, p. 130.

Finger-test.—Under ordinary circumstances it is impossible to measure the pressure in the living human eye, and we have to content ourselves with measuring, as an index to it, the resistance of the tunics. We apply the tips of the two forefingers with light alternate pressure to the upper eyelid while the eye is directed downwards, and we record our observations by means of Bowman's symbols : Tn, T+1, T+2, etc. For daily use no better method than the finger-test has been, or is likely to be, found, and it must be employed constantly by everyone who would deal successfully with diseases of the eye ; but it is obviously inexact. We cannot state with precision the resistance which we feel, and we cannot rely upon the constancy of our sense of touch.

Tonometer.—to obviate this uncertainty, many attempts have been made to substitute for the fingers an instrument of precision—a mechanical tension-measurer or tonometer. I will not attempt to describe the various instruments which have been invented for this purpose ; not one of them has hitherto found general acceptance. I will ask your attention rather to the principles which underlie and limit every effort of the kind, my object being not to show how every difficulty may be overcome, but to distinguish the possible from the impossible.

Principles of Tonometry.—This bladder (Fig. 1) will serve to represent the eye. It is filled with water, and is in open communication with the reservoir which stands at a higher level. The pressure in the bladder depends upon the height of the reservoir, and is constant so long as this remains unchanged. Our object is to estimate the internal pressure from the behaviour of the tunics under external pressure. The instrument used, therefore, must press upon the eye, and must measure both the force employed and the impression produced. It is easy to measure the force employed. It is less easy to measure the impression produced, for it has more than

one dimension; it varies in area and in depth. Are we to measure the area, or the depth, or both?

Area of Impression.—When this flat disc (Fig. 1) is loaded with a certain weight and placed upon the bladder, it flattens the membrane over a certain area; it sinks until it meets with precisely the same pressure from below as from above; then it remains in equilibrium. Hence, if we employ a known external

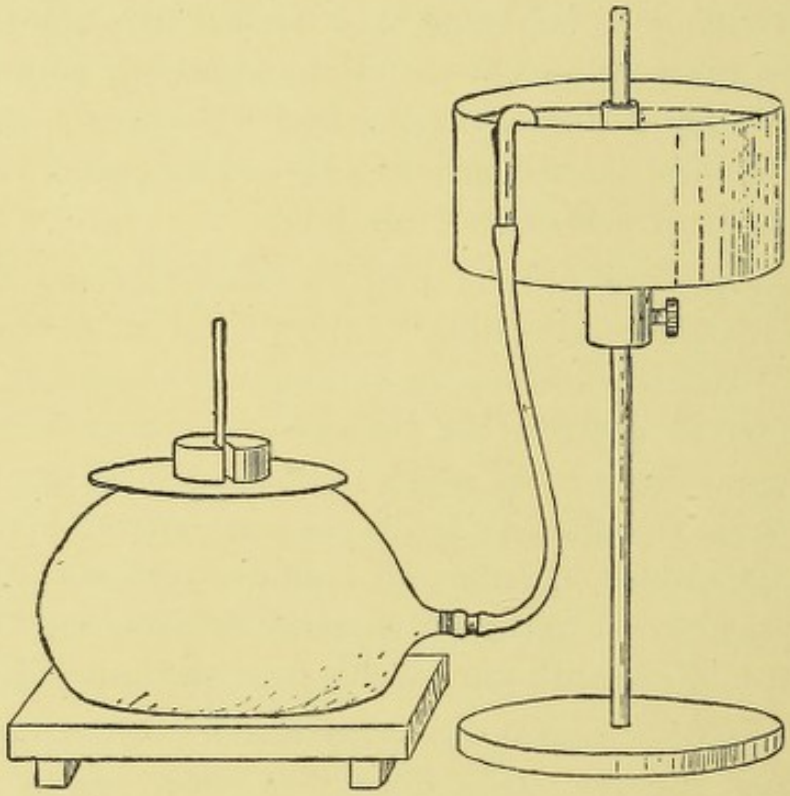


FIG. 1.

Apparatus to illustrate the mode of estimating the internal pressure from the area depressed by a known external pressure.

pressure, and measure the area of contact, we can calculate the internal pressure. For example, if we load the disc with a weight of 100 grammes, and find that the area of contact equals 100 square centimetres, we know that the internal pressure is 1 gramme to 1 square centimetre. If we were to take a second bladder of different size, or of different curvature at the point

of contact, or of more extensible membrane, and place it at the same height and in connection with the same reservoir, we should find that the disc, if loaded with the same weight as before, would have the same area of contact as before. We see then that, *assuming the membrane to be perfectly flexible, the area of the impression produced by a known external pressure is a true index to the internal pressure.*

It might appear, then, that the problem of ocular tonometry could be solved in this way. More than ten years ago I arrived by experiment at the principle which has just been stated, but concluded that the difficulties of applying it to the eye were insuperable.[6] Quite recently Professor Fick, of Würzburg, following the same theoretical considerations, has invented a tonometer which is correct in principle and very simple in construction.[7] This rough, enlarged model (Fig. 2) will

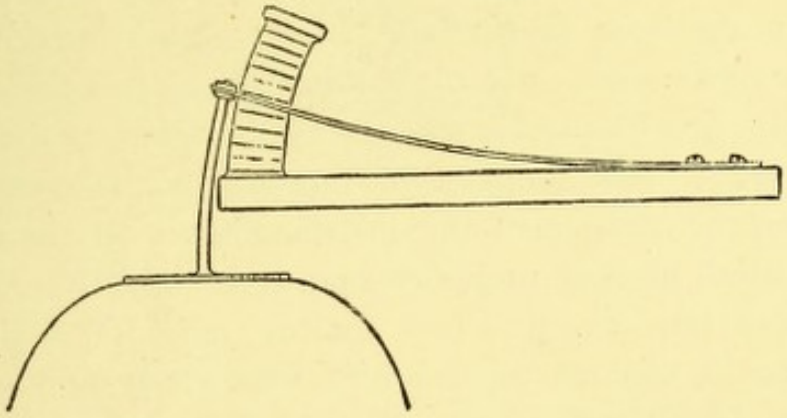


FIG. 2.
Model illustrating the action of Fick's tonometer.

explain its action. A flat disc is pressed against the eye through the medium of a spring, and the force exerted by the spring is indicated by the scale behind it. The area of the disc is known ; let us say that it is 50 square centimetres. I now

[6] "Glaucoma, its Causes, etc." London, Churchill, 1879, p. 48.

[7] "Inaugural Dissertation" by R. A. Fick, Würzburg, 1888, and "Transactions of International Ophthalmic Congress, Heidelberg," 1888, p. 289.

press the disc against the bladder until it depresses and flattens exactly its own area of the membrane, neither more nor less, and I see by the scale that the force employed in doing this is, say, 50 grammes. We know that the internal pressure against the flattened area is equal to the external pressure, namely, 50 grammes. We have, therefore, an internal pressure of 50 grammes on an area of 50 square centimetres, or 1 gramme to 1 square centimetre.

Now the value of this instrument obviously depends upon one's ability to depress the membrane to an extent precisely equal to the area of the disc. A small error in this respect gives a larger error in the estimate of the pressure ; thus, if the diameter of the depressed membrane be larger or smaller than that of the disc by one-tenth, the area will be at fault by one-fifth, for the areas of circles vary as the squares of their diameters ; and the estimate of the internal pressure will be at fault in the same proportion. In the actual instrument the diameter of the disc is about 7 millimetres, so that the difficulty of applying it with precision to the eye, covered as the latter is by a more or less compressible conjunctiva, is considerable. Moreover, the readings of the instrument are at the mercy of the operator's wish or prejudice, and it is difficult to apply it with strict impartiality when a little more or a little less pressure will confirm or falsify a preformed opinion. The principle of estimating the internal pressure from the *area* of the impression is unquestionably a true one, and Professor Fick's tonometer is the only one clearly based on this principle. I fear, however, that the difficulty of applying it with precision will be found to cause uncertainty in the results obtained.

Depth of Impression.—The depth is more easily measured than the area ; this model (Fig. 3) will serve to show how it may be done. When the three rods are placed vertically upon the bladder without pressure, they adapt themselves to its curvature. When the middle rod is loaded with a certain

weight it sinks through a certain distance, as indicated by the scale behind it. This distance varies with the internal pressure, and is therefore to some extent a measure of it; but it varies with other things as well, namely, with the curvature of the surface and with the extensibility of the membrane. Thus if the instrument were placed upon another bladder having the same internal pressure, but a flatter curvature, the impression would be shallower than before; and if the curvature were the same, but the membrane more extensible, it would be deeper than before. Now the sclera of the human eye varies somewhat both as to curvature and extensibility in different individuals, hence *the depth of the impression produced by a known external pressure is not a perfectly true index to the internal pressure.*

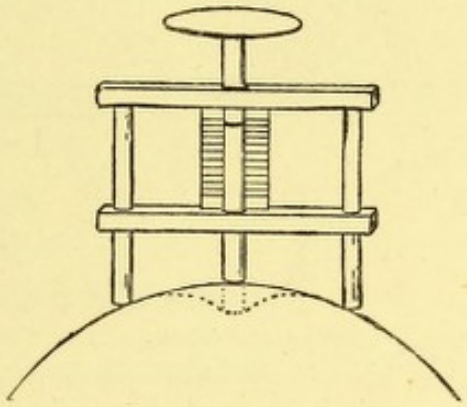


FIG. 3.

Model illustrating the mode of estimating the internal pressure from the depth of a pit made by external pressure.

And there are other sources of error which cannot be avoided, whichever method we employ. The tunics of the eye are not perfectly flexible, and their rigidity varies at different parts and in different individuals. Again, the thickness of the conjunctiva must be taken into account; a swollen conjunctiva pits deeply, irrespective of the yielding of the sclera. And again, it must be remembered that the tonometer itself raises the intraocular pressure for the moment, so that, even if it could measure the internal pressure accurately, we should learn,

not the pre-existing pressure, but the pressure as modified by the application of the instrument; the amount of this modification remains unknown, and it varies in different cases according to the ease with which fluid is extruded from the eye.

No tonometer, then, however ingeniously devised, can accurately measure the intraocular pressure, or accurately compare one eye with another. Are such instruments of no value? Permit me to exhibit a tonometer which is the outcome of my own study of the subject, and to state what I believe to be its utility. This instrument[8] (Fig. 4) exerts a

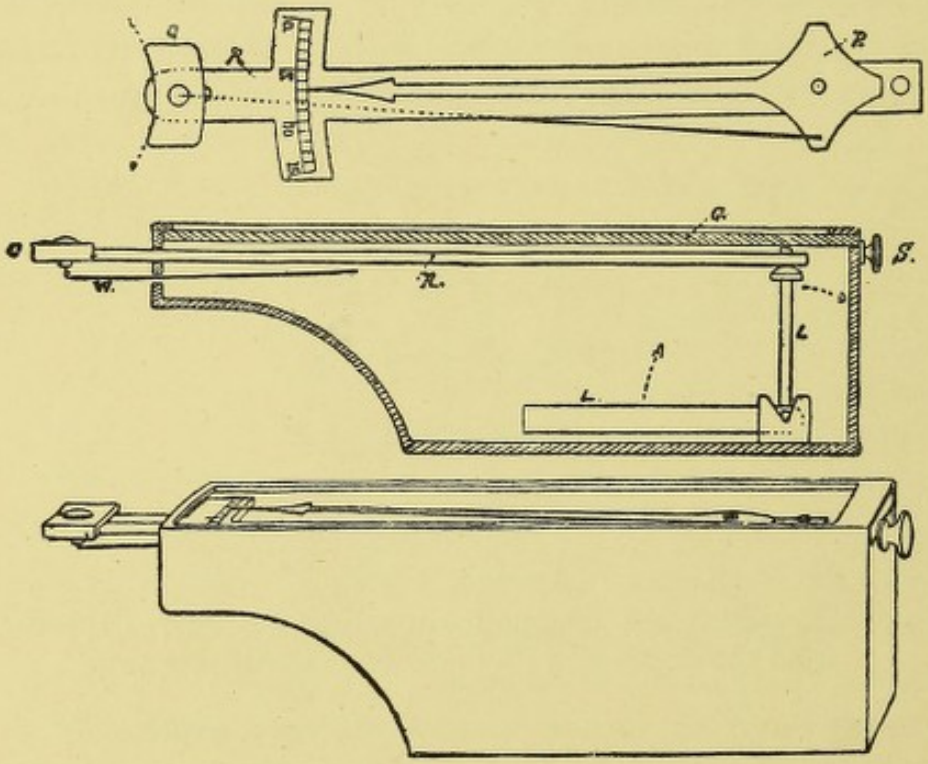


FIG. 4.

Author's Tonometer.—The upper figure shows the ram R, carrying the crescent C and the pointer P, these two being connected by a wire—seen from above. The second figure shows the instrument in section, the end of the ram resting on the upright arm of the rectangular lever L. It is made by Messrs. Curry and Paxton, of Great Portland Street, London.

[8] For details, see "Ophthalmic Review," 1887, p. 33.

known and invariable pressure on the eye, and indicates the depth of the impression produced. The cross-shaped bar or ram is brought into contact with the eye. The crescent which lies upon it slides backward with a minimum of friction, and communicates its movement to the pointer. The pointer magnifies the movement of the crescent ten times. When the ram is applied to an unyielding surface no impression is produced, and the pointer stands at 0 ; when it is applied to a yielding surface it impresses it, the crescent slides back, and the pointer indicates the depth of the pit produced. It only remains to apply this mechanism to the eye with the same force in every case. This is effected by means of a weight and rectangular lever. An important point in the instrument is the mobility of the crescent about its own centre. If the tonometer is applied obliquely instead of perpendicularly to the surface of the eye—and this happens more or less in every instance—the crescent adjusts itself to the surface before it begins to actuate the pointer. Without some such adjustment very accurate measurements cannot possibly be made. The curvature of the crescent is adapted to the average curvature of the eye, and makes no allowance for individual variations ; it is impossible to make such allowance without complicating the mechanism. The consequence is that in a sharply curved eye the tonometer under-estimates the depth of the impression, while in a flatter eye it over-estimates it ; but these are errors in the right direction, for, as we have seen, a given pressure pits more deeply in a sharply-curved surface than in a flatter surface, so the one error tends to neutralise the other. If, however, the curve of the eye varies greatly from that of the crescent, a considerable error will arise.

If properly handled, this instrument does its work well—that is to say, it makes the same pressure in all cases ; it measures the depth of the pit with considerable accuracy ; and it will not lend itself to the operator's prejudice. Has it

any practical utility? For the purpose of comparing the eyes of one person with those of another, it is, I think, not much better than the educated finger, for the differences which it may indicate, if they be so slight as to elude the finger, may be due to differences in the curvature or flexibility of the membranes. For comparing the two eyes of the same individual, when they are presumably alike in structure, it is of much greater value. But its chief use is in determining the changes which occur in one and the same eye. Applied to the same spot on successive occasions, it reveals slight changes of the intraocular pressure with much greater certainty than the finger, and its indications can be recorded and compared. I now use it habitually when in doubt whether the tension is quite equal in the two eyes, whether it is influenced in the right direction by the treatment employed, or whether an operation is necessary. It is not an instrument for rough and ready diagnosis in unskilled hands, for it is more difficult to use than the finger-test; but to those who would accurately control the indications afforded by the finger, it may, I think, be recommended.

The manometer, the finger-test, and the tonometer in combination, have been useful in determining the pressure which is normally present in the living human eye. If we connect the eye of the dead subject by means of a hollow needle and elastic tube with a column of mercury or other fluid, we can ascertain what pressure is required to produce a degree of tension resembling that of the living eye; we can compare the artificial with the natural tension, either with the finger or more accurately with the tonometer. In like manner we can ascertain what pressures correspond with our own ideas of T+1, T+2, and T+3, or with the various degrees upon the scale of the tonometer.

AMOUNT OF THE INTRAOCULAR PRESSURE.

The foregoing are the means by which the intraocular pressure has been estimated. We may next consider the results obtained. Concerning the normal pressure in the eyes of animals, we have a considerable mass of evidence and a very close agreement. The animals chiefly experimented on were rabbits, cats, and dogs, and the precautions taken against error were very minute. In nearly all cases the pressure was between 20 and 30 millimetres of mercury; the average was about 25 millimetres. Slight oscillations were found to accompany pulse and respiration, and a more marked rise was produced by contraction of the external muscles of the eye.[9]

Experiments upon the dead human subject with my own tonometer gave a closely corresponding result. I found that in order to produce a tension resembling that of the living eye, a pressure of about 30 centimetres of water—that is, 25 millimetres of mercury—must be employed, and the same result was obtained on testing with the finger. Other observers employing a tonometer in the same way have placed the normal pressure rather higher than this—in one case even as high as 50 millimetres of mercury; but that this latter figure represents a distinctly glaucomatous tension will be manifest to anyone who will take the trouble to employ the finger test in the mortuary in the manner described. Adolph Weber, trusting the finger rather than the tonometer, estimates the pressure, as I have done, at about 25 millimetres of mercury.[10]

This estimate has lately been confirmed by Wahlfors, who applied the manometer to several human eyes immediately before excision. One of these was a healthy eye, which had to be sacrificed on account of an orbital tumour. The intraocular pressure was equal to 26 millimetres of mercury.[11]

---

[9] "Graefe-Saemisch Handbook," vol. ii., p. 371.

[10] "Glaucoma, its Causes, etc.," p. 98.

[11] "Trans. of Internat. Ophth. Congress," 1888, p. 268.

Again, in the lower animals, numerous experiments with the double manometer have proved that the pressures in the aqueous and vitreous chambers are equal, or so nearly equal that the difference is not discoverable.[12] It has been asserted that the vitreous pressure is considerably higher than the aqueous pressure, and some observers have even declared themselves able to detect the difference by placing the fingers on different parts of the globe. This idea, though disproved by accurate observation, is not yet quite abandoned. Experiments of my own upon the freshly excised eyes of animals have shown that the lens is displaced from its normal position by a slight excess of pressure either before or behind it. Thus, an excess in the vitreous chamber equal to 5 millimetres of mercury causes a considerable displacement of the lens and iris, and an excess of 10 millimetres almost abolishes the anterior chamber.[13] Such differences in the two chambers are quite too small to be detected by the finger, and it is obvious that any comparison of the scleral with the corneal tension by means of the tonometer is fallacious. When such differences do occur in the living eye they produce, and are manifested by, displacements of the lens and iris forwards or backwards as the case may be.

Concerning the height to which the intraocular pressure rises in the human eye under abnormal conditions we have little direct evidence. In a case of chronic glaucoma in which the tension had been slightly reduced by an unsuccessful iridectomy, Wahlfors found with the manometer a pressure of 71 millimetres of mercury. Experiments with my own tonometer seem to indicate that the tension which we call +3 corresponds to a pressure considerably higher than this. Much higher pressures have been produced experimentally in the eyes of animals. In

---

[12] Bellarminoff, Abstract in "Centralbl. f. Prakt. Augenheilk," 1887, p. 54; and in "Ophthalmic Review," 1887, p. 361.

[13] "Ophthalmic Review," 1888, p. 207.

one instance, it appears, a pressure equal to 200 millimetres of mercury was produced by compression of the aorta and simultaneous irritation of the fifth nerve.[14]

SECRETION AND EXCRETION OF THE INTRAOCULAR FLUIDS.

The maintenance of the normal pressure in the chambers of the eye depends upon the due secretion and the due excretion of the fluids which traverse them. These physiological processes have been most carefully studied during the last fifteen years, and it will be well to review the evidence upon which our knowledge stands at present. It is only by a careful study of these processes that we can hope to unravel the complex pathology of glaucoma.

The intraocular fluids flow, like all other secretions, from the blood-stream. Studying their origin from the anatomical point of view, we find, as possible sources, two vascular membranes, the uveal tract and the retina. We may at once eliminate the retina, for clinical observation shows that complete blockage of the retinal vessels by embolism causes no discoverable change in the fulness of the chambers. The uveal tract consists of three distinct parts, namely, the choroid extending forwards as far as the ora serrata, the ciliary body reaching from the ora serrata to the base of the iris, the iris ending at the margin of the pupil. The functions of these several parts may to some extent be inferred from their structure and relations.

The choroid, by its internal surface, is applied to the external layers of the retina. Its capillaries feed the pigmented epithelium, and the epithelial cells keep the rods and cones in a state of functional activity. The meshes of the capillary plexus

---

[14] Von Hippel and Gruenhagen. "Von Graefe's Archiv," vol. xiv., part 3, p. 219. See also "Glaucoma, its Causes," etc., p. 103.

become progressively less and less close from the posterior pole to the anterior limit, and this arrangement accords with the varying sensibility of the corresponding zones of the retina. There is no evidence that the choroid nourishes the vitreous body, and it is obviously improbable that a highly organised membrane like the retina, which has many differentiated layers and a separate vascular system of its own, should transmit nourishment from the choroid to the vitreous.

The iris has a well-defined optical function, that, namely, of regulating the entrance of light. It is possible that its posterior surface, like the rest of the uveal surface, may have a secretory function as well, and take some part in the formation of the aqueous humour, but this part must be quite a subordinate one, for there are many cases on record in which the iris has been completely absent, either from birth or through injury, and yet there has been no discoverable insufficiency of the aqueous fluid ; the tension has been normal.

The ciliary portion of the uveal tract shows a special adaptation for the supply of fluid to the vitreous body, the lens, and the aqueous chamber. Where it is in relation with the vitreous, its secreting surface is extended by a series of grooves and ridges, and where it is in relation with the aqueous chamber it assumes a still more convoluted arrangement well suited for rapid secretion.[15] The vitreous body has its chief attachments in this region ; its limiting membrane adheres firmly to the ora serrata, and in advance of this point is separated from the secreting surface only by a single layer of cylindrical cells. The membranous septa which spring from the limiting membrane in this region are arranged in a manner peculiarly favourable for the entrance of fluid.[16] (Fig. 5).

---

[15] See description by Brailey, "Brit. Med. Journal," September, 1882, p. 577.

[16] See description by Straub, "Von Graefe's Archiv," vol. xxxiv., part 3, p. 7.

Pathological anatomy points to the same conclusion. If we examine eyes which have been excised during the first stage of vitreous infiltration, we find an inflammatory exudation entering the vitreous body from this portion of the uveal tract, the region of its influx being limited posteriorly by the ora

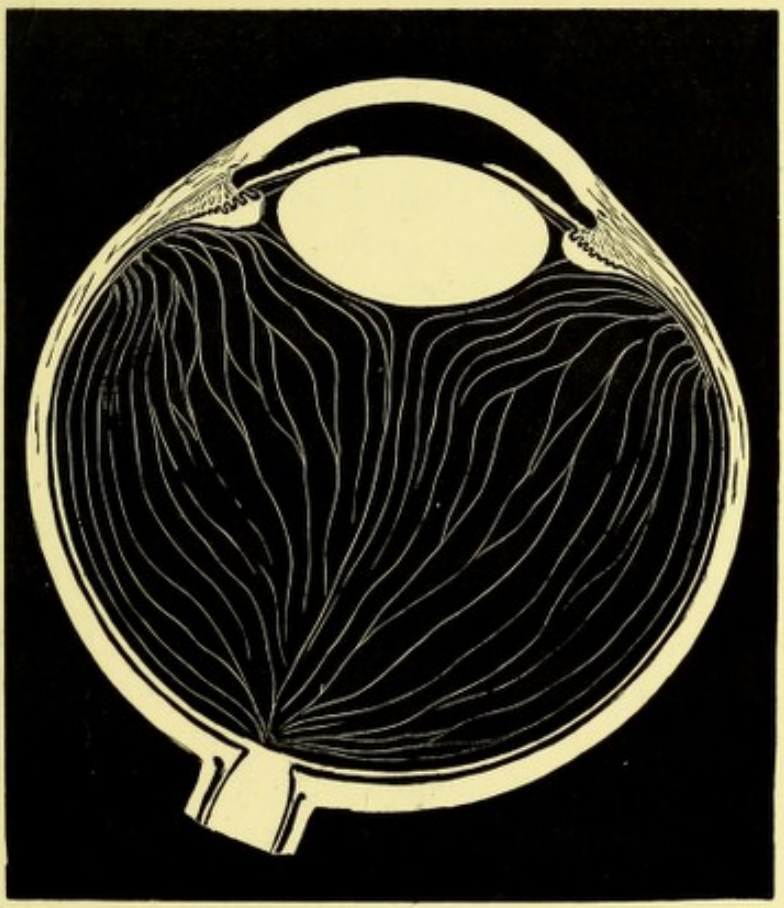


FIG. 5.

Horizontal section through right eye, seen from above. Enlarged 3 diameters.—Somewhat diagrammatic. The septa of the vitreous body are transparent and invisible in the normal condition, but their general arrangement can be made out in eyes which have been long hardened in Müller's fluid and divided in the frozen state.

serrata. We find also that while a shrinking vitreous readily separates from the retina it always retains its firm connections in the region of the ora serrata. And, again, we find that while very extensive atrophy of the choroid does not necessarily affect

the transparency or the volume of the vitreous body, disease in the ciliary region always tends to its destruction.

Experiments upon animals have amply confirmed the inferences previously drawn from these anatomical and pathological facts. Deutschmann found that removal of the ciliary processes together with the iris, which can be effected in the rabbit without loss of lens or vitreous, and without causing inflammatory destruction of the eye, is followed by a total arrest of the secretion of the aqueous, and by atrophy of the vitreous body and lens.[17] Schoeler, Uhthoff, and others have found that after subcutaneous injections of fluorescine there is a speedy coloration of the aqueous fluid, and a more gradual coloration of the vitreous body, and that the coloured secretion proceeds from the ciliary body, and perhaps to a small extent from the posterior surface of the iris also.[18] Leplat has demonstrated the same thing by a somewhat different method. He gave subcutaneous injections of iodide of potassium, enucleated the eyes after various intervals of time, froze them and divided them into zones, and made a quantitative test for the iodide in each zone.[19] These and many other experiments agree in showing that the fluids which nourish the vitreous body and lens and fill the aqueous chamber are secreted chiefly, if not entirely, by the ciliary portion of the uveal tract.[20]

In what direction do these fluids travel, and where do they escape from the chambers?

---

[17] "Von Graefe's Archiv," vol. xxvi., part 3, p. 117; and "Ophthalmic Review," 1882, p. 149.

[18] "Report of Professor Schoeler's Clinic for the Year 1881," Berlin, 1882 p. 52; and "Ophthalmic Review," vol. i., p. 413.

[19] "Annales d'Oculistique," Sept.-Oct., 1887, p. 1; and "Ophthalmic Review," 1888, p. 84.

[20] Long before any of this experimental evidence was available, Mackenzie, speaking of the crystalline lens, said:—"Its nutrition, as well as that of the vitreous humour, is derived unquestionably from the capillaries of the ciliary body." "Diseases of the Eye," 4th Edition, p. 897, 1854.

The aqueous fluid passes from the posterior chamber forwards through the pupil into the anterior chamber. It has been asserted that there is a current passing forwards through the base of the iris,[21] but the evidence is by no means conclusive. It is contrary to physical reasoning to suppose that while the pupil remains open the fluid will traverse the tissues of the iris ; moreover, the stream through the pupil is proved by the fact that when the pupillary margin becomes entirely adherent to the lens capsule the fluid collects behind the iris with disastrous consequences.

The aqueous fluid escapes at the angle of the anterior chamber, by filtering through the ligamentum pectinatum into Schlemm's canal and the veins connected with it. This fact was established by Leber's well-known experiments. Employing freshly excised eyes of dogs, pigs, and cats, Leber found that diffusible coloured solutions injected into the anterior chamber pass readily into Schlemm's canal and the veins of the iris, causing a visible injection of the episcleral venous plexus and the conjunctival veins, and escaping through the cut ends of these vessels and through the vortex veins. Colloid solutions, on the other hand, which will not pass through animal membranes, caused no injection of the vessels. This indicated the absence of any open communication between the chamber and the vessels, and a very beautiful crucial test gave confirmation. A mixed solution of carmine and Prussian blue, violet in colour, was injected. The carmine, which filters readily through animal membranes, passed through, causing a pink injection of the vessels ; the blue, which does not so filter, was arrested, and was afterwards found by the microscope collected in the meshes of the ligamentum pectinatum. Leber showed also that the cornea, so long as its posterior epithelium remains entire, is not permeated by the aqueous fluid. Certain experiments of my own,

[21] "Von Graefe's Archiv," vol. xxvi., part 3, p. 35.

to be described immediately, showed that when the base of the iris is pushed forwards so as to close the angle of the anterior chamber, the exit of fluid is arrested.

The escape of fluid from the vitreous body has been the subject of much experiment and discussion, and is still not determined with absolute certainty. The question is whether it escapes at the papilla, or through the aqueous chamber, or in both these directions. Schwalbe has described certain lymph passages within the sheath of the optic nerve, which, he says, "find exit in the lymph passages of the skull, and convey not only the lymph formed in the optic nerve, but that also of the retina and vitreous."[22] Stilling has seen a considerable escape of fluid in this region under artificial pressure.[23] The injection experiments of Leplat, already referred to, appear to show that iodide of potassium injected subcutaneously enters the vitreous from the ciliary body and travels very slowly backwards, and leaves it at the papilla. Still more positive evidence is afforded by some experiments made by Gifford.[24] He injected with special precautions small quantities of water containing indian ink or cinnabar in suspension into the vitreous of rabbits and other animals. The results were definite and constant. On the second day, or later, according as the injection was made farther backward or forward in the vitreous, the ophthalmoscope showed the particles collecting in the hollow of the papilla. On killing the animal a day or two later the microscope showed the particles passing backwards through the papilla, along the lymph spaces around the central vessels, leaving the nerve trunk with the vessels, and passing towards the sphenoidal fissure. They did not enter the sheath of the optic nerve. The current within this sheath has been proved by other experiments to move from the brain towards the eye.

[22] "Graefe-Saemisch Handbook," vol. i., p. 50.

[23] "Report of Heidelberg Congress of 1885," p. 42.

[24] "Archives of Ophthalmology," 1886, p. 153; and "Ophthalmic Review," 1886, p. 217.

In spite of the evidence put forward by these investigators, it has been difficult to accept the view they advocate, for certain facts seemed to point the other way. Schoeler found that artificial occlusion of the supposed posterior outlet caused no discoverable reduction in the amount of fluid escaping from the eye in a given time.[25] More recently, in experiments made by himself and Uhthoff, he failed to find any transit of fluorescine from the eyeball into the optic nerve or its sheaths. I myself, ten years ago,[26] injected solutions of carmine and of aniline purple into the vitreous of freshly excised pigs' eyes, maintained the pressure for five or six hours, then froze the eye, and cut microscopic sections through the posterior pole. There was no colour deeper than the superficial layers of the retina. Again, I injected a coloured fluid into the sheath of the optic nerve, after ligaturing the nerve behind the point of injection. The pressure employed was about double the normal intraocular pressure, and was maintained at this height during five hours. Microscopic sections showed no trace of fluid having entered the eyeball, although the tissues were deeply stained up to the anterior extremity of the subvaginal space.

In favour of the supposition that the vitreous fluid escapes through the aqueous chamber is the fact that it *can* easily pass in this direction. Leplat has lately proved, with regard to the living eye, that the fluid which rapidly refills the aqueous chamber after this has been emptied by puncture of the cornea, is derived in large measure from the vitreous chamber ; and this same refilling of the aqueous chamber occurs also in the dead eye, in which the vitreous is the only possible source of the fluid.

In order to test this matter still further, and to obtain, if possible, some information as to the amount of fluid which

---

[25] "Von Graefe's Archiv," vol. xxv., part 4.

[26] "Glaucoma, its Causes, etc.," p. 142.

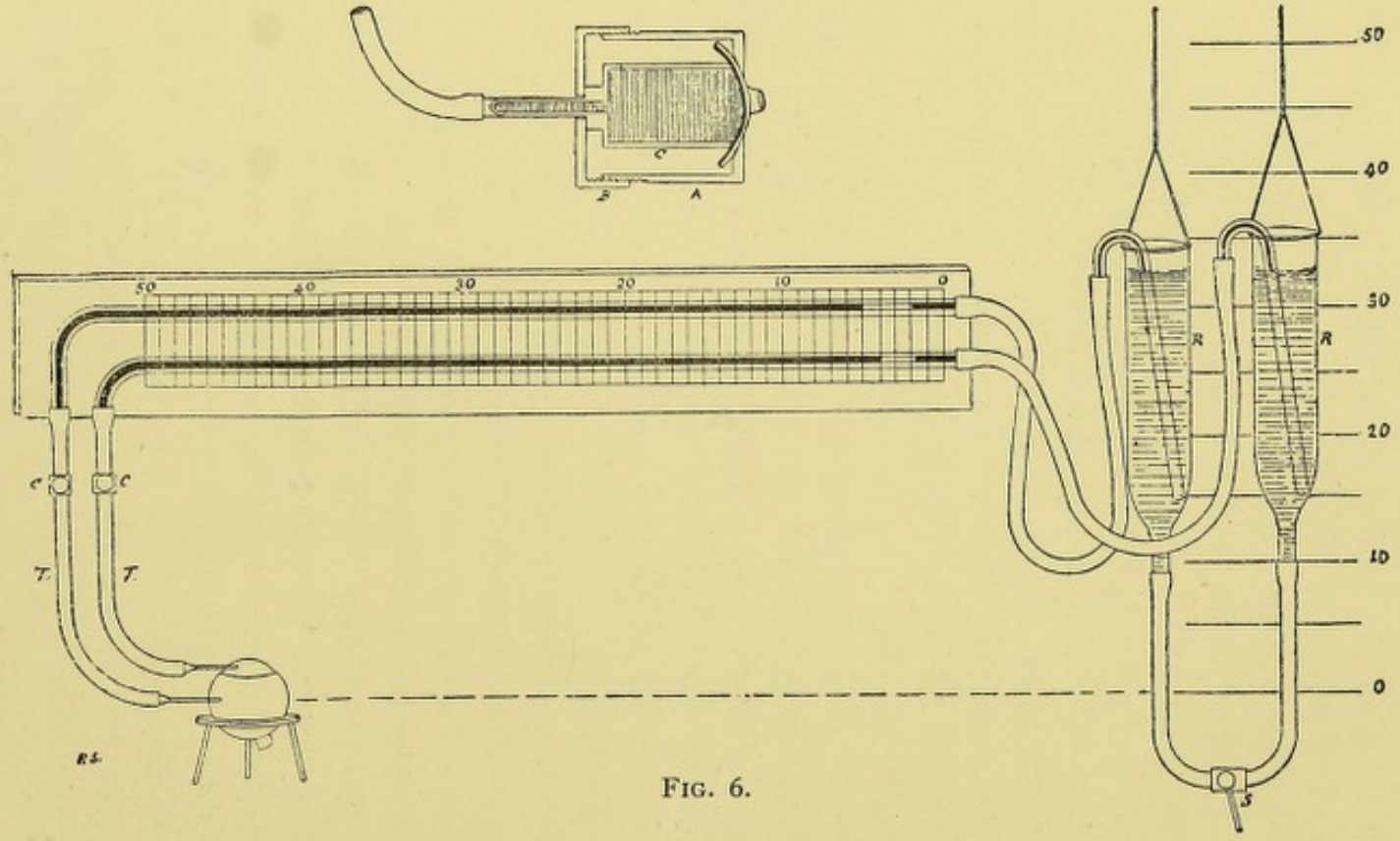


FIG. 6.

Apparatus for injecting fluids under various known pressures into the aqueous and vitreous chambers of the eye, and for measuring the amounts of fluid which pass through the chambers in a given time.

The smaller figure exhibits an apparatus for testing the escape of fluid through the papilla under artificial pressure.

traverses the chambers in a given time, I undertook lately another series of injection experiments on the freshly excised eyes of oxen, sheep, and pigs, and, in a few instances, on the human eye after death. The most trustworthy results were obtained with sheep's eyes, for I could have an unlimited supply of these within a few minutes of death. The apparatus employed is shown in Fig. 6, and need not be described in detail. It enables one to make a continuous injection of one or both chambers under a known and constant pressure, and to measure the amount of fluid which passes through the chambers in a given time. The pressure can be varied at will, by raising or lowering the reservoirs, and it can be kept absolutely equal in the two reservoirs by opening the tube which connects them below ; or, when this tube is closed, the reservoirs can be adjusted to give different pressures. An air-bubble in each of the graduated horizontal tubes serves to indicate the movement of the fluid ; its position is noted at regular intervals of time. The cubical content of the tube is known. A pressure of 30 cm. of water, that is, 25 mm. of mercury, was adopted as the standard pressure throughout, this being about the normal pressure.

The experiments were of several kinds. In one series I injected the anterior chamber only ; in another the vitreous chamber only ; in a third series, aqueous and vitreous were injected simultaneously under precisely equal pressures ; and in a number of other cases I employed different pressures in the two chambers simultaneously, and varied the conditions in many different ways. The details have been described elsewhere[27] ; here I need only deal with the results. The following were the averages of ten experiments of each kind on the eyes of sheep ; the variations were not great :—

---

[27] "Ophthalmic Review," July, 1888, p. 193.

When the anterior chamber only was injected, the escape during the first half-hour was 785 cubic millimetres.

When the vitreous only was injected it was 275 cubic millimetres.

When the aqueous and vitreous were injected simultaneously under the same pressure it was 485 cubic millimetres, that is, considerably less than during injection into the aqueous chamber only.

This result, at first sight paradoxical, is explained by the varying position of the iris, and its effect upon the patency of the angle of the anterior chamber—the "filtration-angle." When the anterior chamber only was injected, the iris was displaced a little backwards; when the aqueous and vitreous chambers were injected simultaneously under equal pressures, the iris remained in its normal position, or nearly so; when the vitreous only was injected the iris was displaced a little forwards. In order to observe these displacements accurately, I froze the eyeballs solid while the injection was still going on, and bisected them.

In the fourth series of experiments the vitreous reservoir was raised to a higher level than the aqueous reservoir. For example, keeping the aqueous reservoir at 30 centimetres, as before, the vitreous pressure was raised to 35 centimetres. Under these circumstances the diaphragm between the two chambers, consisting of the lens, zonula, processes, and iris, is displaced forwards until the increased tension of the zonula balances the excess of pressure behind it. Equilibrium being established in the diaphragm, a stream passes through the zonula from the vitreous to the aqueous chamber, and fluid continues to enter the eye through the one needle, and to leave it through the other. The amount which escapes by filtration is then ascertained by subtracting the backward movement of the one bubble from the forward movement of the other. Now, under these circumstances, although the pressure in the aqueous chamber was as high as in the previous experiments, and the vitreous pressure

was actually higher, the total escape was reduced from 785 to about 50 cubic millimetres in the first half-hour. In the frozen and bisected eye the iris-base was found to be pushed forwards, and in places visibly in contact with the periphery of the cornea. By placing the vitreous reservoir 15 centimetres higher than the aqueous reservoir it was possible to empty the anterior chamber almost completely, and to apply the whole iris closely to the cornea ; and under these circumstances the escape by filtration from the whole eye appeared in some instances to be entirely arrested.

Similar experiments upon the freshly excised eyes of oxen, and in one instance on a freshly excised healthy human eye, removed with an orbital tumour, gave closely similar results.[28]

The last observation, the closure of the filtration-angle by a slight excess of pressure in the vitreous chamber, has an important bearing on the pathology of glaucoma ; I therefore ask your attention particularly to it. Anyone who desires to test the point for himself may do so easily by using the very simple arrangement (Fig. 7) with which all my own earlier experiments were made.

With regard to the escape at the papilla I made some further experiments with the smaller apparatus, shown half-size, in section, in Fig. 6. A circular piece, including choroid, sclera, retina, and optic nerve, is cut from the back of a sheep's eye

[28] Since these experiments were published, Leplat has carried the matter further by experimenting in a similar manner on the eyes of living rabbits. ("Annales d'Oculistique," Jan.-Feb., 1889.) He found, as I had done, that the amount of fluid escaping at the papilla is extremely small as compared with that escaping from the anterior chamber. He found also that injection of fluid into the vitreous of the living eye does not displace the lens forwards to any discoverable extent. This appears to show that, in the normal living eye, a rise of pressure in the vitreous chamber at once produces an equal rise in the aqueous chamber ; it does not show that the aqueous pressure is unaffected by changes in the vitreous pressure. The lens cannot hold its place against any inequality of pressure on its two surfaces. When such an inequality actually arises, as in the artificial conditions of my own experiments, and in certain pathological states, the lens yields easily to the higher pressure.

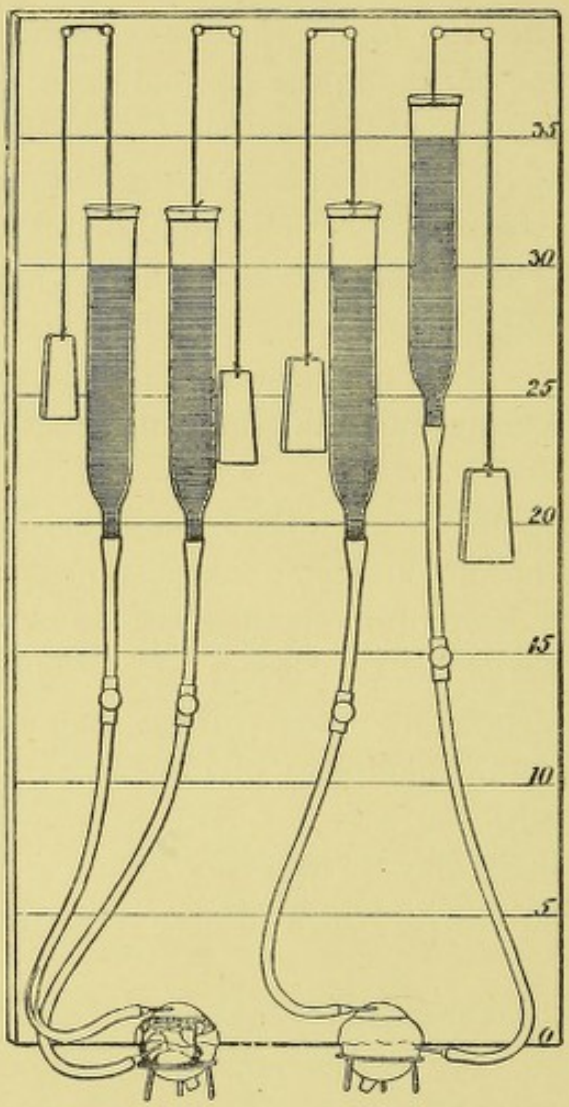


FIG. 7.

Simpler apparatus than the foregoing for demonstrating the escape of fluid from the eye when the aqueous and vitreous pressures are equal, and its non-escape when the vitreous pressure is a little higher than the aqueous. (One-fourth the actual size.)

The eyes of a pig or sheep are excised immediately after the animal is killed, conveyed to the laboratory in a warm closed vessel, and injected both at the same time. The fluid is diffusible, strongly coloured, and preferably of a colour easily distinguishable from that of blood—a one per cent. solution of aniline-black answers well. Care is taken to remove all air from the tubes. The aqueous tube is opened a few seconds before the vitreous tube so as to ensure a full anterior chamber at starting.

(A.)—Both reservoirs stand at 30 cm. above the eye. The fluid in the anterior chamber completely hides the iris. In the course of a few minutes it injects the external vessels and escapes from their cut ends.

(B.)—The aqueous reservoir stands at 30 cm., the vitreous reservoir at 35 cm. The greater part of the iris is hidden by the fluid in the anterior chamber, but the periphery remains visible; it is in contact with the cornea. There is no visible injection of the external vessels, and no escape of the coloured fluid at any part of the eye even after several hours.

immediately after death and placed in the box, and the inner tube is tightly pressed down upon it by screwing on the cap of the box. The inner tube is then filled with coloured fluid and connected by a flexible tube with a graduated glass tube and reservoir, as in the other apparatus. Under a pressure of 30 centimetres, as before, there was no perceptible exudation and no coloration on the outer surface after several hours.

These experiments show several points of importance with regard to the freshly excised eye. They show firstly, that fluid injected into the vitreous chamber escapes chiefly, if not entirely, through the aqueous chamber ; secondly, that a slight excess of pressure in the vitreous chamber displaces the lens and iris forwards, compresses the filtration angle, and impedes the escape of fluid ; thirdly, that there is no escape through the vortex veins when the filtration-angle is compressed ; fourthly, that there is little, if any, escape at the papilla. With regard to the currents in the living eye, they give, of course, no positive information.

If we compare these various observations it is not difficult, I think, to reconcile the apparent discrepancies.

It appears certain, from the movements of solid particles observed by Giffard, that there is a current passing backwards in the vitreous body, and escaping along the lymph-passages which surround the central vessels of the optic nerve. It appears equally certain that this current moves very slowly, and that the amount escaping in a given time is extremely small as compared with that from the aqueous chamber. Whether the small perivascular spaces in the papilla are rendered impervious by excision of the eye, or whether a small quantity of fluid does actually traverse them during artificial injection of the vitreous chamber, it is difficult to determine. In any case the absence of visible filtration here presents a striking contrast with the free escape which occurs at the filtration angle. It remains uncertain, I think, whether there

is a persistent stream from the vitreous to the aqueous chamber. But we have seen that fluid *can* pass very readily in this direction under a slight excess of pressure in the vitreous chamber, and it seems probable that so long as the partition remains permeable, and the fluid diffusible, any excess of pressure in the vitreous chamber will relieve itself in this way. An intercommunication of this kind between the two chambers appears to be necessary for the maintenance of the lens in its normal position.

The lymph passages which, according to Schwalbe, surround the vortex veins and convey lymph from the inner to the outer surface of the sclera, appear to have no connection either with the vitreous or the aqueous chamber. The fluid which is secreted by the ciliary processes after subcutaneous injection in living animals, and which can be traced through the chambers of the eye, by its artificial coloration, has not been found in these channels. The fluid which sometimes escapes through the vortex veins during injection of the anterior chamber in the excised eye appears to reach them through the veins of the iris ; it never passes between choroid and sclera.

Our knowledge of these currents may, then, be briefly summarised as follows :—

The fluids which nourish the vitreous body and lens, and fill the aqueous chamber, are secreted chiefly by the ciliary portion of the uveal tract.

The larger part of the secretion passes directly into the aqueous chamber, forwards through the pupil, and out at the filtration-angle.

A very much smaller portion passes backwards through the vitreous body, and escapes at the papilla.

The hyaloid membrane and zonula which separate the two chambers are readily permeable by the vitreous fluid.

The pressure which the fluid exerts against the walls of the chambers is equal to about 25 millimetres of mercury, and is the same, or very nearly so, in the two chambers.

To conclude this physiological part of the subject, I may point out that while the *circulation* of the intraocular fluid is necessary to the eye as a living organ, its *pressure* is necessary to it as an optical instrument—an instrument which is built entirely of soft materials, but which, nevertheless, can adjust itself with precision both for direction and for distance. In the larger chamber, where there are no moving parts, the fluid is supplemented by a stroma of transparent connective-tissue, which adds to the safety of the organ. In the smaller chamber there is fluid only, so that the curtain which regulates the admission of light may have perfect freedom of movement. In both chambers the fluid exerts that particular degree of pressure which suffices to maintain the form of the globe, and to give precision to the action of the muscles, external and internal, but which does not embarrass the circulation of the blood, the nutrition of the tissues, or the transmission of nerve-currents within the organ.

When the intraocular pressure rises above these physiological limitations, we get that complex disturbance which we call glaucoma.

LECTURE II.

Increase of the Intraocular Pressure: physical illustration of its various possible causes.—Physiological Experiments.—Evidence as to the Actual Causes of Glaucoma: hypersecretion; serosity of the fluids; obstruction of the filtration-angle.—Distinction between Primary and Secondary Glaucoma.—Causes of Secondary Glaucoma in connection with the following conditions: Annular posterior synechia. Perforating wounds and ulcers of the cornea with anterior synechia. Dislocation of the lens into the anterior chamber. Lateral dislocation of the lens. Traumatic cataract. Operations for cataract. Intraocular tumours. Detachment of Retina. Intraocular hæmorrhage and exudation.

INCREASE OF THE INTRAOCULAR PRESSURE.

In my first lecture I spoke of the normal intraocular pressure, and the processes by which it is maintained. I have now to bring to your notice the conditions under which this pressure becomes abnormally increased. It will be well to approach the subject from the physical side, for though we may be unable at present to explain all the phenomena of secretion and excretion by physical laws, we cannot be too careful to build our knowledge of them upon a foundation of this kind.

This apparatus (Fig. 8) will serve to illustrate in an elementary fashion the chief causes of an increase of pressure within the eye. The glass globe filled with water represents the eye. The red rubber tube (A), conveying the water from the reservoir, represents the arteries which bring blood to the ciliary processes. The black tube (V) represents the efferent veins. The short piece (C), which joins them inside the globe, has permeable walls, and permits fluid to escape into the chamber; this represents the capillaries of the ciliary processes. The other tube (O) represents the outlets through which the

intraocular fluid escapes from the eye, of which the angle of the anterior chamber is much the most important. The height of the water in the vertical glass tube indicates the pressure in the chamber. A continuous stream of fluid passes in through the artery and out through the vein ; as it passes the capillaries it gives off a certain quantity into the chamber, and a corresponding amount escapes through the outlet of the chamber.

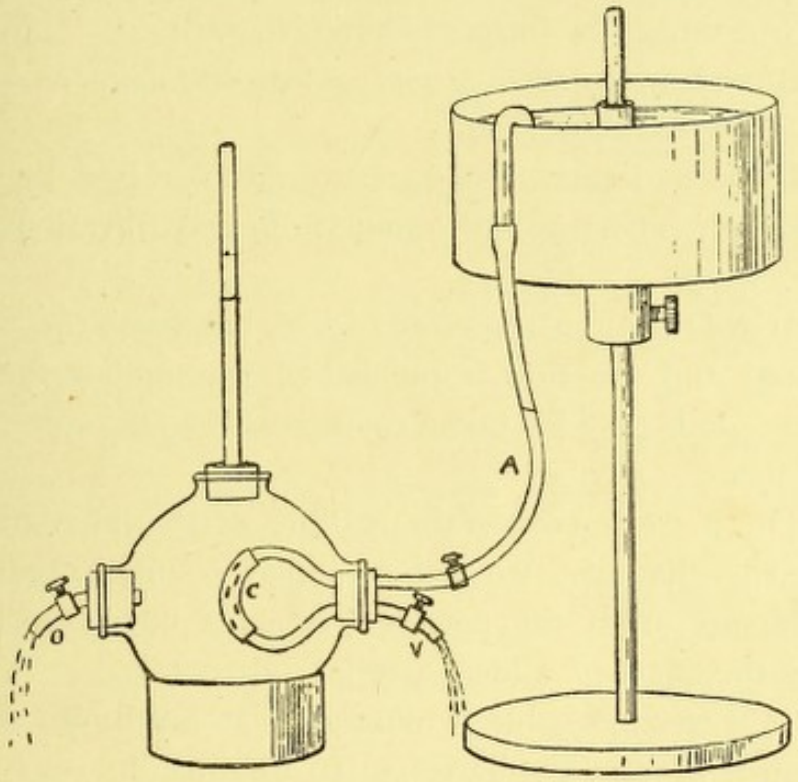


FIG. 8.

Apparatus to illustrate various physical causes of an increase of pressure in the chambers of the eye.

Now the pressure in the chamber may be increased by the action of various causes.

In the first place, it might be temporarily increased by a simple encroachment on the cavity of the chamber. Please to imagine that the vessel has a flexible wall, and that I indent it with my finger. For a moment the internal pressure will be

raised, the inflow checked and the outflow quickened, until compensation is made ; then the pressure will fall to its normal level, and the inflow and the outflow will proceed as before. Or, if one could cause the wall of the vessel to contract upon its contents, or could intrude some foreign substance into it, there would, in like manner, be a temporary rise of pressure, lasting only until compensation was made for the encroachment. Just in the same way the pressure in the eye may be increased by external pressure or by internal hæmorrhage, but the excess will soon disappear, *unless there be some permanent disturbance of the secretion or excretion processes.*

In the second place, the pressure my be raised by various causes directly affecting the circulation and filtration of the fluids.

1.—If we raise the reservoir to a higher level, the pressure is increased and the flow is quickened throughout the whole apparatus. This may be taken to represent a stronger impulse from the heart.

2.—Or, if we increase the calibre of the afferent vessel close to the chamber, we get a similar result, although the initial pressure from the reservoir remains unchanged. This illustrates the effect of a local arterial hyperæmia.

3.—If it were possible to enlarge that portion of the tube which represents the capillaries, or to diminish its resistance to filtration, we could, in that way also, increase the pressure in the chamber.

4.—On the other hand, if we diminish the calibre of the efferent vein, we get, as before, an increased pressure in the capillaries, an increased pressure in the chamber, and a more rapid escape through the outlet of the chamber.

5.—Once more, if we leave untouched the tubes which represent the vascular system, and diminish the outflow from the chamber itself, again the pressure in the chamber rises. This represents an obstruction at the filtration-angle.

6.—Finally, we may cause any two or more of these disturbances to act at the same time. For instance, we may increase the secretion-pressure either by accelerating the arterial, or by retarding the venous, stream, and at the same time, we may diminish the outflow from the chamber. Under these circumstances, the fluid secreted into the chamber will be lessened in quantity but greatly increased in pressure. This would represent hyperæmia of the ciliary processes in association with an obstructed filtration-angle.

Here I must call attention to an important point in which my apparatus fails to represent the living eye. These rubber tubes are sufficiently incompressible to bear with impunity the pressure which falls upon them as they pass through the chamber. The choroidal veins, on the other hand, have thin walls which offer little resistance to external pressure, and it is evident that high pressure in the chambers of the eye must react on the choroidal circulation. The so-called venous pulse often seen on the papilla of healthy eyes, which is really a rhythmical compression of the retinal vein at the point of lowest resistance, due to the arterial wave, shows that the vitreous pressure and the venous pressure are here very nearly balanced. Why, then, does not the higher pressure of glaucoma occlude the retinal vein entirely? Because the pressure of the venous stream necessarily rises when its movement is obstructed. The arterial stream cannot be arrested by any pressure inferior to its own, and can in face of venous obstruction raise the venous pressure to a height nearly equal to its own. In like manner, an excess of pressure falling on the uveal tract tends to compress the vortex veins, to raise the pressure in the veins connected with them, and to congest the ciliary processes; this reaction is very evident in the more acute forms of glaucoma.[29]

[29] Many years ago Körner, of Vienna, and more recently Birnbacher and Czermak, made a number of experiments bearing upon this point. They showed that if a current of fluid be conducted through a closed chamber in a tube of thin and

Now, the pressure changes which I have attempted to illustrate represent the action of well-known physical laws; they could be foretold from a mere inspection of the apparatus.

---

pervious membrane (Fig. 9), such as the intestine of a small animal, the pressure in the chamber gradually arrests the circulation through the tube; the tube becomes dilated near the point (A) where it enters the chamber, compressed near to its point of exit (B). (Von Graefe's Archiv, vol. xxxii., part iv., p. 1.) The cause of the phenomenon is simple. According to a well-known physical law, fluid moving along a horizontal tube of uniform diameter exerts a pressure against the wall of the tube, which decreases as the distance from the reservoir increases. In the case of the membranous tube (A B), which permits filtration along its whole length, the fluid exudes at higher pressure at A than at B; the pressure in the chamber rises until it is equal to the highest pressure in the tube; the further end of the tube is then necessarily compressed. The movement of the fluid through the tube can only be maintained by providing the chamber with an outlet sufficiently large to keep the pressure in the chamber lower than that in the tube.

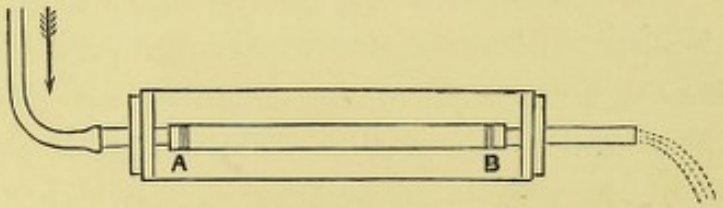


FIG. 9.

Applying this observation to the case of the eye, we might suppose that if the escape of fluid from the chambers were to be arrested the choroidal circulation would be arrested also. But this is not necessarily true. We do not know that the pressure of the fluid in the chambers can rise to a height equal to that of the blood in the ciliary processes, and we do not know that the pressure in the vortex veins is less than that in the ciliary processes. The law stated above refers only to tubes of uniform calibre. In tubes of varying calibre the pressure at any given point is modified by the diameter at that point, being increased by a larger diameter, diminished by a smaller. For example, in a tube having the form shown in Fig. 10, the

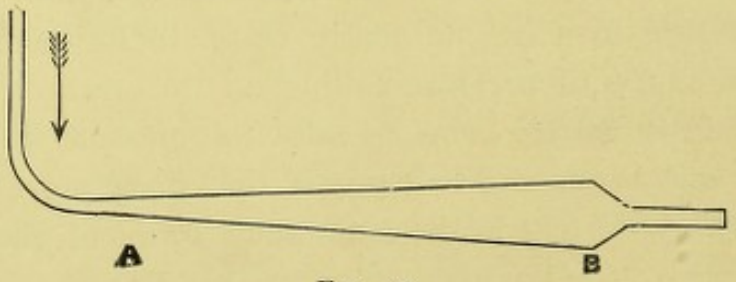


FIG. 10.

pressure may be greater at B than at A. The pressure in a vein is not necessarily lower than that in the corresponding capillaries. It is possible that the aggregate

In the living eye the conditions are, of course, much more complex. There we have to deal not with a mere filtration of fluid, but with a more subtle process. The secretion which enters the chambers differs from the fluid circulating in the vessels, and under certain conditions its own constitution changes and its filtration power is lessened. Here, again, is a possible cause of pressure change. But, in spite of their greater complexity, the processes in the living eye are unquestionably governed by the same physical laws, and even this rough analysis may serve to illustrate the directions in which we should seek the causes of an abnormal intraocular pressure.

Passing now to physiological experiment, we find that pressure changes do actually occur in the living eye from such causes as have just been described. Experiments on the lower animals have established the following facts :—

An increased flow of blood to the head, produced by compression of the descending aorta, raises the intraocular pressure in a marked degree.

A local dilatation of the ophthalmic artery, induced by irritation of the fifth nerve within the skull, raises it in a still more decided manner, and if this nerve be irritated during compression of the aorta, an additional rise occurs.

Venous obstruction in the neighbourhood of the eye produces a similar effect. Ligature of the vortex veins outside the eye may raise the pressure to more than twice its normal height. Obstructions at a greater distance from the eye have

---

sectional area of the choroidal veins may be so proportioned to that of the capillaries that the pressure in the former may be greater than that in the latter, in which case the choroidal circulation can never be absolutely arrested by the pressure in the chambers. As a matter of fact, the veins of the choroid are remarkably large and numerous where they have to bear the intraocular pressure, while at the points where they perforate the sclera and escape from the pressure they show a sudden diminution in calibre. This looks like a special adaptation.

much less effect ; for instance, compression of the large veins of the neck, and even ligature of the superior vena cava, raise the intraocular pressure but little.[30]

Obstruction of the outlets through which the intraocular fluid escapes can hardly be made the subject of satisfactory experiment in the living eye. We have seen that the chief outlet is at the angle of the anterior chamber, and that a very much smaller escape occurs at the papilla. With regard to the papilla, the results of experiments are negative : ligature of the optic nerve produces no noteworthy change in the intraocular pressure ; and as we know that papillitis, with swelling of the disc, constriction of the vessels, and dropsy of the sheath, is not commonly associated with any discoverable rise of tension, we may safely conclude that the posterior outlet cannot have much influence on the intraocular pressure. With regard to the filtration-angle, my own experiments on the freshly-excised eyes of animals, described in the previous lecture, have shown that artificial closure of this outlet almost entirely stops all filtration from the chambers ; and this fact has been confirmed by Leplat's more recent experiments on living animals.[31]

Changes in the constitution of the intraocular fluids, as a cause of increased pressure, are also almost beyond the reach of satisfactory experiment, but it is easy to prove that albuminous fluids escape from the eye much less easily than the normal intraocular fluid. Using the apparatus exhibited in my former lecture (page 22), and experimenting with sheep's eyes immediately after the death of the animal, I found that a limpid albuminous fluid drawn from the abdomen in a case of dropsy escaped from the anterior chamber much less rapidly than a normal salt solution.

---

[30] "Graefe-Saemisch Handbook," vol. ii., p. 371.

[31] "Annales d'Oculistique," Jan.-Feb., 1889.

From these observations, physical and physiological, we may safely conclude that the chief conditions which are capable of causing persistent high pressure in the chambers of the eye are the following:—

(*a*) Hypersecretion by the ciliary processes.
(*b*) Serosity of the fluids.
(*c*) Obstruction at the filtration-angle.

Moreover, we can understand how, in the presence of such disturbances, the inflow and the outflow act and react upon each other. It is obvious that, except when the eye is actually altering in size, the inflow and the outflow must be equal under all degrees of pressure, high or low. Now comes the question, What evidence have we of such disturbances in the various forms of glaucoma?

EVIDENCE AS TO THE ACTUAL CAUSES OF INCREASED PRESSURE IN GLAUCOMA.

Hypersecretion.—What evidence have we of hypersecretion? We may expect it to arise from some kind of irritation in the region of the fifth nerve, and from dilatation of the ciliary vessels.

More or less pain in the fifth nerve is usually present during glaucomatous attacks, and occasionally the neuralgia is distinctly antecedent to the outbreak of the glaucoma.[32] Some attacks are referable to injuries which presumably involve nerve disturbance in the brain or in the eye itself.

Ciliary hyperæmia is almost always present in the more acute forms of glaucoma, and is often very intense. In certain cases vascular engorgement is manifestly the cause of an increase of tension. For example, in persons who have had premonitions of glaucoma, or who have been partially relieved

[32] See case recorded by Hutchinson, R.L.O.H. Reports, vol. iv., p. 127.

by rest in bed or other palliative measures, any disturbance which flushes the face or congests the head is apt to bring on a recurrence; indeed, there are cases in which the surgeon can tell from day to day, by the colour of the patient's face, whether he will find the eye tense or slack. In such instances we may safely ascribe the glaucoma, in part at least, to nervous and vascular disturbance, and perhaps to hypersecretion.

On the other hand, the pain and injection which characterise acute glaucoma appear usually to be consequences, more than causes, of the high pressure in the globe, for they subside quickly when the excess of fluid is allowed to escape. In many cases there is no evidence whatever of antecedent nervous or vascular disturbance; as, for example, when an intense glaucoma follows the use of a drop of atropine solution to an apparently healthy eye. Moreover there is sometimes no sign of nerve-irritation or active hyperæmia throughout the whole course of the disease.

Further, we see many persons suffering from well-marked congestion of the head and eyes who show no tendency to glaucoma, and we see countless eyes with injected ciliary vessels which have the same immunity.

We find that trigeminal neuralgia even when very severe and when accompanied by watering and injection of the eye does not, as a rule, but only as a rare exception, induce glaucoma. Dental caries causes a very severe peripheral irritation of the fifth nerve, which certainly sometimes induces reflex disturbance in the eye; but there is not, as a rule, any discoverable increase of the intraocular pressure. Hermann Schmidt,[33] finding that paroxysms of tooth-ache appeared to diminish the range of accommodation, assumed that this was due to an increase of pressure within the chambers. In pursuit of this idea I made careful measurements with the tonometer at

[33] "Von Graefe's Archiv," vol. xiv., part 1, p. 107.

the Birmingham Dental Hospital, in sixteen persons who were suffering at the moment, or had very recently suffered, from severe pain in the teeth. I found a complete absence of pressure change.[34]

It is possible, of course, that hypersecretion of the intraocular fluids may occur through the action of some undiscoverable nerve-irritation or vascular change, but this is pure hypothesis. So far as clinical observation enables us to judge, nervous and vascular disturbances, act under certain exceptional conditions, as exciting causes of glaucoma, but do not in themselves suffice to account for the high pressure in the chambers. The hypothesis that glaucoma is the expression of a persistent hypersecretion remains a hypothesis and nothing more ; and, as Adolph Weber has pointed out,[35] it is insufficient even as a hypothesis. A rise of pressure, due simply to hypersecretion, would tend to correct itself by a gradual dilatation of the outlets, whereas the glaucoma process tends to intensify itself. And, more important still, the idea of a persistent hypersecretion is negatived by the presence in glaucomatous eyes of certain changes, to be described immediately, which render hypersecretion a physical impossibility.

Serosity of the Intraocular Fluids.—Secondly, what evidence have we of serosity of the intraocular fluids as a cause of high pressure ? I have already mentioned an experiment which shows that albuminous fluid escapes from the anterior chamber much less easily than a normal salt solution. (See page 36.) Now, in all forms of iritis there is probably more or less albuminous exudation into the aqueous chamber, but so long as the inflammation is limited to the iris, there is usually little alteration in the tension of the eye, for the aqueous humour comes, as we have seen, almost exclusively from the ciliary processes, and is

---

[34] "Glaucoma, its Causes, etc.," 1879, p. 11.

[35] "Von Graefe's Archiv," vol. xxiii., part 1, p. 1.

secreted so copiously that a slight morbid admixture from the iris does not greatly affect its filtration power; but if the ciliary processes are involved changes of tension are sure to follow. If the inflammation be intense, with plastic or suppurative exudation, the secreting organ is rapidly impaired and minus tension soon sets in; if, on the other hand, it is comparatively mild, with serous exudation, then we get the cloudy aqueous, the punctate deposits on the cornea, the dilated pupil, and the high tension which characterise the so-called serous iritis, a condition which should, I think, be called serous cyclitis. Fig. 11 was drawn from a typical specimen kindly given to me some years ago by Mr. McHardy. The filtration-angle, far from being closed,

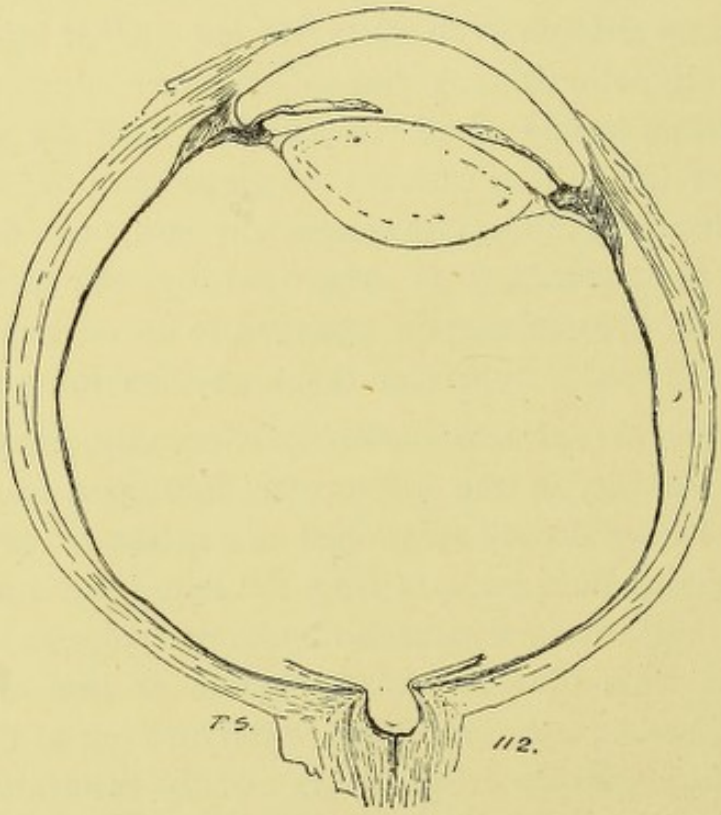


FIG. 11.

From an eye blinded by high tension due to serous cyclitis.
Glaucoma with a widely open filtration-angle.
Filtration retarded by serosity of aqueous fluid.
(Mr. McHardy's case.)

is widely distended ; the disc is deeply cupped. This is a glaucoma which owes its high tension to the serous nature of the fluid, and also perhaps to tissue changes around the filtration-angle. In many other forms of glaucoma serosity of the fluids is probably a supplementary cause of the high tension, for whenever the circulation of the blood is obstructed serum is apt to escape from the capillaries. In many eyes blinded by high tension, we find, after hardening and bisection, signs of serous exudation into the aqueous and vitreous chambers.

Obstructed Excretion.—Finally, what evidence have we of obstruction at the filtration-angle? In the year 1876 Max Knies[36] and Adolph Weber,[37] following the clue given three years earlier by Leber, and working independently of each other, pointed to a well-marked obstructive change at the angle of the anterior chamber. This discovery marks an epoch. In my opinion, the most important addition to our knowledge of the subject since the time of von Graefe is to be found in Weber's work, "Die Ursache des Glaucoms." Hundreds of eyes have now been examined, and in the very large majority this obstruction of the filtration-angle has been found. I have myself examined with the microscope more than eighty eyes, representing glaucoma of many varieties, and, with the exception of three or four, of which I will speak immediately, I have found the filtration-angle compressed or closed in every instance.

To emphasise the importance of this pathological change, I may remind you of the experiments described in my first lecture, which proved that the escape of fluid from the anterior chamber varies greatly in amount according as the filtration-angle is more or less widely open, and that when this outlet is closed the escape from the whole eye is almost completely arrested.

---

[36] "Von Graefe's Archiv," vol. xxii., part 3, p. 163.

[37] "Von Graefe's Archiv," vol. xxiii., part 1, p. 1.

It must be noted also that Leber has proved by injection-experiments that fluid escapes with great difficulty from eyes which have been blinded by glaucoma.[38]

Now this obstruction of the filtration-angle is sometimes found in eyes which have no history and present no sign of glaucoma, and on this ground some writers have urged that it cannot be an essential factor in that disease. But in such eyes we can always find, I think, other changes which explain the absence of the glaucoma. I have many specimens of the kind in my collection. In some the fluid appears to have found an abnormal outlet. Fig. 12 was drawn from an eye in which the

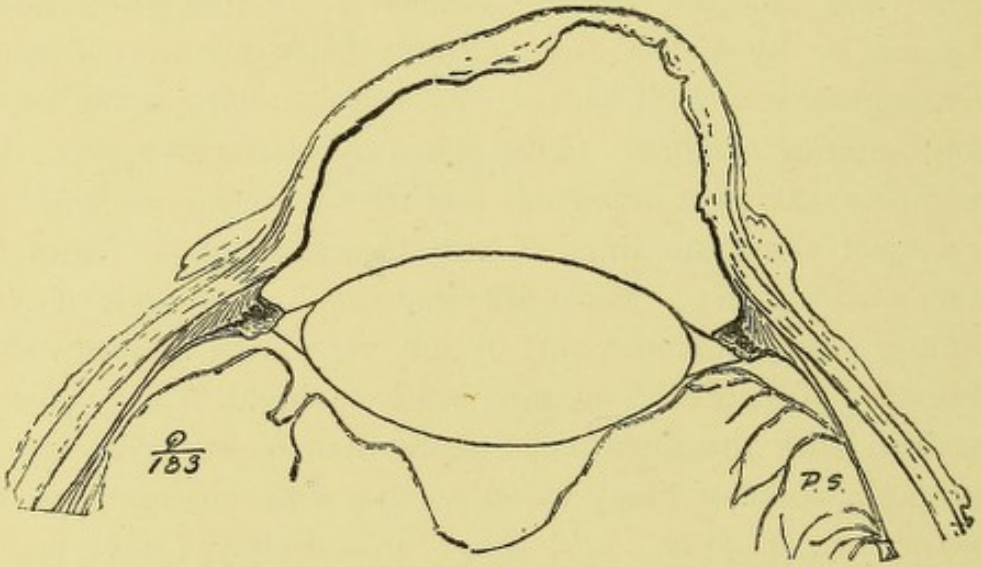


FIG. 12.

From an eye lost by suppurative keratitis leading to staphyloma. Abolition of the filtration-angle, but no glaucoma. The fluid has probably found abnormal outlets.

cornea had been destroyed by suppuration and replaced by new tissue, lined throughout by the remnant of the iris. Here the filtration-angle is abolished, and yet there has apparently been no glaucomatous tension, for the disc shows no trace of excavation. The secretion of the intraocular fluid has not

[38] Report of Seventeenth Meeting of Ophth. Soc. of Heidelberg, p. 131.

been suppressed, for the retina is not detached, and the soft pseudo-cornea has been distended by pressure from within. I suspect that in such cases the intraocular fluid finds exit through some part of the pseudo-cornea. Vessels permeate the new tissue ; the endothelium, which, as Leber has shown, renders the healthy cornea impervious, is here destroyed ; and the lining of iris tissue is extremely attenuated. Normal tension in such eyes is, however, the exception rather than the rule. (See page 49.) We very frequently find, together with the distension of

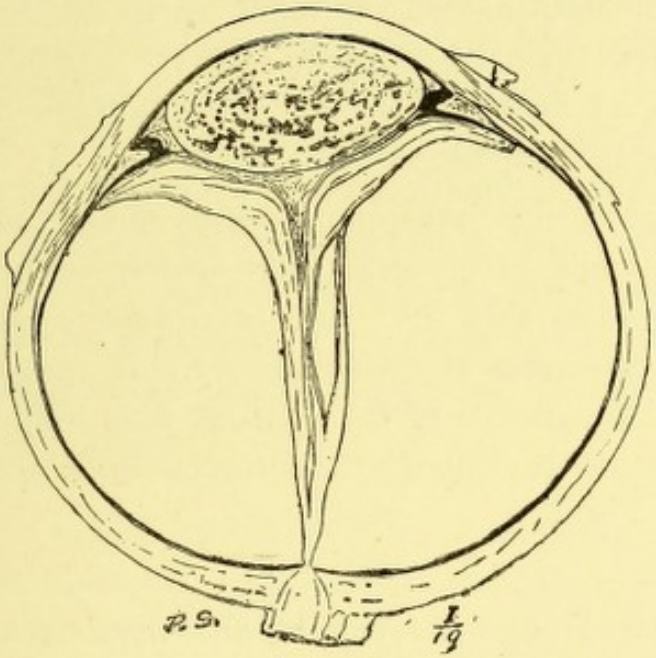


FIG. 13.

From an eye lost by cyclitis and vitreous-infiltration after fever, six years before excision. Abolition of filtration-angle, but no glaucoma. Secretion of aqueous and vitreous fluids suppressed.

the cornea, an enlargement of the whole globe, and, on dissection, an excavation of the disc, manifestly due to high pressure in the chambers. These differences of tension probably indicate differences in the permeability of the pseudo-cornea and in the integrity of the secreting organs.

In other specimens presenting closure of the filtration-angle without glaucoma (see Fig. 13) secretion has evidently

been arrested. When the ciliary processes are damaged by inflammatory changes the vitreous shrinks, the lens degenerates vicarious exudations fill the spaces which are left, and the eye becomes too soft rather than too hard. Fig. 13 was taken from an eye which was blinded by cyclitis and infiltration into the vitreous, after fever, six years before excision. The vitreous body has disappeared, the lens is a white degenerated mass, the aqueous chamber is abolished—the normal secretion-process has been totally suppressed. In such an eye glaucoma is impossible, unless, indeed, through some accident, a blood vessel ruptures ; then blood pours into the closed eyeball, and the tension may be very high for a considerable time, for the outlets, long disused, have become useless.

Again, in a few exceptional cases of glaucoma we find the filtration-angle open—even more widely open than in the healthy eye—and this also has been used as an argument against the so-called retention theory. It has been shown already that the escape of the aqueous fluid may be retarded by its own serosity, although the filtration-angle itself is widely open. (See Fig. 11.)

It appears from the foregoing that nervous and vascular disturbances, and presumably hypersecretion, are often concerned in the onset of glaucoma ; that serosity of the fluids plays an important part in those forms which present a deep anterior chamber and a wide filtration-angle, and probably in other forms as well ; and that closure of the filtration-angle is a part of the glaucoma-process in the great majority of cases. Now some writers on ophthalmology tell us that this last named change—the closure of the filtration-angle—has no very direct or essential connection with the high pressure of glaucoma. We are informed that it occurs at a late stage of the disease ; that it is a consequence, rather than a cause, of the high

pressure ; that the essential cause of the high pressure still remains to be discovered. Laying aside generalities, let us, so far as it may be possible, examine each variety of glaucoma in turn, and ascertain its mode of onset.

Distinction between Primary and Secondary Glaucoma.—Here it will be convenient to divide all forms of glaucoma into two groups, the primary and the secondary, and a very simple definition will suffice to distinguish them :—The primary forms are those in which we cannot see any previous disease in the eye which accounts for the glaucoma ; the secondary are those in which we can see such a disease. Some cases occupy a doubtful position between these two groups, but as a rule the distinction is easily made, and it is certainly useful. Beginning with causes which are visible rather than with those which are invisible, I shall endeavour, in the first place, to sketch the sequence of events in various forms of secondary glaucoma, and I am able to lay before you specimens and drawings of nearly all the forms described.

CAUSES OF SECONDARY GLAUCOMA.

Annular Posterior Synechia.—After repeated attacks of iritis, the pupil margin remains widely adherent to the lens. The surgeon, foreseeing the evil consequences of complete exclusion of the pupil, proposes iridectomy, but the patient refuses, for as yet vision is fairly good, the tension is normal, and there is no pain. Some months later the patient returns, the eye now being painful, injected, and hard, and the iris bulging forwards against the cornea. An iridectomy is at once performed ; fluid escapes freely from behind the iris ; the iris returns to its normal plane ; and the glaucoma is gone. Or, if the eye be already blind, we remove it and find the iris-base widely in contact with the cornea, as shown in Figs. 14 and 15. Here the cause of the glaucoma is obvious. The passage from

the posterior to the anterior aqueous chamber becomes closed or insufficient; the iris is pushed forwards by the fluid imprisoned behind it; the filtration-angle is closed, and with that closure high tension sets in.

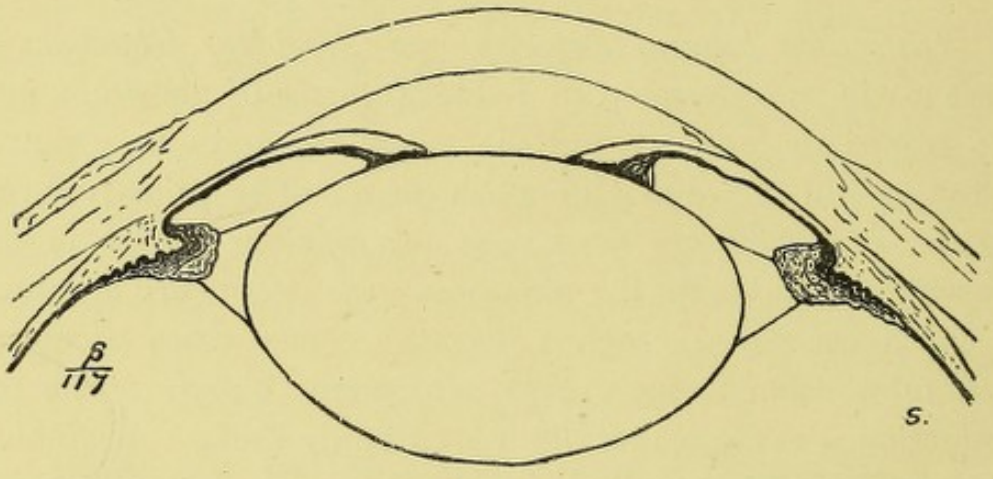


FIG. 14.

From an eye blinded by secondary glaucoma following neglected iritis. Exclusion of the pupil; accumulation of fluid in the posterior chamber; displacement of the iris; closure of the filtration-angle.

When there is a hope of restoring useful vision by iridectomy, a considerable aperture in the iris should be made if possible, but even a very small opening may suffice to liberate the imprisoned fluid, to re-open the filtration-angle, and permanently banish the high tension. In a case of my own, in which the bulging iris was widely in contact with the cornea, I punctured the iris through the margin of the cornea, but was unable, the patient being unsteady, to seize or excise any portion of it. The bulging quickly disappeared, however, and the high tension did not return.

In some of these cases the tension remains permanently sub-normal after the operation, showing that the ciliary processes are permanently damaged: they have sufficient secretory power to produce high tension while the outlet is obstructed, but, when it re-opens, their insufficiency becomes manifest. In eyes

blinded by this form of glaucoma, a complete detachment of the retina is frequently found when the excised eye is opened—a further consequence of disease in the ciliary processes. (Fig. 15.)

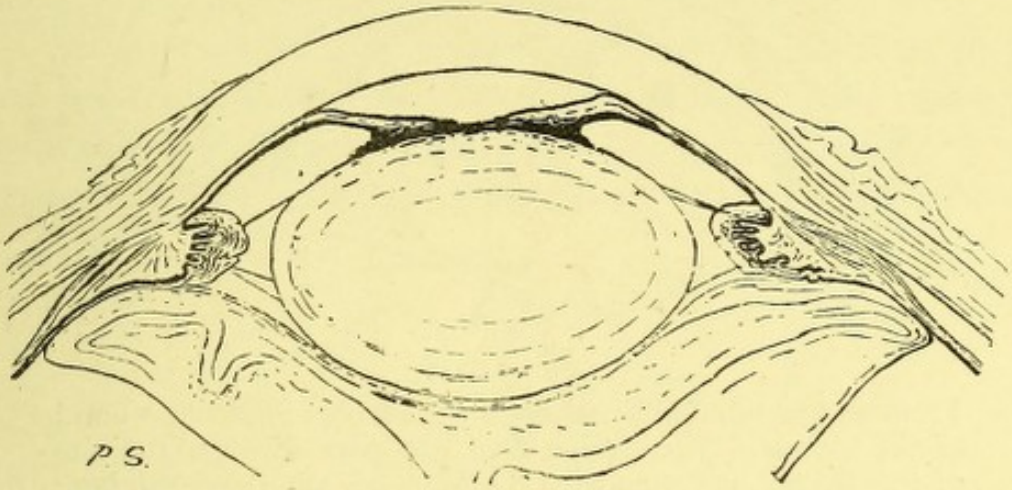


FIG. 15.
The same changes as in Fig. 14 together with complete detachment of the retina. (4-409.)

Perforating Wounds and Ulcers of the Cornea with Anterior Synechia.—The anterior chamber is opened by a wound or ulcer, the aqueous escapes, the iris applies itself to the cornea and prolapses through, or adheres to the margins of, the wound. So long as the aperture remains open, the aqueous drains away and the eye remains soft; when it closes it becomes hard, for the aqueous fluid has now insufficient access, or no access, to the filtration-angle. If, as sometimes happens, the lens is carried forward so as to quite abolish the anterior chamber, the re-establishment of the normal current between iris and cornea is hardly possible. Fig. 16 was drawn from an eye excised six weeks after receiving a jagged wound in the cornea. The tension was very high. The anterior chamber had not been re-established: the filtration-angle remained closed. The blockade appears to have been confirmed and rendered irremediable by inflammatory exudations into the posterior chamber.

A puncture through the ciliary region had been made by the surgeon who first saw the patient, with the object of relieving the high tension, but with no permanent effect.

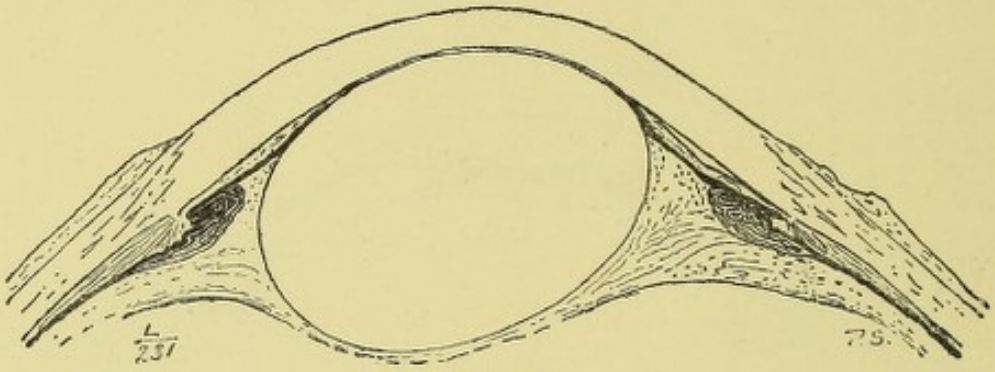


FIG. 16.

From an eye blinded by secondary glaucoma following wound of the cornea. The section does not pass through the site of the wound. Permanent abolition of anterior chamber; access of fluid to filtration-angle completely cut off.

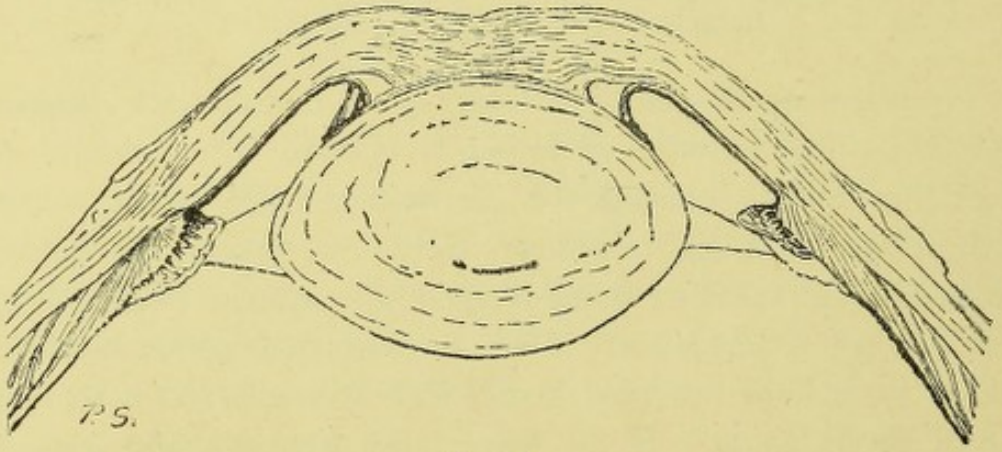


FIG. 17.

From an eye blinded by secondary glaucoma following central perforating ulcer of the cornea. Permanent abolition of anterior chamber; access of fluid to filtration-angle completely cut off. (P. 52.)

Fig. 17 was drawn from an eye blinded by glaucoma, following central ulcer of the cornea. Here the lens had become adherent to the corneal cicatrix and the pupillary

margin of the inflamed iris had adhered to the lens. When the leakage stopped, the lens retired a little from its advanced position, the adhesions becoming stretched, and the iris being doubled upon itself and pushed forwards by the imprisoned fluid. Here again the filtration-angle is occluded.

Staphyloma of the cornea, partial or total, is a very frequent result of secondary glaucoma, arising in the manner above described from perforation of the cornea. The disaster is especially frequent as a sequel of neglected purulent ophthalmia

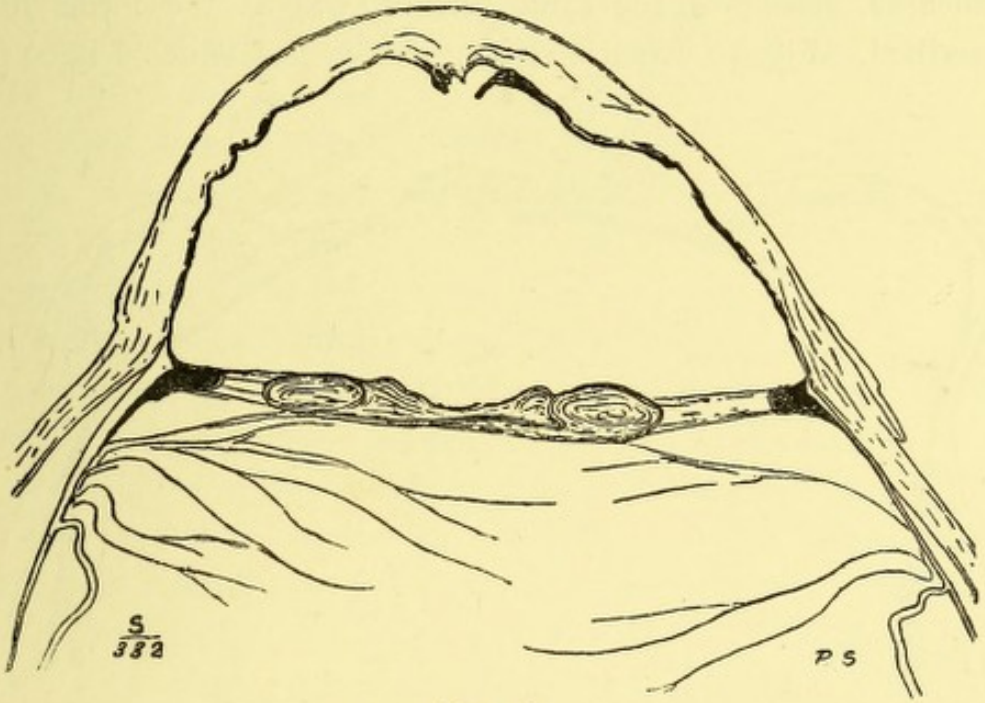


FIG. 18.

From an eye blinded by secondary glaucoma, following central perforating ulcer of the cornea in an infant. Abolition of anterior chamber, as in Fig. 17. Corneal staphyloma with distention of the globe (antero-posterior diameter, 31 millimetres; transverse, 27). Disc deeply cupped. Disorganisation of the lens through adhesion with cornea, and subsequent separation, with rupture of its capsule.

and phlyctenular ulcer in children, and at this time of life the sclera, being far less resistant than in the adult, is distended by the increased pressure, and the globe enlarges in all diameters.

Fig. 18 was drawn from the greatly distended eye of a child, aged 5. The history showed that the eye had been first inflamed when the child was four months old, and had been under treatment a month later for corneal ulcer. The changes are an exaggeration of those seen in Fig. 17; the lens has adhered in the early stage of the disorder to the centre of the cornea, but has been torn from it and thereby disorganised during the distension of the tunics.

Ciliary staphyloma is a less frequent result of secondary glaucoma, arising in the same kind of way as the forms just described. Fig. 19 was drawn from an eye of which I have no

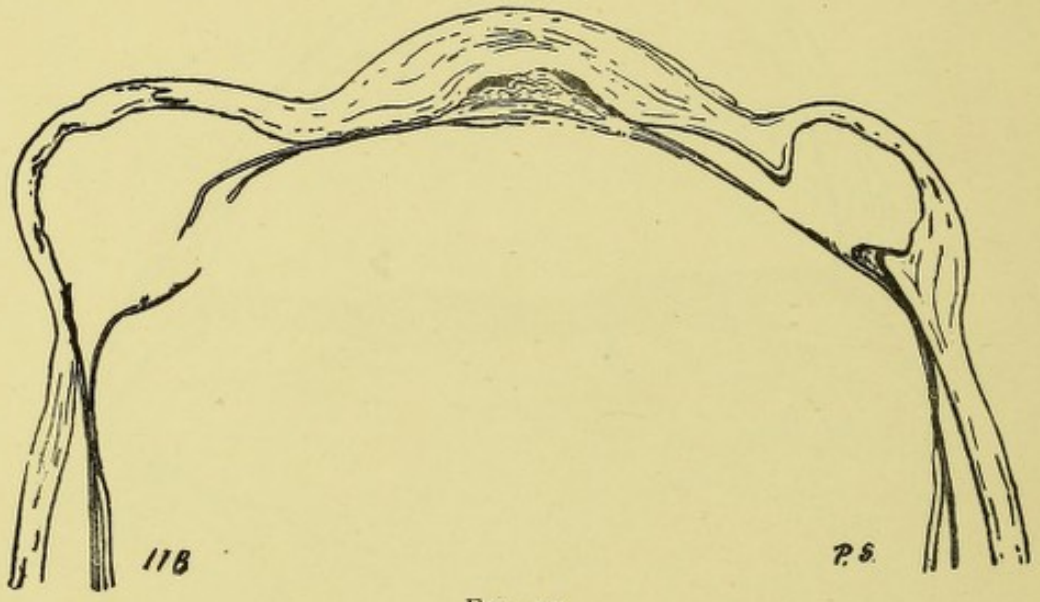


FIG. 19.

From an eye blinded by secondary glaucoma, probably following wound or ulcer of the cornea. Three staphylomata in the ciliary region; the section passes through two of them. These and the cornea are lined throughout by the attenuated iris. Remnants of lens adhere to cornea. Disc deeply cupped. The thickened cornea has withstood the pressure, while the ciliary region, weakened at certain spots by inflammatory changes, has yielded.

(Mr. McHardy's case.)

history, except that it had high tension at the time of excision. The disc was very deeply cupped, and to a certain extent the changes tell their own tale.

In the advanced stages and severer forms of this morbid process, when the anterior chamber is irrecoverably lost, as seen in Figs. 16, 17, 18, and 19, no operative treatment is likely to give relief short of excision, or perhaps evisceration, of the globe, but in the earlier stages an iridectomy will in some cases both reopen the pupil and re-establish a large part, at least, of the anterior chamber, and thereby banish the high tension, and preserve useful vision.

Staphyloma is not necessarily associated with glaucoma. Any part of the wall of the eye, probably, which has been much weakened by morbid changes, may become distended under the action of the normal intraocular pressure.

Dislocation of the Lens into the Anterior Chamber.—Both lenses have been imperfectly suspended and oscillating from birth, but the patient has never had pain or inflammation in the eyes. One day, while stooping over the wash-hand basin, she suddenly feels something wrong in one eye. Within an hour an intense glaucoma is established. The surgeon sees that the lens is in the anterior chamber; that outside the margin of the lens the iris is in close contact with the cornea, and that the rest of the iris bends backwards over the posterior surface of the lens. An operation is proposed, but before it can be carried out the lens slips back through the pupil, and the glaucoma is gone. The dislocation recurs several times, and each time with glaucoma. Ultimately the lens is extracted and the glaucoma appears no more.[39]

In another case of spontaneous dislocation followed by intense glaucoma, the eye, having been nearly blind before the accident, is excised. The conditions seen on bisecting the frozen globe are shown in Fig 20, and a microscopic section through the ciliary region in Fig 21. Here again the filtration-angle is closed in a manner which cannot be misunderstood. Under

[39] For details of such a case see "Glaucoma, its Causes, etc.," pp. 262, 263.

normal conditions the intraocular fluid meets with no resistance in passing forwards through the pupil, but no sooner is the relation of the iris and lens reversed, than the stream which previously kept the passage open closes it with a constantly increasing pressure. The iris is firmly applied to the posterior surface of the lens ; its periphery, where not supported by the lens, is driven forwards against the cornea. In some cases, as we have seen, the high pressure effects its own cure by driving the iris forwards so strongly that the distended and paralysed sphincter offers no resistance to the return of the lens when the

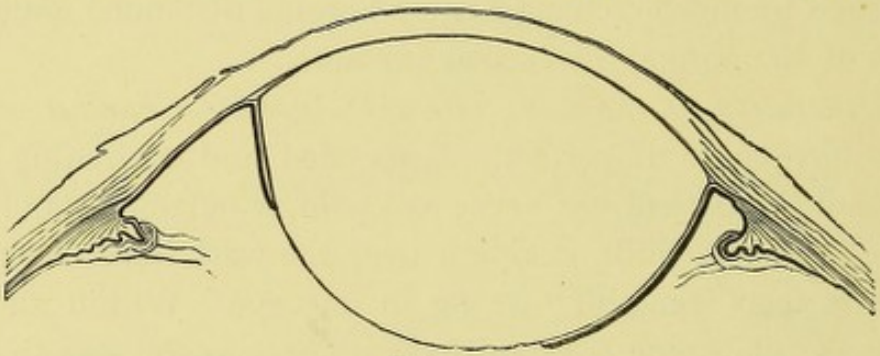


FIG. 20.

From an eye blinded by secondary glaucoma, following spontaneous dislocation of the lens into the anterior chamber. The patient, a tailor, aged 55, had retinitis pigmentosa in both eyes ; this eye had been nearly useless for some years, but never red or painful. A sudden pain in it occurred while he was stooping over his work, and became more severe from day to day. Excision on the ninth day, the eye being very hard, greatly injected and without perception of light. Access to filtration-angle cut off. Iris and lens firmly pressed against the cornea and moulded to its curvature by the pressure of the imprisoned fluid. (See "Ophthalmic Review," 1882, p. 209).

patient lies upon the back. The great force exercised by the fluid in such cases is shown by the remarkable moulding of the lens to the curvature of the cornea. I have two such specimens in my collection. In other instances this same displacement occurs without glaucomatous complication : there is no pain, no injection, and no rise of tension. In such cases I believe it will always be found that the pupil is not entirely

occluded by the lens; a passage remains through which the fluid can still pass into the anterior chamber, and the base of the iris is not pushed forwards around the lens-margin. One case has been placed on record in which no rise of tension occurred until, by the use of eserine, the iris was contracted and tightened up against the posterior surface of the lens; then an acute glaucoma at once supervened.[40] This suggests the practical lesson that if eserine be employed in such cases to facilitate the extraction—and it is very useful for that purpose—it should be applied only a short time before the operation.

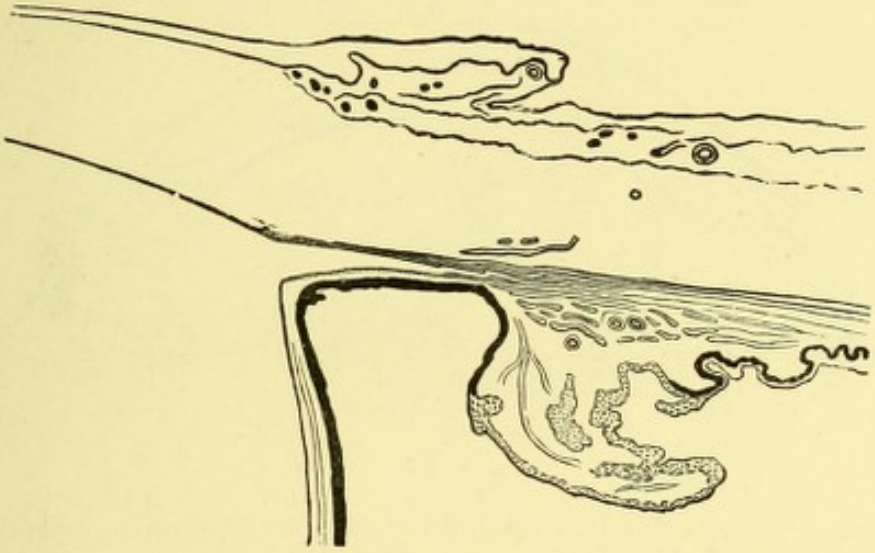


FIG. 21.

From same eye as Fig. 20.
Section through ciliary region enlarged.

It is hardly necessary to add that not every glaucomatous eye which presents a lens in the anterior chamber has become glaucomatous in the manner here described. In other forms of glaucoma the lens, usually degenerated and shrivelled, may fall forwards through the pupil.

Lateral Dislocation of the Lens.—The patient receives a severe blow upon the eye. When the swelling of the lids has

[40] J. J. Minor, "New York Medical Journal," 1881, p. 194.

subsided so far as to permit thorough examination, we find the tension increased and the vision much impaired. The pupil is dilated, but less so at one side of the circle than elsewhere, and at the less dilated side the iris lies very near to the cornea, or in contact with it. Looking into the eye very obliquely with the ophthalmoscope we see the edge of the lens behind the more

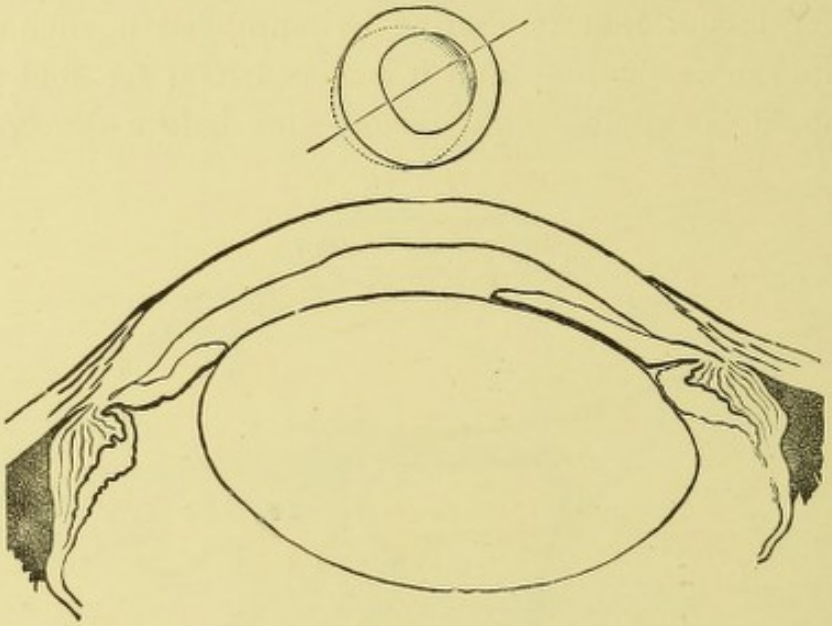


FIG. 22.

From an eye blinded by secondary glaucoma, following lateral dislocation of the lens by a blow. The smaller figure shows diagrammatically the direction of the dislocation, the alteration of the pupil, and the direction in which the frozen globe was bisected. In the larger figure the specimen is reversed as regards right and left. Filtration-angle completely closed at the one side of the circle by direct pressure of lens against ciliary processes and iris; at other parts of the circle it has probably been more or less compressed by the displaced vitreous. (See "Ophthalmic Review," 1883, p. 257.)

dilated side of the pupil; it is dislocated laterally, and bears at the one side against the ciliary body. Possibly we are able to reduce the tension by means of eserine and cocaine, and a useful eye is retained for years; or in view of irreducible

tension, we attempt to remove the lens, and perhaps succeed. Possibly the patient declines operation until excision of the hard and painful eye becomes inevitable.

I have been able to examine two specimens of this kind. Figs. 22 and 23 were taken from one of these. The lens was pressed against the processes and iris at the one side of the circle, and very firmly pressed, for it was indented by the processes. It was wedged in, so to speak, between the ciliary body in front and a consistent vitreous behind. The filtration-angle appeared to be closed, not only where it was compressed

FIG. 23.

From same eye as Fig. 22.
Section through ciliary region enlarged.

by the lens, but more or less throughout the whole circle. It seems probable that the vitreous body, displaced at the one side by the intrusion of the lens, was driven forwards at the other in such a way as to effect this closure.

Injury of the Lens.—In performing preliminary iridectomy in a case of senile cataract, the surgeon accidentally touches the lens with the point of the keratome. Twenty-four hours later the eye is very hard; the incision is healed; the lens is swollen and manifestly presses the iris against the cornea throughout a large part of the circle. The cataract is extracted

without delay, and the eye recovers perfectly, with normal tension and a good anterior chamber. In the same way swelling of the lens after a needle operation for cataract sometimes causes compression of the filtration-angle and high tension; and in the same way evacuation of the lens matter, or even the escape of a portion from within the capsule into the anterior chamber relieves it. I have had no opportunity of dissecting an eye blinded in this manner; no surgeon permits the glaucomatous complication in such a case to go unrelieved.

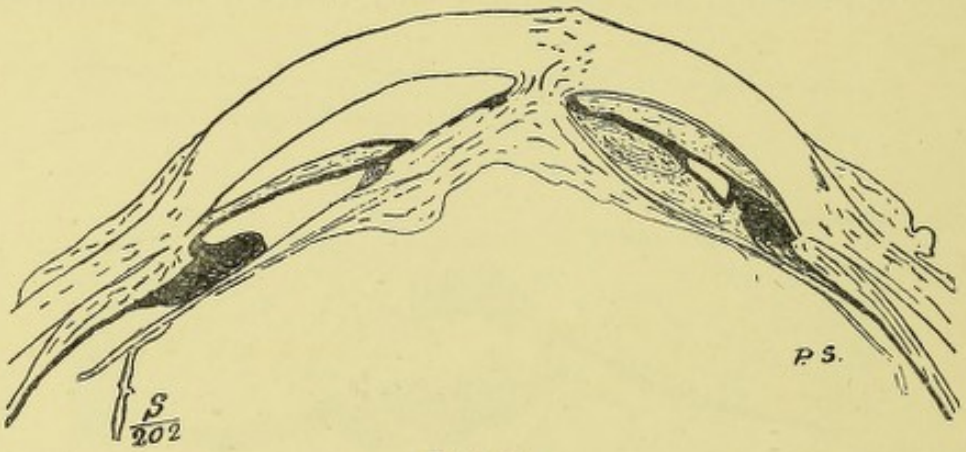


FIG. 24.

From an eye affected with secondary glaucoma, following a puncture of the cornea and lens with a pair of scissors. Synechia; adhesion of capsule and vitreous to corneal cicatrix; closure of filtration-angle. Disc deeply cupped.

Ordinary wounds of the eye which involve the lens often lead to glaucoma, though here the manner of its occurrence may be less easy to distinguish, for the swelling of the lens is commonly complicated with synechia or prolapse of the iris, or with inflammatory exudations into the chambers. Fig. 24 was drawn from the glaucomatous eye of a boy aged sixteen. It had been injured seven years before excision by a puncture with a pair of scissors. Some operation had been performed, but no accurate history was obtainable.

The following case, the notes of which were kindly given to me by Mr. Lloyd-Owen, appears to show that, under excep-

tional circumstances, an acute glaucoma may depend on firm plugging of the pupil by lens-matter. A boy, aged seven, presented in one eye a cataract, probably of inflammatory origin, but with no sign of present inflammation ; the pupil was only moderately dilatable by atropine; perception of light and projection were good. A needle-operation, the centre of the anterior capsule being opened, was followed next day by a severe acute glaucoma. An iridectomy done forthwith banished the glaucoma, and showed that the cataractous lens was of small size, and pressed against the iris only at the margin of the pupil. The iris was remarkably thick and tough, and the bulging lens-matter forced the capsule into the rigid pupil in a manner recalling the protrusion of the membranes through a rigid *os uteri* during labour.

Removal of the Lens.—Secondary Glaucoma sometimes occurs after the lens has been removed by operation. The manner of its occurrence is not the same in all cases.

In an infant, four months old, I needled both lenses for lamellar cataract.[41] Both pupils cleared centrally, but remained small through posterior synechia. Seven years later, I saw the child again with high tension in one eye. The history showed that the high tension was probably of recent occurrence. Vision was still useful, but deteriorating. The pupil was small, undilatable, and deeply seated ; the rest of the iris bulged forwards against the cornea. A small iridectomy gave exit to the fluid retained behind the iris ; the iris retired from the cornea ; the eye recovered with normal tension and useful vision. In this case an annular posterior synechia had united the pupillary margin with the capsule or hyaloid, locked up the posterior chamber, and caused bulging of the iris with closure of the filtration-angle, just as when the lens is present (see page 46).

[41] Kate C———. $\frac{2}{3\frac{1}{4}2}$.

Fig 25 was drawn from an eye blinded by this same form of secondary glaucoma. Two or more needle-operations for congenital cataract were performed by an eminent ophthalmic surgeon, when the child was nine months old ; the result appears to have been a clear but adherent pupil, and a persistently increased tension. The blind and distended eye was removed by myself seven years later. The pupillary margin was adherent throughout to the remains of the lens-periphery, and the iris bulged forwards in the manner already described. The pupil was not blocked by any visible false membrane, but the vitreous body appears to have opposed the free escape of the fluid from behind the iris. The iris adhered to the cornea in two places where the needle punctures had been made.

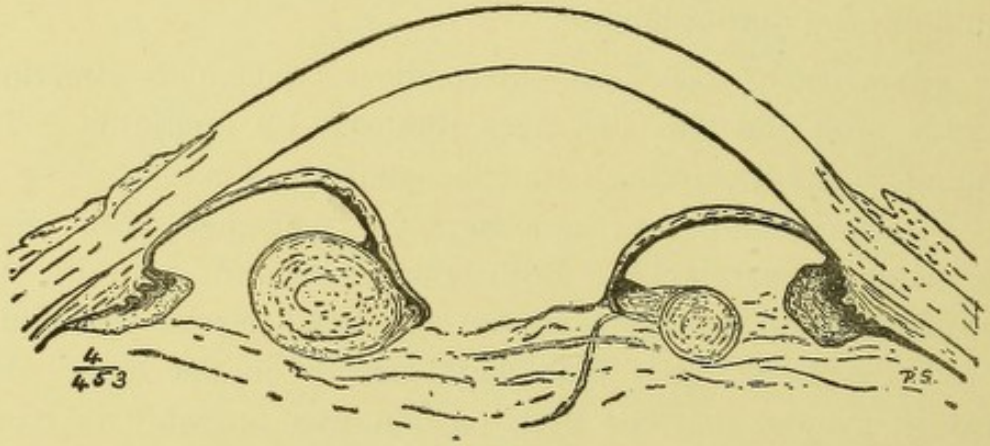


FIG. 25.
From an eye blinded by secondary glaucoma, following needle operations for cataract in infancy—seven years before excision. Annular posterior synechia ; accumulation of fluid behind iris ; closure of filtration-angle, as in Figs. 14 and 15.

After extraction of senile cataract high tension may occur at any time during the after-treatment ; it may occur after the eye has made a good recovery ; it may even occur after the good result has been maintained for many years. Two important papers on this subject have appeared lately. Natanson has collated and analysed the records of thirty-seven cases ;[42]

[42] "Ueber Glaucom in aphakischen Augen." Mattieson. Dorpat, 1889.

Treacher Collins has microscopically examined ten eyes blinded by this form of secondary glaucoma.[43]

Natanson's cases show, in the first place, that immunity from subsequent glaucomatous complication is not ensured by any particular method of operation : glaucoma may occur after the flap operation without iridectomy ; after iridectomy in combination with variously placed incisions ; after an extraction preceded by preliminary iridectomy ; and after extraction in the capsule. They show also that in the majority of the cases there was some visible complication involving the iris or the capsule or both, namely, iritis or iridocyclitis with occlusion of the pupil ; prolapse or adhesion of the iris at the wound ; or a similar entanglement of the capsule. In some cases, on the other hand, the eye appeared to be quite free from any complication of the kind. This negative evidence is, however, not quite conclusive, for slight adhesions of the kind in question may be quite undiscoverable in the living eye, and that they are frequent even in satisfactory cases has been proved by Becker. Becker examined with the microscope thirty-eight eyes from which cataracts had been extracted, and in only one-third of these was the iris free from adhesion with the scar, although thirty-two of the thirty-eight eyes were removed, not on account of any trouble during life, but after the death of the patients. He expressly states that minute adhesions of the iris or capsule with the scar may be quite invisible in the living eye.

It is obvious that an entanglement of the iris or the lens-capsule in the wound may lead to closure of the filtration-angle in its immediate neighbourhood, but this does not suffice to explain the occurrence of glaucoma. We cannot assume that obstruction of the filtration-angle, confined to a small part of the circle, is sufficient to cause high tension ; on the contrary,

---

[43] "Trans. of Ophthalmological Society of United Kingdom," vol x., p. 108.

we know that such entanglements after cataract extraction are common, while glaucoma is rare.

Treacher Collins's microscopic observations give more positive evidence as to the cause of the glaucoma. In nine of the ten eyes examined by him the capsule was adherent to the scar; in the remaining one, from which the lens had been

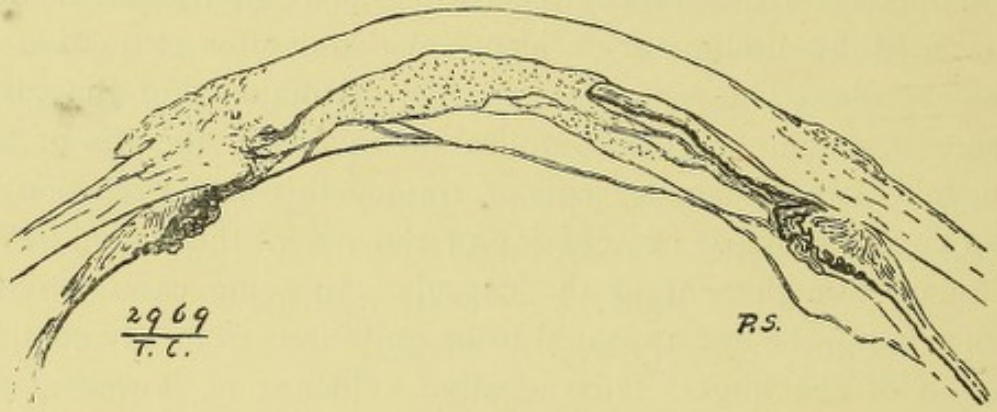


FIG. 26.

Extraction of senile cataract. Secondary glaucoma. Upward incision at sclero-corneal junction with large iridectomy. Anterior capsule opened and partly removed with capsule forceps. Iritis noted ten eight days after operation. On tenth day patient knocked the eye and the wound reopened. Injection and chemosis followed, and the anterior chamber was long in reforming. On return to hospital four months later, no anterior chamber; cloudy cornea; perception of light only; T+ 2. Attempted evacuation of lens-matter with curette gave no relief. The substance which coats the posterior surface of the cornea appears to be organising blood-clot, inflammatory exudation, and soft lens-matter. Filtration-angle closed at both sides of the eye.

(Specimen and notes from Mr. Treacher Collins.)

removed in its capsule, the hyaloid was adherent in the same manner. The filtration-angle was closed in the neighbourhood of the scar in every case; moreover, it was closed at the opposite side of the eye also, and probably throughout the whole circle, in seven if not in eight cases; and in those cases in which it was not closed by apposition of the iris and cornea, it was blocked by exudation.

Through the kindness of Mr. Collins, I have been able to examine his specimens, and to make drawings of some of the more typical examples. Figs. 26, 27, and 28, show how an adhesion of the capsule, and even of the hyaloid, with the scar may lead to compression of the filtration-angle at the most distant part of the circle.

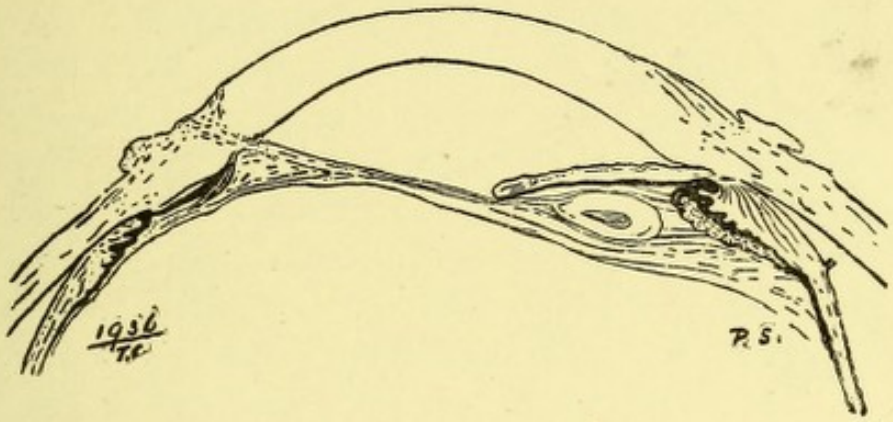


FIG. 27.

Extraction of senile cataract. Secondary glaucoma. A "modified Graefe" extraction, uncomplicated. Slight iritis followed. The patient could see to read and sew with this eye until one year and nine months after the operation; then the vision rapidly failed without much pain. Excision a month later. T + 1½. Adhesion of capsule to cicatrix. Filtration-angle closed at both sides of the eye.

(Specimen and notes from Mr. Treacher Collins.)

In the living eye, also, we can sometimes, I think, make out the cause of a glaucomatous complication after cataract extraction. In some cases the iris and posterior capsule, being united and coated by inflammatory exudation, appear to form an impermeable or insufficiently permeable diaphragm across the eye, which checks the passage of fluid from the ciliary process into the aqueous chamber. An excess of fluid becomes imprisoned behind this diaphragm. This may happen, although a good iridectomy has been made. In a case of this kind, on

the eighth day after extraction, and in presence of acute iritis, with free exudation into the aqueous chamber and very high tension, which had twice rapidly returned after paracentesis of the aqueous chamber, I made an iridectomy downwards, tearing completely through the adherent membranes, and obtaining for

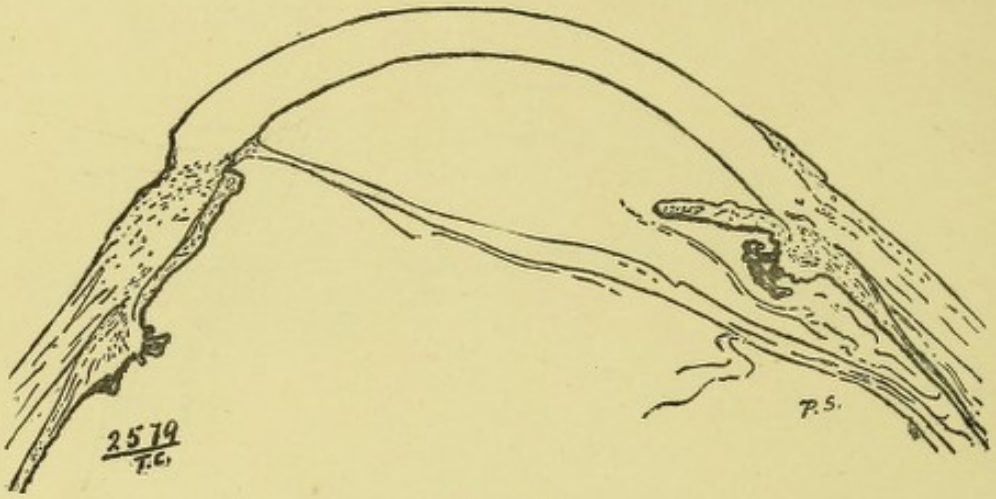


FIG. 28.

Extraction of cataract. Secondary glaucoma.

A cataract, probably of traumatic origin, had been removed, apparently with good result, eighteen months before patient came under observation. Pain and inflammation began without known cause twelve months after the operation. At the time of excision the cornea was cloudy and rough ; vision reduced to perception of hand ; T normal. After excision, disc found to be deeply and widely excavated. Iris and hyaloid adherent to cicatrix. Filtration-angle closed at both sides of eye.

(Specimen and notes from Mr. Treacher Collins.)

the moment a jet-black pupil. The eye recovered normal tension and good vision, which are still retained after thirteen years.[44]

It is not easy, even with the help of the pathological specimens here figured, to explain the occurrence of glaucoma

[44] For details of case, see "Glaucoma, its Causes, &c.," p. 273.

after a long interval of time during which the eye has enjoyed useful vision. It appears probable, however, that a transparent membrane, stretching across from the ciliary processes on the one side to the cicatrix on the other, as in Fig. 27, may, in the course of time, undergo some slight contraction which draws the processes forwards so as to compress the filtration-angle. Or such a membrane may become less permeable than at first. In this way, or perhaps through some change in the intraocular fluid itself, filtration from the vitreous to the aqueous chamber may be checked. This is not mere conjecture. In an elderly woman I performed a preliminary iridectomy, and later an extraction, apparently with complete success. A few months later an insidious glaucoma began, which at first yielded to eserine, but later became persistent. The field contracted; the disc became cupped. Sclerotomy with a Graefe knife was performed in the region of the extraction-wound. On the withdrawal of the knife, hardly any fluid escaped, and the iris applied itself closely to the cornea, showing that fluid was imprisoned behind the aqueous chamber. The point of the knife was then passed in again through the same wound, and through the coloboma into the vitreous; a gush of fluid escaped, the iris retired from the cornea, and the globe became slack. The eye recovered, with normal tension. Curiously enough, I operated later on the fellow eye of the same patient, and encountered almost exactly the same sequence of events. In some cases of this kind the high tension may be banished by passing a cutting needle through the area of the pupil, so as to divide the posterior capsule and anterior part of the vitreous.

With regard to glaucoma following cataract extraction, we can, therefore, assert that there is usually a closure or blockage of the filtration-angle, although we cannot in every case ascertain the precise manner of its production. The point of practical importance is that such an obstruction can be remedied only

while it is recent; when the base of the iris has become adherent throughout to the periphery of the cornea, the glaucoma is incurable.

Intraocular Tumours.—Eyes which contain tumours usually become glaucomatous if excision be long delayed. If the eye be excised while the tension is still normal, the filtration-angle will be found open ; if it be excised after high tension has come on, the filtration-angle will be found closed. I have met with no exception to this rule. In a series of cases of intraocular tumour with increased tension Brailey found the periphery of the iris applied to the cornea in every instance.[45]

Clinical observations show that the onset of the high tension corresponds in point of time with a displacement of the lens and iris towards the cornea. This displacement is not caused by direct pressure of the tumour against the lens or ciliary body, for it occurs when the tumour is limited to the back part of the eye, as seen in Fig. 29. It is caused by a general overfulness of the posterior segment of the globe. The growth of a choroidal tumour gives rise, either by compression of the choroidal veins, as Fuchs suggests,[46] or in some other way, to an outpouring of serum from the choroid. The retina is detached thereby, and pushed inwards. There is at first no discoverable increase of tension, for space is gained at the expense of the vitreous fluid;—we know that fluid contained in the vitreous body can pass very easily through the hyaloid into the aqueous chamber (see page 21);—but when the retina has become folded together in the axis of the eye, and little of the vitreous body remains beyond its fibrous stroma, then the lens, the ciliary processes, and the iris, are driven forwards and the filtration-angle is closed ; and with this closure the glaucoma begins. I

---

[45] R. L. O. H. Reports, vol. 10, p. 281.

[46] "Das Sarcom des Uveal Tractus." Vienna, 1882, p. 210.

would draw particular attention to these changes and to the order and time of their occurrence, for they throw light on the more obscure process which we call primary glaucoma.

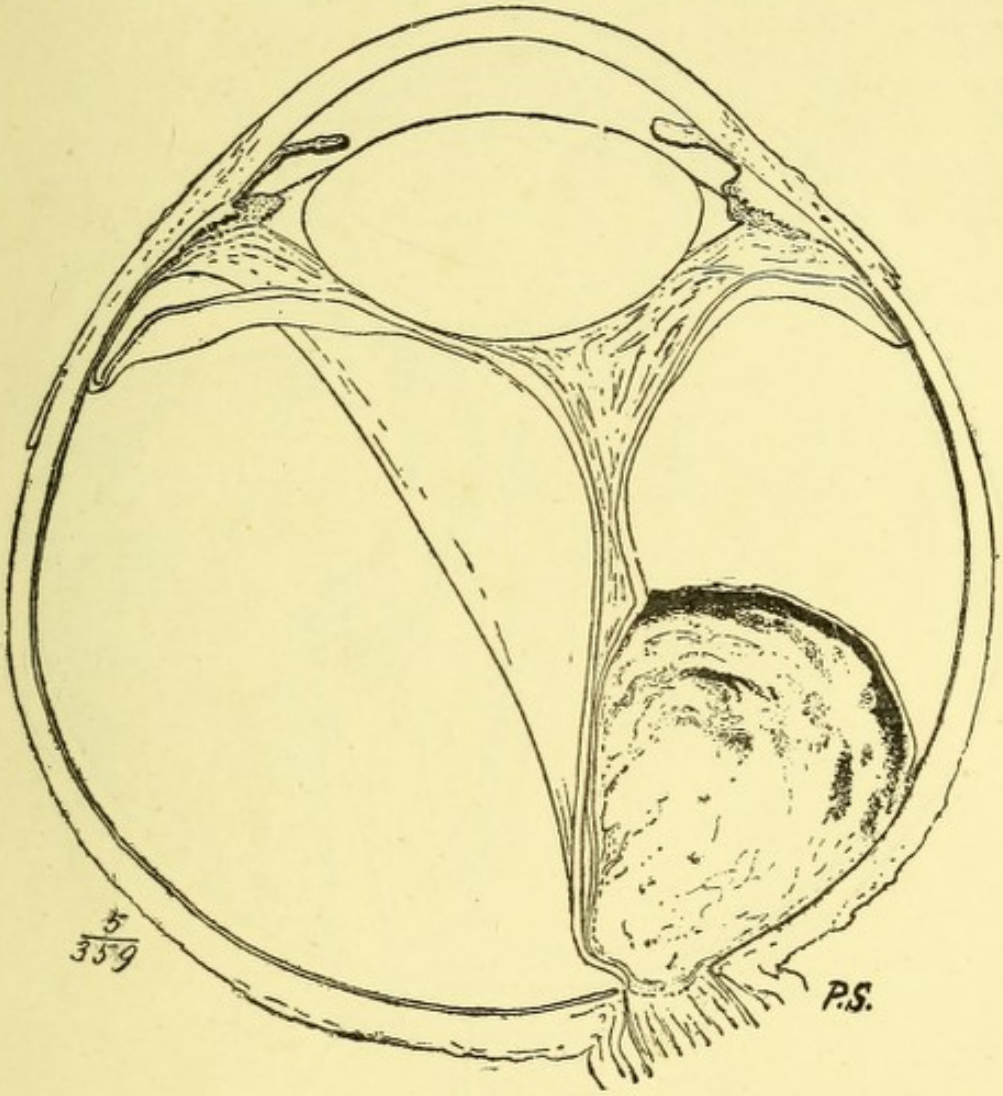


FIG. 29.

Sarcoma of choroid. Secondary glaucoma. Filtration-angle closed.

Tumour limited to posterior half of eye. Retina totally detached. Vitreous represented only by its fibrous stroma. First diagnosed as simple detachment of retina. Acute glaucoma with very shallow anterior chamber seventeen months later. Excision on the eighth day.

Fig. 30 was drawn from an eye in which a tumour of the choroid, with detachment of the retina, was diagnosed several months before the patient would consent to excision. While

the eye was still quite free from pain and disfigurement, the young lady went to a ball. The same night an agonising glaucoma began. Called to the patient seven days later, I

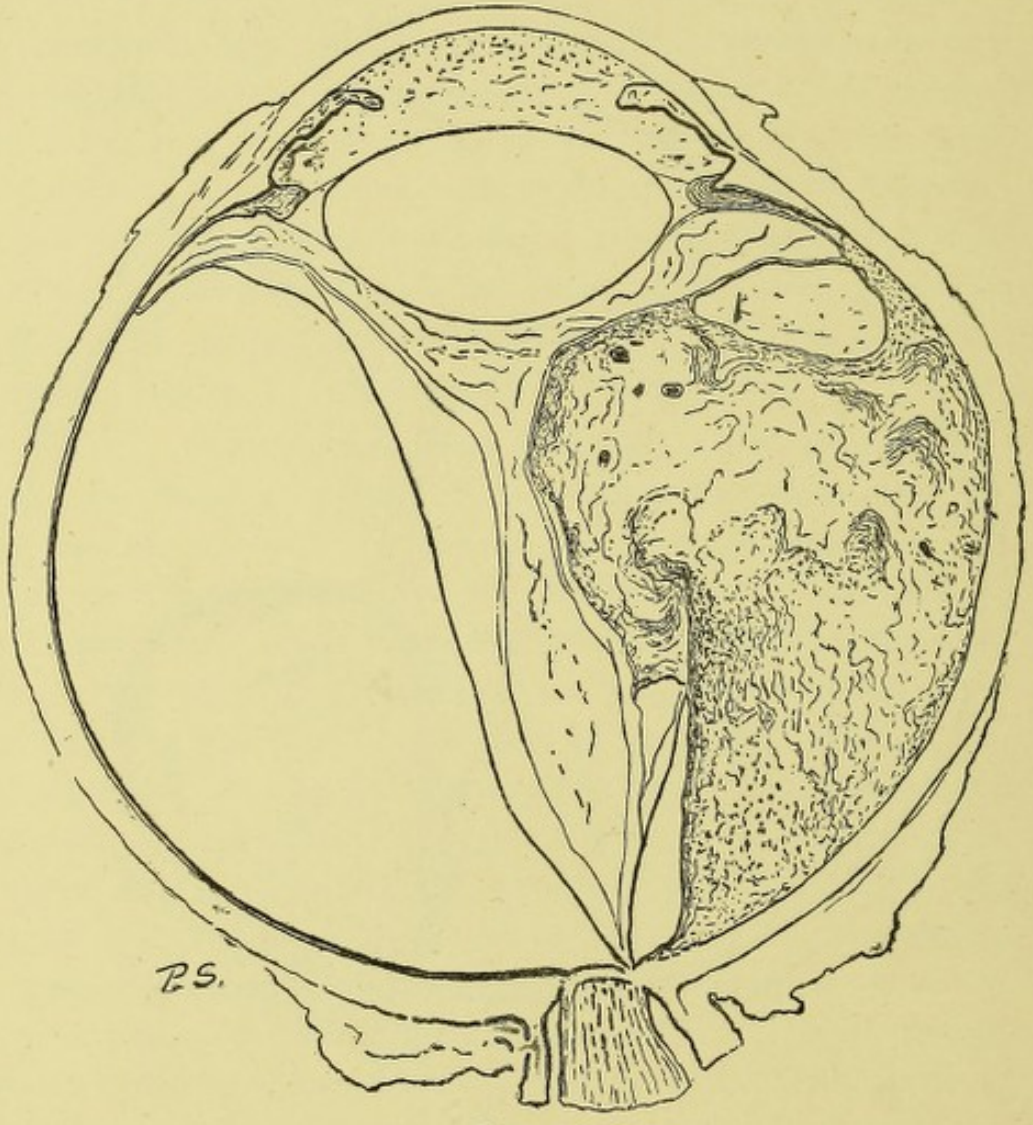


FIG. 30.

Sarcoma of choroid. Secondary glaucoma.
Filtration-angle closed.
The glaucomatous attack was unusually severe; the anterior chamber was almost completely abolished. The eye was excised on the seventh day.

found the eye greatly injected and stony hard; the aqueous chamber very shallow; the periphery of the iris visibly in contact with the cornea, its colour and surface here being very

clearly seen, while elsewhere they were obscured by the turbid contents of the chamber. This example is important, because it represents a very early stage of a very acute glaucoma. The

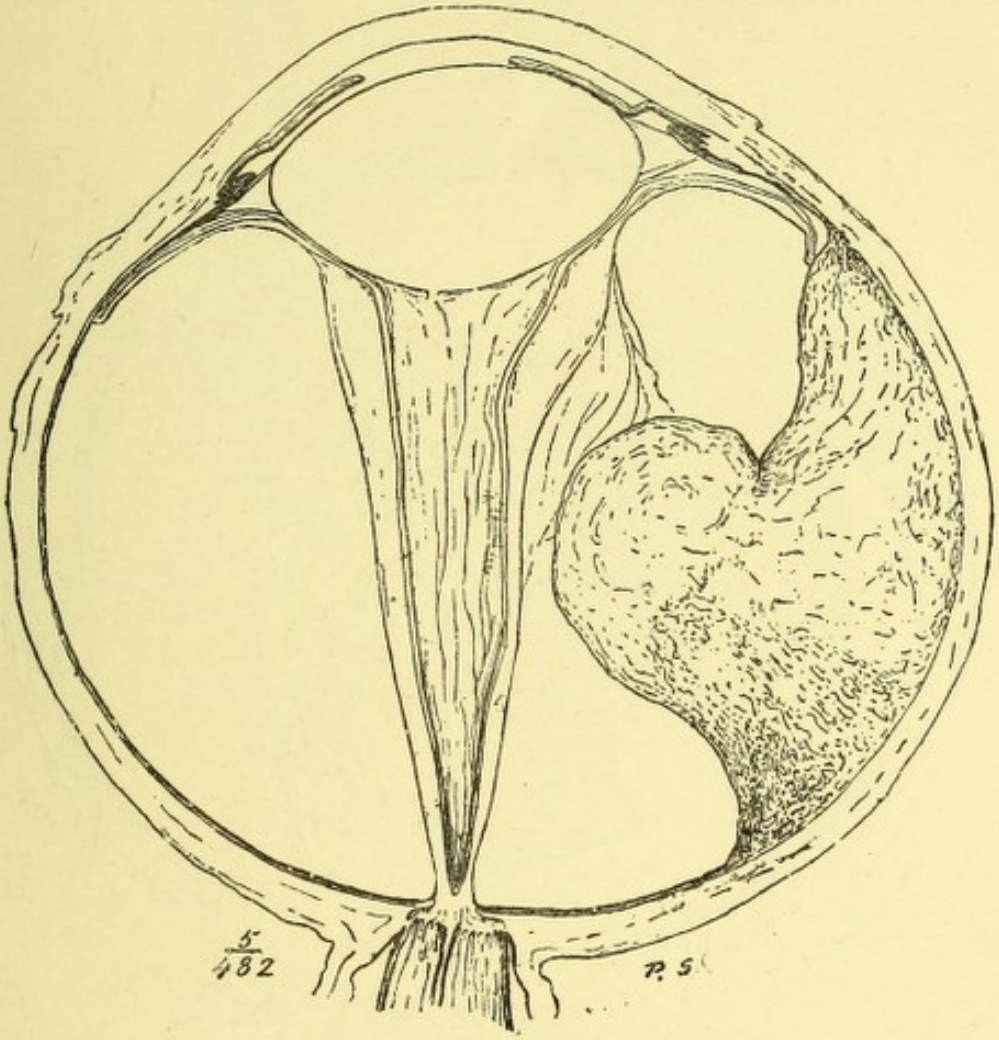


FIG. 31.

Sarcoma of choroid. Secondary glaucoma.
Filtration-angle closed.
The angular bend in the iris probably indicates the point to which the tips of the ciliary processes extended when the glaucoma was at its height. Excision on the sixth day.
(Mr. Hodges's case.)

mechanism of the outbreak is evident. The lens was driven forwards till it nearly touched the cornea; the ciliary processes were pressed against the iris-base, the iris against the cornea.

Later, either during or after the excision of the eye, when its tension was somewhat lessened by the escape of blood from the divided vessels, the lens and processes retired to their normal condition, leaving the iris in contact with the cornea.

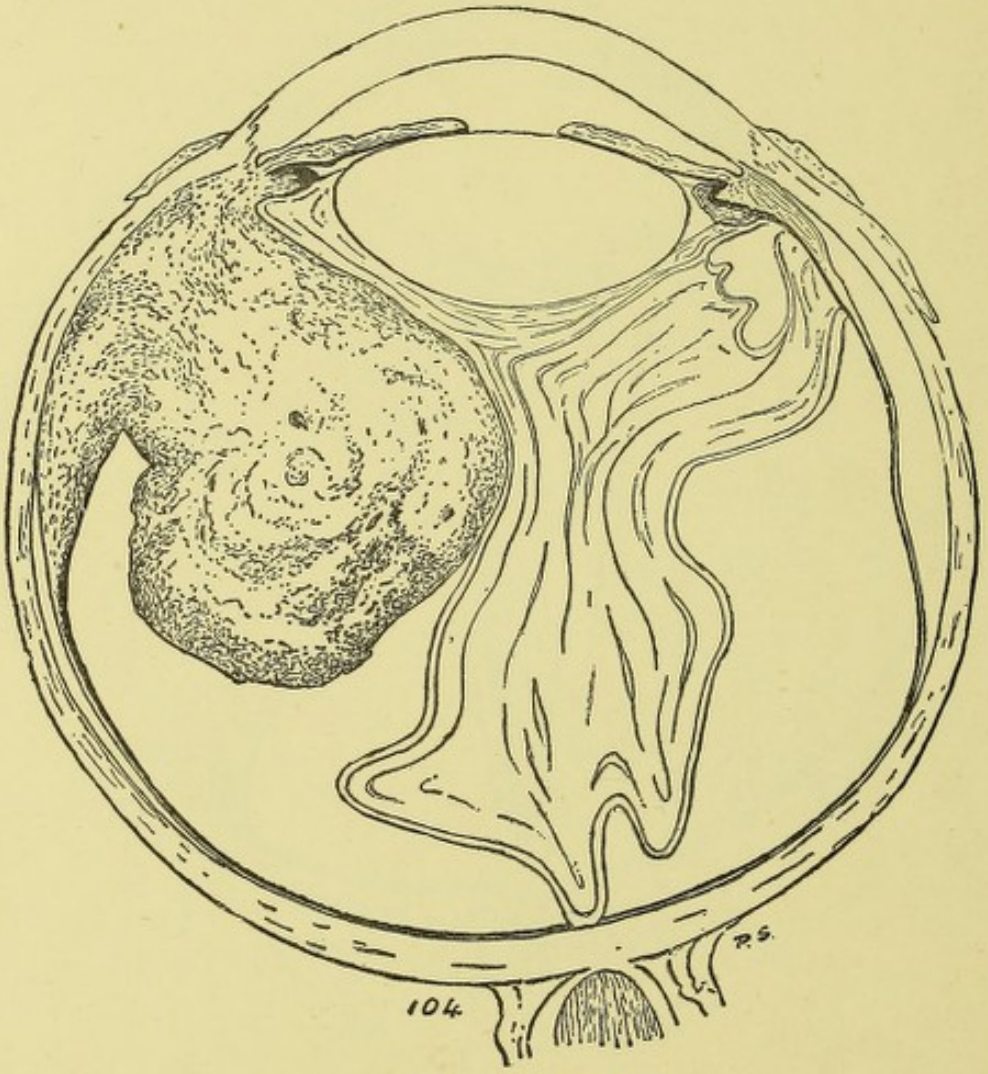


FIG. 32.

Sarcoma of choroid. No glaucoma. Filtration-angle open.
(Mr. McHardy's case.)

Fig. 31 was drawn from an eye which, like the foregoing, was excised very soon after the beginning of the glaucoma. The attack came on a few hours after the use of homatropine for diagnostic purposes, and was probably induced thereby, but could not in any case have been long delayed.

Fig. 32 was drawn from an eye much resembling the two foregoing as regards the size and position of the contained tumour, but differing from them in not being glaucomatous. It was excised while the tension was still normal, and the filtration-angle was found to be open.

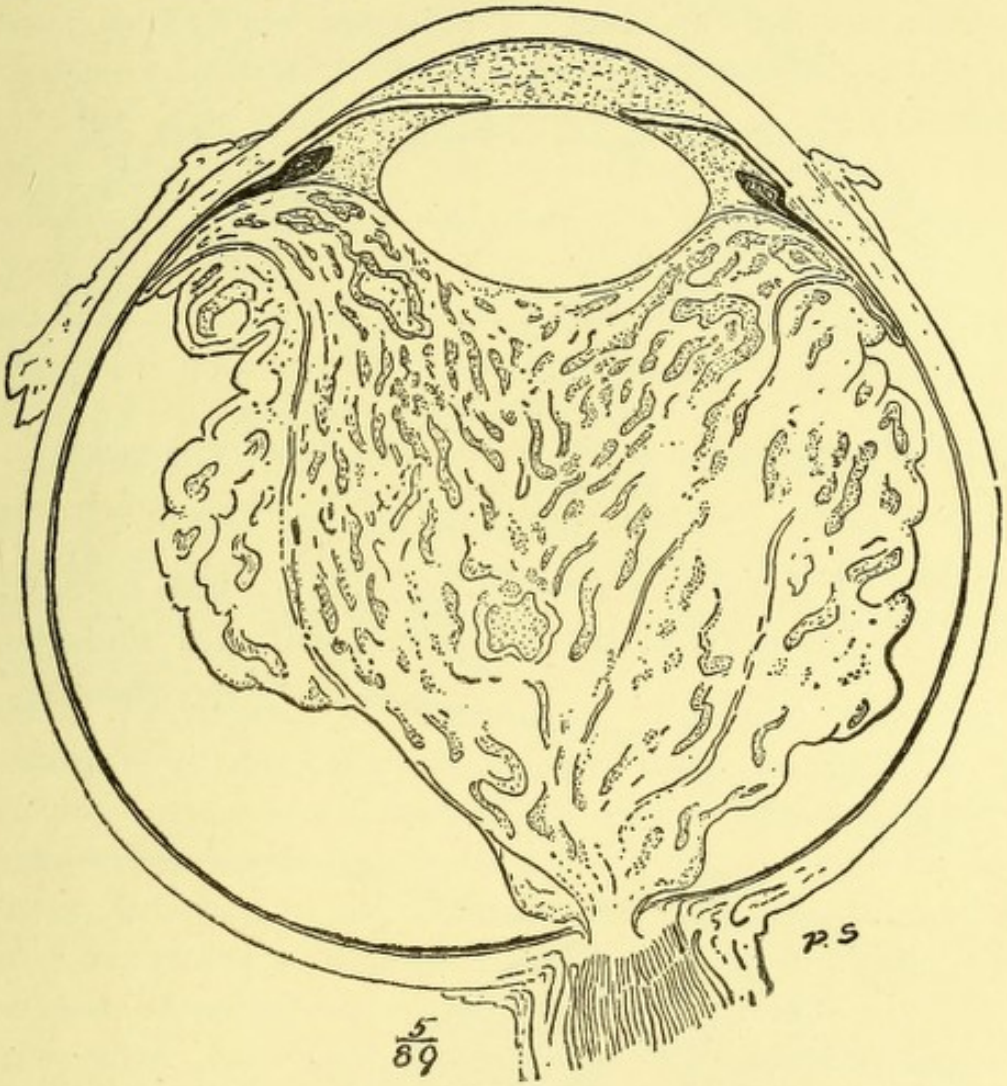


FIG. 33.

Glioma of retina. Secondary glaucoma. Filtration-angle closed.

Fig. 33 was drawn from the eye of a child, aged two-and-a-half years, which I had under notice for six weeks before excision was consented to. The iris and lens advanced, and the increase of tension began within the last fortnight, probably within the last few days, before excision.

Fig. 34 was drawn from an eye affected with a wide-spreading infiltration of the iris and ciliary body—probably a syphiloma. The filtration-angle is completely blocked. The glaucoma was intense.

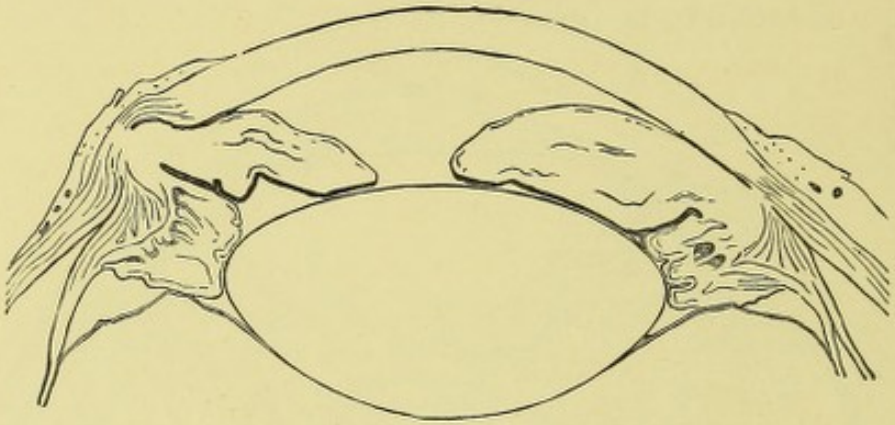


FIG. 34.
Tumour of iris and ciliary body. Secondary glaucoma. Filtration-angle closed.
(Mr. McHardy's case.)

Fig. 35 was drawn from an eye affected with sarcoma of the iris. The angle of the chamber is infiltrated throughout

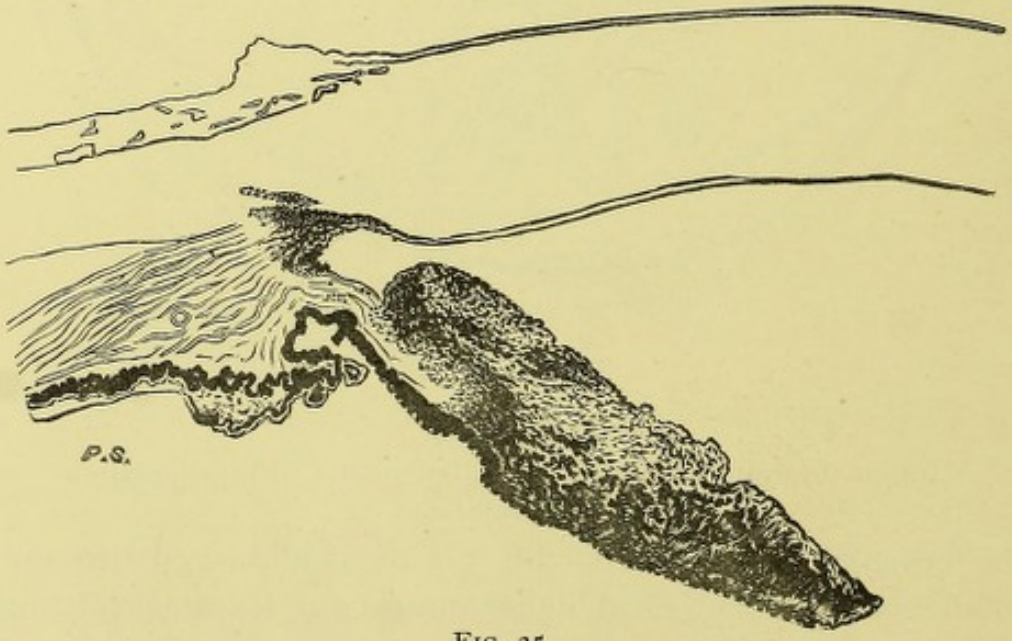


FIG. 35.
Tumour of iris. No glaucoma. Filtration-angle open, but infiltrated with cells from tumour.
(Mr. Solomon's case.)

the whole of the circle by a mass of cells which have no direct continuity with the primary growth, but have evidently been brought there by the current of the aqueous humour, and, being caught in the meshes of the ligamentum pectinatum, have begun to proliferate in the immediately adjacent structures. In some sections the cells appear to have entered and passed some distance into the sclera, following the course of Schlemm's canal ; they have found their way very freely into the anterior limit of the ciliary muscle, and are infiltrating between the muscular fasciculi ; they are also passing, though more sparingly, into the base of the iris. The ciliary processes are not invaded. Apart from the infiltration by the cells which are caught in its meshes, there is no closure of the angle of the chamber. The tension was normal at the time of excision.[47]

Detachment of the Retina.—Idiopathic detachment of the retina, such as occurs in myopic eyes in elderly persons, though not a direct cause of high tension, is occasionally associated with it. Leber's clinic at Göttingen has supplied important statistics concerning this malady.[48] 126 instances of spontaneous detachment of the retina, diagnosed with the ophthalmoscope, and not including any cases associated with intraocular tumour or a history of injury, were recorded during a period of six years. In the cases which were examined soon after the detachment had occurred, the tension was usually normal, but in many of these it became subnormal later ; in those first examined some time after the occurrence it was usually subnormal. Increased tension was present in only six of the 126 eyes ; in one of these iridocyclitis had supervened, and caused blocking of the pupil with bulging of the iris ; in three there was acute iridocyclitis, with deepening of the anterior chamber; in the two others, which belonged to one

[47] For details of case, see Trans. of Ophthalmological Society, vol. ii., p. 257.

[48] E. Nordenson, "Die Netzhautablösung." Wiesbaden. J. F. Bergmann. 1887. See abstract in "Ophthalmic Review," vol. vii., p. 74, etc.

patient, the detachment was associated with "glaucoma," the character of which is not stated.

The occasional occurrence of secondary glaucoma in such eyes appears to depend upon an extension of inflammation from the choroid to the anterior parts of the uveal tract. According to Leber and Nordenson, choroiditis is always present in cases of idiopathic detachment of the retina. Cyclitis is very common, as indicated by the subnormal tension. More or less iritis often supervenes. In a case under my own care,[49] a spontaneous painless detachment of the retina in a myopic eye was followed some months later by acute iritis leading rapidly to annular posterior synechia, exclusion of the pupil, and bulging of the iris. The eye was excised on account of severe pain. The specimen shows a displacement of the iris closely resembling that seen in Fig. 14, except that the filtration-angle appears not to be absolutely closed. Nordenson gives an excellent drawing[50] of an eye affected in the same way, in which the tension was very high. In an eye kindly sent to me by Mr. Richardson Cross, which was lost by spontaneous detachment of the retina, followed two years later by violent inflammation with very high tension, the filtration-angle is open, but the aqueous chamber as well as the subretinal space is filled with a highly albuminous fluid which would escape from the eye with great difficulty.[51]

When a retinal detachment is followed by a glaucomatous attack which has the general character of an acute primary glaucoma—dilated pupil, shallow anterior chamber, &c.—the presence of a choroidal tumour must be strongly suspected.

Serous Exudation and Hæmorrhage. — The form of secondary glaucoma which is caused by serous exudation from the ciliary body into the aqueous chamber has been referred to already (page 40). It is commonly characterised by a dotted deposit upon the posterior surface of the cornea, and by more or less deepening of the anterior chamber. The filtration-angle

[49] Specimen Q. 16. [50] Nordenson, plate xx., case iii. [51] Specimen 131.

is not closed. The excess of fluid appears to be retained in the eye by reason of its own diminished filtration-power; perhaps also by tissue changes around the filtration-angle.

An exudation of serous fluid or of blood into the vitreous chamber is sometimes followed by glaucoma. In such cases, the depth of the anterior chamber, if at all affected, is diminished rather than increased, and the condition may bear a very close resemblance to primary glaucoma; indeed, it is probable that many of the cases which we call primary are, to some extent, dependent upon effusions of this kind. It will be convenient, therefore, to discuss these cases in the third lecture.

Intraocular hæmorrhage is a frequent cause of sudden high tension in eyes previously blinded by iridocyclitis—eyes with a closed pupil, a shrunken vitreous, a detached retina, and until the moment of the hæmorrhage, a subnormal tension. In such eyes the filtration-channels are usually blocked by impervious membranes, and the sudden addition to the contents of the globe finds compensation with great difficulty.

In all these various forms of secondary glaucoma, then, we find changes which, in one way or another, obstruct the escape of fluid from the eye. In the very large majority of cases the filtration-angle is closed. In some its function is impaired or annihilated by morbid exudations. In most instances it is clear that the obstruction at the filtration-angle is not a mere complication of the glaucoma-process occurring after the high pressure has been definitely established, but is itself the immediate cause of the high pressure. It is clear, also, that the only way to relieve the high pressure is to restore the patency of this outlet The causes of the more obscure condition, which we call primary glaucoma, will be considered in the concluding lecture.

LECTURE III.

Primary Glaucoma. Obscurity of its causes. Structural changes found after excision. Closure of the filtration-angle.—Predisposing causes :—Growth of the crystalline lens. Liability to glaucoma at different periods of life. Size of cornea in healthy and glaucomatous eyes. Size of globe. Refraction.—Exciting causes :—Congestion and inflammation of the uveal tract. Forward displacement of the lens ; accumulation of fluid in the vitreous chamber. Dilatation of the pupil ; thickening of the iris-base. Influence of accommodative effort. Influence of sex.—Consequences of increased pressure in the eye :—Œdema of the cornea. Paralysis of the ciliary muscle. Obstruction of circulation in the uveal tract. Obstruction of circulation in the retina. Excavation of the optic disc. Changes in the size and shape of the globe.—Principles of treatment :—Value of eserine ; atropine ; cocaine ; morphine ; sleep, warmth, aperients, etc. Iridectomy. Sclerotomy. Other incisions in the ciliary region. Scleral puncture. Depression of the lens in malignant glaucoma.—Conclusion.

PRIMARY GLAUCOMA.

We call a glaucoma primary when we are unable to point to a previous disease of the eye as its cause. The obscurity of its origin distinguishes the primary from the various forms of secondary glaucoma which we have already considered. It is important to observe, however, that, apart from their initial causes, primary and secondary glaucoma are, to a large extent, one and the same morbid process. Pressure, the essential factor, is common to both. The other leading symptoms of primary glaucoma—the progressive loss of vision, the contraction of the field, the cupping of the disc, and, in the more acute forms, the pain, the injection, and the rapid abolition of sight—are all to be found in one or other form of secondary glaucoma also. They are absent only in so far as they are hidden, or modified, or forestalled by the pre-existing disease of the eye. The special interest which belongs to the pathology

of primary glaucoma lies at present, I think, not so much in the evil *consequences* of high pressure—these are now fairly well understood—as in the mysterious nature of the *causes* which can give rise to a formidable disease to-morrow in an eye which looks perfectly healthy to-day.

Clinical observation alone, however acute, fails to explain the malady. I will, therefore, in the first place, ask your attention to certain conditions which are found in eyes which have been blinded by primary glaucoma, and excised. Many minute descriptions of such specimens have been published, notably by Brailey in this country, by Knies, Weber, and Birnbacher and Czermak in Germany. I have myself, up to the present time,[51] examined 34 specimens of primary glaucoma, and 7 others which I believe to be primary, but about which there is some doubt, either through the absence of a proper record or an uncertainty whether an injury of the eye was the starting point of the disease—41 in all. Of these specimens 27 were from my own practice, 14 were given to me by friends. They represent various types of glaucoma—the chronic or simple, the subacute, and the acute—but I have no specimen of the chronic variety which had remained free from pain and congestion up to the time of excision; in every case of this kind it was the ultimate occurrence of pain which led to the excision. The list includes many cases in which hæmorrhage had occurred at one or other stage of the disease, and several in which it had perhaps been the starting point In 3 eyes at least the glaucoma was associated with high myopia, in 3 with well-marked senile cataract. In 9 instances an iridectomy had been performed without success, and in one a successful iridectomy had been performed twenty years before excision, but the eye had latterly become blind and painful. The youngest of the patients from whom these eyes

[51] March, 1891.

were removed was only 21 at the time of the excision ; five were between 40 and 50 ; the rest were over 50 ; the oldest was 80. The large majority of the specimens are permanently preserved.

One word as to the method of examination. My own specimens were hardened gradually in Müller's Fluid, and divided when frozen solid.[52] I believe this to be the best way of avoiding disturbance of the internal parts, but even this does not enable us to see the parts exactly in the positions which they have occupied in the living eye. If the lens be elastic, it will become more globular when the tension of the suspensory

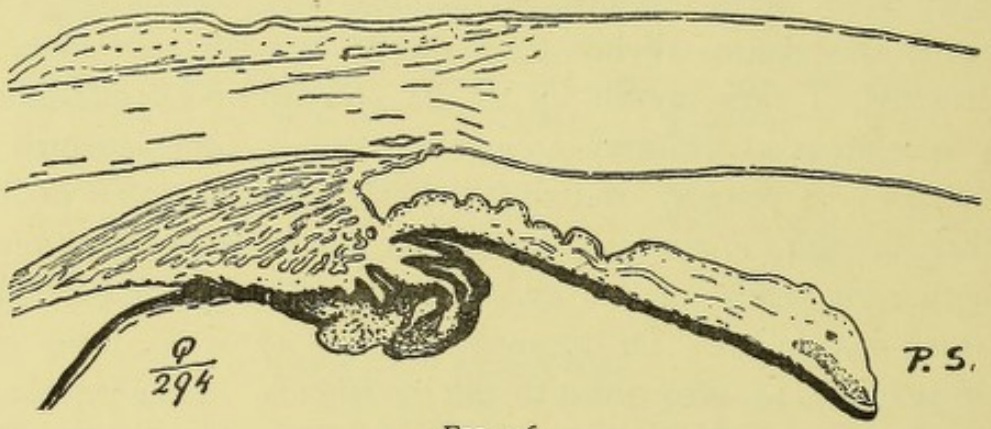


FIG. 36.

From the healthy emmetropic eye of a man aged 57.
For comparison with Figs. 37 to 41.

ligament is relaxed ; if it has been pushed forward by an excess of pressure in the vitreous chamber, it will tend to return to its normal position when the pressure falls after the removal of the eye, and in so doing will permit the ciliary processes to alter their shape and position. If the processes have been turgid with blood, they will tend to shrink when some of this blood escapes through the divided ciliary vessels at the moment of excision. It is important to bear these disturbances in mind in considering the changes now to be described.

---

[52] The methods employed in examining, drawing, and preserving the specimens are described fully in the Appendix.

The filtration-angle is closed in the great majority of cases. When it is not closed it usually shows signs of compression. If the glaucoma have been of short duration the iris-base may be merely pressed against the margin of the cornea and ligamentum pectinatum (Fig. 37), and may separate easily in the divided

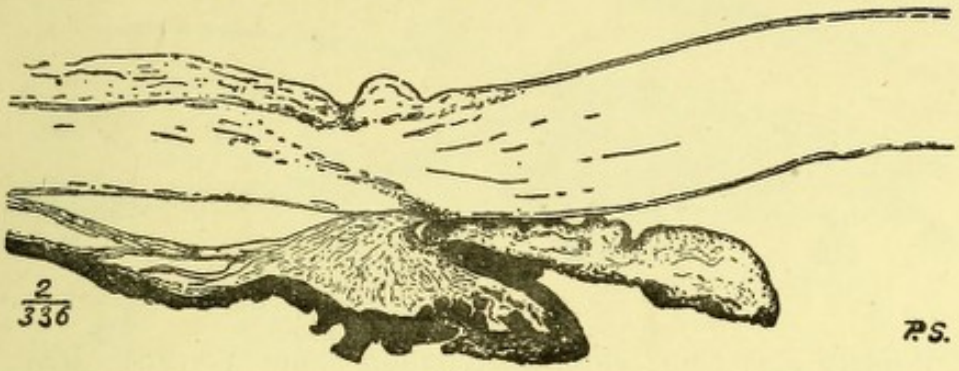


FIG. 37.

Primary glaucoma; acute; recent. Ciliary processes swollen and advanced; iris-base pressed against cornea, but not adherent to it. The eye was excised ten days after the beginning of the attack. It had been affected previously with senile cataract, nearly mature. At the time of excision: T + 3; great pain and injection; V = 0; anterior chamber very shallow; pupil dilated and irresponsive to eserine.

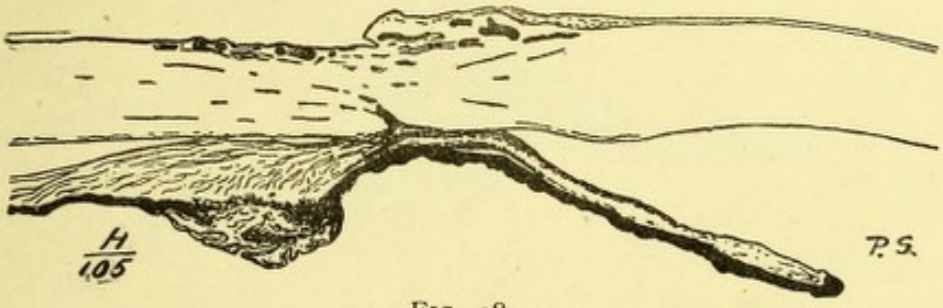


FIG. 38.

Primary glaucoma; chronic; duration about 12 months. Iris-base adherent to cornea; ciliary processes atrophied. The eye went blind gradually, without pain or redness; it had been quite blind for five months, and pain and redness had been present for three weeks before excision.

eye. In older cases it is generally adherent (Figs. 38, 39, 40), and often much atrophied. The extent of the adhesions varies much in different eyes and in different parts of the same eye. In some cases it involves the iris-base to a width of 1 millimetre

or more ; in others it is so slight as to be easily overlooked (Fig. 39). Sometimes the iris-base adheres to the processes, and is drawn backwards when they retract, so that its adhesion with

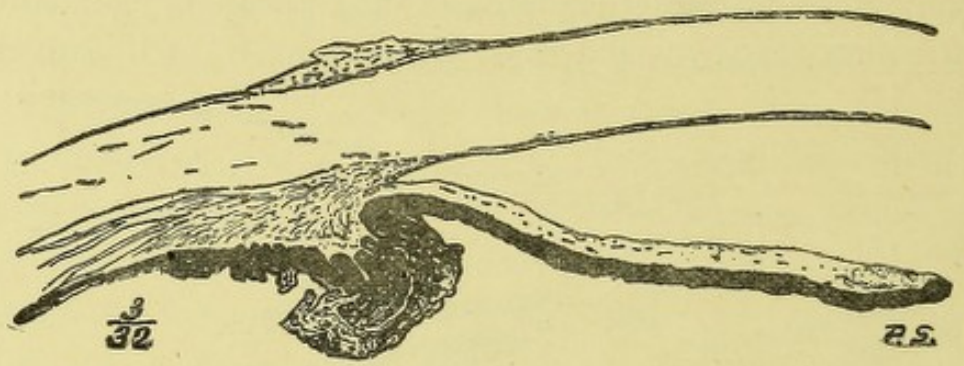


FIG. 39.

Primary glaucoma ; chronic ; duration about 5 years. Iris-base and ligamentum pectinatum adherent to a very small extent ; a very much wider adhesion exists at the opposite side of the eye. (See R.L.O.H. Reports, vol. x., p. 42, Fig. 9.)

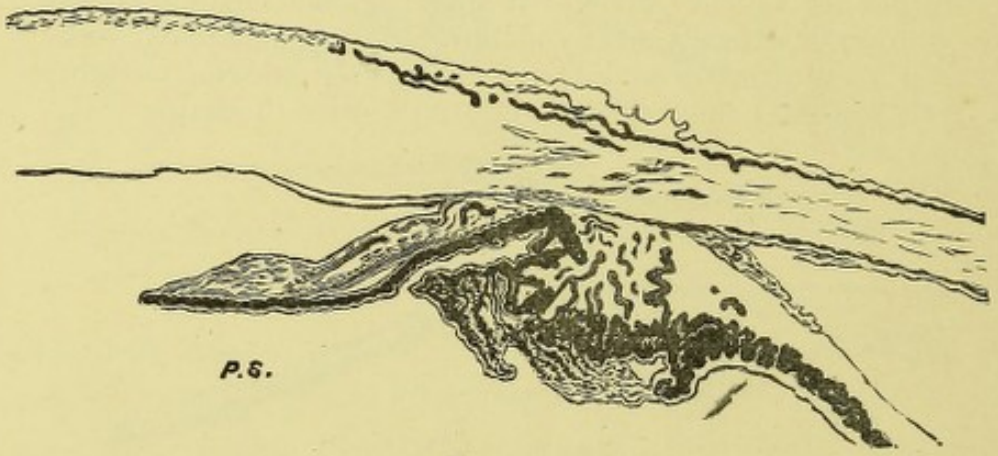


FIG. 40.

Primary glaucoma ; acute ; duration about 12 months. Filtration-angle closed by adhesion ; ciliary processes moulded into a wedge-like form by compression between lens and iris. (See R.L.O.H. Reports, vol. x., p. 36, Figs. 6 and 12.)

the cornea is more or less torn through ; or, if it is not torn through, the iris-base may be stretched in the direction of its thickness, and present a peculiar bend or notch in its anterior surface just at the limit of the adhesion (Fig. 41). The

adhesion seems to form most constantly and most rapidly in the acute and subacute cases—that is to say, the tendency to adhesive inflammation varies in degree with the vascular

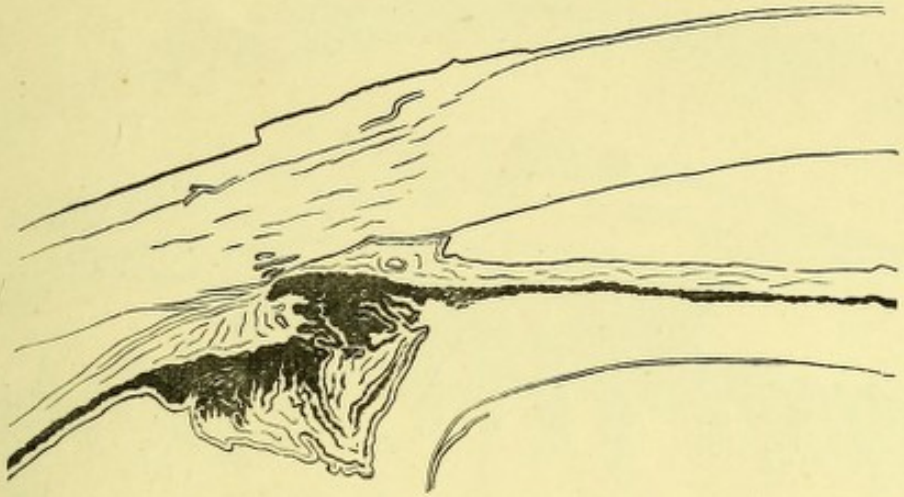


FIG. 41.

Acute glaucoma resembling the primary form, but induced by a blow on the eye. Before excision the anterior chamber was very shallow, the iris being pushed forwards by the lens. In the divided eye the lens has receded; when pushed forwards it fits closely against the ciliary processes as well as the iris. The ciliary processes have receded slightly, pulling upon the adherent iris-base. (See "Ophthalmic Review," vol. i., p. 273.)

congestion. In the chronic non-irritative form it occurs later and sometimes not at all, but even in these cases signs of compression of the filtration-angle are usually to be seen.[53]

---

[53] In 29 of my 34 specimens the filtration-angle is closed either by firm adhesion or close contact of the iris-base and the cornea. The 5 cases in which it is open claim particular notice. In one of these it has certainly been closed during life, for cells and pigment from the iris adhere to the cornea throughout precisely that region where the adhesion is usually found (F. 174, Haywood. See R.L.O.H. Reports, vol. x., p. 29, and drawing 11). In 3 other cases it is narrowed in a way which strongly suggests compression during life, followed probably by more or less re-opening after excision (122 ; K 133 ; S 411). The fifth case is altogether exceptional. The patient presented herself with the anterior chamber three-fourths filled with blood and an intense glaucoma of eight days' duration. She stated that the eye had failed without pain two years before, and had been quite blind more than a year. She had albuminuria. The eye was excised. In the bisected globe the anterior chamber was nearly filled with blood, and the filtration-angle was of normal width. The disc was excavated, which proved that the glaucoma was not very recent, and

The ciliary processes are usually altered, both in size and in position. If the glaucoma have been of recent date and congestive type, they are enlarged, their apices extending forwards

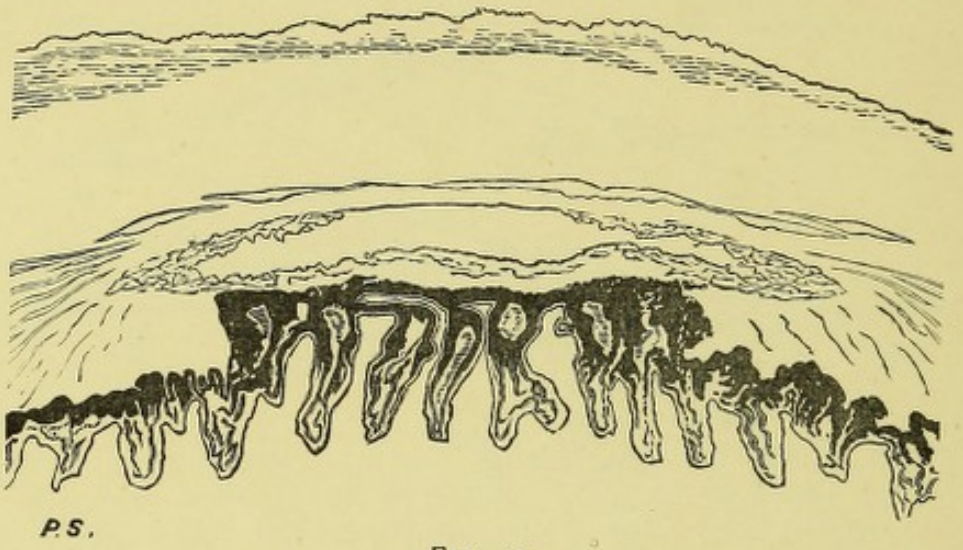


FIG. 42.

Healthy eye. Transverse section through ciliary processes. (See R.L.O.H. Reports, vol. x., p. 42.)

far beyond the customary limit (Figs. 37 and 40). In such cases they are usually in close contact with the iris anteriorly, and sometimes with the margin of the lens internally ; or, if no longer in contact, their wedge-like shape shows that in the living eye they have been tightly pressed between these structures Viewing them in transverse section (compare Figs. 42 and 43), we see that they are increased in thickness also, the spaces between the processes being narrowed, or even obliterated, by the swelling of the lateral convolutions. If, on the other hand, the glaucoma have been of long standing, the processes are sometimes much shrunken and retracted, being then far removed from the base of the iris ; but even in such cases the iris-base often bears the impress of their former contact. Between

---

was not due merely to the hæmorrhage, which occurred eight days before the excision. The case may perhaps be explained by supposing that a chronic, painless glaucoma, with some compression of the filtration-angle, had been present for many months, when a profuse hæmorrhage from the iris distended the anterior chamber, blocked the outlet completely, and set up acute symptoms (T 88).

extreme hypertrophy on the one hand, and extreme atrophy on the other, any degree of swelling or shrinking may be present.

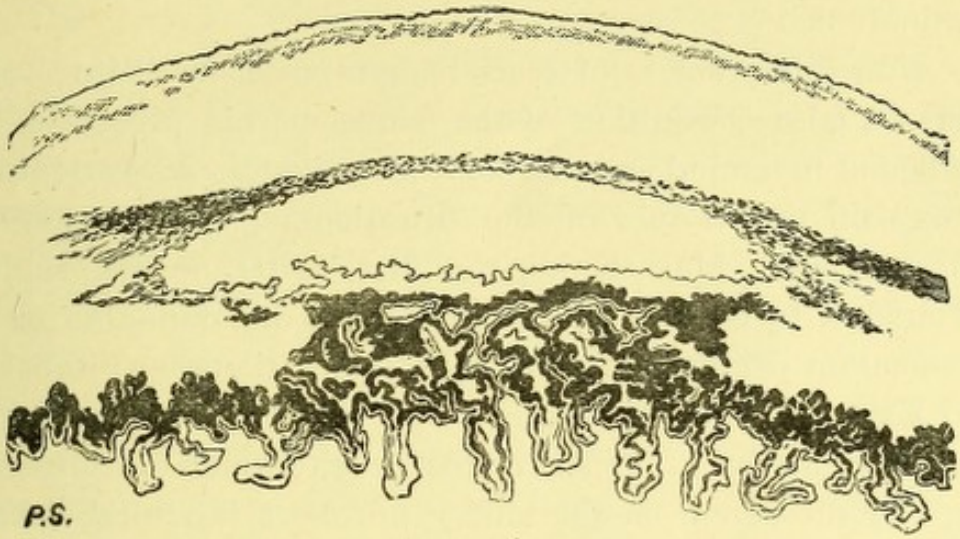


FIG. 43.

Primary glaucoma. Transverse section through ciliary processes. (See R.L.O.H. Reports, vol. x., p. 42.)

The ciliary muscle participates in these changes; at first it is drawn forward with the processes; later it retracts and atrophies.[53]

The lens is sometimes found in close contact with the processes and iris; more often it is a little separated from them, for, being pushed forwards during the glaucomatous attack, it recedes when the pressure is removed. Its relations should be studied in the frozen globe immediately after its bisection. In some cases there is an obvious disproportion between the size of the lens and the size of the eye which contains it. The significance of this will be discussed a little later on.

A careful study of these changes in specimens of recent primary glaucoma leaves, I think, no uncertainty as to the manner in which the filtration-angle becomes closed:—The iris-base is pushed forwards by the ciliary processes, as Adolph

[53] See description by Brailey, R.L.O.H. Reports, vol. x., p. 89.

Weber asserted in 1876, and adheres, sooner or later, according to the degree of vascular disturbance and inflammatory action, to the opposing surface of the cornea and ligamentum pectinatum.

Now this point is of crucial importance, for it involves a true or a false conception of the whole morbid process, and I am bound to remind you that some investigators have attempted to explain the closure of the filtration-angle in an entirely different way. Max Knies, the first writer on the subject, declared it to be due to a circumscribed inflammation of the ligamentum pectinatum and the adjacent tissues. Birnbacher and Czermak have maintained the same opinion, and have even gone so far as to assert that a closure of the filtration-angle by pressure from the ciliary processes is entirely out of the question.[54] In view of the conditions which are to be seen in many of the drawings and specimens which I am able to lay before you, this assertion is quite astonishing, until one examines the evidence upon which it is based. Drs. Birnbacher and Czermak examined seven eyeballs. Two of these were without clinical history. In the remaining five, the duration of the glaucoma had varied from seven months to twenty years. There was no very recent or acute case. The eyes were opened without freezing, and in one case without hardening. The material and method were obviously ill adapted to determine the point in question, and I feel sure that, with more favourable opportunities, these most careful observers would have come to a different conclusion. Indeed, Birnbacher, in describing a specimen of recent acute glaucoma in a later paper, expresses himself very differently. He admits that in this case the closure of the filtration-angle did not originate in an inflammation around Schlemm's canal, for no trace of such inflammation could be found, and the extremity of the angle was still open (this

[54] Von Graefe's Archiv, vol. xxxii., part ii., p. 1.

condition may be seen in Fig 37), and concludes that the iris-base must have been pushed forwards by an advance of the ciliary processes.[55]

The condition of the filtration-angle in the earlier stages of chronic glaucoma (glaucoma simplex) can hardly be ascertained. Probably there is no actual closure, but a slight, and in some cases a variable, degree of narrowing, which gradually becomes greater or more persistent. But this cannot be proved. Eyes in an early stage of chronic glaucoma are never excised, and, even if they were, the very act of excision would probably permit the parts to resume their natural positions (see page 76). Some authorities who have studied this question chiefly from its clinical side tell us that there are cases of primary glaucoma in which there is certainly no compression of the filtration-angle, for the iris can be seen, in the living eye, to lie in a normal position. But this view is invalid. An eye blinded by chronic glaucoma may show during life no fault whatever in the position of the iris, and yet may present on bisection a well-marked closure of the filtration-angle. In the living eye the base of the iris is completely hidden.

We learn, then, from pathological anatomy, that in primary glaucoma the ciliary processes push forward the base of the iris, and thereby compress the filtration-angle. We have already seen that a similar displacement with a similar result is caused by the growth of choroidal tumours (see page 64), and that it may be induced artificially by raising the pressure in the vitreous chamber a little above that in the aqueous chamber, so as to cause a slight advance of the lens (see page 24). Concerning primary glaucoma, we have still to enquire what it is which brings the processes into this mischievous position, or, in

[55] Alois Birnbacher, "Ein Beitrag zur Anatomie des Glaucoma acutum." Graz : Leuschner and Lubensky, 1890 ; pp. 24 and 25.

other words, what are the initial causes of the disease. It will be well to distinguish between predisposing and exciting causes.

PREDISPOSING CAUSES OF PRIMARY GLAUCOMA.

It can, I think, be shown that a want of space between the crystalline lens and the parts which surround it is a frequent predisposing cause of primary glaucoma. This idea was put forward in a work published twelve years ago,[56] being at that time little more than a hypothesis, and it has served the true purpose of every hypothesis, good or bad—it has suggested fresh lines of enquiry. It now rests upon the following facts :—

a. The size of the lens increases throughout life.

b. The liability to primary glaucoma increases throughout life.

c. The liability to primary glaucoma is greatest in eyes of exceptionally small size.

d. A disproportion between the size of the lens and the size of the globe can be demonstrated in some eyes blinded by primary glaucoma.

It will be well to deal with these several points in the order in which they were investigated.

Growth and Size of the Lens.—In order to ascertain whether the relation of the lens to the surrounding parts varies at different periods of adult life, I examined, in the year 1880, five pairs of healthy eyes taken from adult male subjects, varying in age from 21 to 90, and found a well-marked increase in the size of the lenses from the youngest to the oldest.[57] A more thorough investigation naturally followed. 156 lenses removed from the dead subject were examined. They belonged, in nearly equal numbers, to the six decades of life between 20 and 80, and in smaller number to the decade 80 to 90. Each

[56] "Glaucoma, its Causes, etc." London : Churchill, 1879, page 158.

[57] R. Lond. Ophth. Hosp. Reports, vol. x., p. 33.

lens was accurately weighed, and then measured as to its volume, by means of an apparatus devised for the purpose (Fig. 44). In most cases the linear dimensions were measured also. Opacity when present was noted. The specific gravity was calculated in each case from the weight and volume. Details of the method, and of the precautions taken against error, have been described elsewhere.[58] Here it is only necessary to state the results:—

The crystalline lens, so long as it remains healthy, increases in weight and in volume throughout the whole of life. During the forty years between 25 and 65 years of age it adds about one-third to its weight, one-third to its volume, and one-tenth to its diameters. The specific gravity appears to vary a little in individual cases, but shows no decided change with the advance of life. Lenses which are becoming cataractous are as a rule, smaller than healthy lenses belonging to the same period of life (see Figs. 45 and 46, and the tables in the Appendix.) These are anatomical facts; physiology explains them.

The lens is derived from the cuticular epiblast, and in its mode of growth is analogous to the cuticle. But its cells, unlike those of the cuticle, are not cast off as they grow old; they are laid down layer upon layer within a closed capsule, the younger fibres surrounding the older. In consequence of this unique arrangement, and in spite of the shrinking of the older cells which form the nucleus, the growth of the lens does not cease with that of the rest of the body, but is continuous, unless some morbid process intervene, throughout the whole period of life. In advanced life the process of growth often fails. Then the shrinking nucleus tends to separate from the softer cortex,[59] and senile cataract begins. Accordingly, the lens with incipient

[58] Transactions of Ophthalmological Society, 1883, page 79.

[59] Becker, "Zur Anatomie der gesunden und kranken Linse," p. 52. See "Ophthalmic Review," vol. ii., p. 269.

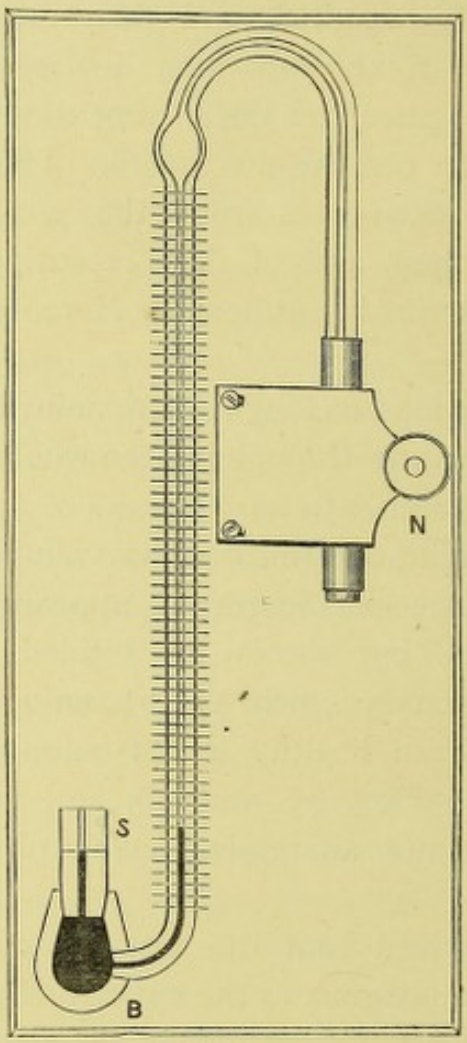


FIG. 44.

APPARATUS FOR MEASURING THE VOLUME OF THE CRYSTALLINE LENS.

The graduated glass tube ends below in a bulb, B, closed by a perforated glass stopper, S. Above, it terminates in an india-rubber tube closed at the end, which can be compressed by means of the plate and nut, N. The cubical content of the glass tube, as ascertained by experiment, is 2·24 cub. mm. for each mm. of length. The bulb and lower part of the tube are filled with rectified oil of turpentine.

In making a measurement, the nut is screwed down until the fluid rises in the stopper exactly to the transverse line. The height of the column in the graduated tube is then noted. The nut is then reversed until by the expansion of the india-rubber tube the fluid is drawn away from the stopper and the upper part of the bulb. The stopper is then removed, and the lens dropped into the bulb; the stopper is replaced; the fluid is again driven exactly up to the mark on the stopper; and the height of the column is again noted. The difference between the present height and the former height, in millimetres, multiplied by 2·24, equals the volume of the lens in cubic millimetres.

For details, see Trans. of Ophth. Society, vol. iii., p. 82.

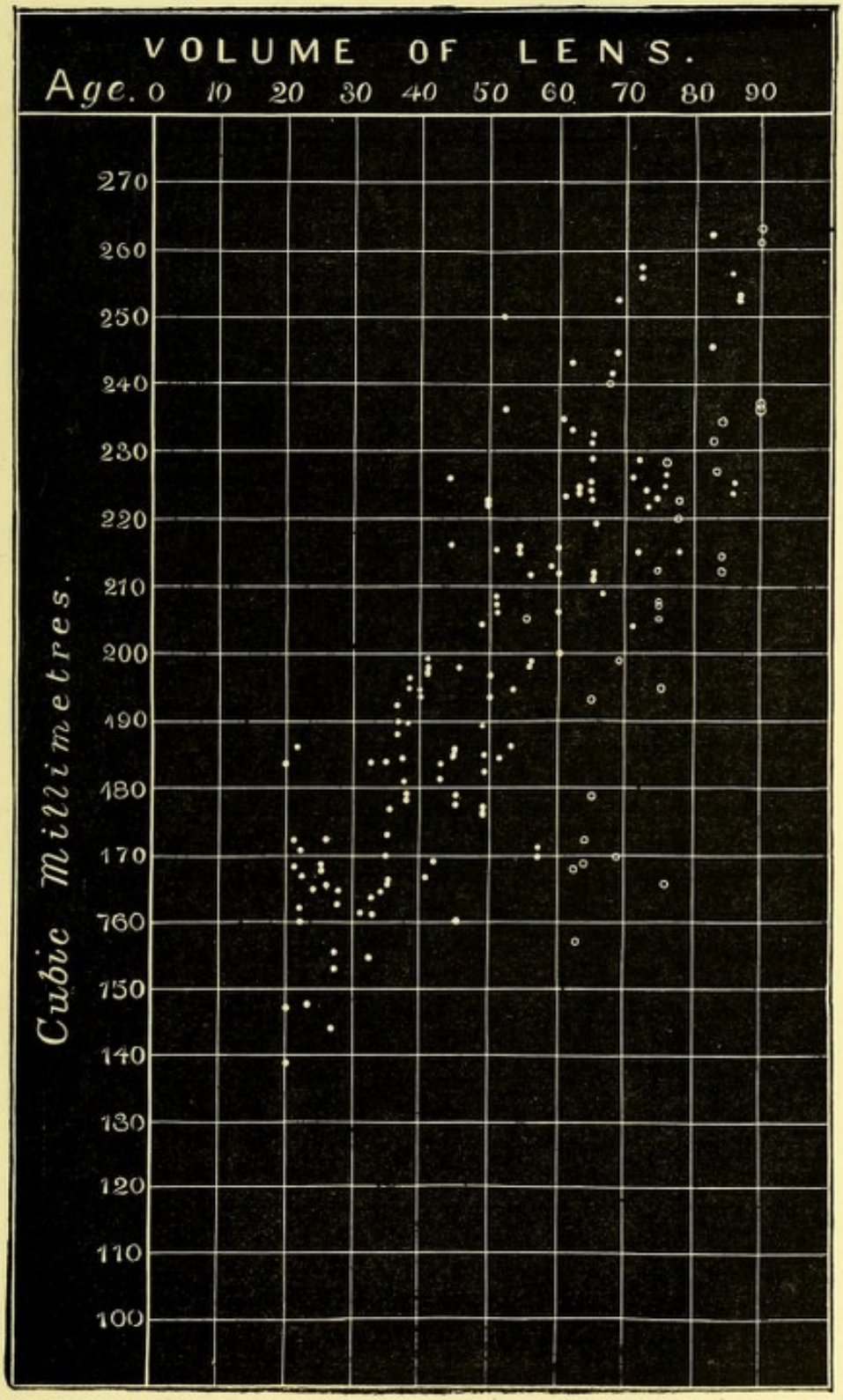


FIG. 45.

This chart shows the volume of each lens examined, and the age of the person from whom it was taken. The white dots show the transparent lenses; the circles those which were partly or wholly cataractous. (See also tables in Appendix.)

cataract is usually smaller than the healthy lens of the same age. To this rule, however, there are perhaps some exceptions; in certain stages of the development of cataract the softened cortex appears to swell.

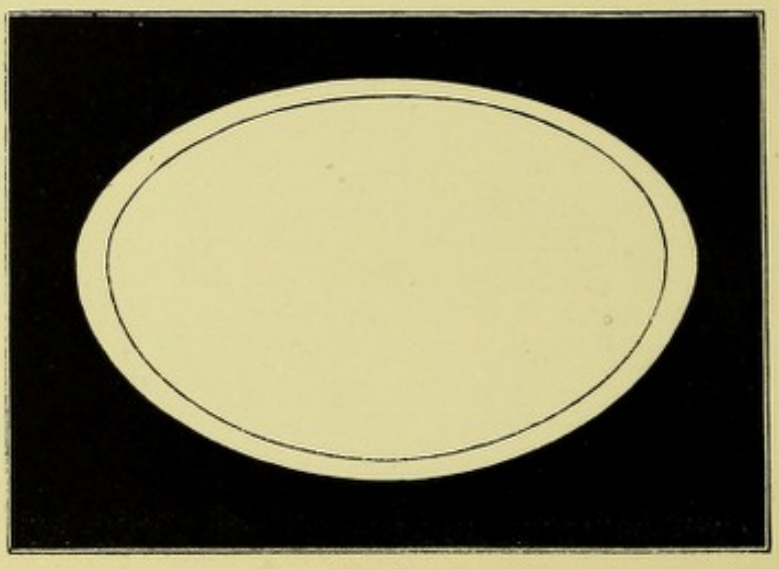


FIG. 46.

Relative sizes of average healthy lens at 25 and 65 years of age.

Now the structures which surround the lens attain their full dimensions at the commencement of adult life, or perhaps earlier I shall have to point out immediately that the diameter of the cornea increases little, if at all, after the fifth year. Hence it comes about that as age advances the lens steadily encroaches upon the space in which it lies. Its margin comes into closer relation with the ciliary processes. The precise relation of these parts in the living eye cannot be ascertained, for it is altered by death and by excision, but the change in question is obvious when we examine eyes of very different ages. Its anterior surface approaches nearer to the cornea, and thereby diminishes the depth of the anterior chamber. The shallow anterior chamber of old age has been supposed to indicate an advance of the whole lens towards the cornea, but such an advance has not been proved or explained, and the idea is not reconcilable with

the change of refraction which actually occurs. A simple advance of the lens in an emmetropic eye would cause myopia, whereas the tendency in advanced life is to hypermetropia.[60]

And here I may point out that the dimensions of the crystalline lens are incorrectly stated in many of our text-books, and that in diagrams and illustrations of the front segment of the eye similar errors are common. For example in the "Graefe-Saemisch Handbuch,"[61] the schematic eye has an antero-posterior diameter of 3·7 millimetres. This is too small even for

[60] The increased size of the lens is itself, probably, to a large extent, the cause of the acquired hypermetropia of advanced life. If a lens be enlarged symmetrically—that is, if its several diameters be increased in equal proportion—its focal length will increase in the same proportion. This may be demonstrated without mathematics. Supposing that the Figure A (Fig. 47) correctly represents the refraction of a given lens, and supposing that we enlarge this figure symmetrically—say by photography

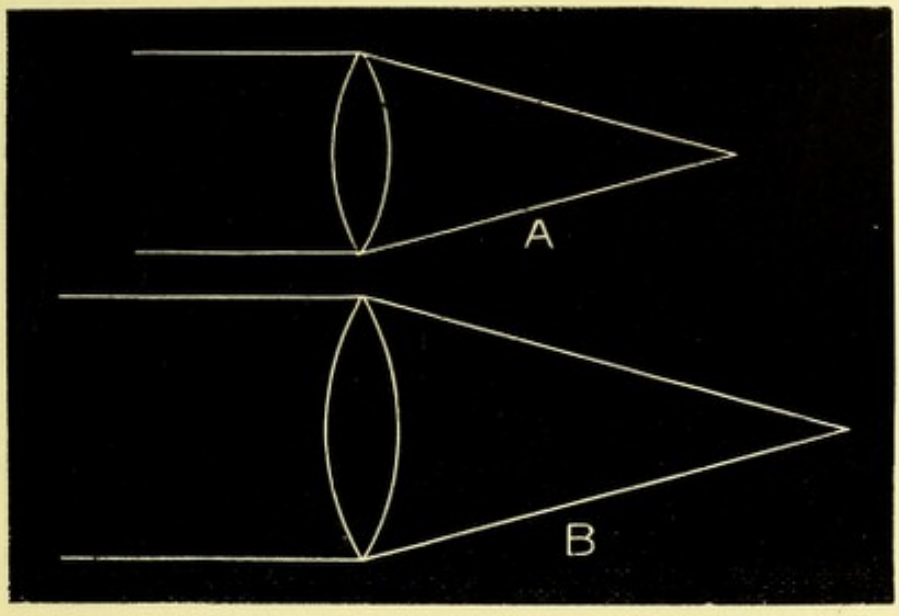


FIG. 47.—Refraction of lens.

—then the enlarged Figure B will correctly represent the refraction of the enlarged lens. Other things being equal, therefore, the focal length of the human lens must increase as its size increases. The result may, of course, be modified by changes in the index of refraction, or by disproportionate enlargement in one or other diameter; but it is clear that the growth of the lens cannot be omitted, as hitherto, from the calculation.

[61] Vol. I., pp. 43 and 45.

the young adult, and for middle and advanced life it is much too small. In elderly people I have found lenses measuring 6, and even 6·5, millimetres in this diameter. (See the series of photographs, Figs. 52, etc.)

As the result of this inquiry the glaucoma question stood thus: The lens steadily increases in size as life advances. If the size of the lens is an important factor in glaucoma, the liability to glaucoma should steadily increase in like manner. Does it so increase?

The Liability to Primary Glaucoma in relation to Age.—With the help of many friends, members of the Ophthalmological Society, I was able to collect accurate data concerning 1,000 cases of primary glaucoma. The cases were tabulated on a uniform system, indicating the sex of the patient, the age at which the glaucoma began, and the type of the disease whether chronic, subacute, or acute. The figures so obtained were then adjusted to the number of persons, males and females living in each period of life, so as to show the liability belonging to each sex and each life-period. The results, both as to frequency and as to liability, are shown in the charts, Figs. 48, 49, and 50. The following are the salient points:—

A.—*Frequency.*—1. Primary glaucoma is extremely rare in childhood and youth; not one per cent. of the cases met with begins earlier than the twentieth year (five per 1,000 belong to the second decade).

2. Its frequency increases, slowly at first, more rapidly later on, in each decade until about the sixtieth year; between sixty and seventy it is about as frequent as between fifty and sixty. After seventy its frequency diminishes.

Frequency of Primary Glaucoma at different Life-periods.

Distribution of 1,000 cases as they are actually met with.

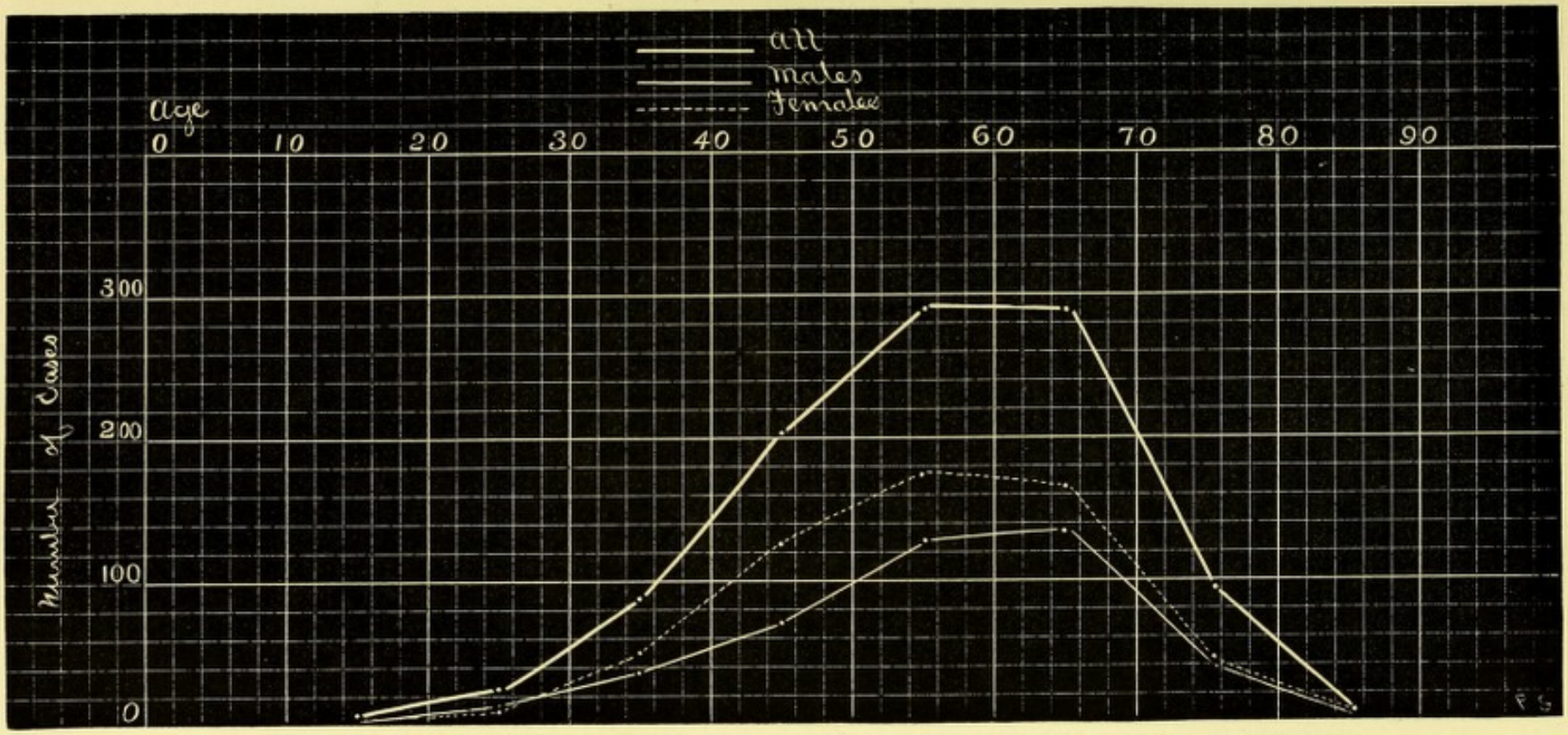


Fig. 48.
(See also the tables in the Appendix.)

Liability to Primary Glaucoma at different Life-Periods.

The same 1,000 cases distributed as though persons of all ages and both sexes were equally numerous.

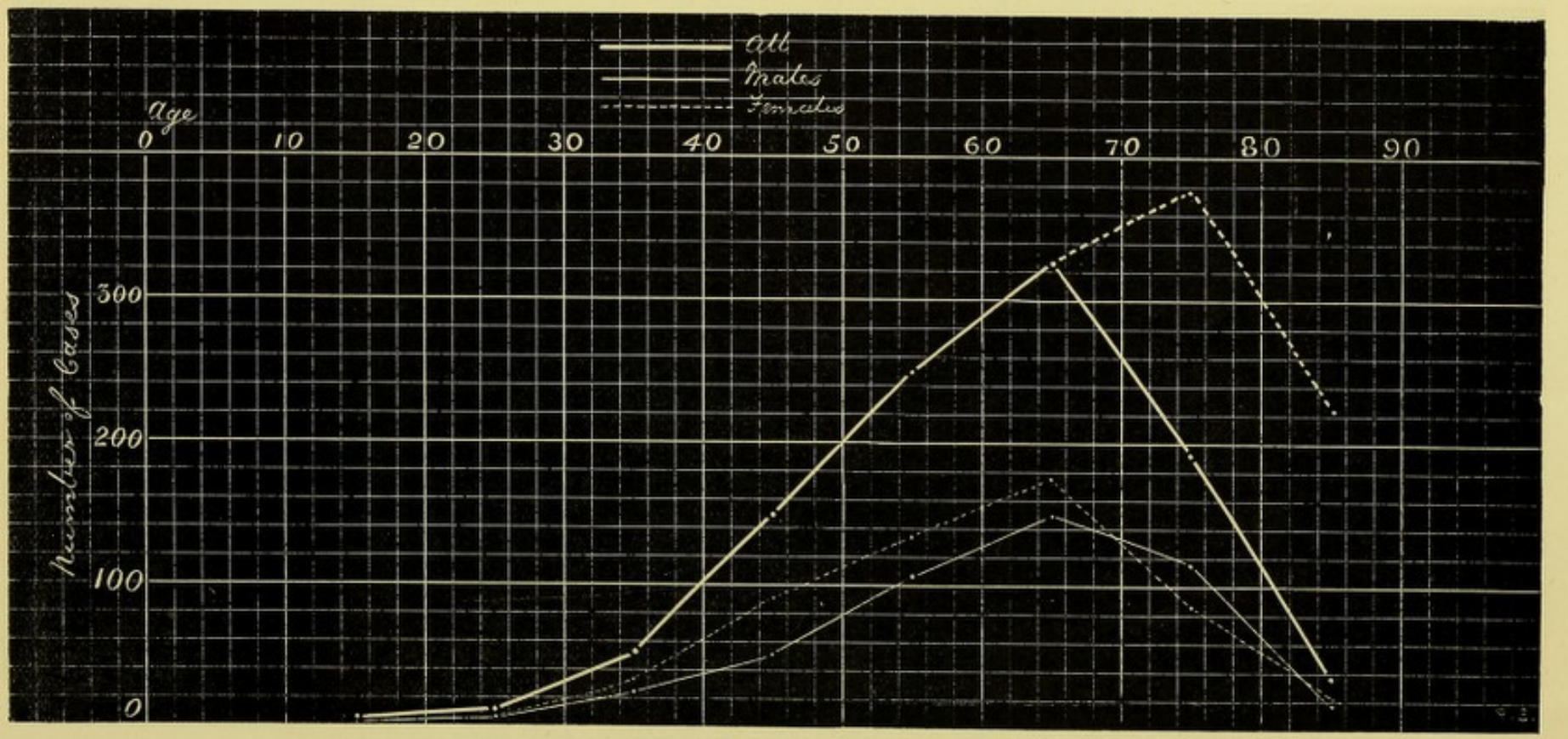


Fig. 49.
(See also the tables in the Appendix.)

Liability to Primary Glaucoma at different Life-periods.

The same as the foregoing, with classification according to types in the two sexes separately.

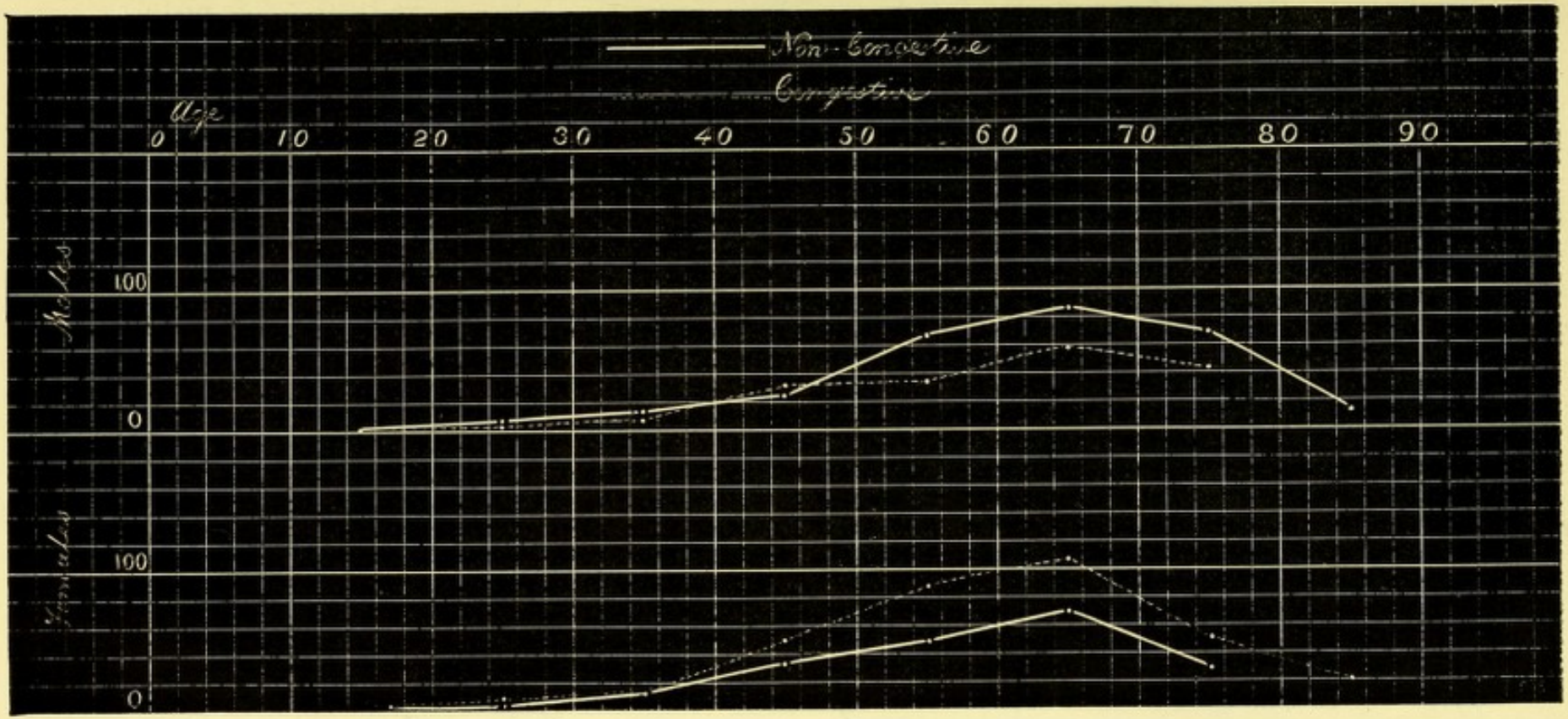


Fig. 50.
(See also the tables in the Appendix.)

3. Cases beginning after fifty are about twice as numerous as cases beginning before fifty (679 and 321 per 1,000).

4. Females suffer in rather larger numbers than males (569 and 431 per 1,000).

5. The chronic non-congestive form is rather commoner in males than in females (253 and 223 per 1,000).

6. The acute and subacute congestive forms are much commoner in females than in males (346 and 178 per 1,000).

The foregoing statements refer to the distribution of primary glaucoma as it is actually met with. When the figures have been modified in proportion to the numbers of males and females living at each period of life, we find, of course, that the relative liability of old people is greater than would appear from the actual numbers coming under notice.

B.—*Liability.*—7. The liability to primary glaucoma is extremely slight in childhood and youth as compared with the later periods of life; thus at fifteen years of age it is at least 100 times smaller than at sixty-five.

8. It continually increases up to and during the seventh decade, *i.e.*, the ten years between sixty and seventy. Between sixty and seventy it is more than twice as great as between forty and fifty.

9. After seventy years of age the liability to glaucoma appears to decline considerably; it is probable, however, that the statistics relating to this period do not correctly represent the frequency of the disease. Very old people do not willingly or easily travel to the centres where oculists are to be found; many are permanent inmates of workhouses; many more, through the infirmities of age, are prisoners in their own homes. Under these circumstances it is likely that the onset of glaucoma

is neglected in a larger proportion of cases than at earlier periods of life, especially when one eye still remains useful.[62]

10.—The liability of females is greater than that of males in a ratio probably of about 6 to 5.

11. The extra liability of females pertains to the whole of life, except, perhaps, the periods before thirty and after seventy, concerning which the data are too few to justify a generalisation.

12.—The extra liability of females relates very markedly to the congestive forms of the disease, not to the non-congestive.

---

[62] In the annual reports of the Manchester Eye Hospital I find some statistics which tend to confirm this supposition. I take the figures from the reports for the years 1882-3-4.

	Total No. of patients.	No. in decade 60--70	No. in decade 70—80	No. in decade 80—90
1882 ..	12,961 ..	515 ..	138 ..	16
1883 ..	14,702 ..	612 ..	158 ..	14
1884 ..	15,427 ..	636 ..	172 ..	13
	43,090	1,763	468	43
Ratio ..	.. ..	.. 1	·265	·0244

Now, according to the life tables the numbers of persons actually living in these decades are in the following proportions :—

	60—70	70—80	80—90
	3,138	1,693	467
Ratio	1	·539	·149

On comparing these ratios it appears that if the number of persons applying for relief had been proportionate to the persons living in those three decades there would have been just twice as many between seventy and eighty, and six times as many between eighty and ninety as there actually were. This suggests either that the liability to eye disease in old people is reduced to one half by an additional ten years of life and to one sixth by another ten years, which is unlikely, or that the ability of the sufferers to come to the oculist is reduced in this same proportion, which is likely. This estimate is of course uncertain as regards any particular disease such as the one here in question, but it is suggestive. Applied to the eighth and ninth decades it would raise the liability to the height shown by the thick dotted line in Fig. 49.

Our question, then, is answered: the liability to primary glaucoma does, like the size of the lens, steadily increase with the advance of life. Permit me now to ask your attention to another anatomical condition which is connected with this liability.

Growth and size of the Cornea.—Every operator must sometimes have been struck, when performing an iridectomy for primary glaucoma, by the unusual smallness of the cornea. The idea suggested itself that a small cornea might indicate an abnormal relation between the ciliary processes and the lens. I therefore attempted to ascertain the frequency of this

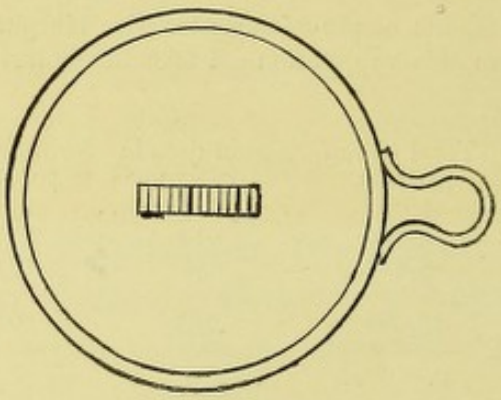


FIG. 51.

Keratometer; actual size.

peculiarity and the nature of its connection, if it have any, with primary glaucoma. For this purpose it was necessary to ascertain by numerous measurements what is the average size of the cornea in healthy eyes, what are the limits within which it varies, and whether corneas of subnormal size are more numerous among glaucomatous than among healthy eyes.

The instrument employed—a keratometer, devised for the purpose—is shown in Figs. 51 and 52. It enables one to measure the horizontal diameter of the cornea without touching the eye or lid and without alarming the patient. It consists of a millimetre scale placed between two plano-convex lenses, in

the form of an eye-glass, having a focal length of ten inches. It is held at ten inches from the observer's eye, and at about one inch from the patient's eye, or nearer if convenient. As the margin of the cornea is not a perfectly sharp line, its position cannot be determined to a small fraction of a millimetre; I noted the nearest half-millimetre in every case: 11, 11·5, 12, etc., as the case might be. I measured the horizontal diameter only; it is difficult in many cases to measure the vertical diameter.

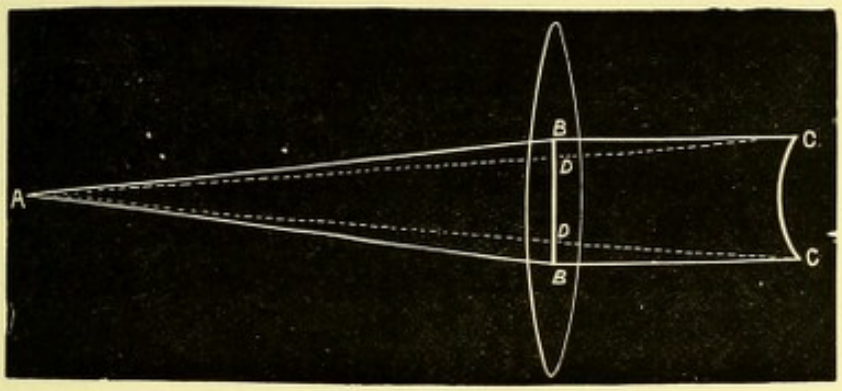


FIG. 52.

Keratometer seen edgeways, with the scale in its interior. the lines C B, C B, represent parallel rays, which when refracted by the lens meet in the point A, ten inches distant from it. To the observer's eye placed at A the points C C appear to coincide with the points B B, irrespective of the distance B C; that is to say, the patient's cornea gives the same measurement on the scale as it would give if it were in contact with the scale. The distance of ten inches between the observer's eye and the keratometer may be accurately obtained by means of a string, one end of which is fastened to the instrument, the other held between the observer's teeth, but after a little practice this control becomes unnecessary; an error of an inch in the position of the observer's eye does not appreciably affect the measurement.

The eyes examined were chiefly those of hospital and private patients suffering from errors of refraction, or from trivial ailments such as could not invalidate the result. In order to see a sufficient number of healthy eyes in very old people, I examined, in addition, forty old women in an almshouse and twenty-four old men in a workhouse.

Apart from these normal cases I measured the corneas of a number of persons suffering from primary glaucoma. The two groups must be considered separately.

The Cornea in Healthy Eyes.—Five hundred persons were examined, 250 males and 250 females, giving a total of 1,000 eyes, belonging in equal numbers to the two sexes, and representing all periods of life from five to ninety years of age. Eyes in which the ciliary region showed any trace of morbid change, and eyes the refraction of which could not be ascertained, were excluded. The age, sex, and refraction were noted in every case. An analysis of the 1,000 measurements is presented in the accompanying Table A. The following are the salient points:

1. The horizontal diameter of the cornea, in persons between five and ninety years of age, usually measures from 11 to 12 mm.; the average is 11·6 mm.; variations beyond these limits are present in about five per cent.

2. The average increases little, if at all, after five years of age. The slight difference between the second and third life-periods seen in the table is too small, in relation to the number of eyes examined, to have any positive value, especially as there is no difference between the first and second periods. The cornea, or at least its visible part, attains its full diameter many years before the rest of the body completes its growth.[63]

[63] It is interesting to compare this precocious development of the cornea with that of the eye as a whole, and with that of the brain. I take the following data from the valuable tables lately published by Hermann Vierordt, "Daten und Tabellen," Jena. Fischer, 1888. p. 17. Dealing with weight in each case, he gives the following proportions:—The full-grown man is to the new-born infant about as 19 to 1; the full-grown brain to the new-born brain as 3·7 to 1; the full-grown eye to the new-born eye as 1·7 to 1. These figures present in a striking manner the great precocity in size of the infant's brain, and the still greater precocity of the infant's eye, in relation to the body as a whole. Tracing the matter further back, we find that in the embryo the eye develops very early, and is disproportionately large. Further, we find that the development of the cornea is precocious in relation to that of the rest of the eye. Manz says, "The extent of the cornea in the very early stage is large in relation to that of the globe, its surface at a certain time being equal to one fourth of that of the globe; at the end of the third month this proportion is altered to one sixth; in the new-born infant also the cornea is relatively larger than at a later age." ("Graefe-Saemisch Handbuch," vol. ii., p. 41.) See also Emmert, "Auge und Schaedel," Berlin, Hirschwald, 1880, p. 21.

3. There is perhaps a slight diminution in the size of the cornea after forty years of age. Statistics show that the average height of the body is about one twenty-fifth less at ninety than at fifty years of age, and that the weight of the brain is less by more than one twenty-fifth.[64] It seems not unlikely that the slackening of nutrition which causes these changes may also slightly lessen the volume of the eye and the extent of its tunics. This, however, is merely a suggestion ; the difference shown by my measurements is too small to justify a positive statement.

4. The average appears to be slightly greater in males than in females. The difference is extremely slight, but as it is found in each separate life-period it is probably real.

5. The average is the same in hypermetropic, emmetropic, and myopic eyes, and there is no difference even between highly hypermetropic and highly myopic eyes. This equality was unexpected. It seemed reasonable to suppose that the ill-developed hypermetropic eye would be found to have a somewhat smaller, the over-distended myopic eye a somewhat larger, cornea than the emmetropic eye. The idea proved incorrect. As a crucial test I tabulated separately ninety highly hypermetropic and ninety highly myopic eyes. In all of these the refractive error was greater than 4 D., and the list contains hypermetropias of 7, 8, and 9 D., and myopias as high as 20 D. The averages are, for the hypermetropes 11·58, for the myopes 11·53. The slight differences between the three types of refraction seen in the table have probably no actual value. It may safely be asserted that the size of the cornea bears no definite relation to the refraction of the eye.

6. There is very rarely any difference between the two eyes of the same individual, and this is true even when they differ widely in refraction. Such a difference was found in only 4 of

---

[64] Vierordt, loc. cit., pp. 8 and 40.

Horizontal Diameter of Cornea. Table A.—*Healthy Eyes.*

500 persons { 250 males / 250 females } 1000 eyes.

General average	11·6 mm.	Number measuring	13·5 mm.	2 =	·2 per cent.
Maximum	13·5 ,,	,, ,,	13 ,,	2 =	·2 ,,
Minimum	10·5 ,,	,, ,,	12·5 ,,	30 =	3·0 ,,
		,, ,,	12 ,,		
		,, ,,	11·5 ,,	949 =	94·9 ,,
		,, ,,	11 ,,		
		,, ,,	10·5 ,,	17 =	1·7 ,,
				1000	

Classified according to age and sex.

Life period	5 to 9		10 to 19		20 to 39		40 to 59		60 and above.	
Sex	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
No. of corneas	54	40	124	132	130	124	100	100	92	104
Maximum	12·5	12·	12·5	13·5	12·5	12·5	12·	12.	12·5	12.
Minimum	11·	11·	11·	11·	10·5	11·	10·5	10·5	10·5	10·5
Average	11·74	11·57	11·7	11·65	11·73	11·7	11·52	11·43	11·54	11·38
Average for each period	11·67		11·67		11·72		11·48		11·46	

Classified according to age and refraction.

Life period	5 to 9			10 to 19			20 to 39			40 to 59			60 and above.		
Refraction	H.	E.	M.	H.	E.	M.	H.	E.	M.	H.	E.	M.	H.	E.	M.
No. of corneas	64	18	12	112	66	78	87	92	75	108	58	34	138	32	26
Average	11·62	11·83	11·66	11·72	11·72	11·55	11·62	11·82	11·7	11·44	11·5	11·54	11·51	11·34	11·34
Av'ge for each period	11·67			11·67			11·72			11·48			11·46		

General average for hypermetropes, 11·58; for emmetropes, 11·67; for myopes, 11·59.
Average for H. greater than 4 D. (90 eyes), 11·58; for M. greater than 4 D. (90 eyes), 11·53.

Horizontal Diameter of Cornea. Table B.—*Primary Glaucoma.*

112 persons (average age 57) { 51 males, 61 females } 216 eyes { 169 glaucomatous, 47 non-glaucomatous.

General average	11·17mm.	Number measuring	13	mm.	2 =	·92 per cent.
Maximum	13 ,,	,,	12·5	,,	4 =	1·85 ,,
Minimum	10 ,,	,,	12	,,		
		,,	11·5	,,	161 =	74·54 ,,
		,,	11	,,		
		,,	10·5	,,	40 =	18·52 ,,
		,,	10	,,	9 =	4·17 ,,
					216	

Classified according to age and refraction.

Life period	Under 20.	20 to 39				40 to 59				60 and above.			
Refraction		H.	E.	M.	?*	H.	E.	M.	?	H.	E.	M.	?
No. of corneas	0	3	9	4	2	33	27	8	26	38	31	11	24
Average		10·66	11·05	10·75	11·00	10·85	11·05	11·37	11·19	11·16	11·40	11·82	11·27
Average for each period		10·92				11·05				11·33			

* Eyes, the refraction of which could not be ascertained, are placed in the fourth column in each life period.

Dimensions of Globe, &c. Table C.—*Healthy Eyes,*

No.	Age.	Sex.	Cornea.		Globe.			Lens.		Refract.
			Horizont.	Vert.	Horizont.	Vert.	Ant. Post.	Transv.	Ant.-Post.	
			mm.	mm.	mm.	mm.	mm.	mm.	mm.	
1.	40	M.	10·5	10·5	24·5	23·	23·5			E*
2. (Fig. 55).....	26	M.	11·	10·	23·	23·	24·	8·	5·25	M. = 2. D.
3.	31	F.	11·	10·5	22·	22·	22·			
4.	69	F.	11·	10·5	24·	23·	24·			
5.	29	M.	11·	10·5	23·	22·5	24·			E. (atropine).
Averages	39		10·9	10·4	23·3	22·7	23·5			
6.	49	M.	11·5	10·5	24·	24·	25·			M. = 1. D.
7.	61	F.	11·5	10·5	25·	24·	28·5			M.=15.D.,about
8. (Fig. 63).....	70	M.	11·5	10·5	23·5	24·	24·	9·5	6·25	
9.	19	M.	11·5	11·	24·	25·	25·			
10.	20	M.	11·5	11·	23·5	23·	23·5			E.
11.	48	F.	11·5	11·	24·5	24·	24·5			H. = 1. cyl. vert.
12.	39	F.	11·5	11·	24·	23·5	24·			E.
13.	67	M.	11·5	11·	24·5	24·	24·5			
14. (Fig. 53)	25	M.	11·5	11·5	26·	25·5	25·	9·5	5·	E.
Averages.....	44		11·5	10·9	24·3	24·1	24·9			
15.	47	F.	12·	11·	24·	23·	24·5			
16. (Fig. 59).....	52	M.	12·	11·	26·5	26·	25·5	9·5	5·5	E.
17.	57	M.	12·	11·	24·	24·	24·5	9·	5·25	E.
18.	35	M.	12·	11·5	24·	24·	25·			
19.	38	M.	12·	11·5	25·5	25·	25·	9·5	6·	
20.	40	M.	12·	11·5	23·	23·	25·			
21.	59	M.	12·	11·5	23·5	23·	24·5			
22. (Fig. 61).....	68	M.	12·	11·5	25·5	25·	25·	9·5	6·	
23.	39	F.	12·	11·5	25·5	25·5	26·5			
24. (Fig. 57).....	39	M.	12·	12·	26·5	26·	27·	9·5	6·	
25.	38	M.	12·5	12·	26·5	25·	26·			E.
Averages	47		12·0	11·5	25·0	24·5	25·3			
General averages	44		11·6	11·0	24·4	24·	24·8			

* It is possible that in some of the eyes noted as emmetropic in this series, slight hypermetropia may have been present, for the refraction was in most cases ascertained by the shadow test only ; considerable degrees of hypermetropia were certainly not present.

DIMENSIONS OF GLOBE, &c. TABLE D.—*Primary Glaucoma.*

No.	Age.	Sex.	Duration of Glaucoma.	Type of Glaucoma.	Cornea.		Globe.			Lens.		Refract.
					Horizont.	Vert.	Horizont.	Vert.	Ant. Post.	Transv.	Ant. Post.	
					mm.	mm.	mm.	mm.	mm.	mm.	mm.	
1.	50	F.	4 years.	Chronic.	10·		22·	21·	21·			Hm. = 5. D.
2.	63	M.	3 years.	Chronic.	10·5		23·		23·	9·	5·	E.
3. (Fig. 62)..	67	M.	4 years.	Chronic.	10·5	9·5	23·5	22·5	23·5	9·5	7·	Hm. = 1·5 D.
4. (Fig. 64)..	68	M.	Some wks.	Subacute.	10·5	9·5	23·	22·	23·	9·5	6·	Hm. = 2 or 3. D.
5.	50	F.	4 months.	Acute.	11·	10·	24·5	23·	23·	9·25	6·25	Hm. = 4. D ?
6. (Fig. 54)..	60	F.	8 months.	Subacute.	11·	10·5	21·5	22·5	22·5	10·	6·25	Hm. = 4. D ?
7. (Fig. 60)..	65	F.	2 years.	Acute.	11·	10·	23·5	23·	24·5	10·	6· *	
Averages	60				10·6	9·9	23·	22·3	22·9			
8.	80	F.	1 month.	Subacute.	11·5	10·5	24·	23·	24·	8·75	5·5 *	
9.	70	F.	3 years.	Chronic.	11·5		22·	23*	23·	9·	5·	M. = 1. D.
10. (Fig. 56)..	61	F.	3 years.	Acute.	11·5		22·5	23·	23·5	10·	6·	
11.	56	M.	?	Subacute.	11·5		23·		23·	9·	6·	
12.	50	F.	Some mths.	Chronic ?	11·5	10·	23·5	23·5	25·	9·5	6·	
13.	43	M.	1 year.	Subacute ?	11·5	11·5	26·	25·	25·	8·75	5·75*	
14. (Fig. 58)..	61	F.	3 weeks.	Acute.	12·5	11·5	24·	23·5	24·	9·75	6·5	Hm. = 3. D.
Averages	60				11·6	10·9	23·6	23·5	23·9			
General Averages ..	60				11·1	10·3	23·3	22·9	23·4			

* Cortical cataract.

the 500 persons examined. In 12 of the 500 persons the refraction of the two eyes differed by at least 3 D.; in not one of these was there any discoverable difference in the size of the two corneas.

I now pass to the other group of measurements.

The Cornea in Primary Glaucoma.—I have been able, since my attention was directed to the point, to examine 112 persons —51 males and 61 females—suffering in one eye or in both from primary glaucoma. Their average age was 57. The number of eyes was 216, some of the patients having only one, or only one which could be properly measured. Of these 216 eyes, 169 were glaucomatous, 47 healthy; the whole are included in the analysis which appears in Table B. The following are the general results :—

7. The horizontal diameter of the cornea in persons suffering from primary glaucoma measures, on the average (including unaffected fellow-eyes), 11·17 mm. This is distinctly less than the average in persons with healthy eyes, viz., 11·6 mm.

8. The percentage of unusually small corneas—I mean corneas measuring 10·5 mm. or less, and it will be convenient to speak of these hereafter as "small corneas"—is much greater in the glaucoma-group than in the healthy group, viz., 22·69 against 1·70 per cent. If we exclude all the healthy persons under 40 years of age, the difference is still very marked, viz.. 22·69 against 3·79 per cent.

9. A horizontal diameter of 10 mm. was found nine times in the glaucoma group, *i.e.*, in 4·17 per cent. of the eyes. It was not found once among the 1,000 eyes of healthy persons, Corneas so small as this may no doubt occasionally be met with in healthy persons also, *e.g.*, in certain persons who, later on, will get primary glaucoma; but they are rare.

These figures indicate some definite connection between smallness of the cornea and glaucoma. As to the nature of that connection they tell us nothing.

Is the smallness of the cornea a consequence of the glaucoma? We know that obstructive changes at the angle of the anterior chamber are a part of the glaucoma-process, and it would seem possible, *a priori*, that an apparent smallness of the cornea might depend on some loss of transparency in this region arising therefrom. But this is not the case, for in nine of my glaucoma-patients both corneas were small, while one eye was still perfectly healthy; in one of these glaucoma made its appearance later. Such cases prove that the smallness of the cornea precedes the glaucoma, and is not caused by it. It will appear immediately that a small cornea usually indicates a small globe.

Relative Sizes of Cornea and Globe.—In order to ascertain whether the size of the cornea bears anything like a constant proportion to the size of the globe, I have carefully measured a considerable number of eyeballs. The results are given in Tables C and D.

Table C shows the dimensions of 25 non-glaucomatous eyeballs. Most of these were quite healthy; all were free from any disorder which could have altered their size or form—the myopic process excepted—so that for the present purpose they may be regarded as a series of healthy eyes. Most of them were obtained from the dead subject, and in such cases the normal fulness was restored by insufflation through a hollow needle passed through the sclera into the vitreous. In some instances the refraction had been ascertained during life, and is noted in the table. The size of the lens, in the instances in which it is noted, was ascertained when the frozen globe was bisected after lying for at least four weeks in Müller's fluid. The cases were not selected in any way; the list includes all the adult normal eyes of which I have yet made systematic measurements. They are grouped with reference to the horizontal diameter of the cornea. The following facts appear:—

Horizontal Diameter of Cornea. Table A.—*Healthy Eyes.*

500 persons { 250 males / 250 females } 1000 eyes.

General average	11·6 mm.	Number measuring	13·5 mm.	2 =	·2 per cent.
Maximum	13·5 ,,	,, ,,	13 ,,	2 =	·2 ,,
Minimum	10·5 ,,	,, ,,	12·5 ,,	30 =	3·0 ,,
		,, ,,	12 ,,	949 =	94·9 ,,
		,, ,,	11·5 ,,		
		,, ,,	11 ,,		
		,, ,,	10·5 ,,	17 =	1·7 ,,
				1000	

Classified according to age and sex.

Life period	5 to 9		10 to 19		20 to 39		40 to 59		60 and above.	
Sex	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
No. of corneas	54	40	124	132	130	124	100	100	92	104
Maximum	12·5	12·	12·5	13·5	12·5	12·5	12·	12.	12·5	12.
Minimum	11·	11·	11·	11·	10·5	11·	10·5	10·5	10·5	10·5
Average	11·74	11·57	11·7	11·65	11·73	11·7	11·52	11·43	11·54	11·38
Average for each period	11·67		11·67		11·72		11·48		11·46	

Classified according to age and refraction.

Life period	5 to 9			10 to 19			20 to 39			40 to 59			60 and above.		
Refraction	H.	E.	M.	H.	E.	M.	H.	E.	M.	H.	E.	M.	H.	E.	M.
No. of corneas	64	18	12	112	66	78	87	92	75	108	58	34	138	32	26
Average	11·62	11·83	11·66	11·72	11·72	11·55	11·62	11·82	11·7	11·44	11·5	11·54	11·51	11·34	11·34
Av'ge for each period	11·67			11·67			11·72			11·48			11·46		

General average for hypermetropes, 11·58; for emmetropes, 11·67; for myopes, 11·59.
Average for H. greater than 4 D. (90 eyes), 11·58; for M. greater than 4 D. (90 eyes), 11·53.

Horizontal Diameter of Cornea. Table B.—*Primary Glaucoma.*

112 persons (average age 57) { 51 males, 61 females } 216 eyes { 169 glaucomatous, 47 non-glaucomatous.

General average	11·17mm.	Number measuring	13	mm.	2 =	·92 per cent.
Maximum	13 ,,	,,	12·5	,,	4 =	1·85 ,,
Minimum	10 ,,	,,	12	,,		
		,,	11·5	,,	161 =	74·54 ,,
		,,	11	,,		
		,,	10·5	,,	40 =	18·52 ,,
		,,	10	,,	9 =	4·17 ,,
					216	

Classified according to age and refraction.

Life period	Under 20.	20 to 39				40 to 59				60 and above.			
Refraction		H.	E.	M.	?*	H.	E.	M.	?	H.	E.	M.	?
No. of corneas	0	3	9	4	2	33	27	8	26	38	31	11	24
Average		10·66	11·05	10·75	11·00	10·85	11·05	11·37	11·19	11·16	11·40	11·82	11·27
Average for each period		10·92				11·05				11·33			

* Eyes, the refraction of which could not be ascertained, are placed in the fourth column in each life period.

DIMENSIONS OF GLOBE, &c. TABLE C.—*Healthy Eyes.*

No.	Age.	Sex.	Cornea.		Globe.			Lens.		Refract.
			Horizont.	Vert.	Horizont.	Vert.	Ant. Post.	Transv.	Ant. Post.	
			mm.	mm.	mm.	mm.	mm.	mm.	mm.	
1.	40	M.	10·5	10·5	24·5	23·	23·5			E*
2. (Fig. 55).....	26	M.	11·	10·	23·	23·	24·	8·	5·25	M. = 2. D.
3.	31	F.	11·	10·5	22·	22·	22·			
4.	69	F.	11·	10·5	24·	23·	24·			
5.	29	M.	11·	10·5	23·	22·5	24·			E. (atropine).
Averages	39		10·9	10·4	23·3	22·7	23·5			
6.	49	M.	11·5	10·5	24·	24·	25·			M. = 1. D.
7.	61	F.	11·5	10·5	25·	24·	28·5			M.=15.D.,about
8. (Fig. 63).....	70	M.	11·5	10·5	23·5	24·	24·	9·5	6·25	
9.	19	M.	11·5	11·	24·	25·	25·			
10.	20	M.	11·5	11·	23·5	23·	23·5			E.
11.	48	F.	11·5	11·	24·5	24·	24·5			H. = 1. cyl. vert.
12.	39	F.	11·5	11·	24·	23·5	24·			E.
13.	67	M.	11·5	11·	24·5	24·	24·5			
14. (Fig. 53)	25	M.	11·5	11·5	26·	25·5	25·	9·5	5·	E.
Averages.....	44		11·5	10·9	24·3	24·1	24·9			
15.	47	F.	12·	11·	24·	23·	24·5			
16. (Fig. 59).....	52	M.	12·	11·	26·5	26·	25·5	9·5	5·5	E.
17.	57	M.	12·	11·	24·	24·	24·5	9·	5·25	E.
18.	35	M.	12·	11·5	24·	24·	25·			
19.	38	M.	12·	11·5	25·5	25·	25·	9·5	6·	
20.	40	M.	12·	11·5	23·	23·	25·			
21.	59	M.	12·	11·5	23·5	23·	24·5			
22. (Fig. 61).....	68	M.	12·	11·5	25·5	25·	25·	9·5	6·	
23.	39	F.	12·	11·5	25·5	25·5	26·5			
24. (Fig. 57).....	39	M.	12·	12·	26·5	26·	27·	9·5	6·	
25.	38	M.	12·5	12·	26·5	25·	26·			E.
Averages	47		12·0	11·5	25·0	24·5	25·3			
General averages	44		11·6	11·0	24·4	24·	24·8			

* It is possible that in some of the eyes noted as emmetropic in this series, slight hypermetropia may have been present, for the refraction was in most cases ascertained by the shadow test only ; considerable degrees of hypermetropia were certainly not present.

DIMENSIONS OF GLOBE, &c. TABLE D.—*Primary Glaucoma.*

No.	Age.	Sex.	Duration of Glaucoma.	Type of Glaucoma.	Cornea.		Globe.			Lens.		Refract.
					Horizont.	Vert.	Horizont.	Vert.	Ant. Post.	Transv.	Ant. Post.	
					mm.	mm.	mm.	mm.	mm.	mm.	mm.	
1.	50	F.	4 years.	Chronic.	10·		22·	21·	21·			Hm. = 5. D.
2.	63	M.	3 years.	Chronic.	10·5		23·		23·	9·	5·	E.
3. (Fig. 62)..	67	M.	4 years.	Chronic.	10·5	9·5	23·5	22·5	23·5	9·5	7·	Hm. = 1·5 D.
4. (Fig. 64)..	68	M.	Some wks.	Subacute.	10·5	9·5	23·	22·	23·	9·5	6·	Hm. = 2 or 3. D.
5.	50	F.	4 months.	Acute.	11·	10·	24·5	23·	23·	9·25	6·25	Hm. = 4. D ?
6. (Fig. 54)..	60	F.	8 months.	Subacute.	11·	10·5	21·5	22·5	22·5	10·	6·25	Hm. = 4. D ?
7. (Fig. 60)..	65	F.	2 years.	Acute.	11·	10·	23·5	23·	24·5	10·	6· *	
Averages	60				10·6	9·9	23·	22·3	22·9			
8.	80	F.	1 month.	Subacute.	11·5	10·5	24·	23·	24·	8·75	5·5 *	
9.	70	F.	3 years.	Chronic.	11·5		22·	23·	23·	9·	5·	M. = 1. D.
10. (Fig. 56)..	61	F.	3 years.	Acute.	11·5		22·5	23·	23·5	10·	6·	
11.	56	M.	?	Subacute.	11·5		23·		23·	9·	6·	
12.	50	F.	Some mths.	Chronic ?	11·5	10·	23·5	23·5	25·	9·5	6·	
13.	43	M.	1 year.	Subacute ?	11·5	11·5	26·	25·	25·	8·75	5·75*	
14. (Fig. 58)..	61	F.	3 weeks.	Acute.	12·5	11·5	24·	23·5	24·	9·75	6·5	Hm. = 3. D.
Averages	60				11·6	10·9	23·6	23·5	23·9			
General Averages ..	60				11·1	10·3	23·3	22·9	23·4			

* Cortical cataract.

1. There is no constant proportion between the two diameters of the cornea; or between the several diameters of the globe; or between the diameters of the cornea and those of the globe. Thus, the ratio between the horizontal diameter of the cornea and that of the globe is in one case 1 : 1·9 (No. 20); in another it is 1 : 2·33 (No. 1).

2. There is, however, on the average, a fairly regular proportion in these respects. The cornea is, as a rule, somewhat larger in its horizontal than in its vertical diameter. The globe is the same, though the difference is smaller in proportion than that in the cornea. The horizontal and vertical diameters of the globe vary in size, as a rule, with the corresponding diameters of the cornea. (The antero-posterior diameter of the globe cannot be regarded as a normal dimension, unless staphyloma posticum is excluded. In the present series, No. 7 showed marked thinning of the sclera at the posterior pole, with considerable distension.)

Size of the Globe in Primary Glaucoma.—It is clear, then, that small corneas belong, as a rule, to small globes; and, in view of the figures previously given (Tables A and B), we may safely assert that small eyes are more liable than large ones to suffer from primary glaucoma.[65] Direct measurements, so far as I have been able to make them, confirm this conclusion.

Table D shows the dimensions of 14 eyes blinded by primary glaucoma. The list includes every specimen of the

[65] This fact is not altogether new. In a Report on Iridectomy-Operations, performed at the Royal London Ophthalmic Hospital during the years 1857-8-9, Bader, at that time Curator of the Museum, says:—"Glaucomatous eyes are generally small." (R.L.O.H. Reports, vol. ii., p. 229). Brugsch-Bey, of Cairo, has lately reported some similar observations among the Egyptians. He found the average diameter of the cornea to be smaller than in Europeans; he found it to be smaller in eyes suffering from primary glaucoma than in healthy eyes; and he found that primary glaucoma formed a larger percentage of ophthalmic cases than is usual in Europe. He also quotes Professor Rimpoldi as having observed glaucoma more frequently in small eyes than in large ones. (Trans. of Internat. Medical Congress at Washington, vol. iii., p. 752, 1887.) Brugsch-Bey's observations were evidently made quite independently of my own. ("Ophthalmic Review," vol. v., p. 319, 1886.)

kind which I have been able to examine since the question of size presented itself in definite fashion—with the exception of one case in which the glaucoma had begun at the unusually early age of 16, and had distended the globe in all diameters. [Some of my earlier specimens were measured in one way or another before they were mounted, and among them are some remarkably small eyes, but the data are too incomplete for citation.] As in Table C, the eyes are grouped with reference to the horizontal diameter of the cornea, and we find, as before, that the smaller corneas belong, as a rule, to the smaller globes. Comparing the general averages with those in Table C, we see that the glaucomatous eyes are smaller in all dimensions than the healthy :—

	Cornea.		Globe.		
	Horizont.	Vert.	Horizont.	Vert.	(Ant.-Post.)
Healthy	11·6	11·0	24·4	24·0	(24·8)
Glaucomatous ...	11·1	10·3	23·3	22·9	(23·4)

Do these small eyeballs contain proportionately small lenses ? If they do, then the supposed faulty relation between the lens and its surroundings does not belong specially to these eyes, and their special liability to glaucoma must find some other explanation. The question is difficult to answer, for not only are specimens rare, and satisfactory measurements of the lens difficult to make, but each lens must be considered in relation to the age of the patient as well as to the size of the globe which contains it, for the normal relations of the lens change with the advance of life.

Looking first to extreme cases, we find that in eyes which are so small as to be called microphthalmic, a disproportionately large lens appears to be common. In a microphthalmic eye, examined by Hocquard and Masson,[66] the lens was found to be much too large for the eye ; speaking more correctly, the eye was much too small for the lens. The same disproportion was noted by Treacher Collins, in a case published by Lang: "The

[66] Archives d'Ophthalmologie, 1883. p. 231.

interior of the eyeball was filled with the lens, which in proportion to the rest of the eye was larger and more circular than usual."[67] And, again, it is to be seen in the diagrams of microphthalmic eyes, published by Kundrat.[68] Evidently an imperfect development of the tunics does not necessarily affect the lens in equal degree, and this, indeed, is what we might expect from the different origin and independent growth of this latter organ. Microphthalmic eyes with transparent lenses are, I believe, very prone to glaucoma.[69]

Looking to the measurements in Tables C and D, we find that the size of the lens bears no fixed proportion to the size of the globe, even at the same period of life, and that in the glaucoma-group there are several instances of a large lens in a very small eye; but the measurements are too few to justify any general statement.

The best evidence which I can offer concerning the relations of the lens in glaucomatous, as compared with healthy eyes, is to be found in the accompanying series of photo-zincographs. Six healthy eyes and six eyes blinded by primary glaucoma are here presented. Each series is arranged in order of age, and, to make comparison easy, a healthy eye is placed above, a glaucomatous eye below, on each page. In the healthy series it will be noticed that the lenses do not present a regular increase of size in accordance with the age; such a regular progression is only to be obtained by taking the average of many lenses in each life-period. (See chart, p. 87). In the glaucoma series the following features are visible in some or in all of the photographs:—A small globe; a disproportionately large lens; a consequent want of space between the lens and its surroundings; a shallow anterior chamber; a closed filtration-angle; an atrophied ciliary body; an excavated optic nerve.

---

[67] R.L.O.H. Reports, vol. xii., p. 292.

[68] "Ueber die angeborenen Cysten im unteren Augenlide, Mikrophthalmie, und Anophthalmie," by Prof. Kundrat. Bergmann, Vienna.

[69] See, for example, the case by Hocquard and Masson, cited above. Also a case of primary glaucoma at the age of 22, with microphthalmos and hypermetropia, Spencer Watson, Transacts. of Ophth. Soc. March, 1890.

Note to Figures 53 to 64.

The photo-zincographs* represent an enlargement of as nearly as possible two diameters, and in order to control the accuracy of the enlargement a very thin millimetre scale was laid upon the surface of the specimen in each case and photographed with it. Each specimen can, therefore, be accurately measured in the print by means of compasses and its own scale.

The globes will be found to agree very closely with the dimensions given in Tables C and D, although they were not photographed until after at least six weeks' preparation, whereas they were measured immediately after excision. Fig. 55 is however, an exception ; the specimen had lain some months in chloral solution, and the sclera has shrunk a little. The lenses, on the other hand, seem nearly all to have shrunk slightly. The dimensions given in the tables must be taken as the more correct, for they were ascertained immediately after the eyes were bisected, and before the specimens were placed in chloral solution. (For details of methods see Appendix.) Moreover, in almost every case the lens has receded a little from the position which it was seen to occupy when the frozen globe was bisected, and is not, as in the living eye, in contact with the iris.

In the healthy series are one or two eyes which were not, strictly speaking, healthy, for one (Fig. 55) had received an injury, and in two others there were traces of albuminuric retinitis, but there were no morbid changes which could affect the size or form of the lens or globe.

---

* The photographs were taken by Mr. James Potts, of 14, Bennett's Hill, Birmingham, to whom I am much indebted for the skill and pains with which he overcame the special difficulties of the case. They have been well reproduced in zincographic blocks by Messrs. Waterlow and Sons, London.

FIG. 53.—Healthy eye. Horizontal section. Age 25. Refraction emmetropic. (No. 14, Table C.)

FIG. 54.—Primary glaucoma. Horizontal section. Age 60 Refraction, Hm. = 4 D., about. (The healthy fellow eye has Hm. = 4 D., and the patient is confident that the two eyes were alike before the attack.) Glaucoma of subacute type began about eight months, and the eye became quite blind with severe pain and congestion about one month, before excision. (No. 6, Table D.)

In order to fully realise the exceptional relations of the lens in this eye, and in others of the glaucoma-series, one ought to imagine the atrophied ciliary body replaced by one of ordinary size.

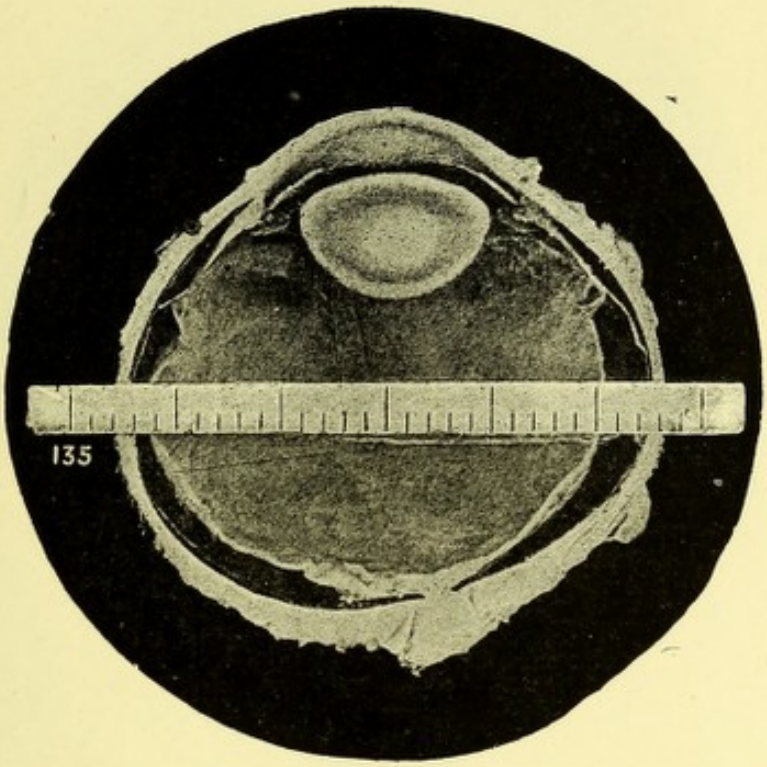


FIG. 53.

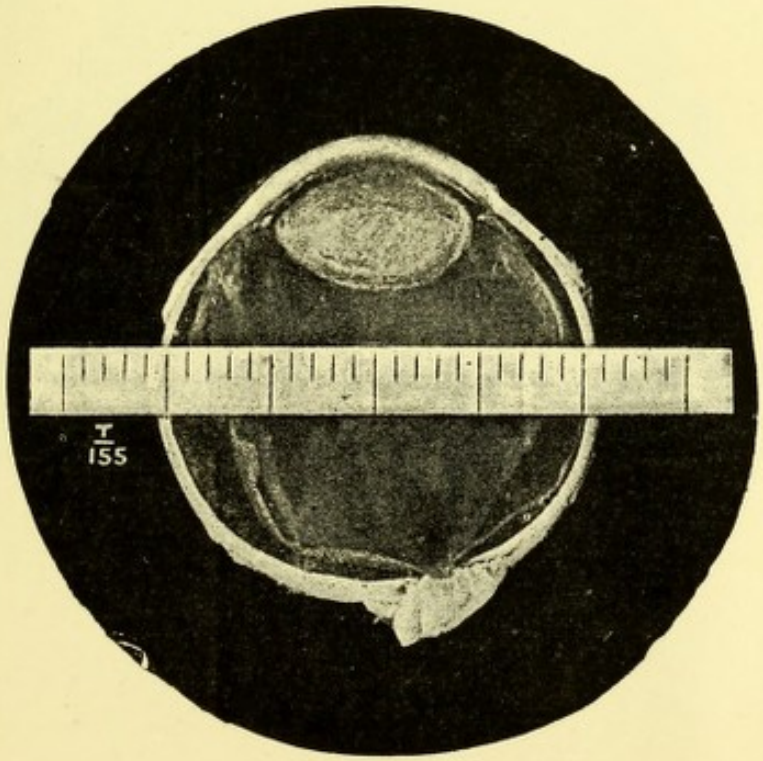


FIG. 54.

FIG. 55.—Healthy eye. Horizontal Section. Age 26. Refraction, M. = 2 D. (No. 2, Table C.)

FIG. 56.—Primary glaucoma. Vertical section passing through site of an iridectomy. Age 61. Refraction unknown. According to the history obtained, the iridectomy had been performed four years before excision, for acute glaucoma, and had resulted in relief of pain but not in recovery of vision. The eye had been quite blind for several years, and painful for some weeks before excision. The lens had become cataractous. Acute glaucoma in the fellow eye cured lately by iridectomy. (No. 10, Table D.)

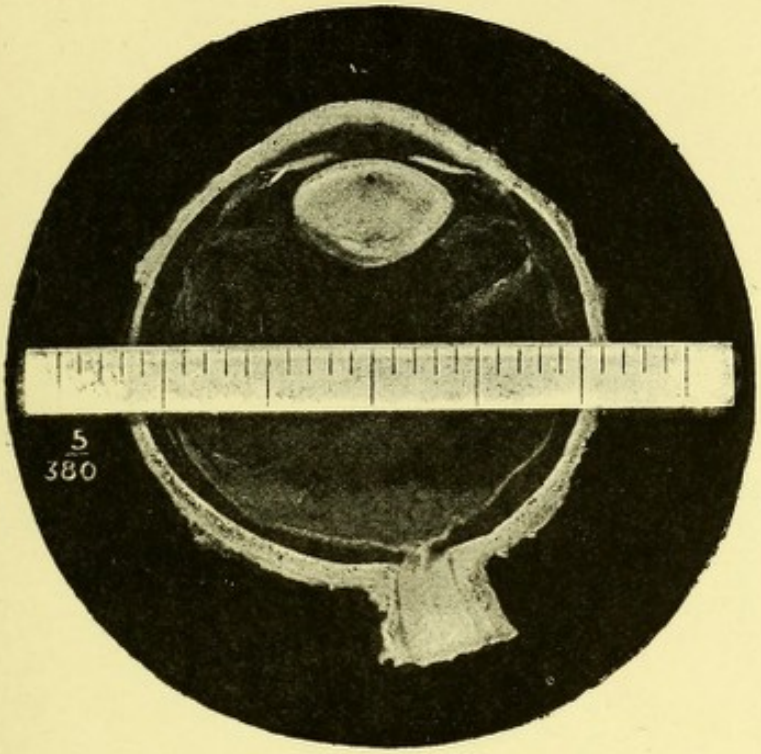


FIG. 55.

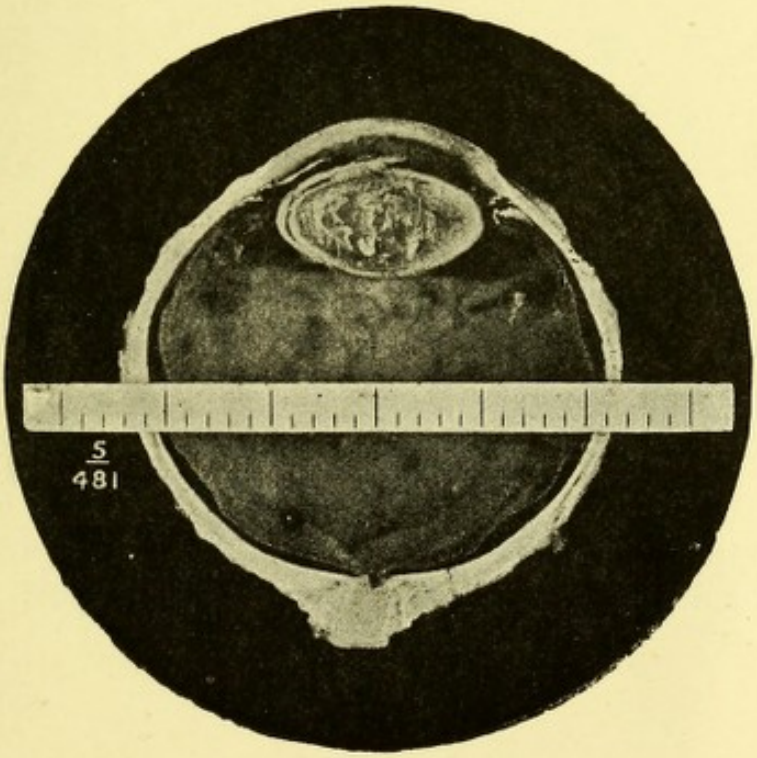


FIG. 56.

FIG. 57.—Healthy eye. Horizontal section. Age 39. Refraction not known. (No. 24, Table C.)

FIG. 58.—Primary glaucoma. Horizontal section. Age 61. Refraction, Hm. = 3 D., ascertained before the commencement of the glaucoma. This eye went blind suddenly without pain, and when seen ten days later showed changes suggesting embolism or thrombosis of the retinal artery, with normal tension. Severe acute glaucoma began four weeks after the sudden loss of sight. Acute glaucoma occurred some months later in the fellow eye, and was cured by iridectomy. (No. 14, Table D.)

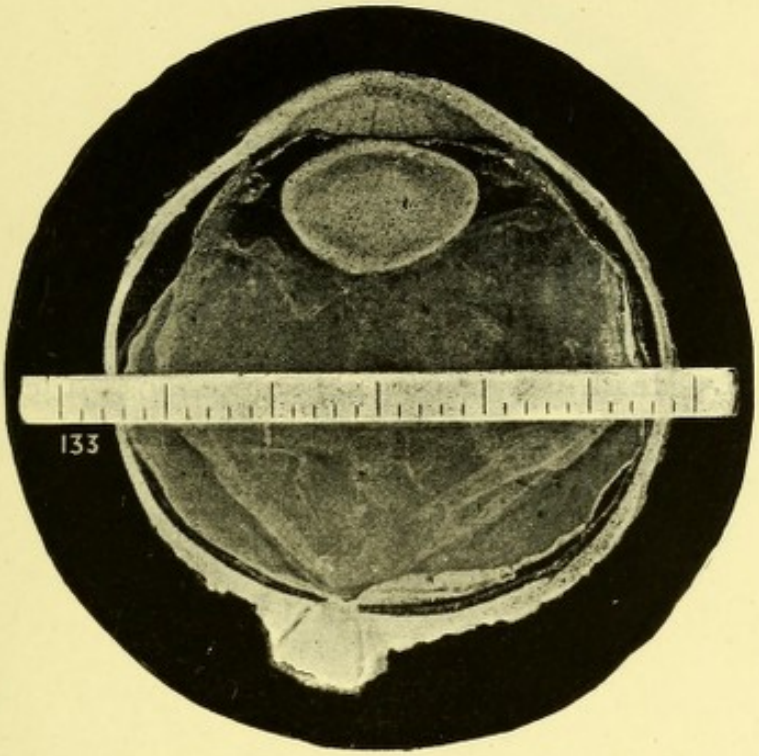


FIG. 57.

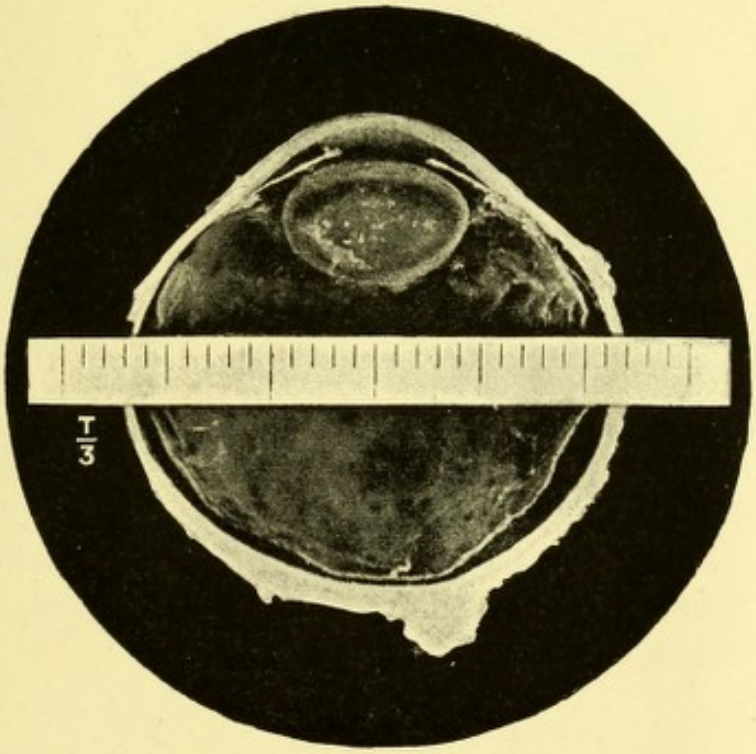


FIG. 58.

FIG. 59.—Healthy eye. Horizontal section. Age 52. Refraction emmetropic. (No. 16, Table C.)

FIG. 60.—Primary glaucoma. Horizontal section. Age 65. Refraction not known. The eye was blinded by acute glaucoma two years before excision. Lens cataractous; a portion of the opaque posterior cortex became dislodged from the capsule when the globe was bisected. (No. 7, Table D.)

The sclera of this eye is thinned and bulging at some parts of the equatorial zone; the section passes through a staphylomatous portion at the right side of the figure, and the globe appears on this account to have a larger horizontal diameter than was actually the case.

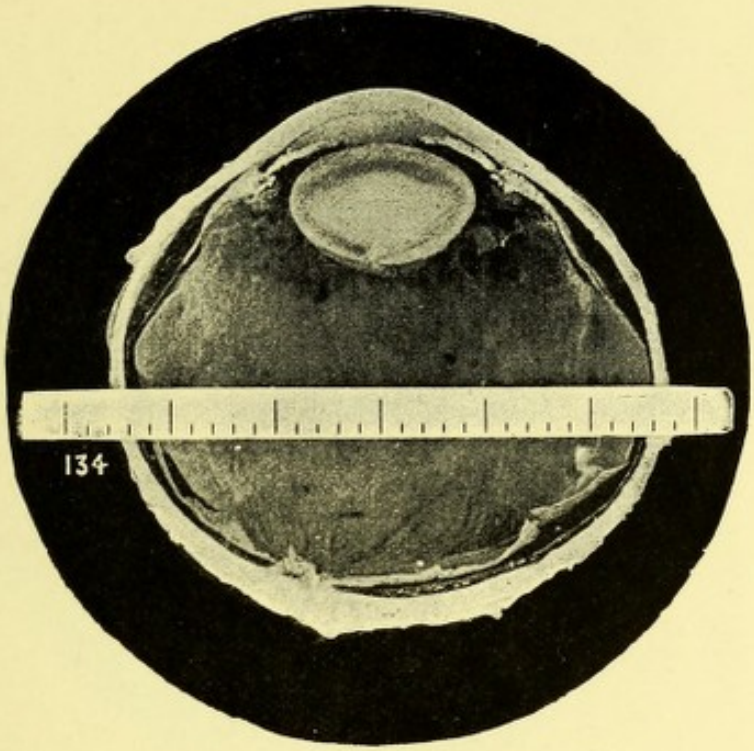


FIG. 59.

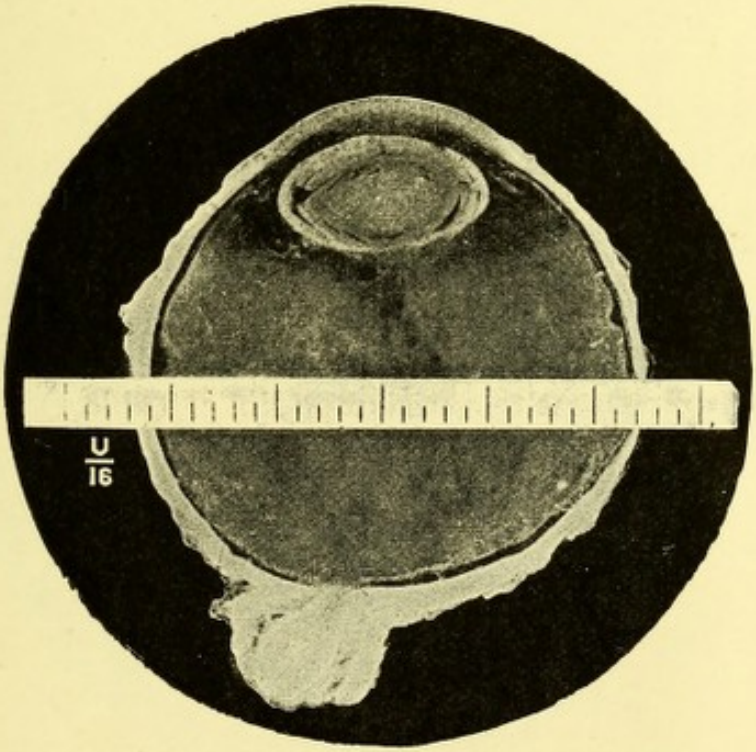


FIG. 60

FIG. 61.—Healthy eye. Horizontal section. Age 68. Refraction not known. (No. 22, Table C.)

FIG. 62.—Primary glaucoma. Horizontal section. Age 67. Refraction, Hm. = 1·5 D., ascertained during the progress of the glaucoma, but before the eye was blind. Chronic glaucoma began at least four years before excision. The patient declined operative treatment while vision was still present. The ultimate occurrence of acute pain and injection when the eye was blind obliged him to consent to excision. (No. 3, Table D.)

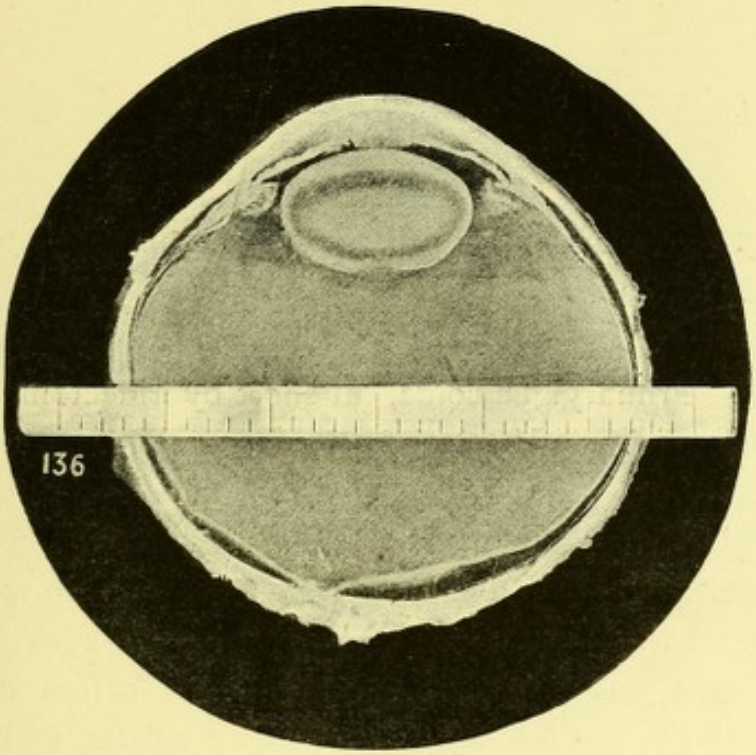


FIG. 61.

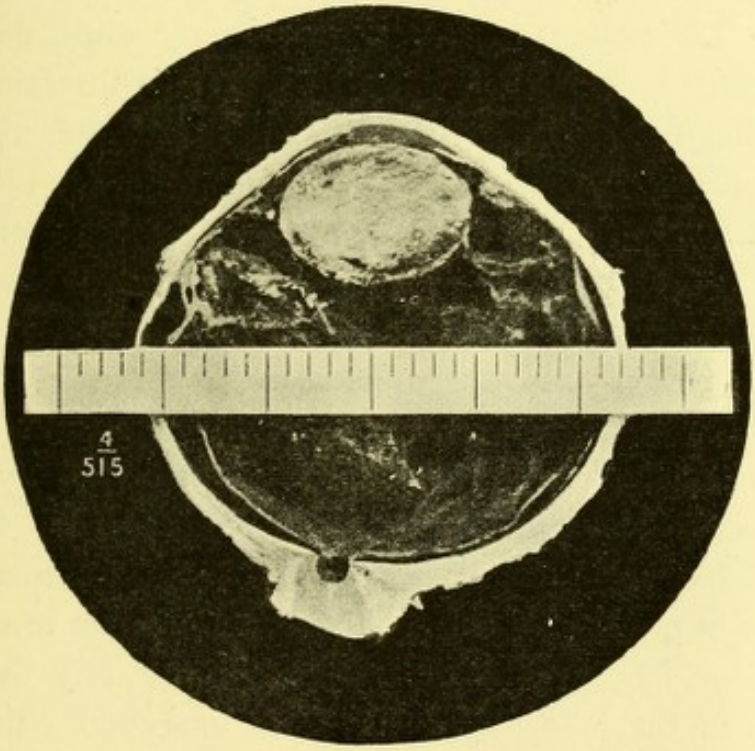


FIG. 62.

FIG 63.—Healthy eye. Horizontal section. Age 70 Refraction not known. (No. 8, Table C.)

FIG. 64.—Primary glaucoma, cut short by iridectomy twenty years before excision. Vertical section passing through site of iridectomy. Age, at time of excision, 68. Refraction, H., amount not known, probably 2 or 3 D., as estimated from the patient's accurate knowledge of the glasses which he wore as a young man. Iridectomy was performed on both eyes for subacute glaucoma, at Moorfields, twenty years before excision. (According to the notes in the Moorfields Register, with which I have been favoured, the right eye—the specimen—recovered more slowly than the left, the wound being still unclosed on the sixth day. The patient was discharged on the ninth day, both eyes doing well.) The left eye recovered, and still retains good vision, so that now, twenty years after the operation, the man still does some work as a clockmaker. The right eye recovered defective vision, so that with it alone he could see, for example, to pick up a tool from his workbench until two years before excision, when it went blind and began to suffer frequent attacks of pain and redness, not due, apparently, to increased tension, —the tension was normal at the time of excision, the disc is not cupped, and there is no atrophy of the ciliary body. The pain appears to have been due to compression of the iris between the lens and the cornea, to both of which structures it had become in places adherent. There is no reason to suppose that the globe has shrunk, for the cornea is exactly the same size as that of the fellow eye, which still has good vision, and which is certainly very small also. (No. 4, Table D. See also Figures 65 to 71.)

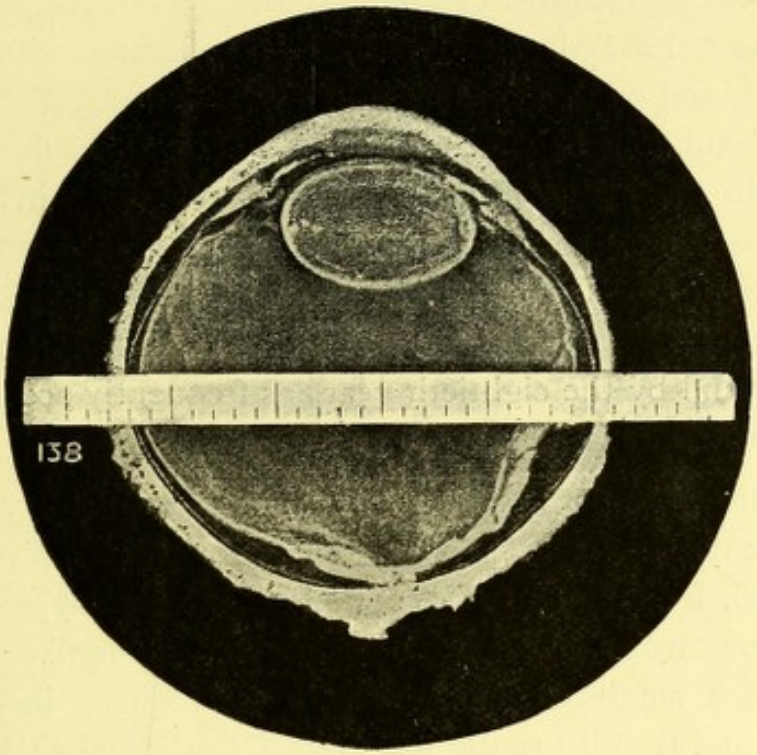


FIG. 63.

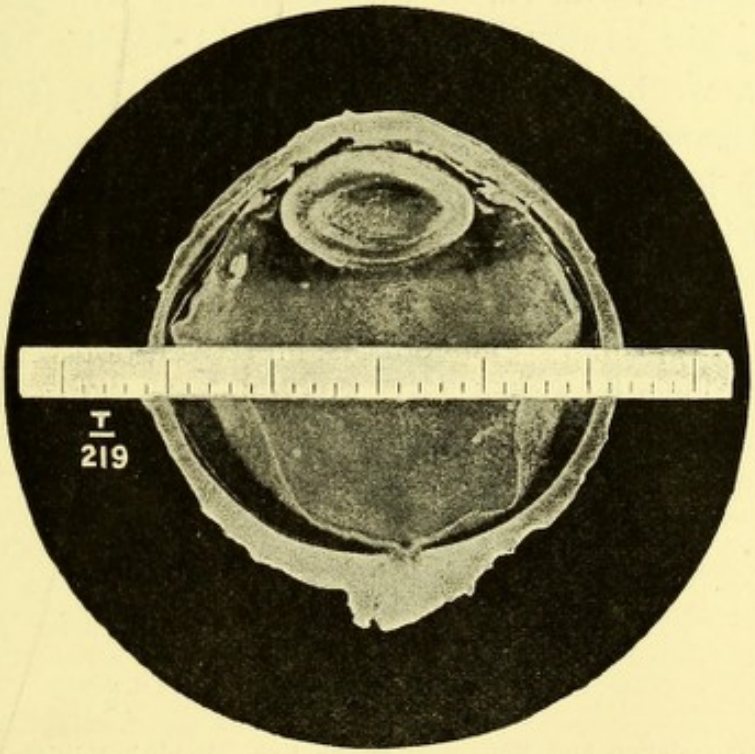


FIG. 64.

Refraction.—Statistics show that hypermetropia is the commonest refractive state in eyes affected with primary glaucoma, and on this account an essential connection between the two conditions has been assumed. We are, perhaps, hardly justified in this assumption. At the time of life when primary glaucoma chiefly occurs, hypermetropia is, in general, very prevalent ; for, as Donders has established, the emmetropic eye tends about the fiftieth year to become somewhat hypermetropic, and by the eightieth year has frequently acquired from 2 to 4 diopters of H.[70]

Further, it must be noted that the special liability of small eyes does not prove a special liability for hypermetropic eyes in general, for small eyes are not necessarily hypermetropic, and hypermetropic eyes are not necessarily small. We have seen that a small cornea is relatively frequent in glaucoma patients, and that it usually indicates a small globe ; but of the 49 small corneas in my own cases (Table B), 21 only were found to be associated with H., 18 with E., and 4 with M. ; in the remaining six cases the refraction could not be ascertained. Moreover, we have seen that hypermetropic eyes have not, as a rule, smaller corneas than emmetropic or myopic eyes. Evidently it is the small eye, rather than the hypermetropic eye as such, which is specially liable to glaucoma.

The preponderance of hypermetropia over the other refractive states in primary glaucoma is, moreover, not very striking. Among my own tabulated cases, excluding the fifty-two eyes the refraction of which could not be ascertained, it is as follows :—

H.	74	=	45·1 per cent.
E.	67	=	40·9 „
M.	23	=	14·0 „
	164		100

[70] Accommodation and Refraction of the Eye. English Edition, p. 208.

And among a much larger number of cases Kryoukoff found almost exactly the same proportion of hypermetropia.[71] Other observers have found a higher proportion; still the figures do not suffice to prove an essential connection between the two conditions.

On other grounds, however, it may be urged that hypermetropia is likely to predispose to primary glaucoma. The ciliary body is, as a rule, more prominent in the direction of the lens in hypermetropic than in emmetropic and myopic eyes, and this, in view of the faulty relations which have been already considered, would seem likely to favour compression of the filtration-angle. And again, the accommodative act is believed by some observers to be much concerned in exciting glaucomatous attacks. (See page 131.)

At present, then, the question of the predisposing influence of hypermetropia must remain open.

Sex.—The differences in liability presented by the sexes appear to be connected chiefly with the exciting causes of acute attacks. They will be considered later. (See page 132.)

Race.—Certain races show a special liability to primary glaucoma Brugsch-Bey found a higher percentage of cases among his patients at Cairo than is common in the clinics of Europe.[72] Moura has noted a much larger percentage among negroes than among whites at Rio de Janeiro.[73] The Jewish

[71] Kryoukoff, in Moscow, found the following refractive conditions in cases of primary glaucoma, H. 43·18 per cent.; E. 28 per cent.; M. 28·78 per cent. Among more than 10,000 consecutive ophthalmic patients tested with regard to refraction, he found H. 43·09 per cent.; E. 27·58 per cent.; M. 29·32 per cent. He therefore concludes that the frequency of H. in glaucoma is not relatively greater than its frequency in general. (Abstract in "Ophthalmic Review," vol. viii., p. 370. See also note by G. A. Berry, vol. ix., p. 58.) But this conclusion is open to the objection that ophthalmic cases in general probably present a higher proportion of H. than does the general population, and therefore afford no fair standard for a comparison of this kind. The only fair comparison would be between a large number of glaucoma patients, on the one hand, and a large number of ordinary persons belonging to the same life-periods, on the other.

[72] Trans. of Internat. Medical Congress, Washington, vol. iii., p. 752, 1887.

[73] The same, p. 755.

race has been declared by some writers to be specially liable but on this point the evidence is conflicting.[74] As to the causes of such racial liability, we must wait for further evidence. In the case of the Egyptians it appears to be associated with smallness of the eye. (See foot-note, page 106.)

The predisposing causes which we have considered up to this point are of a physiological, not of a pathological kind. They are connected with the growth of the lens and with variations in the size of the globe. They appear to favour the occurrence of glaucoma simply through an undue crowding together with the structures which surround the filtration-angle. But here it must be expressly stated that these structural peculiarities are not necessarily present. In some cases of primary glaucoma the cornea and the globe are of full size, and in some the age of the patient precludes the supposition of a full-sized lens. They are in my opinion, conditions which give to certain eyes a more than ordinary predisposition to the disease.

In addition to the foregoing, we might reckon among the predisposing causes of primary glaucoma various morbid conditions of the system, and probably certain latent morbid changes in the eye itself, but these can hardly be separated from the exciting causes of the disease.

EXCITING CAUSES OF PRIMARY GLAUCOMA.

Any disturbance which congests the ciliary processes, or displaces the lens forwards, or thickens the iris, is apt, in a predisposed eye, to cause a dangerous compression of the filtration-angle. To realise this danger it is only necessary to observe the relations of these parts in an extreme case, such as is presented in Fig. 54. Clinical observation shows that disturbances of the kind in question are very frequently associated with the onset of glaucoma.

[74] Graefe-Saemisch Handbook, vol. v., p. 66.

Congestion and Inflammation of the Uveal Tract.—The common antecedents of glaucomatous attacks are exposure to cold, constipation, hunger, sleeplessness, bodily and mental fatigue, heart-weakness, bronchitis, hepatic congestion,—in short, various conditions which disturb the circulation and tend to congest the venous system. Evidence of such disturbance is often to be seen in the pinched and sallow countenance and the overfilled veins of the temple and forehead. Moreover, the influences under which the milder attacks subside spontaneously are pre-eminently those which tend to relieve venous congestion, namely, warmth, rest in bed, sleep, purgation, food, and so forth.

Acute glaucoma is often spoken of as an inflammatory disease, and undoubtedly its course is constantly attended by inflammatory changes, but there is no evidence to show that it commonly originates in inflammation. On the contrary, the suddenness with which the earlier and milder attacks appear and disappear, the fact that a drop of an atropine solution can excite a violent attack in an eye which has not previously shown the slightest sign of inflammation, and that a surgical operation can at once and permanently put an end to the process, are strongly opposed to such a doctrine. More than five-and-twenty years ago Sir William Bowman wrote :—"Glaucoma is in its essence not an inflammation, and, when inflammatory, only so as it were by accident or complication."[75] To my mind, a typical acute glaucoma is just as much an inflammatory disease as a strangulated hernia, and no more. There is, I think, considerable likeness between these two diseases, and I am told that the late Mr. George Critchett was in the habit of drawing the same comparison. In the displacement of the bowel we have a condition, mechanical in its origin, which, for a long period of time, may have no serious consequences, but which may, at its very outset or at any later time, under a slight constriction, be transformed into one of acute and dangerous

[75] R. Lond. Ophth. Hosp. Reports, vol. v., p. 10.

strangulation, with intense engorgement of vessels and outpouring of serum in the directions of least resistance. In the eye threatened by glaucoma we have an unfortunate relation of parts, which, though not itself a disease, may lead, through a little further encroachment upon the already narrowed space, to one of the most formidable of ocular disorders, involving painful distension of the tunics, stoppage of the intraocular currents, throttling of the circulation, and escape of serum into the transparent media and the conjunctiva. Just as the taxis will occasionally remedy the displacement and terminate the danger in the one case, so in some instances will eserine reopen the outlets and relieve the tension in the other; but just as in strangulated hernia it is generally necessary to at once relieve the constriction with the knife, so in most cases of acute glaucoma our only means of cure is to promptly unlock the eye by iridectomy. If, on the other hand, the glaucoma continue unrelieved and the eye be ultimately excised, the pathologist will find in it just such inflammatory changes—exudation of serum and leucocytes, cell-proliferation, adhesion of contiguous surfaces, and so forth—as are found in the sac of a hernia, and neither in the one case nor in the other do these changes reveal the origin of the disorder.

But there are many cases which differ in one way or another from the ordinary type.

An injury is sometimes the starting point of a condition which very closely resembles typical primary glaucoma, and the injury may be of a kind which under ordinary circumstances does little or no damage to the eye; for instance, a wound or contusion of the head without lesion of the eye, a contusion of the eye itself without discoverable lesion, or a slight burn or abrasion of the cornea. In such instances we may, I think, generally assume that the eye was predisposed to glaucoma. In a patient of my own,[76] aged 66, a typical attack followed a

[76] C——G——, $\frac{P.}{304}$

slight abrasion of the cornea by a fragment of coal. Ultimately corneal suppuration set in and the eye was excised. It measured 23·5 mm. horizontally, 23·5 vertically, and 23 antero-posteriorly In another patient, aged 57, the eye was blinded in like manner by the blow of a cork from a soda-water bottle. The globe, on excision, was found to be remarkably small; unfortunately its exact dimensions were not noted. The condition of the filtration-angle is shown in Fig. 41. In the case of a man aged 56, seen with my friend Mr. Hodges, acute glaucoma was excited in one eye by the presence of a foreign body on the cornea. This eye was cured by iridectomy, and very shortly afterwards glaucoma arose spontaneously in the fellow eye; a sequence strongly pointing to predisposition. In these traumatic cases the origin of the outbreak is probably an active hyperæmia rather than a passive congestion, and there is a special tendency to inflammatory exudation, but the direct cause of the high tension appears to be the same as in the more typical cases.

Morbid exudations from the uveal tract are sometimes concerned in the outbreak of glaucoma. If the attack is obviously dependent on cyclitis or choroiditis, as revealed by keratitis punctata or opacity of the vitreous, we should, of course, class the glaucoma as secondary (see page 72); but there are cases, probably of the same nature, which can hardly be distinguished from the primary group. For example, an anæmic woman,[77] aged 25, presented in one eye a well-marked glaucoma of nearly four weeks' duration. The cornea was cloudy, and there appeared to be punctate deposits on its posterior surface; the anterior chamber was shallow; the pupil was free but not dilated; the injection and pain were trivial; the patient was obstinately constipated. Under treatment by purgatives and the local use of cocaine and eserine the tension rapidly fell to the normal, and the ophthalmoscope then revealed

[77] J—— L——, $\frac{T}{212}$

much vitreous opacity, and, when this cleared sufficiently, a large patch of choroiditis. A very similar attack, apparently due to a sudden serous effusion from the ciliary body, and rapidly cured by the same treatment, was observed in a man,[78] aged 54, after a bout of drinking. Again, patients who have been cured of primary glaucoma by an iridectomy, especially those who are anæmic, ill-nourished, and troubled with bronchitis, sometimes return during the cold foggy winter months with an increase of tension in the operated eye. They usually improve when well housed and fed. The likeliest explanation of such recurrences is, I think, that the ciliary body has been secreting an abnormal fluid. Possibly the secretion-process in this organ may be subject, under certain circumstances, to disturbances analogous to those which occur in the kidney. Certainly in dealing with such cases it is very important to attend to constitutional conditions such as anæmia, constipation, lithiasis, bronchitis, &c.

Hæmorrhage from the uveal tract or retina is still more evidently connected with the onset of glaucoma in a considerable number of cases; and while the rupture of a blood-vessel is sometimes, no doubt, a consequence and sign of glaucomatous pressure and obstruction in the eye, we see other cases in which it precedes any symptom of glaucoma by weeks or months. Whether the hæmorrhage is itself the origin of the glaucoma-process, or whether it is merely an expression of a blood or vascular disease which leads also to abnormal secretion into the chambers, it is hardly possible to say. In either case the direct cause of the high pressure appears to be the overfilling of the vitreous chamber and the consequent advance of the lens and compression of the filtration-angle. The condition thus induced bears a close resemblance to primary glaucoma, and in many cases cannot be distinguished from it unless by a knowledge of the antecedent hæmorrhage.

Phlebitis affecting the vortex veins in their course through the sclera has been described as a cause of primary glaucoma.[79] An obstruction of these main trunks would, no doubt, congest the whole of the uveal tract, and might prove a potent cause of glaucoma—I think I have seen high tension induced in this way in the early stage of suppurative tenonitis[80]—but the evidence of its occurrence as a primary lesion in glaucoma is extremely scanty. I have lately examined the vortex veins in 13 eyes lost by glaucoma—primary in 10 cases, secondary in 3—and, for comparison with these, in 6 non-glaucomatous eyes, 3 of which were healthy in all respects. In most cases only one half of the globe was available, so that not more than two of the four venous trunks could be examined. Microscopic sections were made through the sclera in a direction parallel with the equator. In one case of subacute primary, and in one of secondary glaucoma, the veins appear to be partly obstructed by formations in their interior such as have been described and figured by Czermak and Birnbacher. In several cases, both primary and secondary, the vein wall is perhaps thickened, and in some the perivascular space seems to be abolished by the close contact of the vein with the wall of the scleral channel which contains it. This close contact is present also in one of the healthy eyes. Regarded as a whole, the glaucomatous group presents, so far as my observation goes, no distinctive changes. Supposing, however, that a slight phlebitis or periphlebitis is actually present in such eyes, more frequently than I have been able to find it, is it not likely to be a consequence rather than a cause of the glaucoma? If the glaucoma originated in blockage of the veins it would hardly be curable by eserine or iridectomy.

---

[79] Birnbacher and Czermak, Von Graefe's Archiv, vol. xxxii., part ii., p. 1.

[80] For a description of primary tenonitis, see a paper by Fuchs, Wiener Klin. Wochenschrift, 1890, No. 11; abstracted in Ophth. Rev., vol. ix., p. 207. In the cases reported by Fuchs, however, a stage of increased tension was not noted.

Forward Displacement of the Lens.—The experiments described in my first lecture (page 25) showed that a very slight excess of pressure in the vitreous chamber drives the lens and ciliary processes forwards, compresses the filtration-angle, and checks the escape of fluid from the eye. A similar displacement is often discoverable in glaucoma. In the living eye we see that the lens lies very close to the cornea ; after excision we often find that it has receded considerably, leaving the displaced iris to bear witness to its previous advance. This proves that the lens is pushed forwards in advance of its position of normal equilibrium : the vitreous is enlarged at the expense of the aqueous chamber. We have seen that in the normal eye fluid can pass readily from the vitreous to the aqueous chamber (see page 27) ; its failure to do so during an attack of glaucoma appears, therefore, to indicate some obstruction to its passage in this direction. Changes in the hyaloid membrane, in the stroma of the vitreous, or in the constitution of the vitreous fluid, are possible causes of diminished filtration, and, as a fact, in eyes blinded by glaucoma the vitreous is often more distinctly membranous than in healthy eyes, its septa being thickened, or coated by albuminous coagula. In some instances, however, the excess of fluid appears to collect not in the vitreous but immediately in front of it, causing a wide separation of the hyaloid membrane from the ciliary body (see Fig. 62). When the ciliary processes are squeezed between the lens and the iris, any secretion which they still emit must pass backwards, and in this way the blockade must tend to aggravate itself.

Again, as Snellen has suggested,[81] an abnormal slackness of the zonula may be the cause of a forward displacement of the lens. In the young eye, during accommodation, the diameter of the lens diminishes with that of the contracting ciliary circle

[81] Trans. of Internat. Ophthal. Congress at Heidelberg, 1888, p. 249; also "Ophthalmic Review," Feb., 1891, p. 47.

and the zonula remains tense; but in advanced life, when the elasticity of the lens is lost, the zonula must be rendered slack when the ciliary muscle contracts strongly, and the lens may then move forward carrying the iris towards the cornea. An abnormal slackness of the zonula is supposed by Snellen to arise also from degenerative changes, and would satisfactorily explain the occurrence of glaucomatous attacks in connection with some forms of senile cataract. Cataractous lenses are on the average smaller than healthy lenses of the same age (see page 35), and should therefore give some immunity against primary glaucoma. If, however, they are more loosely hung, the occasional association of senile cataract with glaucoma is explained. In a cataractous eye blinded by successive attacks of acute glaucoma, which Dr. G. A. Berry kindly sent me for examination,[82] the lens showed no undue proximity to the ciliary processes, but its anterior surface lay, in the area of the pupil, almost in contact with the cornea; during life it had, in all probability, brought the iris into absolute contact with the cornea over a large area.

[Other observers have maintained that strain of the accommodation in connection with hypermetropia is a potent exciter of glaucoma.[83] The connection can by no means be denied, but, as we have seen, it is rather doubtful whether glaucoma has any special relation to hypermetropia (see page 122), and it must be remembered that the liability to glaucoma is greatest at a time of life when the accommodation is in abeyance. Much book-work is likely to be injurious, apart from the accommodative act, by reason of the stooping and the congestion of the head which it often involves.]

It must be observed here that a shallow anterior chamber does not necessarily indicate a displacement of the lens; it may

---

[82] No. 122 in catalogue. Age of patient, 80.

[83] Walker, Trans. Internat. Med. Cong., 1881, Sect. Ophthal., p. 88; Schoen, Trans. Internat. Ophth. Cong., 1888, p. 257.

be due simply to a lens of excessive thickness (see Fig. 62). An advance of the anterior surface of the lens by natural growth, and in some cases by cataractous swelling, may lead to the same disaster as the forward displacement of the whole lens.

Dilatation of the Pupil; Thickening of the Iris-base.—Atropine, homatropine, duboisin, cocaine, in short, all the drugs which dilate the pupil, must be included amongst the exciting causes of primary glaucoma. The use of any one of these may not only rapidly intensify an incipient glaucoma, it may induce a severe attack in an eye which has previously shown no sign of the disease. It is easy to see that when the filtration-angle is already dangerously narrow, the thickening of the iris-base which accompanies dilatation of the pupil may suffice to complete the blockade.

Influence of Sex.—Finally, in connection with the exciting causes of primary glaucoma, it is interesting to compare the liability of the two sexes as shown by the statistics already given (pages 93 and 95). Men appear to be chiefly liable to the non-congestive, females to the congestive, forms, and the latter seem to be on the whole rather more liable than the former. Probably it would be more correct to say that while the liability to the simple non-congestive form is about equal in the two sexes, women are much more prone than men to congestive exacerbations, or even to anticipate the onset of the chronic disease by attacks of the congestive kind. This special tendency may reasonably be referred to the greater instability of the vasomotor system in women, and particularly to the disturbances of circulation which emanate from the generative organs. So far as regards the predisposition which depends on the build of the eye and the growth of the lens, men and women probably stand on the same footing, but, as regards the exciting causes, women have the disadvantage.

The causes of primary glaucoma, then, are various and complex, and are not yet completely known; but they are alike in this: they all lead to compression of the filtration-angle. With that compression the actual glaucoma-process begins. The escape of fluid is retarded and the intraocular pressure rises; the increasing pressure hinders the flow of blood through the choroidal veins, and augments the swelling of the ciliary processes; this, in its turn, increases the compression of the filtration-angle. The fluid which still exudes from the turgid ciliary body is albuminous and less diffusible than the normal secretion; it tends to accumulate behind the lens, and this latter, being pressed forwards, intensifies the mischief. Thus cause and effect react upon each other in a vicious circle.

In acute glaucoma we see the vascular element at its maximum, in chronic glaucoma at its minimum; in the sub-acute and intermittent forms it seems to ebb and flow under various influences which aid or embarrass the circulation in the uveal tract. The vascular element finds its complement and auxiliary—more marked in some cases, less marked in others—in the predisposing structural condition of the eye.

CONSEQUENCES OF INCREASED PRESSURE.

It is not within the scope of these lectures to discuss in detail the various disturbances which are induced by an excess of pressure in the eye. They are, for the most part, the expression of obstructed circulation, impaired nutrition, and excessive tension of the tunics, and they include the well-known symptoms of glaucoma. Briefly described, they are as follows:—

Œdema of the Cornea.—The cornea receives its nutrient supply from the vessels of the conjunctiva and sclera. The

nutrient streams appear to traverse it in a centripetal direction, meeting at the centre, and passing backwards into the anterior chamber.[84] During the occurrence of high pressure in the chambers, the fluid tends to collect in the form of minute drops beneath the anterior epithelium, and between the fibres immediately beneath Bowman's membrane. Hence the corneal haze, most dense at the centre, which occurs during attacks of glaucoma. The opacity much resembles that which is seen when the freshly excised eye of an animal is firmly squeezed between the finger and thumb, but is not really of the same nature, for while the artificially produced opacity subsides instantly when the pressure is relaxed, and does not involve any discoverable tissue-changes, the glaucoma-opacity requires a definite though short time for its disappearance, and is associated with well-marked tissue-changes. It is distinguished from all forms of inflammatory opacity by the rapidity with which it appears and disappears—sometimes within an hour—in connection with a rise and fall of the tension of the eye.[85]

The loss of sensibility in the cornea which occurs during glaucomatous attacks is due, according to Fuchs, to the maceration and compression of the nerve-filaments by the fluid which collects in the nerve-canals in Bowman's membrane, and to the rupture which they suffer when the epithelium is raised from its surface.

The iridescent vision, which is one of the earliest symptoms in many cases of gloucoma, appears, from the recent observations of Treacher Collins,[86] to be due to the slight disturbance of the epithelium, which is the first stage of the corneal œdema. By applying to the eye a single drop of a 0·125 per cent. solution

---

[84] Pflüger, "Zur Ernährung der Cornea," Klin. Monatsbl. f. Augenheilk., March, 1882. Abstract in "Ophthalmic Review," vol. i., p. 246.

[85] Fuchs, "Ueber glaukomatöse Hornhauttrübung." Von Graefe's Archiv, vol. xxvii., iii., p. 66. Abstract in "Ophthalmic Review," vol. i., p. 126.

[86] "Ophthalmic Review," July, 1890, p. 186.

of hydrochlorate of erythrophloeine, Collins produced slight anæsthesia and slight haze of the cornea, together with blurring of the sight and the appearance of well-marked coloured rings round a flame, resembling those seen in glaucoma. He obtained this result both with a dilated and with a contracted pupil; also in eyes from which the lens had been removed; and in all cases the red circle was the outermost, as in glaucoma. It appears that it is only in the very earliest stage of the corneal œdema, when the haze is very slight, that the coloured rings are seen. When the opacity becomes more pronounced they disappear.

Paralysis of the Ciliary Muscle.—In the act of accommodation the choroid is drawn forwards by the contraction of the ciliary muscle. An increase of the intraocular pressure puts the choroid more tightly on the stretch, increases the resistance which it offers to the ciliary muscle, and thereby lessens the power of accommodation. Stretching and compression of the ciliary nerves may further diminish the power of the muscle. The pressure which falls directly on the muscle no doubt tells in the same direction. In the later stages of the disease the muscle atrophies.

Obstruction of the Circulation in the Uveal Tract.—We have seen that high pressure on the choroid leads to swelling of the ciliary processes, to serous effusion from these organs, to dilatation of the external ciliary vessels, and to œdema of the conjunctiva. During severe attacks the choroid itself appears to become more or less œdematous, and this is, no doubt, partly the cause of the rapid loss of function in the retina, the outer layers of which are nourished by the capillary plexus of the choroid. The compression of the iris-base tends, by interference with the arteries and nerves, to cut off the blood supply to the iris, and to paralyse the sphincter, and thus leads to dilatation of the pupil. The oval dilatation, which is seen in some cases, probably indicates that the pressure on the iris-base is greater at some parts of the circle than at others.

Under long continuance of high pressure, congestion gradually gives place to atrophy. The iris, ciliary processes, and ciliary muscle shrink and retract (see Fig. 38); the choroid becomes thinner and less deeply pigmented, especially in the zone surrounding the optic disc, and in the neighbourhood of the vortex veins. Alterations in the blood vessels—sometimes dilatation, sometimes thickening of the coats with narrowing and even obliteration of the lumen—accompany these changes. The secretion of the intraocular fluid is reduced to a minimum, stagnation prevails in the chambers, and gradually the lens and vitreous degenerate.

Obstruction of the Circulation in the Retina.—The pressure on the retina obstructs both the entrance of the arterial and the exit of the venous stream. The arteries are incompletely filled, the veins congested. Arterial pulsation, invisible in the healthy eye, is often to be seen in the area of the disc: during each diastole of the heart, the high pressure in the vitreous chamber compresses the artery and causes a momentary regurgitation from the eye. In like manner the veins upon the disc may be rhythmically compressed by the transmitted pressure of each incoming arterial wave. This so-called venous pulse occurs only in the small portion of the vein which is close to the point of exit, for the blood-pressure is lowest here, and compression of this portion prevents the expulsion of blood from the adjacent part of the vein. It is doubtless favoured by any structural condition which retards the venous stream near to the margin of the disc, for instance by a sharp bend in the vessel, or by the pressure of an artery crossing it. It is often to be seen in eyes of normal tension. Capillary hæmorrhage occurs not unfrequently as a result of the embarrassment of the retinal circulation.

The lowering of the blood supply and the pressure on the nerve-structures involve loss of sensibility in the retina, and this is no doubt increased by the disturbance in the choroidal circu-

lation which occurs at the same time. The failure of vision is usually greatest at the periphery, least at the centre of the retina, in accordance with natural differences in the force of the blood supply to these regions. It sometimes happens, however, that certain areas are spared, while others which are nearer to the disc are blinded in an apparently capricious manner. Peculiarities in the distribution of the vessels and in the force of the capillary circulation in the respective areas probably underlie these seeming irregularities. If the pressure on the retina be removed in time, nutrition and function are re-established; if it be allowed to persist, the temporary paralysis passes on to atrophy of the nerve-structures, and absolute loss of vision.

Excavation of the Optic Disc.—Under a continuous excess of pressure, the disc—the weakest spot in the envelope of the eye—gradually yields and becomes excavated. Cupping of the disc is not to be discovered during or after a first attack of acute glaucoma, unless the outbreak has been preceded by a chronic excess of pressure of some months' duration. It is not a purely mechanical result of pressure; it includes atrophic changes which, though essentially due to pressure, take time for their development. But even in cases of very short duration, microscopic examinations of the disc in longitudinal section have shown that the first step towards excavation, namely, a depression of the lamina cribrosa, has already been effected. A similar depression of the lamina cribrosa can be produced by artificial pressure in the excised eye.[87]

The gradual contraction of the field of vision which characterises chronic glaucoma appears to be due to the damage which is done to the nerve-fibres in the area of the disc during the process of excavation. The paralysis of the retina is different in character from that which occurs in acute glaucoma. In acute glaucoma there is not the same absolute loss of vision at the

[87] See foot-notes, p. 4.

periphery with comparative immunity at the centre ; the function of the retina suffers more as a whole. In one's own eye it is easy—though, perhaps, not very safe—to annihilate the whole field by external pressure, but it is not possible to obtain a sharply defined peripheral limitation such as occurs in chronic glaucoma. When the excavation of the disc proceeds very slowly, the damage done to the nerve-fibres and the corresponding impairment of the field are sometimes very slight as compared with the depth of the cup.

The retinal circulation suffers additional disturbance through the excavation of the disc: the blood vessels are sharply bent, attenuated, and laterally displaced.

Changes in the Size and Shape of the Globe.—In early life a persistent excess of the intraocular pressure gradually enlarges the globe in all diameters. The curvature of the cornea is flattened and approximated to that of the sclera ; the diameter of the cornea is often notably increased ; the sclera is visibly thinned in the ciliary region ; the eye becomes prominent and highly myopic by reason of its increased length from front to back. The enlargement of the globe often causes a corresponding enlargement of the orbital cavity. To such eyes the name *buphthalmos* is commonly applied ; the term has some practical convenience, but is misleading if it be supposed to indicate a particular disease distinct from glaucoma in general ; buphthalmos is the expression of a persistent glaucoma, usually a secondary glaucoma, in a young eye.

In adult life such changes are much less common, for the cornea and sclera have acquired a resisting power which they do not possess in childhood ; even a long continuance of high pressure appears in many cases to cause no enlargement of the globe, and little if any change in the curvature of the cornea. When any part of the wall of the eye has been weakened by injury or inflammation a circumscribed bulging is likely to occur.

Staphylomata of the cornea, of the ciliary region, and of other parts of the sclera, from this cause, are common in some forms of secondary glaucoma. Such weakening of the tunics may end in rupture and collapse of the globe.

On the other hand, an eye which for a time has been very tense may ultimately soften and shrink by reason of disorganisation of the ciliary processes and arrest of their secretion.

These several results of high pressure vary greatly according as the onset of the pressure is sudden or gradual. The severe vascular disturbance and pain, which commonly occur in the former case, are absent in the latter, presumably because the vessels and the nerves have time to adapt themselves to the new conditions. They vary also with the age of the patient, for reasons already given. Lastly they vary according as the glaucoma is of the primary or secondary form : in many forms of secondary glaucoma the pressure-changes are hidden or modified by the antecedent disease in the eye.

PRINCIPLES OF TREATMENT.

It will be well, in conclusion, to inquire how far our present knowledge of the pathology of glaucoma accords with the rules and methods of treatment which have been established by clinical experience.

If it be true that an increase of the pressure within the eye is the essence of the glaucoma-process, a reduction of this pressure must be the essential object of our treatment. Experience shows that the only measures which retard or arrest the process are those which reduce the tension of the eye. Moreover, seeing that the

morbid process is essentially progressive, and that in its later stages it always annihilates the function of the eye, treatment must be employed early if a permanent disaster is to be avoided. In the advanced or absolute stage, when the eye has lost all perception of light, a reduction of the pressure can avail nothing beyond the relief of pain, and for this purpose excision of the eye is at present the simplest, the most certain, and the safest proceeding.

The treatment of various kinds of secondary glaucoma has already been referred to; that which remains to be discussed is chiefly, though not exclusively, the treatment of the primary form of the disease.

Glaucoma usually calls for operative treatment, for the due escape of the intraocular fluid can seldom be permanently re-established by any other means; but there are some cases which can be successfully treated without operation, and there are very many in which certain auxiliary measures are of great value.

Eserine (Physostigmine) sometimes rapidly relieves the high tension. We owe this important discovery to Laqueur[88] and to Adolph Weber,[89] who appear to have made it independently of each other. Eserine is the antagonist of atropine, which, as we have seen, sometimes induces or aggravates high tension. Other myotics and mydriatics have similar effects. It is important to notice that these drugs cause no decided changes of tension in the healthy eye, and that such small changes as they do produce are usually the opposites of those here in question, eserine tending to raise, and atropine to lower, the intraocular pressure. Their action in glaucoma, therefore, must depend on some abnormality in the glaucomatous eye. This abnormality lies unquestionably in the altered relations of the iris.

[88] Centralbl. der Med. Wiss., 1876, No. 24; and Von Graefe's Archiv, xxxiii., part 3, p. 149.

[89] Von Graefe's Archiv, xxii., part 4, p. 216.

Eserine, by contracting the sphincter of the pupil, thins the iris, flattens its folds, and pulls upon its peripheral insertion. If the filtration-angle is compressed it tends to reopen it. Accordingly, we find that eserine is chiefly useful when this compression is slight or recent. In the sudden, but comparatively mild, attacks which come and go during the premonitory stage of primary glaucoma, it acts with admirable effect. In severe acute attacks, also, it may be useful if applied without delay; but in such cases the sphincter of the pupil is soon paralysed, and the filtration-angle is very firmly closed, hence the period during which it can give relief is soon past. In chronic non-congestive glaucoma, eserine often lowers the tension for a time, but the improvement is seldom great or lasting.

In some forms of secondary glaucoma also—for example, in that which sometimes follows hæmorrhage into the vitreous, and that which is caused by lateral displacement of the lens—eserine sometimes acts just as in primary glaucoma.

In all forms of the disease in which it is unable to contract the pupil, and thereby to increase the patency of the filtration-angle, eserine is, I think, quite useless. When it is useless it is likely to be harmful, for it increases the hyperæmia and often causes pain. On this account the strength of the preparation employed and the frequency of its application should be the minimum which suffices to contract the pupil and to keep it contracted. One grain of sulphate of eserine to the ounce of water (about one-fifth per cent.) is the strongest solution ever required, and a weaker one is often better. Nitrate of pilocarpine is preferred by some surgeons to sulphate of eserine, on account of its feebler action. It is doubtful whether it has any advantage over the latter in sufficiently weak solution.

In one form of secondary glaucoma, namely, that due to the presence of the lens in the anterior chamber, eserine has been known to act as the immediate cause of the attack by

occluding the pupil and thus leading to closure of the filtration-angle, the very complication which, in the reversed position of the parts, it is often able to relieve—a striking proof that its effects depend upon the position of the iris. (See page 53.)

Eserine, therefore, is not a specific for high tension in general. It is a means of combating a particular displacement of the iris, which is very often, but not always, the immediate cause of high tension.

Atropine is injurious in many, but not all, forms of glaucoma. It is harmful precisely in those conditions in which eserine is useful; it induces high tension by dilating the pupil, slackening and throwing the iris into folds, and thereby helping to obstruct the filtration-angle. Hence it is mischievous in the earlier stages of primary glaucoma, and may accelerate the onset of high tension in cases of intraocular tumour and hæmorrhage. When it cannot dilate the pupil it never, I think, raises the tension. In certain cases of secondary glaucoma, in which the high tension is due to serous exudation and not to displacement of the iris, atropine, by subduing the inflammation, tends to restore the normal tension. This is not mere hypothesis. A lady had severe secondary glaucoma from annular posterior synechia, and was iridectomised in both eyes with good result by a well-known surgeon, in another part of the country. Nearly a year later she came under my care, with a recurrence of inflammation and high tension in one eye. She had been strictly warned by two oculists against the use of atropine. In spite of this warning, and against the patient's inclination, I ventured to prescribe atropine, for it could not dilate the pupil, and was likely to subdue the inflammation. The injection and the tension quickly subsided. Again we see cases of serous iritis or cyclitis, with keratitis punctata and no synechia, in which atropine subdues the inflammation and lowers the tension, although it dilates the pupil widely. In these cases the anterior chamber is usually deep, and the tension is due not to a com-

pression of the filtration-angle, but to the serosity of the aqueous fluid, and it may be necessary to empty the chamber by paracentesis in order to re-establish a normal filtration. Eserine is apt to do harm in such cases by increasing the hyperæmia and exudation.

In some cases the choice between a myotic and a mydriatic is very difficult to make. The one or the other must be given tentatively, and the eye must be re-examined after an hour or two, or at latest on the following day.

Cocaine, like every other dilator of the pupil, has been known to induce glaucoma. On the other hand, this drug has the power, invaluable in glaucoma, of contracting the ciliary blood-vessels and diminishing the sensibility of the ciliary nerves, effects which tend to lower the intraocular pressure. By combining cocaine with eserine, in such proportions that the eserine shall have the mastery over the pupil, we may get the advantages of both without the disadvantages. One grain of sulphate of eserine, five grains of hydrochlorate of cocaine, and one ounce of distilled water (equal to about 1-5th, 1, and 100 parts respectively) represent good proportions; in many cases a still smaller proportion of eserine is desirable. Whenever eserine is used in the treatment of glaucoma it should, I think, be combined with cocaine.

Morphine, given internally, will occasionally cut short, and will often alleviate, a glaucomatous attack. It eases pain, lowers the blood pressure, and promotes contraction of the pupil and sleep.

Sleep, even though of very short duration, often dispels the mild premonitory attacks with which primary glaucoma begins. During sleep the pressure in the cerebral vessels falls and the pupil contracts.

Warmth, food, and rest relieve, just as cold, hunger, and fatigue induce, these early and slight attacks.

Aperients sometimes produce a decided effect on the tension and congestion of the eye in those cases, not very uncommon in women, in which an acute or subacute glaucoma is associated with prolonged constipation.

These palliative measures judiciously combined will sometimes restore a glaucomatous eye, for a time, and occasionally for a very long time, to an apparently healthy state. In very many cases they are useful as a preliminary to operative treatment. Even a partial reduction of the tension and congestion of the eye lessens the difficulty of the operation and the risk of after accidents. A night's sound sleep, together with the local action of eserine and cocaine, are often very beneficial in this way. (If cocaine-anæsthesia is likely to be insufficient for the operation—and it usually is so when the eye is painful or injected—it is well, I think, to give a dose of sulphonal or of chloral hydrate an hour before the operation, so as to produce some degree of drowsiness; the patient will then take ether or chloroform more quietly, will need less of it, and will have little tendency to vomiting or excitement afterwards—an important point in these cases. Only in drinkers is this preliminary dose commonly without effect.)

Palliative measures, however, have seldom more than a transient effect, and any discussion of them would do more harm than good if it obscured the fact that the true remedy for glaucoma in the great majority of cases is a timely iridectomy.

Iridectomy, as performed for the relief of glaucoma, consists in the formation of an incision which opens the anterior chamber very near to its periphery, together with the excision of the corresponding segment of the iris. Von Graefe, to whom we are indebted for this invaluable remedy, was unable to explain its mode of action, yet every detail which he laid down with regard to its performance is sanctioned and confirmed by the more advanced pathology of the present day.

In the earlier stages of primary glaucoma, iridectomy appears to act in the following way:—In the first place, the escape of the aqueous humour and the simultaneous advance of the lens immediately reduce the pressure in both chambers. The obstructed circulation in the uveal tract is relieved by the removal of the pressure, and in many cases it is further relieved by a free escape of blood from the divided vessels of the iris. Fluid drains away through the wound for some hours, and the overfilled vitreous chamber is depleted. The ciliary processes recede, the pressure on the iris-base is relieved, and as the anterior chamber re-forms the filtration-angle reopens.

Iridectomy acts with greater certainty in acute congestive, than in chronic non-congestive glaucoma, and it is effective chiefly in the earlier stages of the disease. The reason of this is clear. The pressure on the iris-base is more likely to be removed when it is due to vascular congestion than when it depends on more permanent causes, and the filtration-angle is more likely to reopen when the closure is still recent than when it has lasted many days or weeks. In two eyes examined by Fuchs, which had been permanently cured of acute glaucoma by iridectomy, the filtration-angle was patent throughout the whole circle (Fig. 65).

Besides the cure for the time being, iridectomy commonly affords immunity from attacks of glaucoma in the future. How is this to be explained?

Snellen attributes it to a reopening of the circumlental space in the neighbourhood of the cicatrix.[90] We know that the operation usually produces a permanent alteration in the contour of the eye, for the ophthalmometer and the shadow test reveal, when the healing process is completed, a considerable flattening of the vertical meridian of the cornea. In other words, the region of the cicatrix remains to a certain extent

[90] "Ophthalmic Review," February, 1891, p. 48.

ectatic, and this ectasia must tend to remove the ciliary body from its contact with the lens. For some years past I have made a practice of examining my glaucoma patients, after operation, with regard to the condition of the circumlental space. Looking very obliquely upwards through the coloboma with the ophthalmoscope, one can often discern the lens-margin and a very narrow clear space beyond it. Moreover, one can some-

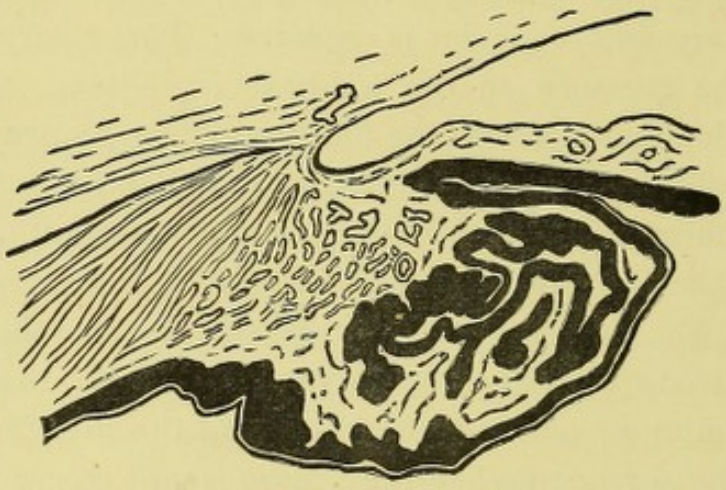


FIG. 65.

From an eye permanently cured of acute glaucoma by iridectomy. (After a drawing by Fuchs.) The patient was a woman aged 66; both eyes were attacked by acute glaucoma at intervals of one year, and both were permanently cured by iridectomy. They were examined after death, seven years later. In both eyes the attack had been of short duration; in the second eye only two days, so that, except for the coloboma, it had probably altered the conditions of the eye but little. The ciliary body was large, both as regards the muscle and the processes. The processes extended almost to the lens on the one side and to the iris on the other. The angle of the anterior chamber was narrowed by the altered position of the iris-base, so that a slight swelling of the processes would have pressed the iris against the cornea. The circumlental space was remarkably small, not through enlargement of the lens, but through enlargement of the ciliary processes. The tissues of the ciliary body were normal, not inflamed.[91]

[91] Von Graefe's Archiv, vol. xxx., part 3, p. 128.

times see marks on the capsule, near to the periphery, which appear to indicate previous contact with the ciliary body or iris. Certainly the operation does often effect a restoration of the circumlental space in the neighbourhood of the cicatrix. Unfortunately, however, this does not always afford immunity from glaucoma in the future, for I have several times been able to discern the lens-margin and the narrow clear space beyond it in eyes which were suffering at that very time from a recurrence of increased tension. This observation, however, by no means shows that Snellen's explanation is generally invalid.

De Wecker, one of the earliest advocates of the retention-theory of glaucoma, maintains that the operation restores a permanent filtration in the region of the wound, either by creating a permeable cicatrix, or by establishing communication with the veins.[92] My own experience leads me to believe that this explanation is true in a very considerable number of cases.

During the healing process the fluid appears to maintain for itself a channel or channels through the cicatrix. The lips of the wound do not come into close apposition, and the overlying conjunctiva remains slightly elevated by the fluid which collects beneath it. In some instances pressure on the globe with the finger, weeks after the operation, will produce a visible extrusion of fluid into the conjunctiva, and a corresponding slackening of the globe—a proof that the cicatrix remains to a certain extent permeable. De Wecker relates the case of a glaucomatous patient who, after iridectomy, was in the habit of relieving the slight recurrences to which he was still liable, by making pressure upon the eyeball with his fingers ; the effect of this, as witnessed by De Wecker himself, was to extrude a small quantity of fluid beneath the con-

[92] "Thérapeutique oculaire," part i., p. 381.

junctiva in the neighbourhood of the cicatrix, and to remove at once the glaucomatous symptoms. Dianoux has recommended the systematic daily application of finger-pressure to the eyeball after sclerotomy, for the purpose of obtaining a less firm and complete cicatrisation of the wound than would otherwise occur.[93]

It is true that in non-glaucomatous eyes, iridectomy causes no permanent reduction of the tension, and that in the eyes of animals it has been found to diminish rather than increase the patency of the filtration-angle.[94] On this ground some writers have maintained that it cannot promote filtration in the glaucomatous eye. The argument is fallacious. In the normal eye the filtration-channel is already patent, and sufficient for its purpose; in the glaucomatous eye the fluid is striving, under abnormal pressure, to find an exit. An iridectomy, if it does not effect the reopening of the whole filtration-angle, is likely to promote the formation of a false passage. What is the behaviour of artificial openings and false passages in other parts of the body—for example, in the urinary organs, the intestine, and the lachrymal sac? If the normal outlet is patent, the artificial opening closes rapidly and completely; if the normal outlet is closed, a vicarious channel is permanently established. It is reasonable to assume that the same rule applies to the eye.

An appeal to the microscope on this point is rarely possible, for the evidence we want is afforded only by eyes which have been cured by the operation, and these very seldom come into the hands of the pathologist. I have lately, however, been able to examine one such eye—the one shown in Fig. 64. As will be seen from the description (page 120), a subacute glaucoma was

[93] "Archives d'Ophthalmologie. Sept.–Oct., 1883, p. 405. Abstract in "Ophthalmic Review," vol. ii., p. 304.

[94] Schoeler "Trans. Internat. Med. Cong., Sect. Ophth.," p. 100; see "Ophthalmic Review," vol. i., p. 216.

cut short in this eye twenty years before excision, and did not return for many years, if at all. The globe was bisected in the vertical meridian through the centre of the coloboma (see Fig. 70). At this point the stump of the iris adheres to the cicatrix, but

FIG. 66.

From an eye cured of sub-acute glaucoma by iridectomy. (See Fig. 64, page 120.) This drawing was made from the bisected globe before thin sections were cut. It shows the condition of the parts at the middle point of the cicatrix. The iris-stump adheres to the lens-capsule and to the cicatrix, but does not pass into the latter, which appears quite solid.

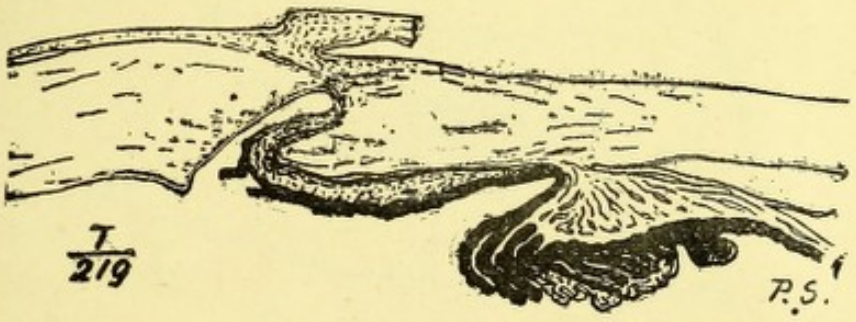


FIG. 67.

From the same eye as the foregoing. This drawing was made from a thin section taken from near one extremity of the cicatrix—the left extremity in Fig. 70. The lips of the wound remain ununited throughout nearly the whole thickness of the cornea. In some adjacent sections the want of union was still more complete, and the parts were held together only by a film of conjunctiva. The iris passes into the aperture.

the latter is solid, and shows no channels in its substance (Fig. 66). But at both extremities of the cicatrix the conditions are different; union is incomplete; iris-tissue is included in the cicatrix; and there are small open channels passing completely through the corneo-sclera, and leading into an open space

FIG. 68.

From the same eye as the foregoing, but from the side of the circle furthest from the cicatrix. This and all other sections examined showed that the filtration-angle had not been properly re-established.

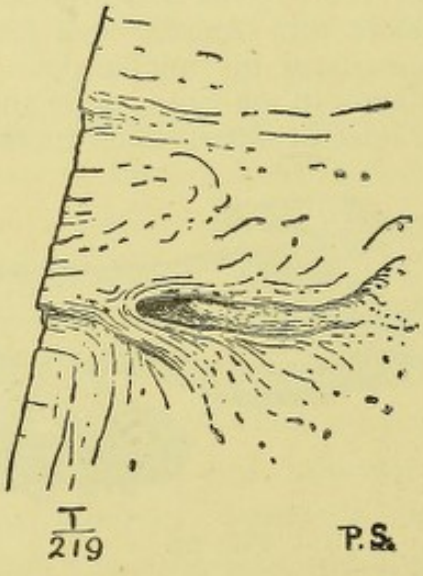


FIG. 69.

From the same eye as the foregoing. This drawing was made from the external surface of one hemisphere, close to the line of bisection—the right hemisphere in Fig. 70. The prominent conjunctiva which covered the cicatrix had been partly snipped off with scissors, and the sub-conjunctival space or channel seen in the drawing was thus exposed.

beneath the thickened and elevated conjunctiva (Figs. 67, 69, 71, 72). This condition is just what a minute examination of the cicatrix in many living eyes which have been cured by

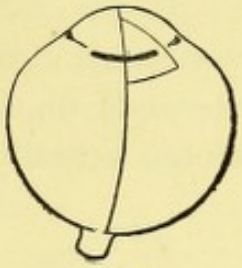


FIG. 70.

Diagram showing the position of the sections represented in the following figures. The globe was bisected in the vertical meridian through the middle point of the cicatrix.

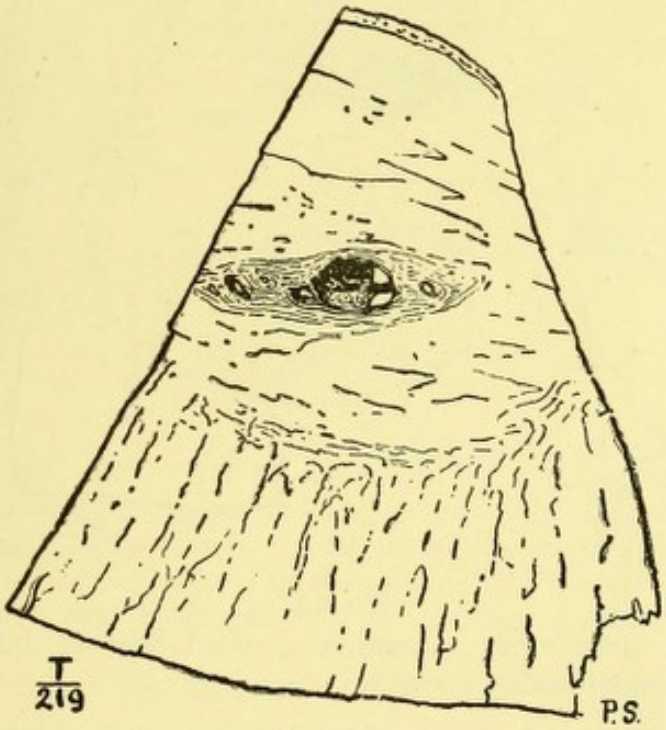


FIG. 71.

From the same eye as the foregoing. A triangular piece including one-half of the cicatrix, as shown in Fig. 70, was cut into thin sections in a direction parallel with its surface. This drawing (Fig. 71) represents a section taken from near the external surface. The lips of the wound at this point have not united, but remain separated by iris-tissue, and by open channels, which lead directly into the sub-conjunctival space seen in Fig. 69. These open channels are obvious in every section.

iridectomy would lead us to expect. Clearly, in some cases, the aqueous fluid maintains for itself, during the healing process, one or more fistulous channels which end externally beneath the reunited conjunctiva.

Until we know to what extent, or in what proportion of cases, a permanent cure depends on these minute fistulas, we cannot decide whether they are actually to be desired or not.

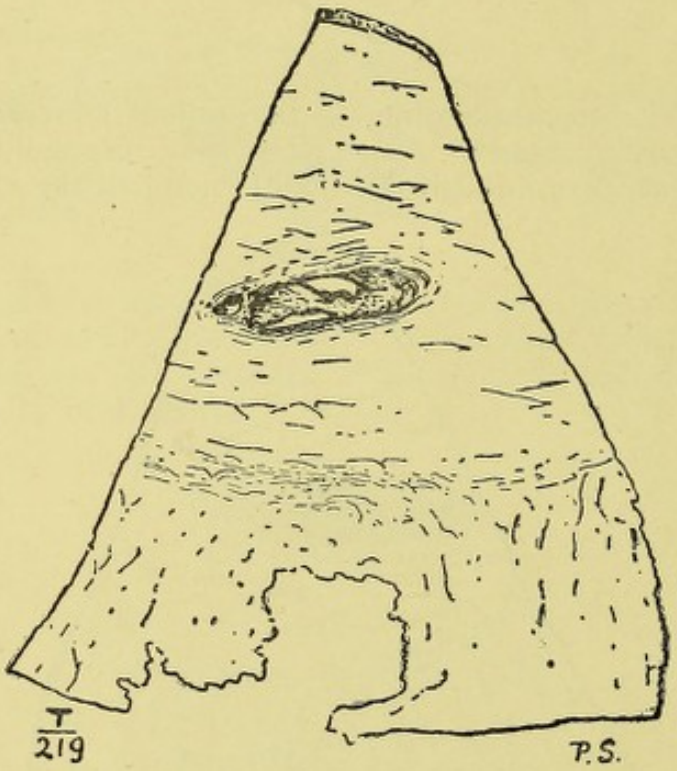


FIG. 72.

From the same series of sections as the foregoing, but rather nearer to the internal surface.

There is no doubt, I think, that the eyes in which they form most frequently, or at least most conspicuously, are those in which the operation is performed late and in which a complete restitution of the filtration-angle can hardly be expected. Moreover the fistulous condition, when strongly marked, appears to expose the eye to some additional risk from septic inflammation in the future.

Iridectomy sometimes fails; it effects neither a reopening of the filtration-angle nor the formation of vicarious channels. The failure may arise in several ways.

The incision may lie too far from the periphery of the chamber; a purely corneal wound is apt to close too quickly, and the resulting cicatrix is less extensible than one which lies largely in scleral tissue. Especially in cases of absolute glaucoma, where a reopening of the filtration-angle is out of the question, is such an operation powerless for permanent good.

The lens may be wounded during the operation and may swell and block the wound. I have found this complication in

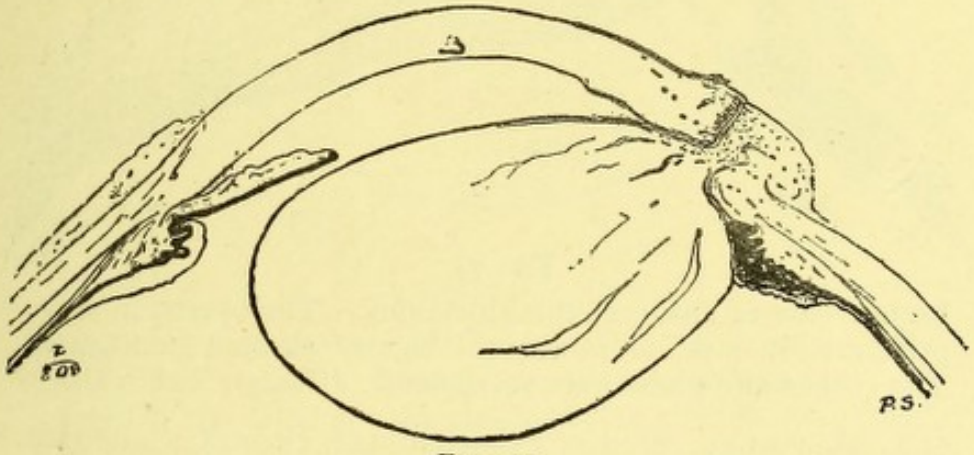


FIG. 73.

From a case of unsuccessful iridectomy. The operation was performed in a very advanced stage of chronic glaucoma, the eye being nearly blind. The lens-capsule was probably ruptured by the pressure of the back of the Graefe-knife, though there was no evidence of the mishap at the time.

a case of my own (Fig. 73), and in several other instances, in all of which, I think, it was unsuspected by the operator.

The uninjured lens may be driven forwards by pressure from behind, and so completely block the wound and annihilate the angle of the chamber that no fluid escapes from the eye after the first hour or two, and the tension is soon as high as, or higher than, before. This formidable complication may occur in spite of the utmost care in performing the operation. It is

most to be dreaded, I think, in cases of chronic non-congestive glaucoma, where the smallness of the cornea shows that we have to deal with an unusually small eye. It indicates retention of fluid behind the lens. Two cases observed in my own practice will serve to illustrate the matter, the second one showing also the way in which, in some cases at least, the anterior chamber may be restored, and the eye saved from destruction :—

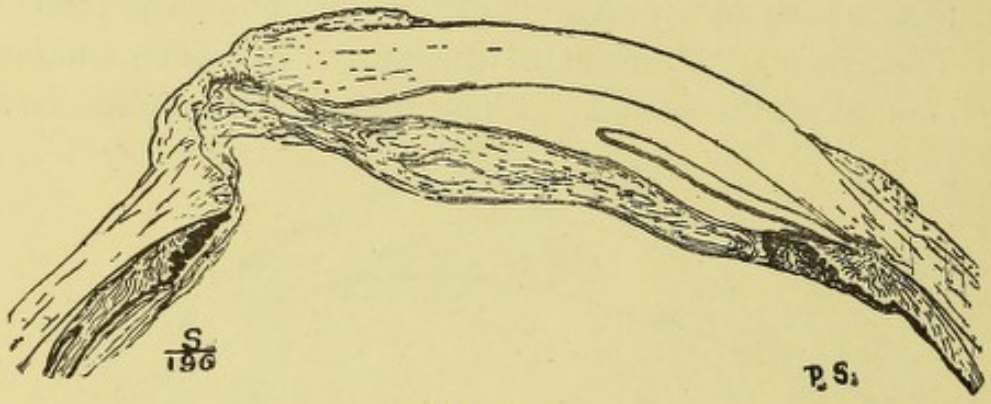


FIG. 74.

From a case of unsuccessful iridectomy. The operation was performed in a rather advanced stage of chronic glaucoma. The cornea and globe were very small. (Case 1, Table D.)

(1.) A woman, aged 50,[94] had simple chronic glaucoma of several years' duration in both eyes. The corneas measured 10 mm. horizontally. The refraction was hypermetropic, 4 D. and 5 D. in the right and left eyes respectively. On the left eye I did what appeared to be a faultless iridectomy, the incision well in the sclera.——The anterior chamber did not re-form. The wound closed at once, and appeared to permit of no drainage from the chambers. High tension persisted. Gradually the lips of the wound separated, and what appeared to be a cystoid cicatrix was formed. I punctured this, and there escaped what I thought at the time was consistent vitreous substance; it was really

[94] J—— A——, $\frac{T.}{196}$

transparent lens matter.——High tension and total abolition of the aqueous chamber still persisted, and the eye was ultimately excised. It proved to be, like the cornea, of exceptionally small size: horizontal diameter 22 mm., vertical 21, antero-posterior 21. (See case No. 1, Table D.) Fig. 74 shows the condition found on bisection.

In this case the disaster was certainly due to a complete obstruction of the outlets by the lens. A glance at Fig. 54,

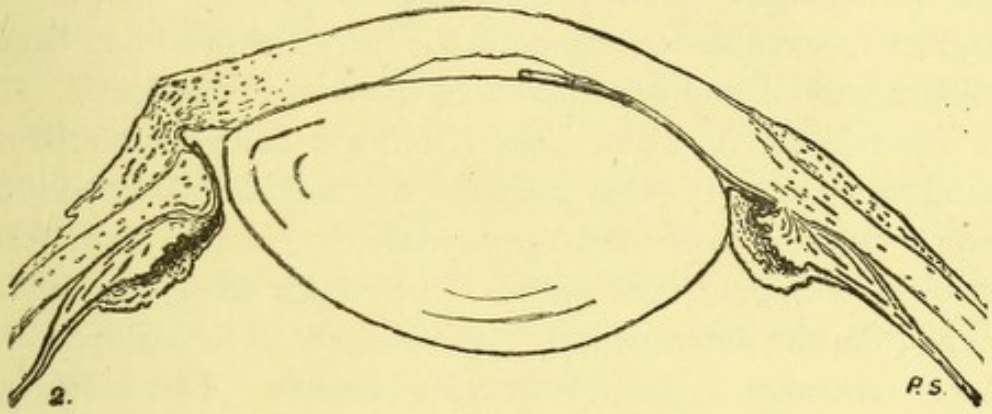


FIG. 75.

From a case of unsuccessful iridectomy. According to the history received with the specimen the operation was performed for the relief of pain, the eye being already quite blind, The relief was only temporary, and the eye was excised a few weeks later. When the frozen globe was bisected the lens was more completely in contact with the cornea than is shown in the drawing; it was pressed against the outer lip of the wound, and completely blocked the aperture.

which represents a very small eye blinded by glaucoma, will show how easily such an obstruction might arise. Fig. 75 was taken from an eye in which the failure of the iridectomy was apparently due to the same cause.

(2.) A woman, aged 70,[95] whose right eye was already lost through neglected primary glaucoma, complained of some premonitory symptoms in the left eye, and had already had one

[95] M—— T——, $\frac{T.}{231}$

distinct attack, an account of which was kindly given to me by Mr. Doyne, of Oxford. This left eye had H 2·5 D., nearly normal vision, an uncontracted field, and a very shallow anterior chamber. I did an iridectomy which, through smallness of the lid aperture, was a little smaller than usual, but satisfactory in other respects.——The chamber did not re-form. On the fourth day it was absolutely empty, the iris being pressed against the cornea throughout; the incision was closed and gave no sign of leakage; elsewhere around the corneal margin the conjunctiva was chemotic; pain in the brow and head had begun 24 hours earlier and was increasing; the eye was hard. The eye was evidently lost unless the anterior chamber could be re-established, and for this purpose the method described by Adolph Weber appeared appropriate.[96] Under chloroform the sclera was punctured with a Graefe-knife at a point about 5 mm. from the outer margin of the cornea, so as to open the vitreous chamber as far forwards as possible. The knife was directed backwards towards the centre of the globe so as to avoid danger to the lens. During its withdrawal it was rotated on its axis so as to effect an L-shaped aperture, more favourable for leakage than a linear puncture. With the vectis or wire loop steady pressure was made upon the cornea and maintained for about 20 seconds. Transparent fluid escaped freely through the scleral wound, and the eye was completely slackened. The usual dressings were applied.——For two days there was a marked improvement, the pain being entirely gone and the œdema diminished, but the anterior chamber did not re-form. Gradually the previous formidable condition returned in full intensity, the iris being closely applied to the cornea and presenting a dome-like curvature such as is rarely seen except in cases of corneal necrosis when the lens is threatening to escape. Again, under chloroform, on the 11th day after the

[96] Von Graefe's Archiv, vol. xxiii., part i., p. 86.

iridectomy, the former proceeding was repeated and carried out more thoroughly than before. The former puncture was reopened. The pressure on the cornea was maintained for ten minutes with occasional intermissions. The effect was very striking. At the end of five minutes the pupillary border of the iris altered in colour, becoming darker and duller than before, and immediately afterwards a similar change took place at the peripheral border. At the end of ten minutes the whole of the iris was altered in colour, and was evidently separated from the cornea by a film of slightly turbid fluid, which also filled the pupil.——The pain did not return, the chemosis quickly subsided, the anterior chamber was permanently reinstated, though shallow, as before the iridectomy ; the tension remained normal. The lens showed some opacities, probably caused by the pressure. Now, five months later, the eye remains free from glaucoma, but the lens is becoming cataractous and will require extraction.

For repression or replacement of the lens in these so-called "malignant" cases, Weber's procedure, as illustrated by the foregoing record, is well calculated to meet the requirements of the case. The escape of fluid through the scleral puncture completely slackens the globe. The pressure on the central region of the cornea forces the lens backwards and removes the pressure of its margin from the ciliary processes and iris-base. Fluid, either from the vitreous chamber or direct from the ciliary processes, insinuates itself around the lens-margin, passes through the pupil and the coloboma, and ultimately between the iris and the cornea. It is, of course, wise to abstain from any such interference so long as we can hope that the anterior chamber will re-form spontaneously. The indications for interference are chemosis around the cornea, with increasing tension.

Should Weber's method fail, it would probably be justifiable to reopen the original iridectomy wound, or to make an

even larger one, and to attempt to extract the lens through a peripheral opening in its capsule. Had the conditions which were actually present in the first of these two cases (see Fig. 74) been clearly intelligible at the time, I should certainly have attempted a more complete removal of the lens, and have followed it, if necessary, by discission of the capsule in the area of the pupil.

With what instrument should the incision for a glaucoma-iridectomy be made—the lance-knife or the Graefe-knife? On this point experienced operators disagree, each claiming advantage for the knife which he is accustomed to use. As a fact, the lens is occasionally wounded by each of these instruments, in spite of irreproachable skill; and, when we consider the conditions with which the operator may have to deal, as shown, for example, in Fig. 54, we cannot wonder that it is so. I now always use a narrow Graefe-knife, because it seems to me to enable one to make a more peripheral incision than could be safely made with the lance-knife. After the point appears in the anterior chamber one can modify the length and the position of the incision according to the space available, by making the counter-puncture a little further forwards or backwards, as may be found practicable. The iris lies between the knife and the lens, and the latter is not likely to be injured if care be taken to keep the plane of the blade parallel to that of the iris, in completing the incision; if the edge be turned forwards in cutting out, the back of the knife may in some cases press upon and rupture the lens-capsule through the intervening iris.

Sclerotomy, whether performed with the linear knife according to De Wecker's method, or with the lance-knife as advocated by Snellen and others, aims at opening the anterior chamber near to its periphery, and leaving the iris intact. Its success in

many cases proves that the essential part of an iridectomy is the incision of the sclera, not the excision of the iris; it does not prove, however, that the latter step is better omitted. The chief disadvantage of sclerotomy is that during the healing process the iris is apt to occlude the wound. In congestive glaucoma it has, I think, the further disadvantage that it does not, like iridectomy, immediately promote a free escape of blood from the turgid vessels. Further, however good the immediate effect of a sclerotomy may be, one would expect the corresponding part of the filtration-angle to be more prone to future occlusion than after removal of the iris-segment. Moreover, a sclerotomy, with incomplete division of the sclera between puncture and counter-puncture, does not produce the ectasia which commonly follows an iridectomy, and which is probably one of the permanent advantages gained by the latter operation. (See page 145.)

The chief use of sclerotomy is as a supplement to iridectomy when the latter fails to permanently reduce the tension. In the absence of the iris-segment it is a simple operation, and seems preferable in every way to a second iridectomy at the opposite side of the circle. In case of need it may be repeated several times, and may, in the end, give a permanently good result. Mr. Anderson Critchett tells me of a case of chronic glaucoma in which, after iridectomy, he performed sclerotomy four times, the benefit proving temporary each time until the fourth sclerotomy permanently relieved the tension.

Sclerotomy is useful also in the scondary glaucoma which sometimes follows cataract extraction. Any capsular or hyaloid membranes which stretch across the pupil should, if possible be divided at the same time. (See page 63.) In these cases also it is sometimes necessary to repeat the operation.

Ciliary punctures have been, and still are, occasionally employed for the relief of glaucoma. An incision which passes in a more or less meridional direction through the corneo-scleral junction, and opens the aqueous and vitreous chambers simultaneously, has been recommended by several operators.[97] Such an incision will give immediate relief in some cases of acute glaucoma and, except in cases of deep anterior chamber, is better calculated to do so than a simple paracentesis of the cornea. But in its effect on the filtration-angle it is obviously inferior to a well-made iridectomy, and it involves the special risks which beset wounds of the ciliary body. The operations in question have not gained general approval.

Scleral punctures or incisions (*posterior sclerotomy*) which open the vitreous chamber only have been recommended for the treatment of advanced glaucoma. The advantage of a scleral puncture behind the ciliary processes is that fluid escapes at once from the overfilled vitreous chamber, and the forward displacement of the lens, which sometimes nullifies the effect of an iridectomy, does not take place. The disadvantage is, so far as my experience goes, that a permanent filtration-channel is not obtained.

In view of some recent commendations of such punctures as a substitute for iridectomy in chronic glaucoma,[98] I tested their effect in five advanced cases in which iridectomy appeared to offer a very uncertain prospect. In each case the puncture was made in the manner already described. (See page 156.) Cocaine sufficed to render the operation nearly painless. In each case a small quantity of clear fluid escaped, and the tension of the globe was immediately reduced. The wound

---

[98] A. S. Buxton, "Hospital Gazette," 1888.
See also Nagel's "Jahresbericht" for 1886, p. 364.

appeared to leak a little for several days, the conjunctiva over it remained elevated, and the eye remained a little slack. The tension-changes were carefully tested from day to day with the tonometer. (See page 12.) On several occasions when the tension was again increasing, finger pressure on the globe produced an additional extrusion of fluid and a reduction of tension. During this time there was no pain, and vision was in some cases a little improved. In the course of from four to fourteen days the tension gradually rose in every case to the same point as had been recorded before the operation.

A scleral puncture is easy to make, nearly or quite painless, and apparently safe, and it fulfils a definite purpose, namely, that of withdrawing fluid from the vitreous chamber so as to lessen for a time the pressure behind the lens. I have many times employed it, and I think with advantage, as an auxiliary to iridectomy in cases of special risk, making the puncture in most instances immediately before the iridectomy, in order to relieve the very high vitreous pressure before allowing the aqueous to escape; in others, immediately after the iridectomy, because there was very little escape of aqueous, and the eye remained unduly hard. Further experience may lead us to employ it methodically in conditions of this kind.

There are, I think, some cases of exceptional gravity in which a preliminary scleral puncture, made some days before the iridectomy, offers the best and, perhaps, the only chance of success :—

A woman, aged 70, whose right eye had been removed eight years previously on account of absolute glaucoma, came last year again under my care with retinal hæmorrhages in the remaining eye. Vision was greatly impaired; but there was no sign of glaucoma, past or present. Acute glaucoma set in six months later, and went on for fourteen days before the patient applied for relief. There was then

great pain, great injection, a very shallow anterior chamber, and only a bare perception of light. Being unwilling to hazard an iridectomy, I made a scleral puncture in the manner already described. Yellowish fluid escaped; the pain and tension were at once relieved. Twenty-four hours later a turbid yellow exudation filled the anterior chamber and hid the iris. Suppurative ophthalmitis appeared imminent, but during the next day or two the exudation rapidly cleared away, leaving a better anterior chamber than before the operation. The exudation was evidently not suppurative, but a result of the sudden removal of pressure from the vessels of the iris or ciliary processes. A week later the patient could count fingers. Under chloroform an iridectomy was then performed without mishap. The patient recovered useful vision, being able to count fingers at arm's length, and to move about with safety, a better result than had appeared possible.

Some years ago, in a case of advanced acute glaucoma, I was obliged to let the disease run its course because the patient was forbidden to take an anæsthetic, and an iridectomy was impossible without it. Under such circumstances I should now make a scleral puncture. It would probably relieve the urgency of the case, and might so far improve the condition of the eye that an iridectomy under cocaine would be possible a week or so later.

Finally, from the pathological point of view, it might appear that primary glaucoma could be radically cured in its early stage by the extraction of the lens in its capsule, but the practical surgeon would shrink from attempting an extraction under such circumstances. The lens being healthy, and sometimes disproportionately large, would demand a larger incision than is required for the cataractous lenses which

we commonly extract, and it would separate from its attachments with greater difficulty. The fact is interesting, however, that the intentional removal of the lens, or its accidental escape after an iridectomy, has proved distinctly beneficial in certain cases of glaucoma.[99]

Our methods of treating glaucoma, and our pathological knowledge are, then, completely in accord, and mutually confirm each other. We need not nowadays speak of the treatment of this disease as empirical. We know, to a large extent, the causes and nature of the morbid process, and we know the action of the measures by which we attempt to arrest it. It may be that our better knowledge of the causes will not supply us with better remedies than those which we now employ, but it will certainly enable us to use them with more discrimination, more confidence, and more success.

---

[99] Rheindorf. Klin. Monatsbl. f. Augenheilk., June, 1887, p. 148. See "Ophthalmic Review," 1887, p. 198.

APPENDIX.

Statistics of Primary Glaucoma in relation to Age, Sex, and Type.*

In order to obtain sufficient material for this inquiry I asked the help of a number of friends, members of the Ophthalmological Society, and provided printed forms for the tabulation of cases on a definite and uniform system. In return I was favoured with a number of lists, some of them very long and representing much labour, and all annotated in a way which showed that care had been taken to make the classification as precise as possible. I desire to express my cordial thanks and my great indebtedness to those who were kind enough to give this help. The following are the names of the gentlemen from whom lists of cases were received:—A. H. Benson, Edgar Browne, E. T. Collins, Richardson Cross, Adams Frost, Hill Griffith, R. Marcus Gunn, F. H. Hodges, G. L. Johnson, Henry Juler, David Little, M. M. McHardy, W. J. Milles, P. H. Mules, Edward Nettleship, Lloyd Owen, A. S. Patton, Henry Power, Simeon Snell, J. B. Story, and H. R. Swanzy. I desire also to express my obligation to the surgical staffs of the Royal London Ophthalmic Hospital, the Royal Westminster Ophthalmic Hospital, the Manchester Royal Eye Hospital, the Liverpool Eye and Ear Infirmary, the National Eye and Ear Infirmary, Dublin, and St. Mark's Ophthalmic Hospital, Dublin, for allowing the hospital registers to be used for the purposes of this inquiry. A series of cases from my own registers completed the list.

* Extract from a paper read before the Ophthalmological Society on March 11th, 1886. Reprinted from the Transactions, vol. vi., by permission of the Council.

The printed form which was sent round read as follows:—

CASES OF PRIMARY GLAUCOMA (NOT SELECTED).

Information is requested on the following points in each case:—

The patient's *sex*.

The patient's *age* when first coming under observation.

The *previous duration of the glaucoma*, if ascertained, reckoning from the first appearance of glaucoma symptoms.

The *type of the glaucoma* in the eye first affected; having regard to the early stages of the disease rather than to its termination.

N.B.—No case is admissible in which the glaucoma followed an injury of the eye, hæmorrhage in the eye, dislocation of the lens, iritis, or tumour of the eye or orbit.

Case.	Sex.	Age.	Previous Duration.	Type.		
				Chronic. Non-congestive; Slow.	Subacute. Congestive; Gradual, or Intermittent.	Acute. Congestive: Rapid, and Intense.

With regard to the three types of the disease it will be seen that below the words chronic, subacute, and acute are placed others in smaller type to indicate the leading characteristics of each variety. In the very large majority of the cases returned to me the type was entered without comment; in a few it was supplemented by a note expressive of some doubt as to the classification; in a few more no type was stated; these last were excluded from the analysis. It is to be noted that the type refers to the character of the disease in its earlier stages; the symptoms which occur in the later stages, when the tissues of

the eye have undergone secondary changes through long continuance of pressure and strangulation, are of less interest in connection with the present inquiry. It must also be observed that wherever in the returns sent to me it was evident that the two eyes of one person represented two cases of glaucoma, I entered the case of the eye first affected, and rejected the other; the inquiry related to persons affected, not to individual eyes.

At the time of closing the list the total number of cases collected was 1,160. Of these it was necessary in the first place to reject 56, viz., 5 on the ground that the glaucoma was stated to be associated with interstitial keratitis, keratocyclitis, or retinitis pigmentosa; 9 because there was no note of the type of the disease; 1 because the sex was not stated; and 41 because the same persons were already entered in connection with the fellow-eyes. In the next place I rejected 99 more for the reason that the previous duration of the disease was not stated. There remained 1,005 cases. Cutting off the last 5 I had 1,000 well noted cases for analysis. The results are shown in the following tables. An analysis of the figures will be found on pages 90 to 95.

TABLE I.—*Frequency.*

1000 Cases of Primary Glaucoma.

Ages in periods of 10 years.		Under 10.	Over 10 and under 20.	Over 20 and under 30.	Over 30 and under 40.	Over 40 and under 50.	Over 50 and under 60.	Over 60 and under 70.	Over 70 and under 80.	Over 80.	Totals.
Males ...	Chronic	...	1	9	23	33	81	74	30	2	253
	Subacute	...	1	3	11	28	25	34	11	...	113
	Acute	...	...	...	5	14	19	19	8	...	65
Total Males		...	2	12	39	75	125	127	49	2	431
Females	Chronic	...	2	4	27	52	58	65	15	...	223
	Subacute	...	1	6	16	47	70	50	13	2	205
	Acute	...	...	1	10	27	40	48	14	1	141
Total Females		...	3	11	53	126	168	163	42	3	569
Males and Females		...	5	23	92	201	293	290	91	5	1000

TABLE II.—*Liability.*

1000 Cases adjusted with relation to the number of each Sex living at each Decade of Life, based upon the foregoing 1000 Actual Cases.

Ages in periods of 10 years.		Under 10.	Over 10 and under 20.	Over 20 and under 30.	Over 30 and under 40.	Over 40 and under 50.	Over 50 and under 60.	Over 60 and under 70.	Over 70 and under 80.	Over 80.	Totals.
Males ...	Chronic	...	·51	4·90	13·84	22·60	66·90	84·42	65·54	16·80	275·51
	Subacute	...	·51	1·64	6·62	19·17	20·65	38·78	24·03	...	111·40
	Acute	...	...	...	3·01	9·59	15·69	21·67	17·48	...	67·44
Total Males		...	1·02	6·54	23·47	51·36	103·24	144·87	107·05	16·80	454·35
Females	Chronic	...	1·02	2·21	16·58	36·22	47·61	70·73	29·36	...	203·73
	Subacute	...	·51	3·32	9·82	32·74	57·46	54·41	25·44	13·50	197·20
	Acute	...	...	·55	6·14	18·81	32·84	52·23	27·40	6·75	144·72
Total Females		...	1·53	6·08	32·54	87·77	137·91	177·37	82·20	20·25	545·65
Males and Females		...	2·55	12·62	56·01	139·13	241·15	322·24	189·25	37·05	1000·00

TABLE III.—*Liability.*

Same as Table II., except that the Subacute and Acute Cases are classed together.

Ages in periods of 10 years.	Under 10.	Over 10.	Over 20.	Over 30.	Over 40.	Over 50.	Over 60.	Over 70.	Over 80.	Totals.
Males ... Chronic (non-congestive)	...	·51	4·90	13·84	22·60	66·90	84·42	65·54	16·80	275·51
Males ... Subacute and Acute (congestive)	...	·51	1·64	9·63	28·76	36·34	60·45	41·51	...	178·84
Total Males	...	1·02	6·54	23·47	51·36	103·24	144·87	107·05	16·80	454·35
Females Chronic (non-congestive)	...	1·02	2·21	16·58	36·22	47·61	70·73	29·36	...	203·73
Females Subacute and Acute (congestive)	...	·51	3·87	15·96	51·55	90·30	106·64	52·84	20·25	341·92
Total Females	...	1·53	6·08	32·54	87·77	137·91	177·37	82·20	20·25	545·65
Males and Females	...	2·55	12·62	56·01	139·13	241·15	322·24	189·25	37·05	1000·00

STATISTICS OF THE WEIGHT, VOLUME, AND SPECIFIC GRAVITY OF THE CRYSTALLINE LENS AT DIFFERENT TIMES OF LIFE.*

One hundred and fifty-six lenses removed after death from the eyes of 91 persons were examined. The interval between the removal of the lens from the eye and its examination was as short as possible, rarely more than an hour, generally much less; and, in order to prevent, as far as possible, any loss or gain of bulk by evaporation or absorption of moisture, the lens was kept, during this interval, immersed in vitreous fluid. When the capsule ruptured, and this happened frequently in the earlier cases, the specimen was discarded.

Each lens was accurately weighed, and immediately afterwards measured as to its volume by means of an apparatus specially devised for the purpose. I am indebted to Prof. Donders for the suggestion which led me to weigh the lenses. Weighing affords an easier and a more accurate means of estimating quantity than does linear measurement, but in the case of bodies like the lens, the specific gravity of which is not constant, it does not suffice to indicate the volume. In addition to weighing, it was necessary for my purpose either to ascertain the specific gravity of each lens, or to measure the volume by some other method. I decided to measure the volume directly rather than to calculate it from the specific gravity. The measurement was made by displacement of fluid along a graduated glass tube. (See Fig. 44, page 86.)

Several possible sources of error were taken into account and eliminated as far as possible. Thus, changes of temperature, occurring during the making of a measurement, would vitiate the result by altering the volume of the fluid. A thermometer

* Extract from a paper read before the Ophthalmological Society, January 11, 1883. Reprinted from the Transactions, vol. iii., by permission of the Council.

was attached to the instrument in order that the absence of such changes might be definitely ascertained. Again, adhesion of the fluid to the internal surface of the graduated tube would cause a considerable error if the column were lowered rapidly and irregularly. To obviate this the nut (N, Fig. 44), was divided round its circumference by eight equi-distant notches, and was turned in all cases at the rate of one notch in a second. The instrument was tested, with the help of Professor Poynting of the Mason Science College, by measuring a series of small bodies of known volumes. The average error was about ·5 cubic mm., the maximum was less than 2· cubic mm. For the purpose in hand, errors of this amount are of little importance.

Immediately after each lens had been measured in the way described, it was laid in a shallow vessel of water and at once measured as to its transverse diameter by means of finely pointed spring compasses. When, as happened several times, the diameter was unequal in different meridians, the mean was stated. In my first few observations this measurement was omitted.

From the age of 20 up to that of 70 I examined more than twenty perfectly transparent lenses in each period of ten years. Beyond 70 years of age opportunities were less frequent, and a large proportion of the lenses examined were imperfectly transparent, so that the desired number of twenty clear lenses in each decade was not reached beyond the age of 70. The whole of the observations are recorded in the tables here appended. In every case in which any opacity was observed in the lens the fact is stated in a foot-note ; the corresponding figures are given in a different type, and are not included in the averages.

In relation to the pathology of glaucoma, the main result of this enquiry is the fact that the growth of the lens does not cease with that of the rest of the body, but is continuous, unless morbid processes intervene, throughout the whole period of life.

(See page 85.) The details of the measurements, &c., appear in the appended tables.

Column D., Table I., shows that the average transverse diameter, like the weight and the volume, continually increases. With regard to this measurement a special source of error has to be noted. When the lens is freed from the traction of the suspensory ligament it undoubtedly alters its form to some extent, the alteration being towards the spherical, and being greater or less according to the elasticity of the lens. It is probable, therefore, that the diameters here given are slightly too small for the living eye, and that the error is greater in the earlier decades than in the later. If, therefore, something must be added to each, and more to the youuger than to the older, the real increase in the transverse diameter during the forty years will be rather less than that indicated by the figures.

The antero-posterior diameter was not systematically measured in my investigations; the measurement is more difficult to make in a trustworthy manner than that of the transverse. The impression derived from inspection was very decidedly that the older lenses were larger in all ways than the younger, and in several instances in which I was able to compare a young and an old lens side by side, there was a more obvious increase in the antero-posterior diameter than in the transverse.

Apart from the main object and result of this research, one or two points which were observed incidentally deserve notice.

The close relation of cataract to senility comes out in a striking manner. Between the ages of 20 and 49 no single instance of any opacity was met with among the sixty-six lenses examined. Between 50 and 59 two lenses* out of twenty-two, *i.e.*, 9 per cent., presented the earliest signs of cataract in the form of slight cortical opacities at the equator. Between 60 and

* These were both from the same individual; one was damaged in extraction, and does not appear in detail in the table.

69 nine out of thirty-two, *i.e.*, 28 per cent., were affected similarly or to a greater extent. Between 70 and 90 thirty-four lenses were examined, and of these no less than sixteen, *i.e.*, nearly 50 per cent. were affected in like manner.

The cataractous lenses are shown on the chart (page 87) by circles, and it is interesting to note that, as a rule, the circles stand at a decidedly lower level than the dots in the same decade; in other words, the lenses in which cataract was beginning were as a rule smaller than transparent lenses of the same age. According to the researches of Becker the commencement of senile cataract is due to the separation from each other of certain of the layers of lens-fibres in consequence of advancing sclerosis and shrinkage, the separation occurring first just where the capsule and subjacent fibres are most affected by the traction of the suspensory ligament, viz., at the equator. My observations are favourable to this view. The subnormal size of the cataractous lenses was observable even in those which presented only very slight cortical opacities, and this suggests that the formation of cataract is preceded by a period in which the rate of growth gradually falls below the normal. In two instances* I was able to compare a completely cataractous lens with a fellow lens which presented only some slight cortical opacities at the equator. In both cases the opaque lens was very much smaller and lighter than its fellow. Here there was evidently a shrinking or shrivelling as the result of the degenerative process.

Finally, I may mention that, in the removal of this series of lenses from the eyes of dead subjects, I obtained constant experience of a fact which has been pointed out by Pagenstecher and others, namely, that the attachment of the lens to its suspensory ligament, and especially to the hyaloid membrane of the vitreous at its posterior surface, is very much weaker in the senile than in the youthful eye.

---

* Table VI., No. 67, and Table VII., No. 31.

TABLE I.—*Averages.*

Age.	A. Weight. mgr.	B. Volume. cub. mm.	C. Spec. grav.	D. Diameter. mm.
20 to 29 ...	174 ...	163 ...	1067 ...	8·67
30 to 39 ...	192 ...	177 ...	1085 ...	8·96
40 to 49 ...	204 ...	188 ...	1085 ...	9·09
50 to 59 ...	221 ...	205 ...	1078 ...	9·44
60 to 69 ...	240 ...	225 ...	1067 ...	9·49
70 to 79 ...	(245) ...	(227) ...	(1079) ...	(9·64)
80 to 90 ...	(266) ...	(244) ...	(1090) ...	(9·62)

N.B.—Above the age of 69 the number of transparent lenses examined was much smaller than in the earlier decades; the averages are given in brackets, and must be taken as less certain than those belonging to the earlier periods. For details see the following tables.

TABLE II.—*Ages 20 to 29.*

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
47	20	M.	159	147	1081	8·5
			—	—	—	—
48	20	M.	—	—	—	—
			195	183	1066	9·
66	20	M.	—	—	—	—
			151	139	1086	8·75
35	21	M.	178	168	1060	9·
			180	172	1046	9·
46	21	F.	167	160	1044	8·5
			180	162	1049	8·5
60	21	M.	—	—	—	—
			195	186	1048	9·
44	22	F.	180	171	1053	8·25
			175	167	1048	8·25
29	23	F.	—	—	—	—
			152	148	1048	8·25
43	24	M.	175	165	1060	9·
			—	—	—	—
41	25	M.	176	168	1048	8·75
			178	168	1060	8·75
68	26	F.	190	172	1105	8·75
			184	166	1108	8·75
4	27	M.	—	—	—	—
			153	144	1062	—
58	27	M.	162	153	1059	8·5
			166	156	1064	8·5
70	28	M.	181	164	1104	8·75
			180	162	1111	8·75
Average			3648 ÷ 21 = 174	3419 ÷ 21 = 163	1067	173·5 ÷ 20 = 8·67

In this and the following tables the upper figures in each bracket represent the right eye, the lower figures the left eye. Where no figures are given, it is to be understood that the lens-capsule was broken during extraction and the specimen thereby rendered useless.

TABLE III.—*Ages* 30 *to* 39.

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
39	32	M.	167	155	1077	8·75
			172	161	1068	8·75
80	33	M.	177	164	1079	8·75
			173	161	1074	8·75
26	34	M.	195	184	1060	9·
			177	165	1073	9·
3	35	F.	197	173	1139	—
			—	—	—	—
7	35	F.	180	170	1059	9·
			—	—	—	—
18	35	M.	183	166	1102	—
			184	166	1108	—
51	36	M.	192	184	1043	9·
			192	177	1085	9·
34	37	M.	196	192	1021	8·75
			—	—	—	—
76	37	F.	206	188	1096	9·
			206	190	1084	9·
42	38	M.	199	184	1081	8·75
			201	190	1058	8·75
8	38	M.	199	179	1111	9·
			—	—	—	—
75	38	M.	197	181	1088	9·
			195	179	1089	9
72	39	M.	215	196	1097	9·5
			216	195	1108	9·5
Average . .			4219 ÷ 22 = 192	3905 ÷ 22 = 177	1085	170·25 ÷ 19 = 8·96

TABLE IV.—*Ages* 40 *to* 49.

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
71	40	M.	217	194	1118	9·
			216	195	1108	9·
33	41	M.	178	166	1072	8·75
			—	—	—	—
86	42	M.	220	198	1111	9·
			—	—	—	—
38	42	F.	217	199	1090	9·5
			215	198	1086	9·5
85	42	M.	189	169	1118	9·
			—	—	—	—
77	43	M.	196	183	1071	9·25
			194	181	1071	9·25
87	44	M.	238	226	1053	9·
			230	217	1060	9·
83	45	F.	194	178	1090	8·75
			191	160	1194	8·5
16	45	M.	—	—	—	—
			194	185	1049	9·5
5	45	M.	204	185	1103	—
			198	179	1106	—
25	46	M.	211	198	1066	—
			—	—	—	—
20	49	M.	199	189	1053	9·5
			—	—	—	—
45	49	M.	193	185	1043	9·
			189	177	1068	9.
1	49	M.	203	183	1126	—
			196	177	1107	—
6	49	F.	214	204	1049	—
			—	—	—	—
Average . .			4696 ÷ 23 = 204	4326 ÷ 23 = 188	1085	154·5 ÷ 17 = 9·09

TABLE V.—*Ages* 50 *to* 59.

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
24	50	F.	208	197	1056	9·5
			213	194	1091	9·5
32	50	M.	240	222	1081	10·
			242	222	1090	10·
13	51	M.	226	208	1086	9·
			222	206	1078	9·
22	51	M.	227	214	1060	10·
			226	207	1091	10·
19	52	M.	—	—	—	—
			191	184	1038	—
64	52	M.	251	237	1069	9·5
			257	250	1029	9·5
23	53	F.	204	186	1097	9·5
			209	196	1066	9·5
11	54	M.	227	214	1060	9·
			227	214	1060	9·
49	55	M.	*220**	*205*	*1073*	*10·*
			—*	—	—	—
10	56	M.	—	—	—	—
			234	211	1109	9·5
17	56	M.	219	198	1106	9·25
			217	199	1090	9·25
65	57	M.	186	170	1094	9·
			188	171	1099	9·
50	59	M.	225	213	1056	9·75
			—	—	—	—
Average . .			4639÷21 = 221	4313÷21 = 205	1078	188·75÷20 = 9·44

N.B.—The *italic figures* in this and subsequent tables represent lenses which were not perfectly transparent; they are not included in the averages.

* Slight cortical opacities at the equator.

TABLE VI.—*Ages 60 to 69*

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
40	60	M.	215	200	1075	9·5
			219	206	1063	9·5
88	60	F.	239	216	1106	9·
			236	213	1108	9·
61	61	M.	248	235	1055	9·5
			242	223	1085	9·5
15	62	F.	*171**	*157*	*1090*	—
			179†	*168*	*1065*	—
30	62	M.	251	233	1077	9·5
			252	243	1037	9·5
52	63	M.	239	224	1067	9·75
			240	224	1071	9·75
84	63	M.	*190‡*	*172*	*1105*	*9·*
			183‡	*169*	*1083*	*9·*
28	65	M.	231	225	1027	9·
			223	211	1057	—
36	65	F.	236	224	1054	9·5
			228	211	1080	9·5
53	65	M.	245	228	1075	9·25
			237	223	1063	9·25
54	65	M.	247	231	1069	9·5
			247	232	1065	9·5
59	65	F.	*199‡*	*179*	*1111*	*8·5*
			200‡	*193*	*1031*	*8·5*
63	66	M.	226	209	1081	9·5
			236	219	1078	9·5
90	68	M.	*263§*	*240*	*1096*	*9·75*
			263	242	1087	9·75
56	69	F.	264	252	1044	10·
			260	245	1061	10·
67	69	M.	*184‖*	*170*	*1082*	*9·5*
			216‡	*199*	*1085*	*9·75*
Average			5524 ÷ 23 = 240	5169 ÷ 23 = 225	1067	208·75 ÷ 22 = 9·49

* Nuclear opacity, with striæ radiating from it.
† Slight nuclear opacity.
‡ Slight cortical opacities at equator.
§ One flake of opacity deep in cortex.
‖ Completely cataractous.

TABLE VII.—*Ages 70 to 79.*

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
12	71	M.	237	226	1049	—
			—	—	—	—
91	71	M.	—	—	—	—
			220	204	1078	9·25
69	72	M.	249	228	1092	9·75
			237	215	1102	9·5
92	72	M.	282	256	1100	9·5
			284	258	1100	9·5
2	73	M.	244	225	1084	—
			243	222	1095	—
55	75	M.	*218**	*205*	*1063*	*9·25*
			*212**	*195*	*1087*	*9·25*
57	75	F.	*229†*	*213*	*1075*	*9·75*
			231	222	1041	10·
81	75	F.	*225†*	*207*	*1087*	*9·5*
			222†	*207*	*1072*	*9·5*
31	76	M.	*252†*	*228*	*1105*	*10·*
			175‡	*166*	*1054*	—
93	76	M.	239	224	1067	10·
			242	226	1071	10·
9	78	F.	230	215	1070	9·25
			—	—	—	—
74	78	F.	*241†*	*220*	*1095*	*9·75*
			244†	*223*	*1094*	*9·75*
Average . .			2938 ÷ 12 = 245	2721 ÷ 12 = 227	1079	86·75 ÷ 9 = 9·64

* Slight nuclear opacity.

† Slight cortical opacities at equator.

‡ Completely cataractous; cortex shrivelled.

TABLE VIII.—*Ages* 80 *to* 89 (*and* 90).

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
73	82	M.	270	245	1102	10·
			283	262	1080	9·75
82	83	F.	*247**	*231*	*1069*	*9·5*
			*245**	*227*	*1079*	*9·5*
95	83	M.	*235**	*213*	*1103*	*9·*
			*234**	*215*	*1088*	*9·*
89	84	F.	*254**	*234*	*1085*	*9·75*
			—	—	—	—
94	86	F.	249	224	1112	9·5
			249	226	1102	9·5
78	87	M.	273	253	1079	10·
			273	253	1079	10·
79	90	M.	*252†*	*237*	*1063*	*9·75*
			254†	*237*	*1072*	*9·75*
62	90	M.	*277**	*263*	*1053*	*10·*
			*277**	*261*	*1061*	*10·*
Average			1597÷6 = 266	1463÷6 = 244	1090	57·75÷6 = 9·62

* Slight cortical opacities at equator.

† Considerable cortical opacities at equator.

METHODS OF PRESERVING AND DRAWING OPHTHALMIC SPECIMENS.*

It is not claimed for the methods here described that they are in all respects the best now available. Of the celloidin method, for example, I have no personal experience. Judging, however, from what I have seen, I believe that the freezing process here described, enables one to bisect the eye with less alteration of its contour, and less disturbance of its internal parts than any other. In the investigations described in this volume, it was all-important to avoid such disturbances.

Preparing and Mounting.—The following are the stages of the process :—

1. The eye is placed immediately after excision, unopened, in Müller's fluid† for at least three weeks, light being carefully excluded. In order to harden the lens satisfactorily, especially in the case of a young eye, six weeks is not at all too long. It is well to change the fluid every two or three days.

† Bichromate of Potash	...		1 part.
Sulphate of Soda	...	...	1 „
Water	...	...	100 parts.

2. It is then wrapped in a piece of thin gutta-percha membrane, the surface of which may be greased to prevent adhesion, and frozen solid by immersion in a vessel containing a mixture of pounded ice and salt. The vessel should have a hole at the bottom, so that water may drain away ; a flower-pot answers well. To freeze an eyeball solid takes at least half-an-hour ; a valuable specimen may be spoiled if cut through before it is solid.

3. When frozen it is divided in the required direction by

* In part reprinted from a paper in the "Ophthalmic Review," Vol. II., p. 69.

means of a sharp table knife. A thicker blade, such as a razor, goes through the frozen globe with difficulty. If the exact position of the section is of consequence the points through which it should pass should be marked with spots of ink before freezing. In spite of this precaution, the section will sometimes fail to pass exactly through some desired point, *e.g.*, the centre of the optic nerve. This is remedied by immediately removing one or more thin slices from the larger frozen hemisphere by means of a sharp scalpel.

4. The bisected specimen is placed in a 3 per cent. solution of chloral-hydrate in order to remove the colour of the Müller's fluid, the solution being changed every two or three days until it is no longer discoloured. (This is a weaker solution than I formerly used, and even this seems to cause some shrinkage in course of time ; a 2 per cent. solution might be better.)

5. It is then placed successively in glycerine solutions, 10 per cent., 25 per cent., and 50 per cent., remaining in each for twenty-four hours or more. These steps are necessary in order to prevent shrinking of the tissues when the specimen is placed in the jelly. The glycerine solutions should contain a small quantity of carbolic acid.

*6. A specimen-jar being partly filled with melted jelly, the half eye is placed in it, the concavity upward. When every interstice is filled it is turned over, care being taken to include no air bubbles. The absence of these can be ascertained by placing a mirror beneath the jar. If the specimen tends to rise in the liquid jelly, it may be kept down in the required position by a pin passed through a strip of card laid across the jar. When the jelly has become firm the jar is placed mouth downwards upon a sheet of white glass.

---

* The first suggestions for preserving specimens of the eye in glycerine jelly were published by Nettleship in 1871 (R. L. O. H. Reports, vol. viii., p. 225).

The jelly is made as follows :—

Best French Gelatine (Coignet and Co., Paris) ...	1 ounce.
Glycerine	8 ounces.
Water	8 „
Solution of Carbolic Acid (10 per cent.)... ...	60 drops.

Soak the gelatine in the water till it swells. Melt it with gentle heat ; add the white of one egg, and boil thoroughly. Filter while hot through flannel. Add the glycerine and the carbolic acid solution.

The glass jars are made by Messrs. Osler, of Broad Street, Birmingham.

When it is desired to mount a ring-section instead of a complete hemisphere, one hemisphere is placed face downwards on the plate of the microtome and frozen to it by the ether spray, and then cut through with a thin knife so as to leave a ring-section of any desired thickness.

One hemisphere being mounted, the other may be kept in the chloral solution, and used at any time for the preparation of microscopic sections. The chloral must, however, be removed by immersing the specimen in water for some hours, if the sections are to be cut by the freezing microtome.

Drawing.—Drawings of the divided globe—*i.e.*, drawings which are intended to represent the shape and relative positions of the various structures, rather than their minute histological appearances—should be made before the specimen is mounted, for a much stronger illumination and better definition can be obtained while the specimen lies in the chloral solution in an open jar than when it is embedded in jelly under glass. The accompanying diagram (Fig. 76) shows an arrangement by which such drawings may be made.

The apparatus consists of a wooden stand, high enough, when placed on the table, to bring the reflector of the microscope about level with the eye ; and having at one end a flap which

can be secured firmly in the vertical position. On the vertical flap is fixed the sheet of paper on which the drawing is to be made. The reflector is a small disc of ordinary looking-glass with a central non-perforating aperture of about 3 mm. diameter, held in a suitable support. It is placed at an angle of 45° over the ocular of the microscope. The eye, looking horizontally, sees, through the central aperture, the paper on which the drawing is to be made; it also sees, reflected from the area around the aperture, the object which is under the microscope. By adjusting the light so that neither image overpowers the

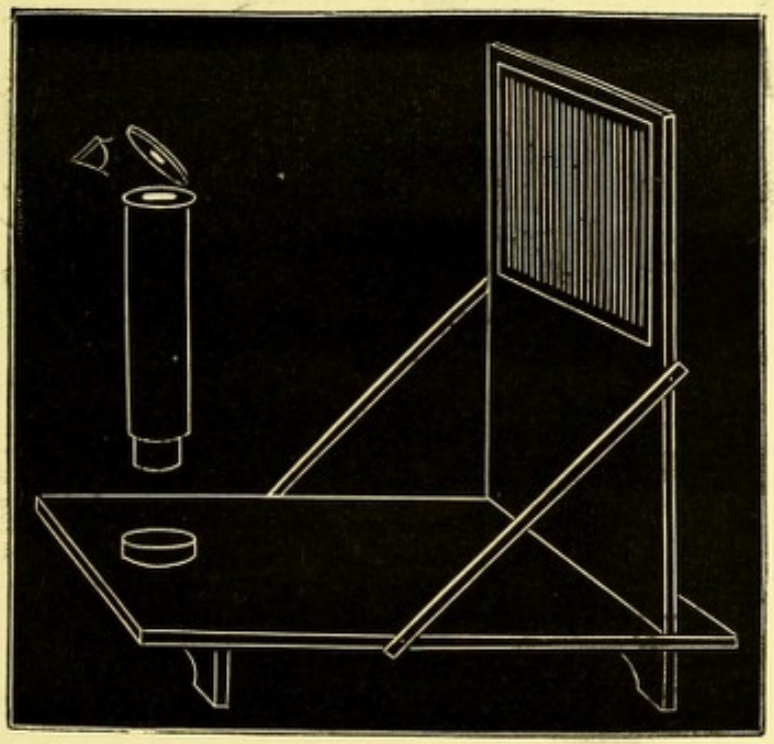


FIG. 76.

other, the picture of the object is seen projected on the paper, and can be readily traced with a pencil. The specimen lies in chloral solution in a glass jar, and in order to secure immobility a small support cut from a piece of thin metal is placed inside the jar; in this the half-eye lies after the fashion of a wash-hand basin in its stand. Its surface is illuminated by means of a condensing lens.

In the case of thin sections, also, it is sometimes well to make a drawing before mounting, for in mounting a section on a microscopic slide it is not always possible to avoid displacement of the several parts. For example, such sections as those shown in Figs. 67 and 68 can hardly be mounted without more or less displacement. The best way to obtain a correct drawing in such cases, is to let the section lie in chloral solution or glycerine and water in an ordinary specimen jar; a cover-glass laid upon it when thus immersed, produces no displacement; the jar can be placed under the microscope and the drawing made in the manner already described.

INDEX.

INDEX.

Glaucoma. Definition How the meaning of the term has changed.
Normal intra ocular pressure. How measured
How maintained.
Origin of intra ocular fluids — 2 vascular systems retinal + uveal. — post pituitary cornt a complete blocking by embolism does not effect T. Bowman
Symbols: Uvea. — Choroid — nutrition of retina — not vitreous
Ciliary body
(Iris — optical function — accomodation
a. Especially adapted
(a) most vascular part of the eye
(2) convoluted for increased surface
(3) Vitreous here most adherent
(4) Membranous septa peculiarly arranged
5 Collins glands
6. Pathological anatomy
Vitreous inept earliest stage
Shrunken vitreous
Disease of ciliary B + choroid
these result in vitreous disease
(6). Experimental evidence
(a) Removal of ciliary body in rabbit
(b) Injection of fluorescein
(c) " " Pot Iod
Normal currents — filtration angle — optic papilla.