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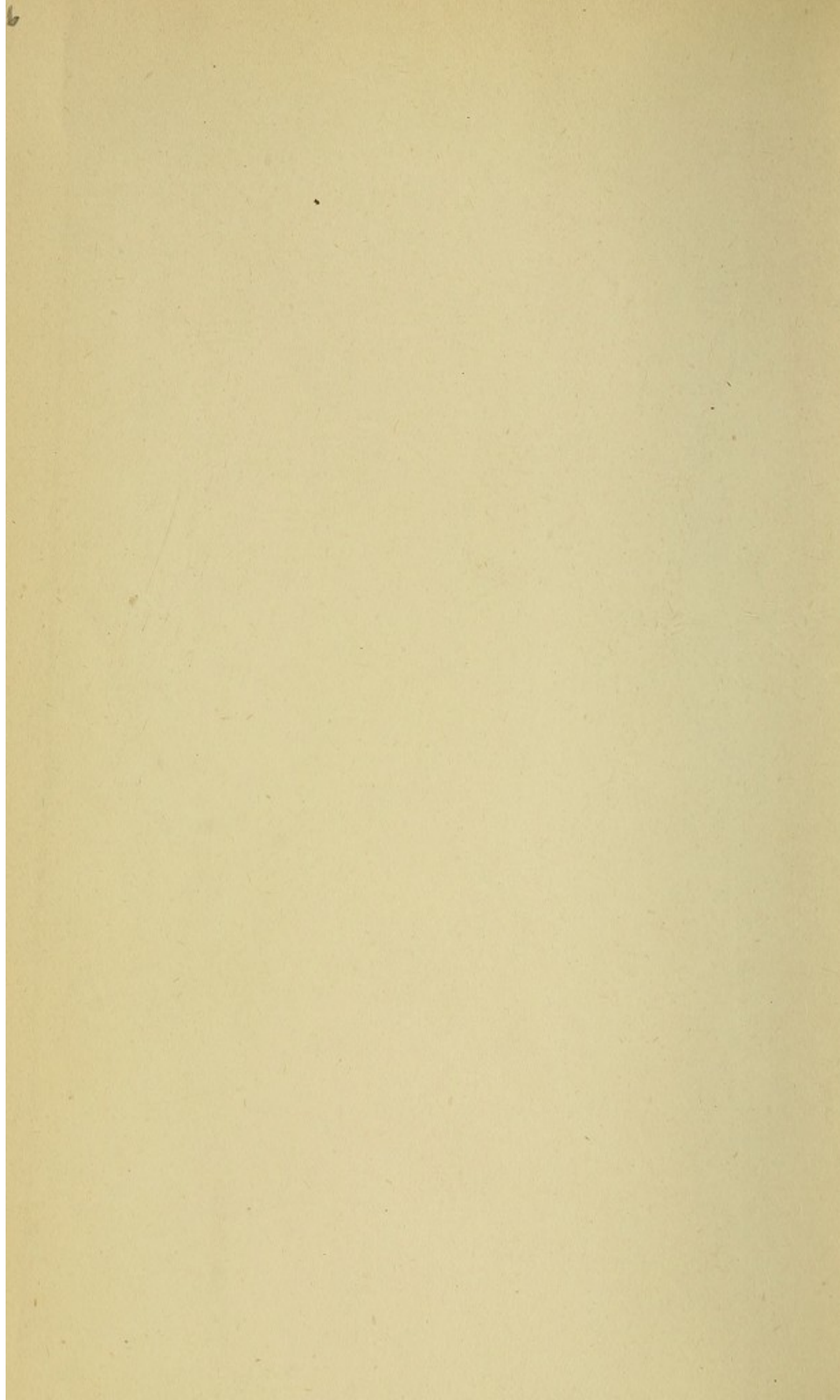
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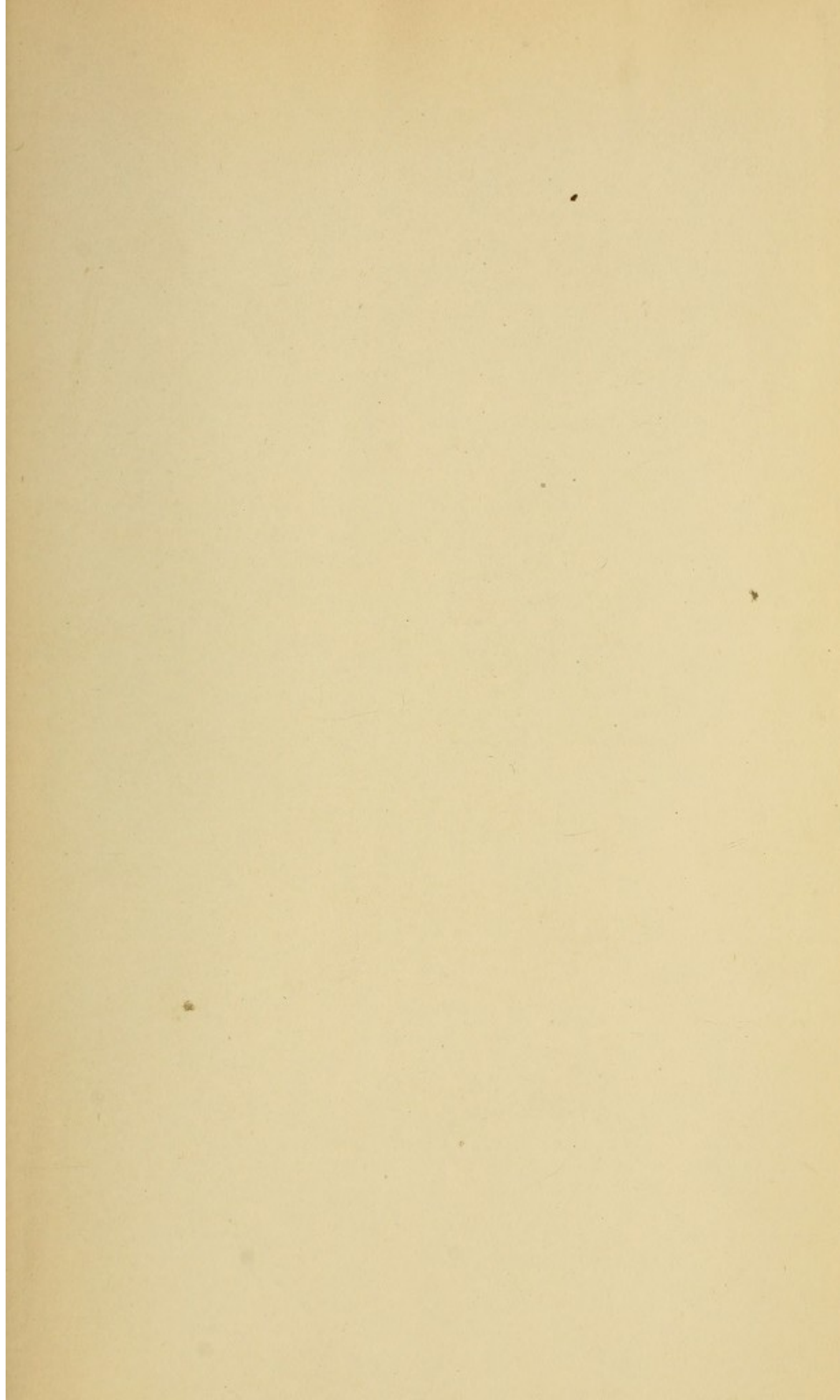
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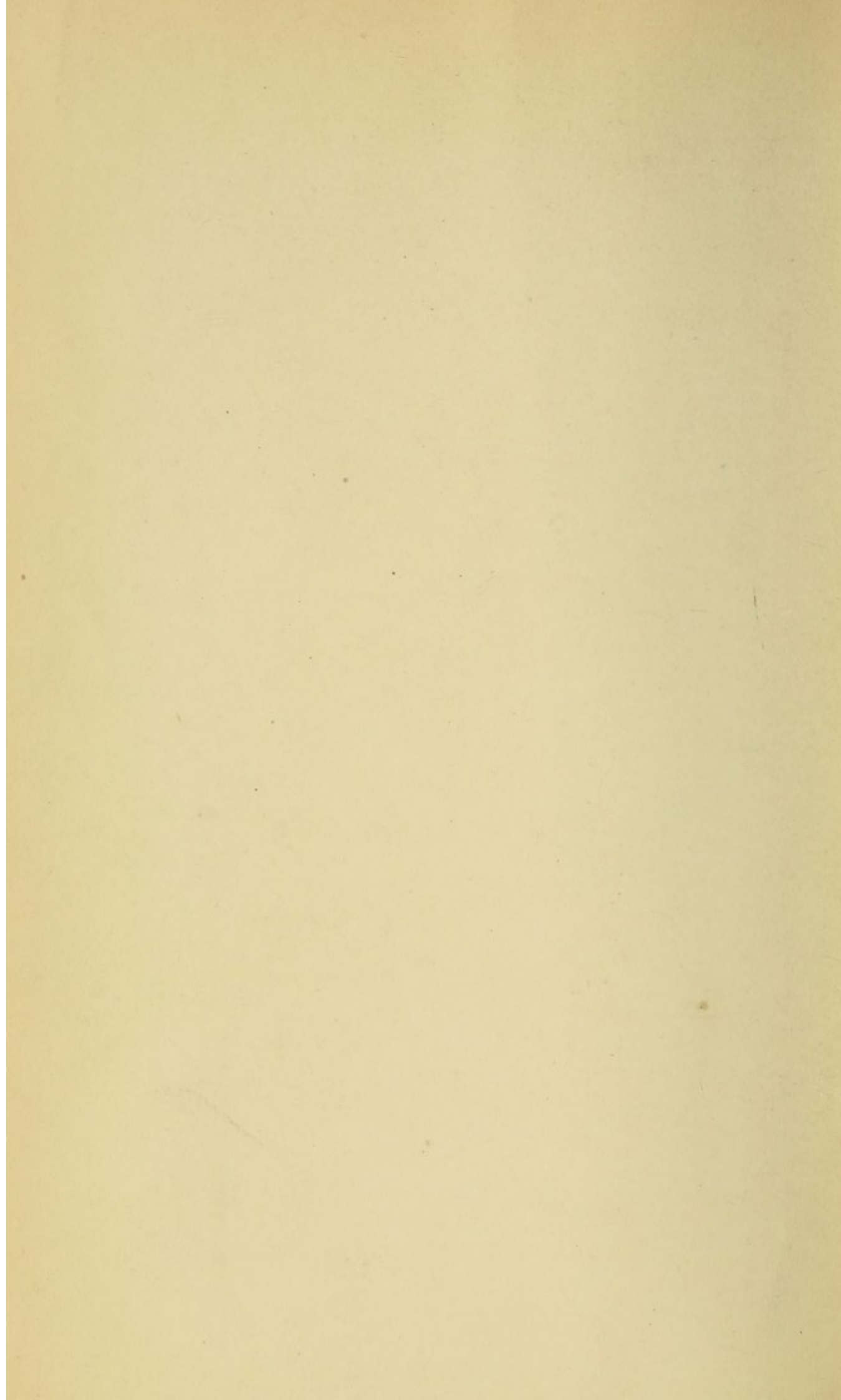
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GLAUCOMA.

GLAUCOMA:

ITS CAUSES,

SYMPTOMS, PATHOLOGY, AND TREATMENT.

BY

PRIESTLEY SMITH,

OPHTHALMIC SURGEON

TO THE QUEEN'S HOSPITAL, BIRMINGHAM.

WITH LITHOGRAPHIC PLATES.

LONDON: ✓

J. & A. CHURCHILL, NEW BURLINGTON STREET.

—
1879.



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PREFACE.

This work presents, in its original form, the dissertation on glaucoma to which the Jacksonian prize for 1878 was awarded by the Council of the Royal College of Surgeons in April of the present year. A few notes bearing a later date have been added, a few superfluous passages have been struck out, verbal corrections, where necessary, have been made, and references to persons and places inadmissible in the anonymous manuscript have been inserted. In other respects the original essay stands unaltered.

Written as it was within a strictly limited period of time, it is, of necessity, an incomplete exposition of the wide subject with which it attempts to deal. I have decided, nevertheless, to lay it before the profession as it is, rather than to incur the delay which an attempt to remedy its defects would involve. My reason for so doing is, that in studying the pathology of glaucoma—a subject which at the present time is exciting much interest both at home and abroad—I have been led, by experiment and otherwise, to certain conclusions concerning the origin of the disease, which I am anxious to submit

without delay to the scrutiny of other workers in the same field.

Among the many works to which I am indebted for help in the preparation of this essay I may mention especially the Royal London Ophthalmic Hospital Reports, v. Graefe's Archiv für Ophthalmologie, and the Handbuch der gesammten Augenheilkunde, edited by Graefe and Saemisch. The latter in particular, consisting as it does of a series of recent monographs in every branch of ophthalmic science, together with a complete index of ophthalmic literature, rendered me invaluable aid.

I desire also to express my indebtedness and cordial thanks to several friends: to Mr. Solomon, and to Mr. Hodges, of Leicester, for the privilege of recording certain points of interest in specimens placed by them in my hands for examination; to Mr. Eales, for most kindly giving to me, for the purposes of this essay, two valuable specimens acquired in the course of his practice at the Birmingham and Midland Eye Hospital; to Mr. Charles Sims and his Assistants at the Dental Hospital, for the opportunity of making a series of observations in that institution; and to the Resident Medical Officers and Students of the Queen's Hospital, for voluntary help rendered to me

on many occasions in making investigations with the tonometer.

The Plates with which the work is illustrated are faithful lithographic copies, by Messrs. West, Newman, & Co., of Hatton Garden, of my own original drawings, which, together with the mounted preparations and microscopic slides which accompanied the original essay, are now in the possession of the College of Surgeons.

Birmingham, August, 1879.

TABLE OF CONTENTS.

INTRODUCTION.

SECTION I.—ETIOLOGY.

PRIMARY GLAUCOMA.

	PAGE
Frequency of occurrence	1
The sexes equally affected	3
A malady of advanced life	4
Usually bilateral	5
The chronic more common than the acute ...	5
Acute attacks more common in women ...	5
The influence of heredity	6
Commonly associated with hypermetropia ...	7
Trigeminal neuralgia—evidence of its association with glaucoma—irritation of fifth nerve in animals—H. Schmidt's investigation as to range of accommodation in cases of toothache— tonometrical measurements in cases of toothache	8
Other nerve disorders	15
Gout	16
The climacteric change... ..	16
Induction of glaucoma by atropine	16

SECONDARY GLAUCOMA.

Iritis and synechia	19
Injury and dislocation of lens	20

	PAGE
Foreign bodies	22
Sympathetic inflammation	22
Intraocular tumours	22
Myopia	22
Retinal hæmorrhage	23
Glaucoma occasionally associated with retinitis pigmentosa, retinitis albuminurica, primary atrophy of optic nerve, detachment of retina, aphakia ...	24

SECTION II.—ESTIMATION OF TENSION.

General principles	27
Bowman's symbols	28
Tonometry—sources of error—variations in elasticity of sclera, size of eye, form of eye, curvature of sclera at point of impression	29
Relative accuracy of different methods—experiments...	33
A new tonometer	38
Action of the tonometer	40
Mode of application	42
Comparison of this with other methods	44
Table of trial measurements	47
Note—further experiments—relation of tonometrical measurements to internal pressure	48

SECTION III.—SYMPTOMS.

GENERAL DESCRIPTION.

*Primary glaucoma	52
Chronic glaucoma	53
Sub-acute glaucoma	56
Acute glaucoma	57
Hæmorrhagic glaucoma... ..	59
Absolute glaucoma	60
Secondary glaucoma	64

ANALYSIS OF SYMPTOMS.

	PAGE
Apparatus for experiment	69
Diminution of accommodation—experiment ...	70
Changes of refraction	72
Iridescent vision—experiment	74
Corneal opacity	76
Corneal anæsthesia	78
Dilatation of pupil	80
Dilatation of anterior ciliary arteries and veins ...	82
Pulsation of vessels of optic disc	82
Contraction of field of vision—experiment ...	87
Cupping of optic disc	90

SECTION IV.—PATHOLOGY.—PART I.

Outline of its history up to present time	93
The intraocular pressure	97
Its normal height—experiments	97
Its relation to blood pressure	100
Influence of fifth nerve	103
Influence of sympathetic nerve	105
Not affected by accommodation	105
Equal in aqueous and vitreous chambers— opposite opinion of Weber examined— experimental evidence	106
Influence of atropine and eserine ..	110
Aqueous humour secreted by ciliary processes ...	112
Aqueous humour escapes at angle of anterior chamber —Leber's experiments	113
Angle of anterior chamber closed in glaucomatous eyes	116
How is this closure produced?—theories of Max Knies and Adolph Weber	118
Experiments—description of apparatus	123

	PAGE
<i>Slight excess of pressure in vitreous chamber closes angle of anterior chamber</i>	128
<i>Glaucoma is due to some condition which exalts vitreous pressure above aqueous pressure</i>	135
Nutrient supply to vitreous humour proceeds from ciliary processes... ..	137
And escapes by passing through suspensory ligament into aqueous chamber—experiments ...	141
Summary of the foregoing	147

SECTION V.—PATHOLOGY.—PART II.

PRIMARY GLAUCOMA.

Glaucoma usually associated with commencing senility	150
Senile changes in lens capable of raising vitreous pressure—evidence in support of this theory, viz., hypermetropic structure, action of atropine and eserine, rapid onset of presbyopia ...	152
Vascular changes—dilatation of anterior ciliary veins—swelling of ciliary processes	156
Theoretical explanation of glaucoma— <i>narrowing of the "circumferential space"</i>	158
Acute glaucoma—sudden change in lens?—sudden swelling of ciliary processes—arterial hyperæmia—neuralgia in relation to acute attacks ...	159
Ultimate stagnation of the intraocular fluid ...	163
Influence of iridectomy	164
In acute glaucoma	167
In chronic glaucoma	168
" Malignant " glaucoma due to excessive diameter of lens—observations of Adolph Weber and H. Pagenstecher	169
Method of cure practised by Weber	172

SECONDARY GLAUCOMA.

	PAGE
Associated with posterior synechia	175
" " anterior synechia	177
" " cataract extraction	178
" " buphthalmos	180
" " staphyloma of cornea	181
" " traumatic cataract	181
" " dislocation of lens	183
" " tumours of retina and choroid ...	185
" " intraocular hæmorrhage	187
Objections to the obstruction theory of glaucoma examined	189
Closure of angle of anterior chamber does not cause glaucoma under all circumstances	191
If secretion of intraocular fluid be suppressed	192
If it find an abnormal outlet	194
If closure be of limited extent	195

SECTION VI.—TREATMENT.

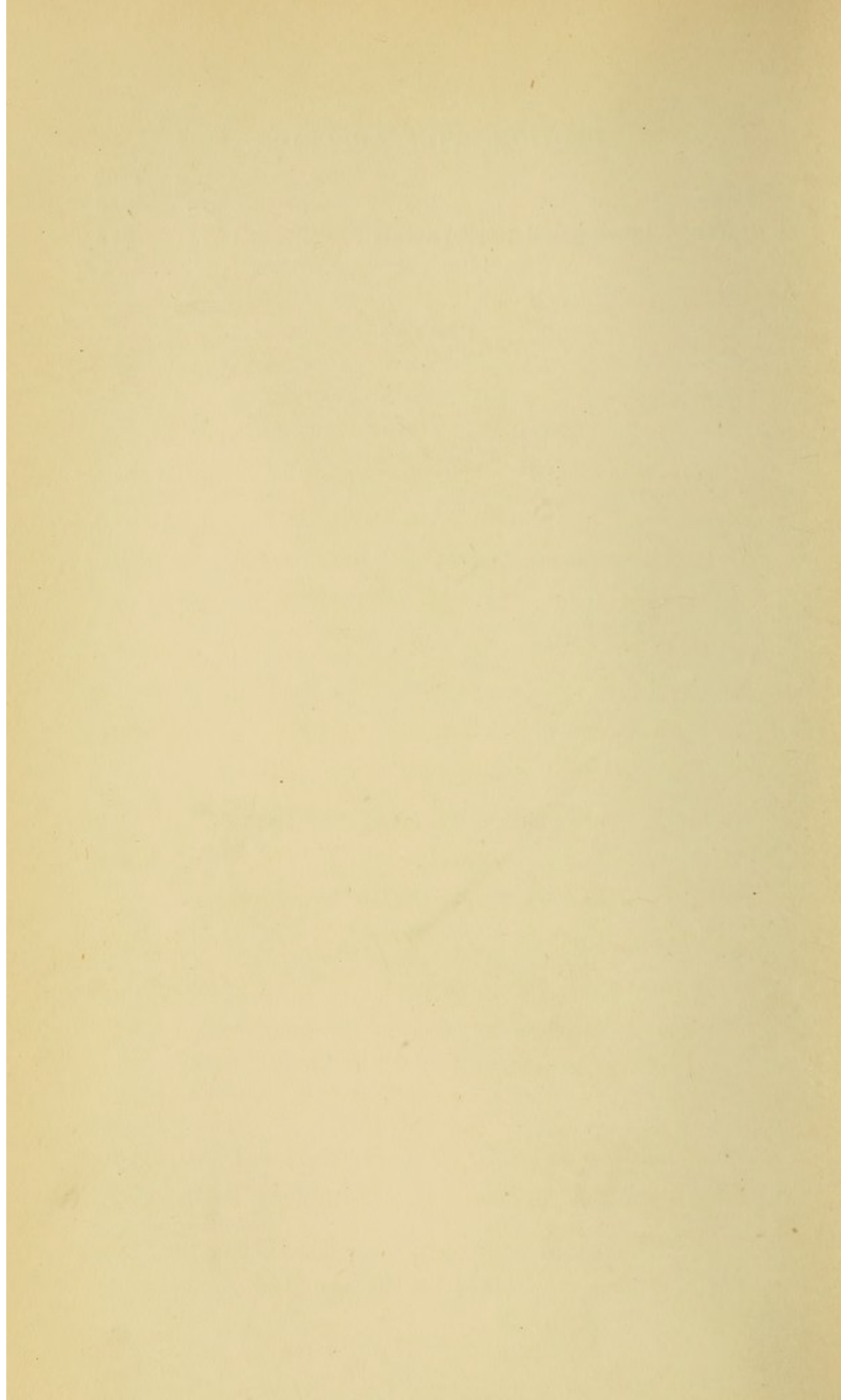
Iridectomy	197
Degree of improvement obtainable	198
Urgency in acute glaucoma	200
Less beneficial in chronic glaucoma	201
Mode of performance	203
After treatment, progress, accidents	207
Malignant course after iridectomy	212
Outbreak in fellow eye	213
Sclerotomy	215
Other substitutes for iridectomy	220
Eserine and other myotics	222
General treatment	228
Note—theoretical considerations as to treatment ...	230
Note—anæsthesia by chloral and ether	231

SECTION VII.—APPENDIX.

	PAGE
Cases 	233
Table of tonometrical measurements 	276

PLATES.





INTRODUCTION.

The nomenclature and the general arrangement adopted in the following pages require a few words of explanation.

The commonly adopted division of glaucomatous diseases into a primary and a secondary group is here retained. Recent discoveries have tended, in some measure, to show that this division is arbitrary, and, on scientific grounds, untenable; that primary glaucoma, if the term indicate a condition in which a rise of the intraocular pressure is antecedent to any structural change, does not exist, and that secondary glaucoma includes conditions the etiology of which entitles them to be classed with the primary. Granting that this is true, I believe that, practically, the division is both simple and useful; and, further, I believe that, with a few slight exceptions, it classifies glaucomatous diseases in strict accordance with fundamental differences in their causes.

I include, under the term glaucoma, or glaucomatous, every condition of the eye characterised by an excess of tension. It is said that not every excess of tension is glaucoma, but it appears to me that to apply or

to withhold the name in accordance with the presence or absence of certain of the secondary symptoms is only to create confusion as to the nature of the essential change. From a clinical point of view, the importance of an excess of tension is doubtless exactly proportionate to its duration and amount; but, to the pathologist, it is the *causes* which are of chief moment, and these must be studied no less when temporary than when persistent.

At the foot of each page are given references to the sources of the information set forth, or to the other parts of the essay where the subject in question is dealt with more fully. I have referred only to those writings to which I myself have had direct access. In many of these, and especially in the article on Glaucoma by Hermann Schmidt in the *Handbuch* of Graefe and Saemisch, very much fuller references to the original papers may be found.

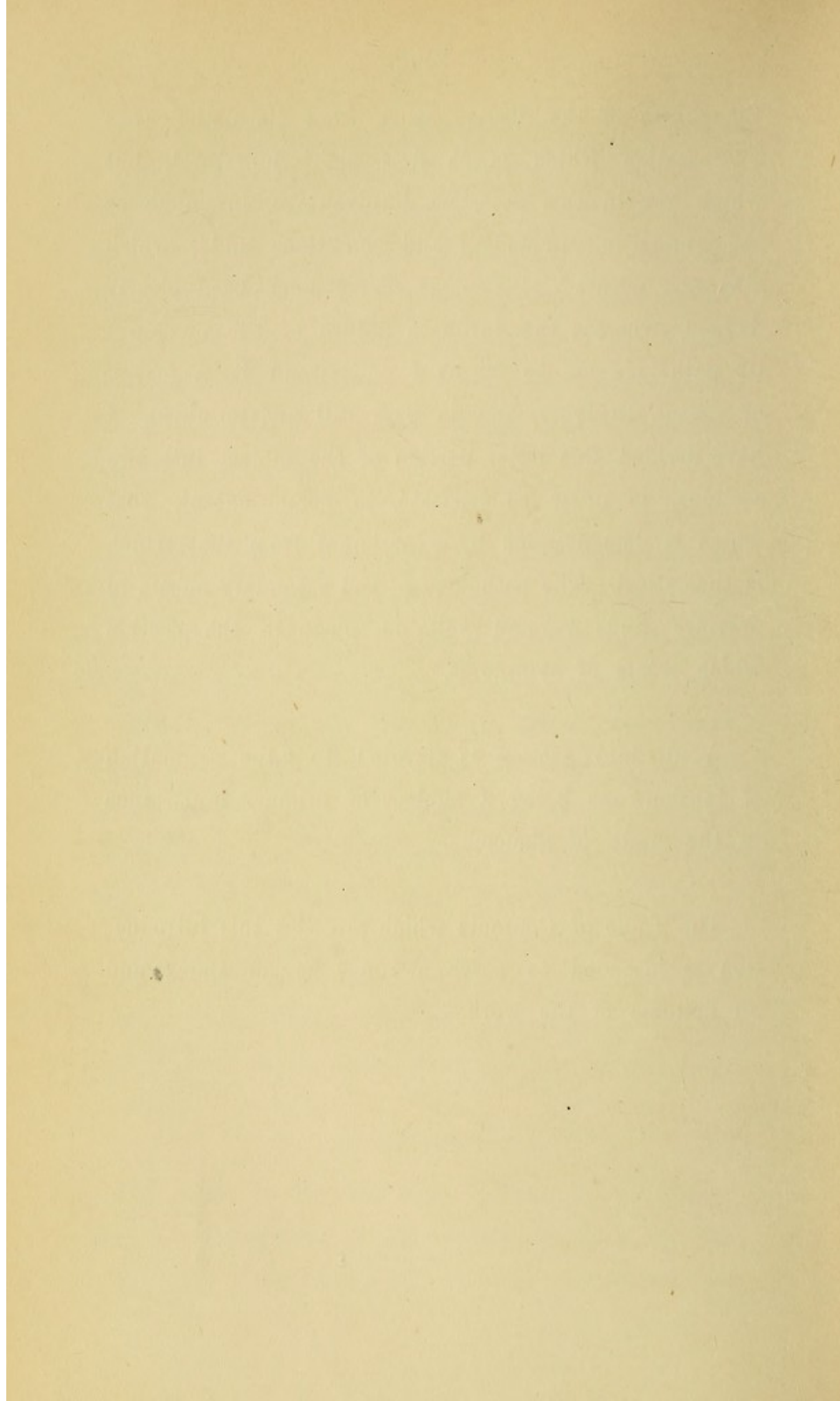
In recording conditions of refraction and the power of lenses, I have employed the system of inches, as being that on which nearly all my clinical records have been made.

The work is divided into sections, in accordance with the arrangement of the subject prescribed by the College of Surgeons. This involves a discussion of

the causes of the disease apart from its pathology—a separation which is at first sight unnatural, but which has, in this case, no disadvantage, inasmuch as the general circumstances and conditions under which glaucoma occurs—its etiology—have long been clearly defined, whereas the intimate nature of the process—its pathology—is, so far as I understand it, a matter of recent discovery, and in part still undetermined. I have divided this latter portion of the subject into two sections, in order to separate, to some extent, that which is demonstrable by experiment from that which is theoretical. The pathology of the secondary effects of pressure finds a more suitable place in the section which treats of symptoms.

A distinct section is devoted to “the estimation of tension,” as being a subject of primary importance in the study of glaucoma.

The Table of Contents which precedes this introduction is intended to serve at once as an index, and an epitome of the work.



SECTION I.

ETIOLOGY OF GLAUCOMA.

Primary glaucoma, though not rare, is one of the less common maladies of the eye. It appears to constitute a little less than one per cent. of all ophthalmic diseases. Secondary glaucoma, if we include under this term all cases of disease of the eye which are complicated by excess of tension, is met with much more frequently. Exact statistics concerning the prevalence either of glaucomatous diseases in general, or of the two classes separately, are hardly to be obtained. My own hospital case books for the last four years show that primary glaucoma occurs once in a little over one hundred cases, but the total numbers are not large enough to justify a general conclusion. The published statistics of our ophthalmic institutions—which alone furnish figures of sufficient magnitude—do not distinguish between the primary and secondary forms, and, moreover, are vitiated, in some cases, by the separate tabulation of diseases of in-patients and out-patients, whereby cases of more than ordinary gravity, like glaucoma, are frequently registered twice over, and *more frequently* so than cases of a class which seldom demands admission. The great majority of the cases registered as glaucoma are doubtless primary, while secondary glaucoma is necessarily scattered under many headings—*e.g.*, iritis, synechia, traumatic cataract, intraocular tumour, &c., &c.

The following table exhibits the registry of glaucoma in five of the chief ophthalmic hospitals in this country, as contained in their published statistics:—

Institution.	Years.	Percentage of Glaucoma.	Equivalent. Proportion.
Royal London Ophthalmic Hospital ...	{ 1876 1877 }	{ .64 ... }	1 in 156
Birmingham and Midland Eye Hospital	{ 1876 1877 }	{ .59 ... }	1 in 169
Manchester Royal Eye Hospital	{ 1876 1877 }	{ 1.08 ... }	1 in 93
Liverpool Eye and Ear Infirmary (Eye cases only)	{ 1876 1877 }	{ .37 ... }	1 in 270
Kent county Ophthalmic Hospital ...	{ 1863 to 1877 inclusive }	{ .50 ... — }	1 in 200
Average	...	.63 ...	1 in 158

The average is .63 per cent., or one case of glaucoma in 158 ophthalmic cases of all kinds.

Statistics from abroad show a higher proportion than this.

The following figures, quoted from H. Schmidt, show the percentages derived from very large numbers of cases treated in three continental cities. ⁽¹⁾

Berlin	...	...	1.27 per cent.	...	1 in 78
Wiesbaden	...	...	1.48	„	1 in 68
Vienna	...	...	1.26	„	1 in 79

The percentage derived from the previous table is, very probably, somewhat too low. Especially in the case of simple chronic glaucoma, in an early stage, is a very searching examination, and frequently more than one, necessary to decide with certainty the nature of the malady. Where registration of the cases is made previous to such complete examination, one of the leading symptoms—*e.g.*, presbyopia, hypermetropia—is likely to be entered as the essential disorder; an oversight which, where entries are very numerous, is not likely to be corrected when subsequent

¹ Handbuch der gesammten Augenheilkunde Band, v. 1., 61.

examination defines the presence of glaucoma. On the whole, I think we may assume that the percentage originally stated—viz., nearly one per cent.—is approximately correct.

The prevalence of the disease differs somewhat, probably, in different localities ; ⁽¹⁾ but, for this country at least, we have no statistics which give certain evidence of the fact.

Secondary glaucoma is certainly met with among the working population of large towns in larger proportion than the primary disease. Its frequency among this class is due, in great part, to mechanical injuries, and to neglected iritis. It is, of necessity, so frequently registered under other names, that statistics are hardly to be obtained.

Primary glaucoma exhibits no decided preference for either sex. It has been asserted that women are more liable to the disorder than men, but there is no satisfactory proof of the statement. The following list, from which I exclude the figures of Laqueur, which refer to both forms of the disease, is given by Schmidt, with full reference to the sources whence it is derived :— ⁽²⁾

			Males.		Females.
H. Schmidt	...	...	154	...	137
Rydel	...	...	46	...	33
Vienna Hospital, 1869			32	...	27
v. Wecker	...	...	50	...	42
Arlt	...	...	45	...	65
Donders	...	...	39	...	56
Förster...	...	...	39	...	54
Totals	...	...	405		414

Having regard to the not very large number of cases tabulated, and to the preponderance of females in the adult population, it cannot be allowed that these figures establish any marked difference in the liability of the sexes.

¹ v. Wecker, *Therapeutique Oculaire*, p. 374. ² *Handbuch d. g. A.*, v. 1, 62.

Secondary glaucoma, on the other hand, is, in my experience, more commonly met with in men than in women, which is doubtless attributable to the greater exposure of the former to injuries. Laqueur gives the proportion for both forms together as three males to two females. Contrasting this with the figures already given, it is probable that the preponderance of males is here due to the inclusion of secondary glaucoma.

Primary glaucoma is pre-eminently a disease of advanced life. About seventy per cent. of the cases met with are in persons over fifty years of age. ⁽¹⁾ Below the age of forty, primary glaucoma is a rare disease. The youngest person in whom I have seen it was a woman aged thirty-one, and in that case the symptoms were only premonitory until the use of atropine induced an acute attack. ⁽²⁾ Statements of its occurrence in early life can only be received on high authority, and with the express exclusion of any confusion between primary and secondary glaucoma. Critchett records one such case in a man aged twenty-five. ⁽³⁾ The media and corneæ were perfectly clear. The discs were deeply cupped. Complete blindness was reached in six months from the commencement of the symptoms. There had been no pain in the eyes themselves. Both were very hard, but not congested. Cases of the disease at the ages of nine and twelve are on record. ⁽⁴⁾

Secondary glaucoma, on the other hand, is not confined to any period of life. Most of the maladies which induce it are less common in advanced than in middle life, and since buphthalmos is essentially a secondary glaucoma, modified only by extensibility of the sclera, childhood is not by any means exempt.

¹ Handbuch d. g. A., v. 1., 63 (deduced from table of percentages).

² Case 19, Section VII. ³ R. L. O. H. Reports, V. 224.

⁴ Handbuch d. g. A., v. 1., 63.

In the large majority of cases primary glaucoma is a bilateral affection. The disease may progress equally in the two eyes from first to last, but more commonly one is persistently in advance of the other. The interval which may elapse between the first manifestation of the disease in the two eyes respectively is various, but we very rarely see a case in which one eye is in an advanced state of chronic glaucoma, as evidenced by deep excavation of the disc and contraction of the visual field, while the other is entirely free from signs of the commencement of the malady.

The more purely chronic and non-irritative, the more likely is glaucoma to be bilateral. In the section on pathology I shall attempt to prove that the cause of glaucoma in its simplest form is a physiological change which presumably affects both eyes alike, while the exciting causes of acute attacks are various, and are capable of influencing either eye alone, and may operate in eyes which previously are not glaucomatous, provided the changes which predispose to glaucoma are already present. Hence the bilateral character of chronic glaucoma, and the unilateral acute attacks.

The chronic is more common than the acute, probably in the proportion of three to one, at least. The absence of exact demarcation between the types, the conversion of one into the other, and the coexistence of the two in the same individuals, render a precise numerical statement of this point unattainable.

Though the two sexes show an equal liability to glaucoma, *yet women suffer the larger proportion of acute attacks.* ⁽¹⁾ This is due, most probably, in part to the vascular congestions so common at the climacteric period of life, in part to the greater sensitiveness and excitability of

¹ Handbuch d. g. A., v. 1, 64.

the nervous system, and possibly to the habitual constipation from which many women suffer. An acute attack in a woman aged sixty-eight, under my own care, was associated with a five days' accumulation of hardened fæces in the large intestine. All causes which produce congestion of the vessels of the head may, under predisposing conditions, induce an acute attack.

It has been asserted, by more than one writer, that primary glaucoma occurs, in the majority of cases, in persons with dark eyes. Granting this to be true, the fact has no significance unless accompanied by statistics as to the relative frequency of dark and light eyes in the particular population which furnished the cases. Should such statistics be found to actually prove that dark eyes are more liable than others to glaucoma, the fact may possibly depend upon the apparent especial liability of Jews as a race.

The influence of heredity has been distinctly traced in a considerable number of instances: thus, by Arlt in a mother and two daughters, and in a father and three sons; by Pagenstecher in a mother and three sons, and in a father and son; and by v. Graefe in one family through three, if not four, generations. ⁽¹⁾ The opportunities for fairly studying the question of heredity are necessarily infrequent, for the patients who form the starting point of enquiry are generally elderly people, and can give no very accurate account of any maladies of sight from which their parents may have suffered. I have asked the question many times, but have obtained nothing but doubtful or negative replies.

Within a stone's throw of the Hospital have lived for some years past an elderly couple, both totally blind from absolute primary glaucoma. They have three children living, aged from twenty-five to forty-four; amongst these no imperfection of sight has yet manifested itself. ⁽²⁾

¹ Handbuch d. g. A., v. 1, 65. ² Cases 22, 23, Section VII.

The theory of glaucoma which I advance in the section on pathology points to peculiarities in the structural conditions of the eye as the cause of glaucoma, especially as regards the size of the lens and the shape of the ciliary muscle. It is certain that such peculiarities, at least as regards the hypermetropic structure, are frequently hereditary.

The foregoing paragraphs treat only of the general circumstances and conditions under which glaucoma occurs. Certain conditions, which appear to be more or less concerned in the *causation* of the disease, must now be noticed. Here it is important to separate, as clearly as possible, the primary from the secondary form. The special associations of secondary glaucoma will, therefore, be noticed later by themselves.

Hypermetropia is present in the majority of cases of primary glaucoma—viz., from fifty to seventy-five per cent., according to different observers. ⁽¹⁾ It is important to ascertain whether it is a result of the excessive pressure, or whether the hypermetropic structure predisposes to glaucoma.

It must be remembered that the tendency of the emmetropic eye at the age of fifty or thereabout is towards hypermetropia; ⁽²⁾ any comparison, therefore, of the relative frequency of hypermetropia in glaucomatous persons and in the general population is fallacious, unless only such degrees are included as cannot be due to senile changes in the lens; otherwise the frequency of hypermetropia in the general population must be estimated by the examination of those persons only who have passed middle life. Seeing however that the effect of glaucomatous pressure upon the refraction of the eye is perhaps as often in the direction of myopia as in that of hypermetropia, and seeing also that high degrees of hypermetropia are often associated with glaucoma, and that

¹ Handbuch d. g. A., v. 1, 4.

² Donders. Accommodation and Refraction (N. Sydenham S.), p. 208.

their pre-existence may be established by the fact that spectacles have been worn at an unusually early age, we may, I think, conclude *a priori*, that the hypermetropic eye is somewhat more liable to the malady than the emmetropic or myopic.

The theory by which I have sought to explain the primary nature of the malady points to the peculiar shape of the hypermetropic ciliary muscle as a predisposing cause. Ad. Weber lays stress upon this same point, but accounts for its effects in a somewhat different way. The reason of the susceptibility of the hypermetropic eye has been supposed to lie in the greater thickness of its sclera, by reason of which it yields with difficulty to an excess of pressure. I will not deny this connection entirely, but I believe that differences of elasticity in the sclera have very little to do with *causing* glaucoma, for we witness the malady in its secondary form in the highly elastic eyes of children. Their effect is seen rather in the *results* of the malady. The youthful elastic eye enlarges to an excessive size under a pressure which cannot extend the more rigid sclera of the adult; the pressure is equal in both, however, and produces results equally disastrous as regards the function of the retina. Another particular in which the hypermetropic eye probably offers a predisposition to glaucoma is the shallowness of the anterior chamber. "A more anterior position of the iris and crystalline lens is part of the peculiarity of the hypermetropic structure." ⁽¹⁾ On this point, however, I lay less stress, than upon the excessive thickness and prominence of the ciliary muscle. ⁽²⁾

Trigeminal neuralgia is a precursor of glaucoma in some cases, occasionally over periods of many years. The question of a causal relationship between the two is of much importance as a point of evidence in the nerve-theory of

¹ Donders, *Op. Cit.*, p. 246. ² See Section V., p. 152.

glaucoma. I believe that trigeminal neuralgia excites glaucoma where the predisposing conditions (to be described later) have already brought the eye to a point of danger, but I do not accept this connection as evidence that glaucoma is the expression of a *persistent* hypersecretion due to perverted nerve influence. The pathology of this question is dealt with in another place. Here I shall deal only with the observations which tend to show that some connection exists. They are of several kinds.

First, we have a series of cases in which the outbreak of glaucoma has been preceded by a neuralgic affectation. Hutchinson records a case in which severe pain in the left cheek, left malar bone, and left forehead occurred very frequently for seven years, and at the end of that time an attack similar in all respects to the previous ones ushered in the outbreak of glaucoma. ⁽¹⁾ With regard to this particular case, and others like it, there can be little doubt that the nerve disorder was distinctly antecedent to the glaucoma; but caution is necessary in this class of cases, lest the effect be mistaken for the cause. A slight excess of pressure suffices, in some persons, to produce considerable pain, and, provided the excess be transient, the eye may remain long free from permanent symptoms of glaucoma. Again, if we assume that the glaucoma is caused by the neuralgia, how is it that the neuralgia entirely subsides, as it sometimes does (*e.g.* Hutchinson's case), directly after the cure of the glaucoma by iridectomy?

Secondly, we know that artificial irritation of the fifth nerve in animals may induce excessive increase of the intra-ocular pressure, although, be it observed, a persistent glaucoma has never been thus induced. ⁽²⁾

¹ R. L. O. H. Reports, IV., 127.

² Section IV., p. 103.

Thirdly, we have, clinically, in dental caries a condition of severe peripheral irritation of the fifth nerve, which has been proved to produce morbid changes in the eye. I have several times detected symptoms of asthenopia in connection with the pain of diseased teeth. Quite recently I have had under my care a young man, the near point of whose left eye was at eight inches, that of the right at five inches. The difference arose during an attack of pain in the teeth, with swelling of the cheek of the left side, brought on by cold, and disappeared completely five days after the subsidence of the pain.

Hermann Schmidt has investigated the point very fully. He accurately measured the range of accommodation, in each eye separately, in ninety-two cases of toothache. ⁽¹⁾ In seventy-three of these cases he found the range too small, in some, much too small in one eye or in both, in proportion to the age of the patient; and in many cases he was able to observe an increase in the range a few days after the cessation of the pain. He states his conclusions as follows:—

1. "Pathological irritation of the dental branches of the trigeminus causes limitation of greater or lesser degree in the range of accommodation."
2. "These limitations may be either unilateral or bilateral; in the former case they occur invariably on the side affected by pain."
3. "These accommodative changes are most common in youth; in advanced life they are rarely, if ever, observed."
4. "They are due to increase of the intraocular pressure."

The fourth conclusion is not proved by the investigation, and must be treated as an assumption. In its favour, however, is the fact that similar limitations of accommoda-

¹ Arch. f. Ophth., XIV., 1, 107.

tion constantly accompany the onset of glaucoma ; and the additional fact, proved experimentally by myself, that they occur when the tension of the healthy eye is artificially raised. ⁽¹⁾ On the other hand, the theory of glaucoma which I have to advance, is intimately dependent upon alterations in the lens in advanced life. Now, the asthenopic symptoms produced by neuralgia may not improbably depend upon nerve-paresis. In fact, we witness total paralysis of accommodation apparently depending upon peripheral irritation of nerves in exceptional cases. From this it appears possible that the limitations of accommodation observed by Schmidt may be quite unconnected with changes of tension, and yet that they may occasionally bear a causal relationship to glaucomatous attacks.

I have endeavoured further to elucidate this matter by tonometrical measurements in a series of cases of toothache at the Dental Hospital. The results obtained give no very decided evidence either way, but, at least, they show that in young persons toothache rarely, if ever, causes a decided rise of tension. I estimated the tension—in each case from a series of measurements—in sixteen persons suffering at the moment or recently from severe pain in the teeth. The position of the near point was ascertained by noting the distance from the eye at which a group of fine lines on white paper lost their definition. It would have been more satisfactory to have examined the eyes, in every case, during the continuance of severe pain ; but, apart from the apparent cruelty of what appears to the patient a needless examination at such a time, it would, I think, be extremely difficult to accomplish, for, over and over again, I was told by patients that the toothache, previously so violent, had vanished on approaching the dentist's chair—a result, I presume, of nervous apprehension.

¹ Section III., p. 70.

Remembering the investigations of H. Schmidt, I was surprised to find no anomaly of accommodation in fifteen out of the sixteen cases. It should, however, be observed that I did not ascertain the state of refraction present, so that it is possible that the presence of myopia may have masked a retrocession of the near point in some cases.

Putting on one side such differences as might be attributed to error in the method, we find a distinct difference of tension in the two eyes in six cases out of the sixteen—(I neglect, for the present, numbers 8 and 17, which were re-measurements)—namely, in numbers 4, 6, 10, 11, 14, 16. But in three of these the eye of the painful side was the *harder* of the two; in three it was the *softer*. This is not to be explained by a previous cessation as opposed to a continuance of the pain at the time of testing, for, of two cases in which the pain continued, the corresponding eye was harder in one, softer in the other. The differences observed are not greater than are occasionally met with in the case of healthy pairs of eyes, and the tension was almost without exception within normal limits. ⁽¹⁾ In case 13 the eye of the painful side overstepped the normal limit slightly in one direction; in case 4, in the other. On taking a general average of all the cases, including the two remeasurements, the tension on the two sides is exactly equal, and is, moreover, of normal medium height. The two cases in which remeasurements were made with closely corresponding result tend to show that the differences discovered actually existed, and were probably proper to the eyes rather than a result of temporary disorder.

Case 16 deserves special notice, as it was more thoroughly examined than the others. It was the only one which presented a sub-normal range of accommodation. The refraction in each eye was exactly emmetropic; the near

¹ See Section VII., p. 276.

point of each was exactly at ten inches distance, a distinct indication of a different tension was given by the tonometer, the eye on the painful side being the harder of the two, but being of perfectly normal tension, while the other was a little below the normal medium. Re-examined on all points, three days later, the conditions were identical. To avoid all chance of error as regards the difference of tension, I measured it in each eye a third time, applying the tonometer to a different part of the eye—namely, 6 mm. to the *outer side*, instead of 4 mm. *below* the corneal margin. Allowing for the greater impressibility of the sclera in this part, I obtained a precisely similar result—namely, *painful side*, 1.0 mm.; *sound side*, 1.1 mm.

I can only conclude, from the foregoing table, that *in young people toothache is not frequently accompanied by noticeable alteration of tension*. A longer series of examinations, made, if possible, during the persistence of pain, in people of middle age or beyond it, and in whom the eye of the corresponding side is distinctly congested—a condition which was only doubtfully present in two of my cases—might, very probably, give more definite results.

Another fact of importance in this connection is that during severe facial neuralgia the eye of the same side is frequently visibly hyperæmic, and that profuse lacrymation occurs at the same time—conditions which point to the probability of a simultaneous increase of the intraocular secretion. Toothache appears to have less power to induce these symptoms than do other forms of neuralgia of the fifth.

On the whole, I think we may safely conclude that *the part played by ordinary forms of facial neuralgia in glaucoma is not one of primary importance*; for, on the one hand, chronic glaucoma occurs pretty frequently in the entire absence of pain in the eye or face, and, on the other, cases of intense trigeminal neuralgia are witnessed in elderly

people over long periods of time without the supervention of glaucoma. One such case, by far the most severe I have ever witnessed, occurred in an elderly gentleman connected with my own family. The pain was agonising, and recurred with the suddenness of a blow, sometimes every few minutes for hours together. The tears flowed profusely, the eye was reddened. During one such attack I could detect no increase of tension. The malady lasted without much intermission for about two years, when it ended in death. Sight was unimpaired, except during the flow of tears, to the last.

Other nerve disorders, less easily defined than the foregoing, are frequently associated with glaucoma, especially in its sudden attacks or exacerbations, and especially in the female sex. Convulsions, hysteria, mental depression, anxiety, excitement, intense headache, have all been known to precede the outbreak of glaucoma. Many such cases are on record. The following is a remarkable one. An elderly lady, passionately addicted to card-playing, and unable to bear a losing game with equanimity, was seized, after playing for heavy stakes and losing, by acute glaucoma in one eye. Under stringent warnings, she forsook for a time her favourite pastime, but yielding after a while to the temptation she played again, lost heavily, became intensely excited, and paid the penalty, within a few hours, of a blinding attack in the second eye. ⁽¹⁾

I have recorded a case of incipient glaucoma, associated with a highly hysterical temperament, at thirty-two years of age. ⁽²⁾

In another of my cases the association of glaucoma with headache is well illustrated. The patient, herself the subject for many years of violent nervous headaches, was the daughter of a woman who had suffered in a similar manner,

¹ Handbuch d. g. A., v. 1., 68. ² Case 19, Section VII.

and died insane. ⁽¹⁾ In another, again, chronic glaucoma was preceded for many years by severe pains in the head, often accompanied by vomiting. ⁽²⁾ I do not attempt to define the relation of these conditions to changes in the intraocular pressure, but would suggest that we may perhaps find a parallel in the vaso-motor disturbances, and secretory derangements which constantly accompany violent emotion, hysterical convulsion, &c.

Gout was considered by many of the older authors to be a frequent cause of glaucoma, especially as regards those forms of the disease which simulate attacks of inflammation. Sichel relates a case of acute glaucoma immediately following the subsidence of gouty inflammation of the knee. ⁽³⁾ The gouty condition is frequently associated with high arterial tension, and it predisposes to the occurrence of sudden hyperæmia, or inflammation, now in one part, now in another. Theoretically, therefore, it would appear favourable to the occurrence of acute glaucoma, but I am not aware of any evidence to show that the connection is frequent. The authority of the older writers is weakened in this instance by the little regard paid by them to the distinguishing feature of glaucomatous diseases—excess of tension. Moreover, it is asserted that in certain neighbourhoods, where gout has of late years become rare, glaucoma is met with in undiminished frequency. ⁽⁴⁾

The climacteric change appears to be occasionally concerned in the production of glaucoma, but its effect is certainly not very great. In the majority of cases the disease first manifests itself at a somewhat later time of life, and, on the other hand, we do not meet with glaucoma in connection with arrested menstruation before the usual time. It is probable that this, and other conditions capable of

¹ Case 23, Section VII.

² Case 20, Section VII.

³ Handbuch d. g. A., v. 1, 67.

⁴ Handbuch d. g. A., v. 1, 68.

disturbing the equable distribution of the blood, and of causing vascular turgescence, especially about the head—such, for instance, as suppression of habitual hæmorrhages or other discharges, arrest of cutaneous perspiration, prolonged bending of the head over work of any kind, constipation of the bowels, &c.—are frequently concerned in exciting exacerbations of glaucoma. On theoretical grounds I believe that they are not, strictly speaking, causes of glaucoma, but that they may excite the malady even in a non-glaucomatous eye, provided the fundamental changes which predispose to it are already present.

Atropine, applied in solution to the eye, has been known, in many cases, to cause an outbreak of glaucoma. ⁽¹⁾ Here, again, it is probable that either the glaucomatous condition itself, as indicated by an excess of tension, or the predisposing change, was already present. In a case under my own care, in which symptoms to some extent suggestive of glaucoma were present, I prescribed atropine tentatively, believing that accommodative troubles might be at the bottom of the mischief. Acute glaucoma of great severity followed. A single application of “calabar” reduced the glaucomatous tension in a few hours to the normal level. ⁽²⁾ The explanation of this phenomenon is discussed in another place. Theoretically, there is reason to believe that *if glaucoma be imminent*, atropine is dangerous in proportion to the youth of the patient. This, however, is an inference, not an established fact. ⁽³⁾ It should invariably be used with caution when any tendency to glaucoma is suspected, and it must be remembered that this tendency, as regards the primary disease, is not very infrequent after fifty years of age. Theoretical considerations must, however, on no account withhold this beneficent drug in the absence of

¹ For a list of cases, see Laqueur. Arch. f. Ophth., XXIII., iii., 152.

² Case 19, Section VII. ³ Section V., p. 154.

good reason. Its omission in the treatment of ophthalmic maladies does, at the present day, I suppose, one hundred times more mischief than its occasional misapplication.

SPECIAL ETIOLOGY OF SECONDARY GLAUCOMA.

When the glaucomatous condition occurs in an eye previously affected with any manifest disorder, it is known as secondary or consecutive.

This complication is met with in the course of many of the maladies of the eye. It may be of short duration, and disappear without effecting any of the changes which characterise a persistent excess of pressure, or it may steadily advance until destruction of the organ is complete. A temporary rise of tension is commonly known as a glaucomatous complication of the pre-existing disease, while the term secondary glaucoma is reserved for those cases in which the hardness of the eye is more persistent, and is followed by its characteristic secondary changes. The distinction is unimportant as regards the subject of this section.

In discussing the pathology of glaucoma, I shall have to refer in detail to certain forms of secondary glaucoma, in illustration of the several modes in which the process may arise; ⁽¹⁾ here I shall only briefly review the morbid states which are liable to this complication, and, as regards the *reason* of the complication, will limit myself to the following general assertion :—

¹ See Section V., p. 174, &c.

Glaucoma, occurring as a complication of other maladies of the eye, is not generally attributable, as formerly supposed, to irritative hypersecretion, but to obstructed outflow of the intra-ocular fluid, caused, for the most part, by altered relations of the iris.

This is asserted as a *general* truth, not as a law without exception.

Iritis of any variety may lead to glaucoma, if the inflamed membrane become adherent to the lens capsule or to the cornea. In the absence of such adhesions, positive increase of tension is rare, except in the *serous* variety.

Serous Iritis is almost always accompanied by an increase of tension. That this is not dependent upon the presence of synechia, is proved by the coexistent wide dilatation of the pupil.

Anterior synechia is liable to be followed by pronounced glaucoma, especially when the adhesion is broad. The danger reaches its maximum in cases of incarceration of the iris in extensive cicatrices of the cornea. It is, I think, hardly correct to assign a separate place among the causes of secondary glaucoma, to "projecting corneal cicatrices." Bulging of the cornea has, *per se*, no tendency to induce a rise of pressure, but is often rather a direct consequence of such a rise. The very frequent association of glaucomatous tension with corneal staphyloma is due to the almost constant presence of iritic adhesions in such cases.

Posterior synechia, if the adhesions are not extensive, has commonly no effect upon the tension of the eye. If the whole circle of the pupil be adherent, so that the passage of fluid from the posterior to the anterior chamber is arrested, glaucomatous complications inevitably occur, unless, as not unfrequently happens, an extension of the inflammation to the ciliary processes impairs or arrests the secretion of the intraocular fluid. In the latter case, a reduction of

tension, generally associated with detachment of the retina, may coexist with total or annular posterior synechia. ⁽¹⁾

Injury of the lens, whether the result of accident or intentionally inflicted by the surgeon, as in needle operations, is very generally followed by a rise of tension. Swelling of the lens, *as a whole*, within its capsule, is, I believe, the essential agent in inducing the complication. Considerable masses of the lens substance may lie in the anterior chamber without any effect on the tension. Inflammatory adhesions of the iris to the capsule, or to fragments of the lens substance, increase the tendency to glaucoma, but are not necessary to its occurrence.

Fragments of the cortex of the lens remaining within the eye are a fruitful source of glaucomatous complications after the extraction of senile cataract. I believe that, in such cases, *rupturing the posterior capsule, so as to destroy the partition between the vitreous and aqueous chambers, lessens this particular danger*. I shall attempt to establish and explain this assertion in another place. ⁽²⁾

Dislocations of the lens are the direct cause of many attacks of secondary glaucoma. The tendency to this complication varies according to the nature of the displacement. If the lens pass entire into the anterior chamber, acute glaucoma of the most violent kind may ensue in a few hours, while in another case of the same kind no glaucomatous symptoms of any kind may arise. I believe the result depends upon the size of the lens in relation to that of the chamber. If it fill the chamber so far as to be firmly in contact with the anterior surface of the iris throughout the whole of its circle, the pupil—*i.e.*, the inlet for the aqueous humour—is closed, and glaucoma inevitably ensues; if, on the other hand, the lens lie excentrically in the lower part of the

¹ Section IV., p. 139. ² Section V., p. 178.

chamber, so as to leave a space, however small, between its posterior surface and the upper margin of the pupil, no such consequence will arise. I found this statement on the experience of four cases in which I have had to extract the lens from the anterior chamber, and on theoretical considerations. The point is discussed in detail in the section on pathology. ⁽¹⁾

Partial dislocations of the lens posterior to the plane of the iris, whether congenital or the result of injury, are apt to be complicated with glaucoma. The way in which an excess of tension in such cases may disappear and reappear many times, in accordance with slight alterations in the position of the lens, is very remarkable. Bowman has recorded and critically analysed a group of these cases. ⁽²⁾

Complete dislocation of the lens backwards into the vitreous humour, when occurring spontaneously, is less prone to induce glaucoma. The lens may lie apparently quite unattached and oscillating freely in the softened vitreous humour for many years without any complication of this kind, until, through some unusual movement or position of the head, it slips forwards through the pupil, when acute glaucoma rapidly sets in. In an old man who had undergone iridectomy in each eye for glaucoma years before, I extracted the lens from the one eye, it having become cataractous, with good result. The lens of the other eye, also opaque, soon afterwards disappeared from the pupil, and was found to have fallen to the bottom of the vitreous chamber. No rise of tension followed the dislocation, although the eye was already rather tense, and the disc distinctly cupped. Artificial displacement of the lens into the vitreous, as in the operation of reclinatio*n*, appears to be less innocuous. It is stated that when this operation was in vogue many eyes subsequently became glaucomatous. It must be remem-

¹ Section V., p. 183. ² R. L. O. H. Reports, V., p. 1.

bered that five-and-twenty years ago the term glaucoma was applied by many writers to internal inflammations of the eyeball, without distinct reference to excess of tension. I am inclined to doubt whether a true glaucomatous condition is very apt to follow such an operation, unless, perhaps, iritic complications are induced. ⁽¹⁾

Foreign bodies lodged within the eye may occasion glaucomatous complications. They do so, I believe, by injuring the iris and the lens, not, as has been stated (probably as a foregone conclusion), by irritating the ciliary processes. When lodged in the latter, their tendency is to cause softness of the eye and detachment of the retina.

Sympathetic inflammation may make the eye glaucomatous. Synechia, as in iritis of other kinds, is, I believe, the agent.

Intraocular tumours, especially sarcomata of the choroid, cause, at some period of their growth, a marked rise of the ocular tension. The symptoms may be acute or subacute, but are always of a more or less irritative type. Glioma of the retina is perhaps less constantly productive of this complication, but is by no means exempt. ⁽²⁾ These cases present simultaneously two conditions which are, for the most part, to some extent antagonistic—viz., high tension, and detachment of the retina.

Myopia of high degree, with staphyloma posticum, is occasionally complicated with glaucoma. Bader records the analysis of a series of cases in which the myopia varied from $\frac{1}{8}$ to $\frac{1}{2}$, and the ages from fourteen to fifty. Myopia of lower degree is, however, not exempt. I have lately had a case under my care in which one eye with myopia $\frac{1}{14}$ became rather suddenly glaucomatous, with pain, engorgement of episcleral

¹ v. Graefe on Glaucoma, New Syd. Soc., p. 372. Bader, The Human Eye, &c., p. 279. Schmidt, Handbuch d. g. A., v. 1, 42.

² Case 6, Section VII.

vessels, "red balls whirling round before the eye," T + 1—impression .53 mm.; the other eye myopia $\frac{1}{36}$, no pain, Tn., impression .73 mm. ⁽¹⁾ Von Graefe witnessed two instances in which several children in one family, when between the ages of twelve and eighteen, were attacked with glaucoma subsequent to the development of rapidly increasing myopia. ⁽²⁾ I am not sure that such cases should be classed as secondary glaucoma. It seems not impossible that the development of myopia by elongation of the eye, and the subsequent onset of glaucoma when the tunics begin to reach the limit of their extensibility, are but two stages of one process, and depend equally upon a pressure slightly in excess of that which the eye can support unharmed.

Retinal hæmorrhage is occasionally followed by the outbreak of glaucoma. Some writers, doubting the possibility of a causal connection in this sequence, consider that the hæmorrhage is a premonitory symptom of the commencement of the malady—that it is, in fact, *caused* by the rise of pressure which has already set in—or that hæmorrhage and glaucoma both are dependent upon disease of the vessels and obstruction to the circulation through them. It is certain that hæmorrhage is not unfrequently the secondary change, and is directly due to the obstructive effects of excessive intraocular pressure, but I think I shall be able to show reason to believe that in an eye in which the predisposing conditions are present, hæmorrhage may at once induce glaucoma. ⁽³⁾

Keratitis, in various forms, has been stated to lead to glaucomatous complications. Their association is indubitable, but I believe that, in all such cases, the disease first becomes a "kerato-iritis," and that inflammation of the cornea, when uncomplicated by extension to the iris, has no such tendency.

¹ Case 34, Section VII. ² Handbuch d. g. A., v. I., 45.

³ Section V., p. 187.

Instances of secondary glaucoma in which the initial malady was the interstitial keratitis of inherited syphilis are not uncommon, in one case of my own the glaucomatous symptoms were so severe that I had to perform iridectomy, but I believe that in all these cases inflammation of the iris precedes the rise of tension. Any attempt to explain the concurrence of glaucoma with corneal changes by assuming an obstruction to the escape of aqueous humour *through the cornea* as the cause, is refuted by the fact, now positively established, that in the living eye no fluid transudes through this membrane. ⁽¹⁾

Glaucoma has occasionally been seen in association with other maladies of the eye than those mentioned, but it is doubtful, in most cases, whether there is any etiological relationship.

Retinitis pigmentosa was, in one case, associated with acute glaucoma in both eyes, and, according to Schmidt, on whose authority I cite this, an excess of tension is frequently discoverable in this disease. ⁽²⁾ Since noting this statement, I have been able to examine only two patients suffering from retinitis pigmentosa. In the first the tension was normal; in the second it was distinctly, though moderately, in excess, as tested by the fingers and by the tonometer, but there was no cupping of the disc, as far as could be seen through a partly degenerated lens. ⁽³⁾

Albuminuric retinitis was discovered by Hutchinson in an eye excised for acute glaucoma, and which was blind previous to the attack. The case is hardly conclusive, for the urine, when examined, contained no albumen, and the other eye was perfect—facts, which, in the absence of a searching microscopical examination of the retina,

¹ Section IV., p. 116. Also own experiments, Section IV., p. 133.

² Handbuch d. g. A., v. 1, 48. ³ Section VII., p. 278-9.

throw some doubt on the nature of the changes observed in it. ⁽¹⁾

Primary atrophy of the optic nerve has been observed as a pre-existent condition in a few cases. ⁽²⁾ It cannot be looked upon as in any way predisposing to glaucoma, but it is possible that the same cerebral conditions which cause the nerve atrophy may be concerned in the onset of the glaucoma, especially if they involve pain, excitement, sleeplessness, &c.

Detachment of the retina is much more frequently associated with a decrease than with an increase of the tension of the eye, the usual conditions of its occurrence being, I believe, essentially opposed to the glaucomatous process. ⁽³⁾ In certain instances this rule fails, and we see detachment of the retina preceding or accompanying the onset of glaucoma—*e.g.*, during the growth of tumours of the choroid, and after the occurrence of extensive subretinal hæmorrhage. ⁽⁴⁾

Aphakia, like the last, is, I believe, in itself opposed to the occurrence of glaucoma; that is to say, in removing the lens, especially if the anterior *and posterior* capsule be divided, we remove a part which is intimately concerned in certain forms of glaucoma. But unfortunately, although the absence of the lens is, *per se*, advantageous in this respect, the procedures by which we attempt to attain this are sometimes very much the reverse. ⁽⁵⁾ They excite secondary glaucoma, I believe, by iritic complications. I have never witnessed the development of glaucoma, after cataract extraction, where the iris has remained entirely free from injury and inflammation. I do not assert that such a

¹ R. L. O. H. Reports, V. 330. ² Handbuch d. g. A., v. 1, 48.

³ Section IV., p. 138. ⁴ Case 7, Section VII.

⁵ Case 36, Section VII.; also Section V., p. 178.

sequence is impossible, but, on theoretical grounds, I believe it to be unlikely to occur. Cases in which glaucoma has been established *before* extraction of the lens was performed are, of course, not to the point.

SECTION II.

THE ESTIMATION OF "TENSION."

The essence of glaucoma is an increase of the intraocular pressure. In the study of this disease, therefore, the means by which changes of this pressure are to be detected are of primary importance alike to the practitioner and the pathologist.

A direct measurement of the hydrostatic pressure of the contained fluids is impossible in the case of the living eye ; we have, therefore, to content ourselves with estimating it indirectly from the tension of the tunics.

Under all conditions of health and disease, it is possible to make an impression in the tunics by external pressure ; the resistance which they offer is the measure of their state of tension, and, indirectly, of the hydrostatic pressure within.

The readiest and, ordinarily, the best means of estimating "tension" is unquestionably the educated touch of the surgeon.

This method was first systematised and supplied with symbols for the record of results by Bowman. ⁽¹⁾ The following is the substance of his instructions : The patient is told to close the eyes gently, as if asleep ; forcible closure of the lids raises the tension of the eye, and renders the lids rigid, and thereby vitiates the examination. The surgeon places the tips of the two index fingers upon the upper eyelid, through which he can feel the upper part of the globe

¹ British Medical Journal, October 11th, 1862.

behind the corneal region. One finger steadies the eye by pressing against it with a suitable degree of force, while the other estimates the tension. The pressure of the fingers should alternate somewhat as in palpation of an abscess suspected of containing pus.

Examinations made by the pressure of a single finger, or even with two fingers of the same hand, though sometimes practised are worthless. The following are the symbols and definitions introduced by Bowman:—

T 3. *Third degree, or extreme tension.* The fingers cannot dimple the eye by firm pressure.

T 2. *Second degree, or considerable tension.* The finger can slightly impress the coats.

T 1. *First degree.* *Slight but positive increase of tension.*

T 1 ?. Doubtful if tension increased.

T n. Tension normal.

— T 1 ? Doubtful if tension be less than natural.

— T 2. First degree of reduced tension. Slight but positive reduction of tension.

— T 2. } Successive degrees of reduced tension, short
 — T 3. } of such considerable softness of the eye as
 — T 3. } allows the finger to sink into the coats. It
 } is less easy to define these by words.

[Like many others, I am in the habit of placing the T first in all cases, and of using the sign + as well as the sign —. Thus, T + 2, T + 1 ?, T — 2, T — 1 ?]

This mode of testing and recording variations of tension has done excellent service in ophthalmic practice, as its general adoption testifies, and will probably continue to be the one best suited for ordinary use. In proportion, however, as the essential importance of such variations has become apparent, and as their origin has baffled discovery, has it become evident that a more precise test

is to be desired by those who would closely study the glaucomatous process, and the means by which it may be controlled. Especially when it is desired to test the validity of opposing views, to ascertain the value or worthlessness of reputed remedies, medicinal or operative, and to control one's own preconceived opinions, is it indispensable to employ some method which, whether more accurate or not, shall, as far as possible, eliminate the effect of the investigator's own wish or prejudice. For the purposes of this essay I have endeavoured, during the last nine months, to check off all my examinations of ocular tension by the mechanical methods of tonometry. Mechanical tension-measurers, or tonometers, of various forms have been devised. In fundamental principle the mechanical is identical with the digital method. Pressure is made upon the eye so as to impress its surface, and the relation between the amount of the pressure and the depth of the impression is the measure of the tension. By the digital method these two factors are estimated by the sense of touch alone; by the mechanical they are both—provided the tonometer be worthy of its name—indicated as known amounts. Even with the most faultless mechanical appliance, however, it is, and must always remain, impossible to *accurately* ascertain the essential condition—*i.e.*, the amount of the intraocular pressure; and for this reason, that there is no constant relation between the intraocular pressure and the tension of the tunics.

When an impression is made in the sclera, the underlying fluid is displaced, and, being incompressible, must be found room for elsewhere. The ease with which this is accomplished depends chiefly upon the degree of pressure already exercised upon the whole internal surface of the sclera by the contents of the eye, but not upon this alone; it is modified by the following circumstances:—⁽¹⁾

¹ See article by Monnik, Arch. f. Ophth., XVI., i.

1. *Variations in the elasticity of the sclera.* If the eye were a perfect sphere, with perfectly incompressible contents, and perfectly inextensible tunics, it could not be indented or impressed by any amount of pressure. Its ability to permit an impression manifestly depends on the stretching power of its coats, and varies therewith. We know that the sclera varies considerably in thickness in different eyes, even in the normal state, and that in diseased conditions the variations are much greater, and it is certain that such differences modify its extensibility. We know also that the sclera is more yielding in early than in advanced life, for a moderate rise of internal pressure, which in persons beyond the period of childhood does not enlarge the globe, suffices in young persons to increase it greatly in all diameters.

2. *Variations in the size of the eye.* Other things being equal, a large eye will accommodate itself more easily to the displacement of a given amount of fluid than will a small one. For just as a long elastic cord will be more lengthened by a given weight than a short one, so will a sclera of large superficies be more increased in area, and therefore in capacity, by a given pressure than a sclera of small superficies.

3. *Variations in the form of the eye.* In the perfect sphere the ratio of content to superficies is at the maximum, and in proportion as the form departs from the spherical does this ratio alter at the expense of the content. This explains the fact that, if into a hollow elastic spheroid fluid be forced under constantly increasing pressure, the spheroid, as it enlarges, tends constantly towards the form of a sphere. It is manifest that an eye which is spheroidal will accommodate a given displacement of fluid more easily than one which is

spherical, for what the former can accomplish by a slight change of form, involves, in the latter, a general extension of the sclera.

These variations, present more or less in every eye, involve unavoidable inaccuracy in the estimation of the internal pressure from the resistance of the tunics. Thus, in a series of eyes with precisely the same internal pressure, *other things being equal*, the old offer more resistance to an impression than the young; the small than the large; the spherical than the spheroidal.

Like other experimenters in tonometry, I have found that the average *tension* of the sclera increases as life advances, but I do not feel justified in asserting, on this ground, that the *intraocular pressure* increases in the same way. This is manifestly an unsafe conclusion, seeing that decreased elasticity produces increased resistance, apart from change of internal pressure.

Another source of error, which appeared to me to be more important than the foregoing, and which I have been at some pains to investigate experimentally, in order to ascertain by what method of tonometry it might best be neutralised, is described at length in the following paragraphs.

4. *Variations in the curvature of the sclera at the point of impression.* The amount of fluid displaced by an impression of given depth is greater as the curvature of the surface is flatter; in other words, the displacement varies with the radius of curvature at the point of contact. The tonometrical error involved in this fact will be best explained by a diagram. Let figs. 1 and 2 represent two eyes similar in size and shape, and having *the same internal pressure*, and let fig. 1 present its flatter curvature, fig. 2 its sharper curvature, to the points of external pressure. Now, if a pressure, say of 10 grammes, be applied at each of these points, the

impression in fig. 2 will be deeper than that in fig. 1 ; or, if an impression of equal depth be made in each, a greater pressure must be employed for fig. 1 than for fig. 2. Therefore, whether we estimate the internal pressures from the *depths of the impressions* in the first case, or from the *pressures employed* in the second, we shall conclude that eye fig. 1 is harder than eye fig. 2. My tonometer, like that of Monnik, ⁽¹⁾ has three points of contact with the eye. A central one, B (fig. 3), by which the impression is made, and two outer ones, A A, which serve as fixed points from which to measure the position of B. Whatever be the position of B, the instrument measures and indicates the distance at which it stands from the imaginary chord A A. Thus, if B be depressed in succession to B', B'', the instrument indicates the distances B' C, B'' C. If no pressure be applied at B, then the three points of the instrument adopt themselves to the curvature of the eye, and measure the distance B C—that is, the versed sine of the arc A B A. In order to ascertain the depth of any impression from the surface two measurements are necessary. For example, to measure the depth B B', it is necessary first to measure the sine B C, and then to subtract from this the distance B' C. In like manner, to measure the depth B B'' we have to add B C and C B''.

This general explanation holds good for Monnik's tonometer also. His system of measurement is this:—He depresses B in every case to a point (B'') .25 mm. below the chord A A, and reads off the pressure employed in so doing. Now, since the sine B C varies in different eyes, it is manifest that the total depression of B (*i.e.*, B B'') will vary also ; that is to say, the impression made by depressing B to a given distance below the chord will have a different depth in different eyes. But it has been already stated that a given

¹ Arch. f. Ophth., XVI., 1.

pressure makes a deeper impression in a sharper curvature than in a flatter curvature, and this fact tends, as Monnik points out, to counteract the error involved in his system of measurement.

In my system the depth of the impression is measured from the *surface* instead of from the *chord*.

The question as to the relative accuracy of the two methods, may be stated thus: *Given a series of eyes of different curvatures—*

1. *If B be depressed in each to a point situated at a given distance from the surface, the displacements of fluid vary.*
2. *If B be depressed in each to a point situated at a given distance from the chord, the displacements vary.*

In which series do the displacements vary the most?

Having failed to obtain a mathematical answer to this question, I have endeavoured to solve it by experiment, as follows:—

Experiment.—See fig. 4.

An india-rubber membrane was tied water-tight over an earthen tea-pot, and the spout of the latter was connected by an india-rubber tube with a horizontal graduated glass tube, fixed at a vertical height of 50 mm. (2 ins.) above the surface of the membrane. The vessel and tube were filled with water so that the upper end of the column stood near the left end of the graduated glass tube. Three straws, tipped with sealing-wax, rested vertically on the transverse diameter of the membrane, and, by means of needles transfixing them near their upper ends, and a millimetre scale, they indicated the height, or sine, of the arc assumed by the india-rubber membrane under the influence of the water pressure beneath it. When the central straw was depressed by the finger to a given extent, as measured by the vertical scale, an impression was made in the membrane, and a certain quantity of water was displaced from the vessel into the glass tube; its amount was measured by the scale attached to the latter. It is obvious that, as the glass tube was open at the distal end, the depression of the membrane would not raise the pressure in the vessel, and that the displaced fluid would not be accommodated by a further distension of the membrane, but would pass in equal volume into the glass tube.

In order to make the experiment as fair as possible, dimensions were adopted proportionate to those of the curvature of the eye, and of the tonometer. The distance between the outer straws was 80 mm.—*i.e.*, ten times the distance between the outer tonometer rods. The mean height of the curvature of the membrane was 5 mm.—*i.e.*, ten times the height of the average sine in the eye, according to Monnik. Monnik states the sine for the outer side of the eye, 6 mm. from the corneal margin, to be on the average .5 mm. The average of a hundred eyes, measured by myself, at a point 4 mm. below the cornea, is .61 mm. These results are to some extent mutually corroborative, seeing that the curvature is quicker as it nears the cornea. For the purpose of this experiment it was more correct to adopt Monnik's measurement.

A series of different curvatures in the membrane was obtained by retying it a little slacker or a little tighter, several trials being generally necessary before precisely the desired curvature was obtained.

In order to avoid fallacy from permanent stretching and change of shape in the membrane, several pieces cut from the same sheet were used at different times; but the precaution appeared to be unnecessary, for the elasticity was so perfect that, after having been tied on the vessel for twenty-four hours under 50 mm. of water, it completely recovered its flatness when released, and showed no trace of extension. A uniform water pressure of 50 mm. was maintained throughout by raising the balanced shelf which carried the vessel, so that the summit of the arc stood, in every case, at the same distance below the glass tube.

The series of curvatures experimented on were the following:—

Sine	=	3 mm.
"	=	4 "
"	=	5 "
"	=	6 "
"	=	7 "

These represent, multiplied by ten, the average sine of the eye—.5 mm.—together with four variations therefrom, two greater and two smaller.

The object in view was, with each sine, to make a series of depressions of different depths, *measured from the surface*, and a second series equivalent to them *measured from the chord*; to ascertain the amounts of fluid displaced in each case; and to show, by comparison, in which series the displacements differed the most from those obtained with the average sine.

With each different sine I made three different depressions measured from the summit of the arc, and three equivalent depressions measured from the chord; in the case of the average sine, 5 mm., these impressions were identical. Thus, with sine = 5 mm.—

- | | | | | |
|---------------------------------------|---|---|---|--|
| 1. Depression to 2.5 mm. below summit | } | = | { | Depression to 2.5 mm. above chord. |
| 2. Depression to 5 mm. below summit | | | | Depression to 0 mm. above chord— <i>i.e.</i> , to chord. |
| 3. Depression to 7.5 mm. below summit | } | = | { | Depression to 2.5 mm. below chord. |
| | | | | |

In the case of other sines, the depths of the corresponding impressions differed. Each observation was several times repeated, and the displacements were noted in every instance. The following table exhibits the results; the average of each group of observations is placed at the foot of the column :—

SINE.	DISPLACEMENTS.					
	Depression to		Depression to		Depression to	
	2.5 mm. below summit. <i>a.</i>	2.5 mm. above chord. <i>b.</i>	5 mm. below summit. <i>c.</i>	0. above chord. <i>d.</i>	7.5 mm. below summit. <i>e.</i>	2.5 mm. below chord. <i>f.</i>
3 mm. ..	48	8	105	54	163	111
	46	8	104	54	167	113
	47		104		163	
	45		104		163	
	— 46	— 8	— 104	— 54	— 164	— 112
4 mm. ..	38	19	96	73	154	128
	38	19	96	71	155	129
	37		96	72	155	128
	— 38	— 19	— 96	— 72	— 155	— 128
5 mm. ..	32		89		140	
	31		87		143	
	32		87		144	
	— 32		— 90		— 142	
	32	= 32	88	= 88	142	= 142
6 mm. ..	32	43	72	93	127	167
	30	43	74	94	129	165
	28	43	75	94	128	162
	— 30	— 43	— 74	— 94	— 128	— 164
7 mm. ..	25	61	72	107	121	172
	24	60	72	107	122	174
	26	59	72	110	122	176
	— 25	— 60	— 72	— 108	— 122	— 174

In the first place, this table proves the statement already made, that the amount of fluid displaced by an impression of given depth is greater as the curvature of the surface is flatter. For example, column *a* shows that the amount displaced by an impression 2.5 mm. deep constantly decreases as the sine increases—*i. e.*, as the curvature becomes sharper. Columns *c* and *e* exhibit the same fact with deeper impressions. I have

not attempted to deduce any mathematical formula for the ratio of this decrease, as the method of the experiment is not sufficiently precise for such a purpose, but the unbroken fall in each of these three columns amply establishes the truth of the statement in question.

Secondly, the table shows that if, instead of measuring *from the summit of the arc*, we make the depressions in each case to the same point as measured *from the chord*, then the displacement varies in the opposite direction; that is, the displacement increases as the sine increases, or as the curvature becomes sharper. Column *b* exhibits this increase in a series of impressions made in each case to a point 2.5 mm. above the chord. Columns *d* and *f* show the same thing in the case of impressions made to the chord, and to a point 2.5 mm. below the chord respectively.

Thirdly, it appears that if we take the displacements in the case of the average sines, 5 mm., as the standard of comparison, the variations from this standard are greater in the series measured from the chord than in the series measured from the summit. The variations are greatest in the shallowest depressions (column *b*), and smallest in the deepest depressions (column *f*).

In order to institute a fair comparison of the two methods, we must consider only their relative accuracy as regards deep impressions, for Monnik always employs a depression to a point .25 mm. below the chord.

On comparing columns *e* and *f*, we see that the displacement with the average sine, 5 mm., is in each case 142, while the maximum and minimum displacements with greater and smaller sines are—

In column <i>e</i>	...	164 and 122
In column <i>f</i>	...	174 and 112

By subtracting the extremes from the average displacements, we can compare the variations incidental to each system of measurement; thus, *in measuring from the summit* :—

Average.		Extremes.		Variations.
142	—	164	=	— 22
142	—	122	=	+ 20

In measuring from the chord :—

Average.		Extremes.		Variations.
142	—	174	=	— 32
142	—	112	=	+ 30

That is to say, the variations by the former method are less than the variations by the latter, in the proportion of about 2 to 3.

The foregoing experiments were made nearly twelve months ago, with a view to ascertaining in what way tonometrical errors, due to asymmetry and varying size of the eye, might best be avoided. The results, though not claiming to be mathematically precise, or to be applicable without any

reservation to the conditions of the eye, appeared to me to show the necessity of adopting a system of measurement in which variations of curvature are not neglected, and led to the employment of my present method.

Subsequent experience, in a large number of cases, has proved to me that by no system can the tonometer yield any but approximate indications. It is in consecutive measurements of the same eye, under various conditions, that the indications are the most accurate, and, for pathological study, the most valuable; and since it is hardly necessary to include the slight changes of curvature due to alterations of tension within short periods of time, I am now less inclined to insist upon the importance of measuring the height of the arc than I formerly was. Still, as it appears that, in principle, the method which I have adopted is the more accurate, it seems worth while to record the experiments which led to that conclusion. (See note, page 48.)

5. *Variations in the fulness of the intraocular vessels.* The blood within the eye, like the fluid humours, is practically incompressible; but, unlike them, it is not limited as to movement by the ocular envelope. Pressure upon the eye is transmitted by the general contents to the outer surface of the bloodvessels, and, by retarding the inflowing while it accelerates the outflowing streams, diminishes the quantity of blood within the globe. Thus space is afforded for a portion of the fluid displaced by a tonometrical impression; and since the reduction in the amount of the blood must depend to some extent on the previous state of fulness or emptiness of the vessels, the depth of the tonometer impression will be modified by the state of the internal circulation of the eye. It is, however, I think, extremely improbable that any appreciable error could arise from this cause. The amount of blood extruded from the exterior of the eye

during the rapid operation of a tonometrical measurement must be extremely small, whilst *variations* in this amount, with which alone we have concern here, must be altogether insignificant.

The foregoing considerations show that the resistance of the sclera is not an infallible index of the intraocular pressure, and consequently that the tonometer, however faultless in construction, and however skilfully applied, cannot determine this pressure with absolute accuracy. But since the same sources of error beset the digital method as have been described for the mechanical, in addition to one or two others peculiar to itself, especially the varying thickness and firmness of the eyelid, it by no means follows that the mechanical method is useless. My own experience is that cases frequently occur in which the tonometer enables one to speak with confidence as to the presence or absence of anomalies of tension, which without its aid must remain doubtful, or be decided rather by attendant circumstances and the surgeon's preconceived ideas as to the nature of the maladies; and that, even supposing the instrument to be incapable of discovering changes smaller than those which practised fingers may detect, it would still be of much value as a controlling test in cases where the action of a remedy or the effect of an operation is on its trial, and where the opinions of skilled investigators are in conflict.

A NEW TONOMETER.

The tonometer with which all the measurements recorded in this essay were made is represented in figs. 5, 6, 7. The mechanism, and, to a certain extent, the principle of the

instrument, are new. The design is my own. A B A are three light ivory rods, parallel throughout their whole length, and capable, each one, of independent movement in the direction of its length. Their anterior ends for a distance of 8 mm. are cylindrical, and slide freely in holes in the thin brass plate which keeps them in position. Throughout the rest of their length the rods are flat. A slot in each, near the posterior end, and a pin, D, keeps them in position. A thread is knotted into a hole in the central rod, near its hinder end, and passes forwards beneath it, and downwards over a small ivory pulley. At the free end of the thread is a ring, R, into which a 10 gramme weight is hooked. When the weight is attached, the central rod is pressed forward with a force equal to 10 grammes. The sectional area of the tip of each rod equals 2 sq. mm. The pin P is the centre of movement for the three pointers indicated by the red and blue lines. Each pointer may be described as a bent lever, of which the pin P is the fulcrum, a fine steel wire (indicated by a coloured line) the long arm, and the short projection, E, at right angles with the last, the short arm. The lengths of the long and short arms are as twenty to one. At the posterior end of each of the ivory rods a cross-piece of very thin steel, F, is attached to it. Each cross-piece bears against the short projection of one of the pointers, so that as the rod slides backwards the corresponding pointer travels along the scale which lies beneath its anterior end. For the sake of clearness, only the upper of the three cross-pieces is shown in the drawing. The movement of the pointer on the scale is twenty times greater than that of the corresponding ivory rod. The scale is graduated so that each degree corresponds to a movement of one-tenth of a millimetre in the rods.

It must be observed that this conversion of a rectilinear into an angular movement involves an error, for the ratio

varies somewhat, according to the position of the pointer. The whole angle through which the pointers travel is, however, so small that the error is practically inappreciable. It is very easy to adopt a means of graduating the scale which shall correct this error. This I actually did in my own instrument, but no perceptible variation in the size of the degrees was obtained thereby. The body of the instrument is of boxwood, and it is protected by a brass cover as far forward as the scale. In fig. 5 it is represented with the cover removed.

ACTION OF THE TONOMETER.

When the tips of the three rods are brought into contact with a *plane* surface, and their direction is precisely at right angles with that surface, the three pointers stand over one another; and if the instrument be advanced, so as to cause the rods to slide back, the three pointers travel across the dial together, neither getting in advance of the other.

If the instrument be inclined slightly to either side, the rod of that side is pushed back the most of the three, and its pointer travels *in advance*; the rod of the other side is pushed back the least of the three, and its pointer travels *in the rear*, while the middle rod and its pointer move intermediately.

So long as the pointer of the middle rod maintains a position equal to the *mean* of the position of the other pointers, we know that the surface is a *plane*.

When the instrument is applied to a *convex* surface, the central rod and its pointer (blue) move *in advance* of the others. The two pointers (red) follow with uniform movement. The distance between the blue pointer and the red

pointers (or their *mean* point, if they are not together) indicates the advance of the central rod, or, in other words, the sine of the curve to which the rods are applied.

Conversely, if the instrument be applied to a concave surface, the outer rods and red pointers travel in advance, and the distance between their mean point and the blue pointer indicates the sine of the curvature.

Each division on the scale measures 2 mm., and therefore corresponds to movements in the rods of $\cdot 1$ mm. In practice, I find it necessary to read smaller measurements than tenths of a millemetre, and I should, therefore, in future, subdivide these divisions ; but after a little practice it is so easy to do this with the unaided eye, that I have not as yet altered my original instrument.

An example will best explain the system on which a measurement is made. Suppose that the instrument is applied to a concave surface, and moved forward until all three pointers are travelling along the scale, the blue, B, in the rear of the red, A A. If it be now removed, the three pointers remain stationary at the several points which they had reached. Suppose that B be found to stand at 70, and A A respectively at 145 and 175 (fig. 5). The mean of 145 and 175 is 160. By subtracting this from 70, we obtain a difference of 90, and we know that the arc of curvature to which the instrument was applied has a height or sine equal to nine-tenths of a millemetre ($\cdot 90$ mm.)

To measure resistance to pressure—*e. g.*, the tension of the eye—the ten gramme weight is attached to the ring. The middle rod is now pushed forward by a force equal to 10 grammes. The two outer rods, as before, offer no resistance, excepting friction, and move back as soon as they are brought in contact with the surface under examination ; the middle rod makes an impression in the surface, and only

moves back when the resistance is equal to the pressure—that is, when the resistance made by the surface at the point of contact is equal to 10 grammes. The depth of the impression thus produced is indicated by the pointers, as already explained. Figure 5 represents diagrammatically the application of the instrument to the eye, the depth of the impression being exaggerated for the sake of clearness.

MODE OF APPLICATION TO THE EYE.

The patient is seated in a chair. If the right eye is to be examined, I stand behind the patient's left shoulder, steady his head against my chest, place my right forefinger on the lower eyelid, depress and somewhat evert it, and tell the patient to look upwards. By this means the sclera is exposed below the cornea to the extent of 5 mm. With the little ivory measure, fig. 7, held in the left hand, I then measure off on the sclera a point 4 mm. below the corneal margin, and notice a bend in one of the minute vessels by which to recognise it again. Then taking the tonometer in my left hand, in the first instance unweighted, I rest it in front, where it is hollowed out for the purpose, on the right forefinger which is depressing the lid, and steady the left hand against the right. In this position I can obtain the absolute steadiness which, in the case of my instrument at least, is indispensable. Advancing the instrument gently, the centre rod is brought against the sclera at the point before ascertained, and its pointer begins to move; as soon as the two outer pointers have also travelled a short distance along the scale—it does not matter how far—the instrument is gently removed. The *mean point* of the two red pointers is noted down in column A of the note book, and the position of the blue one in

column B. Three such measurements are usually made in succession. The weight is then attached to the ring, and three more are made in precisely the same way.

	Unweighted.		Sine above chord = B-A	Weight.		Pit below chord = A-B	Total Impression.
	A	B		A	B		
John J—s. Eyes healthy. Left {	·50	·100	·50	·82	·58	·24	74
	·58	·111	·53	·74	·52	·22	—
	·62	·116	·50	·88	·67	·21	·74
Sarah N—m. Chronic glauc. Right { after iridectomy.	·47	·105	·58	·62	·67	—·05	·56
	·40	·97	·57	·63	·67	—·04	—
	·81	·142	·61	·63	·65	—·02	·55

A simple subtraction gives, in the first case, the height of the sine; in the second, the depth of the pit below the chord. The sum of these two results gives the “total impression.”

There is generally some difference between the consecutive measurements, and, when this is so, the *highest sine* and the *shallowest pit* are selected as being the most correct. For it is manifest that any unsteadiness of the hand in applying the instrument will affect the position of the outer rods, one or both, more than that of the centre rod, and that any extra movement of either outer rod will make the sine appear smaller, or the pit deeper, as the case may be. It frequently happens that, if this selection be reversed, and the *lowest sine* be added to the *deepest pit*, the result is identical with that obtained in the opposite way. The foregoing cases are examples in point. In each case the highest sine and the

shallowest pit are *once* underlined; their sum is equal to that obtained by adding the figures which are *twice* underlined, which are respectively the lowest sine and the deepest pit. It will be seen that in the second case (chronic glaucoma) the pit below the chord is indicated by minus quantities, for the middle rod, though weighted, does not reach the level of the two outer rods, that is to say, the "pit below the chord" does not exist. The minimum of these minus quantities is $-.05$, the maximum $-.02$.

The estimation of "tension" by this instrument is, it must be confessed, a tedious, and, apparently, a complicated process. As regards the reckoning which is required, a little practice has, in my own case, rendered it easy and rapid; but the necessity for recording so many figures is, undoubtedly, a disadvantage. Up to the present time I have had no practical experience of any other tonometer than my own, and I am far from wishing to undervalue those which have been designed and used by other experimenters. The ingenious and convenient tonometer of Monnik, described by him in detail in Graefe's Archives, vol. XVI., I., appears to be an efficient instrument, and to have yielded to its author some important results. That my own is practically superior to this, I am by no means in a position to assert; but I believe that the principle upon which my method was founded is, in certain respects, the sounder of the two. It has already been stated that the tension must be estimated from two data:—1. *The amount of pressure employed*; 2. *The depth of impression produced*; Monnik makes a uniform impression, and measures the pressure employed. I employ a uniform pressure and measure the impression produced. It appeared to me, in view of the sensibility of the eye, that a uniform pressure, of such amount as experience should show to be readily borne, would be preferable to a variable pressure which would be greatest in the very cases where it

might be most objectionable—viz., acute glaucoma, with severe pain. I have met with no objection to the employment of my tonometer, even in the case of acute glaucoma in nervous women, and am constantly answered in the negative when I enquire whether its application is painful. I have no experience of the comparative merits of other tonometers in this respect. Again, it appeared that the unvarying and easily ascertained effect of a weight would be preferable to the less constant effect of an elastic spring, especially as some arrangement was necessary by which the agent, whichever it might be, could be readily thrown in or out of gear. The feature in my instrument, upon which I lay the most stress, however, is the connection of each of the outer rods, A, A, with a separate pointer. This complicates the readings, it is true, but greatly increases their accuracy. Experience shows that it is seldom possible, even with the greatest care, to bring the two outer rods into contact with the sclera at precisely the same moment, for one of the corresponding pointers is almost always left more or less in advance of the other. It follows that, if the outer rods were in one piece, like a fork, so that the contact of either moves a pointer common to both, as is the case in Monnik's tonometer, an error in the reading must occur very frequently—that is to say, whenever the outer points do not reach the sclera simultaneously, the sine will be indicated too low, or the pit too deep, as the case may be.

The following table of measurements, ⁽¹⁾ made on a polished boxwood cylinder, under the most favourable conditions as regards position and steadiness exhibits the necessity of eliminating this source of error. The positions of the two red points (outer rods) were noted down after every application, and appear in the columns *a a*. It will be seen that

¹ See p. 47.

only thrice (*) in the twenty measurements did they exactly coincide; and remembering that the figures in the first decimal place represent the position of the rods to a tenth of a millimetre, $= \frac{1}{250}$ inch, and that those in the second place represent, though, of course, not with any precision, hundredths of a millimetre ($\frac{1}{2500}$ inch), it is manifestly impossible to bring the rods into contact with the eye with a precision at all approaching that which these readings represent.

Of the twenty measurements of the sine, column B—A, the maximum is .61 mm., the minimum .58 mm., giving a difference of .03 mm., $= \frac{1}{850}$ inch. It must be confessed, in measuring the curves and the *tension* of the eye, a series of measurements will frequently, though not always, contain wider differences than this; but, by selecting the maximum sine and the minimum pit, as before explained, I believe that a very satisfactory degree of accuracy is obtained.

It remains only to state the amount of friction in my tonometer. To ascertain this I placed each rod in succession against the long vertical indicator of a very sensitive pharmaceutical balance, taking care that the point of contact was the same distance from the fulcrum as the point on the horizontal bar, from which the pan was suspended. By loading the scale, the weight necessary to move each rod was found. The resistance was as follows:—

Each outer rod	...	5 grains.
Centre rod		7 grains.

The greater friction of the centre rod is attributable to the thread attached to it.

Table of measurements of the curvature of a polished cylinder of boxwood, made to ascertain the amount of error essential to the tonometer:—

<i>a.</i>	<i>a.</i>	A.	B.	B—A.
82	102	92	117	·25 mm.
66	82	74	101	·27 "
79	81	80	108	·28 "
57	67	62	87	·25 "
99	113	106	133	·27 "
* 111	* 111	111	137	·26 "
100	108	104	130	·26 "
117	123	120	147	·27 "
120	130	125	151	·26 "
66	76	71	98	·27 "
* 77	* 77	77	104	·27 "
104	112	108	135	·27 "
83	87	85	113	·28 "
115	119	117	143	·26 "
120	124	122	150	·28 "
112	120	116	143	·27 "
* 109	* 109	109	135	·26 "
158	166	162	188	·26 "
112	120	116	143	·27 "
112	118	115	142	·27 "

Columns a a denote the positions on the scale of the two pointers corresponding to the *outer rods*.

Column A, the mean position of these.

Column B, the position of the pointer corresponding to the middle rod.

Column B—A, the height of the arc.

The extreme measurements are .25 mm., .28 mm. The difference between these = error = .03 mm.

For a table of measurements indicating the relation between the tonometer readings and the commonly employed symbols, see Section VII., page 276.

NOTE.—*May, 1879.* Having quite recently, by means of further experiment, obtained a better insight than before into the relationship between tonometrical measurements and the intraocular pressure, I hasten, just as these pages are going to press, to add a note, which is to some extent a correction of my former conclusions, especially as to the relative merits of the methods adopted by Monnik and myself. In the previous experiments my attention was directed to the relationship between the depth of the impression and the amount of fluid displaced under various curvatures of surface, and various pressures, internal and external. The results obtained afforded no rule by which to calculate the internal pressure from the two known quantities—the depth of the impression and the external pressure employed. I now find that, by measuring the diameter of the impression instead of its depth, we obtain a nearer approximation to such a rule.

Certain terms, made use of in the following paragraphs require definition.

If to the surface of the sphere in which an impression is produced by external pressure we imagine a plane surface to be applied, the line of contact is a circle. The term *impression-plane* is used to mean the imaginary plane bounded by this circle; *diameter of the impression* to mean the diameter of this circle; *depth of the impression* to mean the distance from the bottom of the pit to the summit of the curve in its normal position. The pressure upon the internal surface of the sphere is increased by the formation of an impression in its surface, unless an outlet exist through which a portion of the contents, equivalent to that which is displaced by the impression, can escape. The term *initial pressure* is used to mean that which is present previous to, and the term *modified pressure* to mean that which is present during the existence of the impression.

The following rule holds good, I believe, for all cases, irrespective of the size of the sphere, the amount of elasticity possessed by its walls, and the degrees of pressure, internal and external. It assumes, however, a perfect pliability in the walls:—

RULE.—*The external pressure equals the internal pressure per unit of area multiplied by the number of such units in the impression-plane.*

For example, if a weight of twenty pounds applied externally produce an impression-plane equal to twenty square inches, the internal pressure equals one pound per square inch. Possibly I am here stating that which is well known, or even self-evident, to mathematicians; but in

tonometrical studies it appears hitherto to have received no notice. Surmising the existence of such an equation, and being unable to test it mathematically, I did so by experiment.

Employing the apparatus already described (fig. IV.), I measured the diameters of the impressions produced by a series of known weights. The internal pressure was produced by a column of water 137 mm. in height, = .005 oz. per square mm. This initial pressure was not modified by the formation of the impressions, because the outlet in connection with the horizontal tube remained open throughout. The measurements were made by blackening the surface of the membrane and laying upon it a circular flat card, slit half-way across, so as to permit of its passing the depressing rod. By this means a circle corresponding to the circumference of the impression-plane, was printed off upon the card. Very accurate measurements cannot be obtained in this way but the results, as shown in the following table, are, I think, sufficiently approximate to establish the principle :—

INTERNAL PRESSURE = .005 OZ. PER SQUARE MM.

Weight employed.		Diameter of impression-plane, as measured.		Area of impression-plane.		Internal pressure multiplied by area of impression-plane.
<i>a.</i>		<i>b.</i>		<i>c.</i>		<i>d.</i>
4 oz.	...	33 mm.	...	853.3 sq. mm.	...	4.18 oz.
6 "	...	39 "	...	1194 "	" "	5.97 "
8 "	...	44 "	...	1520.5 "	" "	7.6 "
10 "	...	50 "	...	1962.7 "	" "	9.81 "
12 "	...	55 "	...	2375.8 "	" "	11.87 "
14 "	...	60 "	...	2827.4 "	" "	14.3 "
16 "	...	65 "	...	3318.3 "	" "	16.59 "

The weights with which the depressing rod was loaded are shown in column *a* ; the diameters of the impression-planes obtained by measurement, in column *b* ; their areas obtained by multiplying the square of the diam. by .7854, in column *c* ; multiplying the number of square millimetres in each impression plane by .005, we obtain the figures shown in column *d*. According to the rule, columns *a* and *d* should be identical. Considering the absence of precision in the method by which the impression-planes were measured, their correspondence is remarkably close. Other measurements, made under different conditions as regards internal pressure and curvature of surface, yielded closely corresponding results.

If we attempt to apply this method of calculating internal pressure to the case of the living eye, we are met by several difficulties. The tonometer supplies us with two known quantities—viz. :

1. *The external pressure employed.*
2. *The depth of the impression produced.*

We require to know also—

3. *The diameter of the impression.*
4. *The amount of rigidity in the tunics, i.e., the amount of external pressure which would be necessary to produce this impression-plane in the absence of internal pressure.*

And, lastly, we require to know—

5. *The difference between the initial pressure and the modified pressure ;*

for, from the data 1, 3, and 4, we could calculate only the *modified* pressure present during the application of the tonometer, whereas it is the *initial* pressure which we desire to know.

To what extent can we ascertain the values of these unknown quantities ?

A direct measurement of the diameter of the impression appears unattainable by any possible contrivance. Can we, in any given case, deduce the diameter from the depth ? I find by experiment that, in the case of any given sphere, the ratio between the diameter and depth of an impression varies in impressions of different depths. In the case of a large india-rubber ball of considerable rigidity, I find that if the *depth* of the impression = $\frac{1}{30}$ diam. of sphere, its *diameter* is about eight times greater ; if $\frac{1}{30}$, about six times ; if $\frac{1}{20}$, about five times. The ratio for an impression in the tunics of the eye could only be ascertained by experiment on the dead subject. If we connect the vitreous chamber with a column of water of known height, we know the internal pressure ; if we then, with a known external pressure, make an impression of known depth, we can calculate the area, and thence the diameter, of the impression-plane, and thereby ascertain the ratio between the diameter and the depth of this particular impression. If we now in every case produce an impression of this depth, we know (assuming similar size and shape in the eyes) the diameter and the area of the impression-plane.

The error due to rigidity of the tunics can also only be ascertained by experiment on the dead subject. If we establish, by means of a column of water, a pressure = 0 in the vitreous chamber, we can then ascertain the pressure which is required to overcome the rigidity of the tunics in making an impression of any given depth. This amount must be subtracted from the external pressure employed before including the latter in any calculation.

Finally, the difference between the initial and the modified pressure must in like manner be determined by experiment. If we connect a column of water with the vitreous chamber, and then, *after closing the outlet*, produce a known impression by means of a known weight, the column represents the initial pressure. If we then raise the column to such a height that, *the outlet being open*, the same weight produces the same impression as before, the exalted column represents the modified pressure as it existed in the former instance with closed outlet.

Supposing the foregoing data to be obtained, the tonometer would enable us to calculate the intraocular pressure. Let me give an illustration, although certain of the data are wanting.

A weight of 10 grammes produces, in the average healthy eye, an impression .8 mm. deep. (This is the actual fact according to my own measurements.) Now, .8 mm. = $\frac{1}{30}$ diameter of the eye. Let us therefore assume, following the indications of the india-rubber ball, that the diameter of the impression = 6 times its depth ; then diameter of impression = .8 mm. $\times$ 6 = 4.8 mm. ; and area of impression-plane = 4.8 squared $\times$.7854 = 18 square mm.

From the 10 gramme weight we must subtract the amount expended in overcoming the rigidity of the tunics—say 1 gramme—and we have effective weight = 9 grammes.

Then, by the rule, internal pressure per sq. millimetre = $\frac{9}{18}$ gramme = .5 gramme. Now, .5 gramme per sq. millimetre = pressure of 50 *centimetres of water*.

This, then, according to our calculation, is the internal pressure of the normal eye during the application of the tonometer.

How much must we subtract from this in order to obtain the initial pressure? I will not even hazard a guess in answer to this question, but will point out that the normal intraocular pressure, as determined by purely experimental methods, appears to be equal to about 30 *centimetres of water*.⁽¹⁾ Our calculation, although including some very uncertain data, has thus led to a result which is probably not very far removed from the truth.

My intention in setting forth this method of calculation has not been to hold out the hope that the tonometer will in the future be an exact indicator of the intraocular pressure, for so long as eyes differ in size, shape, rigidity, and elasticity, exactitude will be unattainable; but rather to lay down a principle which shall enable us to perceive where mathematical certainty ends and where physiological uncertainty begins.

As a practical outcome of the investigation, I submit the following conclusions:—

The employment of a uniform impression (Monnik's tonometer) affords a simpler basis for calculation of the internal pressure than does the employment of a uniform weight (my tonometer). The former system has, moreover, a practical advantage—viz., that the sensitiveness of the instrument increases with every rise of the intraocular pressure; it varies, in fact, with the amount of the difference between the initial and the modified pressure, and thus increases with the tension of the tunics.

If an impression of .8 mm. (or .75 mm. Monnik) be adopted for all cases, the distance between the outer rods of the tonometer need not be more than 6 mm., for the diameter of such an impression is certainly less than this. This reduction in the *chord* would diminish the average *sine* from .5 mm. to .3 mm., and would diminish its variations by one half, in which case they might probably be neglected with advantage, certainly with much less disadvantage than when the chord of .8 mm. is employed, as in Monnik's and my own tonometer.

When leisure and opportunity permit, I hope to work out experimentally the average values of the unknown quantities set forth above, and to remodel my tonometer in conformity with these later conclusions.

¹ Section IV., p. 97.

SECTION III.

THE SYMPTOMS OF GLAUCOMA.

Glaucoma is characterised by one constant and essential symptom—increased tension of the eyeball. Let this physical condition be added to any eye, healthy or diseased, and straightway it becomes glaucomatous, and will, under continuance of the pressure, manifest those changes which are peculiar to the disease. On the other hand, let the excess of pressure be taken away from the glaucomatous eye, and, whatever morbid processes or structural changes may be left, glaucoma exists in it no longer. Glaucoma is, then, strictly speaking, a symptom—a complex one, truly, but still a symptom—of certain hidden processes which lead to excess of pressure within the eye.

Those forms of the disease in which increase of tension is the first discoverable change are termed primary, whilst those in which it has been preceded by other evident morbid processes are classed together as secondary, or consecutive.

Primary glaucoma is the uncomplicated type, and is very generally to be understood when the word glaucoma is used without a qualifying adjective. The long series of symptoms, subjective and objective, met with in this disease, are referable, perhaps without exception, to the fundamental change—the increase of pressure within the eyeball; and the varying degrees of intensity which they manifest depend upon the rapidity with which the pressure rises, and the height to which it reaches.

In accordance with these variations we recognise three forms of primary glaucoma—the chronic, the sub-acute, and the acute. Continental writers commonly employ the terms simple, chronic-inflammatory, and acute-inflammatory, but these are not free from objection on pathological grounds. Von Wecker expresses the same differences more happily by the words irritative and non-irritative. Each possesses distinctive clinical features, but is not divided from the next by any hard and fast line. I propose, in the first place, to describe the general characters of each form, and subsequently to analyse more closely certain of the individual symptoms.

Chronic glaucoma begins almost imperceptibly, progresses slowly, with little tendency to exacerbation or remission, and leads inevitably, unless arrested by treatment, to blindness. The patient, when first seen by the surgeon, is usually at least fifty years of age. He complains of a dimness of sight, which has come on gradually in one, or in both eyes. One eye is almost invariably somewhat worse than the other in this respect, and amongst the poorer classes relief is frequently unsought until the second eye is seriously impaired; indeed, it occasionally happens with unobservant persons that one eye is blind, or nearly so, before its owner discovers that anything is going wrong. We are frequently told, if the question is asked, that spectacles have been used from an early age, and that recently they have been changed for stronger ones, once or more, with only temporary benefit. The patient complains of little or no pain in the eyes, but will perhaps have suffered more or less pain in the temples, forehead, or face. On examining the functions of the eyes, we find the range of accommodation small in proportion to the age, if not totally wanting; the acuity of vision more or less impaired; and the field of vision peripherally contracted—a sign that the corresponding portion of the retina is no longer sensitive. A subjective phenomenon frequently,

though not constantly, met with, is the appearance of a ring of prismatic colours around any luminous object at which the patient looks. Externally the eyes exhibit little or nothing amiss. The episcleral vessels—anterior ciliary arteries and veins—which, especially the latter, are barely visible in the healthy eye, are usually somewhat enlarged, and appear as four single or double trunks opposite the insertions of the four recti muscles. The depth of the anterior chamber is reduced by an advance of the lens and iris towards the cornea. The pupil is frequently large in proportion to the age of the patient, and responds imperfectly to light; but this is by no means a constant symptom. *The tension of the eyeball is increased.* The excess at first may be so slight that, apart from other symptoms, it would hardly justify a diagnosis, and yet it may suffice, if continuous, to induce the whole chain of secondary changes; and, on the other hand, it may, in the later stages, amount almost to stony hardness, and yet, if its advance have been even and slow, it may be entirely unaccompanied by pain, and alter the appearance of the eye but little. It is, however, in every form of glaucoma, the chief diagnostic symptom. The second in importance is revealed by the ophthalmoscope. The optic disc, and the vessels lying on it, exhibit changes which are quite characteristic of glaucoma. As the result of pressure, and consequent atrophy of tissue, the disc is depressed to a lower level than that of the surrounding retina—it becomes excavated, or “cupped.” The rim of the cup is firmer, and, consequently yields less than the other parts, so that it comes gradually to be undermined in such a way that the sides of the cup are hidden from the observer’s view by the overhanging margin. The undermining is made evident by the interrupted course of the vessels, which, visible at the bottom of the cup, are lost to view as they ascend the sides, and reappear, changed as to number and position, as they bend

round the rim of the cup to gain the retina (figs. 47, 49). The excavation of the disc involves its whole area quite up to the scleral margin—a point of much importance in the differential diagnosis between this and other forms of cupping. Around the margin of the disc there is almost always a narrow circle of rather irregular contour and of a lighter colour than the adjoining parts—a circumscribed choroidal atrophy. The vessels of the disc and retina exhibit changes as regards size, course, and pulsation. Those portions of the veins which traverse the retina are generally enlarged, and sometimes tortuous, whilst the portions situated within the hollow of the disc may be reduced in size. The arteries are usually reduced in size throughout, but not to any great extent in the earlier stages of the disease. In advanced glaucoma, accompanied by elongation of the eyeball, all the retinal vessels are attenuated (figs. 48, 49). The peculiar interruption of course which the bloodvessels appear to undergo at the margin of the disc has already been mentioned. There is frequently another abnormality of distribution—namely, a displacement of all the vascular trunks towards the nasal side of the disc, or, if viewed by the indirect method, an *apparent* outward displacement. Pulsation of one or more of the venous trunks on the disc is commonly to be seen in glaucoma, and, if not present, may readily be induced by light pressure on the eyeball; but it has little diagnostic value, for it occurs in eyes of normal tension under certain circumstances, which will be discussed later on. Pulsation of the arteries of the disc is a more trustworthy sign of increased pressure within the eye, but this, too, is occasionally seen in connection with other conditions, and is absent in many glaucomatous eyes.

The whole of these symptoms are not to be met with in every case of chronic glaucoma, nor are they necessary for the recognition of the disease. By far the most significant

are the increase of tension, the cupping of the disc, and the contraction of the field of vision.

Sub-acute glaucoma is characterised by the intermittent nature of its course. It may be described as chronic glaucoma, plus periodic exacerbations, of an irritative, quasi-inflammatory type. The symptoms coincident with each of these attacks are as follows :—

A rise of tension, remarkable rather for the rapidity of its occurrence than for its amount, is the most important, inasmuch as it is the precursor and cause of the others. There is a rather sudden obscuration of vision, amounting sometimes only to a mistiness, sometimes to almost total darkness. A ring of colours around luminous objects is generally perceived. Pain is felt in the eye, and often in the forehead and the temple. The conjunctiva is injected, the anterior ciliary vessels are engorged and tortuous. The pupil is unusually large and sluggish, and the anterior chamber shallow. On attempting to view the fundus of the eye with the ophthalmoscope during such an attack, we observe some impairment of the normal transparency of the cornea, a change which has been generally attributed to turbidity of the media, but on insufficient grounds.

After these symptoms have persisted for some hours, or it may be for a day or two, the attack subsides. The first few attacks may leave the eye apparently but little damaged, but with their repetition the glaucomatous state becomes permanently established. The tension remains persistently in excess, the optic disc succumbs to pressure and becomes cupped, and all the other changes already described ensue. Each occurrence tends to intensify the mischief and to render the subsequent remission less complete. Months may elapse between, and during the quiescent period the eye may exhibit only such symptoms as are met with in the earliest stages of chronic glaucoma; or, on the other hand,

the attacks may recur from day to day, and rapidly produce destructive changes. The acuter symptoms tend to become persistent; the enlargement and tortuosity of the episcleral vessels increase; the pupil remains large and fixed, in shape frequently oval rather than round; the tissue of the iris atrophies; the disc is deeply excavated with extensive destruction of its nerve fibres; pain is frequent and severe; and sight, long since limited to a small central area of the retina, is totally extinguished, though subjective phenomena simulating light may still continue.

The structural changes which ensue still later, in this and in other varieties of glaucoma, will be described in another place. ⁽¹⁾

Acute glaucoma is distinguished from the varieties already noticed by the suddenness of its onset, the intensity of all its symptoms, and its little tendency to spontaneous remission. While chronic glaucoma is liable to be mistaken for simple atrophy of the optic nerve, acute glaucoma simulates a general inflammation of the eyeball. The resemblance, although only superficial, has led too often to disastrous results. Here, again, it cannot be too forcibly reiterated that the whole malady depends upon excessive pressure within the eyeball. We have here to deal with no true inflammation, and cannot cure the disease by any antiphlogistic measures—as many a patient could testify in years gone by; a lowering of the pressure, and that only, can break the chain of evil consequences. The onset of the attack is frequently at night, or in the early morning, while the patient is in bed, and appears in many cases to be immediately induced by some form of exhausting nervous excitement, such as anger, grief, or prolonged anxiety. The patient states that she—acute glaucoma is somewhat more frequent in women than in men—that she was seized with sudden pain in the eye,

¹ See “Absolute Glaucoma,” p. 60.

which soon extended to the forehead and temple, or even affected the whole side of the head and face; that this grew more and more severe hour after hour, and seemed almost to blind the eye; that vomiting set in, and that in a few hours she was reduced to a state of utter misery and prostration. The ocular changes during such an attack as this are very striking. The eyelids are somewhat swelled, the conjunctiva reddened, and perhaps œdematous; the scleral vessels are much injected, not only as regards the four groups of episcleral vessels, which are prominent in the less acute forms of the disease, but as a general hyperæmia over the whole surface of the membrane, and especially in the neighbourhood of the cornea. In the most severe cases a protrusion of the eyeball is discoverable. The tears flow profusely, and the eye, though nearly blind, is intolerant of light. The cornea has lost its polish, and "looks like a glass which has been breathed upon," and, provided a high degree of tension has already persisted some time, its sensitiveness to touch is generally diminished or lost. The pupil is widely dilated and totally inactive—a condition which at once indicates that we have before us no ordinary inflammation; it has, moreover, a grayish or greenish hue, instead of the black of the normal eye. Telling the patient to look downwards, and placing the tips of the two forefingers upon the upper eyelid, *we find the eyeball almost stony hard*. The vision of the affected eye is always much impaired. In very acute cases it may, in the course of a few hours, be reduced to the barest perception of light, or it may be totally extinguished; more frequently, a day or two, or more, must pass without relief before this state is reached. The field of vision, tested by the movements of a candle before the eye, or by throwing light upon it from various points by means of the ophthalmoscope, will be found reduced in size. On attempting an ophthalmoscopic examination of the fundus we are baffled, in spite

of the dilatation of the pupil, by the imperfect reflex. If a view of the optic disc can be obtained, signs of obstruction to the circulation will be discovered; the arteries are small, and almost empty themselves by a regurgitation of blood during each ebb of the arterial wave; the veins are engorged, and their main trunks, close to the centre of the disc, collapse with each arterial pulse. Enlargement of the veins is not invariably present, however. In a case of acute glaucoma, induced in a predisposed eye by the use of atropine, I observed the disc to be pale, and both arteries and veins to be reduced in size (see case 19, appendix). If the fundus can be thoroughly examined, extravasations of blood situated in the choroid or retina may sometimes be discovered, though they are less frequent during the advance of the disease than subsequent to its relief by iridectomy, when a lowering of the pressure external to the vessels causes them to rupture.

Excavation 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. As will appear later, cupping of the disc cannot be looked upon as a purely mechanical result of exalted pressure; it probably implies atrophic changes which, though essentially due to pressure, take time for their development.

Hæmorrhagic glaucoma.—In certain cases of glaucoma, a tendency to intraocular hæmorrhage is an early and prominent feature; so much so, indeed, that a distinct variety of the disease has been described as hæmorrhagic glaucoma. For clinical purposes the distinction is useful, inasmuch as the proclivity to rupture manifested by the vessels renders the sudden lowering of ocular pressure by iridectomy extremely hazardous, and may, consequently, counterindicate the operation. Pathologically, these cases appear to differ

from the others chiefly in being complicated with advanced degeneration of the blood-vessels. The subjects of this form of glaucoma "suffer from the head symptoms usual in sclerosis of the cerebral arteries, and often die from apoplexy tolerably soon after the commencement of the disease." (¹)

Numerous minute aneurismal dilatations and varicosities have been discovered in the retinal vessels in such cases, and these, under the conditions of obstruction brought about by rise of the intraocular pressure, are prone to rupture. Hence it happens that hæmorrhage is sometimes the first sign of mischief, the excess of tension being still too slight to have called attention by the production of other symptoms, and being barely discoverable with the finger. The development of higher tension with all the other symptoms of glaucoma, a little later, will not unfrequently establish the true nature of the case. Such cases hold a doubtful place between the classes of primary and secondary glaucoma. If we hold, in accordance with what has just been said, that a rise of pressure precedes the extravasation, they belong to the former; while if the rise of pressure first becomes positive at a later period, and is a consequence of the hæmorrhage, then they must be placed in the latter group. The pathology of hæmorrhage in glaucoma will be more fully dealt with in another section. (²)

Absolute glaucoma.—The condition of a glaucomatous eye which has already passed unrelieved into total and irremediable darkness is thus designated. The ultimate effects upon the eyeball of an excess of internal pressure are manifested in this stage. They are chiefly alterations in the size and shape of the organ, dependent upon degeneration of its tissues. The symptoms belonging to the absolute stage of glaucoma vary somewhat in accordance with the previous

¹ v. Graefe. See translation R. L. O. H. Reports, VII., 95.

² See Section V., p. 187.

character of the disease. If it have been chronic throughout, the eye, though totally blind, may appear at first glance to be perfectly healthy, and may be entirely free from pain. Its extreme hardness to the touch, however, at once declares the cause of the blindness, and the ophthalmoscope reveals a deep excavation of the optic nerve, with contracted arteries, and a surrounding ring of choroidal atrophy. The anterior chamber is usually shallow, but the pupil, even in this stage, is not always dilated; it may be of normal size, and retain its power of consensual action. Occasionally the anterior chamber appears of normal or more than normal depth, and the iris is traversed by one or more large blood-vessels. ⁽¹⁾ In this condition an eye blinded by chronic glaucoma may remain for years without undergoing any visible external change; ultimately, however, it will almost certainly suffer certain of the alterations described below. When the course of the disease has been sub-acute or acute, the ultimate changes are usually much more prominent. The anterior ciliary vessels are large, tortuous, and of a deep purplish red colour, the large trunks appearing in front of the insertions of the recti muscle, and ramifying and inosculating as they approach the situation where they perforate the sclera. Their appearance is all the more striking because the general surface of the sclera is unduly pale and the conjunctiva thin. The transparency of the cornea is more or less impaired, its epithelium is thickened, rough, and sometimes raised in little vesicles; and the true corneal tissue is often more or less densely opaque, especially throughout that portion exposed by the opening of the eyelids. Its sensibility is greatly lowered or abolished. If the structures behind the cornea are visible, the iris is seen to be reduced to a narrow circle by reason of the wide dilatation of the pupil; its characteristic markings are gone, and at the peripheral

¹ Section VII., case 22, fig. 42.

line it is in contact with the cornea, its surface being altered in colour by destruction of the epithelium. The lens sooner or later becomes cataractous, and, provided the cornea be transparent, is then strikingly conspicuous, by reason of its advanced position and the wide dilatation of the pupil. It is most important, however, to remember that, in any stage of glaucoma, the lens may appear at first sight to be cataractous when it is not so. The inexperienced observer may well be astonished to find that he can view with the ophthalmoscope the minutest details of the fundus through a crystalline lens, which to the naked eye looks like a cataract almost ready for extraction. At every ophthalmic institution, probably, instances of bitter disappointment due to previous mistakes in diagnosis from this cause are witnessed: patients suffering from advanced glaucoma, but hoping to be cured of what they have been told is cataract, have to be informed that their malady has been mistaken, and is in an incurable stage. In those cases in which the lens becomes really cataractous the opacity is often extreme, having a brilliant white or yellowish appearance, such as occurs in the secondary cataract associated with disease in the ciliary region. Pain often continues long after the total extinction of sight. This is so chiefly when the disease has previously run a sub-acute or acute course, with sudden painful exacerbations. So long as the sensory nerves retain their function, and the conditions capable of inducing sudden rise of tension persist, so long will the eye be liable to pain. It is chiefly for the relief of this that advice is sought when glaucoma is already absolute. Subjective visual sensations are not infrequent long after the eye is blind, and these also, though more rarely, may bring the patient to the surgeon, in the vain hope that they indicate a chance of improvement.

The last stage of glaucoma is marked by changes in the size and shape of the eyeball. These occur in two opposite

directions, according to whether the integrity of the tunics or the processes of secretion within hold out the longer. If secretion persist in sufficient amount, the eyeball gradually enlarges, the tunics are thinned, the curvature of the cornea flattens and approximates to that of the sclerotic, the ciliary sclera is extremely attenuated, and has a bluish look in consequence, and the conjunctiva is atrophied and rotten. Instead of a general enlargement, or simultaneously with it, a more circumscribed bulging of the sclera frequently occurs in one or more places. Such a weakened state of the tunics is liable to lead to rupture. This occurs usually during some excessive muscular effort—such as coughing, sneezing, or the like—which is accompanied with forcible contraction of the eyelids; it is followed by profuse hæmorrhage from the eyeball, and generally causes violent pain until the distended tunics are relieved by the free escape of blood. Ultimately the eyeball collapses and contracts to a small size, in which condition it may unfortunately still be liable to recurrences of pain. ⁽¹⁾ If, on the other hand, the atrophy of the intraocular vessels, especially those of the ciliary processes, have proceeded in advance of the wasting of the sclera, so that the secretion of the intraocular fluids is insufficient to cause extension of the latter, changes of an opposite kind occur—viz., shrinking, with loss of tension. The cornea contracts in all diameters, the sclera is indented by the traction of the tendons of the recti, its resistance to the finger is greatly lessened, and a small, atrophic, sunken eye remains. Even in this condition also there is sometimes a liability to attacks of pain. Inflammation of the choroid, followed by ulceration and rupture of the cornea, extrusion of necrosed tissues, and even hæmorrhage of considerable amount, may complete the destructive process.

¹ Case 23, Section VII.

SYMPTOMS OF SECONDARY GLAUCOMA.

The symptoms of secondary glaucoma are essentially the same as those already described as belonging to the primary form, for the cause is identical in the two cases—an excess of pressure within the eye—but they are necessarily modified or masked by the other changes present. To the description already given I shall, therefore, add only a brief notice of these modifications.

A rise of the tension of the eye above the normal limits is the essential and characteristic symptom. It has the same importance, and must be investigated in the same way, as when it is the primary symptom.

Anomalies of accommodation are seldom discoverable, by reason of antecedent changes affecting the cornea, iris, lens, &c.

Changes of refraction, for the same reason, are not often observed. In young people, however, in whom the persistence of high pressure invariably increases the size of the eyeball—the so-called buphthalmos—the production of extreme myopia may occasionally be demonstrated, when the condition of the media will allow. ⁽¹⁾

Impairment of vision is usually extreme, for, in addition to the pressure upon the retina, there are usually present conditions affecting the transparency of the cornea, lens, or vitreous, and the patency of the pupil. Such eyes often come to total blindness, and it is sometimes a question of importance whether this depends upon pressure or upon obstruction in the path of the rays of light. Persons occasionally present themselves whose blindness, optically

¹ Case 4, Section VII., specimen 4.

considered, might be remediable were it not physiologically hopeless. Especially is this the case after neglected iritis. The margin of the pupil becomes adherent throughout to the lens capsule; fluid collecting behind the iris bulges it forward till its periphery touches the cornea; from that moment glaucomatous complications supervene, the function of the retina succumbs, and the eye becomes hopelessly blind. The formation of an artificial pupil which, earlier in the disease, would have lowered the tension and readmitted the light, and thus preserved a useful organ, is now no longer able to restore the sight.

Iridescent vision is occasionally met with—*e. g.*, in the secondary glaucoma of myopics. Usually the optical conditions are too much deranged to allow of its occurrence.

Contraction of the field of vision probably occurs precisely as in primary glaucoma, in such cases as do not previously involve disease of the retina. In one of my own cases of buphthalmos with high tension, perception of light was retained only in a very small area of the retina, corresponding, as far as one could tell, with the situation of the yellow spot. ⁽¹⁾

Pain is a very frequent symptom. In many cases it has been already present as a part of the original malady, and is then invariably intensified by the onset of the glaucomatous attack. As already stated, its intensity depends much upon the rapidity with which the tension increases. Very severe pain accompanies the onset of glaucoma during attacks of iritis, with occlusion of the pupil. ⁽²⁾ Intense pain, with vomiting and prostration, are induced by simple displacement of the lens into the anterior chamber, in the absence of any injury or previous inflammation. ⁽³⁾ The same occurs

¹ Case 1, Section VII. ² Case 28, Section VII.

³ Cases 24, 25, Section VII.

during the high tension produced by effusion of blood into the chambers, or between the tunics. ⁽¹⁾ When the tension rises gradually and only to a moderate degree, as in some cases of intraocular tumour, and in myopia with glaucomatous complication in elderly people, the pain is trivial.

Shallowness of the anterior chamber is present in many cases. Total abolition of the chamber, by compression of the iris and lens against the posterior surface of the cornea, may often be observed on making a section through eyes which have been excised for absolute glaucoma originating in iritic adhesions with lens or cornea; but this condition is usually masked, during the life of the eye, by opacity of the cornea. As a warning that glaucomatous complications are likely to arise, an advanced position of the iris, whether due to the direct pressure of the lens or to the accumulation of fluid imprisoned between the iris and the lens, is a symptom of much importance.

Dilatation of the pupil only occurs in the absence of synechia—*i.e.*, in the minority of cases of secondary glaucoma. It is generally to be observed during an attack of serous iritis, and will be found to occur simultaneously with the increase of tension. During the growth of intraocular tumours, a wide dilatation of the pupil, due to the amaurotic state of the detached retina, occurs independently of excess of tension, but the dilatation becomes still more extreme when glaucomatous complications supervene. In such cases we see the iris reduced to a mere dark line behind the periphery of the cornea.

Dilatation of the episcleral vessels is one of the most prominent symptoms of the secondary as of the primary

¹ Case 7, Section VII.

form of glaucoma. It is especially well seen in the later stages—absolute glaucoma—when the general redness of the conjunctiva and sclera, from iritis, &c., has subsided. A single glance at a blind eye in which this condition is well marked sometimes convinces the surgeon that excess of pressure has been the cause of blindness. It is, however, not an absolutely certain indication. In a case of complete destruction of both corneæ by small pox, ultimately resulting in repair by opaque cicatrices, in which no trace of the tissue or the form of the cornea was visible, the episcleral vessels were enormously enlarged in both eyes, and presented an almost exactly similar appearance in each. One eye was excessively hard, the other as remarkably soft. The vascular dilatation was probably, in the latter case, a remnant of a former glaucomatous condition.

Cupping of the optic disc occurs sooner or later in every instance of persistently excessive tension. In eyes excised on account of secondary glaucoma it is constantly to be discovered on section, if the tension has been in excess for some months previously. As a symptom during the life of the eye it is very frequently beyond the reach of observation, by reason of obstructions of the pupil, &c., but, when discoverable, is always of value as an indication of the share which pressure has had in producing the impairment of vision. Fig. 48 represents the disc in a case of buphthalmos, and fig. 49 in a case of myopia with excess of tension. The similarity in the two drawings is striking, and depends upon the fact that in both cases the eyeball had undergone much elongation, with attenuation and atrophy of the tunics. The small size of the blood-vessels, and the remarkable absence of sharp curves or tortuosities in their course, are noteworthy indications of the stretching to which they have been gradually subjected. Speaking of cupping of the disc in

association with posterior staphyloma, v. Graefe says, "the excavation of the optic nerve is never so strikingly and clearly marked in these cases as where there is no posterior staphyloma. . . . The sclerotic around the optic nerve is diseased, and hence, its power of resistance approximating to that of the entrance of the optic nerve far more than in the normal eye, there cannot be formed by pressure a deep cavity in the papilla, but only a shallow depression, with a little displacement of the vessels at its margin." ⁽¹⁾

Changes in the form of the globe occur as the ultimate effects of pressure in secondary as in primary glaucoma; indeed, their varieties are more numerous and irregular in the secondary form, because of its frequent connection with inflammations and mechanical injuries of the tunics. Thus, localised staphyloma, single or multiple, occurs in the ciliary region or at the equator when an inflammation of the uveal tract, involving also the sclera and retina, is complicated with excess of pressure. Again, partial or total staphyloma of the cornea, or of the cicatricial tissue which replaces it after ulceration or injury, is a frequent condition in secondary glaucoma. Such prominences are not certain indications of excess of pressure from within, for they may occur merely under the action of the normal pressure upon weakened tunics, but they should always suggest an examination for the presence of morbid tension.

The ultimate fate of such eyes, if the glaucomatous condition pass unrelieved, differs little from that to which primary glaucoma leads, as already described. The tendency to loss of tension, with detachment of the retina and shrinking of the eyeball, is probably more frequent in this class of cases than in the primary, because of the common occurrence of conditions which diminish or arrest the intraocular secretion-processes,

¹ v. Graefe on Glaucoma, New Syd. Society, p. 375.

especially inflammatory disorganisation of the ciliary body. Destruction of the eye in the opposite way—namely, by distention, attenuation, rupture, and collapse—may also occur. Eyes blinded by secondary glaucoma should be, and usually are, excised before such a result is reached. They are capable, in many instances, of exciting sympathetic inflammation in the fellow eye.

ANALYSIS OF THE SYMPTOMS OF GLAUCOMA.

In order to test experimentally the supposed causal relationship between increased intraocular pressure and certain of the phenomena observed in glaucoma, I have constructed an apparatus by which the living eye may be subjected to pressure of known amount, variable at pleasure, for any desired period of time, while at the same time subjective and objective examinations of the organ may be made.

Fig. 8 represents diagrammatically a side view of the apparatus, a portion of the frame, which would otherwise hide the face, being removed. The head is fixed steadily by means of adjustable supports, *a, a*, for the chin and forehead. Pressure is made upon the eyeball by means of a polished wooden ring, shown in section and with exact dimensions in fig. 9. The radius of the concavity which is towards the eye is 14 mm.—*i. e.*, 2 mm. in excess of the average radius of the sclera—so as to allow for the thickness of the eyelids; the hole is 12 mm. in diameter—*i. e.*, the average diameter of the cornea. This wooden ring is carried by a rigid wire, fixed into a light wooden bar, *c*, which oscillates upon a fulcrum situated vertically above the eye. The bar carries a movable weight, and is graduated so as to indicate the pressure which this weight exercises, through the medium of the ring, upon the eye. The bar is so balanced that, in the absence of the weight, the ring hangs close in front of the eye, but without contact. When the weight is on the bar, the ring compresses the eyeball through the lids without obstructing the entrance of light. A string attached to the other end of the bar enables the person under examination to remove the pressure and to reapply it at pleasure. For the examination of refraction and accommodation, a graduated scale, *d*, carrying a sliding block, *e*, provided with a sight-object, is fixed before the eye. The block is moved to and fro by the person under examination, by means of a string and counterweight, *f*.

In ascertaining the position of the far point, a convex lens, *g*, is placed before the eye. In order to measure variations in the size of the pupil I availed myself of the ingenious method devised by Landolt. ⁽¹⁾ A double prism is fixed upon the sliding block. The observer, looking from the other end of the graduated bar, through the prism, sees two images of the pupil. The prism is moved nearer to or further from the eye by the observer, until the two images of the pupil exactly touch without overlapping; its position on the scale is then read off, and indicates, in accordance with values previously ascertained for each degree of the scale, a given deflection—*i. e.*, the diameter of the pupil.

1. *Diminution of the range of accommodation* never fails to accompany the onset of glaucoma in such eyes as previously possessed accommodative power. In the old, where extreme presbyopia already exists, it is not discoverable, but in early middle life a retrocession of the near point, to the extent of several inches, representing a loss of accommodation equivalent to a lens of 20 or 15 inches focus, occurs with even a moderate rise of tension, while, during an acute attack of glaucoma, the accommodative power appears to be entirely abolished. By experiment with the pressure apparatus already described, I have obtained strong evidence that the excess of pressure is the direct cause of the limitation. I accurately ascertained the positions of the far and near points of my own right eye—first in the normal condition, then under a pressure of 40 grammes. In measuring the far point a convex lens of 40 cm. focus (16 ins.) was interposed at 5 cm. (2 ins.) from the eye. It is necessary, therefore, to subtract 5 cm. from the measurements obtained, and to consider the focus of the lens 35 instead of 40 cm. I made ten successive trials of the far and near points in each case, and as the readings on the scale were invisible to me, and were noted down by an assistant without comment, the conditions of an unbiassed experiment were ensured. The following were the measurements obtained, variations of less than half a centimetre ($\frac{1}{2}$ inch) being disregarded:—

¹ Annales d' Oculistique, 1878, Mars-Avril, p. 146.

UNDER NO PRESSURE.		UNDER PRESSURE OF 40 GRAMMES.	
Far point (with lens 40 cm. focus, 5 cm. from eye).	Near point.	Far point (with lens 40 cm. focus, 5 cm. from eye).	Near point.
31.5 cm.	16. cm.	28.5 cm.	17.
32. „	15. „	30. „	18.5
31.5 „	15. „	29.5 „	17.5
31.5 „	15. „	27.5 „	17.5
31.5 „	15.5 „	28. „	19.
32. „	15. „	29.5 „	17.5
31.5 „	15. „	30. „	17.5
30.5 „	15. „	29. „	18.5
30. „	15.5 „	30. „	17.
30.5 „	15. „	29.5 „	17.
Average 31.25	15.2	29.15	17.7

Subtracting 5 cm. in each case from the measurements of the far point, and deducting the influence of a lens of 35 cm. focus, we find the *real far points* to be—

<i>Under no pressure.</i>	<i>Under pressure of 40 grammes.</i>
105 cm. = 41 inches.	... 79 cm. = 31 inches.

Reckoned in inches, the pre-existing $M \frac{1}{40}$ was increased by the pressure to $M \frac{1}{30}$. This was an unexpected result. It appeared probable that pressure applied to the front of the eye would shorten the antero-posterior axis, and thereby produce hypermetropia. The actual result was, I believe, due to an increased convexity of the cornea through the pressure on the ciliary region, for a distinct myopic astigmatism occurred very readily in the vertical meridian unless the edges of the lids were approximated so as to leave only a

small gap of the ciliary region at each side free from pressure. The position of the near point was displaced in the opposite direction, viz. :—

<i>Under no pressure.</i>	<i>Under pressure of 40 grammes.</i>
15.2 cm. = 6 inches.	... 17.7 cm. = 7 inches.

Reckoned in inches, this represents a loss of accommodation = $\frac{1}{40}$ (approximately), and to this must be added a further loss equal to the increased refraction, viz. $\frac{1}{120}$, by approach of the far point; thus, the total diminution of the range of accommodation induced by a pressure of 40 grammes applied in the manner described = $\frac{1}{40} + \frac{1}{120} = \frac{1}{30}$.

The probable explanation of this symptom, as it occurs in glaucoma, is this: The rise of pressure in the vitreous chamber increases the tension of the choroid and suspensory the ligament, and thereby diminishes their power to undergo the forward movement which is essential to the accommodative act. ⁽¹⁾ Paresis of the nerves to the ciliary muscle is a less feasible explanation; later in the course of the disease the nerves doubtless suffer atrophy from long-continued pressure, but it is unlikely that they are so affected at a time when the sensory nerves exhibit no sign of impaired function, and under a pressure very much lower than that which we often see, at a later stage, to be quite compatible with free movements of the iris.

Changes of refraction—i.e., of the position of the far point—are less frequent than changes of accommodative range. Hypermetropia is found in eyes affected with glaucoma in 50 to 75 per cent.; but it cannot be inferred from this that the refractive error is caused by the excess of tension, especially as there is reason to believe that the hypermetropic structure predisposes to glaucoma. Again, comparisons of the refractive state before and after iridectomy, which have been adduced

¹ Hensen and Völckers, Arch. f. Ophth., XIX., I., 156.

in this connection, are, I think, entirely fallacious. In a given case, it is said, hypermetropia was present before iridectomy; myopia was discovered after it: and, since iridectomy cured the disease, the subsequent condition was the normal one, the previous, a product of the disease. The occurrence of myopia after iridectomy is certainly, in many cases, due to the advance of the lens on the evacuation of the anterior chamber, a displacement which often disappears very slowly, and sometimes remains as a permanency. Case 21 illustrates the conversion by iridectomy of a slight H into a slight M, and the ultimate establishment of E. Cases occur, however, in which, without operation, a development or an increase of H or a reduction of M are discoverable during the progress of glaucoma. ⁽¹⁾ In case 19, H $\frac{1}{38}$ appeared in both eyes, previously emmetropic, immediately before an acute attack. Here, however, the change was attributable, in part at least, to the use of atropine.

The lowering of refraction by increased tension is readily explained in the same way as was the reduced accommodative power—viz., by the increased tension of the suspensory ligament, whereby the convexity of the lens is diminished.

Increase of refraction is also sometimes observed. In advanced glaucoma, especially in the secondary form met with in young persons, the so-called bulphthalmos, the antero-posterior axis of the eye is often greatly lengthened (specimen 5, case V., measures 32 mm. = $1\frac{1}{4}$ inch), which in a seeing eye constitutes a high degree of M. The slight increments of refraction which often usher in glaucoma are probably due, however, rather to an advance of the lens and to a slight change in the form of the globe than to a positive distension of the tunics. With every rise of tension the spheroid tends to become a sphere: therefore, *ceteris paribus*,

¹ Handbuch d. g. A., V. I., 3.

the short, flattened, hypermetropic eye is lengthened, the elongated myopic eye is shortened, by rise or fall of pressure within.

The modes, then, in which the refraction of the eye may be altered by an increase of the intraocular pressure are numerous and mutually antagonistic—*e.g.* :—

- Flattening of the cornea ;
- Shortening of the antero-posterior axis ;
- Lengthening of the antero-posterior axis ;
- Advance of the lens ;
- Reduced convexity of the lens.

It is, therefore, not surprising that the refraction should in one case be increased, in another be diminished, and in a third be left unchanged. ⁽¹⁾

Iridescent vision—*i.e.*, the appearance of a ring of prismatic colours around luminous objects—is frequent in the early stages of glaucoma, especially during periods of temporary exacerbation. It is stated that the red end of the spectrum is usually to the outer side of the circle, the violet to the inner, and that between the light and the coloured circle there is an intervening non-luminous space. The cause of the phenomenon is not clear. Donders describes it as an “interference” effect, due to clouding of the refractive media; ⁽²⁾ von Wecker, observing that a somewhat similar anomaly of vision occurs in some conjunctival affections, attributes it to “very slight alterations in the

¹ The foregoing paragraphs were written before the theory which attributes the causation of primary glaucoma to a senile change of form in the lens had been arrived at (see Sections IV. and V.) I will not now attempt to bend the facts into conformity with the theory, but will merely suggest that, should the theory prove to be correct, these accommodative and refractive changes may turn out to be, in part at least, precursors rather than consequences of a rise in the intraocular pressure.

² Handbuch d. g. A., V. I., 6.

corneal epithelium, caused by a temporary exaggeration of pressure." ⁽¹⁾

It must be remembered that the normal eye is by no means achromatic. The existence of chromatic aberration is readily made manifest in several ways—*e. g.*, covering one half of the pupil with an opaque shield while viewing the boundary line between white and black surfaces; intercepting the middle rays of the spectrum by cobalt blue glass, &c. ⁽²⁾ It appears probable that in glaucoma the latent physiological aberration is in some way rendered manifest or exaggerated.

By means of the pressure apparatus (fig. 8) I subjected my right eye to a pressure of 40 grammes while looking steadfastly at the flame of a candle at ten feet distance. The effect upon the refraction was such as to produce polyopia; the flame appeared to be triple, a central bright one, with a fainter one on each side, and in contact with it. After a minute or two the outer margin of each lateral image was seen very distinctly prismatic, the violet to the outer side, within that blue, shading into green; the other colours of the spectrum were not distinguished. An increase of pressure almost sufficient to abolish vision, made by the finger on the bar, did not increase the iridescence. On removing the pressure the flame instantly recovered its normal form, and the iridescence vanished. On looking out of the window, at least two minutes later, I was instantly struck with an iridescent appearance round the moon, and round a street gas lamp, visible against a pitch-black background. The contrast presented by each of these to the two eyes was striking and unmistakeable. The gas flame was seen with each eye as a symmetrical star of brilliant rays; to the *left* eye the rays appeared devoid of colour except the yellowish tint of the gaslight; to the *right* they were

¹ *Thérapeutique Oculaire*, p. 352.

² Helmholtz. *Handbuch der Physiolog Optik*, 125.

brilliantly coloured—violet, blue, and red were very evident, the violet was furthest from the flame. On making the eye hypermetropic with a — 10 inch lens, the order was reversed, the red being then to the outer side. This difference between the two eyes lasted at least six or seven minutes. During this time I examined my pupils twice in a mirror to ascertain whether the chromatopsy might be due to an inequality in the size of the pupils, but found them exactly equal. ⁽¹⁾ I would point out that to my eye, which has a refraction $M = \frac{1}{50}$ or less, the spectrum appeared with the violet end outwards, but that when the M was converted artificially into H the order was reversed. These are the positions in which the normal chromatic aberration of the healthy eye disposes the colours in M and H respectively. In glaucoma the red is outwards in the majority of cases, presumably because the refraction is most frequently hypermetropic. This would appear to indicate that the phenomenon is closely allied to the chromatopsy which is always present, though seldom perceived, in the healthy eye.

Corneal opacity is of very frequent occurrence in glaucoma, especially in the acute form, and in the later stages. In the latter the epithelium is very often the seat of marked changes, looking as though it were raised in little blisters or were partially peeled off; but in the early stages of the disease no such changes are discoverable. The surface retains a high polish, and presents a sharp reflected image; the cloudiness which is here discoverable, and which has frequently been mistaken for opacity of the vitreous humour, appears, beyond all doubt, to be situated in the true corneal tissue. Time fails me for a review of all the facts and

¹ This experiment was carefully made, and was noted down at the time, now some months ago. I felt sure that the phenomenon observed was a pressure effect. Quite recently, wishing to confirm the observation before recording it here, I repeated the experiment with slight alterations several times, and, to my surprise, failed to obtain the same results.

opinions which have been recorded in connection with this subject, ⁽¹⁾ and I must confine myself to calling attention to one or two points of importance.

It has been suggested recently that such opacity is due to "an increase of the osmosis which occurs through the healthy cornea, to such an extent as to separate and infiltrate the corneal tissue." ⁽²⁾ Leber has proved, and my own experiments have fully confirmed the fact, that no such osmosis occurs even under a pressure thrice as great as that of the healthy eye. ⁽³⁾ It is exceedingly unlikely that such osmosis ever occurs in the living cornea, unless it has been partially destroyed or injured, for, apart from experimental proofs, the very existence of enormously high tension is evidence of the completeness of the obstruction to the escape of the intraocular fluid. ⁽⁴⁾ Again, the theory which attributes these corneal obscurations to infiltration with altered fluids along the normal nutrient channels, though doubtless true for cases of considerable and persistent opacity, is hardly satisfactory for those not uncommon instances, in which the haze appears and disappears with great suddenness and completeness.

It is probable, I think, that the change is a purely physical one, affecting the minute relations of the corneal elements, and, consequently, the transparency of the membrane as a whole. Evidence in support of this opinion may be obtained by observing the change effected in the cornea of an excised eye by considerable pressure on the globe. If the perfectly fresh eye of a pig be squeezed between the finger and thumb the whole cornea is dimmed, and if much force be employed the dimness is so great as almost to completely hide the iris. The instant the pressure is relaxed,

¹ See especially Schnabel's article in the *Archives of Ophth. and Otol.* (English edition), vol. V., pp. 342-381.

² *Arch. f. Ophth.* XXII., III. ³ Section IV., p. 113. ⁴ See Section IV., p. 116.

perfect transparency returns. This change may be effected as many times in a minute as the fingers can be made to act. If the eye be illuminated with the ophthalmoscope meanwhile, an obscuration very much resembling that which is witnessed in acute glaucoma is seen during every compression of the eye. The pressure necessary to effect this obscuration is certainly considerable, but it is not unlikely that the slight cloudiness met with in the cases in question may be evoked by a pressure much less intense.

By oblique illumination of the cornea, opacities in the superficial epithelium, in the substance of the membrane, and dotted over its posterior surface, are also frequently discoverable. These do not appear and disappear suddenly as does the diffuse cloudiness above referred to. They depend upon nutritive disturbances, which take time for their production and recovery. The dotted opacities on the posterior surface are doubtless minute deposits of lymph from the altered aqueous humour. It must be observed, however, that certain observers have disputed the existence of any such alteration in the aqueous. ⁽¹⁾ The fact that the evacuated aqueous humour received into a watch-glass has been found to be discoloured, if confirmed by sufficient trials, and if not rendered fallacious by the chance admixture of a minute quantity of blood, appears to be capable of deciding the question. ⁽²⁾ I have no experience which throws light on this matter; excepting of the well-known fact that in serous iritis with glaucomatous tension—a condition constantly accompanied by dotted opacities on the posterior surface of the cornea—the fluid which escapes on paracentesis is often thick and yellow.

Diminished sensibility of the cornea may often be observed during acute attacks, and in the advanced stages of glaucoma-

¹ Schnabel, Schweigger. See Arch. for Ophth. and Otol., vol. V., p. 335.

² Handbuch d. g. A., V., I., 10.

tous degeneration. It is generally attributed to a partial impairment of the function of the corneal nerves, due to the direct action of pressure upon them. Some additional cause, however, appears to be at work, for in chronic glaucoma, in which very high tension has come on gradually, the sensibility of the cornea is frequently little, if at all, impaired. These variations appear to find a parallel in the greater and smaller degrees of paralysis present in the iris in these different forms of glaucoma. It is not improbable, I think, that *stretching* of the nerve fibres plays some part in diminishing their conducting power. A child was under treatment at my hands for severe interstitial keratitis, which had reached a formidable stage before he came under notice. At his third or fourth visit I was surprised to find that the pain and photophobia, previously severe, had ceased suddenly and entirely. I found that both corneæ had become rapidly infiltrated to such an extent as to threaten rapid destruction, and had undergone a very marked distension in all diameters at the same time. The eyes ultimately recovered excellent sight, but the corneæ remain considerably above the normal size. The sudden cessation of nervous symptoms appeared to me to be due, in all probability, to the sudden *nerve-stretching*. Again, corneal anæsthesia occurs during "rapidly-forming protrusion of the eyeball." (¹) Ad. Weber concludes, as the result of certain observations made during his experiments on rabbits, that loss of corneal sensibility depends more upon bulging of the cornea than upon high pressure. (²) In the glaucomatous human eye a positive bulging of the cornea is entirely exceptional, but it is likely that the nerves undergo a considerable stretching when the sclera is suddenly made tense, as in an attack of very acute glaucoma; and, in the latter stages, when staphylomatous conditions ensue, they

¹ Bader. The Human Eye, &c., p. 171.

² Arch. f. Ophth., XXIII., I.

manifestly do so. In chronic glaucoma time is given for the nerves to adapt themselves to the altered conditions, as we know, from recent experiments in nerve stretching, they are capable of doing. In the later stages of absolute glaucoma the ciliary nerves doubtless pass into a state of atrophy, in common with the other structures within the eyeball.

Dilatation and inactivity of the pupil occur in the same classes of cases as those in which the foregoing symptom is met with, and are probably caused, to some extent, in a similar manner. Certain other causes, however, are certainly in operation. The presence of a peripheral adhesion of the iris and cornea, produced by the bulging forward of the former by the enlarged ciliary processes, must of itself tend to draw the pupillary margin outwards. ⁽¹⁾ In advanced glaucoma the narrow atrophic iris may be seen, during life, to be adherent to the cornea throughout more or less of the circle. The somewhat irregular or oval form of the pupil in the glaucomatous eye is probably due to the more advanced peripheral changes which are present in certain portions of the circle. The impairment of retinal function in the glaucomatous eye *tends* in the same direction, but appears to exercise no great influence on the size of the pupil; for cases of chronic glaucoma in which both pupils are of normal size, and active, *consensually*, although one eye is already blind, are not uncommon. ⁽²⁾

There is, under ordinary circumstances, a tolerably close relation between the size of the pupil and the amount of blood circulating through the iris, and it appears probable that the dilatation of the pupil which occurs in acute glaucoma is due in part to a mechanical emptying of the vessels of the iris. ⁽³⁾ This explanation is by no means completely satisfactory, however, without further evidence.

¹ See Section IV. ² Case 21, Section VII.

³ Leber. Handbuch d. g. A., II., I., 362.

A variety of experiments, made in order to ascertain the effect of injection of fluid into the vessels and into the chambers of the dead eye, have given contradictory results as regards alterations in the size of the pupil. ⁽¹⁾ Again, very high pressure, as in chronic glaucoma, may exist with small pupil and active iris, and intensely high pressure may exist with extreme venous hyperæmia of the iris (fig. 42, case 22); in fact, if the circulation through the iris is affected by an excess of intraocular pressure, in the same way as the circulation through the ciliary processes, we should expect to find a condition of turgescence or venous hyperæmia, rather than one of vascular emptiness during a glaucomatous attack. ⁽²⁾ In spite of these difficulties, I will venture to offer a suggestion which may help to explain the different behaviour of the iris under sudden and gradual rise of intraocular pressure. We know that, under increased resistance to the propulsion of the blood, the arterial walls become greatly hypertrophied, and, further, that such a hypertrophy has actually been discovered in the internal arteries of the glaucomatous eye. ⁽³⁾ This may be the reason why, under a *gradual* rise of pressure, the blood supply to the iris is maintained unimpaired, while, when a *sudden* stress falls on the external surface of the comparatively yielding healthy arteries, their calibre is so far diminished as to induce arterial emptiness in the iris, and consequent dilatation of the pupil. And it is to be noticed, further, that the arteries of the iris are much more exposed to pressure than are those of the ciliary processes, which pass directly into these organs at the point where they perforate the sclera. This applies, of course, only to the anterior ciliary arteries. ⁽⁴⁾ I suggest the foregoing merely as an hypothesis. Certain experiments on

¹ Op. cit. p. 363. ² See Section V., p. 156.

³ Hulke. R. L. O. H. Reports, vol. III., 70, 71, &c.

⁴ See diagram, Handbuch d. g. A., II., I., 313.

this matter which I had in view when the pressure apparatus and pupilometer before described were constructed have not as yet been undertaken, through want of time.

Dilatation of the anterior ciliary veins is a characteristic and important symptom of the glaucomatous condition. It will be fully discussed in connection with the primary causes of glaucoma, to which it bears an intimate relation. ⁽¹⁾

Enlargement of the anterior ciliary arteries is a very nearly constant symptom in glaucoma, though it has not attracted very much attention. In consequence of the increased resistance to the entrance of blood into the eye, these minute trunks undergo more or less hypertrophy. They are to be distinguished from the veins by their greater tortuosity, by their very abrupt disappearance at the points where they perforate the sclera, by the greater pressure required to empty them by the finger, and by the fact that the current re-establishes itself, after being interrupted, from the equator towards the corneal margin. The difficulty with which these vessels are emptied by finger pressure is a characteristic sign of glaucoma, and may occasionally assist an otherwise doubtful diagnosis.

Pulsation of the veins of the optic disc is a phenomenon not confined to glaucomatous eyes: it frequently occurs in eyes of normal tension. But inasmuch as it is intimately dependent upon the mutual relation of the blood pressure and the intraocular pressure, and is especially liable to occur when the latter rises, it deserves consideration here. It is not a pulsation in the ordinary sense of the word, for it is not a direct continuation of the arterial blood-wave. It is, as Donders has pointed out, the result of *external* pressure upon the veins, produced by the incoming arterial wave, and transmitted by the contents of the globe—the vitreous humour. Under ordinary conditions the systolic influx of

¹ Section V., p. 156.

arterial blood is made room for, presumably, by a minute enlargement of the globe, and by an acceleration of the venous efflux, so small in amount as to produce no visible change in the calibre of the veins. When the intraocular pressure rises, and the sclera approaches the limit of its elasticity, the effect of the arterial wave falls more and more upon the veins—the only structures capable of undergoing compression. Hence the presence of venous pulsation in the glaucomatous eye.

Why, then, is the venous collapse limited, as it invariably is, to a small central portion of the retinal vein? why is it often absent in glaucoma? and why is it often present in eyes of normal tension? I venture to think that a completely satisfactory answer to these questions has not yet been given. Let fig. 56 represent a portion of a compressible tube (a vein) transmitting fluid. If a localised pressure fall suddenly upon the portion *b*, the adjacent portions *a*, *c* undergo an instantaneous dilatation, in order to accommodate the fluid expelled from the portion *b*. Let fig. 57 represent a portion of another such tube passing through a partition, *p* (a retinal vein, passing at *p* through the lamina cribrosa into the optic nerve). If a general and equable pressure fall suddenly upon the whole of the portion to the right hand of the partition *p*, a compression and diminution of the tube in portions *a*, *b*, *c*, &c., can only occur by means of a great acceleration of the amount escaping at *p*, and can not occur, to any visible extent, until after the lapse of a certain interval of time. Thus displacement of fluid from portion *b* is resisted by the pressure falling simultaneously upon portions *a*, *c*. Portion *d*, adjacent to the partition, is the only one which can collapse under the influence of sudden general pressure, for portion *e*, beyond the partition, being exempt from the pressure, is able to dilate so as to accommodate the fluid which is driven into it from *d*. This, I think, explains

the collapse of the small portion of the vein immediately adjacent to the aperture in the optic disc, synchronously with each arterial pulsation. When we remember that the venæ vorticosæ pass into a dense firm structure like the sclera, and that they pass obliquely and at considerable length through this unyielding channel, the absence of any visible "pulsation" or collapse in these trunks appears to be explained. Secondly, as regards the appearance of this phenomenon in eyes of normal tension, I would point out that the tendency to collapse of the central portion must be greatly increased by any condition which tends to check the force of the stream at the *distal* end of this portion—*i.e.*, near the margin of the disc. I believe that when a main arterial trunk crosses a main vein near the margin of the disc the dilatation of the former compresses, or may compress, the latter in such a way as to greatly favour the collapse of its central portion. Fig. 58 expresses this idea diagrammatically, the dotted lines showing the conditions of artery (A), and vein (V) during the arterial wave. Imagine this arrangement to exist at the point *f*, fig. 57, and the increased liability to collapse in portion *d* is manifest. This is not a mere hypothesis, but was suggested to me by the appearances observed in several cases.

Fig. 45 was sketched from an eye affected with premonitions of glaucoma, tension very slightly in excess, T + 1 ? Impression .69 mm. Portion *a* of the vein "pulsated"; no other pulsation was discoverable in either disc, although the tension of the other eye was precisely the same; in no other spot did an artery overlies a vein. ⁽¹⁾

Fig. 44 illustrates the point in a more striking way. The tension of the eye was strictly normal. I was led to look carefully for venous pulsation in portion *e*, solely because the

¹ Fig. 45, case 19.

peculiar arrangement of the arterial and venous branches appeared to be so favourable to its occurrence. The portion *e*, when viewed by the direct method, was seen to pulsate strongly. ⁽¹⁾

Another way in which the vein may be predisposed to suffer this "pulsation" is, I believe, by being bent very sharply in the immediate neighbourhood of the disc. It is well known that the friction of a fluid traversing a pipe is greatly increased by bends or angles in the latter. Fig. 46 exhibits the disc of a myopic eye, the tension of which was strictly normal, and, as indicated by the tonometer, inclining to softness rather than hardness. Portion *a* of the large vein turned suddenly backwards, and collapsed slightly with every arterial wave. It is possible, I think, that it may have been so stretched across the underlying artery as to have suffered some slight compression by the dilatation of the latter.

I think it is probable that some mechanical cause of this kind—perhaps not always a visible one—is present in every case in which a vein pulsates visibly under normal pressure.

Finally, we have to enquire why this phenomenon is often absent in glaucomatous eyes. In these also I suspect that its occurrence varies considerably with slight variations in the mutual relations of the vessels, the angles which they make, and the condition of the portion of the nerve into which the vein immediately passes. If this is abnormally tense, an obstacle similar to that offered by the sclera in the case of the *venæ vorticosæ* may be offered to the compression of the vein. A more important factor, however, must be noted. The venous pulse depends essentially on the elasticity of the arteries, whereby they are enabled to yield to the pulse wave. In chronic glaucoma the arterial

¹ See account of the case, Sec. VII., p. 266.

muscular wall is greatly hypertrophied. ⁽¹⁾ And this doubtless materially diminishes the impulse which is transmitted to the vitreous. A certain elasticity in the arteries being essential, it is not surprising that venous pulsation occurs much more constantly in the healthy eye when subjected to pressure than it does in cases of chronic glaucoma.

Pulsation of the arteries of the optic disc is a symptom of more import than the foregoing, as regards the presence of an excess of intraocular pressure. The pulse becomes visible when, by reason of an excessive pressure on their external surface, their diastolic contraction is greater than in health. It is the expression of the contention between the pressures in the arteries and in the ocular chambers, and the alternate supremacy of each. During the cardiac systole, or as its immediate consequence, the retinal arteries are filled, and, in extreme cases, may be seen to undergo slight movements of unbending or elongation on or close to the disc; during the diastole they collapse, a sudden pallor is visible in the area of the disc, and movements the opposite of those which occur during the systole take place. Klein, ⁽²⁾ who has recently investigated this symptom in a large number of healthy and glaucomatous eyes, states that it may be induced by external pressure in almost every healthy eye, the amount of pressure necessary being very various; and, in common with other observers, assigns no very precise value to the so-called "easily-induced arterial pulsation" in the diagnosis of glaucoma. ⁽³⁾ Spontaneous arterial pulsation, on the other hand, though not absolutely limited to glaucoma, is, in the great majority of instances, a sign of increased intraocular pressure. It has been observed, also, in cases of aortic insufficiency, in Graves's disease, and even in a state of

¹ R. Hulke, L. O. H. Reports, vol. III., 70, 71. See also some of my own sections of optic disc.

² Arch. f. Ophth., XXII., IV., 157. ³ Handbuch d. g. A., V. I., 9.

health. Arterial pulsation is decidedly more frequent during periods of exacerbation in an early stage of the malady than it is in chronic glaucoma, however high the pressure may become. This fact, and the readiness with which it may be induced in healthy eyes, is presumably dependent upon the varying degrees of elasticity present in the coats of the arteries. In chronic glaucoma, as already stated, extreme hypertrophy of the muscular coat opposes the development of the pulsatile movements in any marked extent.

Contraction of the field of vision is, next to excess of tension and cupping of the optic disc, the most characteristic sign of the glaucomatous process. It invariably proceeds, unless complicated by other morbid processes, from the periphery towards the centre. Leber has shown that the peripheral parts of the retina receive the fibres which occupy the centre of the optic disc. The excavation of the disc occurs at the expense firstly, and mostly, of these central fibres; moreover, it is only in cases of cupped disc that we see the gradual slow contraction of the field. In acute glaucoma, in which, as it is well-known, no cupping is present there is not any concentric extension of the blindness at all comparable with this; the function of the retina suffers, as a whole, more than in chronic glaucoma. If central vision be but little impaired there will, I believe, usually be little or no contraction of the field in purely acute glaucoma. These facts point to atrophy and destruction of nerve fibres in the disc as the cause of the progressive contraction of the visual field. The peripheral deterioration is discoverable at the onset only in subdued light. The temporal, the upper, and the lower quadrants of the retina usually suffer more than the nasal quadrant; in other words, vision *outwards* remains unimpaired the longest. Statistics obtained from the examination of the field in 119 cases of glaucoma are given

by H. Schmidt. ⁽¹⁾ For ordinary purposes a blackboard suffices for the mapping of the field. I use a circular board which hangs on the wall, with pulley and counter-weight, so as to be readily adjusted for any height. A bent iron rod fastened to the board above, and bearing a small cushion on its free end, supports the forehead of the patient, and keeps the eye at ten inches from the centre of the board. A series of circles painted on the board indicate the angles from 0° to 50° , and a series of lines passing diametrically through the centre indicate the meridians. With this board all defects except those which lie more peripherally than 50° can be mapped out.

Experiment. The pressure apparatus (fig. 8) was modified in such a way that pressure could be applied without narrowing the field of vision laterally. Instead of the wooden ring (fig. 9), a small steel rod, tipped with a flat ivory knob, having a diameter of 5 mm., made the pressure on the eye. It was directed at an angle of 45° upwards against the lower lid, and, by means of a pulley and a series of weights, could be made to exercise a pressure of any desired amount. A stage placed just below the level of the eye carried a series of small opaque white glass balls, arranged in a semicircle, with the eye as a centre. The balls were separated from each other by an angle of ten degrees, and were backed by a dead black surface. The head being fixed, the light was lowered until the balls, though still distinctly visible, disappeared after about twenty seconds, if the eye remained perfectly motionless. The ball on each side of the central one (the fixation point) generally disappeared first; the remainder of the series, excepting the "fixation" ball, immediately followed; the latter remained persistently visible. The lateral limits of the field were invariably, on many careful examinations, 90° outwards, 55° inwards. My object was to employ an

¹ Handbuch, d. g. A., V. I., 20.

amount of light only just sufficient to excite the retina under normal blood supply, and to ascertain whether, by making a gradually increasing pressure upon the eye, I could (by embarrassing the blood supply or otherwise) obtain a gradual centripetal contraction of the field of vision similar to that which occurs in glaucoma. As will be seen immediately I entirely failed to do so. The eye was continually fixed on the central ball of the semicircle. The pressure rod was weighted, beginning with 5 grammes, and proceeding with successive additions of the same amount. Twenty grammes being reached, it was noticed that the balls disappeared after a shorter interval than before. They disappeared in the same order as before stated; the slightest movement of the eye—viz., through perhaps two or three degrees (which could be effected without removing the pressure)—instantly restored the images, and restored, moreover, the excitability of the parts of the retina on which they had previously fallen, for they remained clearly visible when the eye again “fixed” the previous point. I need not here give all the notes which were taken down from my dictation while I was thus experimenting upon my own right eye. Suffice it to say that I successively loaded the rod up to 40, 60, 70, 80, 90, and 100 grammes. With each increase, the duration of the perception of the balls, after beginning to fix them, became shorter; that is to say, the tendency to “fatigue” of the retinal function was constantly augmented. With the high pressure of 100 grammes, the images were lost after about two seconds, but still instantly reappeared when a slight movement of the eye caused them to fall on fresh parts of the retina. In no single instance was I able to perceive that the images situated in the periphery suffered more than those at the centre of the field. *All* returned into view simultaneously when the eye moved, and all except the middle one appeared to vanish almost simultaneously. In every

instance, when the row was visible, the lateral limits of the field were as before—90° outwards, 55° inwards. If, while the images were lost to view, the pressure was removed (which could be effected instantaneously, without movement of the eye), the whole row reappeared with absolute instantaneousness. It appears probable—but I assert this only as a probability—that the loss of function thus produced was due to a *direct* influence on the nervous structure of the retina (whether on percipient elements or on fibres I can give no conjecture) rather than to an indirect influence by means of arrested circulation. The simultaneous affection of all parts of the field—and the instantaneous restoration, are, I think, in favour of this supposition. Unfortunately, the knowledge of the weight employed and of the area of the compressing surface does not indicate the height to which the intraocular pressure was raised (for physical reasons, which need not be discussed here; ⁽¹⁾ otherwise, a comparison of this latter with the normal blood pressure in peripheral arteries would help to show whether the phenomenon has any direct relation to blood pressure.

[I would observe that the experiment was one which should not be incautiously repeated. I judged the safety of proceeding with additional weight by the sensation of my eye, and felt nothing to be called pain even with the great weight of 100 grammes (= 3 ounces, about); but I did not consider the danger involved in such extreme distortion of the surface near the lens, &c., and would urge that all artificial compression should be made, as in my other arrangement, by the much safer equable pressure of a ring.]

Excavation of the optic disc. The characters of the glaucomatous cup have already been noticed. It remains only to speak of the mode of its production. In spite of some recent suggestions to the contrary, ⁽²⁾ supported, I think,

¹ See note at end of Section II. ² Arch. f. Ophth. XX., IV., 157.

on very incomplete evidence, there can be no doubt at the present time that an excess of pressure is the essential cause of the depression of the disc.

The objections to this view are two: Firstly, it is a well-established fact that high pressure may pass away and leave the disc apparently unchanged. This happens, however, only when the high pressure has lasted a few days or weeks at the outside; and in cases even of such short duration microscopic sections have shown that the first step towards excavation—viz., a retrocession of the lamina cribrosa—has already set in. Brailey has recorded some important observations on this point. He says: "Though the formation of a cup, and especially one with undermined edges, takes a long time, yet in all cases in which increased tension has been noted before excision I have found distinct evidence of it in the disc. The first result of the pressure is a pushing back of the centre of the lamina cribrosa, so that its front surface, instead of being nearly straight as in normal eyes, is markedly concave." ⁽¹⁾ Secondly, we occasionally see excavation of the disc resembling that of glaucoma, in eyes the tension of which is not distinctly in excess. I will not venture to assert that such an excavation *never* occurs in the absence of an excess of pressure, but when we know that a very slight excess, discoverable perhaps only by means of the tonometer, as in one case which came recently under my own notice will under long continuance excavate the disc, we should, I think, be very cautious in assuming the presence of a different cause.

The truth of the matter appears to be that the glaucomatous cup is not a purely mechanical result of exalted pressure, but is in part at least an atrophic condition which, though primarily due to pressure, includes vascular changes and impaired nutrition in the area of the disc and

¹ R. L. O. H. Reports, vol. IX., 208.

around its margin, which require a considerable period of time for their full development.

Figures 50 to 53 exhibit several varieties and degrees of cupping, in microscopic section.



SECTION IV.

PATHOLOGY OF GLAUCOMA.

PART I.

A history of the various opinions which have been held concerning the nature of the glaucomatous process is beyond the scope of this essay. Between the days of Hippocrates, when the green pupil was the symptom by which the disease was known, and to which it owed its name, and that epoch in the annals of our science when the ophthalmoscope of Helmholtz, in the hands of von Graefe, established the fact that the essence of glaucoma is an excess of intraocular pressure; between these points of time, almost every part of the eye—lens, vitreous, retina, choroid, sclera—was selected in its turn as the primary seat of this mysterious malady. For abstracts of the theories of the older writers, and for references to their works, Mackenzie's admirable treatise ⁽¹⁾ and the more recent article by Hermann Schmidt ⁽²⁾ may be consulted.

To Mackenzie is due the credit of having called attention to the over-fulness of the eyeball, and of having been the first to suggest a mode of treatment directed against this physical condition. He notes "an abnormal fulness of the eye to the touch," and, when discussing treatment, says, "it is not unreasonable to conclude that occasionally puncturing the sclerotic and choroid might prove serviceable, by taking off the pressure of the accumulated fluid on the retina."

¹ Diseases of the Eye, 4th Edit., p. 889. ² Handbuch d. g. A., V. 1, 72.

In the year 1855, v. Graefe, correcting the erroneous impression which he and others had acquired from the first ophthalmoscopic examinations of the glaucomatous optic disc—viz., a belief that it was prominent beyond the surrounding retina—demonstrated the fact of its excavation in a scientific manner. He saw, in this excavation, an evidence of excessive pressure, traced the action of the same agent in many of the other secondary changes, and deliberately set himself to treat the malady by reducing the internal pressure of the eye. His beneficent discovery of the curative action of iridectomy had the additional advantage of furthering the knowledge of the disease by proving that excess of pressure is the essential cause of the other symptoms. This point being gained, the lines for further investigation were at once made clear. The pathologist, disregarding the secondary changes, had now only to address himself to the question, "Whence arises the excess of pressure?" Twenty years, however, have not sufficed to give a thoroughly satisfactory answer.

Assuming that the overfulness of the eye indicated hypersecretion of the intraocular fluid, v. Graefe and Donders, each on a different line, advanced theories to account for the excessive secretory activity.

V. Graefe, taking acute glaucoma as the type of the disease, attributed the hypersecretion to a serous inflammation of the choroid. Evidence of such inflammation was hardly to be obtained, even in the so-called "inflammatory" cases, while for "glaucoma simplex" it soon appeared that some other explanation was necessary. Senile rigidity, or contraction of the sclera, and vascular degeneration, were indicated as auxiliary causes.

Donders, on the other hand, regarding the non-inflammatory form as the true type of the disease, attributed the hypersecretion to perverted nerve influence—an irritation of

certain specific nerve fibres which, according to hypothesis, preside over the secretion of the intraocular fluid. The acute attacks were regarded by him as superadded inflammation.

Bowman, writing in 1865, says of glaucoma, "It is essentially an altered balance of secretion, in which intraocular secretion, even if of only the natural fluids of the eye, is in excess of the normal quantity, and removal by the vessels does not keep pace with the effusions from them."

* * "Glaucoma is in its essence, not an inflammation, and when inflammatory, only so, as it were, by accident or complication. (1)

During the following ten years many conjectures were put forward as to the starting-point of the malady; many instances of its occurrence in connection with other local and general changes were recorded; and some important facts, already noticed, concerning its more remote etiology, were established; but, so far as I am aware, no very material addition to its pathology, as expressed in Bowman's definition, was made until quite recently.

Leber, in 1873, demonstrated, by a series of admirable experiments, that fluid in the anterior chamber passes, under slight pressure, through the ligamentum pectinatum into Schlemm's canal and the adjacent veins, and furnished an almost absolute proof that the aqueous humour normally escapes by the same channel. (2)

Max Knies, in 1876, published a series of observations, showing that in eyes which have been blinded by glaucoma, this channel—the angle of the anterior chamber—is almost invariably closed and impermeable. (3)

Adolph Weber, working independently of the foregoing, came to the same conclusion as to the path of the aqueous

¹ R. L. O. H. Reports, vol. V., p. 10.

² Arch. f. Ophth. XIX., II., 87-185. See Section IV., p. 28, &c.

³ Arch. f. Ophth., XXII., III., 163.

humour, and discovered in glaucomatous eyes obstructive changes similar to those observed by M. Knies. ⁽¹⁾

V. Wecker appears to have anticipated theoretically, though not by demonstration, something of the principle which underlies these discoveries, inasmuch as he had previously maintained that glaucoma is the result rather of diminished excretion than of increased secretion, and that iridectomy cures by re-establishing a channel of escape. ⁽²⁾

We can now place side by side three very significant facts, viz. :—

1. The aqueous humour escapes from the eye at the angle of the anterior chamber ;
2. In glaucoma the angle of the anterior chamber is compressed or closed ;
3. The only operative procedures which have a definitely curative action on glaucoma are practised at the angle of the anterior chamber.

These, in conjunction, form a strong argument against the nerve theory of glaucoma, as formerly understood ; but they do not suffice to explain the primary origin of the malady. We have yet to make clear the mode in which the compression of the angle of the anterior chamber is induced. Max Knies attributes the obstruction to a primary inflammation ; Weber, to swelling of the ciliary processes. While I believe that such changes, the latter especially, are more or less concerned in the glaucomatous process, I am, I believe, in a position to show that neither the one nor the other is the primary starting-point, and to advance a theory, founded, in great part, upon facts which are demonstrable by experiment,

¹ Arch. f. Ophth., XXII., I., 1. ² Arch. f. Ophth., XXII., IV., 209.

which affords a satisfactory explanation of the clinical features of the disease.

The foregoing paragraphs indicate, in outline merely, the position of the pathology of glaucoma at the present time. I shall now proceed to review in detail, and necessarily at considerable length, a series of facts, physiological and pathological, without which, as a groundwork, no trustworthy theory of glaucoma can be built up. For the sake of brevity and precision, I shall introduce each step by a more or less dogmatic statement of the facts, as far as they appear to be decided, and then proceed to state the evidence in its favour.

The normal intraocular pressure is equal to about 25 millimetres of mercury (i.e., 30 centimetres of water—1 inch of mercury—12 inches of water).

In certain of the lower animals the normal pressure of the intraocular fluids has been determined with approximate accuracy by means of manometrical measurements. It is equal to 25 mm. of mercury. The following figures show the results obtained by several experimenters ⁽¹⁾ :—

Gruenhagen, in rabbits	...	25	—	26·5 mm. Hg.
„ in cats	...	26	—	29 „ „
Adamük „	...	24	—	25 „ „
Wegner ...	...	18	—	35 „ „
Leber ...	...	18·5—	29·5	„ „

The differences are considerable, but we may safely conclude that 25 mm. of mercury is not far from the average pressure in these animals.

In the case of the living human eye the manometrical test is inapplicable, and, consequently, a less direct method

¹ Leber. Handbuch d. g. A., II., I., 371.

has to be employed. By applying the manometer to the eye of the dead subject, we can produce, at will, a series of variations of tension, corresponding, in each case, to a known internal pressure; by measuring the "tensions" thus obtained with the tonometer, we learn the measurement which corresponds to each degree of internal pressure; and, by comparison of these with the results of measurements of the living eye in health, we can estimate the normal pressure. The accuracy of this method is, however, not very satisfactory, if judged by its results. The following are the conclusions as to the normal pressure thus determined:—⁽¹⁾

Adolph Weber	...	...	30 — 40 mm. Hg.
Dor	...	...	37 " "
E. Pflüger	...	...	50 " "

That these figures, the latter especially, are too high anyone may convince himself, as Weber appears to have done, by simple palpation of any eyeball under such pressure.

Experiments of a similar kind with my own tonometer lead me to believe that the normal pressure is about 25 mm. Hg.—the same, that is, as in the lower animals. I employed a hollow steel needle, from a subcutaneous injection syringe, attached to a fine india-rubber tube, the other end of which was connected with a vessel of water (the tube of a glass ear syringe) suspended at any desired height above the eye under examination. A small stopcock near the lower end of the tube served to prevent regurgitation of water into the tube during the application of pressure by the tonometer. This is an indispensable precaution. The needle was passed through the sclera into the vitreous chamber; no ligature, or other means against leakage was required, for the needle filled the puncture, and was water-

¹ Handbuch, d. g. A., II., 371.

tight. An assistant closed the stopcock immediately before each application of the tonometer, and noted down the readings from my dictation. The table which follows shows the results. In the first and second cases I found some difficulty in applying the tonometer at the same point as that selected in testing the living eye—viz., 4 mm. below the corneal margin, and had to content myself with applying it about 2 mm. below. The tunics are less yielding here, therefore the impression is less deep, and the tension of the eye appears a little higher than when the measurements are taken in the usual place. In the other cases I overcame this difficulty very easily, as follows:—A fine sewing needle was passed beneath a small bridge of the conjunctiva and the denser tissue beneath it, close to the lower margin of the cornea. A second was passed in a parallel direction through the skin of the forehead. By winding a thread round these, so as to approximate them, I could rotate the eye upwards and expose the sclera to a sufficient distance below the cornea. I would observe that no movement of the eyeball is admissible after the stopcock is closed, as changes of tension are thereby readily produced. I found that near the cornea the curvature of the sclera varies but little under different degrees of pressure; I have, therefore, in each case adopted a constant value for the height of the curve, estimated from a number of measurements taken with each degree of pressure.

The results of the measurements of the tension by the tonometer were compared with those obtained by simple palpation with the fingers, and obtained corroboration therefrom. A water pressure of 60 cm. (= 50 mm. Hg.) produced a tension which appeared to myself and to two assistants to be distinctly glaucomatous, while that of 30 cm. (= 25 mm. Hg.) appeared to be about normal. In the last column of the table I have placed side by side the ordinary

symbols of plus tension, and the readings on my tonometer with which they appear to correspond :—

<i>Water Pressure, in Centimetres.</i>	<i>Mercury Pressure, in Millimetres (equivalent to last).</i>	<i>Male subject, aged 70. Tonometer impression, including sine, '69 mm.</i>	<i>Female subject, aged 4. Tonometer impression, including sine, '66 mm.</i>	<i>Male subject, aged 48. Tonometer impression, including sine, '55 mm.</i>	<i>Female subject, aged 23. Tonometer impression, including sine, '60 mm.</i>	<i>Male subject, aged 32. Tonometer impression, including sine, 60 mm.</i>	Tonometer impressions corresponding to symbols ordinarily used.
30 cm.	25 mm.	'70 mm.	'74 mm.	'82 mm.	'76 mm.	'90 mm.	'95 mm. } Tn.
							'70 " } T+1 ?
60 "	50 "	'52 "	Needle afterwards entered lens, and got plugged.	'56 "	'50 "	'53 "	'60 " } T+1
90 "	75 "	'38 "		'44 "	—	—	'50 " } T+2
120 "	100 "	'36 "		'35 "	—	—	'40 " } T+3
150 "	125 "	'27 "		'24 "	—	—	

The intraocular pressure is much lower than the blood pressure, but is intimately dependent upon it.

The pressure of the blood in the arteries is equal to about 150 mm. mercury (= 6 inches Hg. or 6 ft. water), subject to considerable variations, depending upon the condition of the heart and vessels, the quantity of blood, respiration, muscular exercise, &c. The mean pressure is slightly lower in the small arteries than in the main trunks, and lowest in the arterioles directly leading to the capillaries. It increases during the systole, falls during the diastole; increases during expiration, falls during inspiration; it is greatly increased in interruption to respiration, by the arrest of the non-aerated blood in its passage through the capillaries. ⁽¹⁾

There is evidence to show that every fluctuation in the pressure of the blood-stream through the eye alters the

¹ Flint's Physiology.

pressure of the intraocular fluids against their envelopes, the changes in the two cases being similar in kind but different in degree.

Compression of the aorta immediately below the diaphragm causes, in the lower animals, immediate and marked rise of the intraocular pressure, by increasing the volume and the pressure of the blood flowing to the head. During the continuance of the compression and of the high ocular pressure, as indicated by the manometer, the mercurial column exhibits oscillations synchronous with the pulse; on the removal of the compression the pressure speedily falls, nearly, but not quite, to the normal level. Again, a rise of pressure in the eye has been observed during general contraction of the small arteries of the body, and consequent high arterial blood pressure, induced by artificial irritation of the vaso-motor system. On the other hand, ligature of the carotid, by reducing the local blood supply, lowers the tension of the corresponding eye; the same result has been observed under conditions which lower the general blood pressure—*e.g.*, failure of the heart's action in curarised animals; considerable losses of blood; poisoning by opium, which dilates the small vessels, and thereby lowers the blood pressure; and, during depression of the heart's action, by irritation of the pneumo-gastric nerve. ⁽¹⁾

Many of the observations just cited were made under such extreme conditions as to be hardly applicable to the question of physiological variations of the ocular tension, but, apart, from these, there is reason to believe that, under comparatively normal conditions, fluctuations in the blood pressure are accompanied by similar changes in the tension of the eye. In anæmic girls the tension has appeared to me to be perceptibly below the average in almost every case that I have examined, but on such a point one is

¹ Leber. Handbuch d. g. A., II., I., 371.

liable to self-deception. Tonometrical measurements in several cases of this kind have given a corresponding result. An old man insufficiently fed, and exhausted by over-walking, applied at the hospital on account of incipient cataract. His pulse was small and compressible, his face very pale, his skin covered with cold perspiration. The tension of the eyes was below the normal average, as measured by the tonometer. Three days of enforced rest in the hospital, with meat and beer, brought the colour to his cheeks, strengthened the pulse, and greatly improved his general condition. A distinct rise of tension was discovered to correspond with these changes. The same sequence of events occurred twice, at different times, in this patient. One or two other instances of the same kind have occurred to me during my investigations with the tonometer. The variations are small, and are hardly beyond the limits of possible error, but, taken together, they point to the influence of varying blood pressure. (¹)

The rise of the intraocular pressure which occurs when the blood pressure is increased, as in the experiments on animals before mentioned, depends upon two separate factors—viz., an increased fulness of the intraocular vessels, by reason of which they encroach upon the space bounded by the ocular tunics, and an increased flow of secretion into the chambers. It is hardly possible to distinguish between the effects of these two factors, inasmuch as, under normal conditions, they vary together—the amount of a secretion being usually in proportion to the blood supply; but it must not be overlooked that this rule does not invariably hold good. It has been proved in the case of certain secretions—*e.g.*, the saliva—that the amount is not dependent only upon the dilatation of the vessels, but is under the control of specific nerve fibres acting directly on the secreting cells, so

¹ See Case 37, Section VII.

that, under certain circumstances, secretion may be arrested even during wide dilatation of the afferent arteries.

Venous obstruction in the neighbourhood of the eye raises the intraocular pressure. ⁽¹⁾ Experiments have proved that ligature of the veins which receive the blood from the interior of the eye may cause a rise of the internal pressure to more than twice the normal height. Obstructions to the venous current at a greater distance from the eye have much less influence; compression of the large veins of the neck, and even ligature of the superior vena cava, have been found to raise the ocular pressure but little; in the latter case especially, this may be due to the diminished return of blood to the heart, and the consequent weakening of its action.

The intraocular pressure is subject, in some degree, to the influence of the fifth nerve. Artificial irritation of the fifth nerve in certain parts causes a marked rise of the intraocular pressure. Von Hippel and Gruenhagen, in a long series of experiments upon animals, invariably obtained this result, when the origin of the nerve in the medulla oblongata was irritated by means of needles transmitting an induced current. ⁽²⁾ The animals were curarised; the intraocular pressure was measured by a manometer connected with the anterior chamber. The rise of pressure was much greater than that produced by compression of the abdominal aorta, and, on the removal of the needles, it remained in excess much longer than after removal of the compression in the latter case. Moreover, irritation of the fifth nerve *during compression of the aorta* produced a marked additional rise. For example, in an experiment on a cat—

Initial pressure	...	...	...	31 mm. Hg.
After compression of aorta...	...	...	...	81 „ „
After irritation of fifth	...	...	...	200 „ „

¹ Leber. Handbuch d. g. A., II., I., 372. ² Arch. f. Ophth., XIV., III., 219.

During this excessively high pressure the carotid was felt to be enlarged, and the mercury in the manometer pulsed strongly. The intraocular pressure thus obtained, be it observed, was much higher than the normal average blood pressure, and itself indicates a strong determination of blood to the eye. The sequence of events was not altered by the previous application of atropine to the eye, by the performance of iridectomy some weeks beforehand, or by the division of the cervical sympathetic. A fall of pressure sometimes preceded the rise when the sympathetic was entire, presumably due to irritation of that nerve. ⁽¹⁾ Adamük also obtained a marked rise of the intraocular pressure, by irritation of the fifth nerve within the skull. Again, superficial irritation of the eye itself by mechanical means, and by certain irritating substances, notably nicotine, has been observed to raise the intraocular pressure in association with injection of the blood-vessels. Of these facts there is no doubt, but, as yet, the precise nature of the influence exerted by the fifth nerve over the tension of the eye is undetermined. The question still at issue is this: Does the trigeminus contain *special* fibres which regulate the secretion of the intraocular fluids by direct action on the secreting cells, like those which are known to be connected with the salivary glands, or is its influence over the tension of the eye an indirect one acting through the vaso-motor system? Although the proved existence of specific secretory fibres in the salivary gland is a point in favour of those who hold the former view, it is worth noting that the secretory requirements are apparently different in the two cases. In the digestive organs secretory activity is required at certain times only, and in reflex response to certain sensory impressions, while in the eye, on the contrary, uniformity in the flow appears to be essential to the well-being of the organ.

¹ Leber. Handbuch d. g. A., II., I., 378.

The results of experiments of an opposite nature—viz., by destruction of the fifth nerve—give no decisive information. Immediately after division of the nerve, the intraocular pressure rises, presumably through irritation of the fibres; later it falls but, according to von Hippel and Gruenhagen, it falls only when neuro-paralytic changes in the cornea set in, by which, they suppose, the escape of the aqueous is favoured; while Snellen and Donders have witnessed a steady fall when corneal changes are completely absent. On this point it should be noted that Charcot disputes the inferences of Meissner and others as to the changes which follow destruction of the supposed trophic fibres, and urges that such lesions are not paralytic but irritative.

An influence of the sympathetic nerve over the intraocular pressure has not been definitely established. Irritation of the sympathetic is stated by von Hippel and Gruenhagen, and by Adamük, to cause first a slight rise and then a fall of the intraocular pressure. The following is the hypothetical explanation of these changes. The function of the sympathetic, as regards the eye, is to contract the intraocular vessels, and also the unstriped muscular fibre in the orbit. By the latter action the orbital space is diminished, the eye pushed forwards, and its internal pressure raised; by the former, the volume of blood within the eye is diminished, and the intraocular pressure lowered. The changes are slight, and the explanation hardly decisive. ⁽¹⁾

Accommodative movements appear to cause no change of pressure either in front of or behind the lens. This has been tested in several ways—in curarised animals by galvanism of the ciliary ganglion, and of the ciliary region of the eye;

¹ Leber. Handbuch d. g. A., II., 1., 376.

and by galvanism of freshly excised eyes in which contractility of the pupil still remained. A manometer was employed in each case to test the pressure; Adamük, indeed, employed two manometers, connected one with the aqueous, one with the vitreous chamber. In no case was any change of pressure observed, and in the latter the pressure was observed to be equal in the two chambers. Monnik tested the tension of the eye with the tonometer in seven persons during strong accommodative effort, and in no case found it different from what it was during relaxed accommodation. The eye under examination was caused to maintain the same position under each condition, so as to avoid any change due to action of the internal rectus muscle during convergence. The advance of the anterior surface of the lens during accommodation is compensated for by the retrocession of the periphery of the iris. This movement of the iris is very readily seen in the dead subject, if the whole lens be made to advance by pressing on its posterior surface with a needle, introduced near the equator of the eye. This I have witnessed many times.

It is probable that the pressure in the aqueous chamber differs but little, if at all, from that in the vitreous chamber. Before stating my grounds for this assertion I will give, almost in his own words, the opinion advanced by Adolph Weber, which is at variance with it. "The suspensory ligament, together with the lens, constitute a firm physiological as well as anatomical partition" (between the vitreous and aqueous chambers). "In the dead eye this partition will bear a pressure of 50 to 60 mm. of mercury, a pressure double that natural to the eye. The arrangement of this partition is such that, under normal conditions, it bears and neutralises a portion of the pressure generated in the vitreous chamber; the tension of the cornea is, on that account, lower 'by several tonometer degrees' than

that of the sclera." (1) I believe this view to be erroneous, and, as the question bears intimately on the pathology of glaucoma, as I understand it, it must be closely considered.

An inequality of pressure before and behind the partition formed by the lens and its suspensory ligament, can only exist on one condition—namely, *that the partition is incapable of yielding, towards the lower pressure, to an extent sufficient to equalise the pressures upon its two surfaces.* Thus, if the vitreous pressure exceed the aqueous pressure, the lens must be incapable of such an advance towards the cornea as would establish equality. Seeing that the aqueous humour is incompressible, such an advance would manifestly be small. Now, if the partition really possess this power of resistance, it must be in one or other, or both, of two ways. Either the tension of the suspensory ligament is in itself sufficient, or it is supplemented by the pressure of the iris against the anterior lens surface, or the latter is in itself sufficient. I cannot find that these requisite conditions exist.

First, as regards the resistance offered by the lens and its suspensory ligament independently of the iris. Weber has ascertained that in the dead eye this partition will, in the absence of support from the cornea, sustain without rupture a pressure on its posterior surface equal to 50-60 mm. of mercury; but obviously this fact proves nothing as to its ability, when in its normal position, to sustain an excess of pressure on its hinder surface *without advancing.* My own experiments prove that a very slight excess of pressure in the vitreous chamber over that in the aqueous chamber will drive the partition forward and apply it to the posterior surface of the cornea.

A hollow needle, connected by a tube with a vessel of water, was introduced into the anterior chamber, another into the vitreous chamber.

¹ Arch f. Ophth., XXII., IV., 216.

Each reservoir stood at first at a height of 30 cm. above the eye (= 25 mm. Hg. = about the normal intraocular pressure). On raising the vitreous pressure to 40 cm. water (= 33 mm. Hg.) the lens at once moved forward, the contents of the aqueous chamber escaped into the tube connected with it, and *the whole of the anterior surface of the iris was applied to the posterior surface of the cornea*. When the vitreous pressure was raised only to 35 cm. water (= 29 mm. Hg.)—that is, when it was only 5 cm. water (= 4 mm. Hg.) in excess of the aqueous pressure—the lens still advanced sufficiently to apply the periphery of the iris against the cornea, though a narrow circle surrounding the pupil remained free. In the latter case, even, I believe that the incomplete contact of the iris with the cornea was not due to the resistance of the “partition” to further advance, but to the intervention of the needle between the surfaces of the lens and the cornea. When the pressure in the vitreous was a little higher, it sufficed to imbed the needle in the surface of the lens, as I discovered by subsequent examination of the latter. These results were seen with the utmost precision when a coloured fluid was used instead of water in the anterior chamber.

A full account of these experiments is given later. They were made upon human eyes and upon the eyes of pigs, in each case very shortly after death. ⁽¹⁾

It cannot be objected that the experiment was vitiated by the open communication between the anterior chamber and its reservoir; this condition was essential, inasmuch as my sole object was to ascertain the behaviour of the “partition” when subjected to unequal pressures before and behind. The difference between the two pressures was not affected by the advance of the “partition,” for the level of the water in the reservoirs was not sensibly affected by the slight flow *from* the one, or *into* the other. Had the anterior chamber been closed, the pressures on the two surfaces of the partition would have been equalised by an imperceptible advance of the lens, and the point at issue would not have been decided.

It appears, then, that the “partition” is so freely mobile in an anterior direction, as to be incapable, *per se*, of retaining its normal position under any discoverable excess of pressure behind it; in other words, it is incapable of neutralising any discoverable excess of pressure in the

¹ Pages 123 to 134.

vitreal chamber. Secondly, as regards the influence of the iris on the position of the "partition," it appears to me that this must be set down as extremely minute, inasmuch as it has never been shown that the lens advances during paralysis of the sphincter of the pupil due to lesion of the third nerve. Did the lens advance to a perceptible extent under such circumstances, the refraction of the eye must necessarily be altered in the direction of myopia, which it never is. Finally, I would cite, in opposition to Weber's theory, an interesting clinical observation recorded by Ad. Mohr, first assistant surgeon in his own clinique. ⁽¹⁾ While experimenting with eserine, he found in some cases that, by the extreme action of this drug, the ciliary muscle was so much contracted as to draw forward and slacken the zonula "beyond the limits of the elasticity of the lens," so that the zonula became loose and the lens hung slack, causing a visible irido-donesis (tremulous iris). Now, this tremulous condition of the partition could not exist if there were the slightest inequality of pressure on its two sides, and yet, according to Weber, it is precisely under these circumstances—viz., maximum effect of eserine—that the excess of pressure in the vitreal chamber is at its greatest.

I do not assert that the pressures in the aqueous and vitreal chambers are *precisely equal*, but I think it is certain that, if any difference exists, it is extremely small—certainly too small for tonometrical demonstration. Measurements with the tonometer upon structures differing so much in curvature and in texture as the cornea and sclera, are, I think, hardly to be compared; certainly such evidence is not to be accredited in opposition to that given by the manometer, which, as already stated, indicated to Adamük an equality of pressure in the aqueous and vitreal chambers,

¹ Arch. f. Ophth. XXIII., II., p. 166.

and in opposition to that which is afforded by the experiments just cited. ⁽¹⁾

The effects of atropine and eserine upon the internal pressure of the healthy eye are not positively determined.

¹ *June, 1879.*—When an incision is made in the cornea, as, for example, in the performance of iridectomy, more or less of the aqueous fluid escapes through it on the withdrawal of the knife; the lens and iris advance to a corresponding extent, and the form of the cornea remains practically unchanged. In exceptional cases, however, a different change occurs. As the aqueous escapes, the cornea collapses, the pressure upon its inner surface being apparently no longer sufficient to maintain its curvature. This collapse of the cornea appears to take the place of the advance of the lens and iris, and would seem, at first sight, to support Weber's statement that the "partition" is able to support the whole of the vitreous pressure, and to refute the opinion which I have put forward as to the practical equality of the aqueous and vitreous pressures. But let us examine the matter more closely. The advance of the lens and iris, which we ordinarily witness, depends upon the pressure of the vitreous; and, since the vitreous is practically incompressible and inextensible, it implies a certain degree of elastic contraction in the sclera, or of pressure upon it by the contents of the orbit. For, if we imagine the case of an eye with absolutely inelastic sclera, subjected to no external pressure, it is obvious that, however high the vitreous pressure may be, an infinitesimal advance of the lens will reduce it to *nil*, precisely as though the globe were made of cast-iron. I believe that the collapse of the cornea occurs just in those cases in which the elasticity of the sclera and the external pressure upon the globe are at a minimum—namely, in aged persons with cavernous hollows between the globe and the orbital margin. I have never, so far as I can remember, seen such a collapse in a young or even in a middle-aged person, or in an elderly person of stout habit of body with a tendency to prominence of the eyes. Since my attention has been especially directed to the matter, I have seen it four times, during the performance of preliminary iridectomy in cases of senile cataract. All four patients were aged, thin, and hollow-eyed. Arlt observes that loss of vitreous during cataract extraction is not to be feared when the cornea collapses, the eye is deep set, and the patient is perfectly tranquil. (*Handbuch d. g. A.*, III., 302.) My own experience of the truth of this statement, and conversely of the liability to rupture of the suspensory ligament in stout people, with turgid veins and prominent eyes, is very decided. The absence of this danger in the former case is due, I believe, to the fact that an extremely small advance of the lens suffices at once to lower the vitreous pressure in an unusual degree—to a degree, in fact, which is actually neutralised, and borne with comparative safety, by the suspensory ligament. That the suspensory ligament has, as Weber asserts, a considerable amount of intrinsic strength is certain; the point for which I contend is, that it cannot neutralise any appreciable excess of pressure on its posterior surface *without advancing from its normal position.*

They have been closely studied, but the conclusions arrived at are not in agreement. Adolph Weber measured the tension of cornea and sclera before and after the use of atropine, and before and after the use of eserine. ⁽¹⁾ He concluded that atropine lowers the vitreous and raises the aqueous pressure, and explains these effects by the slackening and tightening of the partition between the chambers. As already stated, it appears to me unsafe to attach an accurate value to a comparison of such tonometrical measurements, and, as regards the explanation given, I can not reconcile it with physiological facts. The iris is, according to Weber, the chief agent in neutralising the pressure of the vitreous upon the lens and its suspensory ligament, and is able sometimes, when tense, "to support the whole of the vitreous pressure"! As evidence of this, he cites the production of irido-donesis by the extreme action of eserine already referred to. Now, it appears to me that any undulating movement in the partition between the two chambers is physically impossible, unless the pressure on its two sides is equal. The slightest excess on either side would cause a displacement towards the other chamber, which would only be arrested when the membrane was put upon the stretch, or when the pressure on its two surfaces was equalised. We must, I think, conclude that any discoverable changes of pressure which these drugs produce are not to be explained in this mechanical manner. It is not impossible, however, that they may effect such alterations by their influence on the blood-vessels. Here again, unfortunately, we are met by such diverse observations that no conclusion is justifiable. Leber reviews the question with the result that it is probable, though not proved, that atropine slightly lowers the tension of the healthy eye, but not in excess of those variations which frequently occur under normal

¹ Arch. f. Ophth., XXII., IV., 216.

conditions; and that eserine (Calabar) probably raises it slightly, though this is contrary to the evidence of von Hippel and Gruenhagen's experiments. Its action is possibly only the same as that of any other irritant. ⁽¹⁾

*In the glaucomatous condition the action of these drugs upon the tension of the eye is in some cases very marked; atropine raises, eserine reduces it. Instances of these effects are given in another section, and the *modus operandi* is discussed in connection with the theory of glaucoma.* ⁽²⁾

In the foregoing paragraphs, I have endeavoured to analyse our knowledge of the intraocular pressure in its more general relations; I shall next discuss the physiological processes upon which it is immediately dependent—namely, the secretion and absorption of the intraocular fluids.

The aqueous humour is secreted by the ciliary processes, and perhaps to a smaller extent by the iris. ⁽³⁾ The highly vascular structure of the uveal tract in the ciliary region, its peculiar plicate arrangement, and the relation of this part to the aqueous and vitreous chambers indicate a special adaptation to secretion. The anterior ends of the processes present a free surface to the posterior aqueous chamber; fluid escaping from them passes into this space, and thence through the pupil into the anterior chamber. The effects of disease confirm this view. When the margin of the pupil adheres throughout its whole circle to the capsule of the lens, so as to cut off the passage of fluid from the posterior to the anterior chamber, the iris soon becomes bulged forward by the imprisoned secretion, and the anterior chamber is annihilated. The iris, if a secreting organ at all, is of subordinate importance. It has not the peculiar plicate

¹ Handbuch d. g. A., II., I., 375. ² Page 153.

³ Leber. Handbuch d. g. A. II., I., 380.

arrangement seen in the ciliary processes, and it may be completely atrophied, or even entirely absent, without any discoverable deficiency in the intraocular fluids. A softened state of the eye, indicative of diminished secretion, is, on the other hand, constantly found to be associated with atrophy, or inflammatory disorganisation of the ciliary processes. ⁽¹⁾

The aqueous humour escapes from the eye by filtering through the meshes of the ligamentum pectinatum into Schlemm's canal, and the veins in connection with it. This important fact we owe to Leber's admirable "Studien über den Flüssigkeit's-wechsel im Auge," ⁽²⁾ a work the value of which has been established by the award to its author of the "v. Graefe prize," by the Ophthalmological Society of Heidelberg. Before describing the way in which the discovery was made, a brief account of the anatomy of the region in question is desirable.

The nature of the circular sinus discovered, or rather rediscovered, by Schlemm is now known to differ from that originally attributed to it, but the old name Schlemm's canal is so concise and distinctive that it is more convenient to retain it than to substitute for it either of its recent synonyms—*circulus venosus ciliaris*, *plexus venosus ciliaris*, *sinus venosus corneæ*.

Schlemm's canal is situated at the anterior limit of the sclera, and close to its inner surface, a little anterior and external to the origin of the ciliary muscle. ⁽³⁾ Its structure differs somewhat at different parts of the circle, and in different eyes. It is a flattened, thin walled vein, generally single, and measuring nearly a quarter of a millimetre in its greatest diameter. It is often accompanied by one or more smaller veins, and sometimes divides into two or more trunks, which, by frequent anastomosis, form a plexus instead

¹ See also page 137, &c. ² Arch. f. Ophth., XIX., II., 87-185.

³ Leber. Handbuch, d. g. A., II., 327.

of a single canal. Venous twigs join it from the anterior part of the ciliary muscle, and from the marginal plexus of the cornea. It is separated from the angle of the anterior chamber by the meshes of the ligamentum pectinatum. For the last-named structure, again, I prefer to retain the old name, rather than to use the term Fontana's space, as several recent writers do. The latter name was originally applied only to the larger meshwork of this structure present in certain of the lower animals, and is inconvenient, inasmuch as it indicates not a part but a space, which, moreover, in the human eye is not a solitary well-defined space. The belief in a direct communication between the anterior chamber and the veins which constitute Schlemm's canal is no longer tenable. Leber has proved that the passage of fluid from the one to the other is not by open channels, but by osmosis. My own experiments support the same conclusion.

Leber experimented with freshly excised eyes of dogs, pigs, and cats. A fine canula was introduced into the anterior chamber, and through it various solutions were injected under a series of different pressures. He found that such solutions as will pass through animal membranes rapidly appeared in the episcleral venous plexus, the marginal loops of the cornea, and the conjunctival veins: for example, a neutral carmine solution rapidly caused a pink injection of these vessels. Colloid solutions, on the other hand, which will not permeate animal membranes, caused no injection. A solution of Prussian blue, obtained by chemical reaction, and which presented no visible particles under the microscope, could not be made to pass from the anterior chamber into the episcleral vessels, except occasionally, at isolated spots, when the pressure was very high. Prussian blue, as is well known, will not pass through animal membranes, and the exceptional instances of injection were doubtless due to rupture of tissue. The opposite behaviour

of these two solutions appeared to prove the absence of an open communication between the chamber and the vessels. A very beautiful crucial test confirmed this. A mixture of the two solutions, violet in colour, was injected. The filtration apparatus of the eye separated them, although, under the microscope, no particles were visible. The carmine passed through, causing a pink injection of the vessels; the blue was arrested, and was afterwards found by the microscope collected in the meshes of the ligamentum pectinatum. Fig. 30 shows a section from the eye of a pig on which I performed a similar experiment. ⁽¹⁾

A second observation of much importance for the subject under consideration was, that in the fresh eye no fluid permeates the cornea. From the list of conclusions with which Leber sums up his observations, I will quote those only which have a bearing upon the pathology of glaucoma. I retain the original numbers:—

1. "The anterior chamber has no direct communication with the blood-vessels, neither is such a communication proved to exist with the lymphatics."
2. "In the dead eye, fluid injected into the anterior chamber filters readily into the veins of the circulus venosus and the iris and the vortex veins." ⁽²⁾
3. "If the injected fluid is coloured with diffusible colouring matter (carmine), a visible injection

¹ Fig. 30., slide. It should be stated that the Prussian blue solution which I used was, through an oversight, not prepared by chemical reaction, as it should have been according to Leber's method, but by trituration and subsequent filtration. It therefore probably contained *particles*, which Leber's solution did not, and was, therefore, no true test of the point at issue. Corroboration of Leber's experiments can, however, hardly be considered necessary.

² In my experiments in which fluid was injected simultaneously into aqueous and vitreous chambers, and especially when the vitreous pressure was the higher of the two, no escape occurred through the vortex veins. See page 126, &c.

of these vessels occurs; if with colloid colouring matter (Prussian blue), no injection occurs if there is no rupture of tissue."

4. "It is probable that, during life, the aqueous humour passes outwards by the same channel."
5. "Artificial increase of the intraocular pressure during life, produced by injection into the anterior chamber, soon disappears, unless the pressure is considerably raised; in the latter case the excess persists for a time at least."
4. "During life, and immediately after death, the cornea, even when its anterior epithelium is removed, does not permit the passage through it of the smallest quantity of the aqueous humour."
14. "The agent by which the living cornea is enabled to retain the aqueous humour is the posterior epithelium. If this is removed, the corneal tissue swells, and allows the passage of fluid through it."

Glaucomatous eyes, when microscopically examined after excision, give evidence of changes at the periphery of the anterior chamber, such as would obstruct the escape of the aqueous humour. The changes which this region undergoes during life, as indicated by the microscopical appearances, are:—

1. Narrowing of the angle of the anterior chamber, with obliteration of the meshes of the ligamentum pectinatum.
2. Contact of the periphery of the iris with the periphery of the cornea.
3. Adhesion of the periphery of the iris with the periphery of the cornea.

The very great majority of the glaucomatous eyes which have been examined with especial regard to these changes have been found to present the peripheral adhesion of iris and cornea. ⁽¹⁾ In a very few cases, in which there has been an undoubted excess of tension before the excision of the eye, this adhesion has been absent. As will shortly appear, it is a change which takes time for its development, and which, therefore, could hardly be found in eyes in which the previous excess of tension has been of very short duration. A narrowing of the angle of the chamber, and even a contact of the iris with the cornea, though existing during the life of the eye, may be undiscoverable after its removal; hence, the absence of marked change in this region in a very small number of the cases examined, does not invalidate a theory of glaucoma in which such changes during life are held to be of essential importance.

In evidence of the very general presence of such changes, I exhibit sections and drawings ⁽²⁾ of nine glaucomatous eyes, including specimens of primary glaucoma, acute and chronic; and of secondary glaucoma, associated with synechia, posterior and anterior—in the latter case traumatic; buphthalmos; intraocular tumour; and detachment of the retina, with hæmorrhage. In every case the obstruction of the angle of the anterior chamber is very evident. The conditions are seen in figures 11 to 23. An individual description is given in another place.

I also exhibit four specimens, with sections and drawings, in which closure of the angle of the anterior chamber is present *without glaucoma* (figures 24 to 27). To these I shall have to direct especial attention, inasmuch

¹ Max. Knies, Arch. f. Ophth., XXII., III., 163; Ad. Weber, XXIII., I., 1; Brailey, R. L. O. H. Rep., IX., 220.

² The original drawings, the microscopic sections, and, in most cases, the specimens from which they were obtained, are now in the museum of the Royal College of Surgeons.

as they appear, at first sight, to be opposed to the obstruction-theory of glaucoma. A more careful examination of the conditions which they present will show, however, that, so far from being in opposition to this theory, they afford material evidence in its support. The examination of these points in detail must follow the general explanation of the theory.

The fact of the peripheral adhesion of iris and cornea in glaucomatous eyes is not, strictly speaking, a discovery of the last few years. It has long since been noted here and there in ophthalmic records, but, until Leber demonstrated the importance of this region as a filtration channel, its real significance was naturally overlooked. To Max Knies belongs the credit of having been the first to point to it as an obstruction to the normal outflow of the intraocular fluid, and a cause of increased intraocular pressure. ⁽¹⁾ Ad. Weber observed the same thing independently, and published, immediately after Max Knies, the statement that narrowing or obliteration of this channel is the essential cause of glaucoma. ⁽²⁾

It cannot be doubted by anyone who accepts the discoveries of Leber as facts, that compression of the ligamentum pectinatum and peripheral adhesion of the iris and cornea, when once established, must tend to maintain an excess of pressure within the eye, so long as the secreting organs retain their function; but, granting that this is so, we need not necessarily accept these changes as the essential cause of glaucoma before we know the period of the disease at which they arise, and the manner in which they are brought about. Especially must it first be proved that they are not a *result* rather than the *cause* of an excess of pressure. Max Knies assumes—as, I believe, on insufficient grounds—that a circumscribed inflammation of the tissues in the vicinity of Schlemm's canal is the starting point of these obstructive

¹ Arch. f. Ophth., XXII., III.

² Arch. f. Ophth., XXIII., I.

changes, and is, therefore, to be considered the true cause of glaucoma. He points to the activity of the filtration process carried on in "Fontana's space" and the corneo-scleral junction, as likely to render this region liable to idiopathic inflammation, and he considers that certain of the minor symptoms of glaucoma hitherto viewed as effects of exalted pressure—*e.g.*, opacity and anæsthesia of cornea, paralysis of iris, shallowness of anterior chamber, paresis of accommodation, hyperæmia of anterior ciliary viens—are to be attributed, like the rise of pressure, to "an inflammatory induration of the neighbourhood of Schlemm's canal." He believes that he has found evidence to support this opinion in a cellular infiltration, or a cicatricial retraction of the tissues in this region, in the glaucomatous eyes which he has examined.

Microscopical examinations can hardly prove or disprove this theory. Supposing the cellular infiltration and the inflammatory adhesion to be discoverable in every case, which in my specimens they are not, the question must still remain: Is the inflammation the cause, or the consequence, of the disturbed relations of the parts? In certain of my specimens, the adhesion of the iris with the cornea is not directly continuous with the occluded angle of the chamber, but exists only where the enlarged apex of a ciliary process pushes the iris forwards against the cornea, while between this point and the ligamentum pectinatum the iris and cornea are still separated by a narrow free space. This condition is hardly consistent with the idea of an idiopathic inflammation spreading from Schlemm's canal or its vicinity as a focus. Clinically, we can obtain, in the majority of cases of glaucoma, absolutely no evidence in favour of the existence of inflammation. The strongest argument, however, which I have to offer in opposition to this theory of Max Knies is the substitution for it of another, which appears to me to stand upon firmer ground.

Adolph Weber, observing especially the relative positions of the parts in question, comes to the conclusion that the periphery of the iris is driven forward against the cornea by the pressure behind it of the apices of the ciliary processes. It can hardly be doubted that in most cases this actually occurs, for we find the periphery of the iris bowed forward in an unusual sweeping curve, into the concavity of which the enlarged ciliary process is accurately fitted (fig. 17). The apices of the processes being bounded posteriorly by the suspensory ligament with which they are in contact, must, if they swell, advance forwards and inwards, and, in so doing, carry the periphery of the iris before them. Such, according to Weber, is actually the process by which the obstructive changes are originated. He believes that swelling of the ciliary processes is the direct cause of all the cases of primary glaucoma. This theory, when judged by the relations of the parts as seen under the microscope, appears to be extremely well founded; but, when we regard it from a clinical point of view, and ask for the causes which bring about swelling of the ciliary processes, it is, I think, less satisfactory. These causes are to be found, according to Weber, in a variety of general changes of slow development, especially "in diseases which are associated with long-continued lowering of arterial pressure—*e.g.*, mitral disease, and valvular disease of the right side of the heart, or other circumstances which dispose to passive hyperæmia, such as emphysema, plethora, menopause, suppression of habitual hæmorrhage; and, further, such occurrences and occupations as lead to temporary venous dilatation, such as repeated and persistent attacks of coughing, frequent and severe parturition, trades like that of the smith, and so forth." Unfortunately we cannot, during life, ascertain the effect of these maladies and conditions upon the size of the ciliary processes; but if we are to believe that these organs are liable to swell under the various

vascular conditions which Weber enumerates, and that their swelling narrows the filtration channels, some excess of ocular tension should surely be very common among the thousands of old people who suffer from bronchitis and emphysema with each return of winter; and, amongst the many severe cases of cardiac disease constantly in our general hospitals, an attack of glaucoma should not be very rare. Though for some years past holding a position in which any such case occurring in the physicians' wards of a general hospital would infallibly have been brought under my notice, I have never seen a case of glaucoma associated with any very pronounced disease of the heart or vascular system, such as advanced mitral or aortic disease, aneurism, &c. Some months since I began to take pulse-tracings in every case of primary glaucoma that I met with, but after doing this in seven or eight cases without obtaining any evidence that the vascular conditions were either better or worse than is usual in persons beyond middle life, I abandoned the investigation for want of time. Again, seeing that, under ordinary circumstances, the effect of a lowering of arterial pressure is a lowering of intraocular pressure, I should hesitate to accept Weber's theory that long-continued depression of arterial pressure is one of the causes of glaucoma. In a case of congenital cyanosis, in which the whole venous system was engorged, and the vessels of the optic discs very numerous, large, and blue, the tension of the eyes I found to be strictly normal.

Having carefully studied Weber's admirable analysis of the physical conditions present in glaucoma, and being convinced, both by his arguments and by my own microscopic sections, that obstructive changes at the angle of the anterior chamber are an essential feature in the disease, I proposed to myself to ascertain, if possible, whether such obstruction might not be artificially produced under

conditions which would preclude its dependence upon swelling of the ciliary processes—namely, in the excised eye.

The following case suggested to me the first point to be tested. I performed iridectomy in a case of advanced chronic glaucoma; ⁽¹⁾ the anterior chamber was thereby totally and permanently annihilated, the iris and lens remaining in close contact with the cornea; the tension rose to more than its former height, the remnant of vision was lost—in short, the glaucoma was aggravated instead of being relieved. The unfavourable result appeared to me to be due to the total obliteration of the angle of the anterior chamber by the forcible advance of the iris and lens, whereby all escape of fluid was arrested (the wound closed almost at once, and appeared to transmit no fluid); any secretion now poured into the eye must enter the vitreous chamber, and make matters worse by intensifying the pressure behind the lens.

Acting on this idea, it was not difficult to devise a means by which these conditions might be imitated in the excised eye, and their ability to arrest the escape of fluid from the anterior chamber put to the test.

It was necessary, in the first place, to know approximately the height of the normal intraocular pressure. As already recorded, my experiments with the tonometer on the dead subject indicated 30 cm. water (= 25 mm. Hg.) as about the normal average. ⁽²⁾ This result agreed completely with the estimate formed by finger-pressure, and corresponds exactly with the results of manometrical measurements in certain of the lower animals. I therefore felt justified in assuming it to be a fair standard by which to regulate the pressures employed in my experiments. I used the eyes of pigs, removed in most cases immediately after death, and *before scalding*. In several crucial confirmatory experiments very

¹ Case 20, Section VII. ² See pages 97—100.

special precautions were taken to avoid the possibility of post-mortem changes. ⁽¹⁾ The experiments were subsequently confirmed on the human eye, very shortly after death. For the sake of clearness, I shall here rearrange the experiments in such a succession as will best elucidate the points at issue. They were made in a more irregular succession, generally two at a time, and repeated for confirmation at various intervals, as fresh questions arose. Many are here omitted.

The apparatus employed was simple. A wooden stand (fig. 10) was prepared, which would receive two eyes at a time. They were fixed by passing the stumps of the optic nerves through the holes in the brass plate, and transfixing with sewing needles; thus, ligaturing the nerve, which would have vitiated certain of the experiments, was unnecessary. For the introduction of fluids into the chambers under known pressures, I utilised the pressures of the columns of fluids, instead of the more complicated manometer. The tube of a glass ear-syringe was suspended by a string and counterweight from a nail in the wall above my table. A flexible tube, several feet long, was attached to the narrow end of the glass tube, and ended below in a small stopcock, to which again was attached a shorter flexible tube, carrying in its extremity a hollow steel needle; the blunt end of the needle was made bulbous with sealing wax, so that it was tightly grasped by the india-rubber tube, but could be very readily removed and replaced without the use of a ligature. Four such tubes and reservoirs were hung side by side on a centimetre scale, indicating the height of the column above the eye.

In order to obtain a visible injection of the vessels clearly distinguishable from blood, I used a solution of anyline purple ⁽²⁾; anyline green, which I also tried, passes too readily, and stains the tissues too diffusely.

¹ See page 129.

² I believe that the technical name of this dye is "blue black."

The first step was to ascertain whether Leber's statements concerning the passage of fluids from the anterior chamber into the episcleral venous plexus hold good for a solution of anyline purple dye. For the sake of brevity, injections of coloured fluid or water into the aqueous and vitreous chambers, and the pressures respectively employed, will be indicated thus:—

Aqueous ... purple ... 30 cm.

Vitreous ... water ... 40 cm.

The pressure may in all cases be considered equivalent to the same column of water.

Experiment 1.—Aqueous ... purple ... 25 cm.

When the stopcock was opened, the anterior chamber was instantly pervaded with the dye, and the iris completely hidden thereby. In two minutes the purple appeared in one of the anterior ciliary veins; in ten minutes there was intense injection of all these veins, and escape of the dye from the cut edge of the conjunctiva.

Experiment 2.—Similar in all respects to experiment 1.

Experiment 3.—Aqueous ... purple ... 30 cm.
Free injection in ten minutes.

Experiment 4.—Similar in all respects to experiment 3.

Experiment 5.—Similar in all respects to experiment 3.

Experiment 6.—Aqueous ... green ... 30 cm.
Well marked injection in fifteen minutes.

Hence it appeared that, under a pressure, probably, about equal to that of the intraocular fluids during life, these coloured solutions pass readily from the anterior chamber into the episcleral venous plexus. The next step was to ascertain whether this escape from the anterior chamber could be arrested by causing the lens and its suspensory ligament to advance, by an excess of pressure in the vitreous chamber.

Experiment 7.—Aqueous ... purple ... 25 cm.
Vitreous ... purple ... 100 cm.

During the introduction of the needles both stopcocks were closed. The vitreous tube was then opened, with the effect of rapidly making the globe

very tense. A few minutes later the aqueous tube was opened. No escape of dye occurred into the aqueous chamber, but the aqueous was driven into the tube by the advance of lens and iris. The iris at once applied itself closely to the posterior surface of the cornea throughout, so as to completely annihilate the anterior chamber. Thirty minutes later there was no discolouration of the iris by the dye—it remained closely compressed against the cornea; no trace of the dye in the episcleral or conjunctival vessels; no escape of the dye from any part of the globe. The pressures were then reversed—viz., aqueous raised to 100 cm., vitreous lowered to 25 cm. Instantly, the anterior chamber was filled, the iris retired from the cornea, and became entirely hidden by the dye. Fifteen minutes later there was free injection of the episcleral vessels.

In experiment 7 the difference between the pressures in the aqueous and vitreous chambers was 75 cm. Successive diminutions of the difference showed that a very much smaller excess in the vitreous suffices to arrest the flow from the anterior chamber.

<i>Experiment 8.</i> —Aqueous	...	purple	...	25 cm.
Vitreous	...	purple	...	50 cm.

As in experiment 7, the vitreous tube was opened first, the aqueous tube some minutes later. Immediately the aqueous tube was opened, the iris advanced and applied itself to the cornea, and the anterior chamber was annihilated. The lens advanced in like manner, and was probably very nearly in contact with the cornea, and received the point of the aqueous needle into its anterior surface in the pupillary area. Thirty minutes later, no injection. The pressures were then reversed, viz. :—

Aqueous	...	...	50 cm.
Vitreous	...	...	25 cm.

and rapid filling of the anterior chamber, with total disappearance of the iris, followed. Five minutes later, injection of vessels and free escape of fluid from their cut ends.

<i>Experiment 9.</i> —Aqueous	...	purple	...	25 cm.
Vitreous	...	purple	...	35 cm.

The aqueous tube was opened two minutes later than the vitreous: the periphery of the iris thereupon at once advanced and applied itself to the cornea; the pupillary margin remained for a short time covered by the dye. During the introduction of the aqueous needle in this, and in almost all the experiments, a drop or two of the dye escaped at once into the chamber and mingled with the aqueous humour, so as to hide the iris unless it became applied close to the corneal surface. Twenty-five minutes later, no trace of injection of vessels. Aqueous pressure raised to 50 cm.; thereupon the anterior chamber filled by a gradual insinuation of the dye between the iris and cornea. Aqueous again lowered to 25

cm. ; thereupon the iris again applied itself to cornea. The same change, several times repeated, with the same results. Aqueous then left permanently at 35 cm.—*i.e.*, the same as vitreous: periphery of iris remained just visible for a few minutes, and then disappeared. Fifteen minutes later very slight injection was visible, which gradually increased during the following half-hour.

It occurred to me that I might obtain a permanent demonstration of the behaviour of the iris, lens, &c., under different pressures, anteriorly and posteriorly, by leaving the experiment in operation sufficiently long for the tissues to become stained by the dye where in contact with it, and subsequently cutting microscopic sections of the parts in question. The next two experiments, with the sections and drawings belonging to them, illustrate the remarkable difference in the effects when a slight excess is produced respectively behind and in front of the lens and its suspensory ligament.

Perfectly fresh *warm* eyes were used.

<i>Experiment 10.</i> —Aqueous	...	purple	...	30 cm.
Vitreous	...	purple	...	40 cm.

The aqueous tube was now opened first, so that an influx of dye, mingling with the aqueous humour, completely hid every part of the iris. The vitreous tube was then opened. In a few seconds the iris reappeared at the periphery, and slowly applied itself to the cornea, quite up to the pupillary margin. The fact of its being closely in contact with the cornea was known by the complete disappearance of the dye from its surface. The pupil remained filled with fluid. *Five hours later, no trace of injection of the episcleral vessels at any part of the circle.* [In looking for this injection, the eye was in all cases illuminated with a strong condensed light.] *No escape of the dye from the vena vorticosæ, in the neighbourhood of the optic nerve, or at any other part of the globe.* Fig. 32 shows somewhat diagrammatically the appearance of the eye. The fluid in the glass tubes had not fallen. Both needles were then withdrawn, and were found to be perfectly patent. The eye was divided into lateral halves. The vitreous was deeply stained throughout. On carefully removing it under water, the retina was found to be unstained. The eye was then frozen, and sections cut. Fig. 28 represents a section of the ciliary region, iris, cornea, &c. (¹) The following parts are deeply stained: the posterior elastic lamina of the cornea, throughout an area corresponding precisely with the pupil; a portion of the posterior surface of the iris, reaching from the pupillary margin to the point where the

¹ Slides. Experiment 10, *a. b.*

ciliary process is in contact with it; the suspensory ligament of the lens, and the tissue by which it is connected with the ciliary process; also the ciliary process itself, where the absence of opaque pigment permits the appearance of the stain in one or two spots. The whole of the anterior surface of the iris, and that portion of the elastic lamina of the cornea opposed to it, as well as the ligamentum pectinatum, are entirely free from stain.

Herein we have ocular demonstration (fig. 28) that the fluid which remained in the central part of the anterior chamber corresponding with the pupil was entirely unable, in the course of five hours, although constantly in open communication with a column 30 cm. high, to make its way to the periphery of the chamber, its normal outlet. We see, further, that the obstruction was due to the contact of the iris with the cornea, caused by an advance of the lens and its suspensory ligament. The periphery of the iris was evidently pressed upon posteriorly by the ciliary process, for no stain has permeated between the two, while nearer to the pupil the posterior surface of the iris was stained by fluid, which reached it either by passing backwards through the pupil, or forwards from the vitreous chamber through the suspensory ligament.

<i>Experiment 11.</i> —Aqueous	...	purple	...	30 cm.
Vitreous	...	purple	...	20 cm.

Aqueous tube opened first, as before; vitreous tube immediately afterwards. Anterior chamber at once filled, so as to completely hide iris. Five minutes later a few small vessels were injected; thirty minutes later an exquisite injection of the venous plexus all round the cornea, and escape of dye from edge of conjunctiva (fig. 31). *Five hours later*, intense injection and staining of the ciliary region; a considerable quantity of fluid had escaped from cut ends of vessels, and collected round the eye. The fluid in the glass tube (aqueous) had fallen $\frac{3}{8}$ inch. Needles withdrawn—both patent. Eye divided, no dye in vitreous chamber. Frozen, and sections cut from ciliary region. The following parts were stained:—The whole of the posterior elastic lamina of cornea, ligamentum pectinatum and its immediate neighbourhood, iris, ciliary processes, and suspensory ligament and capsule of the lens. (Fig. 29. Slides. Experiments 11, *a*, *b*.)

These two experiments (10 and 11) carried on simultaneously, exhibited, in striking contrast throughout, the

different consequences of an excess of pressure in the vitreous and aqueous chambers. Figs. 32 and 31 exhibit roughly the appearances of the two eyes about thirty minutes after the commencement of the experiments. The two following (12 and 13), in which the excess of pressure was still smaller, gave respectively the same results, except that the whole of the anterior surface of the iris did not reach the cornea.

<i>Experiment 12.</i> —Aqueous	...	purple	...	30 cm.
Vitreous	..	purple	...	35 cm.

Aqueous tube opened first, so as completely to fill chamber before opening vitreous tube. Iris returned to view more slowly than before, first as a very narrow peripheral line here and there; in ten minutes its periphery was visibly in contact with cornea all round the circle; the pupillary margin remained permanently hidden.

<i>Experiment 13.</i> —Aqueous	...	purple	...	30 cm.
Vitreous	...	purple	...	25 cm.

The tubes opened in same order as before. Iris remained permanently hidden. Injection of ciliary plexus in fifteen minutes.

It appears then that an excess of pressure in the vitreous chamber equal to 5 cm. of water pressure (=4 mm. mercury, or 2 ins. water) suffices to close the channel through which the aqueous humour normally escapes. Seeking the points at which this conclusion might be assailed, I thought that two objections might be raised—namely: (1) the tension of the suspensory ligament is believed to depend upon the resistance offered by the choroid, ⁽¹⁾ and since the choroid may lose its elasticity or contractility soon after death, the suspensory ligament may be more lax in the dead than in the living eye; (2) the tensor the tunics—sclera and choroid—the tensor, probably, is the suspensory ligament; if, therefore, the pressures employed in these experiments are lower than the normal intraocular pressure, the results as regards the advance of the suspensory ligament are not applicable to the living eye. I therefore experimented again

¹ Hensen and Völckers. Arch. f. Ophth., XXIX., I., 156.

with much higher pressure, and on eyes which were above all suspicion of post-mortem change. The two following eyes were rapidly excised immediately the animals were killed, placed in a receptacle previously prepared—viz., a wide-mouthed glass bottle, plunged to the neck in a box full of hot salt, corked, and at once brought to my laboratory, where they arrived, not warm merely, but hot and steaming as they left the orbit. Their temperature was maintained without much diminution during the few minutes occupied by the experiments, by a hot brick placed close to the apparatus. I know well, from former experiments in galvanising freshly-excised eyes, that, with such precautions, the contractility of the iris may be preserved for an hour or more. ⁽¹⁾

<i>Experiment 14.</i> —Aqueous	...	purple	...	90 cm.
Vitreous	...	water	...	95 cm.

The aqueous tube was opened about two seconds first, so as to get the iris hidden before pressure was applied behind it. The tension of the eye to the touch was enormous; the pressures employed I believe to be about treble the normal pressure in man and several of the lower animals. The iris reappeared quite slowly, but after two minutes its extreme periphery was visibly applied to the cornea throughout the whole of the circle, and gradually appeared more and more, but, as in experiment 12, the pupillary margin remained permanently hidden.

<i>Experiment 15.</i> —Aqueous	...	purple	...	90 cm.
Vitreous	...	water	...	92 cm.

i.e., excessive tension of the tunics, and a difference of *only 2 cm. water*. After three or four minutes the iris reappeared through about half the circle, and ten minutes later it was just visible here and there throughout the remaining portion. It was evidently not in *close* contact with cornea, as in former experiments, for its surface did not entirely clear itself of the dye. The experiment was continued, in order to ascertain whether injection would occur. Faint injection of one vein was visible after about twenty minutes. An hour and a half later there was a visible injection all round the cornea, but no escape of fluid, to any perceptible amount, from the cut ends of the vessels.

In addition to, and as a necessary corollary of, the conclusion before stated, the foregoing experiments appear to

¹ See British Medical Journal, December 6, 1873.

prove that the pressures in the aqueous and vitreous chambers are normally equal, or so nearly equal that the difference, if any, must be entirely undiscoverable by any tonometrical test, and hardly discoverable by the manometer.

To ascertain, by a corroborative test, that the closure of the angle of the anterior chamber is brought about by an advance of the partition which separates the aqueous from the vitreous chamber, I resolved, during the occurrence of the said closure, to abolish the partition by displacing the lens into the vitreous.

<i>Experiment 16.</i> —Aqueous	...	purple	...	30 cm.
Vitreous	...	purple	...	40 cm.

Aqueous tube opened a few seconds before vitreous tube. As before, the iris reappeared, cleared of dye, and applied itself to the cornea. Fifteen minutes later, no injection. I then attempted, with a bent needle introduced near the equator, to perform "reclination." The lens was so soft as to offer no sensible resistance, and to displace it whole proved impossible. Free movements were made through it with the needle in all directions. The dye entered the anterior chamber, and slowly insinuated itself between the iris and cornea, completely up to the periphery; ten minutes later, visible injection; twenty minutes later, intense injection. On opening the eye, the débris of the lens was found nearly in its normal position, but so completely divided as to form no barrier between the vitreous and aqueous chambers.

<i>Experiment 17.</i> —Aqueous	...	purple	...	30 cm.
Vitreous	...	purple	...	40 cm.

Aqueous tube opened first, as before. On opening vitreous tube, iris at once reappeared and applied itself to cornea. Lens was at once incised, and broken up as before. Iris immediately disappeared, and aqueous chamber filled with dye. Vitreous pressure was then raised successively to 60 cm. and 90 cm., in order to ascertain whether the periphery of iris could be made to advance after the destruction of the partition between vitreous and aqueous chambers. The iris did not reappear. Rapid and intense injection occurred. Eye opened; lens found freely divided, and displaced backwards.

<i>Experiment 18.</i> —Aqueous	...	purple	...	30 cm.
Vitreous	...	purple	...	40 cm.

Tubes opened in same order as before. Iris applied itself to cornea; five hours later, no injection. Lens divided and displaced as before; one hour later, free injection.

The three last experiments proved that, if the partition between the vitreous and aqueous chambers be abolished by the removal of the lens, the escape of fluid through the angle of the anterior chamber is not arrested, but accelerated, by an increase of the pressure in the vitreous chamber.

In one important particular, the conditions of the eye in all the foregoing experiments were essentially different from the conditions of the living eye—viz., an artificial outlet, the aqueous needle, allowed the escape of fluid from the anterior chamber, when an excess of pressure occurred on the vitreous side of the partition. Such an outlet was an essential necessity for these experiments, inasmuch as without it the pressures on the two sides of the partition would be equalised by an advance of the lens and its suspensory ligament, and the behaviour of the partition under a *permanent* excess on its hinder surface could not be tested.

It was necessary, in the next place, to ascertain the consequences of applying pressure to the vitreous surface of the partition, *the aqueous chamber being unopened*.

The results already obtained enabled me to foresee these consequences with tolerable certainty. It appeared indubitable that the partition and the periphery of the iris would advance and cause an escape of the fluid of the anterior chamber through the natural channel, until, by this very advance, the angle of the chamber should be so much compressed as to preclude further escape, and that then a certain amount of fluid would be permanently imprisoned in the anterior chamber, and prevent the iris and lens from advancing into contact with the cornea.

Experiment 18.—Aqueous chamber filled with purple under a pressure of 30 cm., and *its tube then closed*.

Vitreous	...	water	...	35 cm.
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After a few minutes a faint injection of the episcleral vessels occurred, but there was no free escape. Sixty minutes later iris had not reappeared,

no fluid was escaping from cut ends of vessels, but anterior chamber evidently still contained fluid. Aqueous tube opened, iris advanced and applied itself to cornea, evidently because of an escape of fluid from anterior chamber through aqueous needle. The experiment was not very satisfactory, because the iris and inner surface of cornea were so deeply stained as to prevent the application of iris to cornea from being distinctly seen.

The following two experiments demonstrated the imprisonment of fluid in the anterior chamber more satisfactorily :—

<i>Experiment 19.</i> —Aqueous	...	purple	...	40 cm.
Vitreous	...	water	...	45 cm.

Higher pressures were employed, in order again to prove that the results did not depend upon the employment of a sub-normal pressure. Aqueous tube opened first, so as to fill anterior chamber as much as possible, then closed; vitreous tube then opened. Injection and escape of dye from cut ends of vessels in a few minutes. Sixty minutes later no more fluid appeared to be escaping from the eye; on wiping away the fluid collected on the surface, no more collected. Eye frozen solid, without disturbing needles, and divided into lateral halves. Anterior chamber still present, and of moderate depth, contains frozen dye. As it was impossible so to illuminate the frozen specimen as to thoroughly examine the condition of the angle of the chamber, I attempted to learn its condition in the following way. With the points of a pair of compasses I measured exactly the external diameter of the cornea in the half eyeball, and pricked the measurement on a piece of paper; then extracting the half disc of frozen dye from the anterior chamber, I measured its diameter in the same way, and found that it corresponded exactly with that of the cornea. The edge of the half disc of ice was sharp and unbroken, and no delay occurred during which it could diminish by thawing, so that it was safe to conclude that some narrowing of the angle of the chamber had occurred.

<i>Experiment 20.</i> —Vitreous	...	water	...	60 cm.
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This was simply a repetition of the foregoing, without the injection of any additional fluid into the anterior chamber, and with a higher pressure in vitreous chamber. The eye was fresh and full, and the aqueous chamber was probably nearly as full as during life; whether any aqueous humour was expressed into the venous plexus could not be ascertained. Sixty minutes later, eye frozen without disturbing needle, and divided as before. Anterior chamber contained frozen aqueous humour. The diameter of the frozen disc of fluid was as nearly as possible the same as that of the cornea—possibly a shade larger. It probably reached nearly as far as the ligamentum pectinatum. Its thickness was exactly 2 mm.

[In any repetition of these experiments, I would give a caution that care is necessary to avoid the slightest entrance of air into the india-rubber tubes. Where the columns for the aqueous and vitreous pressures are very nearly equal in height, the entrance of a bubble of air into the longer column may easily reduce its actual pressure below that of the shorter column, and thereby give results exactly the opposite of what is here described. This happened more than once in some of my earlier experiments. Moreover, the last-mentioned experiments gave results entirely different if performed with eyes which were not quite fresh. In three such I found the anterior chamber empty, the iris and lens compressed against the cornea, and the *cornea stained throughout its whole thickness* by the passage through it of the dye, the anterior epithelium peeling off, and fluid exuding from the cornea. In all my experiments, I obtained confirmation of Leber's statement that the *fresh cornea* allows no fluid to pass through it. The anterior epithelium remains perfectly polished, and *perfectly dry*, for several hours under high internal pressure in the fresh eye.]

Thus it was proved that *pressure on the posterior surface of the lens and its suspensory ligament, unless counterbalanced by an equal pressure on the anterior surface, arrests the escape of aqueous humour, by compressing the angle of the anterior chamber, and that this arrest occurs while a considerable quantity of the aqueous humour still remains in the chamber, and while the chamber is open, peripherally, at least as far as the corneal margin, and probably, rather farther.*

The fact here stated requires to be confirmed, as regards the human eye, before it can be applied in the explanation of glaucoma. Inasmuch as a complete repetition of these experiments would require, in that case, the employment of human eyes very shortly after death, their discoloration with staining fluids, and, in some cases, their excision, it would manifestly be a work of much difficulty and time. At present I have had opportunity for a few experiments only. They are however, I think, sufficiently convincing.

Experiment 21.—Male subject, aged nineteen, two hours after death.

Aqueous	...	purple	...	30 cm.
Vitreous	...	water	...	40 cm.

Aqueous tube opened first, so that dye mixing with aqueous humour completely hid iris from view. Then vitreous tube opened. The iris

slowly reappeared at the periphery and, gradually clearing, applied itself throughout the whole of its surface to the cornea. The pressures were then reversed, so as to cause a refilling of anterior chamber and the total disappearance of iris ; pressures then re-established with an excess in the vitreous of 5 cm. only, instead of 10 cm. as before ; thus—

Aqueous	...	purple	...	30 cm.
Vitreous	...	water	...	35 cm.

The iris appeared, the whole circle of the periphery became applied to the cornea, but the pupillary margin remained more or less hidden by the dye—a result precisely like that obtained in the pig's eye under similar conditions.

Experiment 22.—The other eye of the same subject.

Aqueous	...	purple	...	30 cm.
Vitreous	...	water	...	20 cm.

Iris remained hidden ; free injection in 30 minutes.

As regards the imprisonment of fluid in the anterior chamber during exalted vitreous pressure, I have had no opportunity of demonstrating the fact by the freezing method in the human eye, but I am none the less confident as to its occurrence. During the experiments on the dead subject already recorded, made with a view to learning the normal intraocular pressure, I observed, as doubtless others have done before, that, after high pressure has been applied to the vitreous chamber, even for long periods of time—*e.g.*, an hour or more—the anterior chamber still contains fluid. This fluid is the aqueous humour, imprisoned by the closure of its normal channel of escape.

If we now consider in conjunction—

- a.* The conditions revealed by the microscope in glaucomatous eyes ;
- b.* The results of the foregoing experiments ;
- c.* The fact that a shallow anterior chamber is a constant feature of glaucoma ;

we may, I think, safely conclude that:—

1. *Closure of the angle of the anterior chamber is an essential part of the glaucomatous process; it checks the escape of the aqueous humour, and raises the tension of the eye.*
2. *It is dependent on, or at least associated with, an exaltation of the vitreous pressure above the aqueous pressure, which does not exist in the healthy eye.*

Referring again to the theories of Max Knies and Ad. Weber concerning the mode in which the closure of the angle of the chamber is brought about, the following difficulty presents itself:—If the *primary cause* of glaucoma be a localised inflammation affecting the permeability of the ligamentum pectinatum, &c., or if it be a swelling of the ciliary processes, causing compression of this region, why are not the lens and suspensory ligament displaced backwards? Why is not glaucoma characterised by a deep, instead of a shallow anterior chamber?

It is not unlikely that in *serous iritis* we have an example of a primary obstruction of the filtration channel, though as there is here, an altered and perhaps increased secretion as well, the condition is not necessarily one of obstruction purely. I think that the theory of Max Knies might explain this variety of the glaucomatous condition, but this only; and in this disease the symptom above mentioned—viz., deepening of the anterior chamber—is actually met with. With regard to Weber's theory, I will not venture to assert that it is incorrect, but I will say that, if correct, it is incomplete. If we assume, with him, that the swelling of the ciliary processes is the primary change, and that it produces narrowing of the angle of the anterior chamber, and thereby retards the escape of the aqueous humour and raises the intraocular pressure, we have yet to ascertain how these conditions of obstruction, *beginning in the aqueous chamber,*

can cause the lens and suspensory ligament to advance—how, that is, they can exalt the vitreous pressure above the aqueous pressure.

There can be no doubt that the peripheral contact of the iris and cornea is in many cases caused directly by the pressure of the ciliary processes against the posterior surface of the iris. This is seen to be the case, for instance, in figs. 11, 15, 17, 18, 26, in all of which the ciliary process occupies a position closely corresponding to the adhesion on the other side of the iris, or would manifestly do so but for the absence of the support of the suspensory ligament on its posterior surface. This, however, does not prove that the primary change is in the ciliary processes. Fig. 28 represents a very similar relation of the parts in the excised eye of the pig, in which, as we have seen, a slight excess of vitreous pressure closed the angle of the chamber as completely as it is closed in the glaucomatous human eye. Moreover, we have seen that the same result is obtained in the human eye after death, when no swelling of the ciliary processes can occur.

Again, we see, in figs. 13, 22 (see also slides 6*a*, 6*b*, 6*c*, 4*b*, &c.), an adhesion of the iris with the cornea, so far in advance of the tip of the ciliary process, and such advanced atrophy of the latter, that it appears impossible for the ciliary process to have caused the adhesion, unless we imagine that a former condition of elongation has given place later on to atrophy with retraction. The appearances here referred to, as well as those to be seen in many others of the slides, seem to be due rather to compression of the ciliary processes by an advance of the suspensory ligament, than to a primary change in themselves. That such an advance actually occurs in glaucoma we know, and we may often observe a direct relation between the intensity of the symptoms and the amount of this advance.

To explain the compression of the angle of the chamber we must then, I think, look for *conditions which are capable of exalting the vitreous pressure above the aqueous pressure.*

Any further step towards the elucidation of the point in question seems to demand a knowledge of the processes by which the vitreous humour is nourished and renewed. This is uncertain ground, and the theory which I have to advance is supported rather by presumptive evidence than by demonstrable facts.

It is probable that the vitreous body receives its nutrient supply exclusively from the ciliary portion of the uveal tract I have been unable to find, even in the most recent and elaborate expositions of the anatomy and physiology of the eye, any distinct statements as to the nutritive processes of the vitreous body. Even Leber in his admirable treatise, "*Die Circulations—und Ernährungs—verhältnisse des Auges,*" leaves the subject entirely untouched.⁽¹⁾ An opinion commonly entertained, I believe, is that the vitreous is nourished by a fluid secreted by the capillary plexus of the choroid, which reaches it by passing through the retina. One recent writer on detachment of the retina goes so far as to assert that "no other mode of nutrition for the vitreous is conceivable."⁽²⁾

In opposition to this view, and in support of the statement enunciated above, I will suggest the following facts for consideration:—

1. It is *à priori* improbable, and at variance with nutritive arrangements in other parts of the organism, that the retina, a highly complex nervous structure, composed of many differentiated layers, and possessing an isolated vascular system of its own, should convey the secretion of an entirely distinct vascular tract situated on its one side to

¹ Handbuch d. g. A., II., I. ² Arch. f. Ophth., XXII., IV., 244.

a structure with which it has nothing in common, either structurally or by development, situated on its other side.

2. The recent discoveries of Kühne render it almost certain that the function of the chorio-capillaris and the hexagonal epithelium is to secrete the "visual purple." ⁽¹⁾

3. The retina proper terminates at the ora serrata; its vascular system extends to this line, and there abruptly returns upon itself; precisely at this line an intimate connection between the uveal tract and the vitreous, of a character perfectly adapted for nutrition, begins. The hyaloid membrane here undergoes a special development (zonula ciliaris), and is very intimately connected, on the one hand, with the vitreous, and, on the other, by the interposition of a single layer of cylindrical cells (pars ciliaris retinae) with the ridges and furrows of the secreting surface of the ciliary body. ⁽²⁾

4. Staining fluids injected into the vitreous chamber pass very readily through the zonula, and colour the cells covering the surface of the ciliary processes; while, after many hours, the retina, though separated from the vitreous by a much thinner portion of the hyaloid membrane, is found colourless. ⁽³⁾

5. Detachment of the retina, in the forms most commonly met with, is more satisfactorily explained by this theory than by any other. This point, which is, I think, a very strong one, would require for its development a very lengthy examination. In this place I can only sketch the evidence

¹ Photo-Chemistry of Retina, &c., Kühne; Transl. M. Foster; pages 9, 10, 101.

² Kölliker. Micro. Anat., p. 573. Ivanoff. Handbuch, d. g. A., I., I., p. 305.

³ See experiment 29, p. 145; also slides, exp. 29a, exp. 29b.

in outline. It is pretty generally agreed that the primary change in most forms of retinal detachment is not, as formerly supposed, an inflammatory exudation from the choroid, which pushes the retina before it, but, on the contrary, an alteration in the vitreous, of which the choroidal exudation is a secondary consequence. The vitreous change has been supposed to induce the retinal detachment, by altering the osmotic relations between it and the normal nutrient supply believed to proceed from the choroid. ⁽¹⁾ This supposition, as I understand it, gives no explanation of the primary change in the vitreous. It appears to me that, in the great majority of cases, the alteration in the vitreous is one either of *malnutrition, and consequent loss of bulk by atrophy*, or of *shrinking produced by inflammation and cicatrisation of its tissue*, and that the flow of a highly albuminous coagulable fluid from the choroid is the natural result of the withdrawal from its vessels of the pressure to which they are normally subjected.

Detachment of the retina in the complete form, so commonly met with in excised eyes, is constantly associated with marked changes in the ciliary body. The zonula is found coated with inflammatory exudation, and, together with the pars ciliaris retinae, converted into a thick opaque membrane, obviously incapable of the transmission of a normal secretion from the subjacent uveal tract. ⁽²⁾ Such a condition is constantly found in eyes which have been destroyed by irido-cyclitis, whether from injury or from the extension of idiopathic iritis. In other instances, the secreting portion of the ciliary body is found to be in a state of advanced atrophy, the removal of the pigment having left the membrane almost transparent. ⁽³⁾ In other cases again, especially those of perforating wounds of the vitreous, it

¹ E. Raehlmann. Arch. f. Ophth., XXII., IV., 245.

² See specimen 10. ³ Specimens 12, 13.

appears likely that the shrinking of its substance is the sequel of inflammation, and that the changes discovered in the region of the zonula are the result, rather than the cause, of the abolition of the vitreous body. ⁽¹⁾ The total annihilation of the vitreous chamber—*i. e.*, of the *intra-retinal* space—coincides with the complete obliteration of the secreting surface from which it normally receives its nutrient supply; for the anterior portion of the retina proper, doubling forwards at the ora serrata, applies itself to the zonula, and ultimately becomes firmly welded to it.

Amongst a considerable number of specimens in my possession, I find no case of detached retina in which there is not marked disease of the ciliary body; and, more important still, I find no instance of marked disease of the ciliary body without detachment of the retina.

While the ciliary portion of the uveal tract is thus constantly the seat of disease, the portion posterior to the ora serrata—the choroid—is often comparatively healthy. That changes in the choroid, inflammatory and atrophic, may also frequently be found in such eyes, is a natural consequence of the proneness of pathological processes to pass from one part of the uveal tract to another; but that they are not in any sense a *cause* of detachment of the retina is, I think, evident, when we remember that, with the ophthalmoscope, we see almost universal atrophy and vascular emptiness of the choroid (productive of total loss of retinal function), without any retinal detachment, and with a presumably healthy nutrition of the vitreous—*i. e.*, with perfect transparency and normal tension. The diminished tension which is sometimes discoverable in cases where the choroid is the seat of visible changes, is, in my opinion, due to a participation of the ciliary portion of the membrane in the disease.

¹ Specimen 11.

6. The fact that changes in the vitreous body may sometimes be traced to the presence of disease in the corresponding portion of the choroid is no proof that the former draws its normal nutrient supply from the latter. Such an extension of disease, demonstrable under the microscope, involves the retina also, and is a not unnatural consequence of the contiguity of the structures concerned.

The foregoing considerations afford, I think, strong presumptive evidence of the truth of the statement enunciated concerning the nutrient supply to the vitreous body. ⁽¹⁾

It is probable that the waste fluid of the vitreous body passes through the suspensory ligament into the aqueous chamber, and, forming part of the aqueous humour, escapes from the eye at the angle of the anterior chamber. A complete proof of this proposition is hardly to be obtained either by experiment or by clinical study, but it can, I think, be backed with much circumstantial evidence. If we ask what are the other modes of escape or reabsorption possible for this fluid, the following may be suggested:—

1. *Osmosis through the retina and absorption by the choroid*, in accordance with the theory already adverted to. The same objections which were urged against the idea of a nutrient supply *from* the choroid hold good for a return of fluid *to* it. The fact that a diffusible coloured fluid injected into the vitreous body, under a continuous pressure, may fail after many hours to produce the slightest staining of the retina, and appears never, in the fresh eye, to reach the choroid, is, I think, highly significant. ⁽²⁾ To this it may very properly be objected that what is true of an artificial injection, is not necessarily true of the normal intraocular fluid; but the objection is somewhat weakened by the fact that this same artificial injection does pass very readily and

¹ See page 137. ² Experiment 29, slides.

rapidly in certain directions which correspond with the normal path of the intraocular fluid—*e. g.*, from the anterior chamber into the episcleral venous plexus.

2. *Through the lymph passages within the sheath of the optic nerve.* Schwalbe says of these lymph passages, "They constitute a very complicated channel, which finds exit in the lymph spaces of the skull, and which conveys not only the lymph formed in the optic nerve, but that also of the retina and vitreous." ⁽¹⁾ Stilling has even suggested that closure of these posterior channels of exit is the cause of a large class of cases of glaucoma. ⁽²⁾ Now, even granting the existence of a direct communication between these lymph channels and the interior of the eye, which is perhaps hardly proved, we have no evidence to show that they afford an exit to the intraocular fluid proper; in fact, we have strong evidence to the contrary. Optic neuritis, in one form at least—namely, "choked disc" (*stanungspapille*)—is known to be constantly associated with dropsy of the nerve sheath, presumably due, in many cases, to an increased pressure within the skull. If Stilling's suggestion were correct, such conditions as these should surely lead to some excess of tension, if not to a very pronounced glaucoma; but, as far as my experience goes—and I have examined many cases with the finger, and several with the tonometer—optic neuritis of the extremest kind involves no excess of tension. ⁽³⁾

In my own injection experiments on the eyes of pigs, I have entirely failed to trace any communication between the vitreous chamber and the lymph channels of the optic nerve. I have injected solutions of carmine and of anyline purple into the vitreous chamber, and maintained the pressure for

¹ Handbuch, d. g. A., I., I., 350.

² Heidelberg Congress, 1877. See *Klin. Monatsblätt. Bericht über, &c.*, p. 16.

³ See Section VII., p. 278.

five or six hours, and then frozen the eye and cut sections, both parallel with and perpendicular to the plane of the optic disc, but have found no trace of the colour deeper than the superficial layers of the retina. ⁽¹⁾

3. *Reabsorption by the secreting surface of the ciliary body through the zonula.* Against this supposition I have no positive evidence to bring forward. It certainly appears unlikely that whereas the free portions of the ciliary processes in the posterior chamber yield a secretion which passes out by the ligamentum pectinatum, the immediately continuous portions in contact with the zonula should reabsorb the fluid which they secrete; but the improbability, to my mind, depends chiefly on the reasons which are forthcoming in favour of the theory originally stated.

Leber concludes the fourth section of his already quoted "Studies on the Interchange of Fluid in the Eye" with the following sentences:— ⁽²⁾

"The vitreous chamber, when compared with the anterior chamber, appears far more completely closed, and less ready to allow the outward passage of fluid. This is manifested in those experiments in which, after a considerable excess of pressure in the vitreous chamber, the subsequent fall is very slow. This is all the more noticeable because the excess of pressure is transmitted, in part at least, to the anterior chamber, and must be diminished by escape of fluid from the latter. At present I only mention

¹ Experiments and slides, 29, 31, 32, see p. 145. ² Arch. f. Ophth., XIX., II.

NOTE.—*June, 1879.*—I have recently endeavoured to obtain evidence of the existence of these channels by injecting a coloured fluid into the sheath of the optic nerve after applying a ligature so as to avoid its escaping posteriorly. The pressure employed was about double the normal intraocular pressure, and was maintained during five hours. Sections of region around the optic disc showed that no particle of the fluid had entered the eyeball. These sections, together with drawings, were recently exhibited at a meeting of the Midland Medical Society.

“this question incidentally, inasmuch I have not made it the object of direct examination.”

It will be seen at once that my own experiments, already recorded, afford the key to this closed condition of the vitreous chamber.

Injection into the vitreous chamber closes the outlet of the aqueous chamber, and thereby, as I believe, closes the outlet through which the waste fluid of the vitreous normally escapes. This explains the slow disappearance of the excess of pressure noted by Leber, for the greater the pressure of the vitreous chamber the more tightly is the common outlet for the intraocular fluids locked. This explains why the vitreous pressure, although transmitted to the aqueous chamber, is not relieved by escape of fluid from the latter.

For the purpose of testing the escape of fluid from the vitreous chamber, the conditions of Leber's experiments were at fault.

In order to keep the angle of the anterior chamber open, as in the living eye, during the injection of fluid into the vitreous chamber, it is necessary to inject fluid under the same pressure into the anterior chamber also, and to maintain a constant supply of the same, whereby that which escapes at the angle of the chamber may be constantly replaced. Under these conditions the closure of the angle of the chamber is avoided, a stream of fluid constantly traverses the aqueous chamber, as in the living eye, and we may presume that the fluid of the vitreous chamber also will follow its normal path.

I thought that, by injecting a coloured fluid into the vitreous chamber and pure water into the aqueous chamber under the same pressure, I might possibly be able to observe the passage of the former through the suspensory ligament into the aqueous chamber, and its exit, mingled with the water in the latter, into the episcleral plexus. The results

which I have obtained so far are by no means conclusive, but are favourable to the theory before stated. ⁽¹⁾ It appeared necessary, in the first place, to obtain some idea as to the readiness with which fluid escapes from the anterior chamber when an equal pressure is maintained in the vitreous chamber. I therefore made six experiments of the following kind:—

<i>Experiments 23, 24.</i> —Aqueous	...	purple	...	30 cm.
Vitreous	...	water	...	30 cm.

It is not necessary to give the results of these in detail. Injection of the episcleral venous plexus occurred—very slowly if both tubes were opened simultaneously, sooner if the aqueous tube was opened a few seconds before the vitreous tube, so as to ensure a full anterior chamber with a well-opened angle to start with.

<i>Experiment 29.</i> —Aqueous	...	water	...	30 cm.
Vitreous	...	purple	...	30 cm.

In ten minutes the dye could be seen with the ophthalmoscope to have spread laterally over the greater part of the vitreous chamber; water escaped from the cut edge of conjunctiva, and collected below the eye. *Five hours later*, no marked injection of veins; a doubtful injection of one; the fluid in the anterior chamber appeared slightly tinged with purple, but this was doubtful; a free escape of water had occurred; no escape of the dye from the *venæ vorticosæ*; the eye was placed in picric acid; the following day it was frozen, and sections were cut from the ciliary region, and the optic nerve at its point of junction with the globe. ⁽²⁾ The whole of the anterior part of the vitreous was deeply stained; from this a line of colour extended backwards through the centre of the vitreous (central canal of Stilling). Numerous sections of the optic nerve and the adjoining sclera, cut perpendicular to the plane of the disc, were examined microscopically. The dye could nowhere be traced deeper than the fibre layer of the retina. In the sections from the ciliary region, the hyaloid membrane, posterior to the ora serrata, was distinctly stained, while the subjacent retina was colourless. The suspensory ligament was intensely stained throughout the part corresponding with the ciliary processes and a little beyond their apices; the unstained portion beyond this was doubtless the anterior capsule of the lens. The cellular membrane between the suspensory ligament and the pigment surface of the ciliary processes (*pars ciliaris retinæ*) was very distinctly stained; no discolouration was observed in the iris, the posterior surface of the cornea, or the ligamentum pectinatum. The choroid was entirely unstained; it had a bright yellowish brown colour throughout, in contrast with which any purple colouration would have been very manifest.

¹ Page 141. ² See slides, experiments 29*a*, 29*b*.

<i>Experiment 30.</i> —Aqueous	...	water	...	30 cm.
Vitreous	...	purple	...	30 cm.

Two hours later, perhaps a very slight discolouration of the water in the aqueous chamber, but this was doubtful. Free escape of water from cut ends of vessels; no visible injection of episcleral veins; no escape of dye from *venæ vorticosæ*. Next morning—twelve hours from commencement—a very doubtful injection with purple of the episcleral veins; distinct purpleness of fluid in anterior chamber; free escape of water, slightly tinged with purple, and collection of the same in the white saucer in which eye lies; *no escape of dye from venæ vorticosæ*. Eye divided into anterior and posterior halves. When the aqueous needle was withdrawn (before dividing the eye) a quantity of fluid escaped from anterior chamber through the puncture; being received into a white saucer, it was seen to be *very distinctly tinged with purple*. The anterior portion only of the vitreous body was stained. *The retina was entirely free from stain*. [When first removed from the eye, and placed in water, it was rosy pink, but rapidly bleached, and was colourless after about a minute. This was the “visual red” of Kühne. The pig had been killed by gaslight, the injection of the densely-coloured solution into the anterior portion of the vitreous had preserved the retina from the bleaching action of light, until the eye was opened twelve hours later.] *The suspensory ligament was stained, and the internal surface of the cornea was very distinctly purple also.*

<i>Experiment 31.</i> —Aqueous	...	carmine	...	30 cm.
Vitreous	...	carmine	...	40 cm.

This was a repetition, with a different solution, of some of the former experiments—viz., experiments 9, 10, 12, &c. It is given here because sections were cut from the optic nerve and its surroundings, to ascertain whether the staining fluid found an exit there. The superficial layers of the retina were intensely stained close to the optic nerve, but no colour was found deeper than this. (¹)

<i>Experiment 32.</i> —Aqueous	...	carmine	...	30 cm.
Vitreous	...	carmine	...	40 cm.

From this eye also, after twelve hours' subjection to the above-named pressures, sections of the region of the optic disc were cut *parallel with the plane of the disc*, and therefore necessarily exposing every channel through which the carmine could pass, if any such existed. No trace of staining was discoverable below the level of the retinal fibres. See Slides, experiments 32, *a*, *b*. [The extreme margins of the sections, corresponding with the outer surface of the sclera, show a little colouration simply from the exposure of this surface to the action of the fluid which escaped from the cut episcleral vessels.]

<i>Experiment 33.</i> —Aqueous	...	water	...	30 cm.
Vitreous	...	carmine	...	30 cm.

¹ See Slides, experiments 31*a*, *b*, *c*.

Twelve hours later, very decided colouration of the fluid in anterior chamber, and *very distinct red injection of episcleral veins*; no escape from *venæ vorticosæ*; on opening the eye, vitreous stained throughout; retina, when separated from vitreous, colourless; a strongly marked ring of colour round periphery of lens (Petit's canal).

<i>Experiment 34.</i> —Aqueous	...	water	...	30 cm.
Vitreous	...	carmine	...	30 cm.

Twelve hours later, colouration of fluid in anterior chamber, and episcleral injection as in experiment 33. No note made of internal condition of eye.

<i>Experiment 35.</i> —Aqueous	...	water	...	30 cm.
Vitreous	...	carmine	...	30 cm.

Aquous tube opened first, so as to ensure full anterior chamber and an open angle to start with. Vitreous tube three seconds later. Ten minutes later a distinct red colouration, apparently, of surface of iris at pupillary margin. This was, however, really due to the entrance of the carmine solution through the pupil, and mixing with the water in the anterior chamber, for when a strong pencil of light was directed across the eye, parallel with the plane of the iris, the further margin of the sclera (which may be illuminated in all eyes by this proceeding) was of a rosy-red colour, due to the passage of the rays through the fluid of the anterior chamber before they reached it. Two hours later the whole of the iris appeared red under illumination, through the colouration of the fluid in the chamber, but there was no visible episcleral injection. Next morning, ten hours later, doubtful episcleral injection; not so much water had collected below the eye as in other similar experiments; it was faintly tinged with red; on withdrawing the aqueous needle, the fluid which escaped from the chamber was strongly tinged. Eye opened; anterior part only of vitreous stained; retina, when removed, colourless; iris, suspensory ligament, and posterior surface of cornea very distinctly stained.

From the foregoing facts and considerations concerning the secretion and escape of the fluids of the aqueous and vitreous chambers, we may, I think, with a fair show of reason, though not with absolute certainty, derive the following general conclusions. See fig. 33.

1. *The ciliary processes secrete the intraocular fluid.* [The iris also, perhaps, in minor degree.] *A portion of this fluid passes directly from the free portion of the processes into the aqueous chamber. Another portion, proceeding from that part*

of their surface which is in contact with the suspensory ligament, passes through this membrane into the vitreous chamber.

2. The function of the suspensory ligament is probably a purely mechanical one—namely, the maintenance of the lens in its proper position, and the control of its accommodative changes. It is readily permeable by the intraocular fluid, which passes through it in both directions in its course to and from the vitreous body.

3. The whole of the intraocular fluid, including that which returns from the vitreous body, escapes from the eye at the angle of the anterior chamber.

4. Under certain circumstances the pressure in the vitreous chamber becomes greater than that in the aqueous chamber, by the retention in it of more than the normal bulk of fluid. When this occurs the lens and the suspensory ligament advance in such a manner as to compress the angle of the anterior chamber, and retard or arrest the escape of the intraocular fluid.

5. Glaucoma is the expression of this condition of obstruction.

This theory of glaucoma is incomplete until we can point to the cause or causes which destroy the equality of the aqueous and vitreous pressures. In secondary glaucoma such causes are readily to be found, and afford a strong argument that the theory, as applying to glaucomatous conditions in general, is correct. For primary glaucoma it has been to me far more difficult to assign such a cause. I believe, however, that I am now able to propound a theory which affords a more satisfactory explanation of all the facts than any that has

been advanced hitherto. It is no mere hypothesis, for it is the outcome of successive steps of investigation; it is, however, a theory of very recent developement, and, as such, I advance it with hesitation, and must trust to the evidence of further observation for its establishment or its overthrow.

NOTE. July, 1879.—I read with much interest in the current number of Graefe's Archives (XXV., I., 1879) an article by R. Deutschmann, in which independent and conclusive corroboration is given to the view which I have maintained as to the passage of fluid from the vitreous to the aqueous chamber by filtration through the suspensory ligament. Deutschmann observed, by accident, in the human eye that the aqueous chamber, if evacuated by puncture shortly after death, is refilled with colourless fluid within a few hours. He ascertained that the same thing occurs in the freshly excised eye, and concludes that the only possible source of the regenerated fluid is the vitreous humour.

SECTION V.

PATHOLOGY OF GLAUCOMA.

PART II.

PRIMARY GLAUCOMA.

Any one of the three following conditions would destroy the equality of the aqueous and vitreous pressures in favour of the latter:—

1. Increased secretion into the vitreous chamber, not counterbalanced by a similarly increased secretion into the aqueous chamber.
2. Diminished secretion into the aqueous chamber, not counterbalanced by a similarly diminished secretion into the vitreous chamber.
3. Retarded passage of the intraocular fluid from the vitreous into the aqueous chamber.

It will hardly be disputed that simple non-irritative chronic glaucoma is the pure type of the disease, and that irritative symptoms are something superadded. It is in this condition, therefore, that we must expect to find the primary changes in their simplest form.

Chronic glaucoma is met with in association with many morbid states, and it is met with in persons in whom no

other malady whatever is discoverable. Let us take the one instance of cardiac and vascular diseases in general, to which the theory of Weber points. In a certain number of the sufferers from glaucoma these maladies are present in greater or less degree; in many they are entirely wanting; and we see innumerable instances of disease of the heart, the blood-vessels, and the lungs, at all ages, and of great intensity, in which no symptom of glaucoma is discoverable. The same is true, I think, of all the morbid states which have been supposed to cause glaucoma.

The one condition to which glaucoma maintains an almost constant relationship, is *commencing senility*. It would seem probable, therefore, that the essential starting point is to be found in one or other of the changes peculiar to advanced life, rather than in the effects of any particular disease or class of diseases. Can such a starting point be discovered? I think it can.

We know that, during early and middle life, the crystalline lens has the power, by virtue of the elasticity of its capsule, of changing its form—namely, its convexity, its thickness, and its diameter; that, under all physiological conditions, its periphery is separated by an interval from the circle of the ciliary processes; and that this interval is greatest when the lens assumes its maximum convexity, smallest when it assumes its minimum convexity. We know that this power of variation continuously diminishes from infancy to age, and is entirely lost at about the age of 60. We know, further, that in advanced life the lens undergoes, in addition to a loss of elasticity, a change of form: its surfaces are flattened, its thickness is diminished, its diameter is increased. The time at which this senile change of form commences is probably about the fiftieth year, or a little earlier. It is made manifest by a loss of refraction, as distinguished from a loss of accommodation. Concerning the position of the far point,

Donders says, "Up to the fortieth year it remains at the same height; but from that time an extremely slow descent occurs, the emmetropic eye becoming, at the fiftieth year, somewhat hypermetropic, which hypermetropia, at the eightieth year, amounts to from $\frac{1}{24}$ to $\frac{1}{10}$." (1)

Now, this senile change, by increasing the diameter of the lens, diminishes, presumably, the interval between its periphery and the circle of the ciliary processes—that is to say, it narrows the channel through which the intraocular fluid passes from the vitreous to the aqueous chamber. Here, then, is a cause capable of inducing one of the conditions previously stated as a cause of glaucoma—viz., *retarded passage of the intraocular fluid from the vitreous to the aqueous chamber*. It will be objected, If this be so, why is not every senile eye attacked by glaucoma? To which I would reply, *the tendency to glaucoma depends upon an abnormal relationship between the diameter of the lens and the diameter of the circle of the ciliary processes*. Concerning this relationship under different conditions there is not as yet much direct evidence to be obtained, but the following facts appear to me to be in favour of the theory.

The hypermetropic eye appears to be especially liable to glaucoma. Now, the ciliary muscle of the hypermetropic eye has a peculiar conformation, by reason of which the apices of the ciliary processes are considerably more prominent, and stand, *ceteris paribus*, nearer to the periphery of the lens than in the emmetropic and myopic eye. The hypermetropic eye is, moreover, to be regarded as an ill-developed eye, the dimensions of which are, in several particulars, smaller than those of the normal eye. Whether the interval between the periphery of the lens and the ciliary processes is actually smaller than in the normal eye, I have no evidence to show.

¹ Refraction and Accommodation. Trans. N. Syd., S., p. 208.

Atropine has been known, in exceptional cases, to induce glaucoma in an eye presumably healthy up to the time of its application, and, in many cases, to aggravate the glaucomatous condition. So long as the lens retains its elasticity, its diameter tends to be increased by the action of atropine upon the ciliary muscle. The aggravation of glaucoma may be thus explained, and we may, moreover, presume that there are eyes free from glaucoma in which the relation of the lens to the ciliary processes is so near the point of danger that atropine may add the little which is wanting to induce the malady. Ad. Weber has offered a different explanation of the phenomenon. He lays great stress upon the resistance which the contracted sphincter of the pupil offers to the advance of the lens, and argues that, by withdrawing this resistance, atropine enables the lens to advance, under the influence of the excess of pressure which he considers proper to the vitreous chamber. For reasons already stated, I believe the resistance of the iris to be infinitesimal, and conclude that its withdrawal can produce none but infinitesimal results. ⁽¹⁾ I have, moreover, proved by demonstration that the aqueous and vitreous pressures are practically equal. There is, however, another way in which, by its action upon the iris, atropine may perhaps aggravate or induce glaucoma—namely, by gathering its tissue together towards the periphery of the chamber, and thus facilitating the closure of the latter.

Eserine lowers the tension of the glaucomatous eye, and has been known exceptionally to reduce it rapidly to the normal level. Here we have the opposite effect upon the lens. So long as accommodative power remains, and possibly even after the normal action of the ciliary muscle has ceased to affect the form of the lens, the strong contraction produced by eserine slackens the suspensory ligament, and effects the

¹ Section IV., p. 109.

utmost diminution in the diameter of the lens of which it is capable. The effect of eserine upon the glaucomatous eye is, therefore, to reopen the channel and re-establish the flow of fluid from the vitreous to the aqueous chamber. The effect of the drug upon the iris doubtless aids in the removal of the glaucomatous condition, by making traction in such a way as to withdraw the iris from its peripheral contact with the cornea, and thus tends to reopen the angle of the anterior chamber. I can hardly agree, however, with Weber in believing that the sphincter of the iris, even when most strongly contracted, can suffice, unaided, to effect this disengagement by pressing the lens backwards, so long as the condition of exalted pressure in the vitreous chamber is unrelieved. It is, in my experience, in the very cases where the excess of vitreous pressure is most pronounced—namely, in acute glaucoma—that the remedial power of eserine is most remarkable. And, again, the theory which attaches so much efficacy to the sphincter of the iris fails to show how eserine can benefit glaucomatous eyes subsequent to iridectomy, which, according to many observers, it most certainly does.

If my hypothesis concerning the action of atropine and eserine in glaucoma be, in part at least, correct, their power should manifest itself in proportion, *ceteris paribus*, to the elasticity of the lens—that is to say, their action should be greatest in the youngest glaucomatous patients, smallest in the very aged. Is this the fact? My own experience does not enable me to determine, but it happens that the only patient in whom I have seen atropine bring the previous premonitory symptoms to acute glaucoma, and eserine still more rapidly reduce the high tension to the normal level, was a woman aged thirty-two. ⁽¹⁾

¹ May, 1879.—According to this hypothesis concerning the action of eserine, it would appear at first sight that the drug can avail nothing (except

The onset of glaucoma frequently occurs during periods of nervous exhaustion and muscular relaxation. It is possible that the explanation of this fact is to be found in the complete relaxation of the ciliary muscle which accompanies such conditions, especially, perhaps, in eyes which, by reason of hypermetropia, maintain, under ordinary circumstances, a more than normal tonicity of this muscle. I merely indicate the possibility of this connection, without stopping, at the present time, to carefully consider its probability. It can, in any case, hold good only for eyes in which a certain degree of elasticity in the lens still remains.

A rapid increase of presbyopia is a frequent forerunner of the other signs of glaucoma. This has generally been regarded as the first of the symptoms which are caused by the increased ocular pressure, and an experiment of my own proves that, with an increase of pressure, diminution of the accommodative range does actually occur in the healthy eye. ⁽¹⁾ The change is, however, not seldom distinctly antecedent to any certain manifestation of glaucoma, and I

by its action on the iris) in eyes which possess no accommodative power; but it is not impossible, I think, that if, by forcible continuous contraction of the ciliary muscle, the zonula be kept slackened over periods of hours or days, a slight and gradual alteration in the form of the senile lens may occur, which during brief and comparatively weak voluntary contractions would be impossible. It occurred to me lately that the *modus operandi* of eserine in glaucoma might be tested by using in its stead the drug muscarine, which is stated to cause strong contraction of the ciliary muscle without affecting the pupil, or, at any rate, with so slight an effect upon the pupil that the latter may be neutralised by a minute addition of atropine, without weakening the accommodative spasm. (Krenchel. Arch. f. Ophth., XX., I.) I therefore obtained some muscarine from Germany through an eminent English firm, and proceeded to test its action, in the first place, on my own eye, but, to my disappointment, failed, after several instillations of the solution (8 grains to 1 oz.), to obtain the slightest trace of accommodative spasm. The pupil was somewhat dilated for ten or twelve hours. With muscarine possessing the selective properties ascribed to it, it would be easy and instructive to test the possibility of slight refractive changes in the normal senile lens, in addition to ascertaining the effect of the drug in cases of glaucoma.

¹ Section III., p. 70.

would suggest that it is possibly a result of the senile change in the lens which, in certain eyes, leads to glaucoma, rather than a purely secondary effect.

Swelling of the lens, as the result of injury, is frequently accompanied by glaucoma. The rapid rise of tension which often occurs after simple laceration of the lens-capsule, and which can hardly be due to anything else than the swelling of the lens, and which, moreover, is much less common in infancy, when the lens is more globular, than in adult life, when its diameter is greater, is, I think, a strong point of evidence in favour of my theory. ⁽¹⁾

So far, this theory of glaucoma has included no reference to vascular changes. These, however, form a very prominent feature in the acute form of the disease, and are not altogether absent in the chronic. Strictly speaking, they are, I believe, secondary changes; but, as they appear to play an essential part in intensifying the obstructive change, and, moreover, to be essentially involved in the curative action of iridectomy, they must be noticed in this place instead of amongst the other secondary effects of glaucomatous pressure.

The effects of an exalted intraocular pressure are witnessed clinically in the vessels of the retina, and in those of the ciliary region. The latter only concern us here. In health the venous blood of the ciliary processes, together with that from the iris and ciliary muscle, flows, for the most part, backwards into the veins of the choroid, and escapes from the eye through the four large trunks of the vortex veins. A communication less direct, and of smaller aggregate calibre, exists between these parts and the veins which perforate the sclera in the ciliary region—the anterior ciliary veins. In glaucoma the flow through the latter is augmented, more or less, according to the type of the disease. Until recently,

¹ See pages 181—183.

this was described by most writers as a result of compression of the other channels of outlet—the vortex veins—during their oblique passage through the sclera. Microscopic examinations have, however, proved, and sections of my own illustrate the fact, that even in very advanced glaucoma these trunks still carry a considerable quantity of blood. Leber has shown that the obliquity of their course does not involve their compression during raised tension, and has a purpose connected with the accommodative movements of the choroid. He assumes the dilatation of the anterior ciliary veins to be rather a remnant of inflammatory change than a pressure effect. ⁽¹⁾ I think this supposition is untenable. The dilatation may be seen to vary precisely in accordance with changes of tension, as, for example, after the application of eserine to a glaucomatous eye, in which it can effect a reduction of the pressure; it is not absent, though often slightly marked, in chronic glaucoma, in which evidence of inflammation is entirely wanting; moreover, the patency of the vortex veins does not prove that the choroidal circulation is unretarded. Ad. Weber has, I think, given the true explanation of the symptom. He points out that the obstruction to the backward venous current is not due to strangulation of the main trunks in their passage through the sclera, but to the increased pressure which falls upon the veins throughout their whole course from the ciliary processes to the points where they escape from the inner surface of the sclera. ⁽²⁾ This pressure must reduce the amount of blood which escapes backwards from the ciliary processes, and thereby create a passive hyperæmia in the ciliary processes, and an increased escape through the anterior ciliary veins. Swelling of the ciliary processes appears, therefore, to be a natural and inevitable consequence of an increased intraocular pressure. As a matter of fact, such a swelling, or traces of its former

¹ Handbuch d. g. A., II., I., 355-6.

² Arch. f. Ophth., XXIII., I.

presence, are very frequently to be found in glaucomatous eyes. In some the ciliary processes are in a condition of extreme atrophy, as a result of long-continued pressure upon them from behind, but even in such instances the peripheral adhesion of the iris and cornea generally marks the point to which they have, at an earlier stage, advanced beyond their normal limit. Ad. Weber, who considers swelling of the processes to be the *primary* change, gives drawings which illustrate its presence in a striking degree; he also points out, in evidence of its occurrence, that small masses of pigment may not uncommonly be discovered, after iridectomy, adherent to the margin of the lens. Further, he demonstrates that, in acute glaucoma, the swelling reaches a greater intensity than in the chronic form. ⁽¹⁾ My own sections, as far as they go, support the same conclusions.

Assuming that an increase in the diameter of the lens is the primary obstructive change, it is manifest that the swelling of the ciliary processes which follows must tend directly to increase the obstruction and to intensify the glaucomatous condition; first, by narrowing still further the interval between the processes and the lens; secondly, by facilitating compression of the angle of the anterior chamber, the immediate cause of which is the compression of the ciliary processes against the periphery of the iris by the advancing suspensory ligament.

I will therefore restate my theory of glaucoma, as follows (see fig. 34):—

The cause of primary glaucoma is a diminution of the distance which separates the margin of the lens from the ciliary processes. Two factors combine to produce this diminution, viz.:

1. *Increase of the diameter of the lens.*
2. *Swelling of the ciliary processes.*

¹ Arch. f. Ophth., XXIII., I., 58.

The former is usually, if not invariably, the primary change, and is itself the indirect cause of the latter.

Before passing on, let me recapitulate my reasons for considering swelling of the ciliary processes to be the secondary and not the primary change.

1. The time of life at which glaucoma occurs corresponds with that at which the lens undergoes a senile change of form.
2. This change explains the swelling of the ciliary processes.
3. It is not proved that such swelling occurs spontaneously, independently of a previous rise of intraocular pressure.
4. The conditions which have been assigned as causes of a primary swelling of the ciliary processes—*e. g.*, cardiac and respiratory disturbances, &c.—are constantly witnessed without glaucoma, and bear no definite relationship to the period of life when glaucoma occurs.

It now remains to be considered whether the acute form of glaucoma can be explained on the basis of this theory, and whether the conditions described as being essential to the glaucomatous process are compatible with the curative action of iridectomy.

In acute glaucoma we see an intense development of those vascular changes which in chronic glaucoma are only just discoverable, and we see something more ; we see active arterial hyperæmia as well as venous obstruction. In acute glaucoma the swelling of the ciliary processes reaches its maximum intensity.

It would appear that when the primary obstruction is established suddenly, so as to allow little time for the blood

stream to adapt itself to the altered conditions, either by a compensation as regards escape, or a gradual diminution of supply, then the ciliary processes swell rapidly, and at once establish those conditions of complete obstruction which, under other circumstances, take months or years for their development.

What are the causes, then, which can suddenly establish the primary change? In what way can the interval between the lens and ciliary processes be suddenly diminished? First, as regards the lens:—Can we ascribe an acute attack of glaucoma to a sudden alteration of its form? I will here repeat the suggestion that in an eye in which the margin of the lens is already in dangerous proximity to the ciliary processes, the action of atropine may add the little that is wanting to embarrass the escape of fluid from the vitreous to the aqueous chamber. Then, with the first access of pressure in the former, the lens and suspensory ligament advance and close the angle of the anterior chamber; the pressure rises, the ciliary processes swell up; the suddenness of the obstruction allows no time for compensatory changes in the blood stream, but, on the contrary, excites an active hyperæmia by reason of the pain and irritation which is set up. The condition is manifestly one which tends to intensify itself, and can find relief only in such a lowering of the intraocular pressure as will remove the venous hyperæmia and permit the swollen ciliary processes to subside. To attack the primary cause of the disaster by counteracting the effect of the atropine by eserine, and thus to withdraw the margin of the lens from the ciliary processes and reopen the filtration channel, appears to be the natural remedy, and I have known it to be followed by complete success.⁽¹⁾ Usually an artificial method—iridectomy—by which the superfluous fluid is rapidly drained away, and

¹ Case 19, Section VII.

the engorged vessels encouraged to recover their normal calibre, is necessary.

Iridectomy cannot directly influence the diameter of the lens; its permanent efficacy, therefore, should depend chiefly upon subsidence of the ciliary processes, and should be most conspicuous when their engorgement is greatest—namely, in *acute* glaucoma. This, as we know, it is.

It is, I think, just possible that changes in the lens may be concerned in inducing acute attacks without the aid of atropine, or, if not typical acute attacks, the periodic exacerbations so frequently observed. Those conditions which in all eyes weaken the accommodative power, and in many, especially the hypermetropic, produce extreme asthenopia—*e.g.*, muscular and nervous exhaustion, due to illness, grief, privation, cold, &c.—are frequently associated with exacerbation. I suggest this, however, with the utmost hesitation; for many of these attacks, especially those associated with obvious nerve disturbance, a better explanation is to be found.

In the second place, we have to consider the occurrence of sudden vascular disturbance. In chronic glaucoma the vascular changes are those of obstruction and retardation; the hyperæmia, which arises at certain points of the blood stream, is purely passive. In acute glaucoma active arterial hyperæmia is superadded; here, as elsewhere in the system, it intensifies the effect of the obstructive change. The capillary engorgement which results manifests itself in exudation of serum and of blood corpuscles.

How is the arterial hyperæmia brought about? It appears to arise in two ways:—

1. As a reflex phenomenon induced by irritation of the eye itself; as, for example, by the action of any external irritant upon a predisposed eye,

or by the sudden induction or aggravation of the obstructive changes, compression of the iris, &c., by the action of atropine.

2. As a part of a more general hyperæmia affecting the vessels of the head, and associated frequently with some form of nervous excitement.

It is hardly necessary to refer to the connection between emotional disturbance and increased afflux of blood to the head ; it is constantly manifested in suffusion of the cheeks, turgidity of the veins, throbbing of the arteries, even proptosis, with simultaneous coldness of the feet. The outbreak of glaucoma after such periods of excitement has been very often noted. I have seen a rapid recurrence of high tension and pain soon after operation, after a restless night, with crying and intense suffusion of the head and neck.

It has been shown that an increased afflux of blood to the eye raises the intraocular pressure, and that if the afflux be extreme, as in certain artificial states produced experimentally, the pressure rises to a very great height. ⁽¹⁾ The excess thus produced disappears, with more or less rapidity, on the cessation of the causes which call it forth. A persistent, self-maintaining excess of pressure, comparable with the glaucomatous condition of the human eye, has never been observed to follow such experiments. In the eyes of animals, then, at any rate, and undoubtedly in the human eye also, something more than a temporary increase of blood supply is required in order to establish the glaucomatous condition. According to the theory I have advanced, this extra factor is a diminution of the distance which separates the lens from the ciliary processes. It is not necessary to

¹ Section IV., p. 100.

assume that the acute attack has been preceded by glaucoma in a simple form: we have only to assume that the lens and ciliary processes are already in dangerous proximity. A little local irritation, or a little cerebral hyperæmia, quickens the blood stream to the eye; secretion flows faster into the chambers; its escape from the vitreous to the aqueous chamber cannot keep pace with the increased supply; the vitreous pressure rises, the angle of the chamber closes, the ciliary processes swell and intensify the obstruction, and acute glaucoma is established. Does this not explain the indubitable but hitherto mysterious connection between neuralgia and glaucoma? One patient suffers for half a lifetime from intense periodic neuralgia, accompanied at every recurrence with more or less pain in the eye; not till she reaches fifty years of age does glaucoma supervene, and then it comes suddenly and in manifest connection with one of these attacks. Another patient suffers the most intense neuralgia at frequent intervals, and still does so until his death at an advanced age, but never becomes glaucomatous. In each case the neuralgic attacks are associated with manifest ocular hyperæmia. The different effect of the malady in the two cases must, presumably, depend upon differences in the eyes which are subjected to its influence; in the eye which becomes glaucomatous the predisposing condition appears to be absent during early and middle life. From clinical observations of this kind, of which many are on record, we may, I think, derive a strong presumption in favour of the theory which has been advanced.

In the later stages of glaucoma the conditions of obstruction which are opposed to the normal secretion and excretion of the intraocular fluid become complete.

The angle of the anterior chamber is totally obliterated by broad adhesion of the iris with the cornea; the fluid in

the anterior chamber is imprisoned there, and offers an unyielding resistance to the further advance of the lens; no escape of intraocular fluid is possible, hence no fresh supply can enter either aqueous or vitreous chamber unless their space is enlarged by distension of their walls. The stagnation of the current of the intraocular fluid—of the nutrient supply, that is, to the vitreous body and the lens—is manifested in the degeneration of these structures.

THE INFLUENCE OF IRIDECTOMY.

Iridectomy lowers the tension of the glaucomatous eye—

Firstly, by the immediate evacuation of the aqueous chamber.

Secondly, by tending to cause the subsidence of the obstructive changes—*i. e.*, the narrowing of the “circumlental space” and the compression of the angle of the anterior chamber.

Thirdly, by the establishment of a permanent artificial channel of exit for the intraocular fluid.

It is unnecessary to consider the hypothesis which ascribes the influence of iridectomy to the section of nerve fibres, for, if this hypothesis be correct, the pathological description of glaucoma given in this essay is fundamentally erroneous.

It appears that, in exceptional cases of very recent and acute glaucoma, the mere evacuation of the anterior chamber may suffice to re-establish the normal tension; as, for example, when a cure is effected by a very faulty iridectomy, the incision being entirely in the cornea, and the iris, with its sphincter undivided, being left incarcerated in the wound. Such a result is intelligible if it be true that, of the two factors primarily concerned—viz., alteration in the lens and

swelling of the ciliary processes—the latter greatly preponderates in acute glaucoma. The rapid draining away of fluid from the eye during the few hours which elapse before the corneal incision closes may cause this swelling so completely to subside as to restore the eye to its previous condition—a condition, doubtless, of proneness to glaucoma, but still compatible with the normal filtration process, provided that no unusual stress is thrown upon them by a sudden afflux of blood or any of the special exciting causes of acute glaucoma.

Experience has shown, however, that for the majority of cases a scleral incision is imperative. The essential importance of the latter, as compared with the excision of the iris, is proved by the complete reduction of glaucomatous tension which the operation of sclerotomy is able to effect when complications with the iris do not ensue. Experience has shown, moreover, that the more nearly this incision coincides with the normal place of exit—the angle of the anterior chamber—the more certain, *ceteris paribus*, is its efficacy; and this, be it observed, is no theoretical inference, for it was established long before the physiology of this region was understood.

The exact nature of the artificial channel of escape produced by the scleral incision is still unknown. It is doubtful whether the intraocular fluid permeates the cicatricial tissue which closes the wound, and, escaping into the subconjunctival space, is absorbed by the vessels external to the sclera, or whether during the healing process new vascular channels are established in this tissue and the sclera so as to reopen a passage for the fluid into Schlemm's canal or the adjacent venous plexus. This point is obviously not to be cleared up by the examination of unsuccessful iridectomy wounds; and eyes which have been cured by iridectomy rarely fall into the hands of the microscopist.

There is some ground for believing that the cicatricial tissue itself is permeable by the intraocular fluid. The cicatrix is not unfrequently somewhat stretched and elevated by a slight return of pressure in excess during the few weeks following the operation, and it appears to gain by this attenuation a degree of permeability, as evidenced by the final subsidence of the excess of pressure, which it did not at first possess. A slight elevation of the conjunctiva, due to fluid beneath it, is sometimes discoverable after union is apparently complete. V. Wecker relates the case of a glaucomatous patient who, after iridectomy, was accustomed to relieve the slight recurrences to which he was still liable, by making pressure upon his eye with his fingers; the effect of this, as witnessed by v. Wecker himself, was to extrude a small quantity of fluid beneath the conjunctiva in the neighbourhood of the cicatrix, and to remove at once the glaucomatous symptoms. ⁽¹⁾

Again, the intraocular fluid appears to find exit with little difficulty through the adventitious tissue which replaces the cornea in cases of total staphyloma with universally adherent iris. ⁽²⁾

The difficulty of explaining the permeability of the incised tissues would be greatly increased if it were necessary to suppose that the whole or even the greater part of the intraocular fluid escaping from the eye must find exit, subsequently to the operation, through the artificial channel. This is certainly not the case. A *complete* obstruction of the angle of the anterior chamber occurs, probably, only in the acutest cases of glaucoma, if even in these; in other cases the condition is that of *gradual narrowing*, with increasing retardation of the filtration process. The effect of iridectomy is not only to relieve the angle of the chamber from further

¹ *Thérapeutique Oculaire*, Part 1, p. 381.

² See Case 9, specimen 9, this Section, p. 194.

compression, but to promote the restoration of its patency, so far as this may still be possible, by abolishing the swelling of the ciliary processes.

If the foregoing theory concerning the primary cause of glaucoma be correct, the curative action of iridectomy must involve two distinct results—namely :

1. The reopening of the angle of the anterior chamber.
2. The reopening of the space between the lens and the ciliary processes.

Its power to effect the former will depend upon the recentness of the closure, and its power to effect the latter upon the degree to which the narrowing depends upon swelling of the processes, rather than upon change in the lens. In each of these particulars acute glaucoma presents more favourable conditions than does the chronic form.

On the basis of this theory, I shall now attempt to sketch the course of events subsequent to iridectomy in each form of the disease.

Iridectomy in acute glaucoma. The aqueous chamber is evacuated, the lens, suspensory ligament, and iris advance towards the cornea, and the vitreous pressure is immediately lowered ; the pressure upon the choroidal veins being diminished, the venous blood from the ciliary processes flows through them in increased quantity, and the swelling of these organs begins to subside ; the space between the processes and the lens (I will call it, for the sake of brevity, the “circumlental space”) is widened, or, if previously abolished, is reinstated, and fluid again passes from the vitreous to the aqueous chamber. As union of the wound begins, the anterior chamber is re-established, the lens, suspensory ligament, and iris recede, and the angle of the chamber, being

now relieved from pressure, reopens. As regards the permanency of the cure, the iridectomy has provided a kind of safety valve, which guards the eye against a repetition of the attack should the special exciting cause at any time recur; the permeable cicatrix facilitates the escape of any temporary surplus of fluid, and the absence of a sector of the iris renders this portion of the angle of the chamber less liable to closure during a slight access of vitreous pressure than it was before the operation.

Iridectomy in chronic glaucoma. The course of events is essentially the same as in the previous case, but the resolution of the glaucomatous condition is less complete for two reasons—namely :

1. The primary obstruction—*i. e.*, the narrowing of the “circumlental space,” depends less upon vascular engorgement, which is reducible, than upon change in the lens, which is irreducible.
2. The secondary obstruction—*i. e.*, the closure of the angle of the anterior chamber—though probably less complete than that which occurs in acute glaucoma, has lasted much longer, and is less capable of rapid and complete resolution.

The anterior chamber is evacuated and abolished, the ciliary processes undergo no rapid contraction as in acute glaucoma, but still retract sufficiently to increase somewhat the flow of fluid through the “circumlental space;” very gradually, and in constant danger from a recurrence of excessive vitreous pressure, the anterior chamber is restored, and its angle is no longer subjected to excessive pressure from behind; its patency is, however, restored but imperfectly, by reason of structural changes due to long-continued pressure, and for

the future, if the eye is to remain free from glaucoma, the artificial channel produced by the iridectomy must provide an exit for a large portion of the fluid which passes through the chamber. Hence, the uncertain action of iridectomy in chronic glaucoma, the persistence of a slight excess of tension, the imperfect restoration of the anterior chamber, the recurrence of the malady after an interval of some months or years.

Malignant glaucoma is understood to mean the aggravated condition which, in exceptional cases, follows the performance of iridectomy. Provided the operation be correctly performed, this malignant course is limited almost entirely to chronic cases (excluding, of course, the hæmorrhagic group). Its occasional occurrence appears to be almost inevitable under the conditions which have been here theoretically laid down.

The main characteristic of this disaster is that the lens remains in close and forcible contact with the iris and cornea, the vitreous pressure increasing rather than diminishing. In normal cases a drainage of the vitreous chamber is effected by the operation; in these malignant cases the escape of fluid from the vitreous chamber appears to be even more impossible than it was before. ⁽¹⁾ Theoretically, we must assume that the "circumlental" space does not reopen. If the lens be of unusually large diameter; if its margin, previous to operation, be in close proximity to the ciliary processes; if the latter present no great enlargement which can immediately subside on the evacuation of the anterior chamber; and, finally, if the vitreous pressure be greatly in excess, and the anterior chamber shallow—we have a state of things which must almost of necessity be aggravated by the withdrawal of the aqueous fluid. Driven forward by the vitreous, the lens advances against the cornea, and compresses the intervening iris; its periphery bears against the insertion

¹ Fig. 35.

of the iris, and lies to the *outer* side of the internal opening of the wound—it thus blocks up the exit through which fluid might otherwise drain away from the portion of the ciliary processes corresponding with the coloboma; the vitreous pressure, but slightly lowered by the escape of the scanty aqueous fluid, soon rises to and exceeds its former height; if the lens have any elasticity remaining, its diameter is still further increased by the excessive compression to which it is subjected between vitreous and cornea, and its periphery is forced into the depression between the ciliary processes and the iris. ⁽¹⁾

What clinical evidence can be advanced in support of this theoretical description? Ad. Weber describes the conditions observed by him in an eye excised on account of “malignant” progress after iridectomy, as follows:—“From the ora serrata forwards the tunics were all in apposition, but the tips of the ciliary processes were bent backwards, and so came to lie behind the equator of the lens, which was wedged fast between these and the origin of the iris; there was thus a complete luxation of the lens in its capsule, and without injury to the suspensory ligament.” ⁽²⁾ In this case hæmorrhage from the choroid had occurred. Weber states it as his belief that “luxation of the lens *into the groove* between the base of the ciliary body and the origin of the iris” is the cause of malignant glaucoma. The term luxation, as here applied, is, I think, a misnomer. The lens is no more luxated than it is after every iridectomy, in non-glaucomatous as well as in glaucomatous eyes, provided that the aqueous chamber be evacuated. The peculiarity, and, as I believe, the *essential cause* of the unfavourable course in the present case is the excessive diameter of the lens. I would especially call attention to Weber’s description, as it proves that the diameter of the lens was considerably greater than the

¹ See note, page 174.

² Arch. f. Ophth., XXIII., I., 84.

diameter of the circle of the ciliary processes. It is obvious that the incision, and especially its internal opening, must, in such a case, lie very considerably within the circle corresponding to the margin of the lens, and that, in the absence of a circle of swollen ciliary process to prevent the close application of the lens to this circle, the incision must be covered and obstructed.

H. Pagenstecher, in describing a specimen of "malignant glaucoma," says, "I think the lens is to blame as the cause of this appearance; it was forcibly pushed forwards, dragging the ciliary body forwards in like manner, pressing the ciliary processes partly into the ectasia, and having its own margin wedged in the ectatic region." ⁽¹⁾ "I am of opinion that the lens acts in such cases as a foreign body, that it presses against the ciliary body and ciliary processes, and that it thereby excites a hypersecretion which is then superadded to the ordinary glaucomatous process." ⁽²⁾ It is unnecessary here to repeat the explanation which I should oppose to this idea of irritative hypersecretion. I quote the passage merely because it notes the contact of the margin of the lens with the ciliary processes.

My own specimen No. 2 exhibits a somewhat unusual form of malignant glaucoma. ⁽³⁾ Here very extensive hæmorrhage between sclera and choroid, and into the substance of the vitreous, followed iridectomy. The lens was driven forwards and tilted on one side, with the more advanced margin forcing the lips of the incision asunder, and protruding from the eye. Such hæmorrhage and such a displacement of the lens would induce a "malignant" course in any case of glaucoma; cases of this description cannot, therefore, be employed as illustrations of the conditions which induce the malignant course in cases of simple chronic

¹ Heidelberg Congress, Bericht über, &c., 1877, page 13.

² 3 Ibid, p. 23. ³ Case 2, Section VII.

glaucoma, in which no intraocular hæmorrhage occurs, and in which the incision cicatrises in the ordinary way.

Convinced that "malignant glaucoma" depends essentially upon the so-called luxation above described, Weber has devised and practised with success a procedure by which the lens may be restored to its normal position and the eye rescued from destruction of sight. He lays stress upon the importance of following the directions given with great exactness. I therefore translate the passage in full. ⁽¹⁾ "The sclera is to be punctured with a double-edged broad needle, at a point 8 to 10 mm. from the corneal margin, and by preference at the outer side, in the horizontal meridian; and while the scleral wound is made to gape by a quarter-rotation of the instrument on its long axis, pressure is made, by means of the upper lid, upon the cornea, in a direction perpendicular to the plane of the coloboma, in the direction of which the lens is usually protruded most; the pressure is to be slight at first, and gradually increased. The patient should persistently 'fix' the operator during this manœuvre, and thereby enable the latter to judge correctly of the position of the cornea, in spite of its being nearly covered by the upper lid; further fixation with instruments, and the distortion of the form of the globe which is thereby produced, can thus be avoided. The maximum pressure is continued for a minute or a minute and a half, so as to allow time for the re-formation of the aqueous humour; blood is generally mixed with the latter, but disappears in four to six hours—a sign that the filtration channels are reopened. A light pressure bandage and rest in the recumbent position for twenty-four hours completely guarantees the result which has been attained. Chlorform is necessary only when the ciliary region is very sensitive, and then the Graefe cataract spoon may be used conveniently as a means of pressure. I have

¹ Arch. f. Ophth., XXIII., I.

hitherto employed, in addition, the following local treatment before and after the operation, and I believe that it greatly aids the reduction. One or two days beforehand atropine is applied a few times, so as completely to relax the sphincter of the ciliary muscle (sphincter ciliaris), and, about twenty minutes before operating, one or two drops of a two per cent. eserine solution are applied; the commencement of pain in the brow is the signal to seize the instruments. The eserine treatment is continued for some days, until the margins of the coloboma, which are almost invariably adherent to the cornea, are freed. The tension, which previously was extreme, is normal by the evening dressing. The vitreous opacities, which are always present, disappear more rapidly than after successful iridectomy, probably because, in addition to the arrest of the morbid process, a considerable quantity of turbid vitreous escapes and is replaced by new. The most suitable time for carrying out the operation is between the tenth and twentieth days after the iridectomy, by which time the corneal wound is already sufficiently firm to stand such pressure. Clear signs that an acute attack is imminent would necessitate an earlier interference. I have not as yet ventured on this proceeding while the incision is still open. Neither have I had opportunity to attempt it in cases of luxation of long standing. I would give a warning against any modification of this proceeding; I have amply proved that neither the pressure nor the scleral puncture alone suffices to bring about the replacement."

It so happened that on the very day when I first read this passage a patient of my own in hospital presented exactly the conditions which Weber has succeeded in relieving. ⁽¹⁾ I performed the operation as nearly as I was able according to the directions given, but did not succeed in removing the lens from its position of contact with the cornea. I am

¹ Case 20., Section VII.

inclined to think that I did not obtain a sufficient escape of vitreous, and that perhaps the pressure was not exercised in a direction perpendicular to the coloboma, but rather over the centre of the cornea. I am anxious, therefore, to throw no discredit upon a proceeding which is stated to be so beneficial, and the non-success of which, in my own hands, may have been due to an incorrect performance. In answer to a letter in which I asked for further information as to the success of the operation, Dr. Weber favoured me with the following reply:— “Concerning the replacement of the lens, it does not succeed in every case; but in my hands at least it has not yet failed; neither does the lens again come forward. Of this I have examples of two years’ standing. The paracentesis of the vitreous must be tolerably broad, so that during the pressure the vitreous may flow out readily. The operation is not dangerous.—⁽¹⁾ * * * *

SECONDARY GLAUCOMA.

Every variety of secondary glaucoma, probably without exception, is associated with changes which retard or arrest the escape of the intraocular fluid through the angle of the anterior chamber.

I shall endeavour to establish the truth of this statement by examining in succession all the principal varieties of the

¹ Since writing the foregoing on the subject of malignant glaucoma, I have been interested to observe the extreme change of form which the lens will undergo when compressed between the cornea in front, and a gliomatous mass occupying the whole vitreous chamber. Fig. 54 represents roughly the form of the lens. This eye was excised by my friend, Mr. Hodges, of Leicester, from a child. The lens was doubtless highly elastic, and would yield to pressure very much more readily than that of an elderly person. It suggests, however, that if the lens of the latter still maintains any elasticity or compressibility, the condition in which it finds itself when pressed against the cornea in the way before described, is one which must intensify itself. The higher the vitreous pressure rises, the greater becomes the diameter of the lens, and the more complete the obstruction.—Case 16, fig. 54, slide 16a. See account of case by Mr. Hodges, *Lancet*. February 8th, 1879.

malady, pointing out the way in which the obstructive change is brought about in each, and demonstrating the facts as far as possible by means of microscopic sections and drawings from the eyes which I have dissected, and by the clinical evidence of certain other cases.

Posterior synechia, when extensive, is a fruitful source of glaucomatous complication. It leads to closure of the angle of the anterior chamber in more ways than one.

When the whole circle of the pupil adheres to the lens capsule, the intraocular fluid from the ciliary processes can no longer pass forward into the anterior chamber. ⁽¹⁾ It collects between the iris and the lens and suspensory ligament, and distends the posterior aqueous chamber. The iris is bulged forward until, by its advance, it so far narrows the angle of the anterior chamber as to preclude the further escape of fluid through it. The fluid still remaining in the anterior chamber is closely imprisoned there, and presents an unyielding resistance to any further advance of the iris, or of the lens. Fluid is still secreted, though in decreasing quantity, into the vitreous chamber, and continues to collect there until its exalted pressure, reacting on the secreting organ, arrests the further supply, or reduces it to a condition of equilibrium with the small amount which still finds a passage forwards through the pupil, and outwards through the narrowed angle of the chamber. Cases of this kind are frequently met with in hospital practice. The effect of iridectomy is most striking. No sooner is a communication re-established between the posterior and anterior aqueous chamber than the disease—*i.e.*, the glaucomatous complication, vanishes. It is not necessary here to adopt the scleral incision and the complete removal of the iris to its insertion, as in primary glaucoma; a corneal incision, through which a satisfactory

¹ Case 28, Section VII.

"artificial pupil" can be made, suffices; for unless the mischief be of long duration, in which case the eye is probably useless, there is probably no actual irremediable adhesion of the iris and cornea, but merely a close contact, which immediately subsides with the restoration of the anterior chamber. There is not, in general, any marked advance of the lens, for the pupillary margin is depressed as a deep central pit, so that the periphery of the iris probably escapes the compression by the ciliary processes which it undergoes in primary glaucoma. Fig. 27 illustrates the distension of the posterior chamber by imprisoned albuminous secretion. The total posterior synechia in this case was caused by a perforating wound of the lens and vitreous. The case will be again referred to. The glaucomatous tension, in cases of annular posterior synechia, if unrelieved by iridectomy, is liable to give place to a subnormal tension, indicating an extension of inflammatory changes to the ciliary processes and a consequent impoverishment of the vitreous humour. ⁽¹⁾

When the whole posterior surface of the iris adheres to the lens capsule and suspensory ligament, the obstructive change is somewhat different to that just described. The posterior aqueous chamber is abolished, and secretion from the apices of the ciliary processes is no longer possible. Secretion into the vitreous chamber still continues, until arrested by the reaction of the exalted vitreous pressure. The lens, suspensory ligament, and adherent iris advance

¹ NOTE.—May, 1879. It happens not unfrequently that eyes in which glaucomatous tension has arisen in connection with prolonged iritis and synechia posterior are reduced by iridectomy to a condition of subnormal tension which is persistent. Such a case has recently been under my care. The subnormal tension is, in my opinion, a certain indication that the ciliary processes have been damaged by the foregoing inflammation. Previous to the iridectomy their secretory activity, although impaired, is sufficient, by reason of the obstruction at the outlet, to raise the tension of the eye to a high pitch; but no sooner is the channel of escape reopened than the impoverishment of the supply becomes manifest.

until, by so doing, they close the angle of the anterior chamber by obliteration of the ligamentum pectinatum, and imprison the remaining aqueous fluid. Here the compression is affected much, as in primary glaucoma, and the effects are the same as in that condition.

Fig. 19 represents a section from an eye blinded by glaucomatous pressure following total posterior synechia. (During manipulation of the specimen the lens, with most of the uveal surface of the iris, became detached.) There is here no peripheral adhesion of the iris, and cornea. Its absence is explained by the total adhesion of the iris to the lens and suspensory ligament; for in the absence of any space between these structures the ciliary processes cannot enlarge anteriorly so as to push the periphery of the iris forwards in advance of the rest of the membrane, as in primary glaucoma. The escape of the intraocular fluid is, however, as completely arrested as though the peripheral adhesion existed, for not only is the ligamentum pectinatum so compressed as to be almost obliterated, but the passage of the intraocular fluid into the remnant of the anterior chamber is rendered impossible by the general adhesion of the iris to the lens.

Anterior synechia, especially if a considerable portion of the circle of the iris is involved, readily leads to narrowing of the corresponding part of the angle of the chamber. A perforating ulcer, or a wound of the cornea, is commonly the starting point. The iris prolapses, and is included in the cicatrix; it is tightly stretched between the new adhesion and its peripheral attachment; the ligamentum pectinatum is compressed at the corresponding portion of the circle by the undue proximity of the cornea and iris. If the compressed portion is large enough to materially affect the escape of the intraocular fluid, the tension of the eye rises. When such a condition is complicated by an injury of the lens, with

posterior adhesions of the iris to the capsule or to the exposed lens tissue, the danger of glaucomatous complication is greatly increased. Fig. 20 represents the conditions inducing glaucoma after a wound of the cornea, iris, and lens. The cicatrix includes cornea, iris, lens capsule, and lens substance in one confused mass. At the far side of the chamber—*i.e.*, in the portion where the iris was left intact by the injury—we see a broad adhesion of the iris to the cornea, evidently produced by pressure applied to the posterior surface of the iris through the intervention of the ciliary process. How does this part, removed as it is from direct connection with the injury, come to suffer such compression? The swelling of the injured lens, together with the iritic adhesion already described, retard the passage of the intraocular fluid from the vitreous into the aqueous chamber; the pressure in the vitreous chamber is exalted above that in the aqueous chamber, the lens and suspensory ligament advance, and compression of the angle of the chamber throughout its whole circumference ensues, precisely as it does in primary glaucoma.

Glaucoma following the extraction of senile cataract appears, at first sight, to be irreconcilable with the theory of glaucoma which I have advanced. A very pronounced case which recently occurred in my own practice, however, appears to me to afford strong evidence in its favour, both by the manner of its onset and the manner of its disappearance. I will here briefly summarise the process.⁽¹⁾ The lens is removed without rupture of the suspensory ligament or posterior capsule; plastic iritis, possibly due to the retention of cortical fragments, sets in; the iris adheres throughout to the suspensory ligament and capsule; the portions of these membranes occupying the pupil and the coloboma are coated over with

¹ Case 36, Section VII.

lymph. A thickened impervious partition is thus created between the aqueous and vitreous chambers; the tension rises in the vitreous chamber; the angle of the anterior chamber is closed by the advance of the partition; a certain amount of turbid fluid remains imprisoned in the anterior chamber; intense glaucoma is rapidly induced. Repeated leeching, atropine instillations, paracentesis of the anterior chamber have little or no influence on the glaucomatous tension; the eye is evidently doomed to destruction unless the tension is quickly relieved. During the intensity of the inflammation the partition which separates the aqueous and vitreous chambers is forcibly ruptured by operation; the pressure in the two chambers is equalised; the compression of the angle of the anterior chamber is removed; the glaucomatous tension subsides; the iritis gradually disappears; the eye ultimately recovers excellent vision. Such is the outline of the case. I have never before seen an eye recover from such a formidable condition, coming on within a week of the extraction of the lens in an old and feeble person. I would draw particular attention to the case, not only as a point in my argument, but as affording a lesson as to the treatment which should be adopted when such complications arise. I have seen no other case of glaucoma immediately following the operation, but I have more than once seen patients, who have been operated on by others, in whom a glaucomatous tension persisted some months after the operation. Adhesions of the iris to the capsule have always been present. The pupil has been reopened, but too late to avoid the occurrence of the peripheral adhesions of the iris and cornea, which inevitably are formed if the vitreous pressure remain long in excess, and which subsequently maintain an excess of tension, although the communication between the vitreous and aqueous chambers be re-established.

Buphthalmos is a term which I think might with advantage be discarded. It in no sense represents an individual disease ; but is applied, so far as I know, to secondary glaucoma from almost any cause, provided it occurs in eyes which, by reason of the patient's youth, are capable of considerable enlargement by the distending force of increased internal pressure. That these cases belong essentially to the class of secondary glaucoma cannot be doubted, for we find cupping of the optic disc, contraction of the field of vision, and enlargement of the episcleral veins ; we find the tension of the eye in excess ; and we find the characteristic changes at the angle of the anterior chamber on which the excess of tension depends. ⁽¹⁾

In three cases which I have examined lately iritic adhesions are the starting point of the glaucoma. In one ⁽²⁾ the disease was probably in the beginning syphilitic keratitis—the patient has the characteristic marks of inherited syphilis—becoming subsequently kerato-iritis. One eye, long blind, bulging, and painful, was excised. Adhesion of the iris and cornea extended round the greater portion of the periphery of the chamber. The lens was shrunken, probably through injury in operation years ago ;—iridectomy had been twice performed, without benefit ; the cicatrices and adherent portions of iris connected with them were visible in the microscopic sections. The anterior chamber was very large and deep—an unusual condition in glaucomatous eyes. It appears probable that the glaucoma was originally caused by iritic adhesions, advance of the lens, and closure of the angle of the anterior chamber with the formation of peripheral adhesions. Some time later, perhaps through the effect of the operations, the lens suffered disorganisation, and, together with the iris, contracted into a flat, tightly-stretched, membranous partition, separating the vitreous and aqueous

¹ See Cases 4, 14. Figs. 21, 22, 23, 36, 37, 38, 48, 51.

² Case 4, Section VII.

chambers. An open connection, through which fluid could pass, may have existed between the chambers subsequent to the operation, but, by reason of the unrelieved obstruction at the angle of the anterior chamber, may have been useless as regards the reduction of the tension.

In the second case, the starting point of which was variola soon after birth, the glaucoma had evidently been brought about by total occlusion of the pupil in the way already described. ⁽¹⁾

In a third case the original mischief was a wound of the ciliary region or adjacent cornea. Plastic iritis led to total posterior synechia in the wounded eye; sympathetic iritis caused a very similar condition in the other. Secondary glaucoma, with much distention of the globes, was the result.

Staphylomatous conditions of the cornea are not a cause of glaucoma, but they are a frequent manifestation of its presence. Iritic adhesions, such as have already been discussed, are usually associated with these changes, and explain the excess of tension. ⁽²⁾

Traumatic cataract is frequently associated with adhesions of the iris. So far as the latter are concerned, its relation to secondary glaucoma need not be repeated; but another agent is here involved, for we see the most pronounced rise of tension rapidly induced by wounds of the lens—*e.g.*, needle operations, *when the iris is entirely uninjured*. The cause is, I believe, the increased diameter of the lens which its swelling brings about, and the conditions which ensue are similar to those which exist in primary glaucoma; the essential change in each is *the increased proximity of the lens-periphery to the circle of the ciliary processes—possibly their contact*. The tension may rise to T + 2 the day after the

¹ Case 14, Section VII.

² See Case 9, Section VII.

operation, remain at that height for a day or two, or more, and then spontaneously subside, having caused no pain, and hardly a trace of vascular disturbance beyond some enlargement of the episcleral veins. Such conditions are certainly more consistent with a theory of obstructed filtration, than with the formerly accepted hypothesis of irritative hyperscretion.

It has been suggested that the cause of the excess of tension is the presence of lens matter—dissolved, or in the form of débris—in the anterior chamber, and a consequent obstruction of filtration through the ligamentum pectinatum. My own observations do not accord with this idea. The tension rises while as yet little or no lens matter has escaped through the capsular opening; it subsides long before the fragments which subsequently fall into the anterior chamber are removed; in fact, the extrusion into the chamber of swollen fragments of the cortex, appears more nearly to correspond with the fall of tension. In short, the onset and the subsidence of the glaucomatous condition appear to be directly related to the general enlargement of the lens, and its subsequent return to smaller dimensions. The process is as follows:—The capsule is opened, aqueous fluid enters, the lens fibres swell by imbibition, the elastic capsule is distended in all diameters; its periphery approaches the circle of the ciliary processes—it may possibly come into close contact with them—the suspensory ligament meanwhile retracting, by reason of the traction of the choroid; the passage of fluid from the vitreous to the aqueous chamber is retarded sufficiently to create an excess of pressure in the former; the lens and suspensory ligament advance, the angle of the anterior chamber is compressed, and glaucoma is established. After two, three, or more days the swollen lens is reduced in bulk, either by solution or extrusion of a portion of its fibres; the elastic capsule contracts, its periphery retires from the ciliary processes, and reopens a channel from the vitreous to

the aqueous chamber; the excess of fluid in the vitreous chamber escapes, the pressures are equalised, the lens and suspensory ligament retire, the angle of the anterior chamber, relieved from the pressure from behind, reopens, and the glaucomatous condition is at an end.

I have described these successive steps in detail, as I hold that they constitute a strong argument for the theory of primary glaucoma which I have advanced.

Partial dislocations of the lens induce glaucomatous complications, I presume, by reason of the direct pressure of the periphery of the lens against the periphery of the iris throughout that portion of the circle towards which the lens is displaced, and consequent closure of the angle of the anterior chamber in this situation. I have, however, no specimen illustrating this condition, and have had no opportunity of examining an instance of the complication since my attention has been directed to this particular point.

Dislocation of the entire lens into the anterior chamber induces glaucoma, frequently of the most intense type, in a way which, I think, affords another convincing proof of the essential dependence of this condition upon an arrested escape of the intraocular fluid from the anterior chamber. Under normal conditions, the intraocular fluid, in passing from the posterior to the anterior aqueous chamber, meets with no resistance at the pupil, the edge of which is raised from the surface of the lens to an imperceptible extent by the minute current; but no sooner is the relation of these parts reversed than the stream which previously kept the passage open, tends to close it with constantly increasing pressure. The iris is applied to the posterior surface of the lens, the intraocular fluid, pressing against it more and more strongly, cuts off the possibility of its own escape, and an intense glaucoma

is rapidly established. At first, no doubt, the angle of the chamber, far from being closed, is more widely opened than usual by the backward displacement of the iris; for the periphery of the lens never, probably, reaches (in the youthful eye) quite to the ligamentum pectinatum. But this openness is manifestly of no avail, since the intraocular fluid can no longer *enter* the anterior chamber. Nothing could be better adapted in form, size, and surface to accomplish this complete blockade than the lens. When the vitreous pressure has become extreme, it is not unlikely that the periphery of the iris, where it is unsupported by the lens, is pushed forwards so as to effect a partial compression of the ligamentum pectinatum; indeed, it seems possible that this very pressing forward of the periphery of the iris may suffice sometimes to draw the pupillary margin round the margin of the lens, and thus effect a spontaneous cure of the glaucomatous condition, such as occasionally occurs. The degree of tension, the vascular injection, the pain, and the constitutional disturbance—*e.g.*, vomiting and nervous prostration—may be quite equal to those which accompany the acutest primary glaucoma, and yet they subside with great rapidity when the obstruction is removed, either by operative extraction or by return of the iris to its former situation. I record four cases of this accident. In two there was intense glaucoma; in the other two there was none, or at most a doubtful history of it in one. ⁽¹⁾ How is the difference to be explained? I think entirely by the varying sizes of the lenses. In the two former the lens was of normal or nearly normal size, its “golden” margin lay close to and nearly concentric with the margin of the chamber, and the area of the pupil was well within the area of its posterior surface; in the latter two the lens was shrunken, it lay quite excentrically in the lower part of the chamber, and a free passage existed between its upper margin and the margin of

¹ Cases 24, 25, 26, 27, Section VII.

the pupil. In one of the non-glaucomatous cases the lens was cretaceous and rough, an additional proof that the glaucomatous complication is not the result of irritative hypersecretion.

Tumours of the retina and choroid almost invariably induce a glaucomatous tension of the eyeball. Let me here refer again to the general conclusion to which my experiments led—namely, that the causes of glaucoma are *those conditions which exalt the vitreous pressure above the aqueous pressure*. The conclusion will, I think, find confirmation in the class of cases now in question. The vitreous substance is not a mere secretion of gelatinous consistency: it is an organised structure containing living elements. The intrusion of a foreign growth upon the space which it occupies is not at once compensated by the escape of an equal bulk of its substance, as might occur were it an unorganised secretion. So long as its nutrient supply flows into it in undiminished quantity we may, I think, assume that its tissue will not yield to the intruding mass without offering some resistance—that is to say, the advancing tumour has not merely to displace a certain quantity of fluid: it has to reduce the bulk of a healthy living tissue. Here, surely, are conditions competent to effect the advance of the lens and suspensory ligament which, as we have seen, establishes the glaucomatous condition. Specimen 6 is an eye effected with glioma of the retina. ⁽¹⁾ The amount of the new growth within the eye is small, but had sufficed to produce a considerable excess of tension, pain, and enlargement of the episcleral vessels. Fig. 13 exhibits the changes in the ciliary region. The iris is peripherally adherent to the cornea, the ciliary process is greatly atrophied, and evidently had little share in effecting the

¹ Case 6, specimen 6, fig. 13, slides. The apparent detachment of the retina and the absence of the lens are accidents of mounting merely—the specimen was dissected before I had learned how to preserve all the parts in their normal situations.

adhesion. The lens itself was probably pressed against the iris quite up to its periphery. The stretched condition of the iris, immediately in front of the ciliary processes (easily seen in the specimen when a candle is placed a little *below* the level of the glass surface) indicates, I think, its former contact with the margin of the lens. I would particularly point out that glaucomatous complications in such cases do not depend upon a mechanical pressure exercised directly upon the ciliary region by the morbid growth, as has been suggested. This specimen (6) and a drawing of another case made several years since, before this question had been raised, convince me that invasion of the ciliary region and compression of the angle of the chamber by the tumour itself are not the causes.

(The following case came under notice most opportunely as evidence of another kind for this statement.)

Curiously enough, the only other specimen of intraocular tumour which I have been able to examine is one of the rare cases in which no glaucomatous tension has been produced. The exception is most fortunate, as it gives striking confirmation to more than one point involved in the theory which I have advanced. ⁽¹⁾ A large spherical melanotic sarcoma nearly fills the space normally occupied by the vitreous (Fig. 55). I am told that there was no excess of tension previous to excision; the optic disc, microscopically examined, exhibits no sign of pressure; the angle of the anterior chamber is entirely free from narrowing or compression. ⁽²⁾ I was at a loss to explain this absence of glaucomatous changes until I found that the pedicle of the tumour is seated in the ciliary processes, and is almost confined to that region. Evidently the tumour sprang from the ciliary processes, and in its very origin diminished the nutrient supply to the vitreous humour, thereby obviating the tendency to glaucoma which its bulky presence must otherwise have created.

¹ Case 15. ² Slides 15*b*, 15*c*, 15*a*.

Another point of interest is this—the anterior limit of the tumour is in contact with the posterior surface of the lens, and has manifestly pushed the latter somewhat forwards towards the cornea (this forward displacement of the lens was very evident when the frozen eye was divided) ; but, notwithstanding this displacement, the angle of the anterior chamber has suffered no closure or compression. This confirms the fact that an advance of the lens produced merely by pressure at one point does not affect the angle of the chamber. In the human eye, soon after death, I have pushed the lens forward by means of a needle introduced near the equator, and have seen very clearly that as the pupillary margin of the iris advances the periphery retires, precisely as it does, in fact, during accommodation for a near point. Closure of the angle of the anterior chamber cannot be produced by such means : it requires a *general rise* of pressure affecting the lens and suspensory ligament alike. ⁽¹⁾

Hæmorrhagic glaucoma, a condition hitherto especially difficult to explain, is readily accounted for by the theory which has been advanced, and affords, I think, a strong point of evidence in its favour. An outbreak of glaucoma, usually of an acute kind, has in a considerable number of cases been preceded by hæmorrhage into the retina or vitreous substance. ⁽²⁾ It has been an open question whether the extravasation of blood actually originates the glaucoma, or whether it is to be regarded as an early symptom of an increasing intraocular pressure. I think we may safely conclude that where an extravasation, in a hitherto presumably healthy eye, is followed within a few days by an attack of acute glaucoma, the former is

¹ The specimen in which these points were observed was placed in my hands for mounting by Mr. Solomon, in whose practice the case occurred. I am indebted to his kindness for permission to publish this description of it.

² For List of Cases, &c., see Handbuch d. g. A., v. I., p. 43.

the cause of the latter, and we may explain the process thus:—The arterial system is atheromatous, the smaller vessels are prone to rupture; (these patients not seldom die a little later by cerebral apoplexy). By reason of the senile alteration in the lens already described, the eye is prone to become glaucomatous on the occurrence of even a small rise of vitreous pressure, such as, earlier in life, would be rapidly compensated by increased escape of fluid from the vitreous chamber; a retinal or choroidal artery ruptures and pours forth its blood until the flow is arrested by the reaction of the increased intraocular pressure upon the vessel. This addition to the contents of the vitreous chamber, which, in healthy conditions, would occasion no permanent excess of pressure, supplies the little that is wanting to establish the glaucomatous condition; the lens and suspensory ligament advance, ever so little it may be (it will be remembered how slight an excess of vitreous pressure closes the angle of the anterior chamber), ⁽¹⁾ the angle of the anterior chamber is compressed, the general pressure rises, the ciliary processes swell and intensify the obstructive changes, and glaucoma is established.

I have no specimen which illustrates exactly this condition, but I have two which bear upon the point. Specimen 2 shows the consequences of hæmorrhage immediately following iridectomy. The lens was firmly pressed against the cornea, and actually driven forward with such force that its margin separated the edges of the wound and protruded between them. Specimen 7 exhibits a hæmorrhage from the site of an old injury of the iris in an eye with detached retina. This eye had been blind twenty years, and had been entirely free from pain. There can be little doubt that the detachment of the retina had existed a long time, perhaps throughout the whole period, and that the tension of

¹ Section IV., experiments.

the eye would be below the normal. Suddenly, during inflammation of the other eye, the blind eye becomes intensely painful and is found to have $T + 3$. A few days later the eye was excised, the tension being a little lower than on admission. I expected to find the ordinary conditions of secondary glaucoma with complete closure of the pupil, but found, to my surprise, complete detachment of the retina, a condition very rarely associated with excess of tension. The latter symptom, however, was explained by the recent extravasation of blood. The amount of blood poured out, be it observed, was by no means large. Its ability to induce a marked excess of tension lasting certainly a week, perhaps longer, I attribute to the fact that the condition of the iris, suspensory ligament, and adherent retina entirely precluded the escape of fluid through the natural passage from the vitreous to the aqueous chamber. The angle of the anterior chamber, moreover, was completely closed (see Case 7, fig. 14).

This latter case especially illustrates the fact that hæmorrhage of no large amount may raise the pressure in the vitreous chamber considerably. Under the predisposing conditions which exist in the senile eye—viz., the increased diameter of the lens, &c.—I think it not unreasonable to suppose that retinal hæmorrhage of moderate amount may induce even an intense glaucoma.

OBJECTIONS TO THE OBSTRUCTION-THEORY OF GLAUCOMA.

At the Ophthalmic Congress in Heidelberg in 1877 the recent theories concerning glaucoma were discussed. ⁽¹⁾

Hermann Pagenstecher—any criticism from whom demands consideration—propounded two statements which appear to be irreconcilable with the theory of glaucoma

¹ See Bericht über, &c., in *Klinisch Monatsbl für Augenheilkunde*.

which I have advanced, and supported them with specimens and drawings. They were as follows :—

1. "There are forms of glaucoma in which no closure of Fontana's space is present." ⁽¹⁾
2. "Closure or obliteration of Fontana's space does not of itself (an und für sich) lead to glaucoma. ⁽²⁾

With regard to the first statement I would point out, as Ad. Weber has done, that the essential change is not closure of the angle of the chamber, but *an obstruction*, be it what or where it may, *to the escape of the intraocular fluid*. The crystalline lens in the anterior chamber effects such an obstruction most completely, and induces glaucoma, while the angle of the chamber is widely open. In serous iritis, again, the angle of the chamber is doubtless widely open, though it is probable that inflammatory changes or deposits in the tissues retard the escape of fluid. In one of the cases (Pagenstecher's) adduced in evidence, serous iritis existed together with indications of previous glaucoma simplex, and "*across the open angle of the anterior chamber was found a new formation of connective tissue.*"

Was not this new formation, I would venture to suggest, the remnant of a former peripheral adhesion of the iris and cornea, stretched by a deepening of the anterior chamber during the attack of serous iritis? Such a condition, or something like it, is seen in figs. 21 and 22, where a deepening of the anterior chamber and a widening of its previously compressed angle undoubtedly occurred.

The other case cited is also entirely peculiar as to the mode of its occurrence and its symptoms—*e.g.*, "exophthal-

¹ See Bericht über, &c., in *Klinisch Monatsbl für Augenheilkunde*, p. 7.

² *Ibid*, p. 9.

mia," "*very deep anterior chamber*," "*uncontrollable bleeding after the excision*," &c.

I will not attempt to explain the exceptional conditions here met with; moreover, I will not deny that increased tension of the eye can and does occur temporarily as the result of other than obstructive changes—we know that increased afflux of blood produces it—but I will assert that such cases of glaucoma, if we must so name them, represent a pathological condition extremely rare, and entirely different in its essential nature from that which is ordinarily known as glaucoma.

With regard to Pagenstecher's second proposition, it is, I think, true or untrue according to the sense in which it is understood. Closure of the angle of the anterior chamber (Fontana's space) is, I maintain, not only a cause, but *the* cause of glaucoma; but it may and does exist in eyes which are not glaucomatous, by reason of the co-existence of other morbid changes which render the occurrence of glaucoma an impossibility. An incision of the skin in a person narcotised by chloroform produces no pain; but this fact would not justify the statement that incisions of the skin are not of themselves the cause of the pain with which they are commonly associated. The truth of the matter may, I think, be stated as follows:—*Closure of the angle of the anterior chamber causes glaucoma, provided—*

1. *That the secretion of the intraocular fluid be not so far suppressed as to render the distension of the eye an impossibility.*
2. *That the intraocular fluid do not escape through some abnormal channel in sufficient quantity to neutralise the effect of the obstruction of the normal channel.*

3. *That the closure affect a portion of the periphery of the chamber sufficiently extensive to materially retard the escape of the intraocular fluid.*

Closure of the angle of the chamber may frequently be discovered in eyes which are not glaucomatous; nay, more, in eyes the tension of which is decidedly below the normal. I believe that every such case is to be explained by the counter-acting influence of one or other of the changes just referred to. If this be not so, the obstruction-theory of glaucoma falls to the ground. I cannot, therefore, pass the matter by without bringing evidence to support the view I have advanced.

Specimens 10 and 11, are eyes destroyed by perforating wounds. ⁽¹⁾ In both the angle of the chamber is totally obliterated, in both the tension was *subnormal* at the time of excision, in both the retina is detached. There is, however, a striking difference between the two as regards the angle of the chamber, which shows, I believe, that in one the obstructive change preceded the shrinking of the vitreous, in the other the shrinking of the vitreous preceded the obstructive change; in the former, therefore, a temporary excess of tension must have occurred, in the other none. Let me briefly sketch the course of events in each case.

Specimen 10, fig. 26. A fragment of metal perforates cornea, iris, and lens; iritis, with adhesion of the pupil to the swollen lens, follows; the passage of the intraocular fluid into the anterior chamber is arrested; the pressure in the vitreous chamber rises, the lens and suspensory ligament advance, the ciliary processes swell, a firm compression of the periphery of the iris against the cornea, resulting in adhesion, is the consequence (fig. 26). It will be seen from the figure that the adhesion involves exactly as much of the iris as

¹ Cases 10, 11, Section VII.

would be pressed upon by the ciliary process when the latter is supported behind by the suspensory ligament; a glaucomatous state of the eye is thus induced; by reason of extension of inflammation from the iris, or through the original injury, cyclitis—inflammation of the ciliary processes—follows; the nutrition of the vitreous substance is impaired; the vitreous substance shrinks; effusion from the choroidal vessels follows the diminution of pressure; the retina is detached, and ultimately shrinks to a thin, central, fibrous chord; the shrunken disorganised lens, the adherent iris, and the displaced retina are more or less united, and form a thick partition between the aqueous and vitreous chambers; the normal secretion of the intraocular fluid is entirely arrested; ossification commences in the posterior portion of the choroid. (A portion of the ossific lamina was exhibited at a medical society some time ago; it showed highly characteristic bone structure). From the moment when the cyclitis sets in, the tension, previously in excess, becomes subnormal, and remains so.

Specimen 11, fig. 27. The eye is punctured by a piece of wire which perforates cornea, lens, and vitreous, and reaches the choroid near the posterior pole of the eye. Whether the iris is wounded is uncertain, and does not affect the result. The lens swells; the pupillary margin of the iris adheres throughout its whole circle to the capsule; the secretion proceeding from the ciliary processes is unable to enter the anterior chamber, it collects in the posterior chamber and bulges the iris forwards till it absolutely applies itself to the cornea *throughout*, [in this case there is a *total* annihilation of the anterior chamber; in most cases of circular synechia posterior a certain quantity of fluid remains *imprisoned* in the anterior chamber by reason of the occlusion of its angle; it is extremely probable that in this case an escape occurred

through the wound or the cicatrix ; the latter was reopened some time after the injury in an unsuccessful attempt to remove the lens matter,] and bridges over and obstructs the ligamentum pectinatum ; simultaneously with these obstructive changes in the aqueous chamber, inflammation and subsequent shrinking of the vitreous substance set in ; the vitreous pressure, which would otherwise have been in excess, is reduced to a subnormal point ; the lens and suspensory ligament are not pressed forwards, and consequently the ligamentum pectinatum, although completely obstructed, is *not compressed* (an unusual condition, see figure) ; the lens shrinks, its capsule is thrown into numerous folds ; the posterior aqueous chamber is thereby still further increased in size, and remains filled by the imprisoned and albuminous secretion poured into it by the ciliary processes ; the retina is converted into a central fibrous cord, connected with which is a cicatricial band indicating the course of the original injury.

As in the previous case, the obstruction to the escape of the intraocular fluid from the anterior chamber fails to produce glaucoma, because the secretion of this fluid into the vitreous chamber is arrested.

Specimen 9, fig. 25, is a case of total staphyloma. I have no note of the nature of the primary malady. The disc presents no trace of excavation. The staphyloma is a result of diminished resistance in the cornea, or in the new tissue which represents it—not of increased intraocular pressure ; the ciliary processes are not atrophied, the vitreous appears healthy, the retina is not detached. We cannot, therefore, attribute the absence of glaucomatous tension to the suppression of the intraocular secretion. The anterior chamber is entirely abolished ; the iris, reduced to a mere web of pigmented tissue, adheres throughout to the pseudo-cornea.

How, then, is the absence of glaucoma to be explained, seeing that secretion continues, and the angle of the anterior chamber is annihilated ?

I suspect that the intraocular fluid here finds an abnormal exit. The staphylomatous tissue, especially at the anterior part, is extremely thin. It consists, in part at least, not of true corneal tissue, but of new material, possibly analogous to the permeable cicatricial tissue which closes iridectomy wounds in glaucomatous eyes ; its inner surface is devoid of the epithelium, which, as Leber has proved, is the agent which prevents percolation through the cornea ; its substance in several places is permeated by blood-vessels. Moreover, it appears likely that, in spite of the superimposed web of iris, an escape occurred through the veins in the neighbourhood of Schlemm's canal. Open channels perforating the sclera close to the anterior limit of the ciliary muscle, and containing pigment which has passed along them from the iris, can, I think, be discovered. (The section which contains these channels is unfortunately much too thick for good examination, but under a low power and good daylight illumination they are tolerably evident.)

Specimen 8, fig. 24, presents closure of the angle of the anterior chamber limited to a portion of the circle. The disc exhibits a doubtful pushing back of the lamina cribrosa, but certainly no excavation. ⁽¹⁾ In other cases we have seen that a partial affection of the angle of the chamber may suffice to raise the vitreous pressure, and thereby to induce closure *throughout the whole circle*, by means of the advance of the lens and suspensory ligament. ⁽²⁾ Why then did the remainder of the circle remain patent in this case ? Because

¹ June, 1879. Certain notes, given in my manuscript essay, concerning the previous history of this eye, are omitted here, because some uncertainty has arisen as to the identity of the case.

² This Section, p. 178.

there existed an open communication between vitreous and aqueous chambers. The lens has undergone a remarkable disarrangement by which its central portion has disappeared, or curled round towards the periphery, together with the capsule, so as to form a ring in contact with the ciliary processes. This open communication appears to have prevented the self-intensifying process by which a slight excess of tension at once causes further compression of the angle of the chamber and increased tension.

In this connection I would recall the case of glaucoma after cataract extraction, in which rupturing the posterior capsule rapidly banished the glaucoma. In that case the eye regained, and now maintains, a normal tension, with good vision, although at least one fourth of the angle of the anterior chamber is closed by adhesion of the iris to the cornea. ⁽¹⁾

¹ Case 36, fig. 43.

SECTION VI.

TREATMENT OF GLAUCOMA.

If it be true that glaucoma is in reality a complex group of symptoms dependent upon one single and essential change—an excess of intraocular pressure—its treatment must manifestly be directed, whatever be the variety or type of the disease, primarily against this physical condition.

Before this fundamental truth was established, and its indications realised by von Graefe's brilliant and beneficent discovery of the remedial power of iridectomy, glaucoma was an incurable disease. It is hardly too much to say that before von Graefe's time every eye attacked by true glaucoma became blind, unless death overtook its possessor before the disease had had time to run its course. Restoration of such eyes to a permanently healthy state, either spontaneously or by any method of treatment then practised, was unknown, and, by the light of our present knowledge, impossible. No sooner was the value of the new surgical remedy made known by its author than it was forthwith put on its trial in this country and elsewhere. With those who tested it fairly, and in accordance with the prescribed rules, its efficacy at once became an established fact, while the opposition with which it was received in certain quarters did not long survive.⁽¹⁾ Since that time hundreds of persons annually have been, and now are, rescued from blindness by its means.

¹ See Correspondence on Glaucoma, *Brit. Med. Journal*, 1862, II.

During the last few years an improved knowledge of the conditions upon which excess of tension depends have led to fresh attempts to relieve glaucoma by therapeutic agents. Myotics, such as the extract of calabar bean, eserine and pilocarpine, applied in solution to the eye, have beyond all doubt the power, in certain cases, of lowering and, exceptionally, even of removing the excess of tension, and, in conjunction with surgical means, are useful in many more. Attempts to substitute other operative procedures for iridectomy have also, within certain very narrow limits, proved successful. But, these facts notwithstanding, the truth remains to-day almost as absolute as when enunciated by von Graefe twenty-one years ago, that *the* remedy for glaucoma is iridectomy.

Although it is sometimes desirable to precede the operation by certain other special measures, or to attempt relief by other means before advising it, or even to abstain from this operation altogether, still its value so far surpasses that of all other measures taken together, and the evil consequences of non-interference are even now so much more frequently witnessed than any which arise from an occasional ill-advised operation, that the discussion of this remedy must take precedence of all else which may have to be said concerning the treatment of glaucoma.

IRIDECTOMY.

The improvement of vision obtainable by iridectomy varies with the type of the disease. It is greatest in the acute form, smallest in the chronic.

Whatever be the defect of vision before the operation, the prospect of improvement depends almost entirely upon the length of time which it has existed. An acute attack of great intensity may, in the course of not many hours, reduce vision from its normal acuity to the barest perception of light.

Iridectomy, promptly performed, will in all probability restore it very nearly, if not quite, to its former condition. Chronic glaucoma, on the other hand, which has been insidiously advancing for several years, may, at the end of that time, have still spared a large portion of the visual field, and have reduced the acuity of central vision by not more than $\frac{1}{2}$ or $\frac{2}{3}$. Iridectomy in such a case, if properly performed, may enlarge the visual field a very little; it will probably preserve the vision which remains, or retard its further deterioration; it may influence the progress of the malady hardly at all; it may even greatly accelerate its progress. Between the brilliant result obtained in the former case and the disastrous consequence occasionally witnessed in the latter, infinite diversities of progress and result are to be observed. Acute glaucoma blinds by the sudden onset of a pressure under which the function of the retina is at once suspended: the prompt removal of the pressure restores the normal conditions before atrophic processes have had time to confirm the loss of function. In chronic glaucoma, on the other hand, the gradual and insidious advance gives time for atrophy to do its work in the peripheral portions of the retina, or the fibres of the disc, before central vision is so far impaired as to lead the patient to seek relief. In acute glaucoma, moreover, the structural changes which lie at the root of the malady are of recent occurrence, and are, in part at least, caused to subside by the prompt provision of an escape for the imprisoned intraocular fluid, and the cure is therefore likely to be complete and permanent. But in chronic glaucoma, as has been shown, the long continuance of the altered relations of parts is associated with adhesions which cannot be abolished, and the effect of which may not be wholly compensated by the operation; it happens, therefore, that the cures in the latter case are both less complete and less certainly permanent than in the former. Hence comes the rule which, with certain reservations, is very generally

applicable: *when the diagnosis of glaucoma is established, operate.*

The urgency for immediate operation naturally depends greatly upon the acuteness of the malady. In acute glaucoma iridectomy should be performed "without the slightest hesitation or the smallest delay." In the interests of the public, and especially of that class to whom "a second opinion" is a luxury not lightly indulged in, this rule needs to be repeated again and again. *An eye which is painful, red, and hard, should be seen within the smallest possible number of hours by a surgeon competent to operate upon it.* This is a simple maxim. If it were known to every student sent forth from our medical schools, "absolute glaucoma" would, I think, appear with less frequency at our ophthalmic hospitals. Cases of acute glaucoma, in which the whole period during which the sight might have been rescued by operation has been spent in the employment of lotions, blisters, fomentations, &c., and in which the blindness, though perhaps only of a few weeks' duration, is already absolute and incurable, are still not unknown. When the symptoms become intense within a few hours of their onset, every hour which the operation is delayed may induce irretrievable mischief. So long, however, as any perception of light remains, and even although it has already been extinguished for some days, iridectomy should on no account be withheld. The prostrated condition of the patient must not be admitted as a reason for delay. Iridectomy is the surest means of procuring ease and sleep. A delay of some hours is generally inevitable from one cause or another: assistance must be obtained, instruments sent for, consent obtained, daylight waited for, though this latter is by no means essential; during the interval a subcutaneous injection of morphia may generally be given with advantage, unless contraindicated on general grounds, and eserine (or calabar) should, I think, in every case, be applied locally to the eye.

This last advice I give with some hesitation; but strong evidence, both practical and theoretical, can already be advanced to support it. ⁽¹⁾

In subacute glaucoma, also, the importance of early operation is great. Each recurrence of the acute symptoms diminishes the degree to which the sight is capable of restoration. When many such recurrences have already happened, and the visual field, *examined during a quiescent interval*, is peripherally contracted, and excavation of the disc is marked, complete restoration will rarely be obtained. But very considerable deterioration of sight *during an exacerbation* does not preclude the recovery of excellent vision after iridectomy. The prognosis must be founded chiefly upon the state of vision immediately before the exacerbation, if this can be ascertained, and upon the duration of the acute attack, precisely as in the case of acute glaucoma.

In chronic glaucoma—the simple, non-irritative glaucoma of Continental writers—a positive improvement of vision is seldom to be hoped for. Atrophic structural changes have here advanced *pasi passu* with the loss of vision, and the most that iridectomy can effect is to remove the cause of the disease and thereby to arrest its further progress. Even this result, however, cannot be depended upon in every case. The reasons of the less certain action of iridectomy in chronic glaucoma have already been discussed. ⁽²⁾ It remains only to consider the expediency of the operation in this condition, and to indicate the cases, if any, in which it should be withheld. Some surgeons go so far as to abandon operation altogether in the purely chronic form of the disease. I think that to do this is to exaggerate the danger which iridectomy involves, and to condemn to blindness a certain number of eyes which might be saved. One cause of

¹ See page 222. ² Section V., page 168.

the bad results which are obtained is, I believe, a preventible one—and I will not make this charge without confessing to personal experience of its truth—namely, an incorrect method of operation. The removal of the portion of iris withdrawn through the incision by one snip of the scissors, more especially if the scissors are entrusted to an assistant, as is done by some surgeons, is not in accordance with the rules, established by careful observation and experience, for the performance of the operation. Tearing the iris from its insertion, or, at any rate, its complete excision from one end to the other of the incision, in the way to be described later, is especially to be insisted on in operating for chronic glaucoma. The danger of inducing serious hæmorrhage, is, I think, of far less moment than the danger of an incomplete removal of the iris, with subsequent adhesion of the remaining portion to the wound. It cannot be denied, however, that the most skilfully performed iridectomy is occasionally followed by the speedy loss of what sight still remains, and that in many other cases the cure is imperfect and temporary only. In every case of chronic glaucoma, therefore, the responsibility of advising operation is a heavy one, and should on no account be undertaken without a full explanation to the patient or his friends of the certainty of blindness on the one hand, and of the uncertainties which beset the operation on the other. If both eyes are decidedly glaucomatous, but both have still a useful amount of sight, iridectomy on one, probably the more advanced, should be performed without delay ; the result may decide the treatment of the other. When one eye is already blind, and the vision of the other is restricted to a small central portion of the retina, the chances of retaining useful vision are very small. Having regard to the age of the patient, the impossibility of great benefit, and the possibility of a painful and accelerated progress, I think a prudent surgeon will only operate on the

express desire of the patient to receive the only possible chance of benefit, however small it may be.

MODE OF PERFORMING THE IRIDECTOMY.

It is desirable, in most cases, to employ an anæsthetic. I have a strong preference for ether, and never use chloroform except in young children. The disadvantages of an anæsthetic—namely, the preliminary resistance, the danger during the period of complete narcotism, and the subsequent sickness and distress—incline many surgeons to withhold it in this and other ophthalmic operations. Until quite recently, I have almost always performed extraction of cataract without ether, but within the last few months I have adopted a measure—viz., *the previous administration of a dose of chloral*—which so very much diminishes these disadvantages, that already I have resumed the employment of ether in nearly every case. This method, upon which I append a fuller note (¹), has especial advantages in the case of iridectomy for glaucoma, in which the avoidance of previous struggling, complete passiveness during the operation, and freedom from sickness afterwards, are points of much importance.

The patient must lie upon a suitable table or couch, the surgeon standing or sitting behind the head. In private houses the most convenient way is to place the patient upon an ordinary one-ended couch, the feet being towards the raised end, the head resting on two pillows at the flat end; the surgeon seated on a chair, and with the end of the couch between his knees, has the patient's head in admirable position for most ophthalmic operations.

The position of the incision, as regards the portion of the circle selected, is unimportant so far as the curative effect is concerned. The upper incision has advantages both cosmetic and optical, inasmuch as the coloboma is hidden by the upper

¹ Page 231.

lid ; but it is perhaps somewhat more difficult to make with perfect accuracy, especially if the lance-shaped knife be used, and the patient be not narcotised, than an incision in parts of the circle more easily exposed. Lateral and lower incisions have been recommended in preference by high authorities, but, in this country at least, it is usual to adopt the upper incision, in accordance with the example set long since by Bowman. The incision is made in such a manner as to open the anterior chamber as near to its periphery as possible, in order to facilitate the removal of the corresponding portion of the iris as near as may be to its insertion. This rule, originally laid down purely as a result of empirical experience, is now absolutely confirmed by the teachings of pathology. The length of the incision, measured on the external surface, should be from 6 to 8 mm. Within the larger limit, the longer the incision is the better ; but its length is of less absolute importance than its peripheral situation. The average diameter of the cornea is 12 mm., its circumference is, therefore, $12 \times 3.1416 = 38$ mm. nearly. Supposing the length of the incision, measured on the sclera, to be 8 mm., then its length on the inner surface of the cornea must be less than this, and it is obvious that not more than one fifth of the circle of the iris, measured at its periphery, can be excised through this incision.

The incision is made with a broad lance-shaped knife, bent at an angle, or with a narrow cataract knife, such as is used in v. Graefe's operation for extraction. Many operators give preference in every case to the lance-shaped knife, others to the cataract knife, and, as the most eminent authorities differ on this point, it must remain a matter for individual taste and experience. I prefer to use the narrow cataract knife in almost every case. Its advantage, in my opinion, is that it enables one, without danger to the lens, to effect a longer and more peripheral incision than can be made

with the lance-shaped knife. The latter, if introduced in a plane parallel with the iris, is liable to enter the chamber at some distance from its periphery, even though the external puncture be made well in the sclera; and if a sufficient length of incision is not obtained by the forward movement of the knife, it is extremely difficult to enlarge it satisfactorily during the withdrawal, for the chamber, already abnormally shallow, is instantly diminished almost to abolition, by the forcible escape of the aqueous humour. With the cataract knife it is possible, even after the appearance of the point in the anterior chamber, to modify the length and position of the incision according to the space available, by making the counter-puncture a little further forward or backward, as may be necessary. It is especially in cases of advanced chronic glaucoma, with very shallow anterior chamber, that these advantages are conspicuous, for it is in this form that a precisely accurate operation is at the same time essential and difficult.

The patient being narcotised to complete muscular relaxation, the eyelids are separated with a stop-speculum, the conjunctiva is seized with toothed forceps near to the corneal margin and opposite to the site of the incision, and the eye rotated, if necessary, so that this portion of the ciliary region is easily accessible. Supposing the broad lance-shaped knife to be used, its point is entered in the sclera about 1 mm. external to the corneal margin, and pushed slowly and steadily forwards between iris and cornea, without touching either, until an incision of sufficient length is effected. It is then steadily but not too slowly withdrawn, taking care that the point is not directed towards the lens, which suddenly advances as the aqueous humour escapes. If the narrow cataract knife be employed, its point is entered at about the same distance from the corneal margin as in the other case, carried slowly and steadily across the upper part of the

chamber, keeping as near to the periphery as may be, and passing very close to the anterior surface of the iris, but without wounding it, until it effects the counter-puncture in a corresponding spot. So far the blade of the knife should be kept parallel with the plane of the iris, but as it cuts its way out, and as soon as the angle of the chamber is incised between puncture and counter-puncture, the edge should be turned somewhat forwards, so as to emerge nearer to the corneal margin than it otherwise would. By this means the object desired—namely, an internal incision situated as peripherally as possible is attained, while a large conjunctival flap, which is undesirable, is avoided. The fixation forceps being then entrusted to an assistant, the iris is seized with the small toothed iris forceps, and a portion, corresponding to the length of the incision, is removed, partly by incising it with scissors, partly by tearing it from its attachment by traction. This portion of the operation, upon the completeness of which its efficacy greatly depends, is performed differently by different operators. “The iris may be found either to remain in the chamber or to prolapse. If the former, the small slightly-curved iris forceps are to be introduced, closed, into the chamber, and made to seize the iris opposite to the middle of the incision, and about midway between its pupillary and its outer border. The iris is then brought outside the chamber, and divided with small scissors, on one side of the forceps, from the pupillary to the ciliary border, the forceps pulling it gently at the same time, so as to ensure this complete division of it. The end held by the forceps is then torn from the ciliary attachment as far as the angle of the incision, and even dragged upon a little, so as to detach it beyond the angle, and then divided with scissors quite close to the angle. The cut end then retreats within the chamber. The opposite side of the prolapsed part is then seized, and dealt with precisely in the same

manner. No iris should be left in the angle of the incision, lest the healing process be imperfect, and subsequent irritation occur. If the iris at once prolapse on the completion of the incision, the forceps need not be introduced within the incision, but may seize it outside." ⁽¹⁾ A result almost as satisfactory as that obtained by Bowman's method is obtained by seizing the iris in the manner above described, dividing it completely to its periphery close to one angle of the incision, then drawing it towards the other angle of the incision so as to tear it from its attachment between these two points, and, finally, again dividing it as close to the periphery as possible at the latter point. Instead of tearing it away, some operators prefer to remove it by a succession of small snips with the scissors as close as possible to the lips of the wound, as by this means the danger of troublesome bleeding is diminished. Others, again, content themselves with removing with one cut of the scissors the portion of iris withdrawn with the forceps. The last-named method is by far the easiest of performance, and in most cases of acute glaucoma will probably give the desired result; but in cases of chronic glaucoma it is liable to most unsatisfactory consequences, and should never be practised. Knowing, as we now do, the essential importance of reopening the angle of the anterior chamber completely up to the ligamentum pectinatum, we can appreciate at its full value the tearing away of the iris from its peripheral insertion, long since advocated on empirical grounds. Bleeding into the anterior chamber is seldom prejudicial to the healing process, inasmuch as the blood commonly escapes within a few hours.

The after-treatment is simple. So long as any danger of sickness exists, it is well to apply a little pressure over the eyelids. If necessary, a light pressure bandage may be applied for some hours; as a general rule, however, it is desirable to subject the eye to no additional pressure beyond

¹ Bowman *Brit. Med. Journal*, 1862, II., p. 382.

that of the closed lids. I am so convinced of the extreme danger to a glaucomatous eye, the tension of which is not immediately reduced quite to the normal point by the operation, of any external pressure, even such as may arise from a well-adjusted bandage during movements of the head upon the pillow, that I prefer in almost every case to simply close the eyelids with two strips of black court plaster. The pain which immediately follows the operation, or occurs within the next twelve hours, when it depends merely upon the wound, and not upon any serious complication, such as hæmorrhage into the vitreous, &c., may be greatly alleviated by the occasional application, over the closed eyelids and forehead, of a few thicknesses of soft flannel dipped into very hot water and squeezed dry in a cloth. It is sometimes desirable to administer a subcutaneous injection of morphia soon after the operation. The plan, already mentioned, of preceding the administration of ether by a dose of chloral will, however, be found, in the absence of any special source of pain, to render this unnecessary.

The use of a myotic—*e.g.*, eserine—before and after operation, is a new and, in most cases, a valuable addition to the treatment. It will be discussed elsewhere.

The immediate effect of the operation is a reduction of the tension of the eye, due to the escape of the aqueous fluid. The greater this reduction—*i.e.*, the more nearly the tension approaches that of a non-glaucomatous eye after iridectomy—the more favourable, as a rule, the prognosis as regards the permanent abolition of glaucomatous tension. Since the amount of the reduction varies exactly with the amount of aqueous fluid extruded, we may express this fact as follows:—The less fluid contained in the anterior chamber before operation, the less favourable, as a rule, is the prognosis. This applies chiefly to chronic glaucoma; it finds a very satisfactory explanation in the condition of the angle of the

chamber, which undergoes obliteration, *ceteris paribus*, in proportion to the shallowness of the chamber.

The period which elapses before the wound closes sufficiently to re-establish the anterior chamber varies greatly. As a rule, the delay is longest in cases of chronic glaucoma, with very shallow chamber, in which an excess of tension is still discoverable immediately after the operation. In such cases three or four days, a week, or even longer, may elapse before the iris is permanently removed from contact with the cornea. The existence of such contact is sometimes made manifest by the way in which small quantities of blood remaining in the chamber follow the furrows in the surface of the iris. A reopening of the wound, accompanied by a sudden sharp pain in the eye, and a gush of hot water from between the lids and down the nostril, may occur once or more during the first ten days.

During the persistent abolition of the chamber the tension of the eye tends to remain in excess, and the unfavourable course known as "malignant" is to be dreaded: great injection of the scleral vessels and chemosis surrounding the cornea (except opposite the incision) are signs of ineffectual relief of tension, and compression of the iris between the lens and cornea. The pathology of this condition is described elsewhere. ⁽¹⁾ A tardy restoration of the anterior chamber, if not accompanied by excess of tension in the vitreous chamber, has no evil significance. In many cases, especially in such as present an immediate and complete reduction of glaucomatous tension, the wound heals almost as rapidly and as naturally as after iridectomy in a non-glaucomatous eye. If the wound be made in the cornea, it heals more evenly and with a narrower cicatrix than if it be in the sclera; but the latter position should always, except in occasional instances of acute glaucoma, be adopted.

¹ See Malignant Glaucoma, Section V., p. 169.

The lips of the incision appear never, or very rarely, to unite by immediate contact. A greyish, semi-opaque cicatricial substance may almost always be perceived, even in such as heal most readily and smoothly. It is characteristic of the glaucomatous eye, and is not found after iridectomy performed for other reasons; it is attributable to the effect of a continued excess of pressure from within, though frequently a very slight excess, upon the new elastic material which unites the cut surfaces of the sclera. In many cases the cicatrix is somewhat elevated above the general curve of the sclera. When this bulging becomes considerable it is a source of irritation, and even danger, to the eye; it is described as a *cystoid cicatrix*. It is precisely analogous to staphylomatous conditions in other parts of the eye, occurring as the result of an exalted pressure from within upon a yielding cicatricial tissue. So long as a cystoid condition of the cicatrix appears to produce no irritation or inconvenience it should not be counteracted in any way, as there is good reason to believe that it is a preservative lesion rather than a disaster.⁽¹⁾ If, however, it is a source of pain and a centre of hyperæmia, or even, as occasionally happens, of serious inflammation, it should be treated first by simple puncture with a cutting needle, repeated from time to time as may be necessary; by this means a lowering of the tension of the eye is obtained, which affords an opportunity for subsidence of the protrusion. Should it obstinately recur it may be removed, like corneal staphylomata, by abscision; but, under such circumstances, it must always be remembered that the cause is an excess of tension which the first iridectomy has failed to remove, and the propriety of a more radical treatment—namely, a second iridectomy—must be considered. In the exceptional instances of this condition in which suppuration occurs, and threatens to extend to the deeper structures, operation can be

¹ Bowman, R.L.O.H., Reports, IV. 58.

of no advantage. The usual treatment of corneal suppuration, by hot fomentations, atropine, &c., must be adopted.

Accidents of several kinds, preventible and otherwise, may occur during and immediately after the performance of iridectomy. Hæmorrhage is inevitable: its importance depends upon its situation and amount. Spontaneous bleeding from the iris, provided it does not interfere with the precision of the operation, is seldom of any consequence. It is undesirable to attempt to remove all blood from the anterior chamber before closing the eyelids; a little pressure on the outer lip of the incision does all that can be done with advantage or safety. If the ciliary processes be wounded by the knife, or if, as probably sometimes occurs, they are injured by the tearing of the iris, more serious bleeding will follow. The application of lint, dipped in ice-cold water, over the closed lids for some minutes, and the sparing use of the spatula, so as to separate the lips of the incision, is the best means of treatment. The withdrawal of clots of blood by instruments, or the application of any pressure to the eye, is liable to do more harm than it can ever do good. Small extravasations of blood from the retinal capillaries are of very frequent occurrence, as the immediate result of the lowering of the pressure on their external surface. In acute glaucoma they are probably never absent, but, as a rule, have no permanent bad effect; they are always to be found with the ophthalmoscope in such cases, as soon as an examination of the fundus is possible, and may be entirely reabsorbed during a few weeks. Occasionally an opportunity occurs of discovering them by dissection of the eye within a few weeks after the iridectomy. Bleeding from the larger vessels of the retina or choroid is a less frequent occurrence, but infinitely more serious. Occasionally during the operation, more frequently perhaps within the next twelve

hours, sudden violent pain is felt in the eye, the tension rapidly rises to more than its former excess, and any sight which existed is totally extinguished. Such symptoms indicate the pouring out of a considerable amount of blood into the posterior part of the eye—between choroid and sclera, between choroid and retina, into the vitreous chamber, or in several of these directions simultaneously. Excision of the eye should generally be undertaken without delay.

Wounding of the lens by the point of the knife is a serious accident, and one which does harm, not only by the creation of opacity in the previously transparent lens, but by its tendency to induce swelling of the lens, and the almost inevitable aggravation of the condition of the eye. Rupture of the capsule of the lens, especially in the equatorial region, may occur independently of any touch of the knife, by reason of the sudden forcible advance of the lens during the escape of the aqueous humour. With due care in the performance of the operation, neither mishap is at all likely to occur.

The incarceration of the cut edges of the iris and their permanent inclusion in the cicatrix is an accident to be carefully avoided. Besides being a source of irritation and persistent hyperæmia in their immediate neighbourhood, such adhesions tend, as has been shown, to perpetuate the very condition on which glaucoma depends—namely, closure of the angle of the anterior chamber.

Malignant glaucoma is a term applied to the exceptional cases in which the malady is intensified instead of being benefited by the performance of iridectomy. This disaster occurs, according to von Graefe, in not more than two per cent. of the cases of chronic glaucoma, and not at all in the other forms. Most of his followers would, I think, have to own to a larger percentage of non-success than this. The tension of the eye, but slightly reduced immediately after the operation, rapidly rises to its former height; the anterior

chamber is not restored for many days ; the iris and lens remain firmly pressed against the posterior surface of the cornea ; little or no fluid appears to escape from the wound ; great injection of the episcleral vessels, with œdema of the conjunctiva, soon set in ; severe pain and lacrymation, and even swelling of the lids, as in the worst forms of acute glaucoma, may occur. Under the increased tension, the remnant of vision is rapidly reduced to a bare perception of light, and ultimately extinguished altogether. Very slowly, during weeks or months, these irritative symptoms disappear ; the anterior chamber is re-established, but remains extremely shallow ; the eye remains permanently blind, probably with a tension somewhat higher than that which existed previous to the operation. The pathology of this so-called "malignant" course has been already discussed, and the methods by which relief may be attempted have, for greater convenience, been described in connection with its pathology. ⁽¹⁾

Iridectomy appears occasionally to act as the exciting cause of an outbreak of glaucoma in the fellow eye. This sequence, though decidedly exceptional, has been too often observed to be a merely accidental coincidence. Its occurrence is, I think, by no means paradoxical, when we consider the surrounding conditions. It may be very safely assumed, in such a case, that the second eye was, previous to the operation on the first, either already glaucomatous or in that condition of danger when any slight cause may light up the malady. Expectation of the operation, the operation itself, the effect of the anæsthetic in some cases, the subsequent anxiety concerning the result, perhaps with unrest and sleeplessness, are all calculated to induce a state of mental excitement, which, as we have seen, may and does induce the rapid development of glaucoma under certain predisposing conditions.

¹ Section V., page 169. (See also Note, page 230.)

Again—and to this cause I attach more importance than to the foregoing—we constantly see that irritation of one eye from almost any cause is accompanied with some slight temporary weakness of its fellow. Especially is this the case where there is considerable irritation of nerve fibres, as, for example, by superficial corneal abrasions. Now, seeing that the iridectomy incision lies in close proximity to parts wounds of which are especially liable to create sympathetic hyperæmia and inflammation, it appears not unnatural that it should occasionally exercise such an influence over the circulation or secretion in the fellow eye as, under strong predisposition, will cause a development of glaucoma. Ad. Weber attributes the occurrence to the diminished arterial pressure which results from confinement in the horizontal position, and points out the frequency with which acute glaucoma sets in during periods of rest and inactivity. On theoretical grounds, it is difficult, as before stated, to believe that a lowering of blood pressure can directly induce a rise of intraocular pressure, even in the glaucomatous eye. My own belief, founded to some extent upon clinical observation, is, that it does precisely the reverse. A rapid *deterioration of vision* does, I believe, occasionally attend a fall of the blood pressure, because the retinal circulation then more easily succumbs to the excess of pressure external to its vessels; and this, I would suggest, is the probable explanation of the striking case cited by Weber, in which depression of the heart's action by veratria caused a rapid loss of vision in a glaucomatous eye. ⁽¹⁾

In a case of chronic glaucoma I made several tonometrical measurements of the non-operated eye (absol. glauc.) during the few days following the iridectomy, and found a distinct increase in its tension. The patient was dressed and sitting up, and, with the exception of the traumatic irritation, was

¹ Arch. f. Ophth., XXIII., I., 55.

under no unusual conditions (see Case 21). A repetition of this test in a series of cases of glaucoma would, I think, throw much light upon the conditions which are chiefly responsible for the affection of the second eye after iridectomy.

Of late years a conviction has gained ground that the operation of iridectomy in glaucoma owes its efficacy to the incision of the sclera, and not to the excision of the iris. This conviction has led to many attempts to substitute for iridectomy an operation which should leave the iris intact. So long ago as 1867 v. Wecker affirmed that he should refrain entirely from the excision of the iris were it possible to effect a large scleral incision near the corneal margin without incurring an incarceration of the iris in the wound. Efforts to accomplish this desired end have since then been made in more ways than one, of which the one least open to objection is certainly that recently practised by v. Wecker himself, and generally known as sclerotomy.

SCLEROTOMY.

A knife of special construction — a “sclerotome” — resembling a Graefe’s cataract knife, but having a breadth of from 2 to 4 mm., and a double cutting edge at the pointed end, is employed in making the incision. This knife is made to incise the angle of the anterior chamber, either at the upper or the lower part of the circle, in the following manner:—Puncture and counter-puncture are made in the sclera at a distance of 1 mm. from the corneal margin, and in such a position that the edge of the knife exactly forms a tangent to the extreme margin of the cornea. [This is the incision prescribed by v. Wecker. In the only cases in which I have considered it desirable to perform sclerotomy I have caused the edge of the knife to incise the sclera more deeply. I have carried it across so as to be hidden in the angle of the

chamber, and to be, perhaps, about 1 mm. removed from the position of a tangent to the circle of the cornea.] The counter-puncture being made and the whole intervening portion of the wall of the chamber being incised simply by the thrust of the knife, without any sawing movement, the aqueous humour is allowed gradually to escape by a very slight rotation of the knife. The presence of the blade within the chamber prevents the prolapse of the iris into the incision during this escape. The knife is then very slowly and steadily withdrawn. Further to guard against prolapse, as well as for its specific anti-glaucomatous effect before adverted to, eserine is thoroughly applied before and after the operation.

Quaglino, at an earlier date, performed sclerotomy with a broad lance-shaped knife bent at an angle; his operation differed from an iridectomy merely in including no excision of the iris. Prolapse of the iris, either at once or during the healing process, is so liable to follow this proceeding that nothing can, I think, be said in its favour. The results of the twelve operations published by Quaglino contrast most unfavourably with the ordinary results obtained by iridectomy. (2) Other operators—in this country especially, Bader, Spencer Watson, and Nettleship—have performed sclerotomy in a manner more resembling that of Wecker already described, but using, instead of the broader sclerotome, an ordinary Graefe's cataract knife, and making, not merely puncture and counter-puncture, but completing the incision of the sclera as in cataract extraction, and leaving the conjunctiva only undivided. This proceeding involves sawing movements of the knife, and, though greatly preferable to the completed incision of Quaglino, is probably inferior to the incision effected entirely by direct thrust, as practised by v. Wecker.

² Schmidt. Handbuch, v. I., 123.

Before attempting to judge the merits of sclerotomy from my own very small experience of it, let me briefly note the conclusions arrived at by others who have practised it.

Von Stellwag says : " The excision of a section of the iris is of quite subordinate importance as regards the curative action, but it is yet to be urgently recommended in every case ; for the simple scleral paracentesis (incision) occasions, often at the time of the operation, and oftener still a little later, a prolapse of the iris, which is generally irreducible, and which leads to injurious irritation, and produces, moreover, a deformity equal to that which results from iridectomy."

H. Schmidt sums up a full review of the results obtained by sclerotomy previous to 1875 with this sentence:—"Sclerotomy offers advantages only in occasional cases of advanced glaucoma, when the atrophy of the iris renders its excision especially difficult. When excision of the eye is in view on account of severe pain, sclerotomy should first be tried, provided, of course, that the tension be still in excess."

Von Wecker, in whose hands the operation has attained its most satisfactory form, writing in the present year says:—"Although I shall, probably, during the whole of my medical career continue to give preference to excision of the iris as being the surest operation against glaucoma, I hold the conviction that our progressive science will substitute for it a simpler and more logical proceeding" ⁽¹⁾

"Under two circumstances only do I strongly advise you to renounce iridectomy and to resort to my operative procedure—first, when you recognise that you are dealing with hæmorrhagic glaucoma, for here the double section with the narrow sclerotome (2 mm.) enables you to avoid the danger of the section for iridectomy ; and, secondly, in cases of absolute glaucoma : in these, sclerotomy ought always to be preferred to iridectomy, the operation being undertaken only with the

¹ *Thérapeutique Oculaire*, Part I., p. 378 (1878).

object of freeing the patient from severe pain." ⁽¹⁾ Spencer Watson, in 1876, expressed a belief that sclerotomy would be the future remedy in the great majority of cases of glaucoma. ⁽²⁾ The conclusion appears to have been founded upon success in two cases. Higgens, speaking on the same occasion, stated that he had several times performed, but finally abandoned sclerotomy. In two cases it had caused sympathetic mischief, and had necessitated subsequent excision. Nettleship reports two cases performed for the relief of pain and tension, and both successful. ⁽³⁾ In one of these iridectomy had been previously performed four times (!) with only temporary benefit.

I have myself performed sclerotomy only twice, and upon the same eye in both cases. The different results which followed these two operations are so characteristic of the disadvantage and advantage of sclerotomy under different circumstances that I repeat the outline of the case here.

Bupthalmos ; cornea very large, T + 2. ⁽⁴⁾ Iridectomy twice, some years previously, had removed the iris from the inner and lower quadrant, but had not removed excess of tension ; throughout this quadrant the suspensory ligament was widely uncovered. Sclerotomy performed at upper part, where the iris was still present in considerable breadth, at once reduced tension below normal point ; five days later, excess of tension and pain recurred suddenly, induced, apparently, by certain *extraocular* exciting causes—viz., constipation, a restless night, headache, with much crying, and great suffusion of the vessels of the head and neck ; the iris at once applied itself to cornea, and evidently covered up and rendered inoperative the scleral cicatrix ; tension fell a little in a day or two, but remained as high as before operation. Sclerotomy repeated below—*i.e.*, in the portion of the circle

¹ *Therapeutique Oculaire*, Part I., p. 386 (1878). ² *Lancet*, May, 1876, p. 673.

³ *Lancet*, Oct. 7, 1876, p. 504. ⁴ Case 4, fig. 39.

devoid of iris—and a puncture made at the same time through the suspensory ligament; immediate reduction of tension as before. During following few weeks, tension rose gradually nearly to its former height, and then again subsided to a normal point, and has since remained strictly normal, in spite of occasional emotional disturbance and suffusion of face as before; the iris which, between the first and second operations, was in contact with the cornea, slowly recovered its freedom; it now stands at a considerable depth below the cornea.

Judging the operation of sclerotomy empirically on the evidence of its results, I think it may be declared without any hesitation to be inferior to iridectomy as a remedy for glaucoma, and to be inadmissible in the great majority of cases as a substitute for the latter. With regard to its adoption in cases of hæmorrhagic glaucoma I have no experience. On theoretical grounds, it appears to me to offer no very great advantage over iridectomy in such cases. The danger which is incurred by either operation is that of rupture of vessels through lowering of the pressure on their external surface. Sclerotomy, so far as I have witnessed its effects, produces an immediate reduction of tension equal to that which follows iridectomy. If hæmorrhage occurs it is, probably, at least as liable to produce a condition of “malignant glaucoma” after sclerotomy as after iridectomy, for the obstructive conditions are more easily reinstated in presence of the iris than when it has been excised. Rupture of the suspensory ligament, displacement of the lens, and extrusion of the vitreous are, doubtless, less likely to occur after the double puncture than after the completed incision. Experience alone must decide whether a larger number of such eyes are ultimately saved by the new than by the old operation.

In absolute glaucoma sclerotomy may, doubtless, sometimes take the place of excision, though even here I think it is not certain that such a substitution is for the patient's

benefit. In my own case the first sclerotomy, which was certainly made as far beyond the corneal margin as could be ever justifiable, was followed by increase of tension and considerable pain, through complications with the iris. The second was perfectly successful, for there was no iris to interfere with the result; but, in the latter, there is no doubt that the ciliary processes were incised, as also in the cases recorded by Nettleship. This constitutes a reason for the well-marked tension-lowering power of the sclerotomy incision, but is not a step to be practised with impunity when the fellow eye possesses sight that may be destroyed by sympathetic inflammation. In my own case I incised fearlessly, because the other eye had already been excised.

The arguments concerning the greater ease with which sclerotomy may be performed, the avoidance of "useless mutilation of the iris," and the optical and cosmetic disadvantages of a large gap in the circle of the iris, are, I think, undeserving of a moment's consideration in ordinary cases of glaucoma. The only ground on which iridectomy can be displaced by any other operation is a distinct proof that the latter suffices in a larger number of cases to remove the glaucomatous pressure with safety to the structure of the eye.

OTHER SUBSTITUTES FOR IRIDECTOMY.

I shall not attempt to review in detail the operations which have been introduced by Solomon, Hancock, Pritchard, and others, and which all consist in various modifications of paracentesis of the cornea or sclera, or both—*e.g.*, "intra-ocular myotomy," "division of the ciliary muscle," "phlebotomy of the choroid," &c.

There is not the slightest doubt that glaucoma has been many times relieved, and occasionally cured, by paracentesis.

in one form or another; and, on theoretical grounds, I believe that the simultaneous opening of the aqueous and vitreous chambers, or even the opening of the vitreous chamber only, may be a preferable proceeding to the evacuation of the aqueous chamber by a corneal puncture. But the disadvantage of these operations consists in this: they do not tend directly, as iridectomy does, to remove the obstructive changes at the angle of the anterior chamber; this region, if incised at all, is incised transversely, and the iris, an important element in the obstruction, is not removed. The operations in question have never gained a wide acceptance. ⁽¹⁾

In 1876 v. Wecker, in an article entitled "Glaucoma and Eye-drainage," gave a preliminary notice of a new method of treating glaucoma—viz., by the insertion through the tunics of a loop of very fine gold wire; and recommended its adoption in certain classes of cases—absolute glaucoma, hæmorrhagic glaucoma—cases in which high pressure persists in spite of iridectomy. ⁽²⁾ In a more recent article he recommends it only under more exceptional circumstances—namely, in cases of absolute glaucoma in which pain recurs again and again, and the patient nevertheless refuses to permit excision of the eye. From this it would appear that the operation has hardly justified the favourable expectations of its author. My own experience of it in three cases is anything but satisfactory, and inclines me to venture the opinion that it is unjustifiable in any case in which blindness is not already complete. For eyes which are totally blind, but which demand operative treatment for the relief of pain, my own conviction is that excision will, in the great majority of cases, be more truly beneficial than any other proceeding.

As regards the so called "drainage," I think it is by no

¹ See note, page 231. ² Arch. f. Ophth., XXII., IV., 209.

means certain that the operation deserves the name. An immediate escape of vitreous relieves the tension as in any other form of paracentesis, and may occasionally cause a glaucomatous condition to subside; but twenty-four hours later it will be found, in some cases at least, that high tension has returned. The subsequent excessive softening which occurs, and which, as v. Wecker states, may be permanent after the removal of the wire, is more suggestive of the lighting up of morbid changes within the eye, probably with detachment of the retina, than of a persistent draining away of the intraocular fluid. The operation appears to have been founded on the assumption that a diminished permeability of the sclera plays a part in the glaucomatous process, and to have been intended to re-establish something in the nature of an equivalent for this obstructed channel. We now know that such changes in the sclera have no existence in relation to the intraocular fluid, and that any channel of escape established through the choroid and retina is an entirely unphysiological one; such, at least, is the view which I have attempted to prove in the foregoing section.

Of the somewhat analogous proceeding—namely, trephining of the sclera—by which Argyll Robertson has attempted to effect a reduction of tension, I have no experience. The results recorded are not, I think, very encouraging for further trials, especially as it must now be granted that the operation, like the preceding one, has no power to restore the natural process of filtration.

ESERINE AND OTHER MYOTICS.

The power which myotics exercise over glaucomatous tension is a fact of comparatively recent discovery. Its importance ranks second only to that of the action of

iridectomy. Adolph Weber and Laqueur appear to have observed it independently of each other and almost simultaneously. ⁽¹⁾

The mode in which eserine acts upon the glaucomatous eye has already been suggested; it remains only to give proofs of its efficacy, and to lay down, as far as may be, the indications for its employment.

I abstract the following tonometrical measurements from two of the cases recorded in the Appendix:—

					<i>Impressions.</i>	
					<i>Right.</i>	<i>Left.</i>
Case 19.—Premonitory symptoms of glaucoma, patient aged 32					·69 mm.	—
					= T + 1 ?	
After using atropine					·40 mm.	—
					= T + 2 or + 3	
After using one calabar disc					·78 mm.	—
					= Tn.	
Same patient four months later, no calabar for 40 hours					·79 mm.	·77 mm.
30 mins. later, one calabar disc having been applied to each					·85 "	·84 "
Case 31.—Myopia, with glaucoma, age 62.						
June 21	...	...	...	...	·50 "	·55 "
" same afternoon	...	...	...	...	·50 "	·53 "
June 25	...	...	...	...	·51 "	·64 "
June 26, began to use pilocarpine, 1 gr. to 1 drm.						
June 27	...	...	...	...	·71 "	·70 "
" 28	...	...	...	...	·71 "	·72 "
July 5	...	...	...	...	·78 "	·70 "
" 12, is now using eserine, 1 gr. to 1 oz.						

¹ Arch. f. Ophth., XXII, IV., 216.

		<i>Impressions.</i>	
		<i>Right.</i>	<i>Left.</i>
Aug. 14, no drops for 28 hours	...	·67 mm.	... ·61 mm.
" " 60 minutes later, two drops of eserine solution having been applied to each eye		·71 "	... ·71 "
Nov. , no eserine for seven days		·46 "	... ·53 "
" " 60 minutes later, one calabar disc having been used to each eye	...	·56 "	... ·59 "
Dec. 6, no drops for 48 hours	...	·52 "	... ·55 "
" 7, eserine used last night	...	·59 "	... ·64 "

In several cases of chronic glaucoma I have obtained no benefit from myotics either as evidenced by visual improvement or by perceptible reduction of tension. In one case of gradual recurrence of tension after iridectomy vision improved slightly during treatment with eserine ; in two others no improvement was affected. In one case of acute glaucoma, probably supervening upon pre-existing chronic glaucoma in a somewhat advanced stage, eserine applied three times during the day before operation increased the chemosis very considerably, augmented the difficulty of the operation thereby, and was perhaps partly to blame for the unfavourable progress subsequent to operation. ⁽¹⁾

If we possessed a certain means of distinguishing the cases in which myotics are likely to be beneficial from those in which they may be hurtful, their value in the treatment of glaucoma would be greatly enhanced. Neither my own observations nor the published experience of others have as yet offered any decided information on this point, so far as I have been able to ascertain. Ad. Weber, in drawing attention to the efficacy of eserine, urged especial caution in its employment, inasmuch as he had observed its tendency to cause swelling of the ciliary processes to be productive of disastrous results. V. Wecker appears to use it before and after iridectomy in every case of glaucoma. Laqueur cites striking

¹ Case 35, Section VII.

instances of its value, especially in acute attacks, and proposes it as a valuable means of reducing the difficulty of iridectomy in these cases. ⁽¹⁾ He anticipates that it will obviate the performance of second iridectomies, and suggests that if its use were made known to the profession at large many eyes which are now lost during acute attacks for want of timely operative interference, might be saved from this disaster by removing or diminishing the urgency of the mischief. In a case of hæmorrhagic glaucoma he had to suspend its use at once by reason of the severe pain induced. He appears to have derived least benefit from it in those cases which, with deeply excavated disc, present but slight excess of tension, and good activity of pupil.

I have suggested, on theoretical grounds, that its efficacy depends in some cases upon a diminution in the diameter of the lens affected through the agency of the ciliary muscle. This explanation however, if true for some, can hardly be true for all cases, for amongst those cited by Laqueur is one of rapid disappearance of an acute attack in a patient aged 79. ⁽²⁾

Another explanation is the withdrawal of the periphery of the iris out of the angle of the anterior chamber—*i.e.*, out of its contact with the cornea, through the agency of the sphincter of the pupil.

The action of myotics upon the vessels of the eye, especially upon those chiefly concerned in the glaucomatous condition—*viz.*, the vessels of the ciliary processes—appears to be of a kind likely to aggravate the condition rather than to relieve it. The ciliary processes swell under the action of eserine, calabar, &c.; and this I think explains the intensification of the vascular symptoms—*e.g.*, chemosis—which sometimes follows their employment.

¹ Arch. f. Ophth., XXIII. III., 149.

² Arch. f. Ophth., XXIII., III., 168.

It appears probable that eserine has a power for good in proportion to the contractile power of the ciliary muscle and iris, a power for evil in proportion to the turgescence which it induces in the ciliary processes. A series of minute observations of the refractive condition of the eye (when possible) before and after its application, together with the effect upon the pupil, and the size of the episcleral veins, would probably throw light upon this question. With regard to my own theory as to the action of the lens, it must be ascertained at what period of life increase of refraction ceases to be producible by eserine. That the tension of the zonula upon the lens is still maintained in old age is manifested by the absence of a tremulous condition in the iris, but what the effect may be of a maximum contraction of the ciliary muscle—*i.e.*, a maximum relaxation of the zonula—upon the form and position of the senile lens is less certain. ⁽¹⁾

With regard to the comparative value of different myotics it has not, so far as I know, been proved that one is better than another except in the matter of strength. The calabar-gelatine discs of Savory and Moore are extremely convenient and efficient. One of my patients used half a disc daily to each eye for some months with only temporary pain, whereas she stated that severe pain followed the use of eserine and pilocarpine, even in very weak solution. To test the comparative "strengths" of the nitrate of pilocarpine and sulphate of eserine supplied to the Hospital I made a rough experiment with them simultaneously in my own eyes. One drop of a solution (1 gr. to 1 oz.) of each drug was used to the two eyes respectively. Alterations in refraction were ascertained by finding the correcting glass for distance; alterations in accommodation by noting the near point for fine lines drawn on white paper.

¹ See note, page 154.

The distances are given below in inches.

	RIGHT EYE.		LEFT EYE.	
	Eserine 1 gr. to 1 oz.		Pilocarpine 1 gr. to 1 oz.	
	Far point.	Near point.	Far point.	Near point.
Normal state. 7 p.m. drops applied.	Infinity.	5 ins.	Infinity.	5 ins.
7.5	50 ins.	4½ "	"	5 "
7.10	40 "	4 "	"	5 "
7.15	36 "	3¾ "	50 ins.	4½ "
7.20	30 "	3½ "	50 "	4½ "
7.25	24 "	...	50 "	4 "
7.30	24 "	3 "	50 "	4 "
7.40	30 "	3¼ "	50 "	4 "
7.50	50 "	3¼ "	50 "	4 "
8.0	Infinity.	4 "	50 "	4¼ "
8.15	"	4 "	50 "	4¼ "
8.45	"	4 "	Infinity.	4½ "
9.15	"	4½ "	"	4¾ "
10.30	"	4½ "	"	4¾ or 5

Contraction of the pupil was considerable in both eyes, but much more so in the right (eserine) than in the left (pilocarpine); it persisted longer than the contraction of the ciliary muscles, and in the right eye had not quite disappeared the next morning. The pilocarpine caused no pain, but reddened the conjunctiva; this I afterwards found to be due entirely to the presence of free acid. The eserine caused

twitching of the eyelids, and some pain in the eye—worst during strong accommodative effort.

This test was too rough to afford any accurate comparative information concerning the actions of the drugs on iris and ciliary muscle, if they differ in this respect; it showed, however, that in equal solutions eserine is much the stronger of the two. I have usually employed them in the following strengths :—

Eserine, one grain to one ounce.

Pilocarpine, eight grains to one ounce.

v. Wecker suggests that they are best applied at bed-time, so that the temporary obstruction of vision which ensues may pass away during the night and cause the patient no alarm.

On physiological grounds, also, I would suggest that if myotics are used at all, bed-time should at least be one of the times of their application. During the 6 to 9 hours of sleep the ciliary muscle is in a state of complete relaxation, and if the form of the lens be really concerned, as I believe, in causing glaucoma, this unbroken period, which has no parallel during waking hours, must be a time of especial danger. The frequent onset of glaucoma during sleep is a well-known fact. [It is worth noting, as regards the part played by the iris in glaucoma, that the pupil is *contracted* during sleep. It seems probable that this depends upon a relaxation of the arteries of the iris, and indicates the presence of an increased quantity of blood in the vascular tunic as a whole. Hence, perhaps, the liability to nocturnal exacerbation of glaucoma.]

GENERAL TREATMENT.

It remains only to indicate the general principles on which persons suffering from glaucoma should be treated, as distinguished from the more special measures which have already been reviewed. They are simple but by no means unimportant.

Experience and theory alike show that all conditions which tend to promote determination of blood to the head and eyes should be avoided as far as possible, and when arising should be counteracted with promptness and energy.

First among these are all sources of direct irritation of the eyes themselves. Exposure to strong wind, dust, great heat, excessive light, must be guarded against, if necessary, by some form of shade, of which the glasses known technically as "globulars" are the best. Prolonged use of the eyes upon any kind of work, especially if it involve a stooping posture, and require artificial light, must be forbidden. Secondly, such habits of life as tend to undue mental strain must be given up. The duties of a laborious profession, or the over-excitement of much society and late hours, especially if they induce restless nights and unrefreshing sleep, are sources of danger if glaucoma threatens or if it have been for the time subdued by operation. Constipation of the bowels, coldness of the feet, excess in eating, and especially in the use of alcohol, must be habitually guarded against. In cases, such as are not infrequent among women, where a tendency to glaucoma, or the actual existence of the malady in an incipient form, is associated with much nervous excitability, the use of bromide of potassium and chloral may do good service. Enemata, hot foot-baths, the artificial leech, may also occasionally give timely aid. All such remedies, however, are, be it remembered, to be used strictly as auxiliaries in the treatment of the malady when its presence is declared, and are on no account to be employed as substitutes for those measures which can immediately reduce the tension of the eye.

Quinine in considerable doses is much employed by some experienced surgeons during the after-treatment of glaucoma when iridectomy has been performed. I believe that I have seen it do good in cases complicated with an independent

neuralgic element. It has been stated to be generally useful by reason of its power to reduce the blood pressure. I have no knowledge of its efficacy in this respect. If such an action be desirable, other drugs could probably be employed with more marked effect. On the contrary, I am quite sure that in more than one case I have seen benefit follow the employment of digitalis. When a sufficient channel of escape for the intraocular fluid has been provided and the tension remains normal, the further deterioration of vision is due to a progression of the conditions of malnutrition and atrophy which the pressure previously induced. Under such circumstances, the use of such remedies as give strength and steadiness to the circulation and stimulate nerve nutrition—*e.g.*, digitalis, iron, strychnia, phosphorus—deserve a trial. It is not probable, however, that very much good will be effected by their means beyond that which is manifested in the improvement of the general condition of the patient.

NOTE, July, 1879.—Although the theory as to the origin of primary glaucoma which has been put forward in this essay is as yet a theory, and nothing more, it may not be improper to enquire whether, supposing it to prove well founded, it will afford any fresh indication for a preventive or curative treatment.

If it be true that primary glaucoma is essentially dependent upon a structural abnormality—undue narrowness of the “circumferential space”—rather than upon any morbid state of the system, or any external influence, then there is little hope that we shall ever be able to class it with those maladies which are preventible *ab initio*.

Extraction of the entire lens, performed at the very commencement of the disease, would seem to offer the possibility of complete and permanent cure; but the practical objections to such a proceeding would be very grave. The transparent state of the lens, its abnormally great diameter in relation to its surroundings, and the excess of pressure in the vitreous chamber, would render extraction difficult and hazardous; and no prudent surgeon, probably, would be willing to subject an eye to such risk at a time when its vision is still good, and when, in the patient's judgment, no heroic treatment of any kind is called for.

The action of the lens in inducing a “malignant” course after iridectomy is already tolerably well established (see page 170). For the relief of this condition Pagenstecher has proposed the extraction of the lens, and Ad. Weber has devised, and practised with success, a means by which the lens may be restored to its normal position. Knowing, as we do, that the mischief is

brought about by the forcible advance of the lens under the excessive pressure of the vitreous humour, and that it can be relieved only by reducing the vitreous pressure, might we not *prevent* the occurrence of this malignant condition by lowering the vitreous pressure before the lens has been driven forwards—*i.e.*, by making a small puncture through the sclera near to the equator, immediately before performing iridectomy? Especially in cases of chronic glaucoma, with very shallow anterior chamber, such a step might probably be taken with advantage. It would certainly not be dangerous, and although the softness of the globe thus induced would render more difficult the making of the incision, the subsequent steps of the iridectomy would certainly be facilitated. Inasmuch as it is not in our power to effect any permanent diminution of the diameter of the lens, any procedure by which we shall succeed in re-establishing or widening the circumlental space must act by causing a retraction of the ciliary processes. There can be little doubt that such a retraction occurs immediately after iridectomy, by reason of the relief of the venous compression, which previously maintains a condition of passive turgescence (see page 167). Is it not possible that, in consequence of the intimate vascular connection between the ciliary processes and the iris, a certain number of the processes, corresponding to the portion of the iris which has been excised, remain permanently of smaller size, and thus maintain that part of the circumlental space in a state of patency? When swelling of the conjunctiva around the margin of the cornea follows iridectomy for glaucoma, the chemosis is absent opposite the incision.

Is it not possible, again, that the incisions in the ciliary region, practised by Solomon and others for the relief of tension, cause a collapse and atrophy of the incised ciliary processes, and thus reopen the space between themselves and the margin of the lens? If it were compatible with safety to make an incision of some length transversely to the great vessels of the ciliary processes—*i.e.*, just in front of and parallel with the ora serrata—it seems probable that the desired object—collapse and retraction of several of the processes—might thereby be attained.

ANÆSTHESIA BY CHLORAL AND ETHER.

NOTE.—During the last six or eight months I have adopted the practice of giving to patients who are about to undergo surgical operations a dose of hydrate of chloral a short time before the administration of ether. This procedure has appeared to me to have, in many cases, such decided advantages that I desire to recommend a trial of it to others.

In the case of a boy, aged seven, it was necessary to employ an anæsthetic several times within a period of ten days. After the first administration of ether the child's dread of its repetition was so intense, and so distressing to his parents, that I determined on the next occasion to abolish his consciousness before my arrival. Ten grains of chloral given twenty minutes previously had the desired effect. A very few inhalations of ether given during a state of extreme drowsiness from chloral (not amounting, however, to sound sleep) enabled him to undergo the dressing of a painful wound without the slightest reflex movement. Being then aroused from the narcosis sufficiently to move and speak, he was allowed to sleep off the effects of the chloral, and finally awoke in ignorance of my visit.

I next adopted the same plan in the case of a highly hysterical girl, whose first experience of ether was followed by violent and prolonged sickness, with great prostration. On the second occasion, some months later, I ordered for her fifteen grains of chloral thirty minutes before the operation. Though not asleep when the administration of ether was begun, she took it with much less excitement than before, was very slightly sick once, and slept of the nausea in an hour or two under the influence of the chloral.

Since that time I have pursued the same course in about forty cases. I am not able to give exact details as to the amount of ether employed, the degree of excitement at the outset, the struggling, the subsequent tendency to sickness, &c., but I can with confidence assert that in these cases, as compared with ordinary ether administrations—(1) the previous excitement and apprehension have been diminished; (2) the resistance at the outset has been almost abolished, and the irritability of the breathing passages has appeared to be lessened; (3) the amount of ether employed has been less; (4) the subsequent tendency to sickness has been very greatly reduced.

At the present time, whenever I operate in a private house I give minute instructions beforehand that at a certain hour the patient shall be placed on the couch or bed in the requisite position, and then receive the draught as prescribed. Arriving twenty to thirty minutes later, I find him, or her, in a condition of tranquility or semi-oblivion, which under other circumstances is rare at that moment, and which greatly facilitates all that has to follow. In the case of children, were it desirable so to do, we might probably promise in almost every instance that the little patient should know nothing of the surgical operation except by its after effects. For them, however, chloroform will probably continue to be the favourite anæsthetic. The operation in which the chloral has rendered me the most signal service is the extraction of senile cataract. Here the tendency to vomiting constitutes so formidable a danger that on this account alone anæsthetics should be withheld unless we can guarantee their being well borne. The only cases in which it has seemed to be almost without effect have been those of chronic alcoholism. Such patients always take anæsthetics ill.

Several of my colleagues who have witnessed this method of inducing anæsthesia have expressed themselves as favourably impressed with its results. Dr. Carter, especially, who has administered ether for me thus many times, speaks strongly in its favour.

The dose for an adult has hitherto been fifteen to twenty grains thirty minutes before the operation. A gentleman who is in his eightieth year took the larger dose last week with excellent effect. Probably a larger dose might generally be given with propriety. As regards the safety of the proceeding experience must determine. To me it has appeared to diminish the danger by reason of the freedom from excitement, the absence of struggling, and the smaller amount of ether employed; but on this point I speak with no certainty, and would urge that in the trial of this method, as in every case where anæsthetics are concerned, due caution be observed.

(This note is reprinted from the *Birmingham Medical Review*, July, 1879. It appeared in the original essay in a different form.)

SECTION VII.

APPENDIX.

For convenience in reference, and in accordance with the arrangement prescribed for the original essay, recorded cases—the evidence upon which much that is stated in the foregoing pages rests—are here placed together in an appendix. The bracketed letter and number which follow the number of the case indicate the place in the hospital registers, where the case is fully recorded. In connection with each case a short description of the figures which illustrate it is given. The specimens and slides, to which reference is also made, are those which accompanied the original essay, and are now in the museum of the College of Surgeons; the descriptions are retained here in order to facilitate reference. At the end of the Appendix is given a series of records of “tension,” normal and abnormal, as estimated by the tonometer.

CASE 1.

(D. 129).—*Primary glaucoma, acute, absolute.*—Eliza P——s, 55. Rt. Hm. = $\frac{1}{8}$. Lt. Absolute glaucoma T + 2, V = 0, disc invisible by reason of semi-opacity of lens; cornea slightly hazy. The sight of this eye “went altogether” in about a week, nine months since; much pain and redness for some weeks: no pain now; incurable. Twelve months later patient returned; acute attack (Lt.) began five days ago; T + 3, severe neuralgic

pains in orbit and head, hyperæmia, ulceration of centre of cornea. Lt. excised.

Specimen 1.—Central corneal opacity; lens somewhat shrunk by the action of the glycerine jelly in which the specimen is preserved, but evidently advanced so as to make anterior chamber shallow; pupil widely dilated; peripheral adhesion of iris and cornea (see slides); extreme atrophy of ciliary processes, except the anterior free portion; equatorial staphyloma; deep excavation of optic disc undermining the scleral margin.

Fig. 11. Slide 1a.—Peripheral adhesion of iris and cornea co-extensive with and evidently produced by pressure of ciliary process; ciliary process swollen anteriorly, atrophied posteriorly.

Fig. 12. Slide 1b.—Peripheral adhesion, as in last; extreme retraction of iris, apparently with an increase of thickness; the section is taken in an interval between two processes, hence the absence of the swollen apex.

Slide 1c.—Similar to slide 1a.

CASE 2.

Primary glaucoma, chronic, iridectomy, hæmorrhage, excision.—(The patient was under the care of Mr. Eales, who gave me the specimen): "Henry H——e, 70. Had been nearly blind some months, perhaps longer, before applying to Mr. Eales. Rt. T + 3, pain, faint perception of light. Lt. T + 3, pain, V = 0. Anterior chamber very shallow in both eyes. Iridectomy performed in each to relieve pain and tension. Subsequent course not accurately known. Five or six weeks later the patient returned, having suffered much pain in left. Lt. excised.

Specimen 2.—(In snipping away some loose cellular tissue from the specimen the apex of the cystoid cicatrix was accidentally cut off, and with it a portion of the margin of the lens which was wedged into it.) Lens tilted on one side, its anterior edge forced into the staphylomatous cicatrix, its posterior edge lying behind and against ciliary processes at opposite side of circle. (The lens was removed in mounting, so as better to display the parts behind it.) Blood

clot between choroid and sclera. Blood clot in vitreous chamber. Retina not separated from choroid.

Fig. 15. Slide 2a.—Peripheral adhesion of iris and cornea corresponding with swollen ciliary process; the latter, pressed forward by the lens or suspensory ligament, has forcibly driven the iris into the cornea with deep indentation. For a short distance beyond this adhesion iris is almost in contact with cornea, but not adherent; nearer to pupil it is again adherent for a short distance; the latter is a recent adhesion due to pressure of lens after iridectomy. On posterior surface of iris and cornea a portion of lens capsule remains adherent. The extremity of cornea shows one swollen lip of iridectomy incision.

Fig. 16. Slide 2b.—Section through incision near to one extremity where lips were not widely asunder. Iris incarcerated and adherent in angle of wound.

Slide 2c.—Similar to last.

Fig. 50. Slide 2d.—Optic disc, shallow saucer-like excavation.

CASE 3.

Glaucoma (? primary or secondary).—Henry P—h, 50. Rt. eye failed gradually twenty-five years ago, and has been quite blind for twenty years; occasional shooting pain in it throughout the whole of that time; six weeks ago it became very painful and red. Sclera much injected, pupil widely dilated, the fibrous coat of the iris retracted so as to expose the uvea at the free margin; lens transparent; immediately behind lens a greenish-yellow appearance resembling purulent infiltration of vitreous. $V = 0, T + 2$. Lt. eye healthy, $V = \frac{20}{20} T n$. Rt. excised.

Specimen 3.—Peripheral adhesion of iris and cornea (the lens was transparent; it dropped out in mounting); considerable atrophy of ciliary processes; considerable morbid change (? inflammatory) in the retina at the ora serrata, perhaps beginning in the “pars ciliaris;” detachment of portion of retina corresponding to this part of the ciliary circle (the detachment appears greater in the mounted specimen than it was when the eye was first opened, in consequence of shrinking of the detached membrane); very large colloid cells

on the surface of the denuded choroid. The other half of the eye is unfortunately destroyed, and no note was made of the condition of the disc. The nature of the primary disease remains obscure. The point of interest is the peripheral adhesion of iris and cornea in connection with excess of tension.

Slides 3a, 3b.—Peripheral adhesion of iris and cornea.

CASE 4.

(*F. 196.*)—*Secondary glaucoma, buphthalmos, hereditary syphilis, iritis or kerato-iritis, unsuccessful iridectomy, excision.*—Sarah T——n, 21, notched teeth, &c. Eyes first inflamed twelve years ago; was a long time under treatment elsewhere; each eye was operated on twice.⁽¹⁾ Rt. (fig. 38) cornea much enlarged; hazy throughout under oblique illumination; iris forms a crescent, the concave edge locks downwards and inwards, leaving a gap equal to $\frac{1}{3}$ of the circle; at each horn of the crescent a cicatrix is discoverable, left by former iridectomies, one downwards, one inwards. General enlargement of globe; anterior ciliary veins and arteries dilated; latter cannot be emptied by finger pressure. Lens transparent, *in situ*; $\frac{1}{3}$ of its periphery exposed to view by absence of iris and increased diameter of cornea. Optic disc (fig. 48) excavated quite up to margin, not abruptly, but with a rounded curvature; its oblique position causes nearer side of excavation to be hidden and to appear undermined; vessels very thin and free from sharp bends or curves; arteries hardly to be distinguished from veins; a large atrophic crescent to outer side of disc, its transverse diameter about equal to half diameter of disc; a narrow atrophic line round remainder of disc. $M = \frac{1}{3}$, $T + 1$ or 2, impression .54 mm., $V =$ perception of lamp in a small central area of retina, the size of which cannot be exactly determined. Lt. cornea much enlarged, semi-opaque, eye very prominent; anterior ciliary vessels dilated; frequent pain in eye and forehead. $V = 0$, $T + 1$, impression .59 mm. Eye excised for relief of pain.

Specimen 4.—Cornea 14 mm. diameter; anterior chamber 5 mm. deep; globe 32 mm. in antero-posterior axis, 24 mm. in

¹ See also Section V., p. 180.

transverse axis. Iris adherent to shrunken remnant of lens ; ciliary processes atrophied ; posterior to ora serrata a band of pigment spots situated in choroid, and adherent in places to retina ; large rounded excavation of disc, with vessels still containing blood traversing its sides. (The excavation is extremely well seen when the specimen is well illuminated.)

Fig. 21. Slide 4a.—Angle of anterior chamber open ; the tissue of ligamentum pectinatum has perhaps undergone some inflammatory proliferation, but this is not certain ; ciliary process atrophied.

Fig. 22. Slide 4b.—Peripheral adhesion of iris and cornea, which has undergone stretching subsequent to its formation, probably after the occurrence of the iritic adhesion to the lens ; ciliary process atrophied.

Fig. 23. Slide 4c.—Extensive adhesion of stump of iris with cornea ; cicatrix opposite the point where iris terminates, evidently the result of iridectomy ; the distance between cicatrix and ciliary process has probably increased since the operation, by extension of cornea. It appears probable that the lens was injured at one of the operations, and that its subsequent shrinking, when adhesion with the iris had occurred, tended to stretch the adhesions at the angle of anterior chamber, and to re-establish a deep anterior chamber.

Slide 4d.—Peripheral adhesion of iris and cornea ; ciliary process atrophied.

Fig. 51. Slide 4e.—Excavation of optic disc.

Slide 4f.—Excavation of optic disc.

Eserine was used to right eye for a week without any lowering of tension (impression .54), and as pain was frequently complained of, and the remnant of vision was worth saving, sclerotomy was performed.

October 11th. Choral gr. 20, ether twenty minutes later ; (fig. 39) the point of a Graefe's knife introduced through sclera on a level with upper corneal margin, carried across anterior chamber so that its edge was hidden in the peripheral angle ; counter-puncture in corresponding situation ; knife withdrawn without any sawing

movement; T — 2 immediately. *Same evening*, hardly any pain, conjunctiva bulging, from effusion of aqueous humour beneath it, around punctures; T — 2.

Fourth day after sclerotomy. No pain, conjunctival injection nearly gone; punctures healed, visible as greyish semi-transparent lines in the white sclera, the adjacent conjunctiva bulged by subjacent fluid; iris and lens unchanged as to position; an extravasation of blood on the atrophic crescent adjacent to the disc; T — 2, perception of daylight better than before.

Fifth day. Pain came on in the night, apparently after a fit of crying; the bandage appears to have made pressure on the eye. T + 1.

Sixth day. Pain increased; T + 2; very little sleep and much crying last night; face and neck deeply suffused this morning; bowels have not acted for four days; enema. During the following few days, the pain entirely subsided, but T + 2 remained, and the lens and iris remained pushed forward, the latter being in close contact with cornea above so as to entirely to occlude the puncture and counter-puncture cicatrices (see fig. 40).

October 29th. Choral and ether as before; sclerotomy repeated at lower part where subsequent obliteration by the iris cannot occur; after withdrawing the knife, a broad cutting needle was passed vertically through cornea close to its margin, and midway between puncture and counter-puncture, so as to incise suspensory ligament, and thereby establish open communication between vitreous and aqueous chamber (fig. 39). T — 2 or — 3 immediately.

Following day, T — 2, no pain.

Second day. Patient very nervous; cries when spoken to; face and neck flushed; T + 1?

Third day. No pain; T — 1.

During the following few weeks tension varied between T n and T + 1, and there was occasional slight pain; the iris gradually retired completely from the cornea and assumed a position at a considerable depth from it (fig. 41).

December 10th. No pain, T n. Impression .82 mm.

December 17th. No return of pain or tension ; perception of light perhaps slightly improved.

CASE 5.

(*G. 6*).—*Secondary glaucoma ; wound of cornea, iris, and lens ; anterior and posterior synechia.*—Charles C——s, 44. Was struck by a fragment of stone four months ago, and almost blinded ; more or less pain ever since. Rt. V = 0 ; T + 2, impression .44 mm. ; a white cicatrix extends from centre of cornea to its lower margin ; iris adherent to it ; anterior chamber almost annihilated ; pupillary margin of iris adherent to lens or opaque capsule ; ciliary vessels injected. Lt. E. V $\frac{1.8}{3.0}$ Tn, impression .74 mm. ; patient complains that this eye is weak and that he cannot work. Rt. excised.

Specimen 5.—Iris adherent to corneal cicatrix, also peripheral adhesion of iris and cornea (see slides) ; shrunken remnant of lens adherent to iris (¹) ; disc, as seen in specimen, shows no cupping, because only a peripheral portion remains ; the central portion of the surface of disc was excavated, though not up to the margin ; the lamina cribrosa somewhat pushed backwards.

Fig. 20. Slide 5a.—Peripheral adhesion of iris and cornea in a part of the circle where the iris is not involved in the injury ; cicatricial mass involving cornea, iris, lens substance (?) and capsule.

Slide 5b.—Similar to last.

Slide 5c.—Shows how the peripheral adhesion depends upon the pressure of the swollen ciliary processes. It is not improbable that the latter have been dragged forwards somewhat during the shrinking of lens.

Slide 5d.—Peripheral adhesion as before ; iris stripped of its posterior pigment layer, corresponding with a similar denuded area visible in the specimen, perhaps occurring during division of the eyeball into halves.

Slide 5e.—Similar to slide 5c.

Slide 5f.—Optic disc ; central depression in the retinal fibres ; slight pushing back of the lamina cribrosa. (The section is so thick

¹ See Section V., p. 178.

as to be almost useless, but is the only one showing the condition of disc. Under low power and strong illumination the conditions may be made out.)

CASE 6.

Secondary glaucoma—glioma retinae; Bevis H—s.—White appearance first observed twelve months ago. T + 2, pupil widely dilated, anterior chamber very shallow; injection of anterior ciliary vessels, slight redness and swelling of lid, came on only four weeks ago. Immediate excision (death four months later).

Specimen 6.—(The detachment of choroid and retina occurred in mounting; lens dropped out.) Amount of glioma in retina not great; great enlargement of optic nerve by new growth.

Fig 13. Slide 6a.—Peripheral adhesion of iris and cornea; extreme atrophy of ciliary process and attenuation of ciliary muscle.

Slide 6b.—Similar to last.

Slide 6c.—Similar to last; includes cornea and ciliary region at each side. From the small size of ciliary processes, the extensive peripheral adhesion, ⁽¹⁾ and the extreme shallowness of anterior chamber observed during life, it is probable that the lens margin was in direct contact with periphery of iris.

CASE 7.

Secondary glaucoma; wound of ciliary region, iridocyclitis, detachment of retina, probably — T; hæmorrhage, + T.—James W—h, 40. Received a shot in right eye eighteen years ago; pain for three or four months afterwards with rapid loss of sight; no trouble with it since then until a few week ago, when severe pain set in. Rt. V = 0, T + 2, pupil small, completely adherent to lens, great injection of ciliary region; violent pain every night. Lt. acute iritis (? sympathetic); pupil adherent in many places; constitutional syphilis. Rt. excised.

Specimen 7.—Total synechia posterior; lens uninjured; retina detached, extensively adherent to choroid and ciliary processes in the neighbourhood of the cicatrix; the space *within* the folds of the

¹ Compare fig. 54. Case 16.

detached retina filled with recently extravasated blood ; atrophy of ciliary processes and choroid. [It is almost certain that up to the time of the extravasation this eye was soft, probably T - 2 or 3 ; the blood was poured out and rapidly raised the intraocular pressure to a point more nearly approaching the blood pressure ; the high tension subsided very slowly, because the angle of the anterior chamber was closed and no relief by escape of aqueous humour occurred ; the tension had, however, fallen nearly to the normal height on the day of the operation.] (¹)

Fig. 14. Slide 7a.—Cicatrix dividing the ciliary process ; peripheral adhesion of iris and cornea infiltrated with blood ; adhesion of iris to lens capsule ; extensive hæmorrhagic infiltration.

Slide 7b.—Similar to last.

Slide 7c.—Optic disc ; doubtful whether the glaucomatous pressure has effected any change ; lamina cribrosa appears slightly pushed back.

CASE 8.

This case is noticed in sufficient detail in Section V., page 195.

Fig. 24. Specimen 8, slides 8a, 8b, (fig. 53) 8c, 8d, 8e.—It exhibits a peculiar disarrangement of the lens produced by injury ; a closure of the angle of the anterior chamber affecting a portion only of the circle ; a pushing back of the lamina cribrosa without true excavation of the disc.

CASE 9.

This case is noticed in Section V., page 194.

Fig. 25. Specimen 9, slides 9a, 9b.—It exhibits total adhesion of iris to a staphylomatous cornea—*i.e.*, *total closure of the angle of the anterior chamber without glaucomatous consequences.* The reason is discussed in the passage referred to, as also of the following cases.

¹ See Section V., p. 189.

CASE 10.

This case is noticed in Section V., page 192.

Fig. 26. Specimen 10, slides 10a, 10b.—It exhibits *total closure of the angle of the anterior chamber* coexisting with *subnormal tension*.

CASE 11.

The case is noticed in Section V., page 193.

Fig. 27. Specimen 11, slides 11a, 11b.—The patient was four years old. The eye was excised just six months after it was injured; T — 2. It exhibits *total obstruction of the angle of the anterior chamber* by the iris, and imprisonment of an albuminous secretion in the posterior aqueous chamber, coexisting with *subnormal tension*.

CASE 12, 13.

Alfred H——n, 27. Lt. blind eight years; no history of accident; T — 2. V = 0, occasional pain; an appearance “like dissolving views in a magic lantern” frequently before the eye; iris tremulous, pupil small, occluded by a false membrane or a cretaceous lens.

Specimens 12, 13, are the two halves of this eye. They exhibit one of the causes of detachment of the retina as described in Section IV., page 139. The ciliary processes are in a condition of extreme atrophy.

CASE 14.

(F. 210.) *Specimen 14, slides 14a, 14b.*—*Buphthalmos*, high tension, spontaneous ruptures of each iris, almost total loss of sight, “drainage” by gold wire, temporary improvement as regards tension, ophthalmitis, sympathetic iritis, excision.—William F——, aged 6, brought to hospital April 2nd, 1878. Variola, at the fifth week after birth, had been immediately followed by inflammation of both eyes, and permanent loss of sight, almost total. No operation had at any time been performed on either eye; of this there could be no doubt, as the child had never been separated from the mother in

any hospital. Both eyes much enlarged ; diameter of cornea in each 15 mm. (measured when under chloroform for operation) ; anterior ciliary arteries and veins very much enlarged ; the former cannot be emptied by finger pressure.

Fig. 36.—Rt. V appears limited to perception of light and colours ; cannot tell fingers or any other object close before the eye, but on receiving some bits of coloured paper holds each in succession close to the eye and names colour correctly. T + 2. Cornea clear ; pupil small, irregular, adherent throughout to lens capsule ; in iris, below pupil, is an oval gap simulating an artificial pupil from iridectomy ; it is, however, a spontaneous rupture limited centrally by the adhesion of the pupillary margin, peripherally by the origin of the iris, or more probably by a peripheral adhesion between iris and cornea ; the rupture has doubtless been produced by stretching of the iris, accompanying enlargement of the circle of its peripheral attachment. With the ophthalmoscope the margin of the lens is seen through this gap. Lt. V = 0, T + 2.

Fig. 37.—Cornea clear, diameter 15 mm. ; iris has parted from its peripheral attachment throughout the outer and lower third of the circle, and appears as a large crescent throughout the remainder ; through the gap thus left a large portion of the margin of the lens is visible with the ophthalmoscope when the eye is viewed in the direction of the visual axis, for the diameter of cornea is considerably greater than that of lens ; near the lower horn of the crescentic iris is a small oval aperture in it, parallel with the free border, evidently another spontaneous rupture, through which lens margin is again visible. The concave margin of the crescent is formed in part by a transparent membrane to which the iris fibres are adherent in an irregular manner. This appears to be a false membrane which has formerly occluded the pupil, and which during the stretching of the cornea and ciliary circle has so held the pupillary margin together as to necessitate a rupture at the periphery. The false membrane has itself undergone rupture in three or four places, visible with the ophthalmoscope as so many clear oval holes in it. If not adherent to the lens, it is probably in close contact with it. A round bright spot—

"anterior polar cataract"—occupies the centre of the lens capsule. The child appeared free from pain, but could not open the eyes steadily; the lids winked constantly, and the brows wore a perpetual frown. If any treatment could be of use it must manifestly be directed to the lowering of the tension of the eyes. Iridectomy appears seldom to produce good results in such cases (probably because of the extensive and long-standing peripheral adhesion of iris with cornea), and I determined to attempt a reduction of tension by v. Wecker's gold wire drainage, as the parents desired that the boy should have the chance of any benefit that might be possible. ⁽¹⁾ My object was rather to avoid disfigurement of the child by further distension of the eyes, than the improvement of the sight, which appeared impossible. By way of precaution I operated first upon the totally blind eye. The tonometrical measurements in this case cannot be relied upon as accurate, for the unsteadiness of the eyes made it difficult to apply the instrument properly, although the child made no objection at any time to its use. Still, allowing a margin for error, their indications are important. *Before operation*—Rt. T + 2, impression .43 mm. Lt. T + 2, impression .43 mm.

May 31st. Chloroform, a single gold thread inserted in left eye by the method elsewhere described. The escape of vitreous which occurred on the making of the counter-puncture at once reduced the tension to T — 3.

June 2nd (second day after operation). The eye now feels again considerably too tense, though perhaps slightly softer than before operation. The swelling up of the conjunctiva which immediately followed operation, through effusion of vitreous fluid beneath it, has disappeared. Around the two points where the wire perforates the sclera there is still a small circumscribed bulging, probably due to subconjunctival effusion; considerable injection of conjunctival and subconjunctival vessels, especially round these points.

June 18th (eighteenth day after operation). Not much change since last note; a little conjunctival vascularity and thickening

¹ Compare Case 4.

around the wires ; no pain or discomfort is evinced ; there was probably some pain during the first twenty-four hours, but not since. The eye still feels too tense. $T + 2$, impression .54 mm. Having thus, as I believed, proved the safety of the operation in the case of the worse eye of the two, I determined to repeat it upon the other, using, however, a double instead of a single wire, hoping thereby to render the complete plugging of the punctures less likely to occur, and to obtain in consequence a more marked lowering of tension.

June 21st. Chloroform. A double gold wire introduced into the right eye ; immediate reduction of tension to $T - 3$, through escape of vitreous beneath conjunctiva.

June 22nd (first day after operation). Little, if any, pain since operation ; slight swelling of eyelids.

June 24th. Subjunctival extravasation of blood over the outer half of the eye ; no pain ; the eye feels a little less hard, but is still too hard.

June 29th (eighth day after operation on right). Rt. $T + 1 ?$ impression .63 mm. Considerable swelling or thickening of conjunctiva remains around the punctures, and increased injection of the vessels in the same neighbourhood ; no pain, no increased secretion. Lt. bulging of conjunctiva around ends of wire almost gone ; a little extra vascularity remains ; no increased secretion, no pain. Tn., impression = .82 mm. Both eyes appear to be free from any discomfort, and are opened more easily than before operation.

July 2nd. Rt. $T + 1 ?$ impression .61 mm. Lt. Tn., impression .82 mm. These measurements are almost identical with those taken three days before, and show a marked lowering of tension as compared with those taken before operation. The difference between the measurements of the two eyes depends in part upon the fact that in the right eye the instrument had to be applied much nearer to the cornea than in the left. The tension is, therefore, more nearly alike than the figures indicate. The child says he "sees better," but his statement cannot be relied on. An accurate test of vision is impossible. It is certainly not worse. *Patient discharged* (the

wires remaining in the eyes), and to be brought frequently for inspection.

The remainder of the case is a history of disasters, and may be very briefly told. Four days after his discharge the child was brought to me with the left eye in a state of acute internal inflammation, threatening suppuration. Pain had come on suddenly two days previously, without known cause, whilst the child was playing. He was at once readmitted, placed under chloroform, and the wires removed from both eyes. The right eye showed no sign of inflammation, but had become very soft since the examination four days previously; T—3. After several weeks in hospital, during which time the inflammation of the left eye subsided, and the globe shrank considerably, the child was again sent home. When next seen, some weeks later, the right eye also was acutely inflamed, probably through sympathetic action. The shrunken left eye was excised (specimen 14). He is still under care for the sympathetic inflammation of the right eye. (May, 1879. From this he recovered in a few weeks. T — 2. V = 0).

CASE 15.

Fig. 55, slides 15a, 15b, 15c.—Large sarcomatous tumour within the eye; no excess of tension. This case occurred in the practice of Mr. Solomon; it has already been referred to in detail. ⁽¹⁾

CASE 16.

Fig. 54, slide 16a.—Glioma of retina, entirely filling vitreous chamber and compressing lens firmly against cornea, with almost complete obliteration of iris and ciliary processes.—The specimen which is now in my collection was sent to me by Mr. Hodges, of Leicester. The slide and figure are exhibited merely to demonstrate the remarkable change of form which the lens may undergo when subjected to pressure. ⁽²⁾

¹ See Section V., p. 186.

² Section V., p. 174, note.

CASE 17.

(*F. 128*).—*Secondary glaucoma, posterior synechia, unsuccessful iridectomy.*—Henry S——s, 45. Rt. circular synechia posterior; great injection of ciliary region; pain; T — 1, V fingers at 6 ins. Lt. (the eye in question) T + 2, V = 0; was first inflamed fifteen years ago; many subsequent attacks; quite blind for five or six years. Iridectomy was performed on this eye three weeks ago by a skilful operator. The incision is now visible in the upper part of cornea, $\frac{1}{18}$ inch within its margin; it is healed, but bulging, and iris appears to be entangled in it; a partly decolourised blood clot in anterior chamber. Excised.

Specimen.—The globe presents three large equatorial staphylomata, which give it a distinctly triangular shape. The retina, choroid, and sclera are all adherent, and greatly attenuated in the staphylomatous parts. In the posterior hemisphere are numerous small round recent retinal hæmorrhages—the consequence, doubtless, of the operation three weeks previously.

Fig. 17. Slide 17a.—Peripheral adhesion of iris and cornea coextensive with, and evidently caused by, pressure of ciliary process.

Slide 17b.—Similar to last.

Fig. 18. Slide 17c.—Section through the position of the iridectomy, as indicated by a complete gap in iris; peripheral adhesion of stump of iris to cornea; the operation has failed to reopen angle of chamber, the incision having been made at a considerable distance from sclera; the cicatrix of the incision was, unfortunately, not included in the portion from which the sections were cut, nearly the whole of the cornea, including the cicatrix, having been removed previously, with a view to testing the intrinsic strength of the “partition.” The result of this experiment proved to be of no value.

Slide 17d.—Similar to last. A portion of iris remains adherent to cornea near to the incision.

Fig. 52, Slides 17e, 17f.—Optic disc.

CASE 18.

Secondary glaucoma, posterior synechia.—I have no notes of the clinical history. The eye was given to me by Mr. Eales, divided into anterior and posterior halves, with note: "A hard eye; posterior synechia of long duration; patient an elderly woman." In preparing the specimen for examination the lens, suspensory ligament, and adherent uveal surface of iris became detached.

Fig. 19, Slide 18a.—The angle of anterior chamber is *not completely closed*; Schlemm's canal remains open. The appearance of the pigment cells on the inner surface of the cornea, opposite to the periphery of iris, indicates, however, that these parts have been in contact, if not absolutely adherent.⁽¹⁾ The position of the ciliary processes suggests that this contact has been occasioned, not by the intervention of the processes, but by the direct agency of the lens, to which the iris was probably adherent throughout the greater part of its posterior surface.

Slide 18b.—Similar to last.

Slide 18c.—Optic disc. Excavation indicates continuance of excessive pressure. The retina was not detached when the eye was first opened, but assumed an altered position while in Müller's fluid.

Slide 18d, 18e.—Portions of sclera, exhibiting large veins perforating it, patent, and containing blood.

CASE 19.

(*F. 173*).—*Premonitory symptoms of glaucoma in a woman aged 31; acute glaucoma induced by atropine, and rapidly subdued by calabar.*—Elizabeth P——, aged 31, married, came to the hospital February 19th, 1878. She complained of dimness of sight coming on every evening, so that sewing and reading were impossible, and an inability to open the eyes properly during these attacks; also of frequent sharp pain darting through the eyes, temples and forehead, especially on left side. She was thin, pale, and nervous, flushing readily when spoken to; had been under

¹ See Section V., p. 177.

treatment for many months, and by several practitioners, for some uterine disorder; at subsequent visits she manifested a highly hysterical temperament; nearly fainted during a sphygmographic examination of the pulse, and was greatly agitated by a physical examination of the chest. Rt. E, $V = \frac{20}{20}$, accommodation very weak. Lt. E, $V = \frac{20}{20}$, accommodation very weak. Considering the case to be one of asthenopia from debility, I ordered iron and strychnia, and glasses (+ 40) for sewing and reading.

March 1st (ten days later). Patient reported the eyes worse rather than better. Re-examined more thoroughly. Rt. E, $V = \frac{20}{20}$, near point 9 ins. Lt. E, $V = \frac{20}{20}$, near point 10 ins. Anterior ciliary veins somewhat enlarged; pupils active and of medium size; optic discs normal; pulsation of a short portion of a vein on the left disc, between the point where it is crossed by an arterial trunk and its disappearance into the disc (see fig. 45). Each eye $T + 1$? (the tonometer was not yet completed); no contraction of visual fields; frequent darting pain in and around eyes; several teeth decayed, but no pain in them.

During the following few weeks patient took quinine, iron, and bromide of potassium. Though suspicious of the commencement of glaucoma, I deemed immediate operation unjustifiable, seeing that increase of tension was not pronounced, and vision was not impaired as regards acuity, and that in any case the action of iridectomy in such conditions and at so early an age would be doubtful.

April 30th. More pain the last week; it is worse at night, and is always increased by stooping and by washing the face, whether with warm or cold water (possibly because of the stooping involved). Occasional sharp pain in teeth for half an hour at a time last week. Rt. E, $V \frac{20}{20}$, near point 10 ins, impression .69 mm. Lt. E, $V \frac{20}{20}$, near point, 10 ins, impression .69 mm. The depth of the tonometer impressions indicated a tension just above the normal limits, but as I had at that time not yet ascertained what these limits are, in the case of my own instrument, I did not appreciate the results obtained. Believing that the complaints of pain were possibly exaggerations, and that a latent

hypermetropia might be concerned therein, I determined to try the effect of atropine, and ordered drops of a 1-grain solution to be used thrice daily.

Three days later patient returned, alarmed by an increased dimness of sight, which, however, was explained by the development of hypermetropia with paralysis of accommodation, thus:— Rt. H = $\frac{1}{38}$, V = $\frac{20}{20}$. Lt. H = $\frac{1}{38}$, V = $\frac{20}{20}$. No difference was observed in the appearance of the anterior ciliary vessels. She was ordered to continue use of drops, and not to alarm herself without cause.

May 9th (eight days after beginning use of atropine). The husband called on me to say that his wife was in bed, suffering violent pain in the eye. I saw her late the same night, with Mr. Potts, who had been attending her for a uterine hæmorrhage. She was in a state of extreme nervous depression and exhaustion; violent pain in the right eye had come on about twenty-four hours previously, and still continued worse rather than better; she had vomited and retched all day, and swallowed nothing. *The right eye was in a state of acute glaucoma.* T + 2 or 3, impression .40 mm. Episcleral vessels and conjunctiva intensely injected; pupil dilated and fixed; cornea slightly hazy, so that a very minute examination of disc and retina was impossible; disc was perceptibly pale, the arteries small, and I thought the veins also were smaller than when previously examined. I decided to perform iridectomy early next morning, and obtained the consent of the patient and her husband. Meanwhile, believing the atropine to be concerned in the acute attack, I applied a calabar disc to the eye, and ordered a draught of choral and morphia.

On returning next morning—*i.e.*, about twelve hours later—with Mr. Potts, we found that every indication for operation had vanished. Patient had slept soundly and waked free from pain. Right eye considerably softer than the other in fact, as shown by the tonometer, softer than at any previous examination. Rt. T n, impression .78. Lt. T + 1, impression .63. Right sclera almost as pale as left; pupil smaller than left; no pain. It was a question whether this rapid disappearance of the

glaucomatous symptoms in the right eye was attributable to the calabar or to the narcotic draught, especially the morphia, which is said to occasionally exercise such an effect. The unchanged tension of the other eye (or rather the increase of tension since the measurement previous to using the atropine), which still remained in excess, although the eye to which calabar was used had fallen quite to the normal, convinced me that the reduction of tension was due chiefly, if not entirely, to the single application of calabar. This opinion was strengthened when, by subsequently applying calabar to the other eye, it also recovered a normal tension. The patient was ordered a calabar disc to each eye night and morning.

May 13th. The tension of both eyes is lower than at any previous examination, and the anterior ciliary vessels are now hardly noticeably enlarged. Rt. Tn., impression .83. Lt. T n, impression .74. Patient complains of pain in the eye after using the discs, but as they have effected so decided an improvement they are on no account to be discarded. Half a disc to be used each night.

The further notes of the case are not important in detail. At different times I substituted solutions of eserine and of pilocarpine for the calabar discs, but the patient gives a decided preference to the latter, as giving her less pain. She is not now, and has not been at any time since she has been under notice, entirely free from discomfort, but she is now generally able to use the eyes in moderation in reading, sewing, &c. The freedom from discomfort and pain appears to vary with, and to depend upon, her general condition. Wishing to test the influence of calabar on the tension of the eyes, I desired the patient to omit its use entirely for a day or two, and then to call on me. On her doing so, I measured the tension before and after the application of a calabar disc, with the following results.

September 17th. Half a disc had been applied to each eye forty hours previously; none since: Rt., impression .79 mm.; Lt., impression .77 mm. Half a disc was then applied to each eye. Thirty minutes later: Rt., impression .85; Lt., impression .84. Thus an unmistakable lowering of tension was obtained. As this

point is important, I will give the measurements in detail. Their close correspondence in each case proves, I think, that I did not deceive myself through chance variations :—

Before calabar—

		Sine.	Pit below chord.	Total Impression.
Rt.	...	52	28	
		<u>53</u>	<u>26</u>	79
		52	26	
Lt.	...	<u>53</u>	<u>24</u>	77
		53	24	
		51	26	

After calabar—

Rt.	...	<u>53</u>	<u>32</u>	85
			35	
			35	
Lt.	...	<u>53</u>	32	
			32	
			<u>31</u>	84

November 8th. The patient has omitted the calabar for fourteen days, as each application caused a good deal of pain. She has, however, suffered a good deal of pain since, and the eyes are frequently a little dim in the evening.

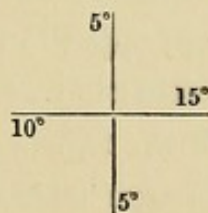
The visible conditions remain unaltered, except that the anterior ciliary veins are again decidedly enlarged ; no change in acuity of vision or in visual fields. Rt. T + 1, impression .65 mm. ; Lt. T + 1, impression .69 mm. I still hesitate to perform iridectomy, on the following grounds : the slightness of the excess of tension, the unaltered structural conditions, the extreme nervousness of the patient, and the good effects which have attended hitherto the use of calabar. She remains under frequent observation and general treatment, and will be subjected to operation should any fresh glaucomatous symptoms occur.

December 24th. No calabar used for several weeks ; to the touch, tension seems quite normal ; very slight dilatation of ciliary

veins ; slight pain occasionally ; any prolonged use of the eyes in sewing, &c., brings on the pain, but, considering the highly sensitive temperament of the patient, their condition may be considered as fairly natural. The general health has improved much the last few weeks, and the eyes have improved simultaneously with it. She is directed to be always provided with calabar discs, and to apply one at once should any sudden deterioration of sight occur in either eye.

CASE 20.

(F. 158.)—*Chronic glaucoma ; iridectomy ; permanent abolition of anterior chamber, increase of tension, pain, chemosis ; paracentesis of vitreous chamber, no improvement ; paracentesis repeated, with pressure on cornea according to Ad. Weber's method, no improvement ; persistent high tension ; total loss of sight.* Ann G——, married, aged 56, admitted into hospital February 2nd, 1878. Patient states that eight years ago she accidentally discovered that left eye was very dim ; it may have been failing before this ; it got very slowly worse, and has been quite blind now for several years. Right eye began to fail about four years ago, and has steadily deteriorated ever since. There has never been pain or redness in either eye, but she “suffered a great deal of pain shooting through the temples nearly all the time she was having her family”—seven children. The pain used to come on after breakfast, and last half the day ; “it often made her vomit when it was very bad.” Fifteen to twenty years ago she suffered frequently and intensely from toothache. Health has been good in other respects. Rt. E (with ophthalmoscope), T + 1, V = perception of movements of hand at two feet distance. Field of vision greatly contracted, thus :—



Lt. V = 0. T + 1.

In external appearance the eyes were nearly normal and quite alike. A little enlargement of the anterior ciliary vessels ; pupils a little above medium size ; both contract slightly when light falls on right eye in visual axis ; anterior chamber in each eye very shallow. Media perfectly transparent. Both discs deeply cupped, the continuity of the vessels apparently broken in many places at the margin of the excavation. The bottom of the cup in the right eye has a refraction = $M \frac{1}{11}$. The patient had been informed some years previously that her malady was *cataract*, and could be cured when the sight was nearly gone. She was now told that her informant had been mistaken, and that operation could now, at best, preserve what little sight remained, though even this was uncertain.

February 4th. Ether ; iridectomy upwards in right eye. A broad lance-shaped knife was used. It was entered 1 mm. external to the corneal margin. Owing to the extreme shallowness of the chamber, it was extremely difficult to keep the point clear of both iris and cornea. It punctured the iris, and reappeared about 2 mm. nearer to the pupil, without, as was afterwards definitely ascertained, injuring the lens capsule. A fair-sized piece of iris was removed. There was scarcely any escape of aqueous humour, and the tension was but little reduced by the operation. It was still certainly not below Tn. The iris was, at the close of the operation, in close contact with the cornea. This was evident from the way in which a minute quantity of blood escaping from the cut iris followed the furrows of the membrane, but did not pass over its ridges, which were in contact with the posterior surface of the cornea.

February 5th. A little pain frequently shooting through eye and temple since operation ; slight chemosis of ocular conjunctiva ; injection of scleral vessels.

February 6th. Pain continues ; chemosis increased, it now surrounds the whole cornea except opposite the incision ; wound appears closed, but there is no restoration of anterior chamber ; a little blood remains imprisoned in the furrows of the iris.

February 7th, (third day after iridectomy). No improvement ; chemosis increased rather than diminished. An incision made in

the most prominent part of conjunctiva; escape of blood and serum.

February 8th. Chemosis gone; scleral vessels continue much injected; T certainly not less than before iridectomy, probably rather greater; occasional shooting pains in eye and forehead, which are always speedily removed by application of hot sponges. Morphia in small doses has been given several nights since operation.

February 9th. No improvement; V = perception of daylight; T + 2; no anterior chamber. A small puncture was made in outer side of eye, about 6 mm. from the corneal margin; a few drops of yellowish fluid escaped.

February 11th. The injection of the sclera is diminished in the part of the ciliary region corresponding to the puncture made two days ago; no other change discoverable.

February 24th (twentieth day after iridectomy). No change of importance during the last fourteen days; no anterior chamber; a little chemosis; V = perception of daylight, T + 2 at least. Having a day or two previously read Ad. Weber's article on the "Cause of Glaucoma," containing an account of a method by which he had succeeded in banishing the untoward symptoms in a similar case, I determined to put it in practice here, knowing that, if left to take its own course, the eye was doomed to total blindness. The interval which had elapsed since the iridectomy indicated, according to Weber, that the present was a favourable time for the operation. After several instillations of atropine the patient was rendered half unconscious with ether; much sickness and distress had followed a full ether narcosis at the first operation, and the patient strongly objected to its employment, but was unfit to undergo another operation without it. With a broad cutting needle a puncture was made through the sclera as far below the cornea as practicable; a little clear colourless vitreous escaped; a considerable pressure was made upon the apex of the cornea by means of a finger on the upper lid, and maintained for half a minute at least. On examining the eye immediately afterwards I was disappointed

to find no appearance of a restored anterior chamber. Eserine drops were immediately applied, and continued many days subsequently. No marked change of any kind was observed as the result of this operation. ⁽¹⁾

March 6th, (thirtieth day after iridectomy). Less injection of sclera; media turbid, a dull red reflex from fundus is just discernible; $T + 2$, perhaps a little lower than a week ago; $V =$ faint perception of lamp placed in visual axis.

March 9th. Discharged from hospital.

April 26th. Right wound well closed, with little visible cicatricial substance; no anterior chamber, or possibly a *very thin* layer of aqueous humour between iris and cornea; no pain; media much clearer, but still rather cloudy; $V = 0$. Rt. $T + 2$ or $+ 3$, impression .39 mm. Lt. $T + 2$ or $+ 1$, impression .52 mm.

[My tonometer was not completed until some weeks after this patient underwent operation, and no measurements were made until her final visit on April 26th. At her first visit the tension was recorded as $T + 1$ in each; if either eye were the harder it would probably be the left, which was in a state of absolute glaucoma. If this were so, then the result of the treatment practised on the right was an increase of its tension.]

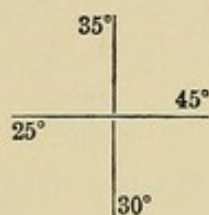
CASE 21.

(*F. 174*).—*Chronic glaucoma; not benefited by pilocarpine; arrested by iridectomy; increase of glaucomatous tension in fellow eye immediately after iridectomy, but no acute symptoms; H before, M after, operation.*—William H——d, 70, bricklayer. Was brought to the hospital by Dr. Monckton, of Rugeley, on February 21st, 1878. Patient stated that left eye began to fail ten years ago, got slowly worse, and had been totally blind about three months. It had never been painful, but some time back had been rather over-sensitive, and “ran water” on any exposure to cold and wind. The right began to fail about one year ago. In both eyes the sight had varied much at different times, being always worse in damp foggy weather. Had never suffered the

¹ See Section V., p. 172.

least pain in either eye. Health had always been excellent; had "never had an hour's illness, and never taken a dose of physic, not even a dose of salts, so far as he knew, in his life." Suffered much from toothache more than twenty-five years ago. Has smoked $\frac{1}{2}$ oz. of shag daily for many years.

Rt. Hm. = $\frac{1}{40}$, V = $\frac{20}{30}$ imperfectly, T + 2. Visual field much contracted, thus:—



Lt. V = 0. T + 2.

Pupils of medium size, and, considering age of patient, very fairly active, in response to action of light on right eye. Anterior ciliary arteries somewhat enlarged; cannot be emptied by bearable finger pressure; anterior chambers shallow; media perfectly transparent; both discs deeply cupped quite up to margin; course of the vessels apparently interrupted at the margin of the excavation; no visible arterial pulsation; in left disc a short portion of a vein, extending from centre to point where an artery crosses it, decreases in size almost to complete emptiness synchronously with each radial pulsation.

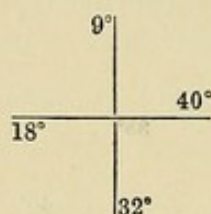
Fig. 47 represents right disc.

Considering the slow progress of the malady in the other eye, the excellent central vision still retained, and the entire dependence of patient upon this remnant of vision, I deemed it better to attempt to arrest or delay the progress of the disease by eserine or pilocarpine than to resort immediately to iridectomy. Patient went home with a supply of pilocarpine drops (two grains to one ounce) for a month. I believe that this solution was too weak; in subsequent cases I have used eight grains to one ounce, which appears to be about equal in effect to eserine one grain to one ounce.

March 26th (four weeks later). Distinct deterioration, both as regards acuity and field. Rt. Hm. = $\frac{1}{40}$, V = $\frac{20}{40}$, T + 2, im-

pression .48 mm. Lt. T + 2, impression .44 mm. Advised to undergo operation at once.

April 9th. Readmitted to hospital for operation. Rt. Hm. = $\frac{1}{40}$, V $\frac{20}{40}$, T + 2, impression .44 mm. Lt. T + 2, impression .42 mm. Field of vision (right) *before iridectomy*—



April 10th, 10 a.m. Ether; iridectomy upwards; incision nearly 2 mm. external to corneal margin, with broad lance-shaped knife; as the point entered anterior chamber aqueous escaped beneath conjunctiva and bulged it forward; operation satisfactory. 5 p.m.; No pain; iris is in contact with cornea except where a little blood lies, chiefly in the furrows of the membrane; to the touch the eye seems nearly as hard as before operation; morphia and chloral draught at night.

April 11th. Sharp pain for an hour during the night, relieved by hot sponge; no pain now; iris in contact with cornea; conjunctiva slightly cedematous all round corneal margin; fingers detect no diminution of tension; the non-operated eye feels harder than before. In view of Weber's theory that the outbreak of acute symptoms in non-operated eye after operation is due to depression of circulation through confinement to bed, patient is to be dressed and seated by the fire. ⁽¹⁾—5 p.m.: Patient has been up all day as usual; no change in operated eye since morning; non-operated eye, measured with tonometer, harder than before operation, viz.:—Lt. 36 hrs. after operation, impression .34.; 24 hrs. before operation, impression .42.

April 13th (third day after iridectomy). Yesterday the surface of iris was entirely free from blood; to-day there is a fresh effusion of very small amount between iris and cornea; as the upper lid was raised and the patient turned the eye downwards this film of blood

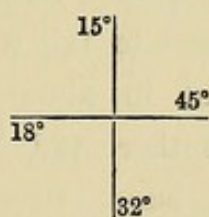
¹ See Section VI., p. 214.

suddenly passed into the pupil leaving, the iris free—no doubt, through the escape of a small quantity of aqueous humour through the incision at the moment when the contact of the upper lid was removed; iris in contact with cornea, no cedema. Left eye still harder—impression .28 mm.

April 15th (fifth day after iridectomy). Incision closed; iris not quite in contact with cornea; slight cedema of conjunctiva; media nearly as clear as before operation; no change discovered in fundus. $M = \frac{1}{20}$, $V = \frac{20}{70}$, Lt. impression .35 mm.

April 17th. Sudden sharp pain in operated eye last night, with flow of water from it—probably a partial reopening of the incision; to-day incision closed, anterior chamber re-established, though very shallow; $M = \frac{1}{20}$, $V = \frac{20}{100}$. Lt., impression .36 mm.

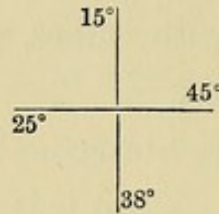
April 23rd (thirteenth day after iridectomy). Sudden sharp pain again, with flow of water from the eye and down the nostril four nights ago; ceased entirely after several hours, and has not recurred; incision closed, cicatrix greyish, very slightly prominent; anterior chamber a little deeper; several small points of synechia posterior; pilocarpine has been used twice daily since operation. Rt. E or $M = \frac{1}{50}$, $V = \frac{20}{100}$ (not tested for astigmatism). Field a little larger, thus:—Tn. impression .81 mm.



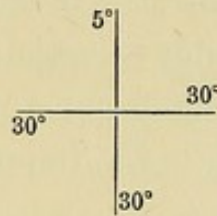
Lt., T + 3, impression .27. Patient discharged; to continue pilocarpine.

May 3rd. Incision firmly healed, not prominent; lips of incision not in immediate apposition, but separated by a greyish, semi-opaque, cicatricial substance; tension higher than when last examined, and decidedly above normal limit, but not so high as before iridectomy. Rt. E, $V = \frac{18}{70}$, T + 1, impression .51 mm.; Lt. T + 2, impression .38 mm. To discontinue pilocarpine.

May 24th. Tension lower in both eyes. Rt., impression $\cdot 63$; Lt., impression $\cdot 48$. Right field a little larger :—



August 23rd. Considerable contraction of visual field since last examination :—



Tension apparently a little higher—Rt., impression $\cdot 57$, E, V = $\frac{18}{100}$; Lt., impression $\cdot 39$. There is now considerable conjunctival hyperæmia, and frequent pain in eye and head (¹).

CASE 22.

Chronic glaucoma; absolute; large veins in iris; deep anterior chamber, &c.—John C—y, 79. Left began to fail seven years ago; it became quite blind in a few months; no pain then or since. Right began to fail a month or two later than left, and became quite blind in about twelve months; no pain then or since. Patient's father and mother died at about 74, with good sight to the last; two brothers died about 70, with good sight. Lt. V = 0, T + 3, impression $\cdot 22$ mm. (the hardest eye I have yet measured). Cornea slightly hazy; non-sensitive; pupil rather above middle size; iris traversed by large veins, which appear to be directly continuous with some enormously dilated episcleral veins (see fig. 42); the latter can be

¹ (June, 1879.—The blind eye, which for some months past has been liable to frequent pain, accompanied with more or less pain and irritability in the operated eye, has been recently excised. The fellow eye has now recovered from all sign of irritation, and in other respects shows no deterioration since last year).

emptied by very firm pressure, and refill *from* the corneal margin *towards* the equator; lens cataractous; anterior chamber deeper than is usual at *middle* age. Right very similar to left; large veins on iris. The patient drew my attention to the fact that his nose was remarkably cold. The whole surface of the nose, exactly up to the line of its junction with the cheek on each side, feels like that of a corpse. It is not anæsthetic, and the coldness is a source of discomfort; it is always cold except when he warms it by direct heat from the fire. It has been so for some years. Hands and feet are not at all cold. General circulation good.

CASE 23.

(A. 142.)—*Primary glaucoma; absolute; preceded many years by violent pains in head and eye; rupture and collapse of one eye; scleral staphyloma of the other.*—Mrs. C—y, 59, wife of the foregoing. States that from childhood she has been subject to very bad “bilious headaches,” always on left side of head, and always accompanied with pain in left eye; the pain in the eye was so bad that she “always had to put wet cloths over it;” the headaches seemed to be brought on by any over-fatigue, worry, or excitement. About eleven years ago the sight of this eye first began to fail; at first the dimness was only occasional, and passed away entirely between times, but when menstruation ceased it became more decided and permanent, and about two years later the eye was quite blind; it still continued painful with each recurrence of the headaches; four years ago, after being red and painful for several weeks, it ruptured, with great loss of blood, and subsequently shrunk; it is still painful during the headaches. The right eye began to fail three or four years later than the left, and became blind in about twelve months; she does not remember ever to have had pain worth mentioning in the right eye or right side of head. Patient’s mother died insane; suffered from melancholy, and was in confinement some years before her death; the family history otherwise excellent—viz., father died at sixty-four, with good sight; three brothers and one sister at about sixty; six maternal uncles and aunts at from sixty to

eighty ; all with good sight and free from mental disease. Rt., absolute glaucoma ; pupil widely dilated ; cornea hazy ; lens imperfectly transparent ; large equatorial staphyloma on each side of the eye ; T + 3, impression .27 mm. Lt. collapsed and shrunken. The children of the foregoing couple, aged 25, 38, and 43, have excellent sight.

CASE 24.

(F. 230.)—*Dislocation of lens into anterior chamber ; rapid onset of glaucomatous symptoms.*—Sarah H——s, 38, admitted to hospital April 30th. ⁽¹⁾ Right eye has been dim from childhood ; yesterday it was quite as usual until, suddenly, in the afternoon, something seemed to go wrong with it ; it felt uncomfortable, as though something were in it ; woke in the night with violent pain in the eye ; began to vomit, and has continued to do so almost constantly until now. Patient much prostrated, pale, perspiring freely ; pulse quick and weak ; severe pain in *right eye* ; lens in anterior chamber, which it fills almost up to the margin ; the periphery of lens, visible as a golden circle, is about 1 mm. distant from corneo-scleral junction above, nearer still below ; pupil, as seen through lens, about medium size ; intense injection of ciliary region. T + 2 at least, impressiom .40 mm. Ether ; lens extracted entire through a peripheral incision (made by carrying point of a Graefe's cataract knife round the angle of chamber), with a cataract scoop and light pressure on cornea. Vomiting continued many hours ; partial suppuration of cornea and shrinking of eye ; final healing with T — 2.

CASE 25.

(F. 153.)—*Dislocation of lens into anterior chamber ; rapid onset of intense glaucomatous symptoms.* Miss Rosetta C——s, 22, seen at her own home, June 18th, 1877. Both eyes have had very imperfect sight for many years. With right eye she has been able to read on holding book an inch or two from face. Yesterday morning, whilst stooping over washing basin, a sudden dimness

¹ See Section V., p. 183, for pathology of these dislocations with glaucoma.

came over right eye ; discomfort all day, becoming severe pain towards evening, with much redness and watering of the eye ; severe pain continued all night, with very frequent vomiting. Rt., considerable swelling and redness of lids ; great injection of ciliary region ; pupil widely dilated ; lens in anterior chamber, its diameter rather greater than that of pupil, the "gold line" being visible all round in front of iris and a little within cornea-sclera junction ; $T + 2$; with ophthalmoscope nothing but a dull red reflex perceptible. Lt., iris tremulous ; pupil small ; lens lies unattached at bottom of vitreous chamber. Two days later the lower margin of lens receded behind margin of pupil, and the severity of the symptoms diminished. With much difficulty patient was induced to consent to operation. The following morning, as Dr. Carter was just about to administer ether for me, I found that the lens had completely disappeared. Operation postponed.

During the three months following, the same accident, accompanied on each occasion by the same glaucomatous symptoms, recurred three or four times ; the pain was less intense each time, but the patient alarmed by the prospect of losing the sight entirely, begged for operation.

November 22nd. Before the commencement of the ether administration the lens was completely in front of the iris, but on introducing speculum I found it had again completely disappeared. Patient, in ether narcosis, was placed in a partially prone position, in order, if possible, to bring lens forward again through pupil ; twice this was done in vain ; finally we placed the head completely over the edge of bed, the face towards floor, when the lens immediately entered anterior chamber. Dr. Carter held the head, with the face looking downwards at an angle of about 45° , while I, kneeling on the floor, made an incision at the lower margin of the chamber with a Graefe's cataract knife. The moment the incision was completed the lens and some vitreous humour were forcibly extruded, and fell on to my sleeve. The incision healed with a slightly bulging cicatrix, to which the iris adheres. Pupil drawn towards cicatrix ; astigmatism. V with

lens + $4\frac{1}{2} = \frac{12}{200}$ (astigmatism not corrected), Tn. impression .72 mm.
Non-operated eye, V with lens + $4\frac{1}{2} = \frac{12}{40}$.

[In this case eserine had no influence on the pupil, and was therefore powerless to retard the slipping of the lens backwards.]

CASE 26.

(B. 115.)—*Dislocation of lens into anterior chamber; no pain; no rise of tension.*—Sarah W—, 28, married. Admitted into the hospital April 21st, 1875. Patient says she has been “very short-sighted” (aphakia) ever since she can remember. This morning she noticed something quite new and strange the matter with the left eye. The husband also noticed a strange appearance about the eye. It was not red or painful. Lt.: The lens lies in anterior chamber. It is perfectly transparent; iris very tremulous; pupil active, does not allow the lens to pass backwards through it when patient is made to lie down; Tn., no pain (according to sketch made at the time, the diameter of the lens is much smaller than that of anterior chamber; its upper edge is about on a level with upper margin of pupil when the latter is under influence of calabar previous to operation; while the pupil still retained its normal size as at the time of admission, the upper part of pupil would be entirely free from contact with posterior lens surface). Rt.: tremulous iris; lens lies unattached at bottom of vitreous chamber. Calabar disc applied; two hours later, pupil well contracted; ether; incision at lower margin of cornea with a Graefe’s cataract knife; lens extruded with very light pressure; rapid healing, without the slightest pain or inflammatory reaction. Previous to operation, eye entirely free from pain, redness, or excess of tension.

CASE 27.

(F. 121.)—*Dislocation of cretified shrunken lens into anterior chamber; no excess of tension.*—A “Sister” in the medical wards of the hospital, aged about 25. Was pecked in right eye by a fowl when eighteen months old; pupil of eye always occupied by a round white substance till

eighteen months ago, when this white object suddenly appeared much larger, and there was some pain in the eye for some days: this occurrence was manifestly the dislocation of lens into anterior chamber. Rt. T n; shrunken, cretaceous lens lies excentrically in anterior chamber, its upper margin being about on a level with upper margin of the moderate-sized pupil, but not in contact with it, for the clear opening of the pupil is seen on looking downwards at the eye from above.

December 4th, 1877. Ether; incision with Graefe's cataract knife at lower margin of chamber; lens withdrawn with sharp hook; portion of iris excised, having been lacerated during escape of lens. Incision healed perfectly. T n. On anterior surface of lens was found a circular raised "boss," evidently the portion which, previous to its displacement, had occupied the pupil.

CASE 28.

(F. 220.)—*Secondary glaucoma; circular posterior synechia and bulging forward of iris; second attack of inflammation with acute glaucoma; tension immediately and permanently rendered normal by iridectomy.* Maria N——, aged 40, married. Came to the hospital April 16th, 1878, complaining of violent pain and impairment of sight in right eye. She had severe inflammation in this eye twelve months before, for which she had no regular treatment. Vision had been very imperfect ever since. The present attack had begun fourteen days ago. Rt. V = fingers at 12 inches, T + 2, impression .42 mm. Intense injection of the vessels of the ciliary region; iris dull, discoloured by exudation on its surface, much pushed forward, so as to be everywhere nearly, if not quite, in contact with cornea, except at the centre, where a pit is formed by the complete adhesion of pupillary margin to lens capsule; pupillary area very small, clouded by exudation. Lt. healthy, T n., impression .72 mm. Patient was urged to undergo operation at once, but desired to defer it a few days. Ordered two leeches to temple, atropine drops, and morphia at night.

April 18th. Conditions but little changed; colour of iris

perhaps a little improved; morphia had given much relief temporarily. T + 2, impression .45 mm.

April 23rd. Admitted into hospital. Pain, injection, and high tension continue. T + 2, impression .50 mm.

April 24th (11 a.m.) Ether; iridectomy downwards and outwards, in which position the bulging of iris appeared the most pronounced. As it would evidently have been impossible to make a good incision with a lance-shaped knife without puncturing the bulging iris and thereby rendering its proper removal less easy, a Graefe's cataract knife was used, the point of which I carried between cornea and iris without wounding latter, so as to make puncture, counter-puncture, and completed incision just external to the corneal margin. Considerable traction on the iris was necessary before it could be withdrawn; it gave way close to the pupillary margin, a small quantity of fluid escaped from behind it, a large piece was removed, leaving a good clear black gap, which almost instantly filled with blood. T immediately reduced to T — 1 or T — 2. 4.30. (5½ hours after operation): Scarcely any pain since operation, incision healed, anterior chamber re-established, bulging of iris, so far as can be seen for blood still in chamber, gone. Atropine.

April 25th (twenty-two hours after operation). T n., impression .78 mm. No pain, much less injection.

May 7th (thirteenth day after operation). T n., impression .78 mm. Injection gone, no discolouration or bulging of iris, vitreous examined through new pupil, turbid; disc not discernible; vision as before.

CASE 29.

(F. 188.)—*Venous pulsation due to peculiar arrangement of vessels; normal tension.*—Sarah W——d, 20. Sallow complexion, much pigment in eyelids; suffers much from headache. Complains that the eyes are painful after prolonged use, especially by gaslight. Rt. E. V $\frac{20}{20}$, T n., impression .77 mm.; Lt. E. V $\frac{20}{20}$, T n., impression .77 mm. Optic disc normal, except that in the left, the vessels have a peculiar arrangement.

Fig. 44.—Near the lower margin of disc the artery divides into three branches, the vein into four; the former overlie the latter in every instance. ⁽¹⁾ On viewing this disc by the indirect method, it at once struck me that if venous pulsation is ever, as I believed, occasioned by the compression of the vein by an artery, here was an arrangement highly favourable to its occurrence. The blood returning through vein *a* has to pass beneath three arterial branches, that in veins *b* and *c* beneath two, and in vein *d* beneath one. On examining the disc by the direct method, with this expectation in my mind, I at once saw marked pulsation of the portion of the vein *e*, which lies between the centre of the disc and the point where it is crossed by the first artery. With each radial pulsation the diameter of this portion decreased by one third at least, and at the point of disappearance into the disc, the decrease amounted apparently to almost total emptying. In no other part of either disc did the arteries lie in front of the veins, and in no other venous trunk was any pulsation discoverable. Re-examined several times after intervals of some weeks, and when the asthenopic symptoms had nearly subsided these conditions remained unchanged.

CASE 30.

(*B. 189.*)—*Venous pulsation with normal tension due to sudden bending of vein; partial excavation of disc with staphyloma posticum.*—Thomas S——s, 21. Rt. M = $\frac{1}{8}$, V $\frac{12}{30}$, T n, (— 1 ?), impression .99 mm. Lt. M = $\frac{1}{6}$, V $\frac{12}{30}$, T n, impression .88 mm.

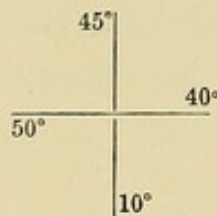
Fig. 46.—The right disc is considerably excavated in its outer half; the vessels bend suddenly backwards in passing from the non-depressed to the depressed portion of the disc, the portion *a* in the lower half of the disc pulsates strongly, the corresponding portion in the upper half very slightly. An atrophic crescent (staphyloma posticum) corresponds to the depressed portion. The excavation extends apparently quite to the margin of the disc, but has a sloping side and an evenly-rounded contour (see p. 85).

¹ See Section III., p. 84.

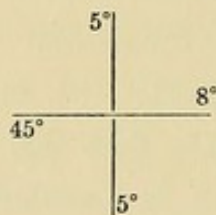
CASE 31.

(G. 25.)—*Glaucoma ; myopia ; reduction of tension by myotics.*
 —John C——r, 62, printer's compositor ; has been short-sighted for many years, if not since childhood ; seven years ago was laid up for three months with a head affection, said to be congestion of brain from over-work—great pain in head and giddiness ; during recovery discovered that sight was much dimmer than before, and believes that contraction of fields—a symptom of which he is well aware—had then begun. A similar attack three years ago laid him by for five months ; sight failed considerably during this time, and has since then steadily deteriorated. By examination of the spectacles which he has worn for work for many years, and which still suit for this particular distance, it is evident that the deterioration of sight during these attacks was not caused by any significant increase of myopia.

Rt. $M = \frac{1}{8}$, $V = \frac{18}{50}$, $T + 1$, impression .50 mm.

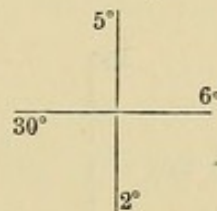


Lt. $M = \frac{1}{8}$, $V = \frac{18}{70}$, $T + 1$; impression .55 mm.



Very slight dilation of anterior ciliary veins ; pupils equal, medium sized, active ; anterior chambers not noticeably shallow, considering age of patient ; media perfectly transparent ; optic discs rather deeply excavated, quite up to the margin ; the nearer (inner) margin (fig. 49) appears somewhat undermined (vessels interrupted), but this is due probably rather to the oblique position of the disc than to a decided undermining, for the further (outer) wall appears to slope up easily to the level of the retina. Blood-vessels considerably

attenuated. It is unnecessary to record this case in detail. The patient being of very corpulent stature, with weak heart and degenerated vessels (occasional cardiac or vascular disturbances, during which sight fails for a few minutes almost completely), a resort to iridectomy was considered hazardous, and treatment by myotics (pilocarpine and eserine) was adopted in its stead with good result. For the last six months he has been using one or other of these myotics once or twice daily without any inconvenience worth notice, and has been taking iron and digitalis with much benefit to the cardiac and general conditions. He has been persuaded to relinquish his work as a compositor entirely. The tension has been measured many times with a view to ascertaining accurately the effect of myotics. Certain of these measurements have already been recorded; they indicate most unmistakably a tension-lowering effect. ⁽¹⁾ The acuity of vision was many times tested; during the first few weeks it improved somewhat, and reached on one occasion, Rt. $\frac{18}{30}$ imperfectly, Lt. $\frac{18}{50}$; it now remains about the same, or slightly better than when first examined; it varies much with the condition of the circulation, appearing to be worst when the patient is exhausted by walking or want of sleep, at which time the tension tends to *fall* rather than to rise; the fields are but little changed; the right remains almost identical with what it was when first examined; the left, always the smallest, has recently contracted considerably at the lower part, viz. :—



CASE 32.

Chronic glaucoma; iridectomy; incomplete reduction of tension; improvement by treatment with myotics.—Ann W—r, 46. Rt., gradual painless failure of sight began eighteen months

¹ See Section VI., p. 223.

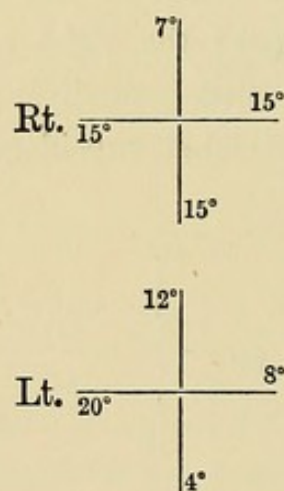
ago ; iridectomy performed elsewhere nine months ago. Lt., similar failure began about six months earlier than in Rt. ; iridectomy twelve months ago. Not much improvement noticed after the operations, but sight has not got worse since, or only very slightly so.

June 18th. Rt. large well-made iridectomy ; coloboma upwards ; disc deeply cupped. $M = \frac{1}{30}$, $V \frac{20}{200}$, $T + 1$, impression .60 mm. Lt. corneal opacity from old injury, $T + 1$, impression .60 mm. ; large coloboma upwards ($V =$ fingers). Ordered Guttæ Pilocarp. (1 gr. to 1 oz.)

June 25th.—Rt. $M = \frac{1}{30}$, $V \frac{20}{100}$, $T + 1$, impression .60 mm. ; Lt. impression .63 mm. Ordered gutt. eserine (1 gr. to 1 oz.)

August 20th.—Rt. impression .64 mm. ; Lt. impression .71 mm.

September 17th.—Rt., $M = \frac{1}{20}$, $V = \frac{20}{70}$, impression .63 mm. ; Lt., tension not measured. The patient expressed herself as quite confident that vision had improved during the treatment. The tonometrical measurements of the right eye exhibit a slight (doubtful) reduction of tension. The fields of vision exhibited no definite change during the treatment ; they were throughout—



CASE 33.

(G. 2.)—*Acute glaucoma following hæmorrhage in an eye previously sound ; acute glaucoma in fellow eye fourteen years later, &c.*—Sarah N——n, 68. Sixteen years ago sight of both eyes quite good, when one day when she was throwing up the

window sash the *left eye* went suddenly dark ; intense pain and redness of the eye came on in a few hours, and lasted several weeks ; she never saw the light again. A well-known ophthalmic surgeon attended her, and told her that there had been bleeding into the eye. This eye has now $V = 0$, $T + 2$, cupped disc, dilated pupil, so that there can be little doubt that the attack described was one of glaucoma supervening on intraocular hæmorrhage. Two years ago the right eye, which until then had been good, went dim rather quickly as she walked home from market. Severe pain came on in a few hours. Four days later she was admitted into hospital under my care.

December 5th, 1876. Rt., acute glaucoma of moderate intensity (no chemosis) ; $T + 2$, V fingers at 1 ft., examination of fundus impossible ; severe pain ; no action of bowels for six days. Ether ; iridectomy upwards ; enema. Twenty-four days later, Rt. E, $V = \frac{20}{70}$, T n. ; two months after operation, $V = \frac{20}{50}$.

May 14th, 1878 (sixteen months after operation), patient applied again, complaining of "moving circles of mist" before the eye. Rt. Hm. = $\frac{1}{30}$, $V \frac{20}{50}$, $T + 1$, impression .50. After three days' rest in bed, with good food and treatment of bronchitis, the "mists" disappeared, the objective conditions of the eye remained the same, or T was slightly lower, impression .53. Guttæ pilocarpine (8 gr. to 1 oz.) were then used for three weeks ; the general condition of patient improved ; she considered her sight better, but any positive change of acuity was not discoverable, and T appeared but doubtfully reduced—viz., impression .56.

CASE 34.

(*G. 13.*)—*Glaucoma with myopia, apparently aggravated by myotics.*—Fanny W——t, 26. Rt. $M = \frac{1}{38}$, no pain, T n., impression .73 m.m. Lt. $M = \frac{1}{14}$, frequent pain, $T + 1$, $V = \frac{20}{30}$, impression .53 mm. Sees little red balls whirling round before this eye, they rest on everything she looks at ; coloured rings round a candle ; the eye is often red. Patient declined to remain in hospital, or to undergo operation under any circumstances. Ordered guttæ pilocarpine (8 grs. to 1 oz.)

Three days later. Says that the pain has been worse ; the eye is certainly redder ; impression .47 mm. Ordered guttæ atropiæ (1 gr. to 1 oz.)

Seven days later. Rather less pain, less redness ; tension seems to be lower ; impression .59 mm.

In this case pilocarpine certainly did no good, atropine did no harm ; patient not seen again.

CASE 35.

Acute glaucoma, intensified by eserine ; iridectomy ; malignant course.—Belinda T——k, 55. Rt. began to go dim a few months (two or three) ago—a cloud in front of it ; ten days ago severe pain set in, spreading from eye over whole side of head and face.

December 17th. Intense pain ; eyelids swollen ; chemosis surrounding cornea ; T + 3, V = shadow ; *pupil very small* ; cornea clear ; aqueous humour does not appear in the least turbid ; anterior chamber not particularly shallow ; ophthalmoscopic illumination scarcely to be obtained. I came to the conclusion that pupil must be small by reason of adhesions of the iris to the lens, though none such could be made out with certainty. Patient was urged to remain in hospital and undergo operation, but preferred to return next day.

December 18th. Conditions nearly as yesterday, but rather less intense ; pain less severe, chemosis nearly gone at the upper corneal margin ; admitted into hospital ; is greatly averse to operation, having been told that her heart is so weak that she may die suddenly at any moment ; the effect of eserine to be tried for twenty-four hours before operating ; morphia at night.

December 19th. Four applications of eserine drops, 1 gr. to 1 oz. were made yesterday ; pain severe after each application. Chemosis considerably increased to-day ; $\frac{1}{60}$ gr. atropia injected subcutaneously to strengthen heart's action (by advice of Dr. Carter) ; ether administered without the least difficulty or apparent danger ; a large iridectomy upwards. By reason of the extreme chemosis it was most difficult to judge the right position for the incision. It was made with a Graefe's cataract knife, and was, I believe, just

external to the corneal margin ; free bleeding occurred during the tearing away of the iris (the iris prolapsed, proving that no posterior synechia existed) ; it was impossible to free the anterior chamber from blood, and the lids were closed without this being effected ; the tension of the eye was considerably reduced, unless, which is not impossible, my touch was deceived by the swollen state of lid and conjunctiva ; subcutaneous injection of morphia.

Same evening. T + 3, chemosis but little diminished ; a good deal of pain the last two hours ; blood in anterior chamber. During the following three days the pain disappeared almost entirely ; the chemosis subsided partially, but high tension persisted, and blood remained in chamber. V = faint perception of strong light.

December 24th. Patient, at the urgent request of her friends and herself, allowed to be removed to her own house for Christmas-day, promising to return for further treatment on the 27th (? Weber's operation—"reposition of the lens").—She did not return.

N.B.—This case is noteworthy, on account of the non-dilatation of the pupil during an intense acute glaucoma, the hurtful effect of eserine, the malignant course after iridectomy.

CASE 36.

(G. 8.)—*Secondary glaucoma following extraction of senile cataract, treated by rupturing posterior capsule.*—Mary F——e, 66. Rt., cataract extracted six years ago elsewhere ; inflammation and protracted pain followed ; eye shrunken. Patient very desirous to have the attempt made to cure the other. Lt., lens opaque ; a very thin superficial layer remains quite transparent ; V = good perception of light in all parts of retina, Tn., impression .73 mm.

June 12th. Chloral, ether ; a narrow preliminary iridectomy upwards ; rapid healing and recovery.

July 1st. Chloral, ether ; lens extracted without accident (upper incision, within corneal margin) ; no escape of vitreous, no visible remnant of lens remaining behind.

July 4th. No pain since operation, no swelling of lids ; on

opening the lids, iritis; surface of iris coated with yellow exudation, which also fills area of pupil and coloboma; leeches, atropine.

July 5th. No improvement; pain last night; T + 1; paracentesis of anterior chamber.

July 7th. No improvement; aqueous humour very turbid; pupil scarcely visible; pain increases; T + 2; paracentesis repeated; leeches.

July 9th. Intense pain last night, in spite of morphia; V = perception of lamp, T + 2 or + 3; the eye was evidently lost unless the glaucoma could be rapidly relieved. Having no distinct rationale in my mind, but knowing the salutary effect of reopening the pupil in such cases, even during the inflammatory period, I determined to operate again. Chloral, ether; broad cutting needle passed through upper margin of cornea close to former wound, and attempt made to divide posterior capsule in pupil; no satisfactory aperture obtained; second incision near lower margin of cornea; iris forceps introduced, the tough membrane (iris and adherent capsule) seized, and with considerable force withdrawn and snipped off; a clear black pupil instantaneously formed, and hidden again by copious bleeding.

During the following week the pain gradually subsided, and the tension became normal; the contents of the anterior chamber remained clouded with blood (and inflammatory exudation?) After an absence of three weeks, I next saw the patient, with a black pupil, the vascular injection nearly gone, normal tension, no pain. She now wears a $3\frac{1}{2}$ -inch glass constantly, and has $V = \frac{20}{70}$.

Fig. 43.—The portion of iris opposite the upper incision adheres to cornea; there is also anterior synechia of smaller breadth at the lower incision. (This case is specially referred to in Section V., p. 178).

CASE 37.

(F. 224.)—*Changes of tension (physiological) apparently dependent upon general bodily condition.*—John F—n, 72. double senile cataract.

April 23rd. Patient anæmic, pale, feeble; pulse weak and very compressible; skin too cool, perspiring very freely; gets insufficient food, and exhausts himself by almost constant walking about, "so nervous he can't sit or lie down." Rt. impression .95 mm.

April 27th. After three days' enforced rest in hospital, with meat and ale, condition much improved, colour in cheeks, pulse stronger; impression .86 mm.

April 29th (forty-eight hours after a narrow preliminary iridectomy). Impression .81 mm.; general condition improved.

May 3rd. Is living at home again. Impression .87 mm.

May 10th. Is evidently again suffering the effects of insufficient food. Impression .93 mm.

May 23rd. Still at home. Impression .92 mm.

May 28th. After four days rest in hospital, impression .78 mm.

[These figures are all within strictly physiological limits.]

CASE 38.

(*F. 183.*)—*Tension not changed by iridectomy in non-glaucomatous eye.*—Sarah H——s, 63. Senile cataract. Rt. impression .80 mm. Rt., forty-eight hours after narrow preliminary iridectomy, impression .80 mm.

The appended list of tonometrical measurements will serve to indicate the significance of the measurements recorded in this essay. It contains—

1. Normal tension $\left\{ \begin{array}{l} \text{in healthy eyes.} \\ \text{in diseased eyes.} \end{array} \right.$
2. Excessive tension.

The limits of physiological tension are wide. This depends partly upon the fact that the tension of the sclera does actually differ considerably in healthy eyes; partly on the fact that my tonometer is, in principle, more sensitive for variations of low than of high tension.

The correspondence which appears to exist between these measurements and Bowman's symbols is as follows:—

T n.	=	impression	·95	—	·70	mm.
T + 1 ?	=	impression	·70	—	·60	mm.
T + 1	=	impression	·60	—	·50	mm.
T + 2	=	impression	·50	—	·40	mm.
T + 3	=	impression	·40	—	·0	mm.

T n. (Healthy eyes.)

			<i>Impression.</i>
F. 216.—Ann D——s, 25.	Rt. E., V $\frac{20}{20}$...	... ·90	mm.
	Lt. E., V $\frac{20}{20}$...	... ·94	„
G. 105.—Alice L——n, 20.	Rt. E., $\frac{20}{20}$...	... ·92	„
	Lt. E., $\frac{20}{20}$...	... ·89	„
Louisa S——, 24.	Rt. E., $\frac{20}{20}$...	... ·85	„
	Lt. E., $\frac{20}{20}$...	... ·88	„
G. 50.—Sarah E——s, 20.	Rt. E., $\frac{20}{20}$...	... ·80	„
	Lt. E., $\frac{20}{20}$...	... ·79	„
Fred. P——e, 12.	Rt. E., $\frac{20}{20}$...	... ·87	„
F. 227.—Emily G——y, 25.	Rt. E., $\frac{20}{20}$...	... ·80	„
F. 92.—Eliza S——l, 30.	Rt. E., $\frac{20}{20}$...	... ·78	„
	Lt. E., $\frac{20}{20}$...	... ·78	„

				<i>Impression.</i>
F. 48.—Wm. M——r, 20.	Lt. E., $\frac{3}{2} \frac{0}{0}$	...	...	·76 mm.
F. 219.—Henry R——n, 23.	Rt. E., $\frac{2}{2} \frac{0}{0}$	...	...	·77 „
Alfred B——y, 28.	Rt. E., $\frac{2}{2} \frac{0}{0}$	...	...	·79 „
F. 188.—Sarah W——d, 20.	Rt. E., $\frac{2}{2} \frac{0}{0}$	...	...	·77 „
	Lt. E., $\frac{2}{2} \frac{0}{0}$	...	...	·77 „
Susan T——n, 62.	Lt., — ?	...	...	·70 „
F. 138.—Hannah H——s, 49.	Rt., — ?	...	...	·71 „
F. 220.—Maria N——y, 40.	Lt., ...	...	...	·72 „
G. 94.—Henry B——n, 56.	Lt., ...	...	...	·75 „
G. 6.—Charles C——s, 44.	Lt., ...	...	...	·71 „
F. 179.—Catherine E——s, 40.	Rt., ...	...	...	·77 „
Alfred H. C——, 28.	Rt., Hm. = $\frac{1}{2} \frac{1}{4}$, V = 1	...	...	·72 „
	Lt., Hm. = $\frac{1}{2} \frac{1}{4}$, V = 1	...	...	·70 „
Joseph H——n, 40.	Rt., Hm. = $\frac{1}{2} \frac{1}{4}$, V = 1	...	...	·86 „
	Lt., Hm. = $\frac{1}{2} \frac{1}{4}$, V = 1 (pain)	...	...	·78 „
G. 85.—Thos. H——l, 13.	Rt., Hm. = $\frac{1}{8}$	...	...	·86 „
Thos. L. H——l, 20.	Rt., M = $\frac{1}{10}$, V = 1	...	...	·85 „
Thos. S——n, 21.	Rt., M = $\frac{1}{8}$	...	...	·99 „
John S——w, 46.	Rt., M. = 1-3 $\frac{1}{2}$, large			
	staphyloma posticum; V = $\frac{1}{6} \frac{2}{0}$	...	...	·83 „
	Lt., M = 1-3 $\frac{1}{2}$, large staphyloma posticum,			
	V = $\frac{1}{2} \frac{2}{0} \frac{0}{0}$	...	...	·75 „
Miss C——n, 50.	Rt., M = $\frac{1}{2} \frac{1}{4}$	...	...	·67 „
	Lt., M = $\frac{1}{18}$	...	...	·70 „
G. 126.—Eliza H——s, 65.	Rt., extremely shallow			
	anterior chamber	...	...	·73 „
	(Ciliary injection of left eye was treated and			
	cured by atropine without causing any			
	increase of tension.) Lt., extremely shallow			
	anterior chamber	...	...	·75 „
G. 8.—Mary F——e, 66.	Lt., senile cataract	...	...	·73 „
Bridget Mac——d, 66.	Rt., senile cataract	...	...	·76 „
	Lt., senile cataract	...	...	·76 „
F. 183.—Sarah H——s, 63.	Rt., senile cataract	...	...	·80 „
F. 224.—John F——n, 72.	Rt., senile cataract	...	...	·85 „

Impression.

G. 116.—John L——s, 30.	Rt., extreme optic	
neuritis		·73 mm.
Lt., extreme optic neuritis		·78 „
G. 39.—Caroline R——e, 37.	Rt., extreme optic	
neuritis		·86 „
Lt., extreme optic neuritis		·83 „
F. 194.—Sarah S——t, 40.	Rt., extreme choroiditis	
disseminata		·80 „
Lt., extreme choroiditis disseminata...		·85 „
Lousia D——s, 42.	Rt., extreme choroiditis	
disseminata		·72 „
Lt., extreme choroiditis disseminata		·74 „
F. 87.—Charles S——r, 34.	Rt., advanced retinitis	
pigmentosa		·74 „
Lt., advanced retinitis pigmentosa		·74 „

T + 1 ?

F. 173.—Elizabeth P——n, 32.	Rt., premonitory glau-	
coma (afterwards acute)		·69 „
Lt., premonitory glaucoma		·69 „
Mrs. H——r, 52.	Rt., Hm. = $\frac{1}{12}$; ? incipient	
glaucoma		·67 „
Lt. Hm. = $\frac{1}{12}$; ? incipient glaucoma		·64 „
G. 25.—John C——r, 62.	Rt., chronic glaucoma	
under eserine		·66 „
Lt., chronic glaucoma under eserine		·68 „
G. 92.—Sarah H——d, 34.	Rt., syphilitic iritis,	
synechia, posterior		·61 „
E. 125.—Charles S——e, 45.	Rt., recurrent iritis,	
posterior synechia		·60 „
F. 237.—Fred. G——n, 30.	Lt. syphilitic iritis,	
posterior synechia		·68 „
F. 174.—Wm. H——d.	Rt., chronic glaucoma	
after iridectomy		·63 „

Impression.

G. 24.—Ann W——r, 46.	Rt., chronic glaucoma	
after iridectomy	·60 mm.	
Lt., chronic glaucoma after iridectomy ...	·60	„
John P——e, 45.	Rt., advanced retinitis	
pigmentosa	·60	„
Lt., advanced retinitis pigmentosa ...	·62	„

T + 1 and T + 2.

F. 174.—Wm. H——d.	Rt., chronic glaucoma,	
$V = \frac{20}{40}$	·48	„
Lt., chronic glaucoma, absolute ...	·44	„
F. 173.—Elizth. P——, 32.	Rt., acute glaucoma ...	·40 „
F. 14.—John E——n, 58.	Rt., chronic glaucoma	
after iridectomy	·53	„
Lt., chronic glaucoma after iridectomy ...	·53	„
G. 13.—Fanny W——t, 26.	Lt., myopia, glaucoma	·53 „
G. 25.—John C——r, 62.	Rt., myopia, glaucoma	·50 „
	Lt., myopia, glaucoma	·55 „
G. 156.—John W——s, 69.	Rt., cataract extraction,	
glaucoma	·52.	„
John M——s, 68.	Lt., absolute glaucoma,	
chronic	·42	„
F. 220.—Maria N——y, 40.	Rt., iritis, circ. synechia	
posterior	·42	„
G. 6.—Charles C——s, 44.	Rt., secondary glaucoma,	
wound, &c.	·44	„
F. 196.—Sarah T——n, 21.	Rt., secondary glaucoma,	
buphthalmos	·54	„
Lt., secondary glaucoma, buphthalmos ...	·59	„
F. 210.—William F——, 6.	Rt., secondary glaucoma,	
buphthalmos	·43	„
Lt., secondary glaucoma, buphthalmos ...	·43	„
F. 230.—Sarah H——s, 38.	Rt., secondary glaucoma,	
dislocated lens	·40	„

*T + 3.**Impression.*

F. 174.—Wm. H——d, 70.	Lt., absolute glaucoma,						
chronic ...						·27 mm.	
F. 156.—Ann G——e, 56.	Lt., absolute glaucoma,						
chronic ...						·39	„
G. 49. — Ann F——n, 60.	Lt., absolute glaucoma,						
acute ...						·33	„
A. 142.—Mrs. C——y, 59.	Rt., absolute glaucoma,						
chronic ...						·27	„
A. 146.—John C——y, 79.	Rt., absolute glaucoma,						
chronic ...						·29	„
	Lt., absolute glaucoma, chronic					·22	„
G. 38.—Aaron W——s, 20.	Rt., secondary glaucoma,						
absolute, total leucoma and synechia anterior	·33	„					

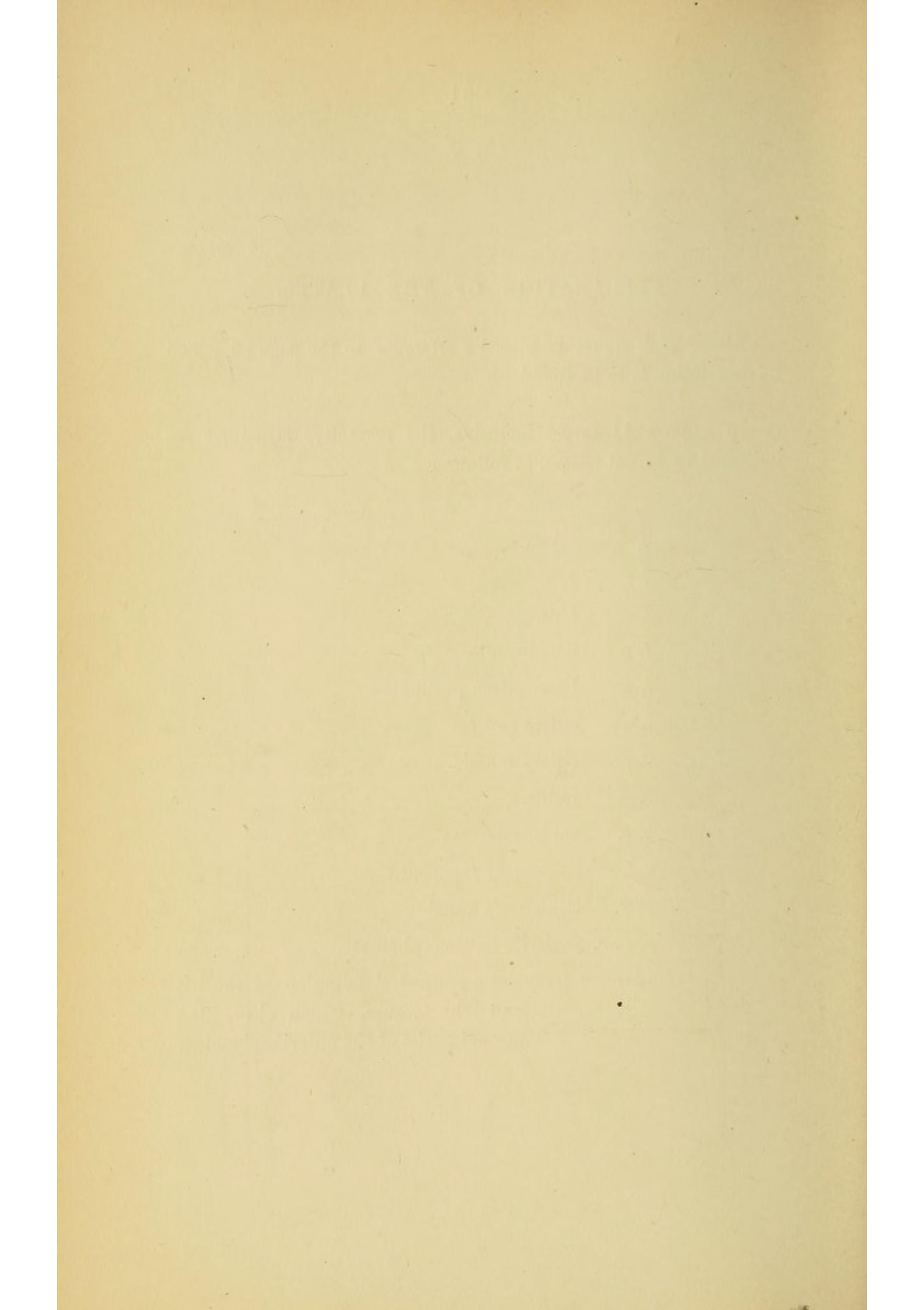
EXPLANATION OF THE PLATES.

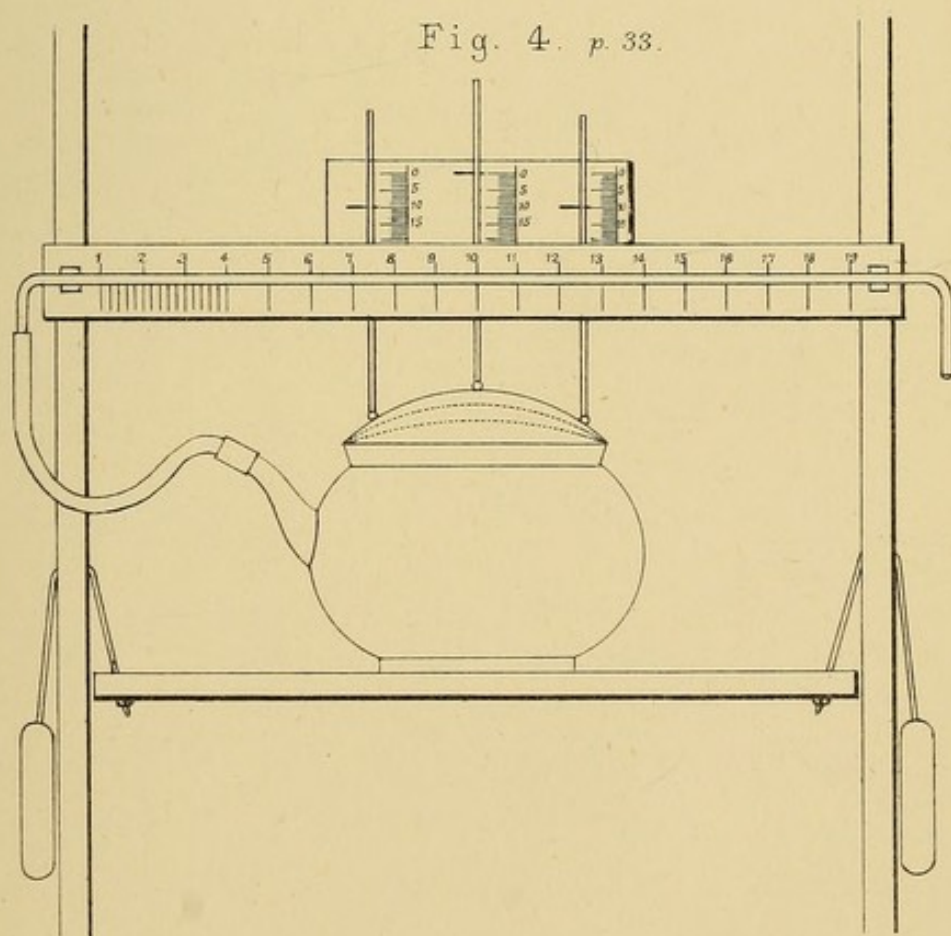
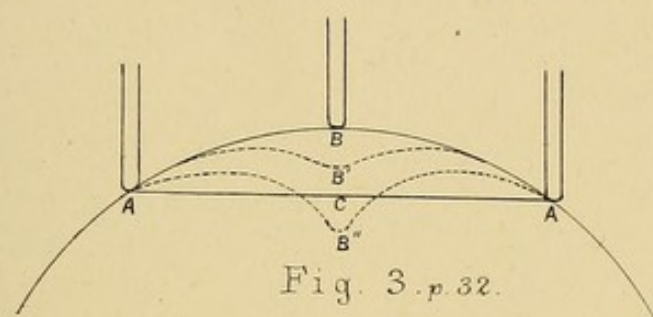
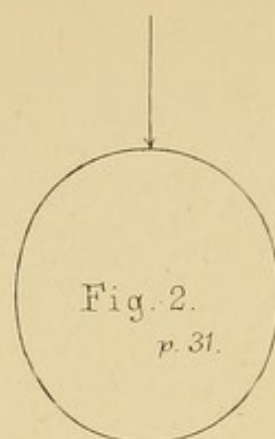
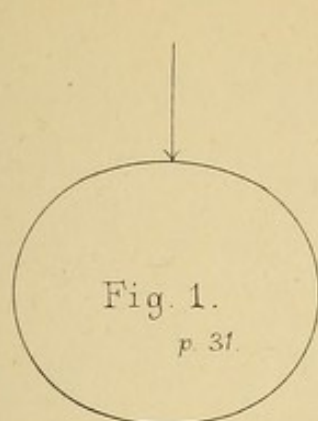
Above each figure is placed a reference to the page on which its description is to be found.

In Figures 11 to 30 inclusive, the respective structures are indicated by initial letters, as follows :—

<i>c.</i>	cornea.
<i>s.</i>	sclera.
<i>i.</i>	iris.
<i>l.</i>	lens.
<i>l. c.</i>	lens capsule.
<i>l. p.</i>	ligamentum pectinatum.
<i>c. p.</i>	ciliary process.
<i>c. m.</i>	ciliary muscle.
<i>r.</i>	retina.
<i>o. s.</i>	ora serrata.
<i>s. l.</i>	suspensory ligament.
<i>S. c.</i>	Schlemm's canal.
<i>p. a. c.</i>	posterior aqueous chamber.

adhesion indicates a peripheral adhesion of the iris
 and the cornea, which closes the
 normal outlet of the anterior chamber.





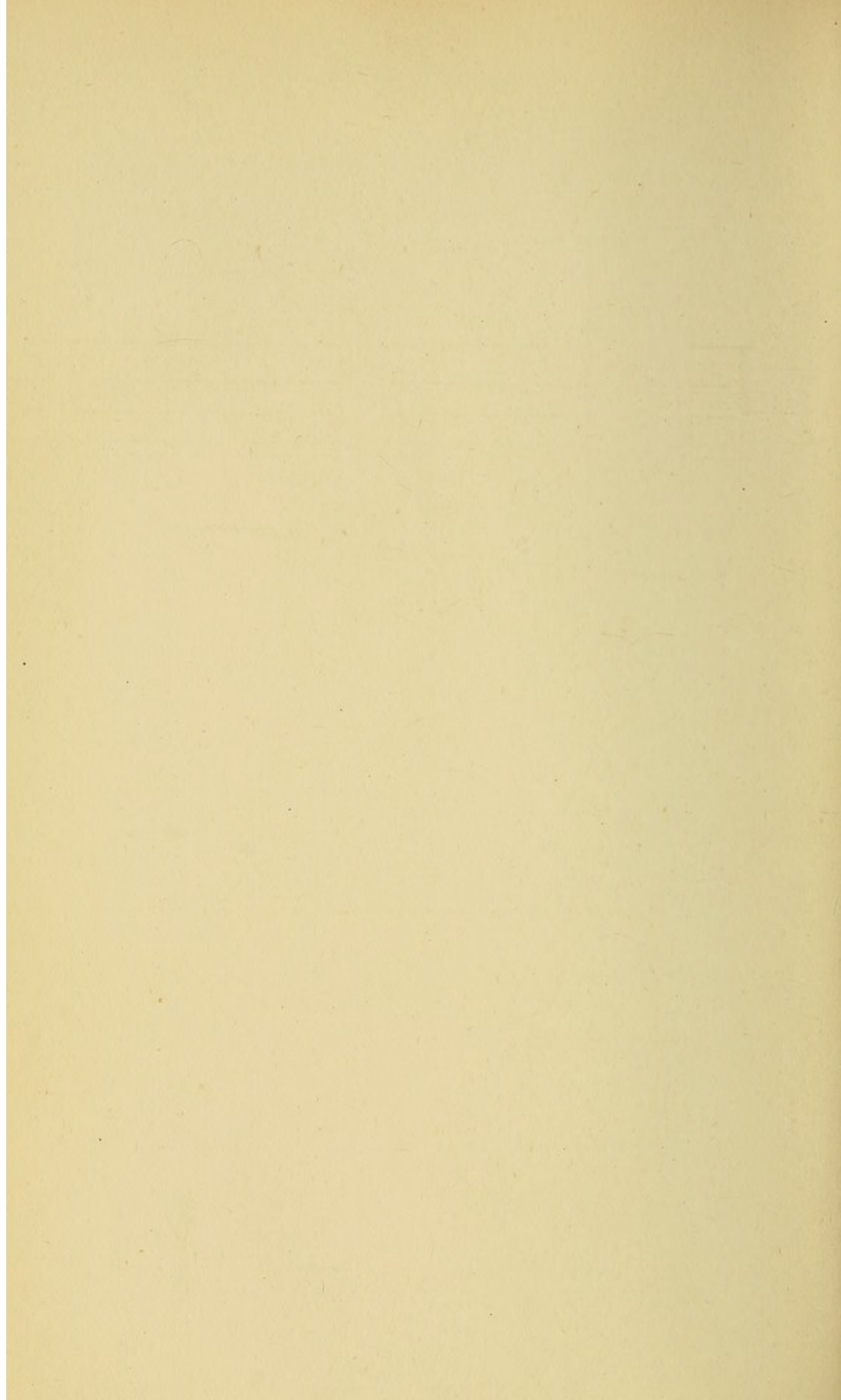


Fig. 5. p. 38.

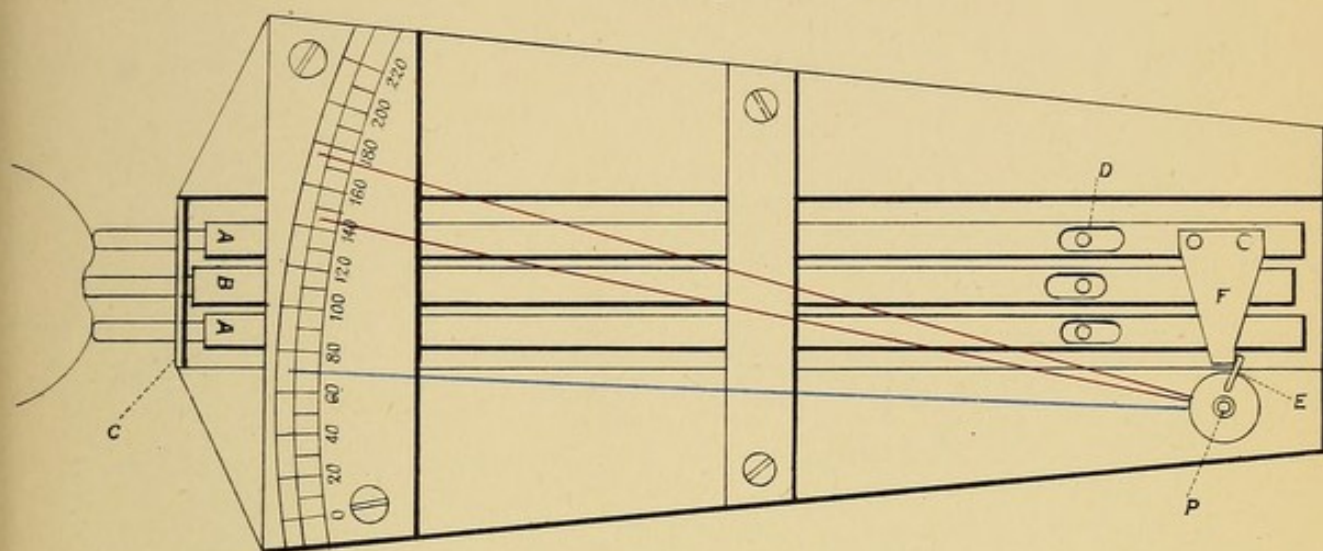


Fig. 6. p. 38.

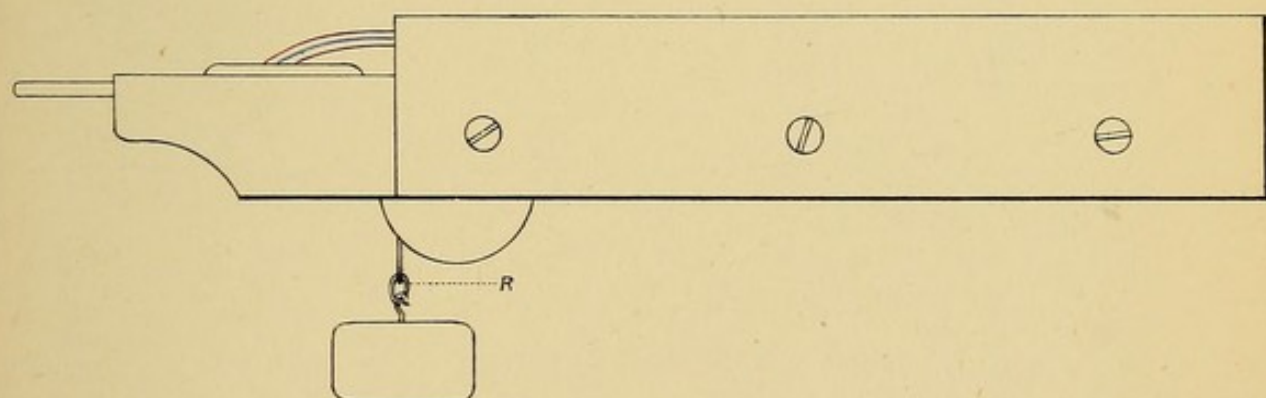


Fig. 7. p. 38.



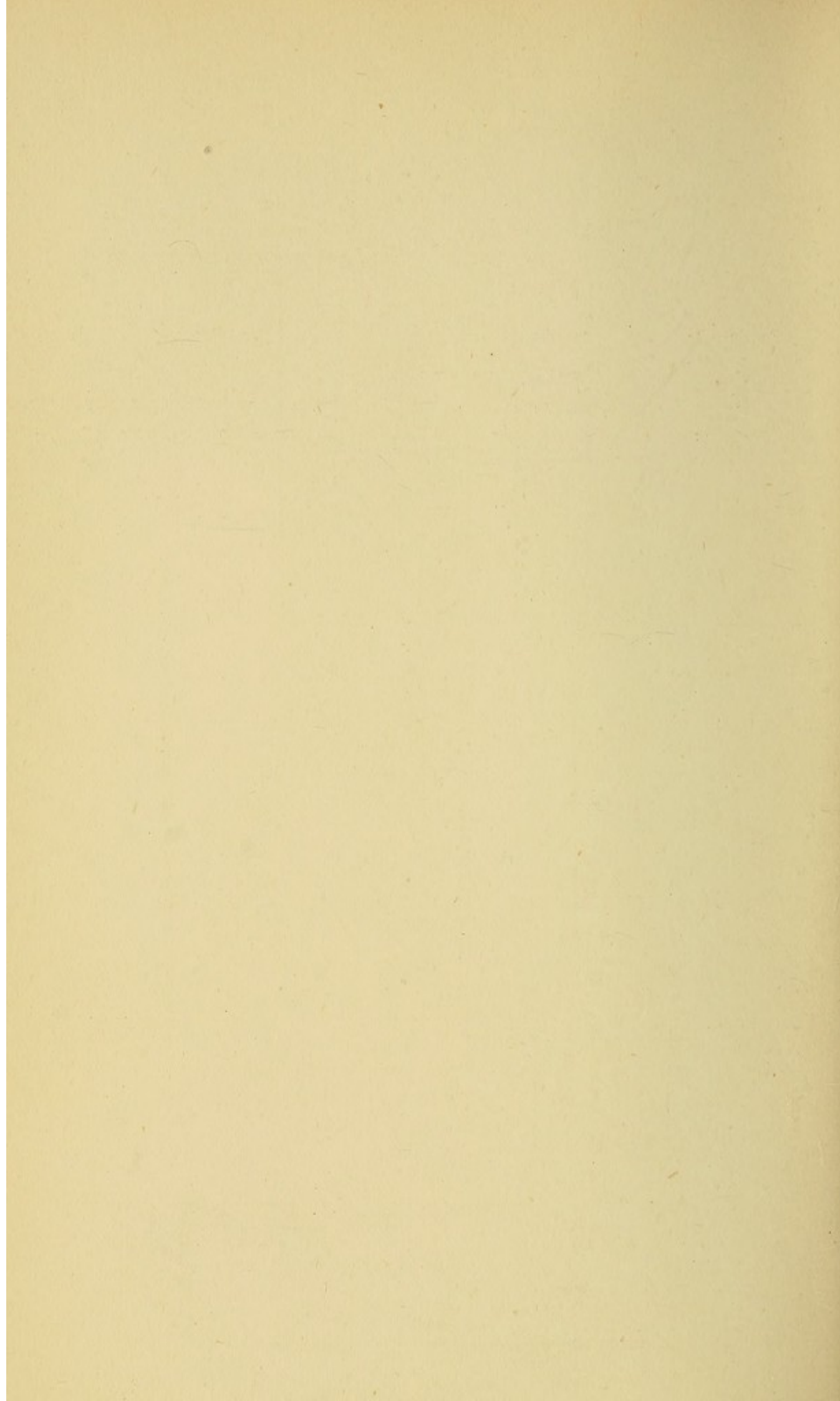


Fig. 8. p. 69.

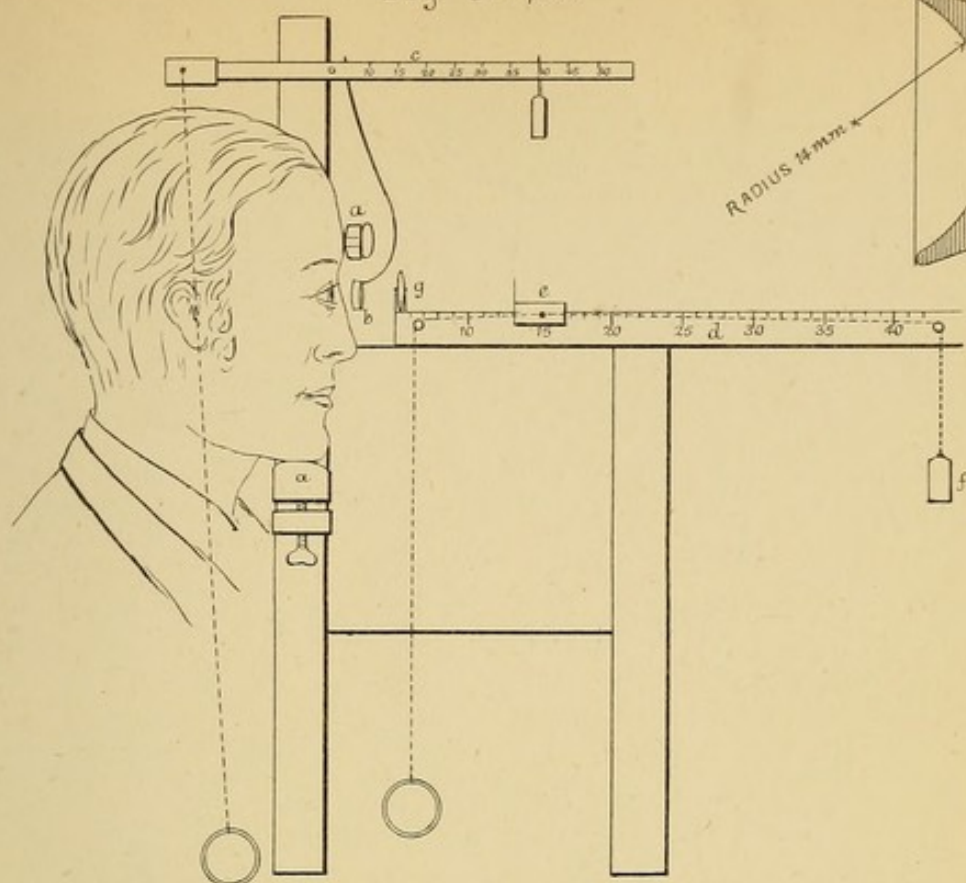


Fig. 9.
p. 69.

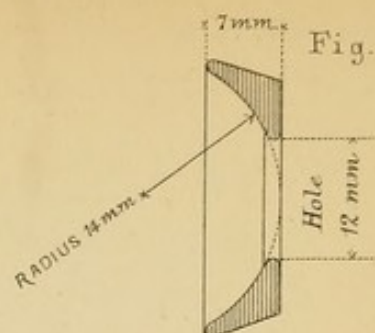
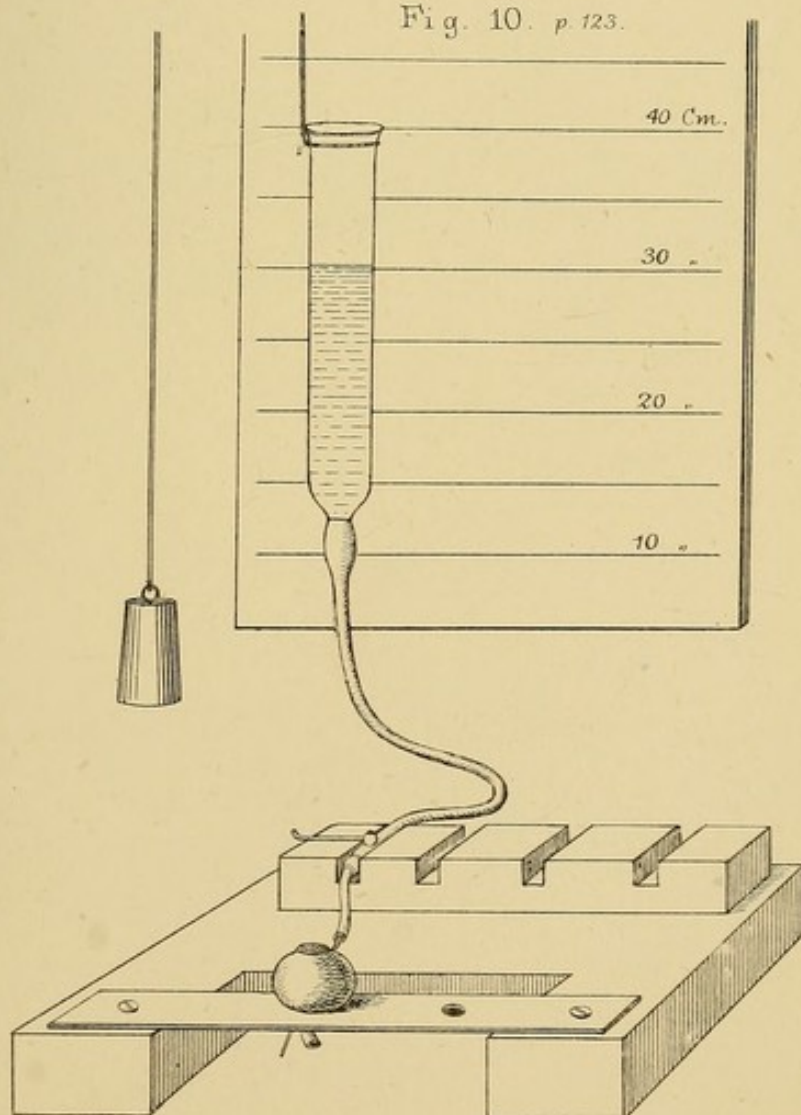


Fig. 10. p. 123.



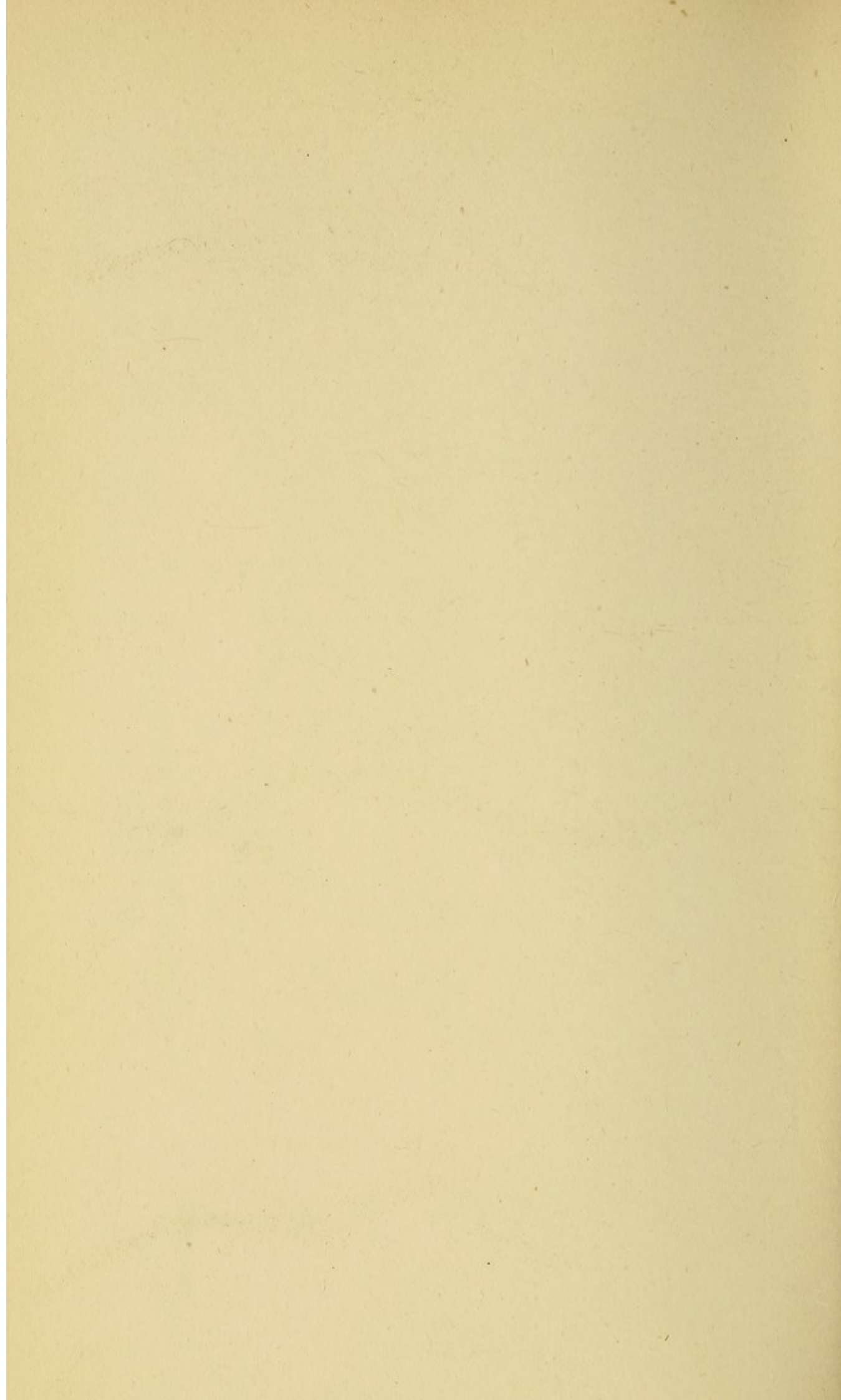


Fig. 11. p. 234

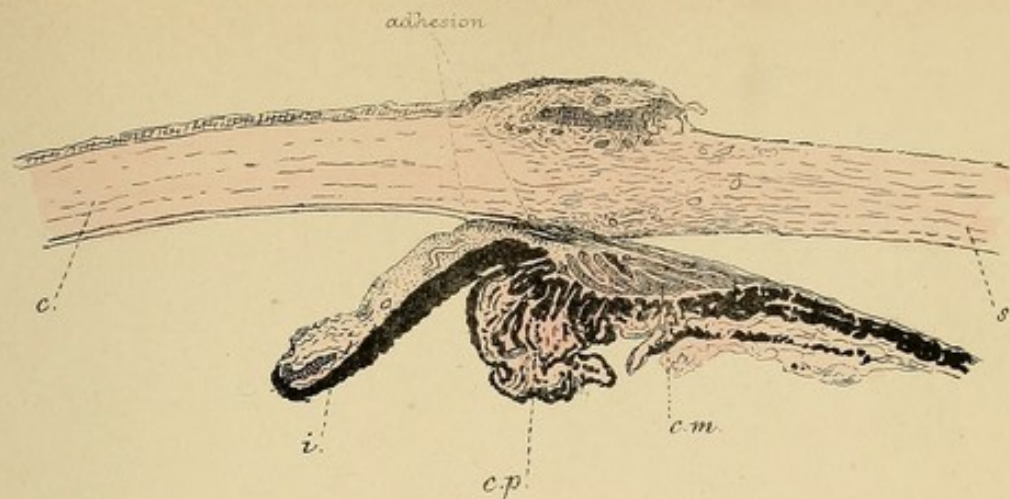


Fig. 12. p. 234

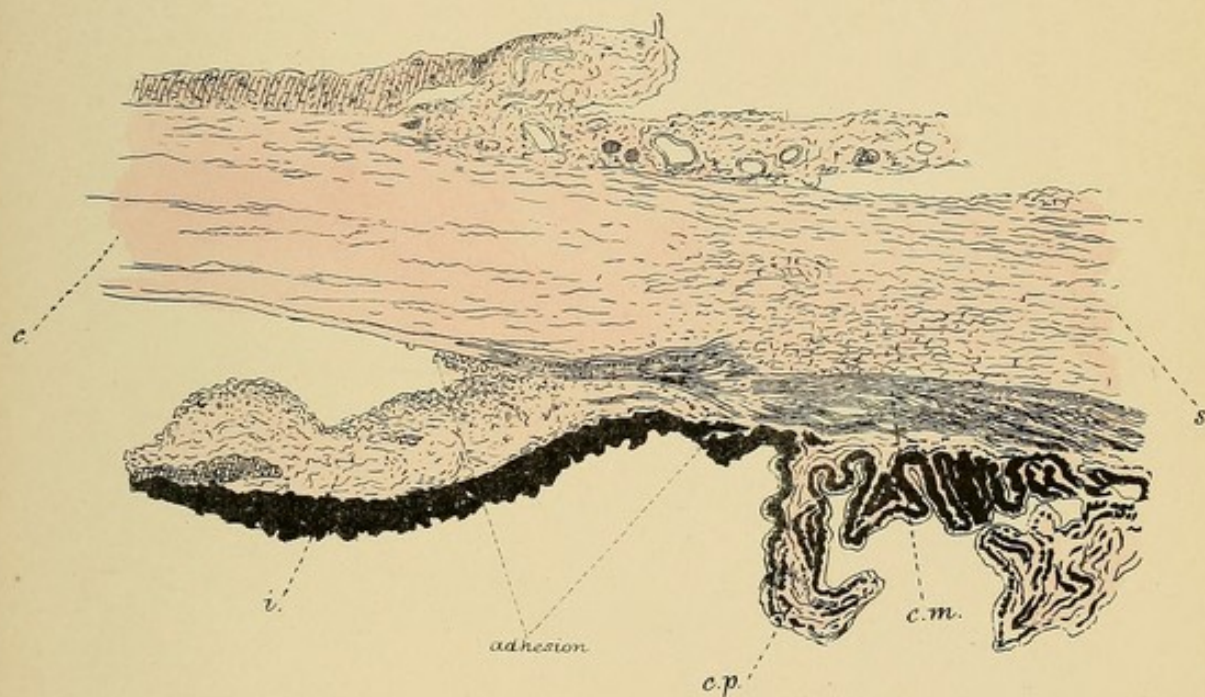
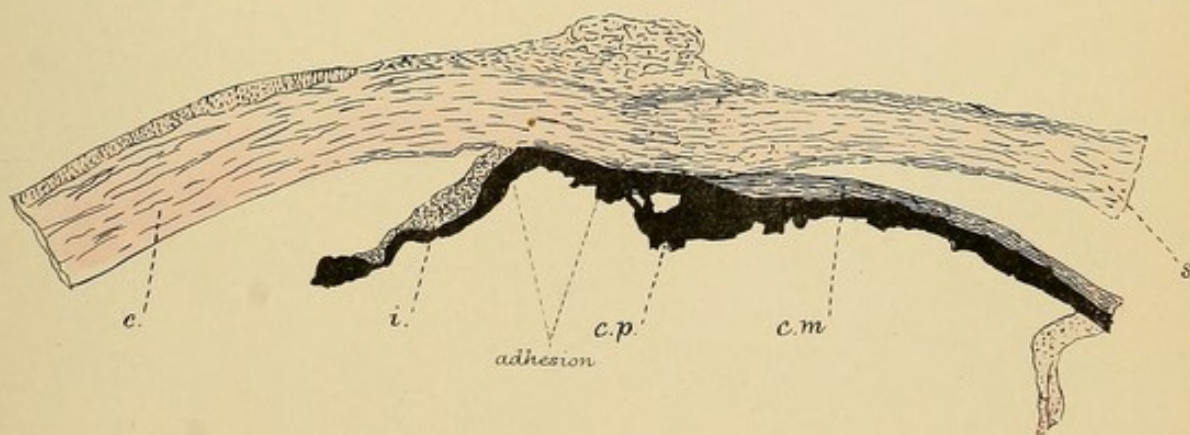


Fig. 13. p. 240



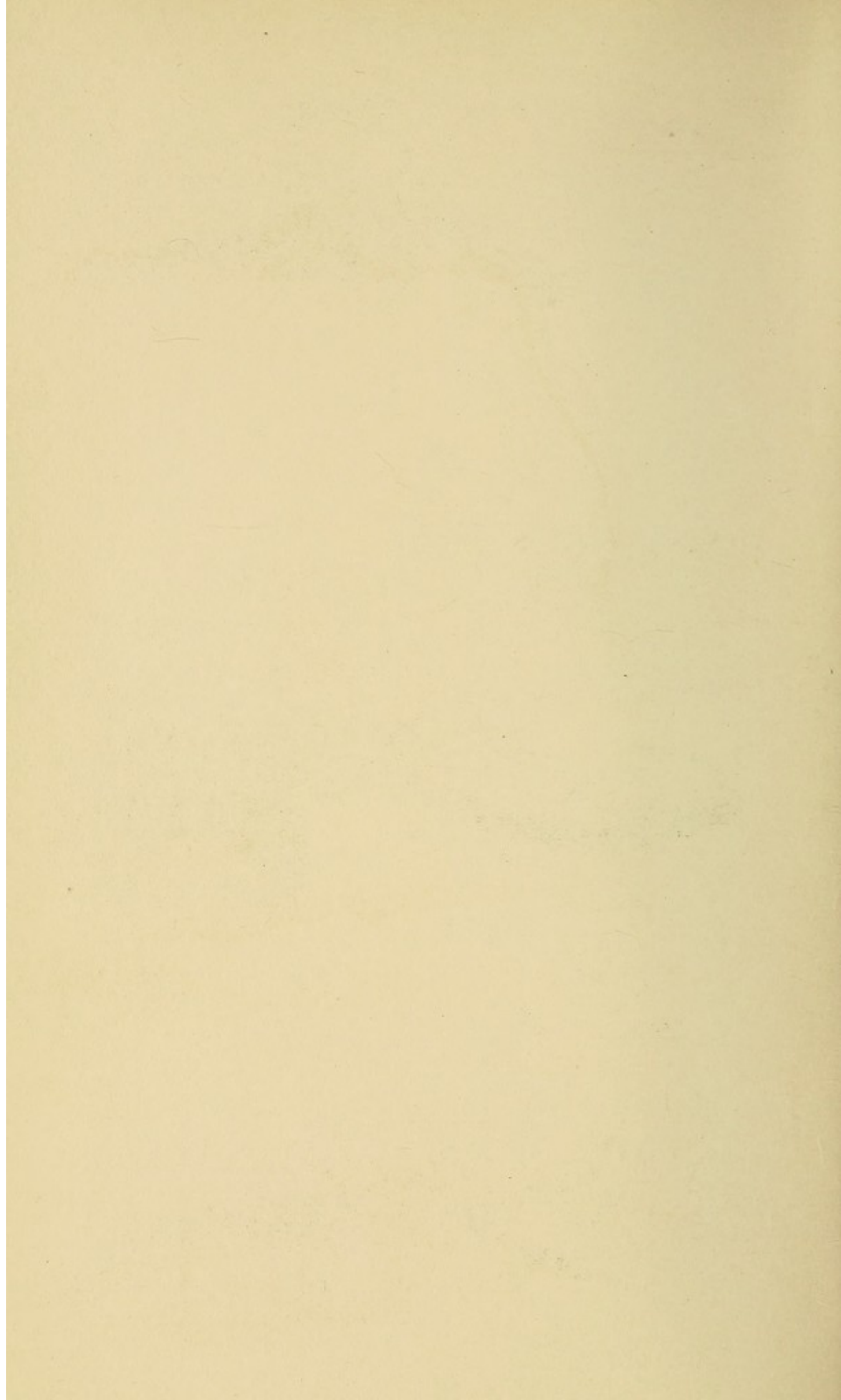


Fig. 14. p. 241.

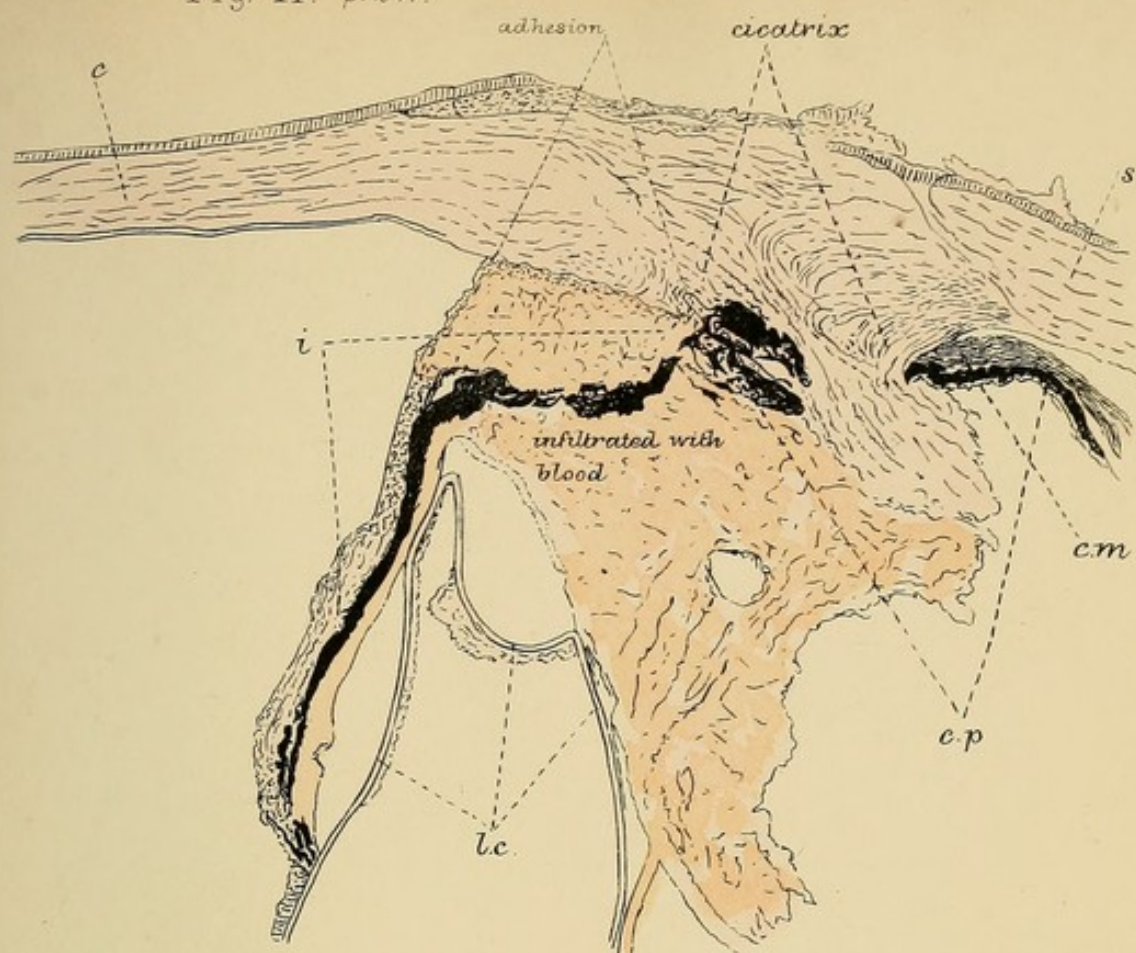


Fig. 15.
p. 235.

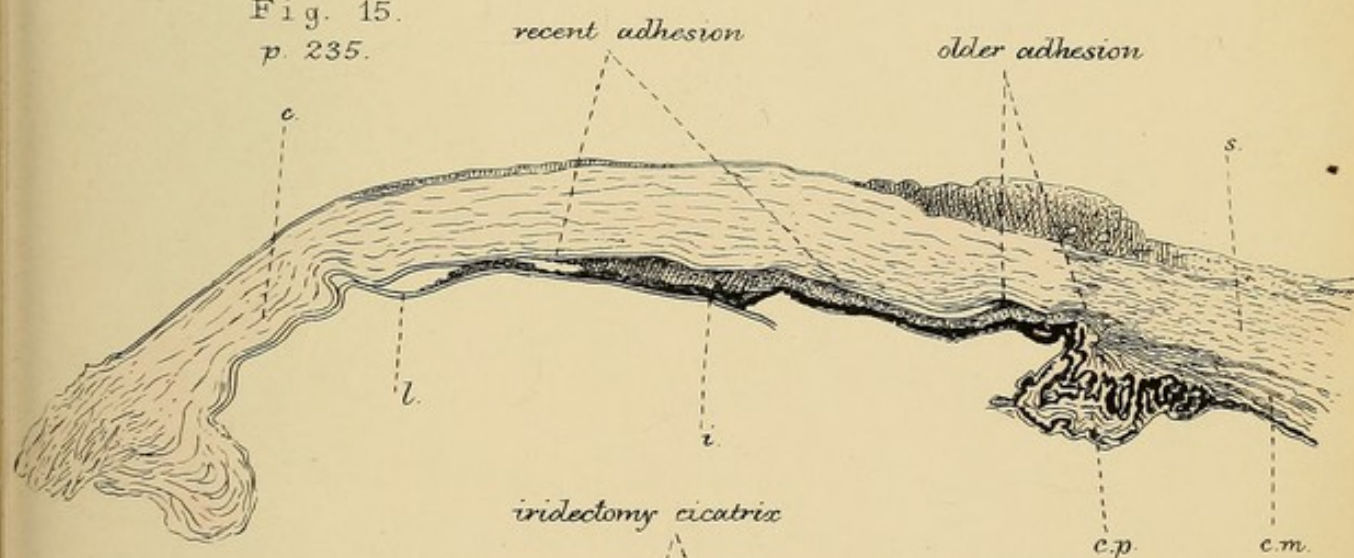
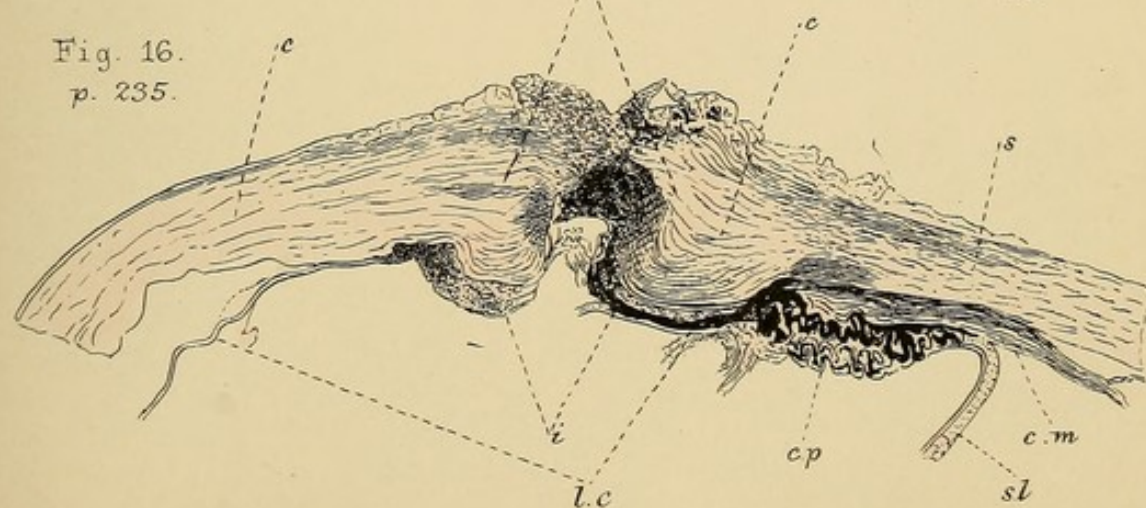


Fig. 16.
p. 235.



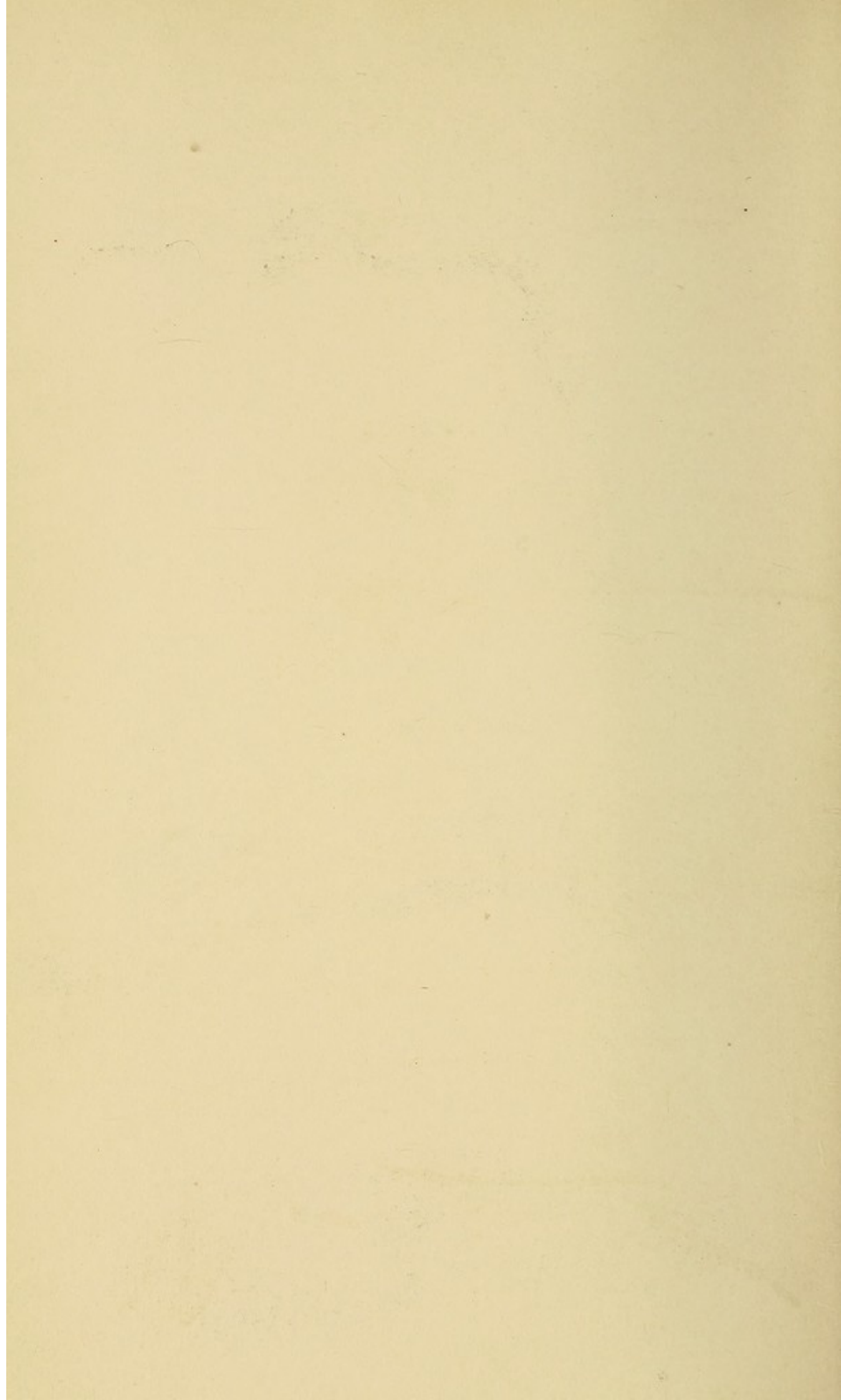


Fig. 17. p. 247.

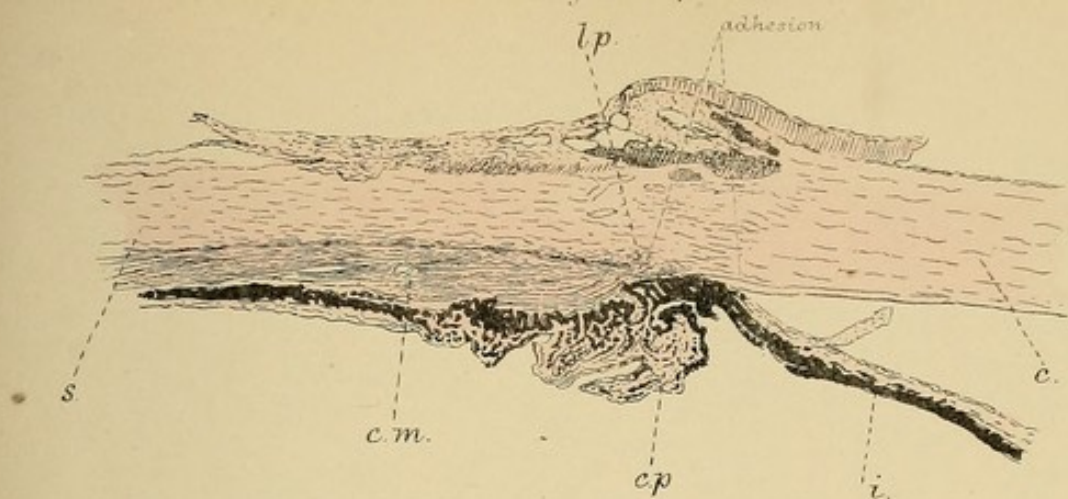


Fig. 18. p. 247.

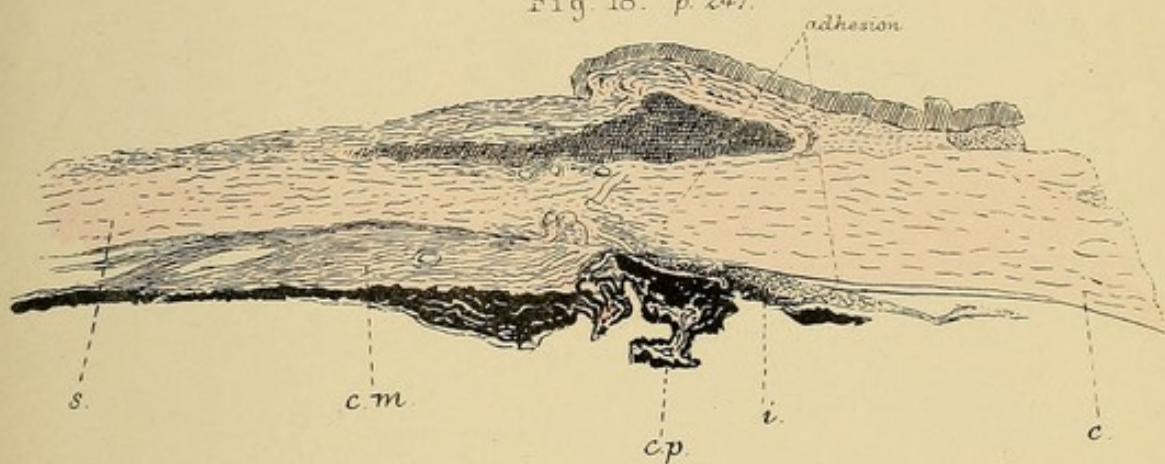


Fig. 19. p. 248.

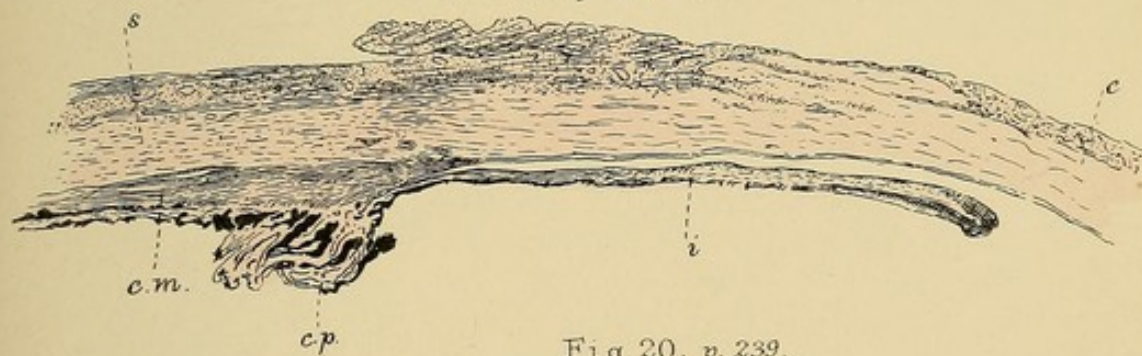
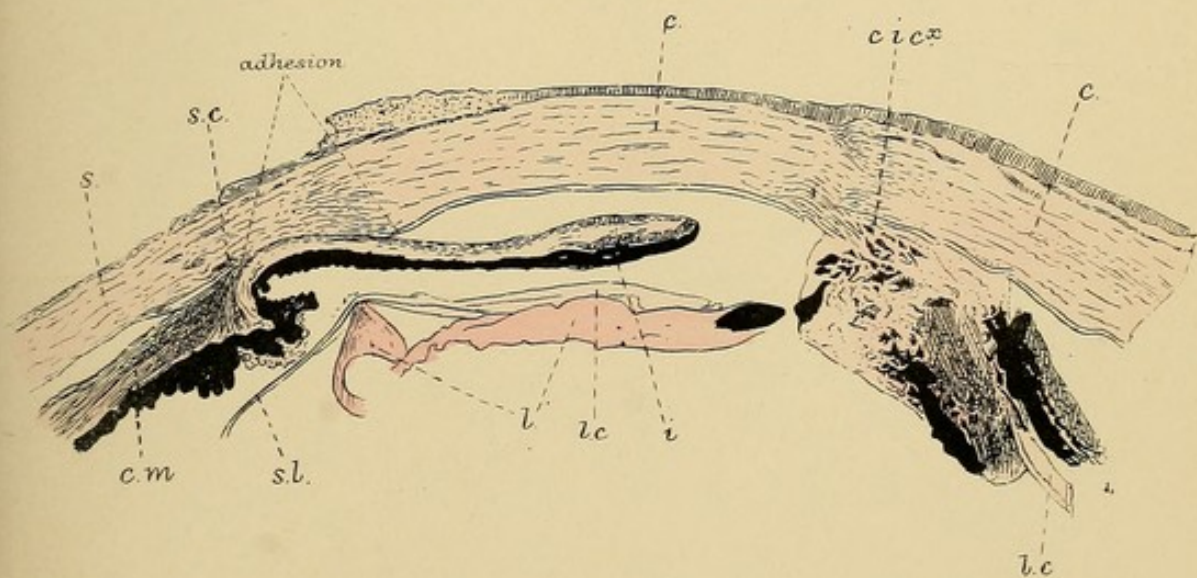


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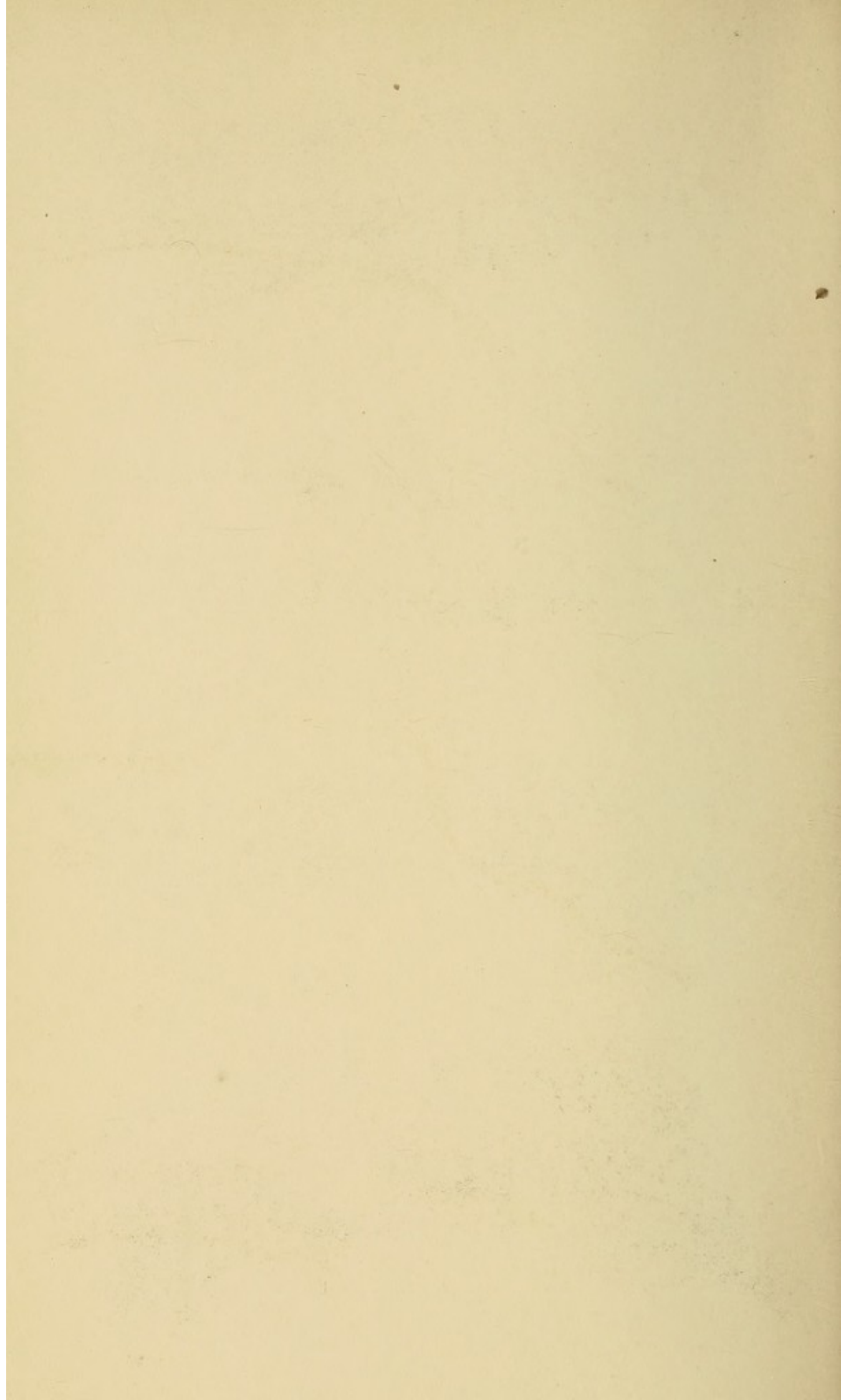


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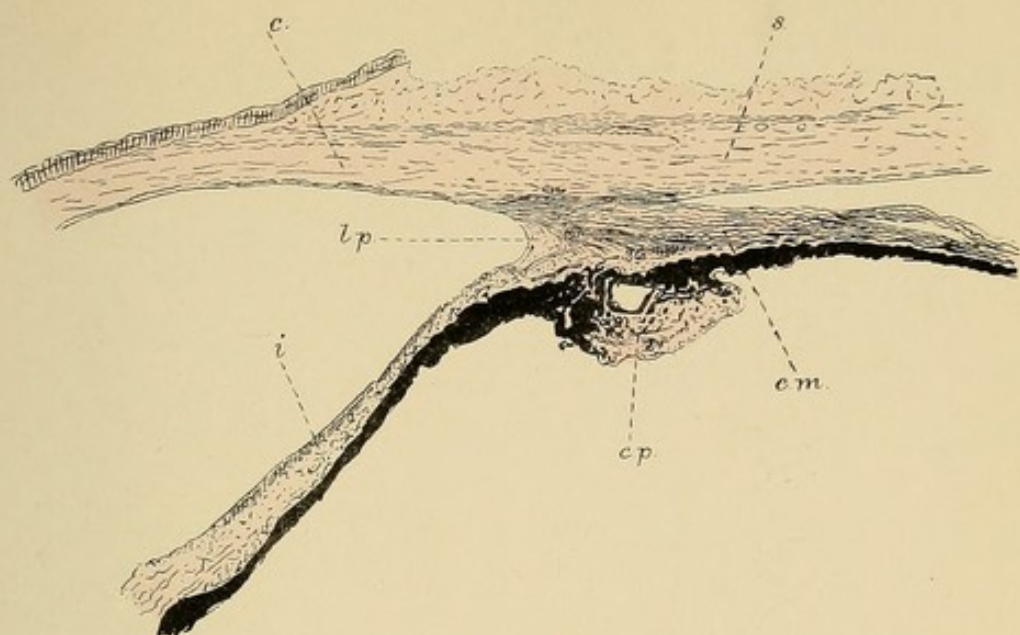


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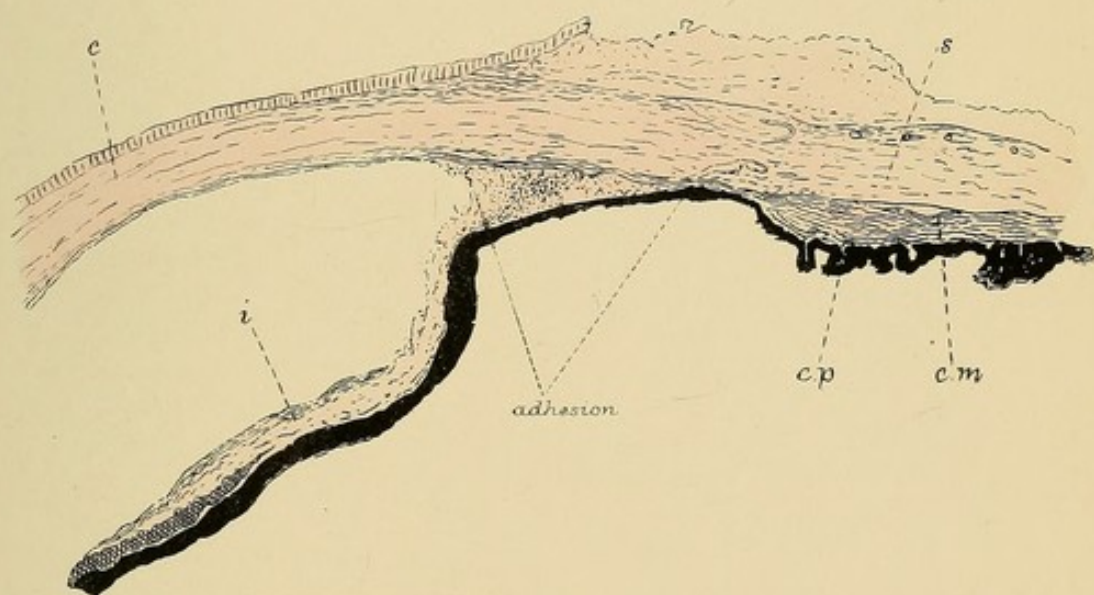
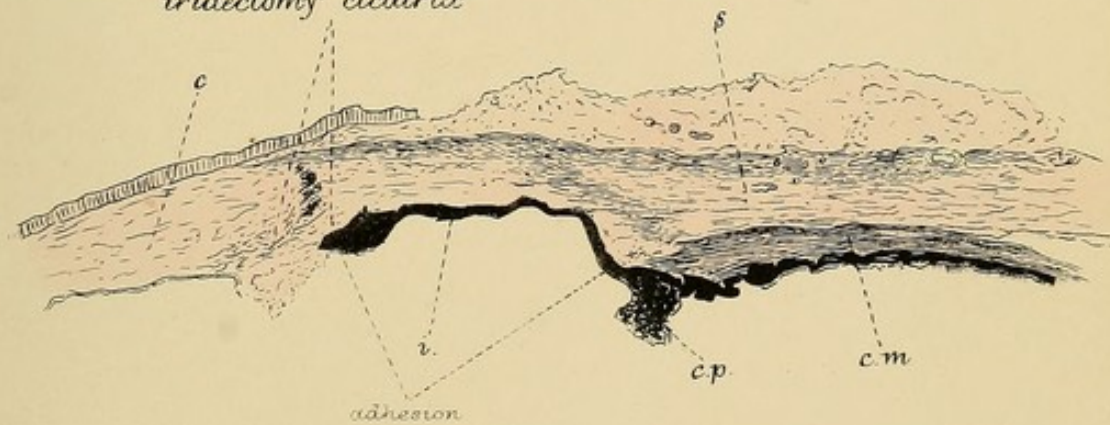


Fig. 23. p. 237.

irideclomy cicatrix



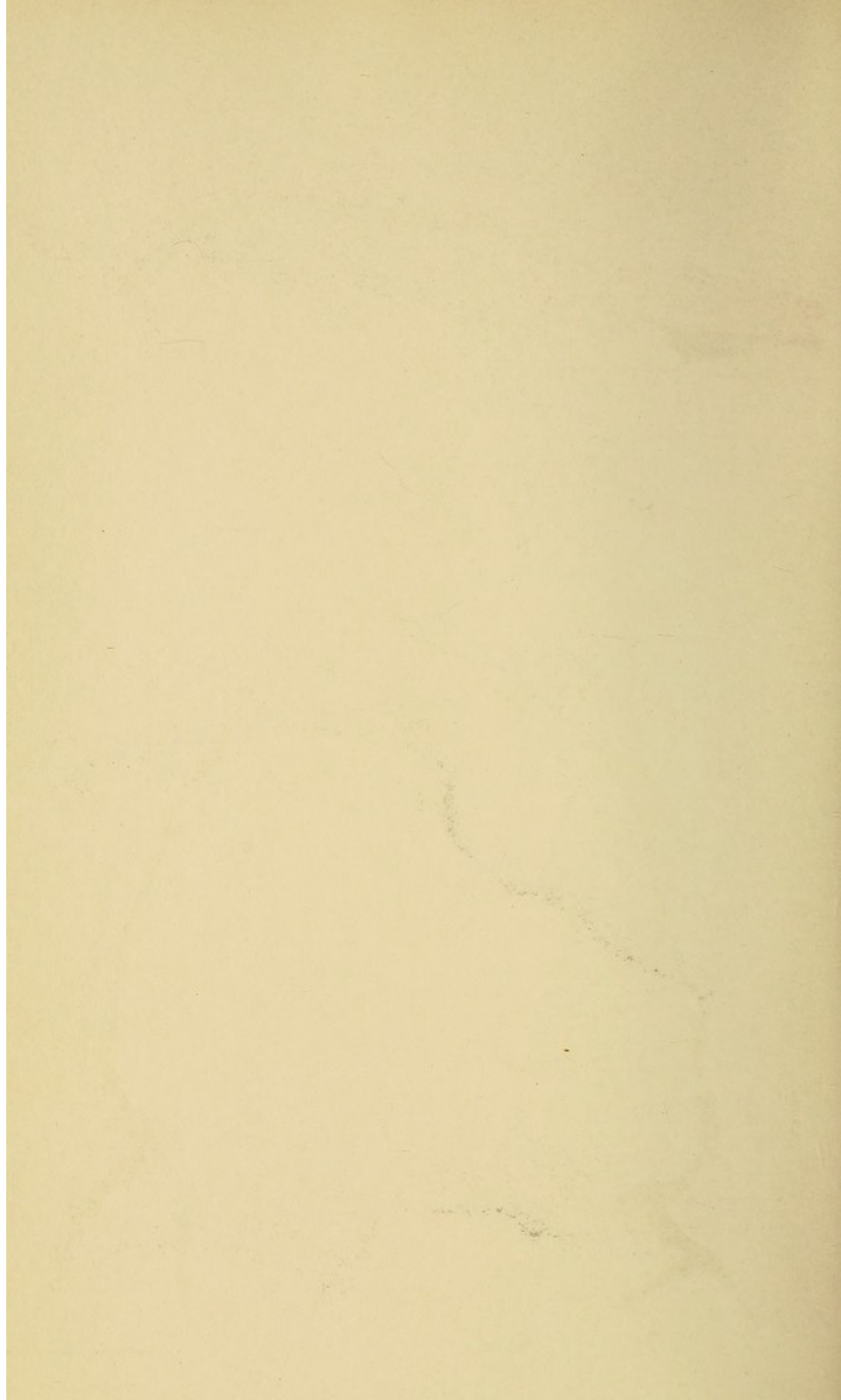


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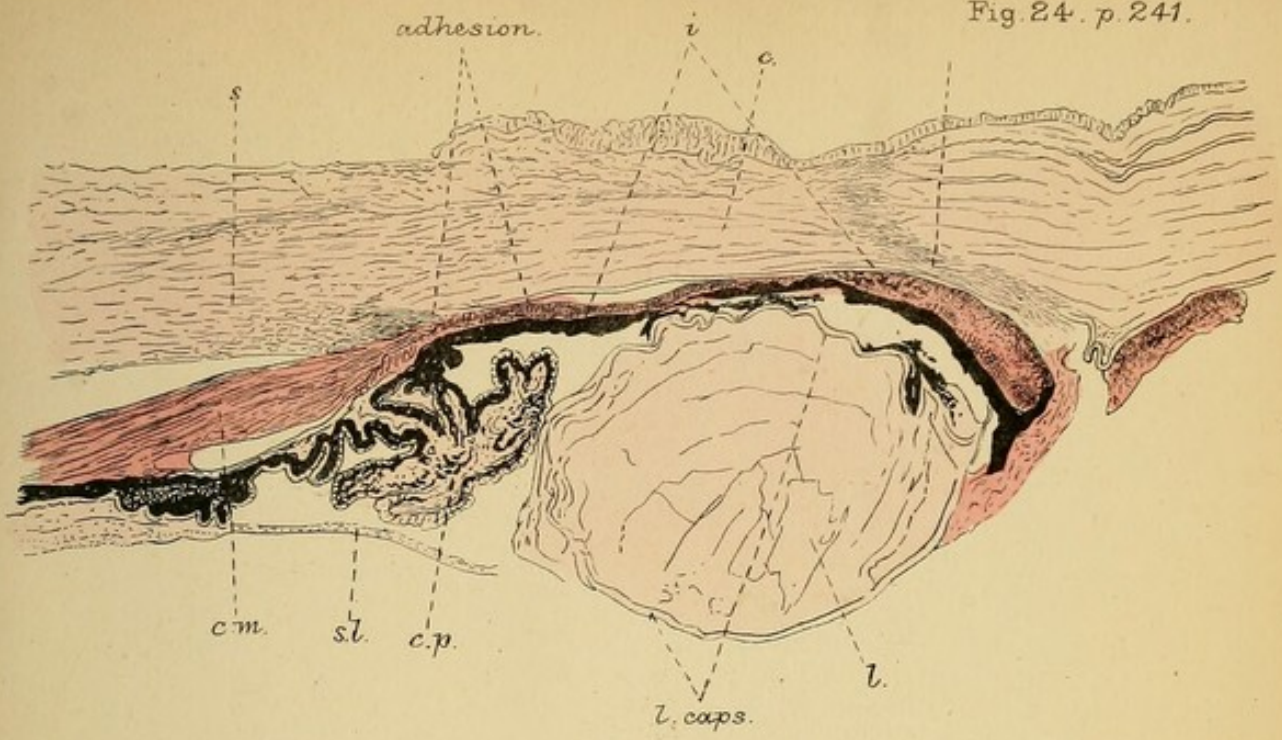
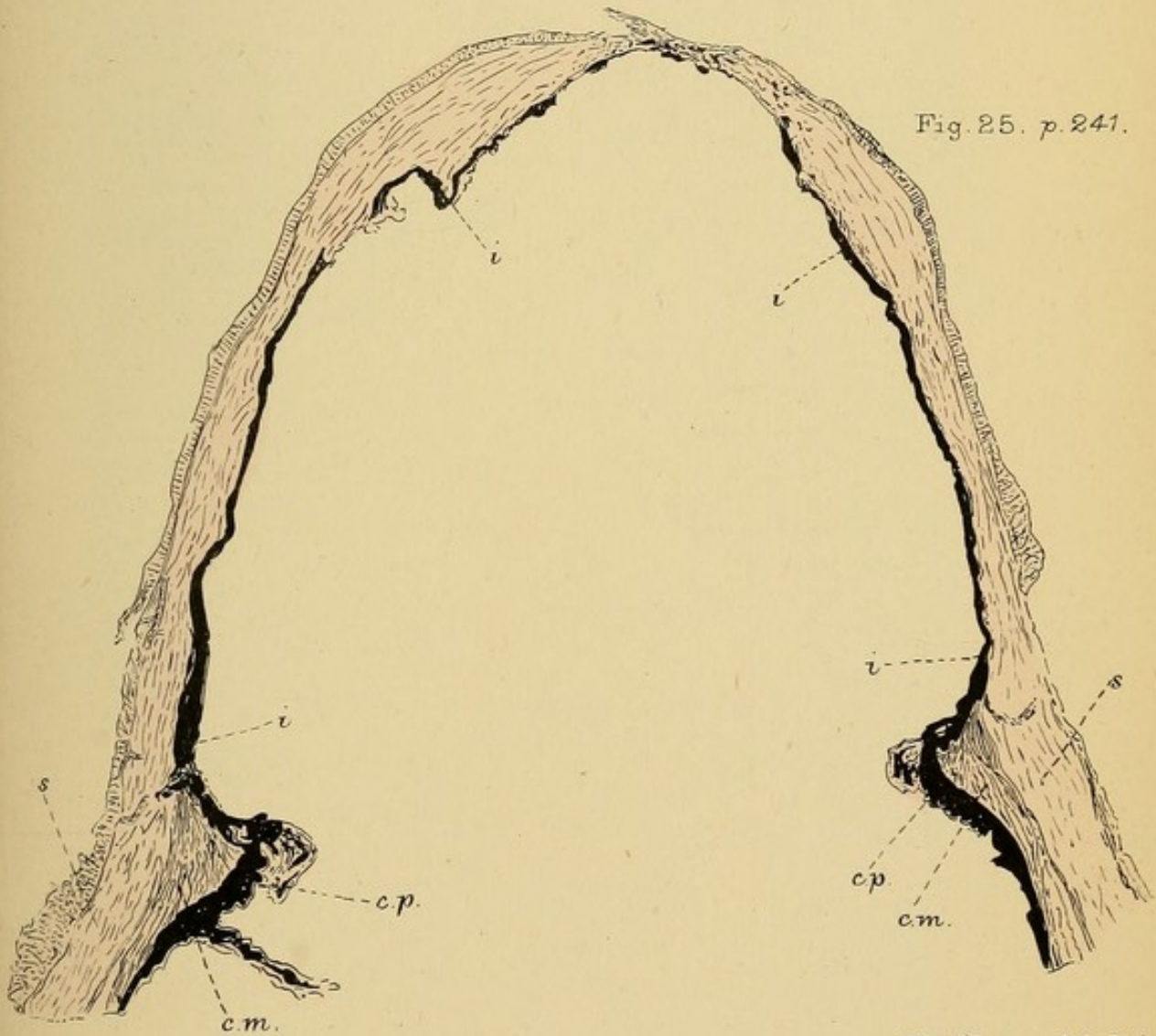


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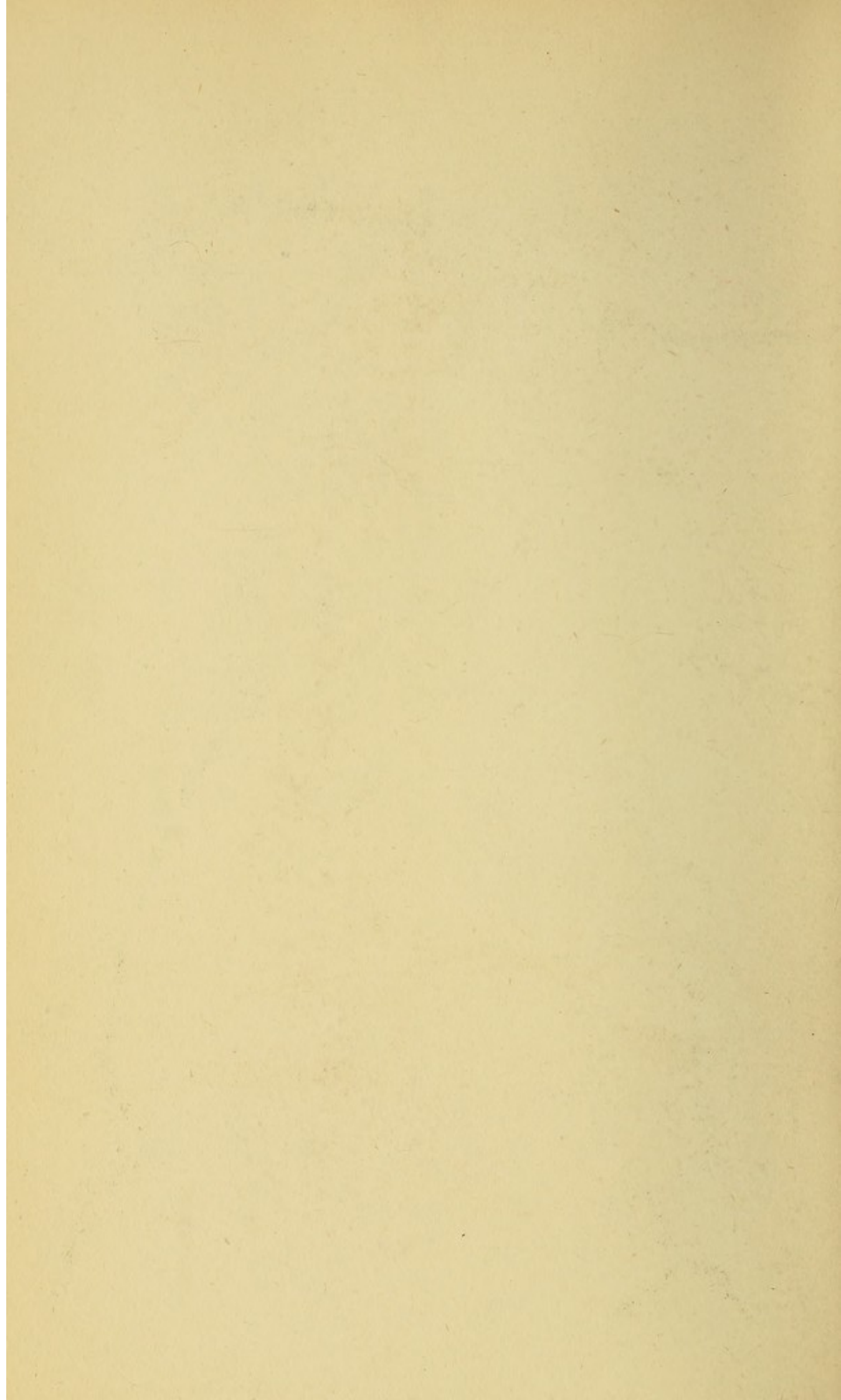


Fig. 26. p.242.

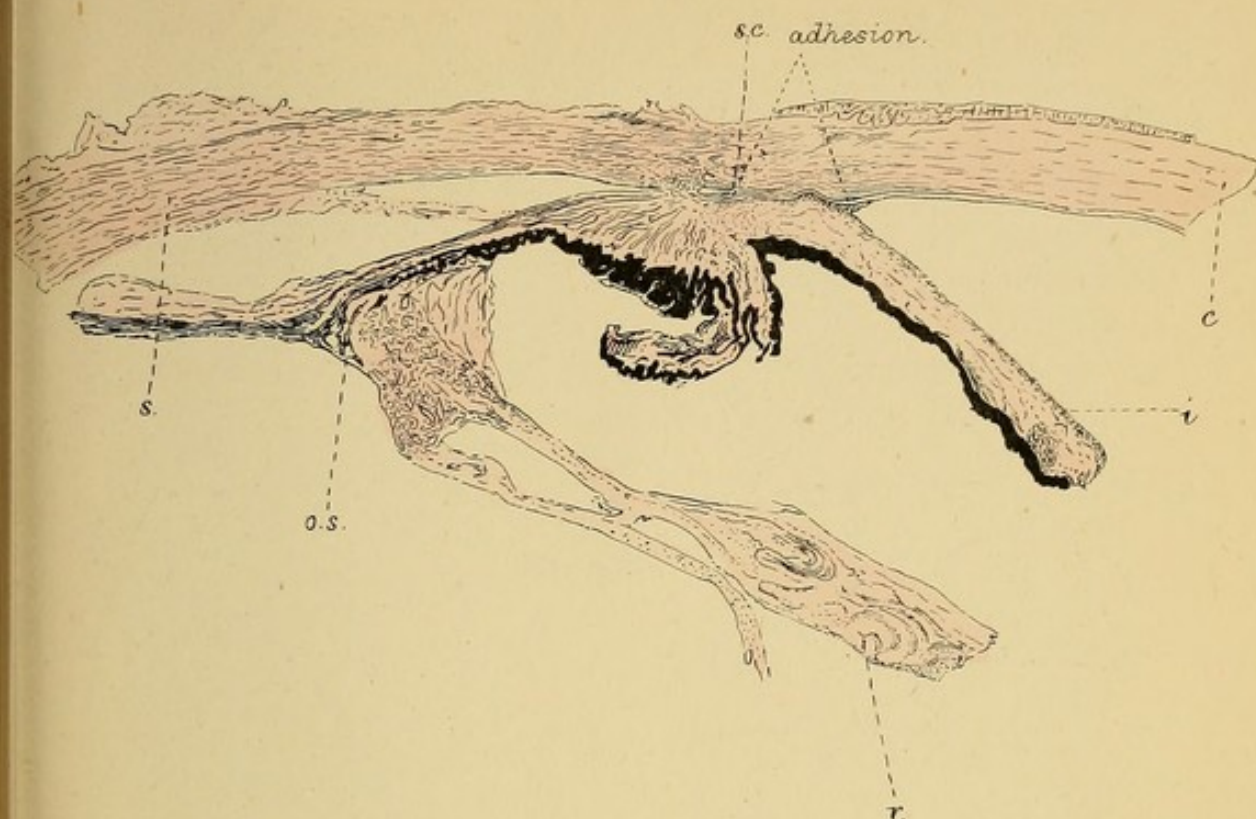
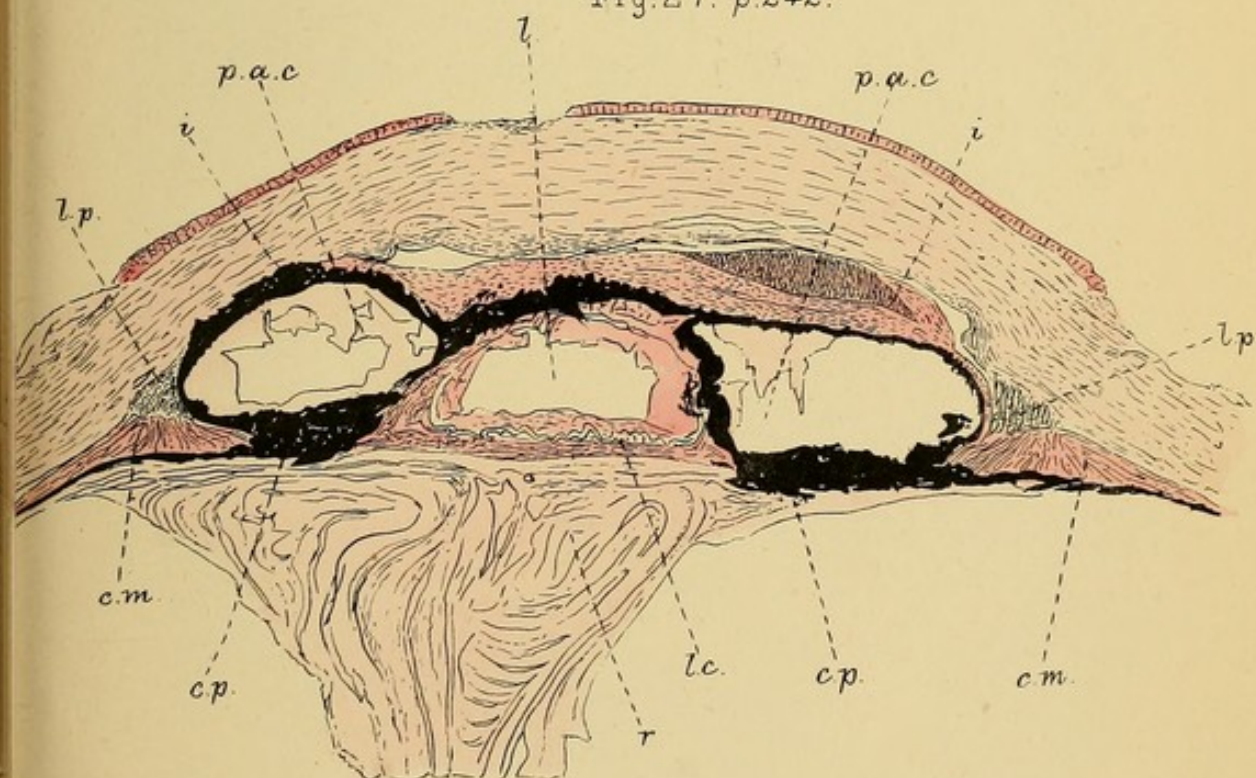


Fig. 27. p.242.



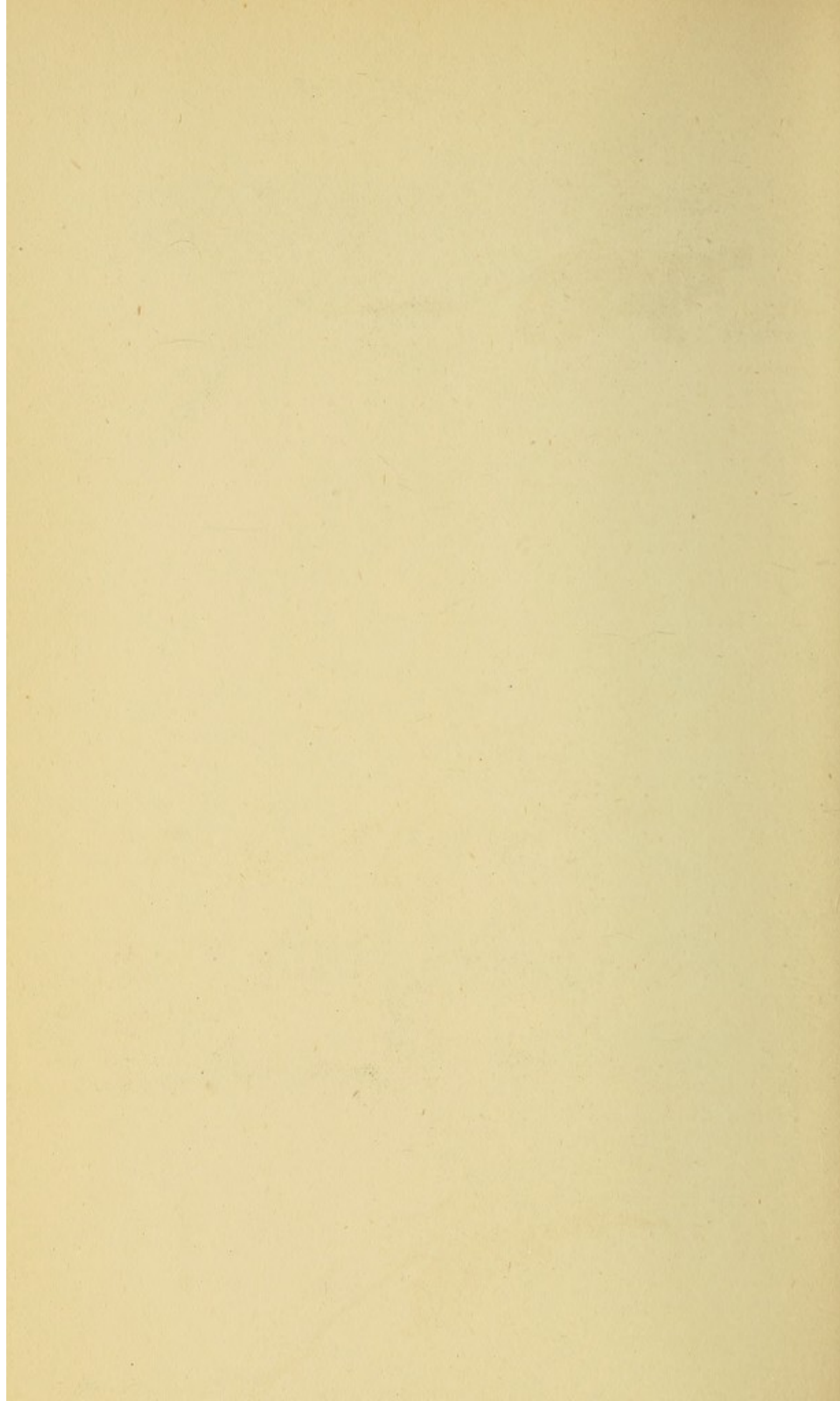


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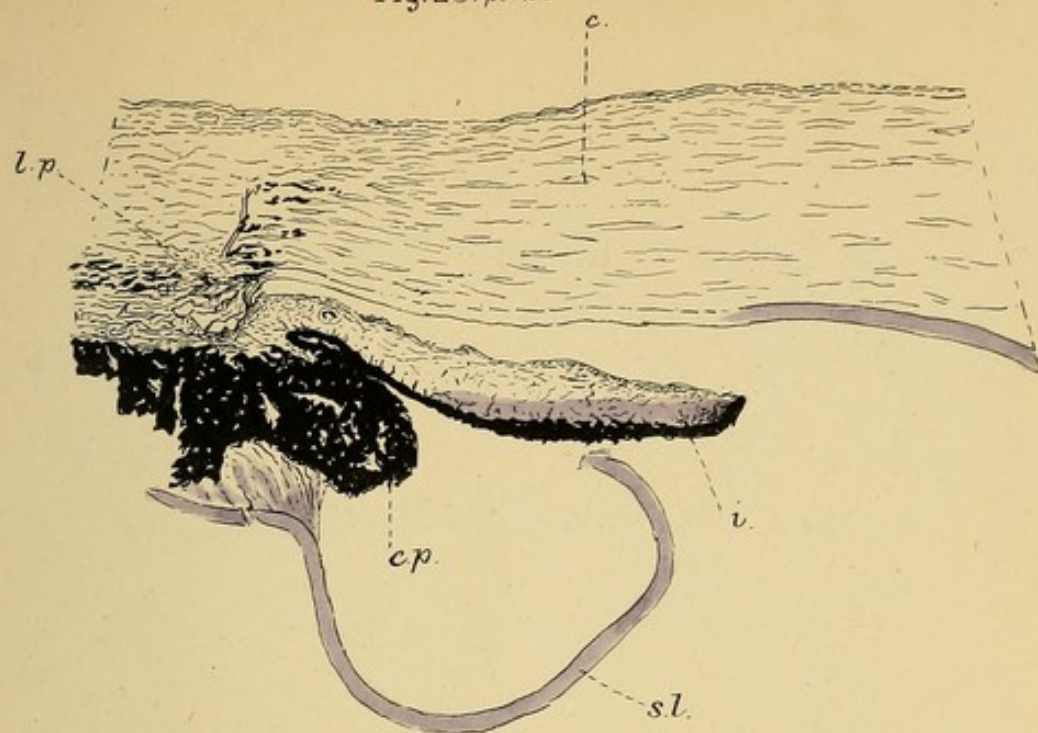


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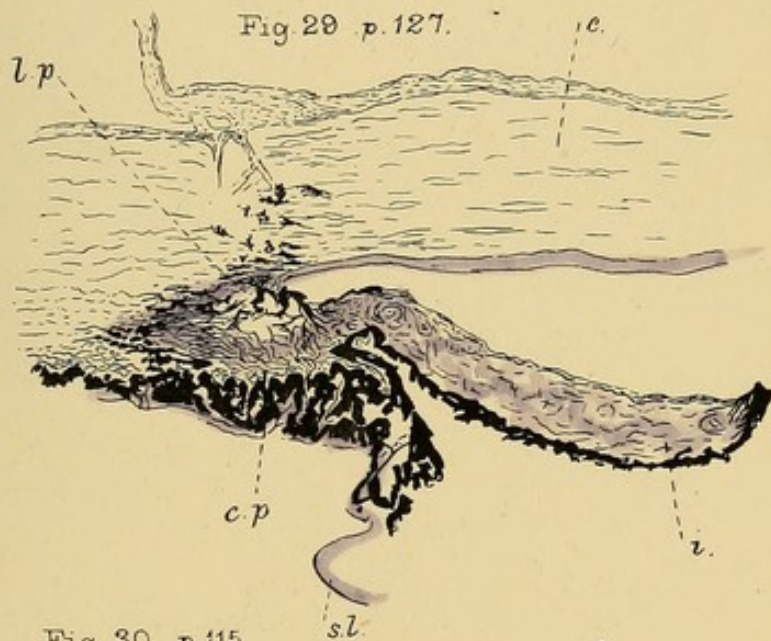
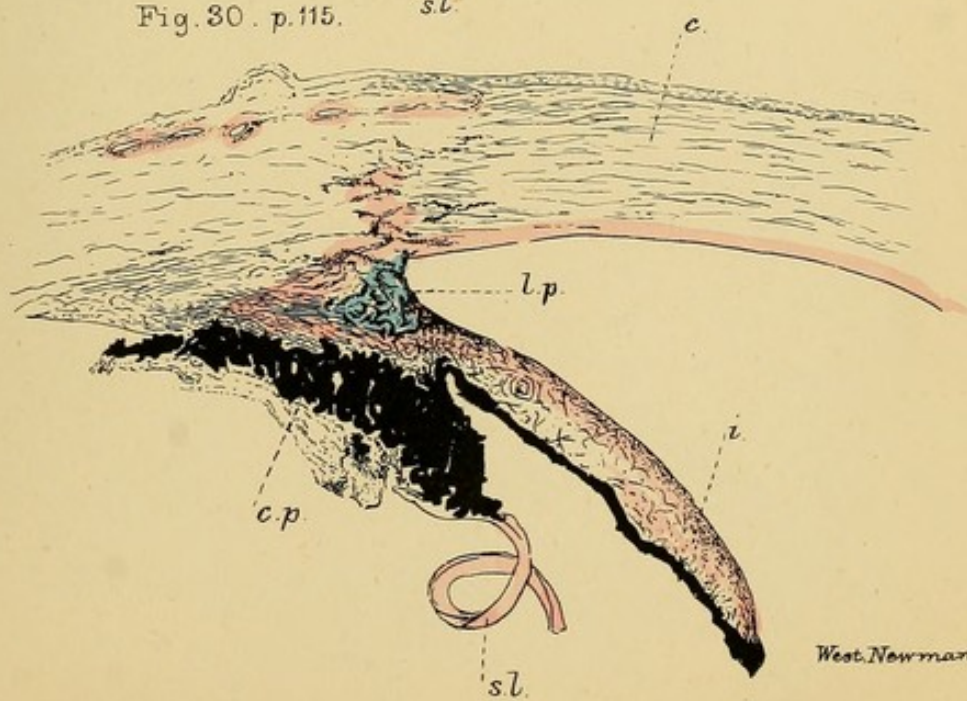


Fig. 30. p. 115.



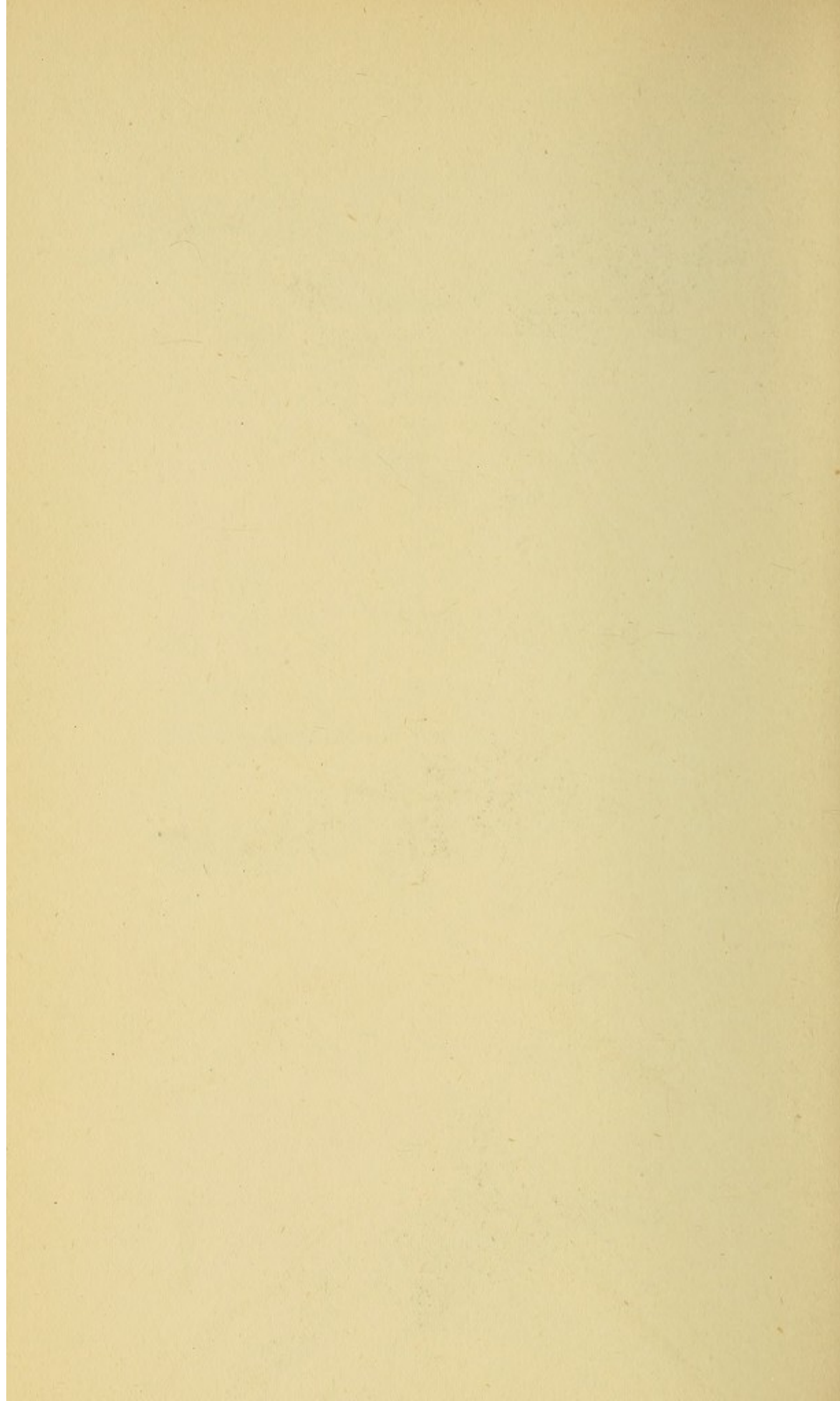


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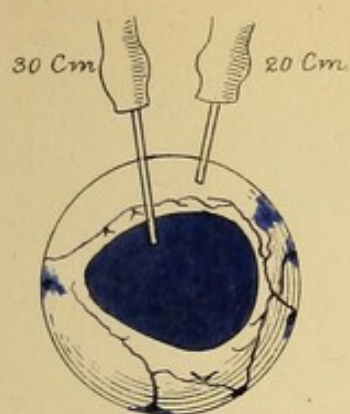


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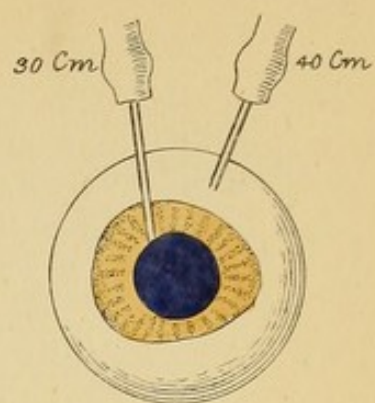


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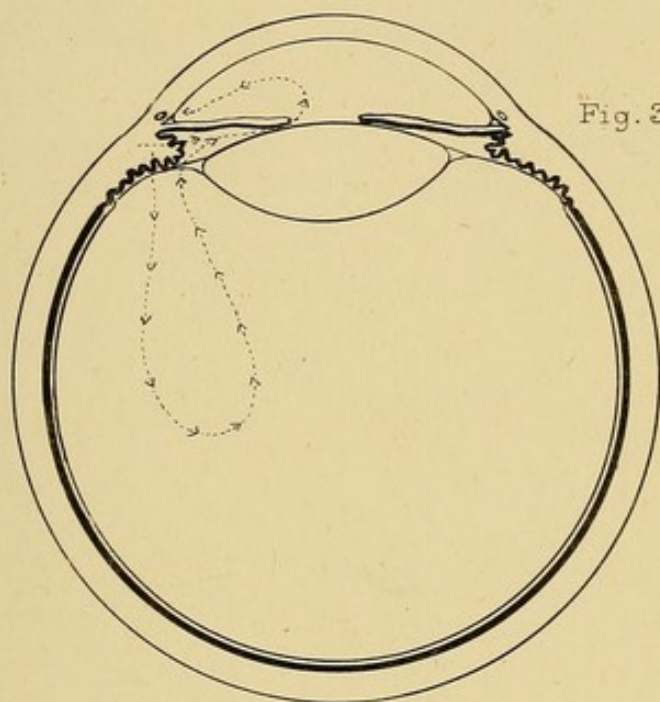


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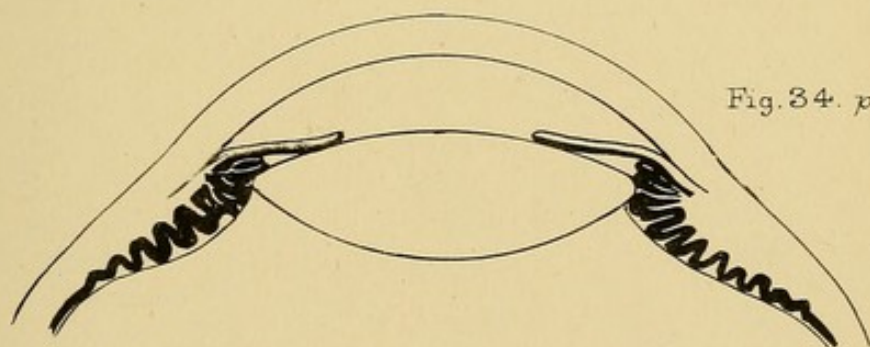
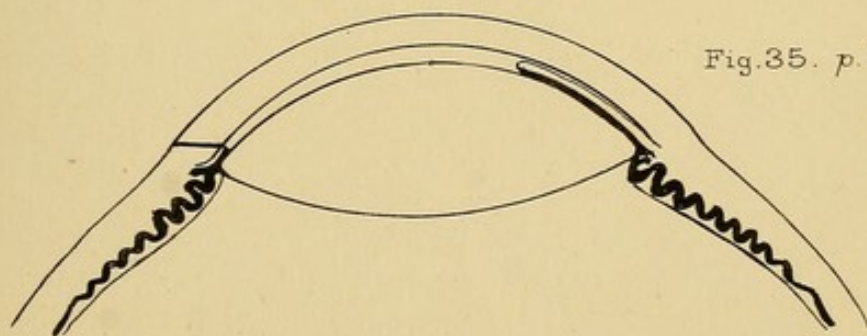


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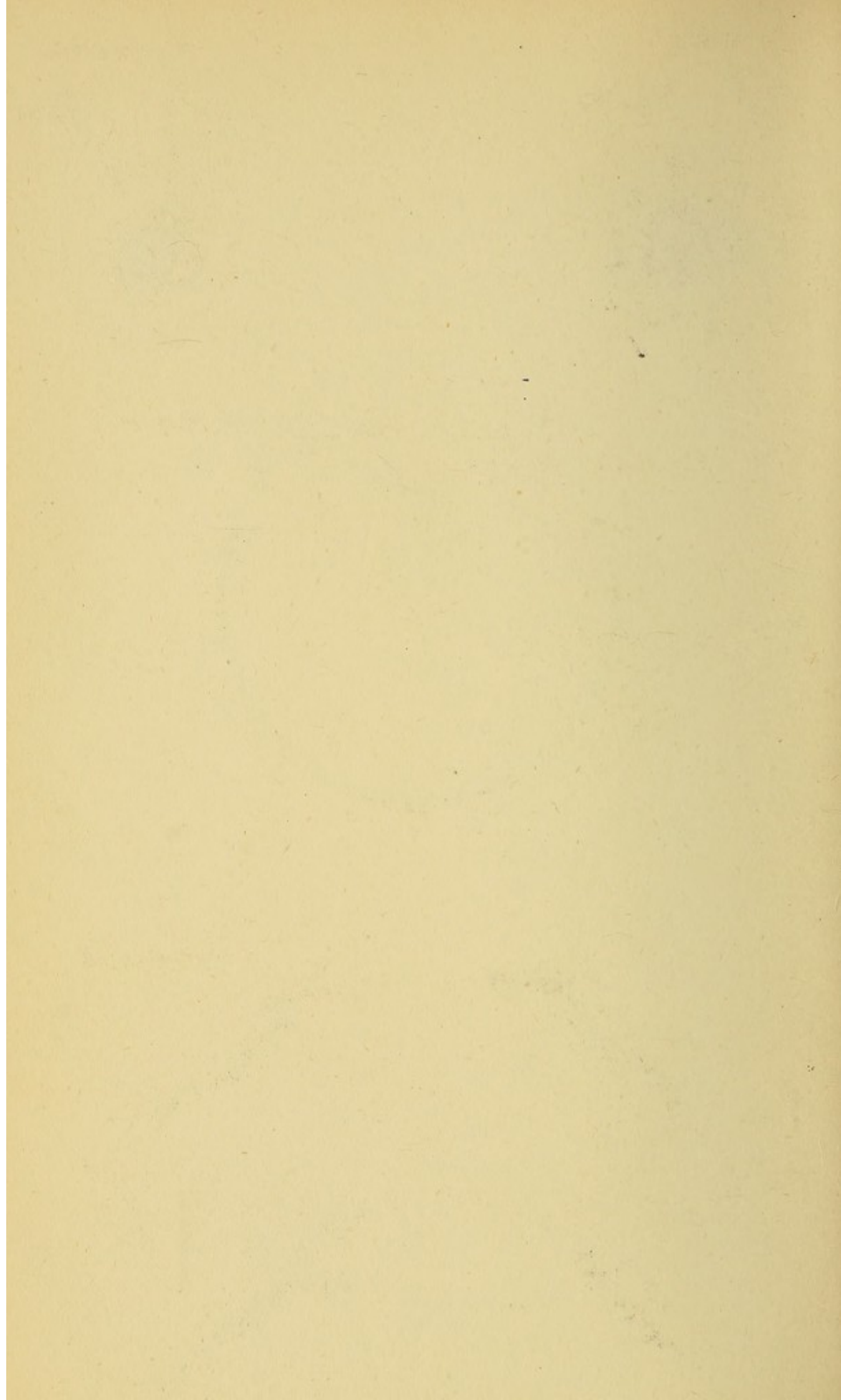


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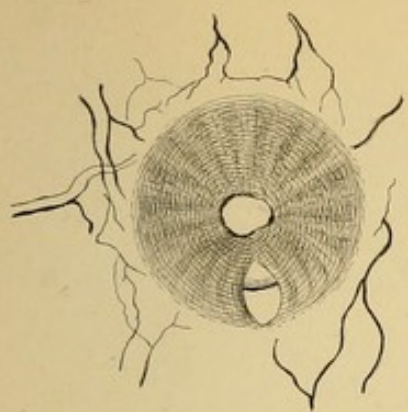


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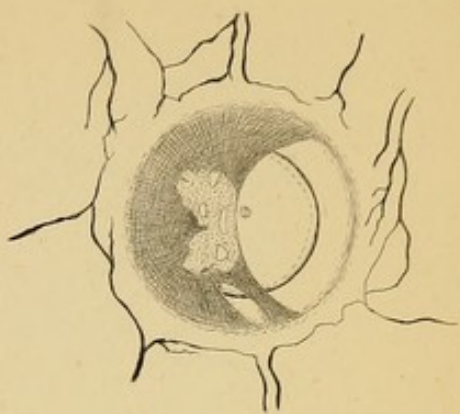


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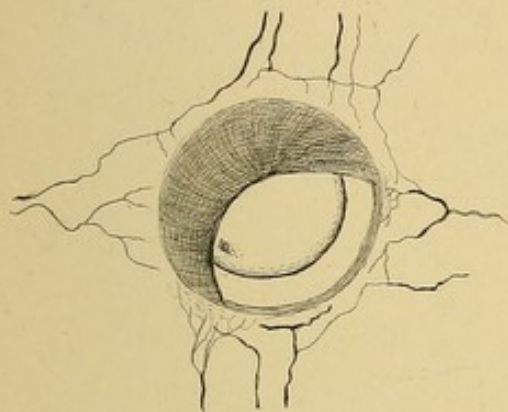


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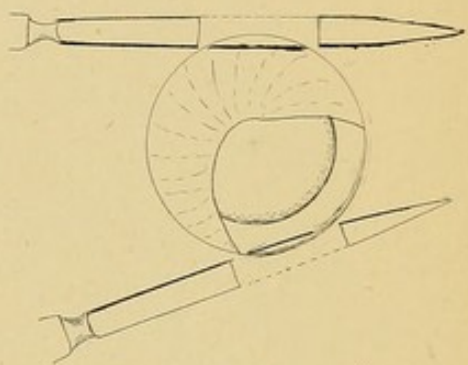


Fig. 40.
p. 238.



Fig. 41.
p. 238.



Fig. 42. p. 260.

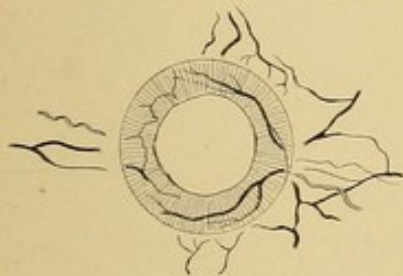
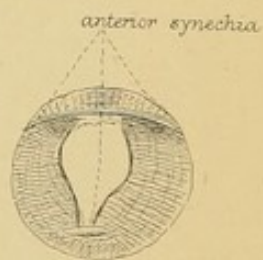


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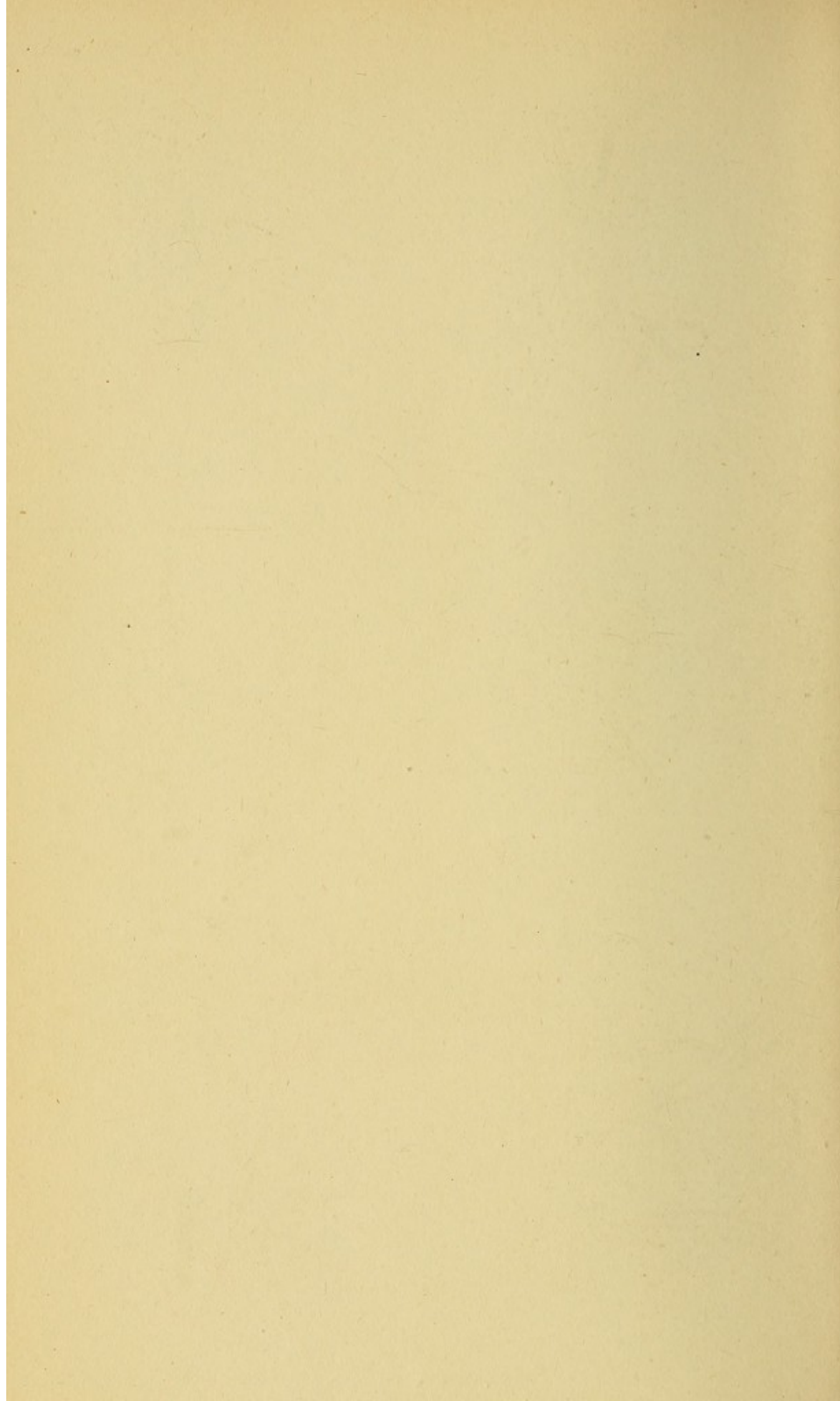


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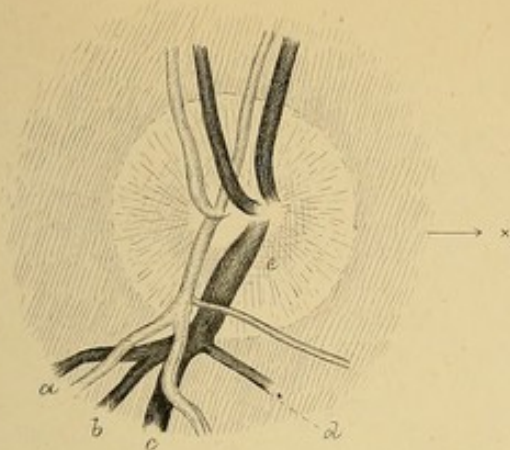


Fig. 45. p. 84.

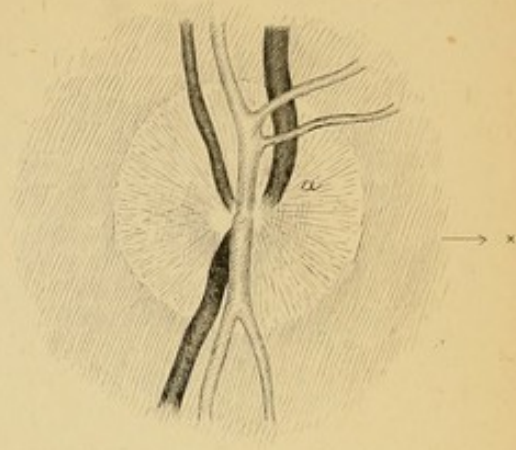


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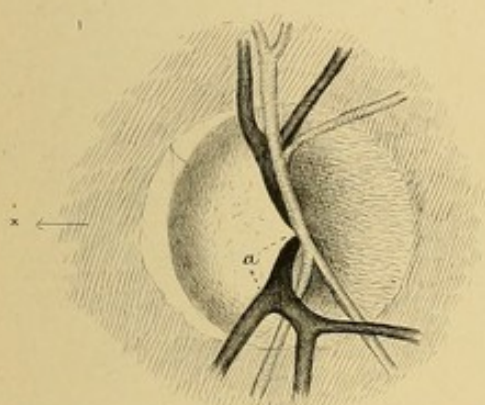


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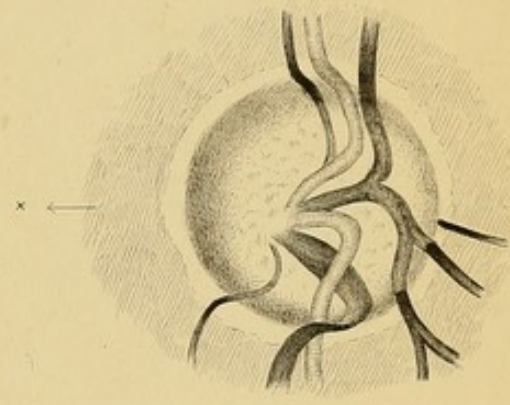


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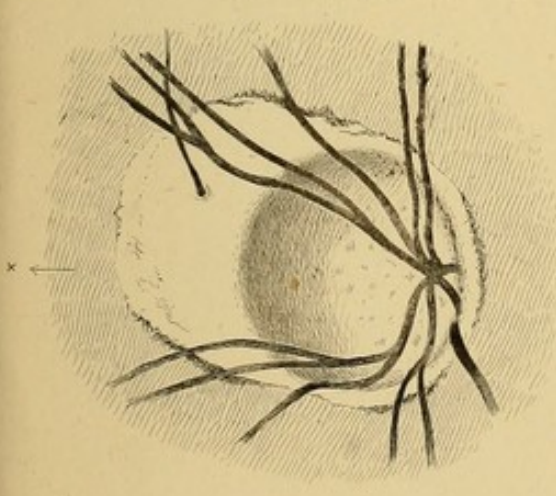
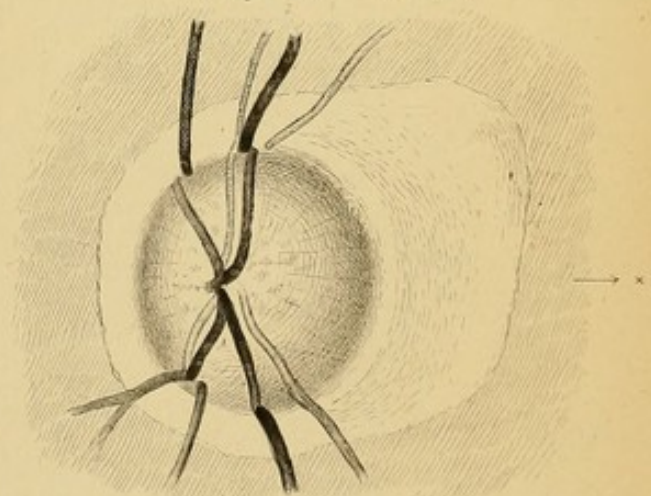


Fig. 49. p. 268.



The arrow ($\rightarrow x$) indicates the direction in which the "yellow spot" is situated.

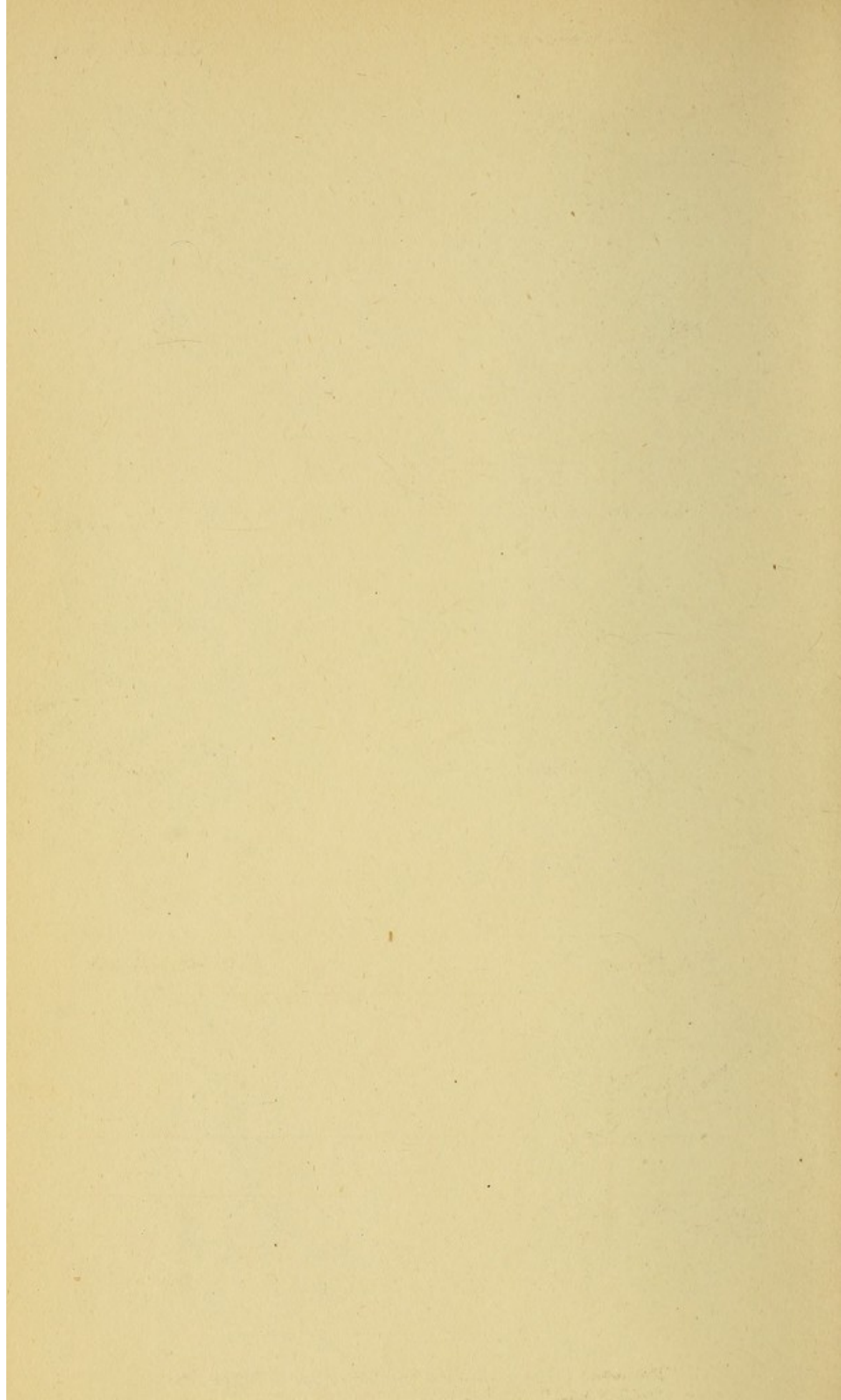


Fig. 50. p. 235.



Fig. 51. p. 237.

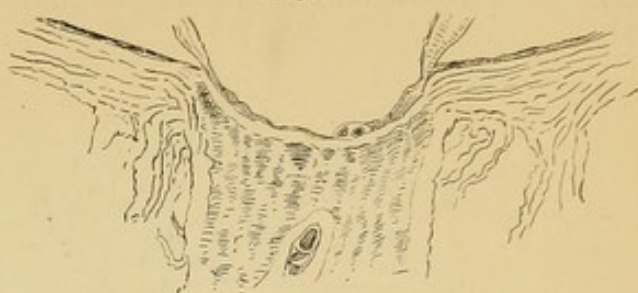


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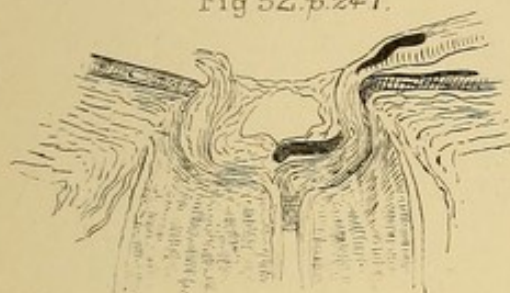


Fig. 52 p. 247

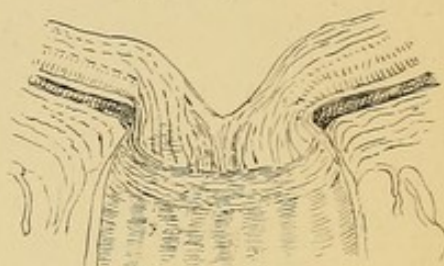


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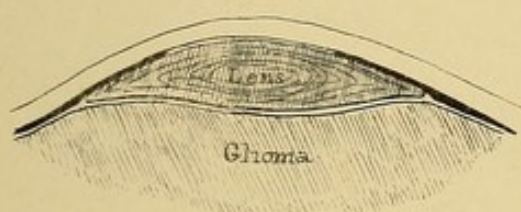


Fig. 55. p. 240.



Fig. 56. p. 83.

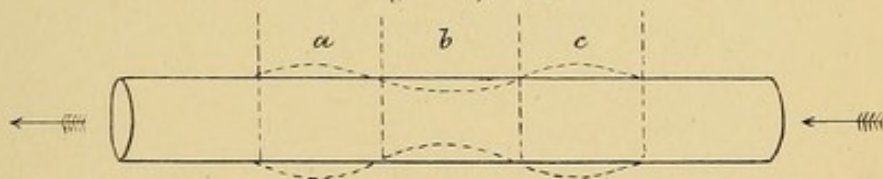


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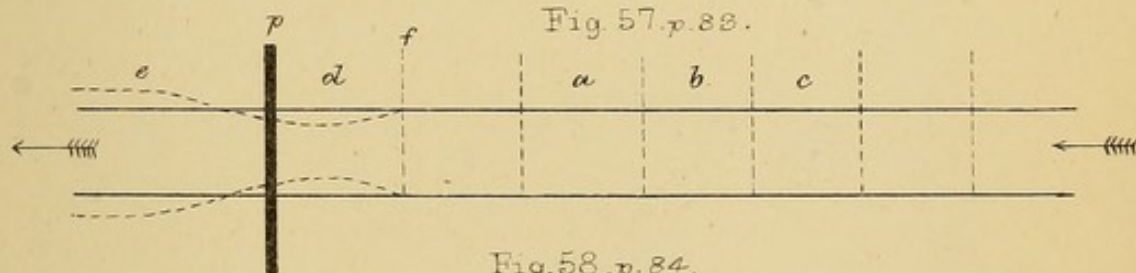
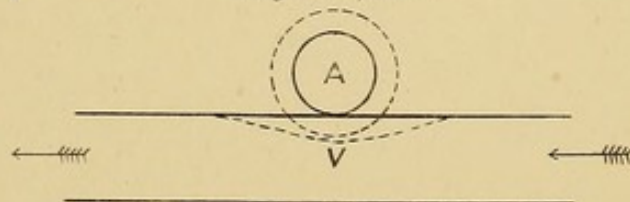
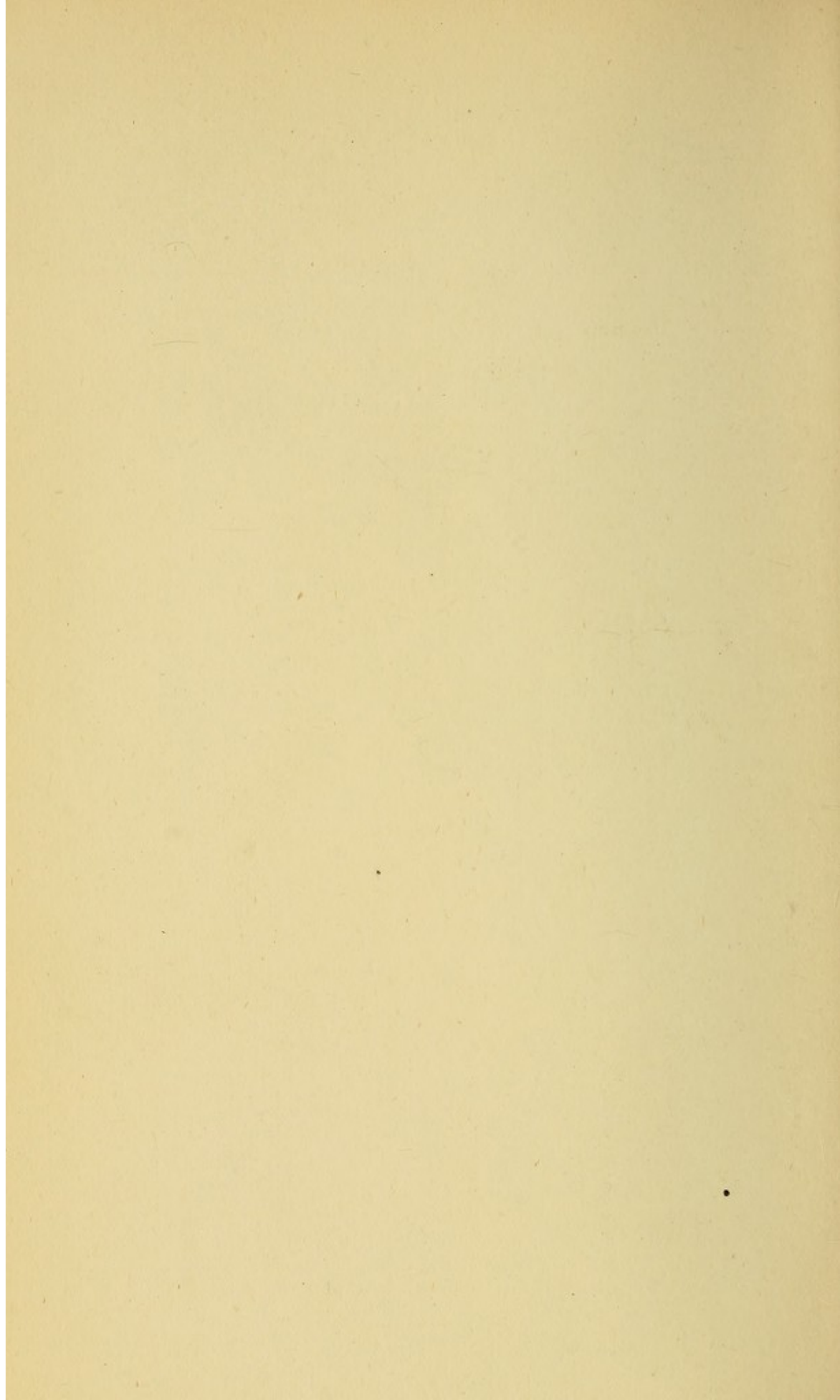
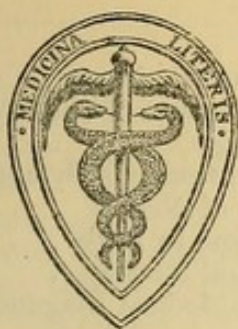


Fig. 58. p. 84.







*London, New Burlington Street.
September, 1879.*

SELECTION
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INDEX

	PAGE		PAGE
Acton on the Reproductive Organs	8	Dunghlison's Medical Dictionary	22
Adams (W.) on Clubfoot	6	Ellis's Manual of Diseases of Children	13
— (R.) on Rheumatic Gout	19	Emmet's Gynæcology	14
Allingham on Diseases of the Rectum	7	Eulenburg and Guttman's Sympathetic System of Nerves	19
Anatomical Remembrancer	11	Fayrer's Observations in India	4
Anderson (McC.) on Eczema	20	Fergusson's Practical Surgery	4
Aveling's Influence of Posture	15	Fenwick's Guide to Medical Diagnosis	12
Balfour's Diseases of the Heart	17	Flint on Phthisis	16
Bantock's Rupture of Perineum	14	— on Percussion and Auscultation	16
Barclay's Medical Diagnosis	12	Foster's Clinical Medicine	11
Barker's Puerperal Diseases	13	Fox (C. B.) Sanitary Examinations	21
Barnes' Obstetric Operations	14	Fox (T.) Atlas of Skin Diseases	20
— Diseases of Women	14	Fox (T.) and Farquhar's Skin Diseases of India	20
Basham on Diseases of the Kidneys	8	Frey's Histology	9
Beale's Microscope in Medicine	11	Galabin's Diseases of Women	14
Bellamy's Guide to Surgical Anatomy	10	Gamgee on Fractures of the Limbs	4
Bennet's Winter and Spring on the Mediterranean	17	— on Treatment of Wounds	4
— Pulmonary Consumption	17	Gant's Diseases of the Bladder	8
— Nutrition	18	Gaskoin on Psoriasis or Lepa	20
Berkart's Asthma	16	Godlee's Atlas of Human Anatomy	11
Bigg's Orthopraxy	6	Gowan on Consumption	16
Binz's Elements of Therapeutics	12	Gowers' Medical Ophthalmoscopy	21
Black on the Urinary Organs	8	Habershon on Diseases of the Abdomen	18
Blakiston's Clinical Reminiscences	11	— on Diseases of the Stomach	18
Bose's Rational Therapeutics	11	— on the Pneumogastric Nerve	18
— Recognisant Medicine	11	Hamilton's Nervous Diseases	18
Bradley's Lymphatic System	15	Hancock's Surgery of Foot and Ankle	6
Braune's Topographical Anatomy	11	Harris on Lithotomy	7
Brodhurst's Orthopædic Surgery	6	Harrison's Stricture of Urethra	7
Bryant's Practice of Surgery	4	Hayden on the Heart	16
Bucknill and Tuke's Psychological Medicine	21	Heath's Minor Surgery and Bandaging	5
Burdett's Cottage Hospital	15	— Diseases and Injuries of Jaws	5
Burnett on the Ear	6	— Operative Surgery	5
Buzzard on Syphilitic Nervous Affections	8	— Surgical Diagnosis	5
Carpenter's Human Physiology	10	— Practical Anatomy	10
Carter (W.) on Renal and Urinary Diseases	8	Higgins' Ophthalmic Practice	22
Charteris' Practice of Medicine	11	Holden's Landmarks	10
Clark's Outlines of Surgery	4	— Human Osteology	10
— Surgical Diagnosis	5	Hood on Gout, Rheumatism, &c.	19
Clay's Obstetric Surgery	13	Hooper's Physician's Vade-Mecum	11
Cobbold on Parasites	20	Horton's Tropical Diseases	18
Coles' Dental Mechanics	23	Hutchinson's Clinical Surgery	5
Cormack's Clinical Studies	12	— Rare Diseases of Skin	20
Cullingworth's Nurse's Companion	15	Huth's Marriage of Near Kin	8
Curling's Diseases of the Rectum	7	Ireland's Idiocy and Imbecility	20
— Diseases of the Testis	7	James' Sore Throat	17
Dalby on the Ear	6	Jones (C. H.) and Sieveking's Pathological Anatomy	10
Dalton's Human Physiology	9	Jones (H. McN.) Aural Surgery	6
Day on Children's Diseases	13	— Atlas of Diseases of Membrana Tympani	6
— on Headaches	18	Jordan's Surgical Inquiries	6
Dobell's Lectures on Winter Cough	15	Lane on Syphilis	8
— Loss of Weight, &c.	15	Leber and Rottenstein's Dental Caries	23
Domville's Manual for Hospital Nurses	15	Lee (H.) on Syphilis	8
Druitt's Surgeon's Vade-Mecum	4	Leared on Imperfect Digestion	19
Duncan on the Female Perineum	15		

	PAGE		PAGE
Living on Megrim, &c.	19	Stocken's Dental Materia Medica	12
Macdonald's (A.) Disease of the heart	16	Sullivan's Tropical Diseases	17
Macdonald's (J. D.) Examination of Water	21	Swain's Surgical Emergencies	5
Mackenzie on Diphtheria	16	Swayne's Obstetric Aphorisms	14
Macnamara on Diseases of the Eye	22	Taft's Operative Dentistry	23
Madden's Health Resorts	17	Tait's Hospital Mortality	15
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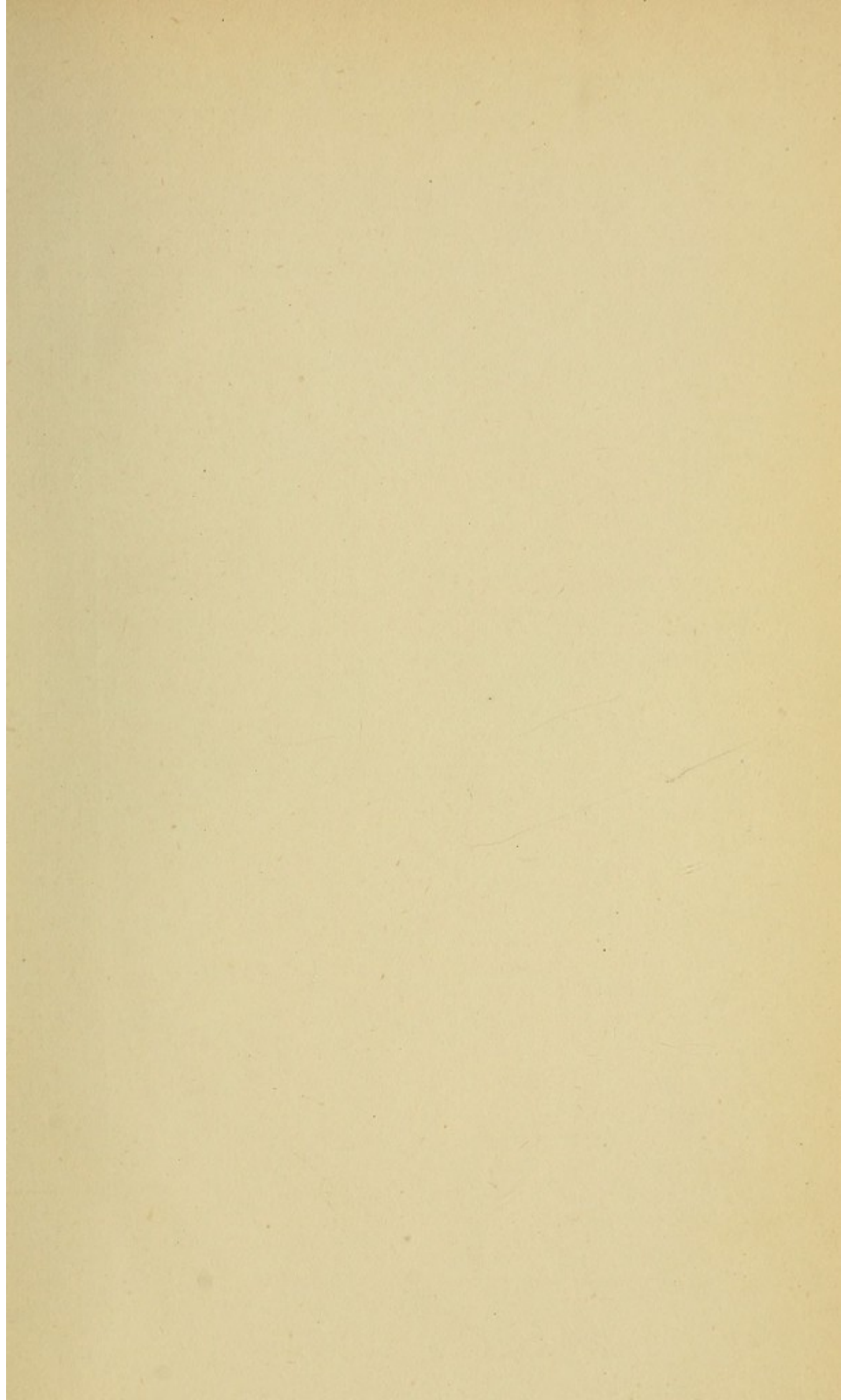
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