Lectures on the anatomy and pathology of the eye : delivered at the Royal College of Surgeons of England, in December, 1894 / by E. Treacher Collins.

Lectures

ON THE

ANATOMY AND PATHOLOGY OF THE EYE.

Delivered at the Royal College of Surgeons of England, in December, 1894.

BY

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Reprinted from THE LANCET.

ANATOMY AND PATHOLOGY OF THE EYE.

LECTURE I.

Delivered on Dec. 3rd, 1894.

MR. PRESIDENT AND GENTLEMEN,—The pathology and anatomy of the eye are large subjects, and it would be impossible to treat of them completely in the course of three lectures. I have chosen it, however, as a title because it leaves me free to speak on several widely different points which I have had exceptional opportunities of investigating, and to which I have devoted special attention. In studying the pathology of the eye I soon became aware how largely a knowledge of the embryology of that organ helped to interpret the changes met with. Not only does embryology help to elucidate morbid processes, but I think I shall be able to show that very valuable suggestions as to doubtful points in the development of tissues may be obtained from the changes which occur in disease. The way in which these two subjects, embryology and pathology, mutually help to explain one another will be the keynote of my lectures.

Taking first the suspensory ligament of the lens, which was at one time supposed to consist of two layers of membrane, but which is now known, and is shown by the specimens on the table, to consist of bundles of fibres, its mode of development has been left by embryologists but very imperfectly explained. According to Kölliker, it is formed in man during the fourth month. Lieberkühn remarks that the zonule is distinctly recognisable in eyes which have attained half their definite size. Angelucci and Löwe believe that the suspensory ligament is derived from the anterior part of the vitreous. My attention was first directed to the matter by the examination of an eye of which I published a

full description in the Royal London Ophthalmic Hospital Reports. It had been enucleated from an infant aged three months, under the supposition that it contained a new growth. Pathological examination revealed that the opaque mass behind the lens, which had been taken for the growth, was really a collection of cells and fibres, into which a persistent hyaloid artery, still patent and carrying blood, passed and broke up. The chief interest of the specimen, however, centred in the fact that it exhibited the fibres of the suspensory ligament in all stages of their development. The fibres of the lens, instead of developing in the normal way, filling and rendering tense the capsule in which they are contained, had undergone retrogressive changes, and had left the capsule lax and wrinkled. Whether this was the result of the persistence of the hyaloid artery, or whether it had persisted as a result of changes in the lens, I am unable to determine. Anyhow, as the result of the lax condition of the capsule, the fibres of the suspensory ligament do not appear to have become stretched to their usual extent, and hence exhibited all stages of delayed development. The ciliary processes were in places nearly in contact with the sides of the lens; adhesions stretched between them composed of spindle-shaped cells, and in other parts fibres like those of the suspensory ligament with nuclei lying on them. These fibres, as they approached their insertion into the ciliary processes, became thicker, and some of them were there composed of several layers of elongated cells, which gradually merged into the unpigmented cells covering the ciliary processes.

The appearances shown in these specimens suggested that the fibres of the suspensory ligament were originally cellular adhesions passing between the sides of the lens and the ciliary processes; that as the eye grew these adhesions became stretched, and lengthened out into fibres with nuclei on them; and that ultimately the nuclei disappeared and left the suspensory ligament as we see it in the adult eye. To test the truth of this theory I measured the diameters of a series of fœtal eyes and the diameters of the lenses contained in them. The result of these measurements I have embodied in Table I. There is a possible source of error in the fact that all the measurements were made after the eyes had been hardened in Müller's fluid. On account of there being necessarily some delay in my obtaining the eyes after their removal this was the only course open to me. In every case an antero-posterior vertical section was made of the eyeball and the lens measured *in situ*.

TABLE I.—*Showing the Diameters of a Series of Fœtal Eyes and of their Lenses.*

Age.	No. of eyes examined.	Diameters of Eyeball			Diameters of Lens.	
		Antero-posterior.	Lateral.	Vertical.	Antero-posterior.	Transverse.
4th month	3	8·1 mm.	7·8 mm.	7·5 mm.	2·8 mm.	3·3 mm.
5th month	1	11·75 mm.	11·5 mm.	10·5 mm.	3·5 mm.	4 mm.
6th month	4	12·5 mm.	12 mm.	11·1 mm.	3·8 mm.	4·5 mm.
7th month	8	14·3 mm.	13·2 mm.	12·6 mm.	4 mm.	5 mm.
9th month	3	16·75 mm.	16 mm.	15·3 mm.	4·3 mm.	5·75 mm.
Adult ...	Merkel	24·3 mm.	23·6 mm.	23·4 mm.	3·7 mm.	9 mm.

This table shows that the diameters of the globe at the ninth month are just double what they are at the fourth month, and in the adult treble that size; that the transverse diameter of the lens is likewise double at the ninth month and treble in the adult what it is at the fourth month. So supposing, as is the case, that the ciliary body touches the sides of the lens at the fourth month—in that the eyeball is much larger than the lens, to begin with, and both increase to double and treble their size—then, unless the thickness of the coats of the eyeball also become double the thickness they were (which they do not), there must be a gradual growing away of the ciliary body from the sides of the lens and stretching of any adhesions which had formed between them. I have recently examined microscopically a human fœtal eye of between the third and fourth months, in which cellular adhesions passing between the ciliary processes and the fibro-vascular sheath of the lens are distinctly shown. I think that there can be little doubt these are the commencing fibres of the suspensory ligament. The hindermost fibre of the suspensory ligament is inserted into the retina in the region of the ora serrata. The retina, as was long ago pointed out by von Ammon, and as I have had abundant opportunities of confirming, is in the fœtal eye thrown into numerous creases; apparently the inner layer of the secondary optic vesicle grows at a greater rate than the outer. The most pronounced of these folds and the last to disappear is in the region of the ora serrata. In fact, folds of retina at this part are at one time in contact with the back of the lens, and while there are quite capable of acquiring adhesions to the fibro-vascular sheath. On the

growth of the globe, the enlargement of the vitreous and the flattening out of the retina these adhesions become stretched and elongated into fibres.

The above description of the way in which the zonule of Zinn develops not only throws light on some of the congenital abnormalities met with in connexion with the lens, but also receives considerable support from its mode of explaining them. Taking first the condition known as coloboma lentis, an excellent *résumé* of the literature of which malformation, together with two new cases, has been published by Dr. Heyl,[1] I cannot do better than quote the passage in which he sums up: "From the preceding it may then be seen that coloboma of the lens is a condition which presents at the place of defect an edge, not rounded as in the normal condition, but either straight in the horizontal direction or incurved; that the amount of deficiency varies from a slight indentation to about one-quarter of the lens substance, the centre of the lens, its poles, and the lens substance in the immediate vicinity being uninvolved; that the lens sometimes, in addition to the defect, is imperfectly developed in all its meridians; that the deficiency is always in the inferior half, and, almost without exception, entirely so; and, finally, that coloboma of the other ocular structures frequently coexists, but very frequently no trace of it can be found."

Now, supposing the pars ciliaris retinæ failed to acquire adhesions to the lens capsule in one part of its circumference, it follows that the suspensory ligament would be absent in that position; and that as the eyeball enlarged that portion of the capsule to which no suspensory ligament was attached would not be held taut, and made to expand like the remainder. Consequently there would be a depression in the lens in that situation. The amount and shape of the deficiency would depend on the extent of the defect in the suspensory ligament. There would not, of course, be any alteration in the nucleus or poles of the lens, for these are formed before the influence of the suspensory ligament comes into play. The most likely cause for the non-adhesion, and so for the defect in the suspensory ligament, would be the absence of the ciliary body or pars ciliaris retinæ—in fact, coloboma of the ciliary body. Dr. Heyl says that this condition does frequently coexist with a defect in the lens, the defect being in the situation of the fœtal cleft. Mr. Marcus Gunn,[2] regarding the condition of coloboma lentis from the clinical

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1 Transactions of the Fifth International Ophthalmic Congress, p. 16.
2 Ophthalmic Review, vol. viii., p. 234.

side, has arrived at a similar conclusion as to its mode of formation. He says : "The notch in the lower border of the lens may be accounted for by the imperfect development of the ciliary processes and suspensory ligament, so that the soft lens is not here drawn outwards into the normal comparatively sharp curve at its equator." The same observer[3] has pictured and described a case of congenitally misplaced and notched lens, in which a distinct gap in the suspensory ligament was visible corresponding to the notch. I have recently examined pathologically an eyeball in which there was a congenital coloboma in the outer margin of the lens. The ciliary processes opposite the notch in the lens, instead of being directed inwards towards the sides of the lens, as they were in the rest of the circumference of the globe, sloped backwards. This might be explained by the suspensory ligament being absent in this position, the ciliary processes not having been drawn forwards by it, as was the case elsewhere. Whether or not there was a gap in the suspensory ligament I was unable definitely to determine, owing to the way in which the eyeball was opened. Ectopia lentis, or congenital displacement of the lens, has been attributed by Becker to irregular development of the suspensory ligament. I think the explanation I have given of the mode of formation of the latter helps us to understand how this comes about. The literature of the subject has been carefully worked up by Dr. D'Œnch,[4] and he ends his article with the following conclusions :—"1. Ectopia of the lens is a malformation, the causes of which thus far remain unknown. 2. It always affects both eyes generally in a symmetrical manner. 3. The direction of the displacement is almost always either upwards and inwards or upwards and outwards. 4 The lenses are generally transparent ; sometimes their size is below the mean. 5. The suspensory ligament is sometimes found and sometimes it is not. 6. In about one-fourth of all cases there is myopia. 7. The position of the lenses may remain unchanged throughout life, but spontaneous dislocation may also result. 8. Heredity has been proven." Supposing the adhesions which take place between the capsule of the lens and pars ciliaris retinæ to form the fibres of the suspensory ligament be absent over a large area, then on the expansion of the globe there would be no counteracting force on one side to hold the lens in position, and it would be drawn

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[3] Transactions of the Ophthalmological Society, vol. ix., p. 166

[4] Archives of Ophthalmology, vol x., p. 89.

towards that side to which the adhesions had formed. The determining cause as to whether a coloboma of the lens is formed or an ectopia would depend on the extent of the deficiency in the suspensory ligament. In some cases, as would be expected, both are present. When the deficiency is very great indeed the lens would be mobile, and might become dislocated. Abnormal density or frailty of the adhesions in part of the circumference would also be likely causes of displacement of the lens, and would account for those cases in which some of the fibres of the suspensory ligament can be seen passing (in a case of displacement upwards) from the lower margin of the lens to the ciliary body. Delay in the closure of the fœtal cleft, though it did not lead to permanent coloboma of the ciliary body, could yet lead to ectopia of the lens, for the eye might have expanded to such an extent that the lens would have been drawn away from the lower part of the globe before the ciliary processes had sufficiently developed to have acquired adhesions to its capsule. Mr. Nettleship, in his Text-book on Diseases of the Eyes,[5] says: "Congenital dislocation of the lens is often accompanied by other defects of development, such as coloboma." It is noteworthy that the lens is almost invariably displaced upwards either directly or with some inclination inwards or outwards; that is to say, it is almost always displaced in the opposite direction to the fœtal cleft, which we know is generally downwards, or downwards with a slight inclination inwards or outwards. The table of measurements of lenses which I have put before you shows that the transverse diameter of the lens is at the fourth month only a little more than the antero-posterior diameter, and in the adult lens is nearly three times as much. This globular condition of the fœtal lens is well known, and the alteration in its shape is due to the laying on of fresh lens fibres at the sides. The cells lining the anterior capsule proliferate and shift round it until they reach a position a little behind its equator, where they commence to lengthen out. The traction which is exerted on the sides of the lens, through the fibres of the suspensory ligament, by the growth of the ciliary processes away from it, apparently influences the position in which new lens fibres are laid on.

[Mr. Collins then went on to show how cells come to line the posterior capsule in cataractous lenses.]

The way in which the capsule of the lens develops has not

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[5] Fifth edition, p. 181.

yet been definitely determined. There are two views respecting it which are thus referred to in the ninth edition of Quain's Anatomy : "The origin of the capsule of the lens appears to be still somewhat doubtful. Lieberkühn, Arnold, and Löwe profess to trace it to a thin pellicle of mesoblast, which at an early period passes in between the lens and the secondary ocular vesicle ; but Kölliker and Kessler are of opinion that it is a cuticular deposit on the surface of the lens cells. Balfour is inclined to adopt the latter view, on the ground that the capsule seems to make its appearance before the introduction of the mesoblast has occurred." The examination of the histological changes in anterior polar or pyramidal cataracts throws, I think, some light on this matter. I have had the opportunity of examining pathologically a large number of lenses with this form of cataract, and I will discuss in detail the changes observed in them and enter somewhat fully into the views which have been put forward as to their mode of origin. I have examined microscopically thirteen lenses with anterior polar cataracts, which have varied from one month up to twenty-one years in the length of time which had elapsed since their formation.

I may here mention that I only regard as anterior polar cataracts opacities which are situated within the capsule ; these are opacities of the lens itself—that is, true cataracts ; and to them the name should be confined. It is possible that there may be opacities at the anterior pole of the lens due either to inflammatory deposit or to the persistence of a portion of the anterior fibro-vascular sheath ; these would be external to the capsule, and therefore not true cataracts. It is well not to confound these latter with the former, but to speak of them as what they really are—viz., pupillary membranes, either inflammatory or congenital. The first change to occur in the formation of an anterior polar cataract is a localised proliferation of the cells lining the anterior capsule and some disturbance and breaking up of the lens fibres in the neighbourhood. As a consequence of the proliferation of the capsule cells, elevation of the hyaline capsule over them is brought about, and from the breaking up of the lens fibres some excavation of the lens substance beneath them occurs. Later the mass of cells and débris of the broken-up lens fibres becomes condensed into an almost structureless substance, showing a slight lamination and having a few flattened epithelial cells stranded here and there in it. When the mass which constitutes the opacity has reached this condition the cells which line the capsule at its margin

spread in between the opaque part and the unaltered lens substance, so that in an anterior polar cataract which has existed for more than seven months a layer of cells is seen between the opacity and the lens substance continuous with those lining the capsule elsewhere. Later still—that is, after several years—a layer of hyaline capsule is found separating the opacity from the lens substance in addition to the layer of cells, so that at the margin of the opacity the hyaline capsule seems to split into two divisions—one going in front of it and the other, with a layer of cells lining it, behind it.

Presumably the newly formed layer of capsule is a deposition from the cells ; and if the capsule cells are in a pathological condition capable of producing a layer of hyaline capsule it seems reasonable to suppose that that structure is originally formed by them. That the changes which give rise to pyramidal cataract are situated beneath the hyaline capsule was long ago pointed out by Stellwag von Carion, Müller Bucke, Bauer, Dixon, and Hulke. That occasionally a hyaline layer is met with behind the opacity as well as in front of it was mentioned by Becker, who considered that it had become split off by the intrusion of new material into the substance of the capsule. More recently B. Gepner, jun.,[6] as the result of examination of two cataractous lenses from fishes' eyes, came to the conclusion that these hyaline layers were not split off the original capsule, but freshly secreted by the epithelial cells. This latter view of their origin I am inclined, as I have said, to believe to be correct, because in all the more recent anterior polar cataracts which I have examined I find there is no distinct layer separating the opaque part from the lens fibres. In those of longer duration a layer of cells is observed between them, which looks as if they had spread in from the unaltered cells lining the capsule. In two lenses in which several years had elapsed since the formation of the cataract, in front of the layer of cells was the layer of hyaline capsule ; it was thicker in the one of twenty-one years than in that of eleven years' duration. Also, because the hyaline layer behind the opacity in one of the cases is a complete layer. I have examined numerous sections of it, but have been unable to find any gap in its continuity through which the intrusion of new material could have taken place. It is sometimes observed that when there is an opacity at the anterior pole of the lens there is also a second opacity, a little depth in the lens substance, separated from the anterior one by some clear lens matter. I have notes of three such cases.

[6] Archiv für Ophthalmologie, Band xxxvi., p. 255.

The first case was that of a man aged twenty-four years who in infancy had had inflammation of the eye, which left a nebula of the cornea. There was a raised opacity at the anterior pole of the lens opposite the nebula, and further back in the lens another opacity. The second case was that of a girl aged nineteen years. She stated that both her eyes were bad when she was born; they were swollen and discharged; they remained bad for about three months and had been in their present condition as long as she could remember. She had slight lateral nystagmus, faint nebulæ of both corneæ, a small central opacity at the anterior pole of each lens, and a second opacity in each lens at a little depth in it, clear lens substance intervening. The third case was that of a man aged twenty years; his left eye was inflamed when he was three years old; he never remembered seeing properly with it. There was a faint nebula of the cornea, and in a position corresponding to it an opacity at the anterior pole of the lens; posterior to this was a layer of clear substance, and then a second opacity.

It will be seen that in all these three cases several years had elapsed between the inflammatory attack, which presumably caused the anterior polar opacity, and the time at which the patient came under observation. These lenses had therefore considerably increased in size, due to the laying on of fresh cortical fibres since the anterior polar cataracts were first formed. These fresh fibres would have separated the opaque part, produced by the mass of proliferated epithelium, from the part of the lens which was in contact with them when the disturbance originally occurred, and which was also probably broken up and opaque. In this way two opacities are formed separated by clear lens substance, the posterior of which would appear to sink deeper and deeper as the lens increased in size. It is well known that anterior polar opacities in the lens are frequently produced by ulceration of the cornea, and frequently follow that very common cause of ulceration of the cornea—viz., purulent ophthalmia of infancy. Though most often originating in early life, they may occur at any age. I have examined one of recent formation from a man aged fifty-one years, who one month previously to excision had had ulceration of the cornea, with hypopyon, for which a Saemisch's section had been performed. I have another specimen from a man aged forty-six years, whose eye was affected with a traumatic ulcer of the cornea one month previously to its enucleation. True anterior polar cataract—that is, where the opacity is situated beneath the capsule and which in every respect resembles those which follow on

ulceration of the cornea—may be congenital and occur in eyes with perfectly clear corneæ. I have two specimens which demonstrate this, and I wish to lay particular stress on it, because it is very tempting to attribute any congenital opacity at the anterior surface of the lens to the persistence of a portion of the anterior fibro-vascular sheath, especially if tags of it are adherent to the anterior surface of the iris in the same eye. The first of the two cases of congenital anterior polar cataract I examined was associated with such tags, and in addition with almost complete congenital absence of the iris in both eyes. The patient died when three years old as a result of a burn, and both eyes were obtained for pathological examination ; one has been mounted as a macroscopical specimen, and sections have been cut of the other. I shall have to refer again to this eye in speaking of congenital abnormalities of the iris. My second specimen of anterior polar cataract is a very remarkable one. The eye was excised when the child was seven years old ; it had, in addition to the opacity of the lens, congenital anterior synechia of the iris and hydrophthalmos. The anterior polar opacity was very large and the rest of the lens imperfectly developed. As I have said, it is quite possible to have congenital anterior polar cataracts without any opacity of the cornea. Often in those in which anterior polar cataracts exist, and in which there is a distinct history of purulent ophthalmia in infancy, only a faint nebula of the cornea is found ; so faint, indeed, sometimes that it seems impossible to believe that it is all the damage that has resulted from a perforating ulcer of the cornea. Though these cataracts are most often seen clinically in cases where there are slight corneal changes, from the great frequency with which I have met with them pathologically in eyes where the cornea is completely opaque, and where there can be no doubt that perforation has occurred, I feel convinced that a perforating ulcer is the commonest cause of them.

Such, then, being the facts with regard to anterior polar cataracts, how can their formation be explained? Mr. Hulke,[7] writing on this subject, says : "When we remember the small size of the anterior chamber of an infant's eye and the spherical shape of the lens at this period of life, it becomes apparent that the distance between the front of the lens and the posterior surface of the cornea must be very small, for a large segment of the lens projects through the pupil and the

[7] Royal London Ophthalmic Hospital Reports, vol. i., p. 190.

zenith is considerably in advance of the plane of the iris ; in these respects the infant's eye presents a striking similarity to that of the fish. In ophthalmia neonatorum, when the cornea has become inflamed and swollen its posterior surface may actually come into contact with the front of the lens, and then a dot of lymph poured out upon the latter by the inflamed cornea, or even the mere pressure contact, may give rise to opacity by preventing the proper nutritional osmose through the cornea ; the little white cones which seem to project forward through the pupil in cataracta pyramidata, have their origin in this way." Mr. Hutchinson[8] says on this matter : "The theory which I would venture to support is this, that the mere proximity of the inflammatory action on the surface of the conjunctiva and cornea suffices to disturb the nutrition of the lens capsule and to produce deposits. It is of course admitted that the lens surface is, during infancy, in very close proximity with the posterior surface of the cornea, so that such disturbance of nutrition may be more easily possible than it would in adult life. Still, if this hypothesis be tenable, we have in the occurrence a most interesting example of the pathological possibility that diseased action may, by a sort of vital catalysis, disturb a structure with which it is not in continuity, and that even when there exists between the disturbing and disturbed tissues a structure (cornea) which is wholly unaffected, and a layer of fluid of greater or less depth." Mr. Hutchinson,[9] in criticising the views of Mr. Hulke on the mode of formation of pyramidal cataracts, says : "I cannot but suspect that we adopt hypotheses which are too mechanical when we attribute these little opacities either to corneal perforation or to prevention, by pressure, of nutritional osmose. As regards the latter, we must remember that there is excess, not deficiency, of growth." My own observations of these cataracts lead me to incline to the view put forward by Mr. Hulke, and to attribute their formation to contact of the cornea with the anterior pole of the lens, and so an arrest in the osmose of the nutritional fluids to it in that position. The contact when the opacity is congenital is due to delay in the formation of the anterior chamber which prolongs the apposition of these structures, normally existing during part of fœtal life, after the fibro-vascular sheath has disappeared. In infancy, when the anterior chamber is very shallow, contact may be brought about by the swelling of the cornea, which is the accompaniment of

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[8] Ibid., vol. vi., p. 138. [9] Ibid., p. 140.

inflammation of it, and need not necessarily be due to its perforation. Later in life the formation of anterior polar cataract is less common, and it would then seem that perforation of the cornea must occur before the lens can come into apposition with it. Mr. Hutchinson's objection to this theory may, I think, be met in this way. The immediate result of the arrest of the osmose of nutrient fluids through the capsule at the anterior pole is to cause the lens fibres in that region to shrink and to break up into hyaline globules and granular detritus ; as a consequence of their shrinking and degeneration, the tension of the capsule at the anterior pole is lessened. There is evidence to show that the only thing which keeps the epithelial cells that line the capsule from proliferating more quickly than they do is the tension to which they are exposed ; therefore, directly the tension at the anterior pole is lessened they commence to multiply at an increased rate and form the mass of cells which is the earliest stage of these opacities.

During the last few years the results of several investigations of minute anatomy of zonular cataract have been published. I have myself had seven lenses presenting this form of cataract for microscopical examination. Before entering into the changes I have found I will briefly review what has been written on the subject. Von Gräfe[10] and Jäger[11] found from section of lenses with zonular cataract which had been removed by extraction that there was an opaque whitish line separating a clear nucleus from a clear cortex. Deutschmann[12] was the first to examine a zonular cataract microscopically. He found the nucleus and cortex clear and unchanged, but that they were separated by a zone in which the lens fibres were altered by the presence of numerous vacuoles and small drops of myeline ; also that there were fissures between the fibres filled with granular detritus. The next observations into the microscopical characters of zonular cataract were made almost simultaneously by Beselin[13] in Germany and by Lawford[14] in this country. Beselin summarises the changes he found as follows : "Scattered throughout the nucleus of the cataract examined we find numerous small masses, which are apparently post-mortem coagulation products. This nucleus is surrounded by two oval zones of cataractous substance, neither complete nor continuous, which

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[10] Archiv für Ophthalmologie, Band i., p. 236.
[11] Staar und Staar-operationen, 1854, p. 17.
[12] Archiv für Ophthalmologie, Band xxxii.
[13] Archiv für Augenheilkunde, Band xviii.
[14] Royal London Ophthalmic Hospital Reports, vol. xii., p. 184.

are separated by a layer of normal lens tissue. These are, however, united here and there: both zones consist in greater part of large and small fissures, which are filled with detritus. In their immediate vicinity lens fibres are found, which are in process of cataractous degeneration. The cortex is composed of normal lens tissue; it presents a number of small fissures apparently accidental, which are filled with detritus, and at the periphery a number of small fissures may be seen, which were apparently produced artificially." Lawford, in the three specimens he examined, found in the cortical layers no changes of importance. The line of demarcation between the cortical and nuclear areas was abrupt, and between them he found a thin layer with breaks in it of granular substance; this he regarded as probably constituting the essential part of zonular cataract. In the nuclear area of all three specimens were a large number of small irregular dots or particles arranged more or less in layers, and which seemed to run concentrically, as if following the laminæ. He was inclined to regard these latter as coagulation masses, the result of the fluids employed in preserving the specimens. Otto Schrimer[15] found a similar dotted—or, as he termed it, "vacuolated"—condition of the nucleus to that discovered by Beselin and Lawford. He found, however, that the vacuoles were more numerous in what he regards as the zone of opacity than in its central portion. He does not agree with Beselin and Lawford that these vacuoles were produced by the hardening reagents, but considers that they are the essential cause of the opacity. Such a band of vacuoles, he points out, is just the very thing to produce an opacity in a lens, as the coefficient of refraction differs in the vacuoles and in the lens fibres; the fact that this layer was not sharply differentiated from the nucleus is no argument against his position, for there is nothing to prove that zonular cataracts have any such sharp line of demarcation. He subsequently[16] brought forward a specimen to show that vacuoles existed in a lens in the recent state; also that the layer of vacuoles corresponded exactly in size and position with the opaque zone seen before and after extraction. The clefts described by Beselin and Lawford he thinks are probably the striæ often seen in connexion with these opacities. Schrimer has further shown that central cataract is due to vacuoles or drops similar to those seen in zonular cataracts. Hess, who has examined five zonular cataracts, confirms

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[15] Archiv für Ophthalmologie, Band xxxv., p. 147.
[16] Ibid., Band xxxvi., p. 185.

Schrimer's results. The seven lenses which I have examined with what were described clinically as zonular or lamellar cataracts were all prepared in the same way. They were all hardened in Müller's fluid, passed through spirit and embedded in celloidin, after which sections were cut of them. They were all lenses which had been removed by the operation of extraction, after the capsule had been opened, from patients over the age of twenty-four years. In all of them some laceration of the extreme cortical layers had occurred. In three only was the transverse diameter of the opacity measured, in two of these it was 3·5 mm. across and in the other 5·5 mm. Microscopically three different kinds of changes are met with in these lenses; all three are not present in each lens, and the amount of change in each lens varies considerably. These changes are: First, fissures between the lens fibres, which may or may not contain a granular substance, they run concentrically to the nucleus separating it from the cortex. Secondly, small vacuoles, mostly round or oval, but in places, where they seem to have run into one another, elongated and beaded. Some of them contain a hyaline substance, which, after prolonged immersion in logwood, stains deeper with it than the surrounding lens fibres. The average size of these vacuoles is about 0·005 mm. across. Thirdly, spaces larger than the so-called vacuoles measuring on an average about 0·02 mm. across, mostly circular, with very irregular margins and containing a granular substance which stains deeply with logwood; apparently some degeneration of lens substance has occurred in their formation. These three changes correspond to the appearances seen clinically in these cataracts—viz., the radiating spokes or striæ, the uniform haze, and the denser dots. None of the specimens showed any changes in the cortical layers, which were not readily accounted for by laceration of them in the removal of the lens. In several of the specimens there was a great tendency for the nucleus to separate from the cortex and to drop out. In these it was impossible to say to what extent fissures between the nucleus and cortex existed prior to extraction. In some fissures existed containing no granular substance. In one the amount of granular substance was very great; it formed a band 0·15 mm. wide encircling the nucleus. The lens fibres bordering on this band were degenerate and had some of the small vacuoles amongst them. This specimen differed from all the others I have examined in that the nucleus showed no changes and was completely free from any vacuoles. In the appearances of the band of

granular substance, and in the absence of changes in the nucleus, it is very similar to that described by Deutschmann. In all the other six lenses small vacuoles were present in the nucleus; in most they were more numerous and closer together in a zone around the nucleus than in the nucleus itself; in two, however, they were distributed uniformly throughout the whole nucleus. If these vacuoles or bodies, as Schrimer suggests, are what give rise to the opacity, then these last two specimens were really central and not zonular cataracts. They were probably incorrectly classed clinically. It would seem that no very sharp line can be drawn between zonular and central cataracts—the same changes are met with in both, differing only in locality and degree. In the two lenses in which in addition to the zonular haze white dots of opacity were seen are the ones, and the only ones, in which I have found the third change mentioned, the larger circular spaces with granular contents. The position of these spaces, outside the zone of small vacuoles, corresponds to that in which the white dots appear to be situated.

Such, then, being the anatomical changes met with in zonular cataract, how are they brought about? Horner's view was that a pathological layer of lens substance is deposited about a normal nucleus. This he attributed to rickets, the convulsions met with in connexion with it being due to cranio-tabes, and the changes in the enamel of the teeth being also of rachitic origin. This view of the rachitic origin of zonular cataract has been widely accepted by writers on the Continent, but none of the text-books in this country mention it. Leber thought that a normally formed layer of lens substance became subsequently changed. Beselin and Schrimer differ from these authorities. The former asserts that alterations in the nutrition affect the whole nucleus, and that this change takes place when the subsequent nucleus constitutes the whole lens, the general contraction of the affected part causes the formation of fissures between it and the cortical layers of normally formed lens substance which are developed later, and that a granular substance subsequently collects in these fissures. Schrimer's theory is as follows: "Some unknown disturbance — probably in its nutrition—affects the lens so as to produce vacuoles in its substance. These may be at first in the fibres, perhaps, but later on between them certainly. Only the newest lens fibres and those formed during the action of the noxious agent contain enough vacuoles to exhibit a visible opacity. The vacuoles in the nucleus are not enough to produce this appearance; but, though the nucleus remains transparent, it

has altered in its physical properties so that it shrinks more rapidly than the subsequently formed cortical portion is able to do, and clefts are thus produced in the lens." Many regard zonular cataracts as really congenital, and a recent paper by Bernard Dub[17] brings forward evidence in favour of this view. The table of measurements of fœtal lenses I have put before you has been used by him, together with measurements of adult lenses by Priestley Smith, and a series of measurements of lenses from the ages of ten months to twelve years made by himself for the purpose of determining the time of life at which zonular cataracts originate. To do this he has compared the measurements of the lens at different ages with those of the opaque zones in a series of zonular cataracts. His method of effecting the measurement of these latter is very ingenious. It is described as follows: "If the left eye be the one affected, a millimetre scale is placed about the patient's left temple behind the outer canthus. The observer illuminates the fundus of the eye by an ophthalmoscope held before his own right eye, and keeps his left eye closed; then, opening his left eye, he squints inwards, so as to fix the scale with the left eye, while the right still observes the cataract against the red background of the illuminated fundus. The position of the scale is such that the homonomous diplopia thus evoked makes the image of the cataract seen by the right eye coincide with that of the scale seen by the left, and the only thing further required is to make the correction necessary to neutralise the enlargement produced by the magnifying power of the patient's cornea. Dub assumes this to be one-seventh (on Helmholtz's authority), and corrects his figures accordingly." As the result of his measurements, he concludes that the commonly received views that zonular cataracts begin in the first two years or so of extra-uterine life, and that the peripheral fibres are the parts affected, cannot both of them be true. This paper gives in figures drawn from a series of cases the views which were expressed by Mr. Hulke[18] in his Presidential Address to the Ophthalmological Society in 1886. He then said: "With reference to cataract in early life, it is a question with some at what age the lamellar form makes its appearance, or rather some excellent observers have formed the opinion that it is infantile, not congenital. I am not convinced by the arguments used by the supporters of infantile origin. The opaque zones are clearly laid down

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17 Ibid., Band xxxvii., p. 26.

18 Transactions of the Ophthalmological Society, vol. vii., p. 27.

during the development of the lens, and instances of zonular cataract occur where the diameter of the opaque zone is certainly less than that of the entire lens at birth." He further goes on to say that the first recognition of lamellar and zonular cataract based on dissection was, so far as he knew, made by Mr. Bowman, the subject being a kitten a few days old.

A number of direct measurements have now been made of the opaque areas in zonular cataract. The following table gives the transverse diameters of ten such, together with the measurements in Dub's ten cases.

Table II.—Showing Direct Measurements of the Transverse Diameters of the Opaque Areas in Ten Zonular Cataracts, with Dub's measurements in ten cases.

Direct Measurements.	mm.	Dub's Measurements.	mm.
Beseline	6	.....	1. 4·4
Lawford (1)	5·25	.....	2. 4.6
Lawford (2)	4·5	.....	3. 4·7
Lawford (3)	4·5	.....	4. 4·8
Schrimer (1)	5·75	.....	5. 4·8
Schrimer (2)	3·25	.....	6. 5
Schrimer (3)	6	.....	7. 5·2
Collins (1)	3·5	.....	8. 5·2
Collins (2)	3·5	.....	9. 5·5
Collins (3)	5·5	.....	10. 5·6

The average transverse diameter of three lenses at the ninth month of fœtal life I found to be 5·75 mm., and Dub found the average of three lenses under a year old to be 7·46 mm. So it would seem from these direct measurements that the zone of opacity in zonular cataract is probably never larger than the lens at birth, that it may be about the size of it at that time, but is more often smaller, and sometimes very much smaller. The direct measurements differ slightly from Dub's and show a greater variety in size, in three the zone being larger than his largest, and in three smaller than his smallest. In the smallest there is so much difference between the size of the zone and that of the lens at birth that I think it hardly possible to believe that the opaque area was the extreme cortex at the time of birth, and that the diminution in size is the result of shrinking. We must, then, either conclude that zonular cataract may be produced before birth or that the part affected, when affected, is not the most peripheral, as has generally been supposed. It is, I think, conceivable that as

the result of some general disturbance of nutrition the lens might become affected after birth, so that the nucleus, which is furthest removed from the nutrient supply, would shrink. Shrinking of the nucleus is what seems to occur first in the formation of zonular cataract, fissures, vacuolation, and degeneration following between it and the cortical layers from which it contracts. (In support of this view the lecturer mentioned several cases in which zonular cataract had occurred as the result of what may be called purely intra-ocular causes.)

There seems no evidence of any connexion between zonular cataract and congenital syphilis. The defective condition of the enamel of the teeth so frequently associated with the former is, as Mr. Hutchinson pointed out, quite different from the notched screwdriver condition found in the latter disease. Mr. Hutchinson[19] differs from Horner in attributing zonular cataract and defect of the enamel of the teeth to rickets; he considers the latter more probably due to a stomatitis from the administration of mercurial powders given for fits. He says: "In reference to the suggestion of the Zurich investigators that the peculiarities of the teeth, in the skull bones and in the general development, are all due to rickets, I must be allowed to say that it is as yet wholly unproved. I am not aware that any author has described what are so freely spoken of as 'rachitic teeth,' if that term be applied, as it is by Dr. Davidson, to the permanent set. Professor Vogel of Dorpat, in reference to the effects of rickets on the teeth, says that, 'as the disease disappears before the second dentition commences, these phenomena are not observed in the permanent teeth.' Yet it is upon the state of the permanent teeth almost solely that the diagnosis of rickets in the Zurich clinique is based; for in only 4 per cent. did Professor Horner find evidence of rachitic malformation in the extremities. The irregular formation of the skull, defects in symmetry of the face, and mental peculiarities when present are perhaps quite as easily explained by reference to the attacks of convulsions as by the hypothesis of rickets." Though, as Mr. Hutchinson says, it is not proved that rickets affects the permanent teeth, still it is very possible that it may do so. The germ of all the enamel organs of the permanent set are present at birth, and just those teeth which are most frequently affected—namely, the first molars, the incisors, and the canines—are the ones in which calcification commences during the first two years of life—i.e., at the time of the initiatory stage of rickets.

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[19] Brit. Med. Jour., vol. i., p. 308.

It occurred to me that possibly some evidence as to the etiological relation of rickets and lamellar cataract might be obtained from our Australian colonies, where I was informed rickets was a rare disease. I wrote, therefore, to my friends, Dr. Barrett of Melbourne and Dr. Symons of Adelaide, asking them for any information they could obtain for me on the subject. The result of these inquiries I recently communicated to the Ophthalmological Society. It may be briefly summed up as follows. In Adelaide rickets is a rare disease, and lamellar cataract very infrequent. In Melbourne rickets was until recently comparatively rare, that it is more common now, but that the severity of the affection is much less than in the Old World; lamellar cataract is exceedingly rare, and the honeycombed condition of the enamel of the teeth is not often met with. In Sydney, the oldest city in Australia, rickets is said to be as common as in England, but I have no statement as to its comparative severity. Lamellar cataract is less frequent than in this country. In Persia, a country I have recently visited, I found both rickets and lamellar cataract very rare diseases.

LECTURE II.

Delivered on Dec. 5th, 1894.

MR. PRESIDENT AND GENTLEMEN, — The late Lord Sherbrooke was an albino. During the latter part of his life he became affected with cataract, and an operation of extraction with iridectomy was performed on one of his eyes. The surgeon who performed the operation brought the piece of iris to the laboratory at the Moorfields' Hospital for examination. It was thought it might show well the arrangement of the epithelium on the posterior surface of the iris, which in the normal eye is densely pigmented. The specimen was a mere fragment, and its preparation was not satisfactory ; but its examination suggested to me the possibility of depriving sections of the eye of their pigment, and so allowing of a more accurate examination of the pigmented epithelium. The best way I found to effect this was to place the section from an eye which had been previously embedded in celloidin, first, into chlorine water, which can be readily made as required by soaking chloride of lime in water and filtering, then into water acidulated with hydrochloric acid (one minim to one drachm). The sections should be passed backwards and forwards between these two solutions until the pigment quite disappears, then after thoroughly washing in several changes of water they can be stained with logwood. The manipulation of the sections requires considerable care, as, after exposure for some time to the chlorine and acid, they have a great tendency to come to pieces—the substance of the cornea peeling off Descemet's membrane, the choroid separating from the uveal pigment layer, and so forth, in spite of the embedding celloidin. Mr. Griffith has recently improved on this method, and has succeeded in bleaching portions of the eye *en masse*. A section of the iris which has been bleached shows quite distinctly that there are two layers of epithelium on its posterior surface. The anterior of these is continuous with the pigmented layer on the inner surface of the ciliary body and the posterior with the unpigmented layer or pars ciliaris retinæ. At the pupillary border of the iris these two layers unite, this union corresponding developmentally with the junction of the inner and outer layers

of the secondary optic vesicle. The anterior layer consists of a single row of flattened nucleated cells. The posterior layer is the most deeply pigmented portion of the whole eye and the last to lose its pigment in the bleaching process; the cells in it at the root of the iris are elongated and wedge-shaped, the bases of the wedges being directed backwards, the result being that the posterior surface of the outermost part of the iris has a convoluted outline, the cells being arranged in little groups with depressions between them. On tracing the iris inwards these depressions become much shallower and separated further apart. From about its centre to the pupillary border it presents a smooth surface, the cells here being of a regular columnar shape. Throughout this layer the nuclei of the cells are situated in their posterior halves. At the pupillary border the cells in the anterior layer are not so flattened and are more quadrate in shape. It is this increase in the size of the cells in the anterior layer which makes the pigment on the posterior surface of the iris thicker at the tip than in the centre. The posterior layer of cells may be termed the pars iridis retinæ; and as in the hinder parts of the eye the two layers of the secondary optic vesicle become separated in detachment of the retina, so in the anterior part they may become separated and a space be formed between the two layers of pigment on the back of the iris. Slight detachments of this sort are seen frequently in various pathological states, especially in eyes of youthful patients. It is of more frequent occurrence than detachment of the whole of the pigment epithelium from the stroma of the iris, for the anterior layer, like the pigment epithelium in the posterior parts of the eye, has firm connexions to the mesoblastic structures overlying it. An effusion between the two pigment epithelium layers, when extensive and localised, will produce a cyst-like tumour of the iris. When not localised, extensive and following iritis, with occlusion of the pupil, a condition indistinguishable clinically from what is known as "iris bombé," is produced. The surgeon in performing an iridectomy in such a case removes the stroma of the iris and anterior layer of pigment, and then is probably surprised to find the coloboma still filled up with a layer of pigment, the posterior pigment epithelial layer. The pigment epithelium terminates usually at the pupillary margin of the iris; in certain pathological conditions it is continued round it on to the anterior surface, constituting what is known as ectropion of the uvea. This is most commonly met with in cases of glaucoma of some stand-

ing, and appears to be due to a shrinking of the iris, which ensues from the pressure of its root against the periphery of the cornea. The shrinking affects the anterior part of the iris more than the posterior, consequently the latter gets drawn round. It is not uncommon to find the ectropion of the uvea more in one part of the circumference of the pupil than in another. In such cases the iris is most shrunken where the ectropion is most marked, and where the root of the iris is most firmly adherent to the periphery of the cornea. The shrinking of the iris more in one part than another is rendered evident by an eccentric position of the pupil. As the result of these observations we may formulate the following rule, which is of some practical utility in operating for glaucoma—that when in a case of glaucoma the pupil is eccentric, and there is ectropion of the uvea, the iris is likely to be less adherent to the periphery of the cornea, in the position opposite to that to which the pupil is displaced, and where the ectropion of the uvea is most marked than elsewhere. This is, therefore, the most favourable position in which to do an iridectomy in order to remove the iris up to its extreme periphery.

Ectropion of the uveal pigment, besides occurring as the result of glaucoma, is sometimes also a congenital condition, and may give rise to a raised pigment nodule at the pupillary border. The arrangement of the pigment epithelium at the pupillary margin of the iris of a horse is very peculiar. The pupil of the horse is oval in shape, and projecting from its margin on one side is an irregular, nodulated, deeply pigmented mass, continuous with the uveal pigment layers on its posterior surface. In the eyes I have dissected this mass measured on an average 8 mm. in length, 3 mm. in width, and 3·5 mm. in height. In unbleached sections of it little can be made out of the arrangement or shape of the cells of which it is composed. It is very cavernous, and bloodvessels are seen coursing through it, continued on from those in the stroma of the iris. Bleached sections show the mass to be made up of flattened epithelial cells, which line and form columns between the numerous and variously sized circular spaces in it. Bleached sections of the human eye show that the pigment epithelial layer from the optic disc up to the ora serrata consists of but a single row of cells. In the ciliary body, protruding from this layer towards the ciliary muscle, are numerous little processes or down-growths, each consisting of a group of cells. These down-growths vary in number and shape in different portions of the ciliary body. The two parts where they are most numerous are just in

front of the ora serrata and at the junction of the plicated and non-plicated parts. In the plicated part there are but very few of them, and those that exist are met with in the recesses between the ciliary processes. Most of the down-growths at the junction of the plicated and non-plicated portions are pear-shaped, the bulbous end of the pear projecting towards the ciliary muscle. Those in front of the ora serrata project outwards and slightly backwards; they are shorter and broader than the others, and some of them can be distinctly seen to consist of two rows of cells. By the examination and comparison of a number of bleached and unbleached sections of the ciliary region cut on the flat I have been able to distinctly make out that the cells of these projecting processes are arranged in rings with a central lumen.

[The arrangement of the pigment epithelium of the ciliary body in the ox, horse, and chimpanzee was shown to present similar down-growths to those which exist in man.]

Many ingenious experiments have during the last few years been performed for the purpose of determining the source of the aqueous humour and of the nutrient fluids of the lens and vitreous. That the aqueous was secreted by some part of the walls of the posterior chamber seemed likely by its accumulation behind the iris when the pupil became closed either by a complete ring of posterior or anterior synechiæ. It seemed improbable that it came from the iris, for in cases of aniridia, either congenital or traumatic, no alteration in its constituents or in its amount was noticed. Deutschmann[1] excised the iris and ciliary body from the eye of a rabbit, and found that the secretion of the aqueous was arrested, and that the vitreous shrank. Schoeler and Uhthoff,[2] adopting a method of subcutaneous injection of a solution of fluorescine originally suggested by Ehrlich, found that from four to ten minutes after such an injection the colouration could be detected coming through the pupil into the anterior chamber. On dissection of eyes excised at various periods after subcutaneous injection they found that simultaneously with, or immediately after, the colouration of the pupil some of the furrows between the ciliary processes were tinged with green. These green lines grew narrower in the direction of the equator of the eye and ended at the posterior limit of the processes; there was also a gradual colouration of the vitreous body proceeding from

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[1] Archiv für Ophthalmologie, Band xxvi., p. 117.

[2] Jahresbericht über die Wirksamkeit der Augenklink, p. 52. Von Prof. Schoeler.

the ciliary processes and passing backwards. In an eye in which the ciliary processes had become atrophied or destroyed in consequence of previous cyclitis no colour entered the anterior chamber through the pupil, and on section the atrophied processes were almost entirely destitute of colouration.

These experiments seem to prove that the aqueous and nutrient fluid of the vitreous are secreted in the ciliary region. They are confirmed by those of Leplat,[3] who injected iodide of potassium subcutaneously, and after enucleation cut up the frozen eyeballs into zones, testing each zone quantitatively for iodine with starch. In the *Archives d'Ophthalmologie* for 1891 there appeared a series of articles by M. Nicati, in which he claims to have established, on histological, physiological, and pathological grounds, the existence of a glandular apparatus for the elaboration of the aqueous humour. His histological researches refer to rabbits' eyes, and he speaks of the pars ciliaris retinæ as the gland of the aqueous humour. These experimental researches go to show that it is in that part of the eye in which these little tubular down-growths of cells exist that the aqueous humour and the fluid which gives nutriment to the vitreous are secreted. These are the only fluids which are known to be poured into the eye. These tubular downgrowths open towards its interior, being only separated from the close vascular plexus of the ciliary region by a basement membrane. The inference that they are glands concerned in the elaboration of the aqueous humour and nutrient fluid of the vitreous seems to me irresistible. There is no evidence at present to show to what extent they are concerned in it; that they are the sole source of these fluids seems improbable, from their absence in rabbits' eyes and from the fact that epithelium similar to that lining the glands covers the rest of the ciliary processes. I first pointed out the existence of these gland-like processes of the pigment epithelium of the ciliary body in March, 1891.[4] Greef, who has since written on the subject, seems to think I referred to the recesses between the ciliary processes in speaking of glands, but can only have done so by failing to have read carefully the description I gave of them. Mr. Griffith,[5] in a criticism of my work read before the British Medical Association this year, agrees that the anterior group of processes are glandular in character, but considers that the posterior—those situated

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[3] Annales d'Oculistique, vol. xcviii., p. 89.
[4] Report of the Heidelberg Ophthalmoscopical Society, 1893.
[5] Ophthalmic Review, vol. xiii., p. 247.

in the non-plicated portion of the ciliary body, which he finds are not always present—are really only rucks in the pigment epithelial layer due to alterations in the state of contraction of the ciliary muscle. The objections to this view of their nature are: (1) that when present they are distinctly tubular; (2) that there are no half-stages between a distinct tube and a single row of cells; and (3) that I have found them present in some and absent in other perfectly healthy eyes, hardened and prepared in precisely similar ways. I am at a loss to explain their absence in some eyes and presence in others. The anterior group of pear-shaped processes is always present. If they be glands we should expect to find them subject to the same diseases as secreting tubular glands in other parts of the body. We should expect to find them subject to catarrhal and suppurative inflammation; we should expect to find overgrowth of them in cases of chronic inflammation; we should expect them to be subject to cystic formation, and for them to be the starting points of tumours presenting a glandular character, either adenomata or carcinomata. I will now proceed to show that these expectations are actually fulfilled.

The disease, which was originally described by Wardrop in 1808 as aquo-capsulitis, and which is now generally spoken of as serous iritis, is, I believe, primarily catarrhal inflammation of the ciliary body and of these glands. The whole course of the disease points, I think, in this direction. I would interpret its symptoms as follows. It commences with congestion of the bloodvessels around the glands, which manifests itself as circumcorneal injection. This is not associated with pain and photophobia, as usually occurs in iritis. The secretion from the glands becomes augmented, causing increase in the aqueous humour and deepening of the anterior chamber. It also becomes altered in character, more albuminous than usual, and contains a few leucocytes, some pigmented epithelial cells which have desquamated from the surfaces of the glands, and some shreds of fibrin. These formed elements tend to gravitate to the lower part of the anterior chamber and to be deposited on the posterior surface of the cornea, forming the spots of "keratitis punctata," some of which are often pigmented, the pigment being derived from the desquamated cells above mentioned. Some of the leucocytes also collect in the meshes of the ligamentum pectinatum; this, together with the alteration in the character of the fluid, tends to hinder its exit from the eye, and, combined with the excessive secretion, readily leads to increase of tension, which is only temporarily relieved by iridectomy or para-

centesis. As a result of these changes in the composition of the aqueous it becomes slightly turbid, which turbidity produces an alteration in the colour of the iris. This latter is not at first affected, and there is no tendency to the formation of synechiæ until in the later stages, when it has become secondarily involved. The vitreous is early noticed to contain flocculi. The course of the disease, as in all catarrhs, is very variable, and there is great tendency to relapses. Severe cases end in disorganisation of the structures which receive their nutrient fluid from these glands. Thus the vitreous shrinks, the lens becomes cataractous, and a condition of phthisis bulbi results. The histological appearances of sections from eyes affected with so-called serous iritis are quite in keeping with this theory of its pathology. There is some increase in the size of the glands, some irregularity and proliferation of their epithelium, enlargement of the bloodvessels in their vicinity, and a variable amount of round-celled infiltration about them. At the stage in the disease at which eyes are usually obtained for histological examination inflammation is not confined to the ciliary body, but has spread throughout the uveal tract. In the eye of a boy aged nine years, in whom the symptoms of this disease followed a wound from a knife, which was excised eighteen days after the injury, I found considerable cell accumulation between the ciliary muscle and the pigment epithelium, just in the region where these glands are situated. The dotted opacities on the back of the cornea are frequently spoken of as "keratitis punctata," a term which is of course inappropriate if this theory of their formation be correct, for they are not caused by an inflammation of the cornea but are deposits of inflammatory products on its posterior surface. In teased specimens of these deposits, besides round cells, pigment epithelial cells similar to those in the tubular down-growths of the ciliary body are occasionally found. The term "Descemetitis" for the condition in which these opacities are found is also inaccurate. I have a section of a cornea with dotted opacities on its posterior surface in which the endothelium lining Descemet's membrane can be traced continuous and unaltered in front of the accumulations of round cells. It is interesting to notice how a supposed analogy between the aqueous chamber and the large serous sacs of the body, such as the peritoneum and pleura, led to what for the patient must have been a very disagreeable form of treatment—viz., the internal administration of the oil of turpentine. Wardrop, Jacob of Dublin, and others thought the aqueous chamber to be a closed sac lined

throughout with endothelium ; this of course it is not, for posteriorly it communicates through the circumlental space with the vitreous, and the anterior surface of the lens capsule has no endothelial cells on it. Moreover, the lining membrane on the back of the cornea is unlike that of a serous cavity, which consists essentially of a layer of endothelial cells with bloodvessels external to them. Perhaps the most striking difference between the aqueous chamber and the serous sacs is that the former is a cavity containing fluid, while the latter in the normal state are only potential cavities whose walls are lubricated with fluid. Dr. Carmichael of Dublin, relying on this supposed analogy and on the apparently beneficial effect which turpentine administered internally had on cases of peritoneal inflammation, recommended the administration of the same drug when the supposed lining serous membrane of the aqueous chamber was the seat of disease. This treatment is still prescribed by some surgeons even at the present day. Besides the glands and secreting area of the ciliary body being primarily affected by a catarrhal inflammation, they may also become secondarily involved by inflammation starting in other structures of the eye ; or in the course of a general disease in which the uveal tract is implicated the ciliary body may be the first part to be inflamed. In sympathetic ophthalmitis the early symptoms are similar to those described as typical of "aquo-capsulitis" or "serous iritis." The most striking and constant change revealed by the microscope in eyes affected with sympathetic ophthalmitis is the presence of groups of round cells scattered throughout the uveal tract. It can be easily understood that such an inflammatory nodule occurring in the situation of the glands of the ciliary body would cause the exudation of inflammatory products from them, which would be partly carried forwards into the anterior chamber and partly effused into the vitreous. In eyes affected with sympathetic ophthalmitis I have frequently found nodules of cells of this character thus situated. In the same way, should one of the nodules of round cells which occur in syphilitic uveitis be located in the glandular region of the ciliary body a like effusion would be expected and similar symptoms. Dots on the back of the cornea, deepening of the anterior chamber, and opacities in the vitreous are often met with in syphilitic affections of the eye.

In suppurative inflammation of the eye it is quite common to find purulent infiltrations of the vitreous obviously starting from the ciliary body, and pus cells can be traced forwards from the latter, between the iris and lens, into the anterior

chamber, where an accumulation is seen at the lower part, constituting a hypopion. When inflammation of the ciliary body is of a plastic character the inflammatory bodies effused into the vitreous organise into fibrous tissue, and a band of fibrous tissue forms behind the lens across the ciliary region. This is spoken of as a cyclitic membrane; to it the detached retina is frequently adherent. In such cases there is often considerable overgrowth of the pigment epithelium of the ciliary body. This overgrowth presents a characteristic arrangement of its cells in the form of cylindrical gland-like tubes. Just as in chronic inflammation of other glandular surfaces hyperplasia of the glandular structure occurs, so here. I cannot do better than quote a description of this condition which has been given by Alt,[6] and with which my own observations quite coincide. Speaking of plastic cyclitis and cyclitic membranes, he says: "Meanwhile the cells of the uveal layer have also undergone proliferation. The whole layer appears very much thickened, and grows into the cyclitic membrane in an irregular way. We also observe, however, frequently a more typical kind of proliferation of the cells of the uveal layer in the shape of cylindrical tubes, which grow into the cyclitic membrane and give off branches. In longitudinal and transverse sections these tubes appear like the glandulæ tubulosæ, or the epithelial cylinders of epithelioma. The cells of these tubes are either free of pigment or pigmented. Their shape and arrangement with their branches have given some writers (Schieff-Gemusens) the idea that they were bloodvessels whose walls were filled with pigment. Specimens in which the bloodvessels have been injected with a coloured fluid, however, plainly show that they are different from bloodvessels. They appear, as stated, just like glands or epithelioma cylinders. Not all the cells originating by proliferation from the uveal layer are pigmented, and I am even convinced that the young cells of this layer are at first always unpigmented and form their pigment only later on. For this reason we find in cyclitic membrane the same tubular formations without any pigment at all, which can nearly always be traced backwards to a pigmented cell tube or the uveal layer itself." He gives woodcuts showing these cylindrical tubes.

It is by no means uncommon to meet with cystic spaces in the region of the ciliary body in eyes affected with very different diseases. Many of these spaces are due to the

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6 Lectures on the Human Eye, p. 106.

detachment of the pars ciliaris retinæ from the pigment epithelium. Brailey,[7] writing on this subject, says: "I have found even in the course of my usual microscopic examination the pars ciliaris retinæ detached from the subjacent pigment layer in about 10 per cent. of the cases of primary glaucoma. These detachments are frequently so localised as to give the appearance of little fluid-containing cysts, but sometimes they are of considerable extent. Very similar changes are found in association with the cyclitis which accompanies the disease, known clinically as iritis serosa. In this condition, however, the cysts contain some corpuscular elements." Kuhnt[8] mentions that he has frequently found cysts in the ciliary body due to the detachment of the pars ciliaris retinæ from the pigment epithelium as a senile change in eyes which have been altered by atrophic processes. Cysts due to the distension of the small tubular glands which I have found to exist in the ciliary body, should they occur, would differ from those due to the detachment of the pars ciliaris retinæ from the pigment epithelium in being bounded entirely by pigment cells. I have one specimen from an eye which had had cyclitis showing such cysts. Alt, in his book entitled 'Lectures on the Human Eye,"[9] mentions and pictures what he calls "a tumour-like new formation of pigmented and unpigmented cell cylinders, starting from the uveal layer of the ciliary body and neighbouring choroid." The woodcut shows very distinctly tubules lined by epithelial cells, cut transversely and longitudinally. This tumour-like new formation growing in the region where I find these tubular glands I regard as an adenoma.

Professor Michel[10] removed the right eye of a woman aged forty-one years who had always enjoyed good health for what was supposed to be a melanotic sarcoma growing from the posterior surface of the iris and invading the ciliary body. Pathological examination of the eyeball showed that the growth, which was grey in colour, sprang from the ciliary processes. Microscopically the iris and ciliary muscle were seen to be quite free from any pathological formation, and the growth was found to consist of a mesh of connective tissue, in some of the spaces of which there were rows of cylindrical cells, arranged, he says, so as to present the appearance of a tubular gland. He concluded by describing

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[7] Royal London Ophthalmic Hospital Reports, vol. x., p. 384.
[8] Klinisches Monatsblatt, 1881.
[9] Ibid., p. 123. [10] Archiv für Ophthalmologie, vol. xxiv., p. 140.

the growth as endothelial and epithelial cancer. Amongst the specimens preserved in the laboratory of the Royal London Ophthalmic Hospital, Moorfields, I found an eye removed from a woman aged sixty-three years, who twenty-five years previously had had a severe blow on it from a fist, and two years subsequently had found that it was blind. It gave her no trouble until nine weeks previous to enucleation, when pain and inflammation set in. Except for the eye trouble she was in good health, and continued so for twenty-three months after its removal; since that time she has changed her address, and I have been unable to trace her. The eye contained a growth which was partially deeply pigmented, and sprang from the ciliary processes, invading the ciliary muscle and root of the iris. It was originally described as a sarcoma which had undergone mucoid degeneration. It was afterwards re-examined and shown by Mr. Solly at a meeting of the Pathological Society of London on April 15th, 1891, as "a melanotic growth from the eye which appeared to be epithelial." I have cut some more sections from this tumour and bleached them; there can be no doubt of the epithelial character of the cells. In some parts of the tumour they have undergone colloid degeneration; while in others, especially towards its base, which is the most pigmented part, they are grouped in parallel columns, cut in various directions. This growth I regard as a melanotic glandular carcinoma. As melanotic carcinoma is practically unknown in other parts of the body and it is extremely unlikely that a primary non-pigmented growth would give rise to a secondary deeply pigmented one, and as the patient made no complaint of any other growth during the two years she was under observation, I think there can be no doubt that the tumour was a primary one of the ciliary body. In March, 1893, Messrs. Badal and Lagrange[11] recorded a case which they described as a primary carcinoma of the ciliary body. The patient under their care was a boy aged eight years. A peculiar appearance was noticed in his left eye soon after birth. When about five years old it became quite blind, and shortly afterwards commenced to enlarge. At the time of excision it was injected, there was a staphylomatous condition in the ciliary region, and by oblique illumination a rose-coloured reflex was obtained. A year after excision the child was in good health, and there was no sign of recurrence. Pathological examination revealed a new growth confined to the ciliary region and consisting of

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[11] Archives d'Ophthalmologie, 1892.

two unequal whitish nodules placed alongside each other. Microscopically the growth was seen to be composed of (1) regular tubules with a central lumen lined by a single layer of cylindrical epithelium ; (2) similar tubules, filled with proliferating epithelial cells ; and (3) collections of atypical and deformed epithelial cells grouped and separated by thin strands of connective tissue. At the December meeting of the Ophthalmological Society in 1893 I showed a primary tumour of the ciliary body exhibiting glandular structure. It was removed by Mr. Rockliffe, who sent it to me for examination. The patient was a woman aged twenty-eight years ; for six months she had noticed failure of vision in the eye, but it had only recently become inflamed and painful. Her health otherwise was quite good, and she had no symptoms of growths in any other part of the body. On section of the tumour the anterior part of it was seen to be deeply pigmented ; the posterior part was devoid of pigment. Microscopically, the cells composing the central portions of the growth appeared very degenerate ; they were much swollen and their outlines ill-defined. The more peripheral parts were less degenerated ; the cells here were of epithelial character, and arranged in a way suggestive of a glandular structure.

To sum up, then, I find that there are situated in the region which experiments have proved to be the part from which this aqueous humour and nutrient fluid of the vitreous are secreted numerous little tubular processes of epithelial cells, which I can imagine to be nothing else than glands concerned in their elaboration ; that the nature of these processes has been hidden by their pigment, and is only rendered evident in bleached sections ; that these glands and other secreting cells of the ciliary body are, like secreting structures elsewhere, subject to attacks of catarrhal inflammation, which gives rise to the group of symptoms generally included under the term "serous iritis" ; that as the result of chronic inflammation there may be considerable overgrowth of their tissue, which overgrowth preserves a glandular type ; that they may become distended into little cysts, and that they may be the seat of tumours, either adenoma or glandular carcinoma. The development of the retina is a subject of great intricacy and one which requires further investigation. I have not myself made any special study of it, but in the examination of microscopical sections of human fœtal eyes I have been struck by the fact that in those about the third month its layers are quite undifferentiated, that it is then composed entirely of a mass of round cells with large nuclei and scarcely any cell protoplasm, some

of them, however, having little spicules projecting from them. These cells closely resemble those which are met with in gliomatous tumours of the retina. So closely, indeed, is this resemblance that I feel sure that if a pathologist were shown a piece of fœtal retina in which the layers were undifferentiated under the microscope, and was told that it was a piece of a tumour, he would have no hesitation in pronouncing it a glioma of the retina.

[Mr. Collins then pointed out that there were great clinical, histological, and embryological differences between gliomata and sarcomata, and suggested that, as sarcoma represents the fœtal condition of the tissue in which it primarily grows, so glioma of the retina represents the fœtal condition of the retina.]

In an early stage in the development of the eye the mesoblastic tissue which surrounds the secondary optic vesicle consists of a mass of cells, there being no differentiation between the sclerotic and choroid coats. Later, in the part which is to become choroid the nuclei stain deeper and blood-vessels commence to appear. The cells of the part which is to form the sclerotic are gradually lengthened out and differentiated into fibres. The tissue between the two coats early becomes spaced out to form the lamina supra-choroidea and lamina fusca. The pigment in the branched cells of the choroid can be detected about the seventh month, but does not reach its maximum until after birth. It is exceedingly difficult to determine the exact date of the commencement of the elastic lamina or membrane of Bruch. I have certainly been able to make it out as a very thin layer as early as the sixth month. Its origin and mode of formation cannot be determined by the examination of embryological specimens alone. As in the case of the lens capsule, I think some valuable suggestions may be drawn with regard to it from a study of some of the pathological processes which occur in connexion with it. It is exceedingly common to meet in pathological specimens of the eye with little nodules of a hyaline substance on the inner surface of the elastic lamina, which present all the physical characters of that membrane. These hyaline or colloid bodies were originally described as a senile change, but they are met with in eyes which have been blind for some time from patients of any age and often quite apart from any signs of inflammation. Various opinions have been expressed as to their origin ; these may be grouped under three heads—viz., (1) that they are thickenings of the hyaline membrane (Müller and De Wecker) ; (2) that they are due to a hyaline

degeneration of the pigment epithelial cells lining the elastic lamina (Donders, De Vicenten, and Dimmer); and (3) that they are an excretion from the pigment epithelial cells (Leber and Meyer). *A priori*, it would seem more probable for these hyaline nodules to be the product of epithelial cells rather than outgrowths from an inert basement membrane like he elastic lamina. Dimmer has pointed out that if they were only thickenings of the elastic lamina we should expect to find them extending outwards towards the choroid as well as inwards towards the retina, which we do not. Their presence always on the inner surface of the elastic lamina is readily understood if they are regarded as products of the pigment epithelium. That they are not outgrowths of the elastic lamina seems to me conclusively proved by the fact that they may be found where no elastic lamina is present. Thus, in a specimen I have examined of a microphthalmic eye which had a cyst connected with it, ill-developed retina was found lining the cyst, but no choroid; in the retina, however, there were hyaline bodies presenting all the characters of those under consideration. Hess[12] also found bodies of a similar character in the lining membrane of cystic protrusions of the sclerotic, where no choroid was present, in two pigs' eyes. That they are the product of epithelial cells I think there can be no doubt; whether they are formed within the cells themselves or are an excretion from them cannot be so conclusively determined.

I have bleached several sections of eyes containing these hyaline nodules, thinking that when the cells were deprived of their pigment it would be easy to detect the presence of degenerative changes in them. The cells, which are often much flattened, are always outside the hyaline nodules, several of them often completely encircling the latter. The usual form of these hyaline or colloid bodies is that of little globules, the largest not so big as a pin's head. Occasionally, however, on the inner surface of the choroid much larger masses are met with, which are composed of layers of hyaline substance, with here and there flattened pigment epithelial cells between them. The appearance presented suggests that of numerous layers of the membrane of Bruch closely packed together. Such masses may occasionally be seen ophthalmoscopically as raised white elevations over which the retinal vessels course. I have known them diagnosed as sarcoma and tubercle of the choroid, the eyeball being enucleated accordingly. Their extreme chronicity is usually

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[12] Archiv für Ophthalmologie, Band xxxviii., Ab. 3.

sufficient to distinguish them from these affections. Their position between the retina and choroid, the disturbance of the pigment epithelial cells around them, and the presence of some of these cells between the layers, all seem to point to the hyaline substance being the product of the pigment epithelium. Now, if it is possible for the pigment epithelium under the influence of a morbid stimulus to produce a substance which stains and reacts to reagents in precisely a similar way to the elastic lamina itself, it is reasonable to suppose that that membrane is originally produced from them, just as the hyaline capsule of the lens appears to be the product of the cells which line it. Additional evidence of this view of the origin of the elastic lamina is afforded by a case of cyst connected with a microphthalmic eye recorded by Rindfleisch,[13] in which the lining membrane of the cyst consisted of retina with well-developed layers, pigment epithelium, and an elastic lamina, but no choroid. If the elastic lamina is derived from the pigment epithelium it is epiblastic in origin, and should, strictly speaking, be classed as a layer of the retina and not of the choroid.

The choroid in places sometimes fails to develop—i.e., the mesoblastic tissue which surrounds the secondary optic vesicle fails to become differentiated into choroid and sclerotic. The inner layers fail to become vascularised or only a few large vessels form. It would seem that when this failure in vascularisation occurs the outer layer of the secondary optic vesicle in the part non-vascularised does not become pigmented, and at the margin of the patch there is often an excess of pigmentation. These patches of failure in the differentiation of the choroid give rise to what are known as colobomata. They occur, of course, most frequently in the region of the fœtal ocular cleft, but may be met with, as pointed out by Lindsay Johnson,[14] in any part of the fundus.

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[13] Ibid., Band xxxvii., Ab. 3, p. 192.
[14] Archives of Ophthalmology, vol. xix.

LECTURE III.

Delivered on Dec. 7th, 1894.

MR. PRESIDENT AND GENTLEMEN,—The pathology of glaucoma may not inaptly be compared to a large child's puzzle with many pieces of various and irregular shapes. Its framework has by the united efforts of many earnest workers been pieced together. Several important centre pieces, however, have yet to be added to make the picture complete. Some of these have probably been placed wrongly; every additional bit, however small, rightly inserted may, by forming some fresh curve or notch, suggest how the centre pieces should lie. I propose to-day to invite your attention to some of these small bits, the shape and situation of which I have carefully studied. Of all the theories which have been put forward to explain the increase of tension in glaucoma, that which attributes it to the obstruction to the exit of the aqueous humour from the eye, and which has been so ably expounded and supported by Mr. Priestley Smith, is the most fascinating and apparently explains best the phenomena met with. There are, however, several classes of cases which at first seem to offer considerable difficulties to its acceptance. If these at first sight exceptional cases can be shown really to be consistent with it, then great additional support will be afforded to the theory. These classes of glaucoma with which I now propose to deal are—(1) glaucoma occurring in eyes in which part or the whole of the iris is apparently congenitally absent; (2) congenital glaucoma, otherwise known as buphthalmos or hydrophthalmos; (3) glaucoma occurring after extraction of cataract; (4) glaucoma in cases in which the whole iris has escaped through a wound—i.e., cases of traumatic aniridia; and (5) cases of primary glaucoma in which the operation of iridectomy has been performed and in which the tension has returned. For the consideration of the first two of these classes it is necessary to refer to the development of the iris. Before the iris is formed there exists between the posterior surface of the cornea and the anterior capsule of the lens the anterior portion of the fibro-vascular sheath. This receives its blood-supply

partly from the ciliary arteries and partly from those in the posterior fibro-vascular sheath, which is prolonged forwards around the sides of the lens to join it. The anterior fibro-vascular sheath is developed by a differentiation and vascularisation of the posterior part of the mesoblast, which grows in to separate the cuticular epiblast from the lens ; the more anterior portion of this mesoblast developing into the substantia propria of the cornea and Descemet's membrane The cornea, anterior fibro-vascular sheath, and lens lie, then, at one time in close contact with one another. Until the separation occurs of the anterior fibro-vascular sheath from the back of Descemet's membrane there is not even a potential anterior chamber. The iris, as it grows out of the ciliary body, consists of some mesoblastic cells and of the two layers of the secondary optic vesicle, the posterior of which is at first only slightly pigmented. It has to insinuate itself between the cornea and the anterior fibro-vascular sheath on the one side, and the lens on the other, pushing the prolongation from the posterior fibro-vascular sheath in front of it. The anterior fibro-vascular sheath, when the iris has grown in, becomes raised from the surface of the lens capsule, and forms the pupillary membrane, which subsequently disappears as far as the small circle or corona of the iris. Tags of it when persistent can be seen proceeding from the toothed edge of this corona. It is not at first evident, if primary glaucoma is due to an obstruction to the exit of the aqueous humour from the eye by the apposition of the root of the iris to the periphery of the cornea, how, when the iris is apparently absent in part of or in its entire circumference, glaucoma can develop.

I have been fortunate in having three specimens which help to explain these cases, for two of which I am indebted to Mr. Nettleship. The first is from the patient I have already referred to in speaking of anterior polar cataract, who had apparent complete congenital absence of both irides. The second is from the mother of this patient, who also had apparent complete congenital absence of both irides, and in one eye a leucomatous staphylomatous cornea, with increased tension, secondary to ulceration of the cornea. The third was from a man aged thirty-six years, who had congenital coloboma of the iris outwards and primary absolute glaucoma. In the first case, though no iris could be detected clinically, on peeling off the sclerotic and cornea a small rudimentary iris was exposed in its entire circumference ; it was broadest on the outer side, where it measured 2 mm. in length, above it measured 0·75 mm.,

and it was slightly broader above than below, and was narrowest of all on the inner side. In removing the cornea it was noticed that it was abnormally adherent at its periphery. Throughout the entire circumference of this rudimentary iris the uveal pigment showed at what corresponded to its pupillary border. Some delicate tags, remnants of the pupillary membrane, could be seen passing from the anterior surface of the iris to the lens capsule. Of the left eye microscopical sections were made; in these the cornea appears perfectly healthy. The ciliary body on each side of the section is seen to end in the rudimentary iris, above measuring 1 mm., but much smaller below; the whole of its tissue appears more condensed than usual. Above, the free margin extends just beyond a line drawn backwards on a level with the termination of Bowman's membrane. This line corresponds to the apparent sclero-corneal margin clinically, the real corneal margin being an oblique line extending posteriorly to the angle of the chamber. Below, the tip of the iris does not extend beyond the termination of Descemet's membrane in the ligamentum pectinatum. A small rudiment of the sphincter muscle is distinguishable in the upper part of the iris, but not below. The uveal pigment ends on both sides in a double fold; above, it extends a short distance beyond the stroma. One section shows a tag of membrane prolonged from the anterior surface and floating free, a remnant of the pupillary membrane. In some sections delicate bands of adhesion are seen to pass from the anterior surface of the iris to the ligamentum pectinatum; these do not, however, go up into the extreme periphery of the anterior chamber. In this case, then, though clinically apparently no iris was present, there was really a rudimentary one, which, if pushed forwards, would be quite sufficient to block the whole of the posterior surface of the ligamentum pectinatum. Owing to the obliquity of the sclero-corneal margin the periphery of the iris is hid from view, and when it is very small, as in this case, none of it may be visible. In the left eye of the second case, the mother of the first, there was increased tension; a rudimentary iris, similar to that in her daughter, had become pushed forwards into contact with the periphery of the cornea and was intimately adherent to it. Why it should have become thus pressed forwards from a perforating ulcer of the cornea was not evident; possibly the extensive hæmorrhage which there had been between the choroid and sclerotic may have had something to do with it.

In the third case, the one with congenital coloboma of the iris outwards and primary glaucoma, the ciliary body in the region of the coloboma ends in a rounded knob, which is in contact with the back of the cornea. The termination of this knob, however, does not reach as far as the end of Descemet's membrane, so that the whole of the posterior surface of the ligamentum pectinatum is not blocked by it. The uveal pigment on the back of the termination of the ciliary body is composed of two layers united at the tip, the posterior being much thicker than the anterior. Just where these end on the posterior surface of the ligamentum pectinatum there is a little accumulation of fibres and cells; from it the endothelial lining of Descemet's membrane passes inwards and a layer of cells outwards beneath the pigment on the termination of the ciliary body; this layer of cells may possibly be a persistence of the prolongation forwards from the posterior fibro-vascular sheath to join the anterior. From the arrangement of this knob at the termination of the ciliary body, with a layer of unpigmented cells behind it, I should think that it had never become separated from the cornea rather than that it had recently become pushed forwards into contact with it; so that probably there had always been obstruction to the exit of fluids in this situation, and the onset of the glaucoma was determined by the root of the iris in the rest of its circumference becoming displaced forwards. In a case like this, then, there would be a congenital predisposition to glaucoma. In the first case, although no increase of tension was ever established, there would seem to have been a predisposition to it, for, as I have said, tags of adhesion, not sufficient to block the angle of the chamber, passed across between the rudimentary iris and the ligamentum pectinatum. Several cases have now been published in which the iris was entirely or in part absent, and in which the eye became glaucomatous. Brailey[1] showed at the Ophthalmological Society a girl eighteen years of age with double microphthalmos and glaucoma; the iris was absent in her right eye except for a narrow crescentic piece on the inner side which occupied about two-fifths of the circle, and absent in the left except for three small isolated bits, also on the nasal side. Armaignac[2] reports a case of glaucoma secondary to dislocation of the lens in a patient with nearly complete aniridia.

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1 Transactions of the Ophthalmological Society, vol. x., p. 139.
2 Mémoires et Observations d'Ophthalmologie Pratique, p. 239.

I recorded in the *Ophthalmic Review*,[3] 1891, the notes of a patient under the care of Mr. Tay who had apparent complete absence of the irides in both eyes and increased tension of both. De Schweinitz[4] read before the American Ophthalmological Society the description of a case of coloboma of the iris, polycoria, and primary glaucoma in a woman aged fifty-two years.

There is a practical point in connexion with these cases which follows as the outcome of my observations—that is, that the apparent complete congenital absence of the iris or the presence of a congenital coloboma of the iris does not diminish the likelihood of relief of tension being obtained by a sclerotomy in the former case or an iridectomy in the latter. It is but seldom that an opportunity offers itself for the microscopical examination of an eyeball with apparent aniridia. I have only been able to discover two in which the result of such an examination is recorded. H. Pagenstecher[5] in 1871 was the first to give a description of the microscopical characters of a case of irideremia. In his case the ciliary body ended in the region of the ligamentum pectinatum in a small, pointed, pigmented, and vascular process ; and a prolongation of Descemet's membrane with the endothelium lining it passed beneath this process to the ciliary body. In the last volume of Graefe's Archives, 1891, G. Rindfleisch has given an excellent microscopical description of a case, together with a *résumé* of the literature of the subject. His case was that of a woman aged fifty-one years, who presented the typical symptoms of irideremia in both eyes. She had nystagmus, some amblyopia, and some opacities of the corneæ and lenses, the latter being displaced upwards. Microscopically there were found to be considerable encroachment of the episcleral tissue on the cornea, some hyaline excrescences on Bowman's membrane, and a triradiate deposit of pigment on Descemet's membrane. At the lower sclerocorneal margin there was the scar of a perforation. A rudimentary iris was present, but its sphincter muscle was completely absent. It was closely approximated to the cornea, the angle of the chamber appearing pointed. The ciliary body was badly developed, and its processes were directed backwards. Manz suggested that the arrest in the development of the iris in cases of aniridia is due

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[3] Vol. x., p. 101.
[4] Transactions of the American Ophthalmological Society, 1891.
[5] Klinik für Augenheilkunde, Band ix., p. 425.

to the contact of lens and cornea mechanically preventing its growth inwards. Rindfleisch agrees with Manz, and attributes the contact of lens and cornea in his case to intra-uterine inflammation, beginning in the choroid, extending forwards, and causing perforation near the sclero-corneal margin and escape of aqueous. In my specimens no sign of a perforation is to be seen either in this region or in the cornea. The sclero-corneal margin is a very unusual position for a perforation to occur in, and it seems unlikely that a bilateral affection, as irideremia usually is, would be caused in this way. It is, moreover, unnecessary to have a perforation for the lens to come in contact with the cornea, for at the time the iris is developing these structures are in apposition, the anterior fibro-vascular sheath alone intervening; there are, then, no anterior chamber and no aqueous. We have only to imagine an abnormal adhesion or late separation of these structures to understand how the iris may be prevented from growing in. The adhesions between the front of the rudimentary iris and the ligamentum pectinatum, the folded condition of the uveal pigment at the tip of the iris, and the presence of an anterior polar cataract in the case I have examined are all in keeping with such a theory. If arrest of development of the whole iris can be explained in this way it follows that, supposing the late separation or abnormal adhesion of lens and cornea to be localised, a localised arrest of development of the iris would be occasioned. In this way might be explained cases in which a coloboma of the iris is situated in a position in which it is inconceivable that the fœtal cleft could ever have existed, and also the rare cases in which two colobomata of the iris have been seen to exist in the same eye.

The anterior fibro-vascular sheath is, as I have said, developed from the posterior part of the mesoblast, which grows in to separate the lens from the cuticular epiblast, the anterior part forming the cornea. It can be easily understood that a portion of the anterior fibro-vascular sheath might remain after birth adherent to the cornea. The iris grows beneath the peripheral portion of the anterior fibro-vascular sheath, which comes to form its anterior layer; an adhesion of the anterior fibro-vascular sheath in this region to the back of the cornea would form an anterior synechia of the iris. The central portion of the anterior fibro-vascular sheath forms the pupillary membrane, which usually disappears before birth; occasionally a portion of it persists, and may be attached to the back of the cornea. Cases of adhesion of persistent pupillary membranes to the back of the cornea

have been recorded by Beck,[6] Samelson,[7] Makrocki,[8] and Zinn.[9] In these cases there seems to have been a possibility that the adhesions were caused by intra-uterine perforation of the cornea. I have a specimen of this sort in which no such possibility was at all likely. It occurred in an eye to which I have already referred. In it the central artery of the vitreous was persistent and patent, ending behind the lens in a fibro-cellular membrane, which was seen through the lens as a grey reflex, which led to the diagnosis of glioma of the retina and excision of the globe. The cornea in this eye was quite clear and microscopically appeared perfectly healthy, but adherent to its posterior surface was a large piece of a persistent pupillary membrane, and also the pupillary border of the iris.

The case already quoted, in which the iris appeared clinically to be absent, but where really a rudimentary one was present from the anterior surface of which adhesions passed to the ligamentum pectinatum, is another example of congenital anterior synechiæ of the iris. I have met with two others. In one the adhesions were situated rather nearer the periphery of the iris than its pupillary margin in its outer half; they were about twelve in number, and appeared as little whitish elevations of the iris tissue coming forwards to the back of the cornea, which latter was perfectly clear. In this eye, besides the anterior synechiæ, there were some slit-like openings right through the substance of the iris, just external to the sphincter muscle. The other case was that of a girl aged seven years, whose mother stated she had noticed that the girl's right eye when she was born was larger than the left and that it had a slight cast, and since then that it had gradually increased in size. At the time of excision it appeared about as large again as the left. The cornea measured 12·5 mm. across It was quite clear, and the iris at the upper part was adherent to it, elsewhere being in its normal position It had no perception of light and the tension was increased. Microscopically the laminæ of the cornea everywhere appear normal. Bowman's membrane in the region of the anterior synechia is replaced by fibrous tissue and Descemet's membrane is thickened. The endothelium lining the latter membrane turns round at the edge of the synechia to the anterior surface of the iris. The periphery of the iris is very thin and on

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6 Ammon's Zeitschrift, Band i., Heft.1

7 Centralblatt für Augenheilkunde, 1880, p. 215.

8 Archiv für Augenheilkunde, Band xiv., p. 83.

9 Klinisches Monatsblatt für Augenheilkunde, Band xxviii., p. 290.

both sides of the section is intimately adherent to the cornea. There are also some posterior synechiæ, but no round-celled infiltration in any part of the uveal tract. In this last case it would appear that the portion of the anterior fibro-vascular sheath which becomes incorporated with the iris had not only failed to separate from the back of the cornea in the region of the pupil, but also at its extreme periphery, so that there was a congenital block to the exit of the aqueous humour, which gave rise to an increased tension and expansion of the elastic tunics of the child's eye. Failure in the separation of the iris from the back of the cornea at its extreme periphery might occur unassociated with anterior synechiæ elsewhere; such a condition of things would offer an easy explanation of cases of congenital glaucoma known under the names of "hydrophthalmos" and "buphthalmos." In an eye from a case of this sort sent o me by Mr. Papillon I have found extensive peripheral adhesion of the root of the iris to the cornea. The patient from whom it was removed was a boy aged fourteen years. When he was six weeks old the medical man first noticed his left eye to be larger than his right; it had gradually increased in size since then. The cornea at the time of excision measured 15 mm. across. No operation had ever been performed on it. I have examined microscopically several other eyes affected with congenital glaucoma in which the operation of iridectomy had been unsuccessfully performed, and in all these the angle of the anterior chamber was closed by adhesion of the root of the iris to the cornea both in the region of the coloboma and elsewhere. The anterior chamber in cases of congenital glaucoma is usually abnormally deep, and I should like to point out that it is quite possible to have a deep anterior chamber and at the same time for its angle to be closed, the iris being considerably bent at the point where the adhesion ceases. In an eye in which the primary obstruction to the circulation of the intra-ocular fluid occurred at the angle of the anterior chamber the fluid would be expected to collect in the anterior chamber and distend it. If, however, the primary seat of obstruction was further back, at the circumlental space, as it probably is in the primary glaucoma of adults, then the accumulation of fluid would occur in the vitreous chamber, which would be enlarged and the anterior chamber shallowed. I think the difference in the condition of the anterior chamber in the primary glaucoma of adults and in that of children can be thus explained by a difference in the seat of primary obstruction.

Cases of buphthalmos or congenital glaucoma sometimes go on to a spontaneous cure. Thus a child may be born with increased tension of the eye which may as the result of the tension expand to a certain size; then the tension may become normal, and, though the eye remains large and the sight defective, still no further enlargement or deterioration of vision may occur. I have sections of the eye of one such case which was excised when the child was four years old on account of pain following a blow. In them the iris is much displaced backwards and the angle of the chamber is open, but stretching across from the ligamentum pectinatum to the root of the iris fine, delicate fibrils are seen. The appearances suggest that possibly originally congenital adhesions existed between the root of the iris and cornea, which checked the exit of aqueous humour from the eye, and that as this accumulated in the anterior chamber and distended it these adhesions became stretched, some of them giving way, so that ultimately normal tension became established.

Glaucoma coming on after extraction of cataract is a subject which presents many points of interest, not only to the pathologist, but also to the practical surgeon. In 1888 I published the clinical history of four such cases, and in 1890 I read before the Ophthalmological Society a paper based on the pathological examination of ten eyes in which sight had been destroyed by increased tension supervening on extraction. Since then I have examined pathologically eleven more eyes similarly affected. From the commencement of 1885 to the middle of 1889 there were extracted at the Royal London Ophthalmic Hospital, Moorfields, 1405 senile cataracts; in nine of which cases the eye was subsequently lost from the results of increased tension, giving an average of 0·64 per cent. It can be easily understood that the fact of the lens and a portion of the iris being absent would not materially affect the course of a serous cyclitis. Supposing such an attack to occur in an aphakic eye, there would be the same tendency to the accumulation of cells in the meshes of the ligamentum pectinatum as the fluid containing them filtered through it, and there would be the same excess of fluid thrown into the eye. There would be the same increase in the albumen in the fluid, which hinders its power of diffusion through animal membranes. I have found many cases of increased tension after extraction to be due to the onset of such inflammatory trouble; it is not these I particularly wish to deal with now, but those in which the

operation apparently goes perfectly smoothly and in which the wound heals well, the eye recovering from the operation without any or with only slight inflammation, and in which increase of tension subsequently ensues. In such cases there can be no obstruction of the circumlental space, for the lens is absent ; the pupil is not blocked, and it might be imagined that, as iridectomy had been done, the angle of the anterior chamber in the region of the coloboma would be open. In these cases the time at which the increase of tension comes on after the extraction varies. It may be noted as soon as the wound is sufficiently healed to permit of its estimation, or it may not occur until a year or more after. Often the operation of discission seems the exciting cause of the glaucomatous attack. How can all these cases be explained? From the examination of my specimens I believe as follows. In all eyes in which glaucoma comes on after extraction of cataract there is adhesion or entanglement of the lens capsule in the extraction scar. This adhesion or entanglement holds the root of the iris, or the anterior of the ciliary processes if the iris has been removed up to the periphery, into close contact with the back of the cornea in the region of the coloboma and so keeps the angle of the chamber blocked in that situation. The advanced position which the capsule takes, by reason of its attachment to the cornea, draws forwards the iris lying in front of it, and in this way approximates the root of the iris, elsewhere than in the region of the coloboma, to the periphery of the cornea. In some cases, especially those in which the extraction scar is very corneal, the advance in the position of the capsule is so great that the apposition of the cornea and iris is actually occasioned. In such cases the increased tension follows directly the wound has closed after the operation. The adhesion of the lens capsule to the extraction scar is sometimes composed of inflammatory exudation; this on organising and contracting tends gradually to advance more and more the position of the capsule and consequently that of the iris. Such a gradual advance would explain those cases in which the increased tension does not make its appearance until some time after the cataract has been removed. In an aphakic eye in which glaucoma followed the operation of needling an opaque membrane I found, besides an adhesion of the capsule to the extraction scar, a second adhesion at the needle puncture. This second adhesion had caused a further advance in the position of the capsule, which led to apposition of the root

of the iris and the cornea, and so to increased tension. A needle operation after extraction of cataract leads sometimes to the onset of increased tension without any second adhesion of the capsule to the cornea, probably either by the swelling of the cortical lens matter, so often left in the periphery of the capsule, through the action of the aqueous humour on it, or by the swelling of the ciliary processes occasioned by dragging on them in tearing through the capsule. This swelling of the cortical lens matter or swelling of the ciliary processes may be just sufficient to push forward the root of the iris into contact with the cornea, to which it is already abnormally close by reason of the advanced position of the lens capsule. I have one specimen of glaucoma occurring in an eye from which a traumatic cataract had been extracted in its capsule; in it the anterior hyaloid of the vitreous is clearly shown adherent to the cicatrix in the cornea. An adhesion of the hyaloid of the vitreous to the cornea would tend to cause a greater advance in the position of the iris than an adhesion of the lens capsule, because normally it is further back I have met with two eyes in which the whole iris had escaped through a wound near the centre of the cornea, and which afterwards became glaucomatous. Until I examined sections of them microscopically the cause of the increased tension in them seemed to me quite inexplicable. In both these eyes the lens had been wounded, some portion of it escaping and a broad adhesion of its capsule to the corneal scar remaining. In the escape of the iris through the wound the anterior part of the ciliary body had become drawn forwards, the advance in the position of which had been maintained by the adhesion of the lens capsule to the wound. In both specimens I find the most anterior of the ciliary processes in contact with the posterior surface of the ligamentum pectinatum.

Since Graefe, by what must be described as a lucky chance, discovered that the removal of a portion of iris would relieve the tension in primary glaucoma, numerous speculations have been made and theories suggested as to its *modus operandi*. Obviously the only way to obtain any accurate data upon which to form an opinion on this subject is to examine sections of eyes on which the operation has been performed, and to compare those in which it has proved successful with those in which it has failed. Eyes in which iridectomy has relieved tension are not often obtained for pathological examination; I have, however, had four such specimens. Those in which the operation has failed—unfortunately, only too frequently—come

into the pathologist's hands. A careful study of these offers many points of practical interest. As with so many subjects, when they come to be investigated closely the issue is found not to be a simple one. Iridectomy does not relieve tension always in precisely the same way—failure in relief of tension by iridectomy is not always due to the same cause. First I should like to point out that iridectomy is performed in two very different ways. So different are these two modes of performing the operation in their possible results as regards the relief of tension, that I think it would be well to designate them by two different names. The term "iridectomy" might be retained for those cases in which an incision is made and the iris simply drawn out and snipped off. The old name of "iridectomedialysis" might be revived for those in which after the incision is made the iris is drawn out, snipped on one side, then torn away from its insertion along the whole length of the wound, and finally cut off.

Before proceeding to the ways in which I have found iridectomy to relieve tension I will dispose of two of the theories which have been put forward with regard to it. It is held by some that in making the incision for iridectomy for glaucoma the knife is passed into the extreme angle of the anterior chamber and made to open up the blocked spaces of Fontana and the canal of Schlemm. In order to be able to determine the exact relative position of a cicatrix to the sclero-corneal margin I have, in several recently excised eyes with healthy corneæ, made a scratch with a needle through the epithelium at what appeared to be the exact junction of the clear cornea with the opaque sclerotic. I then hardened the eyes and cut sections of them, and found that my scratch corresponded to the usually well-defined termination of Bowman's membrane. The distance of this landmark to a point on the surface of the globe on a level with Schlemm's canal is 1·61 mm., and to a point level with the commencement of the ligamentum pectinatum—i.e., where Descemet's membrane begins to split up—1 mm. On account of the obliquity of the line of junction of the cornea and sclerotic it follows, that an incision which passed external to the termination of Bowman's membrane and internal to the commencement of the ligamentum pectinatum would be partly corneal and partly scleral. One which passed internal to the termination of Bowman's membrane would be purely corneal, and one passing through the ligamentum pectinatum almost entirely in the sclerotic. The iridectomy cicatrices in the eyes which

I have examined microscopically are nearly all oblique, the gap left in Descemet's membrane on the posterior surface of the cornea being more internal than the commencement of the cicatrix on the surface of the globe. The amount of obliquity varies considerably, and, speaking generally, it is more in those cases in which a keratome is used than in those in which the incision is made with a Graefe's knife. In only two of the eyes I have examined had the incision been made sufficiently peripheral to penetrate the ligamentum pectinatum. In one of these the lens escaped, and the ciliary body prolapsed the day following the operation. In none had the canal of Schlemm been reached. In one eye the cicatrix was entirely corneal. In the remainder, including those cases where the glaucoma was cured, it was partly corneal and partly scleral. Another theory is that the removal of the iris has little or nothing to do with the relief of tension, but that it is occasioned by the formation of a band of scar tissue, which, being situated at the sclero-corneal margin, allows of the filtration of the aqueous humour through it. *A priori*, it is unlikely that the fibrous tissue of a cicatrix would be more permeable by fluid than the fibrous tissue of the cornea or sclerotic. Experimentally, Schoeler[10] has shown that in rabbits' eyes upon which sclerotomies have been performed filtration is retarded, not promoted, and that when he increased the pressure in these eyes the cicatrices remained dry. Pathologically, I find in several eyes iridectomised for glaucoma good bands of cicatricial tissue, well at the periphery of the chamber free for some part of their extent from any entanglement of the iris, in which the tension returned and persisted. Starting now with the known fact that whatever is the first factor in the causation of primary glaucoma the tension is kept up by the apposition of the root of the iris to the posterior surface of the cornea preventing the exit of the aqueous humour through the spaces of Fontana, I find that in the four specimens I have examined in which normal tension was re-established (three of them being subsequently excised for some intercurrent malady and the other removed post mortem) the arrest in the exit of fluid from the eye was relieved in somewhat different ways.

The first case was that of a woman aged fifty-seven years, in whom iridectomy was performed for acute glaucoma of

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[10] Transactions of the International Medical Congress, 1881, vol. iii. p. 100.

four days, standing ; tension was relieved and the eye excised ten years later on account of an ulcer of the cornea with hypopyon. In this eye the angle of the anterior chamber was round and open, both in the region of the coloboma and elsewhere. The iris had not been removed to its extreme periphery, a portion of its root had been left. It would seem that in this case the apposition of the root of the iris to the cornea having only been of recent origin, slight means were sufficient to dislodge it from its faulty position, and that the escape of the aqueous and the drag on the iris incident to the iridectomy had effected this. The second case was that of a man aged sixty-nine years, who had an iridectomy performed on his right eye for a glaucoma of two months' standing. The eye was excised five weeks afterwards on account of ulceration of the cornea ; during the time which had elapsed since the operation tension had remained normal or slightly subnormal. Microscopical examination of this eye showed that the iris had been removed quite up to its point of junction with the ciliary body—that is, in this eye the iris had torn through at its thinnest part, as it usually does when drawn upon in the normal state. The filtration area in the region of the coloboma had thus been opened up through which the aqueous humour could escape. A similar result had been obtained in the third case : that of a woman aged eighty-eight years, who had had repeated attacks of increased tension. An iridectomy was performed and the tension remained normal up to the time of her death. The angle of the anterior chamber elsewhere than at the seat of the iridectomy in this eye remained closed. The fourth case was a private patient under the care of Mr. Tweedy, to whom I am indebted for the specimen. The patient was a woman who had chronic glaucoma in both eyes. In each an iridectomy was performed, and in each the tension was relieved. In the left the cicatrix became cystoid. Eleven months later, while out driving, she caught cold in the eye, and an attack of iritis ensued. Thirteen months after the iridectomy the left eye was excised for the relief of pain. In the region of the iridectomy scar of this eye there was a gap in the sclero-corneal tissue, over which the conjunctiva stretched and around which the conjunctival tissue was swollen and œdematous. Around the outer part of the gap in the sclero-corneal tissue the anterior part of the ciliary body turned, lining the outer wall of the gap, while remnants of the atrophied root of the iris were found lining the rest of it. Relief of tension in this case had evidently been occasioned not by the opening up of the normal passages for

the exit of fluid from the eye, but by the formation of a fresh one in the shape of a fistula in the fibrous tunic of the globe, through which aqueous humour passed into the subconjunctival tissue. I have examined microscopically several cystoid cicatrices and find they are always lined by more or less atrophied iris tissue. If the cut end of a piece of iris becomes entangled in a wound, the wound in closing incorporates the iris in the cicatrix. If, however, a fold of iris tissue becomes involved, the anterior surface of the iris unites to the cut edges of the sclero-corneal tissue, the conjunctiva heals over it, and thus a gap is left lined by iris tissue in the walls of the eyeball. It is known that the iris tissue is impermeable by the intra-ocular fluids, for when the pupil becomes closed by total posterior synechiæ they accumulate behind it and bow it forwards, instead of passing through it. Hence it follows that an eyeball may have a gap in its coats, but that no escape of fluids would occur into the subconjunctival tissue through the gap so long as it was lined by impermeable iris tissue. Should, however, there be any tendency to accumulation of fluids in such an eye, the gap, being a weak spot, would yield and expand, the iris tissue lining it would become atrophied and stretched, and breaks would occur in the continuity of its uveal pigment layer, which is probably the chief obstacle to the passage of fluid through it. There would then be formed a thin bulging or cystoid cicatrix lined by very atrophied iris tissue, through which any excess of intra-ocular fluid would be able to pass into the subconjunctival tissue. It is not uncommonly observed, and was pointed out by Graefe, that these cystoid cicatrices periodically discharge themselves subconjunctivally, the explanation being that when the accumulation of fluid has reached a certain stage a rent occurs in the iris tissue which allows of its escape. Mr. Bader, in the form of sclerotomy which he advocates, seeks to produce a condition such as the above. He says : [11] "To secure success it is desirable to obtain, and to maintain, a staphyloma of the conjunctiva, with or without prolapse of the iris." De Wecker and most other operators endeavour to avoid a prolapse of any kind.

Such, then, being the ways in which I have found iridectomy to relieve the increase of tension in glaucoma, I will now describe what I have found to be the causes of failure in the operation. By far the commonest would seem to be the non-removal of the iris up to its extreme periphery, so that a

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[11] Ibid., 1881, vol. iii., p. 98.

portion of it is left blocking up the filtration area, sometimes rendered additionally secure in its faulty position by the healing of the cut end of the iris in the corneal cicatrix. In cases where glaucoma has been of long standing the root of the iris becomes absolutely adherent to the cornea, and when drawn upon, instead of tearing at its junction with the ciliary body, tears at the point where it ceases to be adherent, so that the filtration area never becomes opened up. Sometimes the adhesion of the iris is so firm that its separation is an absolute physical impossibility. In acute glaucoma, when the root of the iris only lies in apposition with the cornea and is not adherent to it, it tears away from its extreme periphery. This is the difference between acute and chronic glaucoma, which renders the operation of iridectomy such a very satisfactory proceeding in the former and so very unsatisfactory in the latter. Another cause of failure in the removal of the iris up to its periphery is the way in which the operation is performed ; thus a simple withdrawal of the iris and snipping it off are not so well calculated to effect this as what I would term, as I have already explained, an "iridectomedialysis." Sometimes after an iridectomy for glaucoma the anterior chamber is very slow in re-forming. In such cases the plastic exudation thrown out to effect the closure of the sclero-corneal wound may unite the lens capsule lying in contact with the wound to it. Then when the anterior chamber does re-form, the part of the lens in the region of the wound will remain adherent to it, becomes drawn forwards, and may hold the anterior of the ciliary processes or the root of the iris if a piece of it is left, in contact with the ligamentum pectinatum in the situation of the coloboma. In such cases, it is needless to say, tension is not relieved. It has been suggested that where the lens is very much pushed forwards in cases of glaucoma, or where it seems unlikely that the anterior chamber will readily re-form, a scleral puncture should be performed as a preliminary procedure, and in some cases this has apparently seemed to answer its purpose well. I should like to point out, however, a possible danger which I have met with in connexion with scleral puncture. In a case in which it was performed suppuration in the vitreous subsequently ensued, and the eye had to be excised. Sections across the line of puncture show a prolapse of vitreous much infiltrated with pus cells lying in it. It must have been along this prolapse of vitreous that the suppurative infection had gained access to the interior of the eye. Prolapse or adhesion of the ciliary processes to the sclero-corneal cicatrix after the

iris has been removed not infrequently occurs when the incision is a very peripheral one. A study of the anatomical relation of the anterior part of the ciliary body and iris shows at once that this is what might be expected. The anterior part of the ciliary processes extends for some distance behind the periphery of the iris, so that if the sclero-corneal incision is a very peripheral one, and the iris is drawn out, the anterior part of the ciliary body is drawn forwards with it; in one specimen I have it was cut off with the iris. An adhesion of the anterior part of the ciliary body to the iridectomy cicatrix I have found to block the filtration area, even after the iris has been removed up to its extreme periphery. In acute glaucoma I do not think there is any necessity, provided an iridectomedialysis be done, to make the incision very peripheral. The iris tears away naturally at its root when drawn upon, wherever the incision is, and a peripheral incision certainly increases the risks of the operation.

In conclusion, I should like to urge, as the result of my observations, the necessity of performing an iridectomy early in cases of chronic glaucoma. I think in some cases, even, it would be well to perform what might be termed a preventive iridectomy for this disease—that is, to perform an iridectomy when only the subjective symptoms are present and when no deterioration of vision has occurred. The risks of an iridectomy at such an eye are very small—they can hardly be considered to be greater than the risks of a preliminary iridectomy for an extraction of cataract. The chances of success, in preventing or relieving increased tension, of an iridectomy performed before adhesion of its root to the cornea has occurred are infinitely greater than after such adhesion is established.

PRINTED AT THE LANCET OFFICE, 423, STRAND, W.C.