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COMMUNICATIONS

ON THE CEMENT SUBSTANCE OF THE INTRA-
OCULAR MUSCLES AND CHRONIC GLAUCOMA

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(Continued)

PART II

THE ORIGIN OF CHRONIC GLAUCOMA

An Anatomical Basis—a Glaucoma Mechanism

There is now no doubt that many chronic glaucomas are characterized by a combination of three outstanding features :

A. The sinus of the anterior chamber has a thick posterior-inner wall, made up mainly of an extension of ciliary body tissues supporting the iris base, instead of iris alone, as in many eyes.

B. The open angle of the chamber has been displaced by pressure of the aqueous, so that it lies abnormally far beyond the level of the scleral furrow and of Schlemm's canal. The position attained may be such as is never seen apart from chronic glaucoma.

C. The meridional bundles of the ciliary muscle fail to reach near to the level of Schlemm's canal, and the middle and inner divisions of the muscle are neither particularly broad nor closely packed. There is the defective inward pull of the muscle on the pec-

tinuate ligament that was emphasized in the *Trans. Ophthalm. Soc. U.K.*, XLV, 1925, i, 340-1. And the glaucoma developing in these eyes may not inaptly be spoken of as the glaucoma of the peripherally placed and poorly developed ciliary muscle.

These three features are very well illustrated in Fig. 9 from my one and only specimen of quite early chronic glaucoma. It is only

FIG. 9. $\times 114$.

by a rare chance that one can obtain an eye showing anything approaching this complete picture, since the high tension of the later stages of the disease blurs the anatomical detail, and the sinus of the chamber becomes closed. But in many of the ordinary painful blind eyes that one has the opportunity of examining under the microscope the type of the disease can still be roughly made out. By varied staining the site of the closed filtration angle may often be quite definitely located, and such structure as can be made out conforms with the above outline.

The Working of the Mechanism

It is a very old observation that the periphery of the iris falls back in accommodation of the eye. It has to do this to compensate for the advance of the anterior pole of the lens.

Mr. Priestley Smith, in kindly giving me the reference to Helmholtz's original observations⁽⁷⁾ on this movement of the iris, sent also the reminder that Helmholtz was unaware of the associated flattening of the periphery of the anterior surface of the lens in accommodation, which must aid in the back-swing of the iris. But the question whether the iris base is mainly pushed back or drawn back in accommodation does not alter the fact that passive movement of the very thick block of combined tissues forming the posterior-inner wall of the sinus of the chamber in Figs. 9, 12, 13, 14, 15, 16, must be greatly impeded, as compared with the movement of a free iris base.

In these eyes the *impeded back-swing of the thick wall of the sinus* must tend to cause a momentary rise of pressure in the chamber in each act of accommodation. Such transient elevations of chamber pressure in these eyes of the triple anatomical combination are rendered the more possible since there is no accompanying powerful opening pull of the ciliary muscle on the pectinate ligament and canal, such as in other eyes might provide a partial or temporary outlet for the aqueous displaced from the middle of the chamber by the lens advance.

The constantly repeated momentary push of the displaced aqueous must lead to a slowly progressive additional displacement of the already peripherally placed iridial angle, increasing the anatomical defects. That the angle in early life already lay well behind the level of Schlemm's canal is to be judged from the similar position of the meridional muscle heads. The resulting increase in length of the thick sinus wall, with decreasing provision for escape of aqueous, must accentuate and prolong the momentary rises of chamber pressure, until some of the elevation of pressure becomes continuous (glaucomatous).

This onset of glaucomatous tension must inevitably accentuate and perpetuate the anatomical difficulties. It is a vicious circle. And the disease, once started, is always, if untreated, permanent and progressive, without complete intermissions.

Supplementary and Explanatory

1. In order to state the main issue as simply as possible, I have omitted one rather important consideration: Before attempting to draw the above inferences from a slowed *passive* backward-inward movement of the thick sinus wall in these eyes, one ought to feel sure that the defect is not made good by an *active* movement, from contraction of the circular muscle bundles seen lying in the sinus wall.

The line of contraction of these muscle fibres, acting as a ring muscle, directly inwards towards the lens, is so nearly (in some eyes, strictly) parallel with the posterior

surface of the iris, that any possible slight deepening of the sinus producible in this way would almost certainly be fully counterbalanced by the increased firmness imparted to the sinus wall by the contracted state of the muscle.

This question of the line of action of the inner fasciculi of the ciliary muscle should acquire particular force with regard to the exceptionally strong muscle seen in the sinus wall of Fig. 15. It is satisfactory, therefore, to have the direction of contraction indicated, so far as is possible, by the general contour of the muscle stretching forwards and inwards from its attachment to the sclera, and more especially by the fine points jutting forward in the sinus wall from the sloping border of the muscle.

It seems certain that the direction of these drawn-out points of the muscle must indicate fairly well the line of movement of the muscle border when the muscle contracts. Otherwise, they could not persist.

2. It scarcely needs emphasis that the suggestion of a transient rise of chamber pressure with accommodation applies exclusively to this type of eye. And it has to

FIG. 10.

a = Fig. 9 in semi-diagrammatic form.

b = From an eye with a Thomson scleral spur and resistant radial ciliary muscle.

c = From an eye with good inward pull, but no localized scleral spur.

do with the active accommodative movement only. It implies nothing with regard to continued accommodation, which, as Grönholm⁽⁸⁾ showed, tends to lower the intra-ocular pressure in glaucomatous eyes. Where there is no impediment to free movement of the iris base, and where the ciliary muscle extends well forwards, the opening pull of the muscle on the ligamentous spaces must be at least sufficient to prevent any rise of pressure in the anterior chamber in accommodation, whether there happens to be an Arthur Thomson type of scleral spur present or not. See Fig. 10*b* and *c*.

3. A reminder is needed of the importance that has been attached to the ciliary muscle in aiding the flow of aqueous from the anterior chamber. Heine⁽⁹⁾ showed the extreme influence of unnatural pull of the muscle on the lumen of Schlemm's canal and on the spaces of the ligament in apes. Professor Thomson⁽¹⁰⁾, in his well-known pump action theory, stressed the value of interrupted action of the meridional muscle in accommodation, with the aid of a mobile scleral spur, such as is shown in Fig. 10*b*. I personally have found an obviously mobile spur to be exceptional. But the opening influence on the spaces of the pectinate ligament exerted by the middle and inner fasciculi of a forward-reaching ciliary muscle is often very obvious, as in Fig. 10*c*.

The other extreme was illustrated by a micro-photograph published in the *Brit. Jl. of Ophthal.* VII (1923), 470, and in the *Trans. Ophthal. Soc. U.K.*, XLV (1925), i, 341, from the early glaucomatous eye that furnished Fig. 9 of this paper.

The meridional muscle heads exert a purely meridional pull, through a definite tendon, on the greater portion of the pectinate ligament, leaving the oblique and circular divisions of the muscle connected only by an attenuated narrow band with quite the

innermost ligamentous trabeculae. It is the extreme of deficient inward traction. If the meridional muscle had any influence at all on the ligament of this eye, it could apparently only draw the lamellae closer together, tending to hinder filtration. And one is reminded of the suggestion of Küsel⁽¹¹⁾, that the influence of the meridional muscle on the ligament is probably to narrow the openings in the lamellae.

Unless the influence of the ciliary muscle on the outflow of aqueous has been much over-rated, one feels fairly confident in claiming that marked and obvious defect in its action on the pectinate ligament, and on Schlemm's canal, may be the crucial influence determining the onset and development of glaucoma in eyes otherwise predisposed to the disease. If this claim is valid, the mechanism for the origin and progressive advance of chronic simple glaucoma is complete.

The Origin of the Mechanism

The above working outline necessarily includes the essentials of the origin and development of the disease. It implies a primary anatomical predisposition embracing some essential portions or degrees of the mechanism, as a basis for the progressive pre-glaucomatous increase. The inevitable automatic advance of the defect appears to be such that some people may be said to be born with the certainty of glaucoma, induced solely in this way, if they live long enough—to middle or old age.

There is much evidence to support this view, although the task of disentangling completely the primary congenital elements from the later acquired additions, is one that will necessitate access to more material from the earlier years of life than has yet come my way. Some broad indications are now available :

A. Low grades of Factor A, the supported sinus wall, are quite common, and higher grades probably not uncommon, representing purely persistence of a foetal phase. But I have not seen anything in early life comparable in length, breadth and solidity, with the formations of Figs. 15 and 16, which may be taken as typical of advanced pre-glaucomatous development.

The forward projecting inner ciliary muscle bundles so constantly seen behind the iris base in such eyes (see also Figs. 9 and 14) may perhaps play a minor rôle in the development of the thick sinus wall, as it is so regular a feature of peripherally placed muscles, and so frequently absent or insignificant in muscles whose meridional bundles extend forward beyond the level of the posterior end of Schlemm's canal. (Fig. 10*b*, however, shows such a forward-reaching muscle with a projecting inner plate).

The projecting inner plate, may be, in some degree, a response of the muscle to the stimulus of accommodation, to make up for the unfavourable peripheral position of the muscle as a whole. This suggestion is merely in accord with the hypertrophy of the muscle that results from the same stimulus in hypermetropic eyes.

A much more definite and certain influence in the progressive development of the broad muscle-containing glaucomatous, or pre-glaucomatous, sinus wall is evidently the zonular tension of Part I of this paper, apparently aided by the pull of the dilatator

pupillae, or of the sphincter pupillae transmitted through the cement substance of the dilatator. *Evidences of traction and displacement* are to be found in the tissues (pp. 348-9) that can be explained only by the advance of the epithelial surface towards the lens. Where this advance seems indicated by general contours and associated conditions, doubt may often be turned into certainty, on looking through a series of sections, by finding one or more narrow localized epithelial depressions at points where the advance has been held back by relatively unyielding bands that cross the hyaline-free space to reach the ciliary muscle directly, or indirectly through the "elastic ring."

Such narrow, and usually pointed, epithelial depressions, seen in Figs. 9 and 15, consisting in part or altogether of the deeper pigment epithelium alone, dipping down towards the ciliary muscle, may, I am sure, often furnish proof of stretching of the hyaline-free space between limiting membrane and muscle, from advance of the surface towards the lens in later life. This is proved by observation of their more rapid formation under abnormal forward-inward traction, *e.g.*, of iris and lens capsule impacted in a corneal wound, or of organizing exudate between the lens and iris.

The chief of these inverted epithelial processes spring (1) from between the bases of minor "wart" ciliary processes, (2) from the angle between the posterior surface of the iris and the anterior surface of the "inner ledge" of the ciliary body, *i.e.*, of the tissues supporting the iris base. And at these sites they are evidently formed by coalescence of the stretched surfaces, drawn tightly together. Those at site (2) necessarily lie near the radiations from the dilatator pupillae, where both are present in the same section, and they may be connected with different parts of the elastic ring. It may be a little difficult to decide whether an isolated process is a true dilatator radiation, or whether it represents the angle between iris and inner ledge of earlier periods of life.

In a few glaucomatous and pre-glaucomatous eyes the long-continued traction, transmitted from the limiting membrane of the epithelial depressions to the muscle, has been sufficient to draw forward isolated bands (Fig. 9 and p. 348) or projecting points (Fig. 15, and p. 349) of muscle, so obviously and so exceptionally as to furnish absolutely certain evidence, I believe, of traction-displacement towards the lens.

In other eyes, the direction of the fibres connecting the limiting membrane with the anterior end of an inner muscle "plate" or "net" suggests that this portion of the muscle has been drawn forward.

The results of this late advance of the inner ledge of the ciliary body, and of the ciliary muscle, are not unlike a reversion to the transient phase of foetal life (seventh and eighth months) illustrated by Seefelder⁽¹²⁾ in the Graefe-Saemisch Handbuch. A considerable strip of circular muscle bundles is there shown in the sinus wall.

B. Increasing displacement of the angle of the chamber (Factor *B*) is brought about by the retraction of the ends of the oblique (radial) fasciculi from their hyaline sheaths, seen in Fig. 6, these muscle heads and their means of attachment forming the main support of the chamber angle. And the influence of chamber pressure in contributing to the displacement seems certain, (*a*) from the common limitation of the retraction to those muscle bundles that have to support the pressure, and (*b*) from the displacement being more marked in eyes in which the muscular development is poor. Note the complete absence of pressure displacement of the very powerful muscle of Fig. 15. The middle (radial) portion of this muscle extends actually in advance of the meridional fasciculi.

Such (meridional) muscle heads as may arise from hyaline sheaths, but which lie in the (anterior-outer) side wall of the sinus, supported by the sclerotic, certainly escape the obvious and marked retraction of Fig. 6, apparently because the chamber pressure on them is lateral.

The very variable pre-glaucomatous muscle displacement does not differ essentially, except in degree, from the obvious result of the glaucomatous pressure of Fig. 9, given in detail on p. 351.

The retraction of the muscle adds to (*C*) the defect in the opening action of the muscle on the spaces of the pectinate ligament and on Schlemm's canal.

The late Congestive Phase

The late ciliary congestion and pain that supervene ultimately in the majority of untreated chronic glaucomas, appear to be due to closure of the periphery of the anterior chamber, as in primary acute and subacute glaucoma.

There is ample evidence among my sections that the closure may take place first at a little distance from the angle, being produced by swollen congested ciliary processes, pressing the iris base forwards immediately beyond the thick, resistant sinus wall, to come in contact with the pectinate ligament.

There is nothing in the chronic glaucoma mechanism to account directly for gross swelling of the processes. There are merely the two influences: (1) the advance of the tissues behind the iris base, tending to carry the ciliary processes forwards and inwards, narrowing the circumferential space; and (2) the general widening of the hyaline-free space, tending to venous dilatation.

These influences appear insufficient to account for the definitely secondary nature of the late congestive phase—for its frequency, its onset after a congestion-free period, not too prolonged, and its association more particularly with the higher grade tensions.

It is, I think, a matter of common observation that the glaucomatous eyes that remain permanently free from all trace of congestion are mainly those in which the disease has progressed most slowly, in some cases having taken many years to result in complete blindness. The explanation seems to be that with plus tension increasing very gradually, atrophic changes in the ciliary body—fibrosis and shrinkage—have time to develop sufficiently to forestall the possibility of secondary venous congestion of the ciliary processes taking place.

The inference is that with more rapid increase of intraocular pressure the pressure itself may, perhaps, impede the venous circulation sufficiently to lead to dilatation of the veins and enlargement of the ciliary processes.

Considering the striking results that have been obtained by experimental ligation of vortex veins in rabbits⁽¹³⁾—marked swelling of the ciliary body, shallowing of the chamber and plus tension—it is difficult to rule out altogether the influence of possible compression of these veins at their points of exit through the sclerotic by high intraocular pressure. Exaggerated claims in this respect⁽¹⁴⁾ may have antagonised general opinion too strongly.

But such general considerations certainly do not cover the whole ground. One is sometimes struck with the high degree of early plus tension found in glaucoma without any trace of ciliary congestion. One has to fall back on the infinite variety of the anatomy of the parts about the filtration angle, including features here dealt with, and possible combinations with portions of the recognised Priestley Smith congestive mechanism.

In Fig. 9 the projection of the iris between the points 1 and 2, and the corresponding slight bulge of the pectinate ligament between 1' and 2', narrowing the chamber, represent an earlier localized adhesive closure of the chamber, that gave way after operative relief of the tension. And that the closure was due to the pressure of ciliary processes is seen by reference to the *Trans. Ophthalm. Soc. U.K.*, XLV, 1925, i, p. 342, Fig. 52, from the same eye.

In Fig. 14 is seen a permanent adhesion at this site doubtless produced in the same way by processes that have retracted in the beginning atrophic stage of the disease.

It is interesting to note that localized closure of the chamber at this point is to be seen in a few glaucomas that are apparently primarily congestive in type, acute or subacute, necessarily associated with a thick sinus wall (Factor A). Such a finding is to be expected, following the recognition of such marked examples of the thickened wall as seen in Figs. 15 and 16, occurring without any acquired peripheral displacement of the chamber angle. I have one example of localized narrowing of the chamber from the pressure of ciliary processes in an eye as yet free from glaucoma. But in no eye is this localized closure or narrowing to be found in all sections examined. Usually it represents an early phase persisting at some points, with complete closure of the filtration angle in other portions of the periphery of the chamber.

Some Mixed Types

1. Primary closure of the filtration angle, the most essential feature of the Priestley Smith mechanism, has been in my mind associated particularly with acute primary glaucoma, and with recurrent prodromal attacks, with "haloes." But, apparently, it may also account for some—possibly only a few—glaucomas that begin quietly and almost imperceptibly. For instance, Fig. 11, from the *Trans. Ophthalm. Soc. U.K.*, XLV (1925), part 1, p. 338, is taken from an eye that is said to have become nearly blind from glaucoma so quietly that the patient did not notice anything wrong with the eye until the painful congestive attack occurred, for which the eye was removed. Her attention had been fixed on the fellow eye, in which prodromal attacks preceded an acute attack, satisfactorily relieved by operation. There is no supported iris base, and there is primary closure of the sinus of the chamber. In one section the swollen ciliary body is seen still pressing the base of the iris forwards.

FIG. 11.

FIG. 12. X 67.

2. Fig. 12, from a glaucoma patient aged 72 years, shows a complete mechanism above the cornea—a very broad sinus wall, chamber angle far back, and an almost purely meridional pull on the pectinate ligament. But, below the cornea there was no supported iris base; the iris alone formed the sinus wall. And there was the closed angle of congestive glaucoma from the pressure of swollen ciliary processes, the angle being situated nearer the level of Schlemm's canal than is here seen.

In this case, there appeared to have been an explosive congestive attack six weeks before, supervening on chronic glaucoma. The specimen, for which I am indebted to P. E. H. Adams, was obtained through death of the patient four days after a Lagrange operation. The tear from X to X' was doubtless due to the iridectomy.

3. In a number of glaucomatous eyes, the (generally closed and sometimes elongated) sinus of the chamber slopes inwards, away from the sclerotic, as shown in Fig. 10b, and slightly in Fig. 11, by the sloping border of the ligamentous terminals. But both the sinus and the ciliary muscle that supports it are placed much more peripherally than in Fig. 10b, so that there is very little action of the muscle on the spaces of the pectinate ligament. The sloping border of the muscle apparently indicates persistence of the primary formation, owing to unusual strength and resistance of the oblique (radial) fasciculi. These are found broad and closely packed, usually forming an obvious meshwork with the meridional fasciculi.

The clinical histories have been defective in the cases that I have seen.

Association with Secondary Glaucoma

Reference need be made only to two of the illustrations published herewith.

1. Fig. 13 is from a painful, congested, hard and nearly blind eye, in which there was repeated hyphaema, in a woman of 59 years of age.

Blood in anterior chamber

Meridional muscle
Blood in angle of chamber

Radial muscle

FIG. 13. $\times 43$.

From a patient aged 59 years. van Gieson staining.

There is a deep anterior chamber such as that with which one is familiar in iridocyclitis with plus tension. But, coupled with the abnormal chamber contents, as cause of the high tension of the eye, there is a fair representation of the chronic glaucoma mechanism. The peripheral displacement of the angle of the chamber is doubtless partly recent and secondary to high pressure in the chamber; but that it is partly primary may be inferred from the thick posterior-inner wall (x) of the sinus, and from the position of the meridional muscle heads, behind the level of Schlemm's canal. Without some anatomical predisposition to glaucoma, there might have been no plus tension.

2. Fig. 14 shows the typical chronic mechanism. The deepened sinus of the chamber extends quite exceptionally far back, as is also the meridional muscle origin, and there is the supported iris base, in a man of 78 years. But the glaucoma followed cataract extraction, and subsequent limited epithelial downgrowth into the anterior chamber, which, however, nowhere extended near the angle of the chamber.

FIG. 14. $\times 43$

Thus the glaucoma was much more primary than secondary. And this agrees with my clinical experience of glaucoma following cataract extraction. There has very rarely been any adequate clinical explanation of the complication, *i.e.*, anything beyond slight impaction or adhesion of a pillar of the iris coloboma, or of a tag of capsule, such as is often seen without ill result.

The Finer Histological Details

Acceptance or rejection of the whole theory above outlined must depend finally upon the support derived from close examination of the tissues. Take first Factor A, the thick sinus wall :

(a) Primary formations. Varying grades of unusual persistence of foetal ciliary processes behind the iris root are frequently met with. Fig. 13 is from my most extensive case, Fig. 4 from an average low grade case, and Fig. 19 from a quite minor example. Separate "warty" processes are found extending forwards in rows meridionally in line with the much larger ordinary ciliary processes.

I have found all cases alike characterised by proportionate defect in the peripheral radiations of the dilatator pupillae. The muscle ends where the processes begin, though it extends farther in the valleys between the rows of processes. In place of the normal radiations reaching to the elastic ring, there are only short, useless, brush-like stumps of fibres. There may be supplementary bands of fibres springing from the ciliary epithelium, nearer to the angle of the chamber, that may, or may not, reach the elastic ring.

This defect has been so constant as to suggest that the persistence of the foetal processes is due to the failure of the dilatator muscle to connect up with the pectinate ligament terminals through the elastic ring. And this failure probably accounts also for the very loose mesh of the thin strip of continuous tissue that is found immediately behind the usually attenuated iris, which is here represented only by vessel layer and anterior border layer.

Hence the slack appearance and uneven outline of the iris root in Fig. 4. And, generally speaking, it is obvious that the presence of separate persistent foetal processes, though they are largely made up of dense fibrous tissue, does not serve to stiffen the wall of the sinus. It is rather the reverse.

This suggestion of weakness of the sinus wall does not extend to the broad ground plate α in Fig. 13, uninterrupted by meridional "valleys," or to other similar congenitally thick sinus walls, unaccompanied by the separate persistent "warty" processes of this case. But the two above-mentioned features—partial or complete failure of the dilatator radiations, and looseness of the tissues—are of some service in distinguishing the purely, or mainly, primary formations.

(b) Contrasting with the above features, there are the very important *evidences of traction and displacement* in the broad sinus wall of the glaucoma mechanism, already claimed as indicating progressive changes, acquired features. They have a slight practical bearing also, in that the tissues bearing the impress of traction seem to be, in parts, more closely packed, tightened and resistant, and therefore suited to bear the interpretation of relative rigidity, given as an integral part of the working of the mechanism.

(1) Signs of displacement are particularly convincing in Fig. 9, which is as it should be, since the glaucoma was of rather severe type, resisting miotics and tending (as judged by the more advanced fellow eye) soon to become moderately congestive. Part of the stretching and displacement in this sinus wall merely indicates resistance to the peripheral displacement of the angle of the chamber, resulting from the high chamber pressure. But after making every allowance for this factor, features remain that are explicable only as results of zonular traction.

The extent and shape of the nearly rectangular mass of elastic tissue (a), the "elastic ring," are quite exceptional. The tissue reaches much farther than is normal from its ordinary site of development, near and between the heads of the radial bundles of the ciliary muscle (see Part III). And this is explained by the structures attached to its angles.

The upper left-hand corner of the tissue in the photograph was found, by examination of other sections, to be connected through the small muscle-containing band (b) with the limiting membrane of the indrawn process of epithelium (c). This very unusual, narrow, isolated strip of muscle, does not represent a developmental anomaly. It has certainly been drawn forward (upward in the photo) to the extent seen, from the radial fasciculi seen below the elastic mass; and the connecting bands lie hidden, because unstained, in the elastic tissue. In other sections, the up-drawn border of the muscle and the disarranged narrow neighbouring fasciculi are well seen.

The upper right-hand corner of the mass of elastic tissue is connected with the dilatator muscle of the pupil, by an exceptionally strong band (*d*). Berner⁽¹⁵⁾ and v. Szili have found in such bands, rarely, processes of ciliary muscle extending forward actually to join the dilatator fibres. The traction of the dilatator fibres (or more probably of the pupillary sphincter transmitted) upon the elastic tissue in this eye seems to have been nearly as effective as that of the zonular fibres, judging from the shape of the elastic mass.

(2) The massive pre-glaucomatous sinus walls of Figs. 15 and 16 are very useful, since there is little or no peripheral extension of the sinuses in these two eyes, to account for any part of the formation of the sinus walls.

FIG. 15.

From Elliot's "Treatise on Glaucoma," fig. 23, facing p. 115; also published in the earlier "Text-book."

For Fig. 15, as a very valuable example of predisposition to glaucoma, I am indebted to Col. Elliot, who published it under the title "Narrowing of Angle of Chamber preceding Glaucoma," the fellow eye being glaucomatous. The specimen was supplied by Treacher Collins.

That there has been no pressure displacement of the chamber angle is shown by the fact of the radial portion of the muscle extending further forwards than the meridional portion. Any lengthening, therefore, of the sinus wall, must mean some definite forward-inward displacement of tissues towards the lens.

The three small, thin processes from the black mass of pigment epithelium between the base of the iris and the ciliary processes are, I am sure, connected by unstained fibro-elastic bands, with the rather-distant, out-drawn points of muscle along the muscle border, already referred to on p. 340. This is shown by the direction of the two opposing sets of processes, epithelial and muscular, and by comparison with selectively stained eyes in my collection. The connection between the pigment epithelium and the muscle is more extensive and more direct than in Fig. 9, corresponding with the greater bulk of the muscle.

(3) In Fig. 16, from a healthy eye at 74 years, there are no clear signs of traction. But the long pigmented radiation from the dilatator muscle, passing down to be connected with a process of the elastic ring, strongly suggests late development, as opposed

to the primary formations with defective or absent dilatator radiations, above mentioned.

And in other sections an indrawn process of pigment epithelium was found, like that of Fig. 9, but nearer to the dilatator radiations, running down parallel with the latter from the irido-ciliary angle, to be connected by a band with the elastic ring.

The direction of the collagenous fibres seen in the inner part (left hand) of the very broad block of tissues in Fig. 16, streaming upwards and to the left from the neighbourhood of the muscle, are suggestive of zonular traction.

(4) In Fig. 12 the only evidences of traction are the elastic fibres streaming upwards from the elastic ring. There is no radiation from the pupillary dilatator to meet

Radiation from dilatator
pupillae

Muscle bundles
Process from elastic ring
Elastic ring

Over-stained inter-fascicular
fibrous tissue

FIG. 16.

them, apparently because the dilatator at this site has (temporarily?) become resolved into a few isolated rounded pigment cells. This is explained by the fact that the iris base seen in the upper part of the photograph represents the stump of iris, left after the iridectomy performed as part of the Lagrange operation a few days before.

Other sections from this eye, stained by van Gieson's method, show the inner plate of the ciliary muscle drawn very far upward in the block of tissue here shown.

(5) The origin of the mechanism has been dealt with only so far as was necessary for the establishment of the mechanism. To go beyond this is too big a task for the present. Thus I am omitting the available evidence indicating that the pull of the dilatator pupillae may aid in the advance of the tissues behind the iris root. And I have made no attempt to investigate the possible influence of variations in the attachments of the short zonular fibres, or to correlate these microscopical observations with measurements of circumlental space, etc.

B. Extension and Distension of the Sinus. The more significant available details are the following :

In Figs. 9 and 14 the sinuses are, I think, a little wider than normal, *i.e.*, deep clinically. During life, they were probably deeper than is here shown, allowance having to be made for the shallowing of the chamber as a whole, that takes place ordinarily after removal of the eye, before and during fixation⁽¹⁶⁾. In Fig. 9

this impression of deepening of the sinus, unaccompanied by any deepening of the chamber as a whole, is confirmed by thinning of the iris base above (x), from compression. Iris tissue only begins at (x). Below this point in the illustration is seen wavy elastic tissue, representing pectinate ligament terminals drawn around from the anterior-outer boundary of the sinus.

That the oblique, or radial, muscle heads in this eye have been pushed back—and not merely retracted—is indicated by the following:

The lowest point reached by the curve of the muscle bundles lying around the angle of the chamber corresponds with the lowest point of the curved boundary of the chamber. And the muscle heads are thickened, as compared with the backward continuations of the fasciculi.

Slackness of the outermost radial muscle head (f) is shown by its curved, crumpled outline. There is a marked sudden drop in the level of this muscle head, as compared with that of the bundle immediately to the outer side of it, shown better in sections stained by van Gieson's method.

The same sudden drop in level is well seen in Fig. 12; less well, owing to the tearing of the tissues, in Fig. 14.

There is a faint indication of the widely open hyaline sheath, slightly out of focus, from which the crumpled muscle head in Fig. 9 has retracted, seen above the muscle. This uncommon finding of a widely-open hyaline sheath, unoccupied by muscle, may perhaps be taken to indicate that the backward displacement of the muscle head has been recent.

C. The peripheral origin of the meridional muscle was dealt with in 1925, when Factor C was the only one of the three that was recognised. The muscle may, as already mentioned, arise from a definite tendon, only laterally attached to the sclerotic. This may be exceptional, even in glaucomatous eyes, but the meridional pull—the “defective inward pull” on the ligament—remains an essential part of the glaucoma machinery.

Though this Factor C is essentially a congenital feature primarily, there is the possibility that where there is no origin of the muscle directly from the sclerotic, the meridional muscle heads may retract further from the level of Schlemm's canal through simple senile wasting.

In the fellow eye to the typical early glaucomatous eye of Fig. 9, there was the atrophic shrinkage of the ciliary muscle of advanced chronic glaucoma. In this eye, the meridional muscle origin was found to be definitely farther back than in the eye of Fig. 9. Apparently, the tendon had stretched.

Brief mention has elsewhere been made of a case of apparently defective action of the ciliary muscle on the deeper spaces of the pectinate ligament and on Schlemm's canal, due to the fact that the canal was more deeply buried in the sclerotic than is usual. The pectinate ligament was also exceptionally short. There was chronic glaucoma with the typical glaucoma mechanism.

Support for the above Observations

A. There are two powerful arguments indicating that the seat of obstruction lies in the pectinate ligament in many chronic glaucomas:

(1) Clinical evidences of open filtration angle sometimes persisting for years⁽¹⁷⁾. Uribe Troncoso⁽¹⁸⁾ has recorded a case of glaucoma of some years' standing, with vision reduced to hand

movements, in which the angle of the chamber was seen with the gonioscope to be entirely free.

(2) Raeder's observations⁽¹⁹⁾ showing that the duration of the influence of eserine on the intraocular tension in chronic simple glaucoma corresponds with the duration of its influence on the ciliary muscle, and not with that of its effect on the pupil.

In glaucoma with open sinus, it is difficult to see how contraction of the ciliary muscle can reduce tension in any other way than by opening up the meshes of the pectinate ligament.

Grönholm's observations⁽²⁰⁾ on the influence of miosis alone, in both chronic and acute glaucoma, were valuable. But more or less confusion is inevitable from such observations, unless one can separate cases with closed filtration angle from those with open angle.

B. If the site of obstruction lies in the pectinate ligament, there are only two available explanations of the mechanism of the obstruction, namely, that given above and Thomson Henderson's theory of "sclerosis of the cribriform ligament," which lacks support.

Thomson Henderson's theory of obstruction by thickening of the trabeculae of the pectinate ligament was a most natural assumption. There is no doubt at all of a general tendency to thickening throughout life—both of the hyaline sheaths and of the white fibrous centres of the ligamentous bands.

But this is not nearly enough. Some definite evidence of a glaucoma connection is required. And so far as my own somewhat limited observation goes, the claim has failed to stand the test of verification on glaucomatous eyes. The degree of thickening varies greatly in different eyes. In some old people the lamellae remain quite thin, and this may apply to glaucomatous eyes equally with others. And the more marked examples of thickening that I have come across have certainly not been in glaucomatous eyes.

I once began a series of measurements of the component hard tissues of the ligament, but soon gave them up, as they did not help in my work. Thickening of the hyaline tissue of the ligament, like that of Descemet's membrane, tends to be most marked peripherally, as is well seen in Fig. 4, *i.e.*, beyond the level of Schlemm's canal, and thus beyond the level of filtration. And it is only here that "warty" elevations of the membrane may occasionally be found, as noted by Ranvier in 1881⁽²²⁾.

The terms "sclerosis" and "fibrosis" are hardly appropriate in dealing with bands that in old people may be two-thirds hyaline and one-third fibrous.

PART III

Anatomical Supplement

The following anatomical facts, supporting statements made, have been collected together here in order to leave the main arguments unencumbered with detail.

The *process of exclusion* must be given, by which one is forced to claim the muscle cement as bearing and transmitting some of the tension of the zonule in the muscle area. The only other tissues available are the following :

(1) The muscle fibres themselves hardly need mention in this connection. An actively contractile tissue, whose function is to relax the zonule, could scarcely serve in its resting phase for exactly the reverse purpose.

(2) The dense blocks of fibrous tissue that, with fine interspersed elastic threads, fill up the broad spaces between the thin bands of the inner two-thirds of the ciliary muscle in old people, are essentially to be regarded as *mere packing material*, to maintain the shape and prominence of the ciliary body. In young eyes, this inter-fascicular tissue, though in much smaller quantity, is equally solid, ending abruptly just short of the inner border of the muscle.

In many old people, the tissue, instead of remaining shut in within the muscle area, extends through gaps in the inner muscle net, to occupy a very variable portion of the outer compartment (see below) of the space between the muscle and the *limitans externa*. But even so, the boundary of the solid tissue remains abrupt, and it is only scantily connected by loose tissue with the limiting membrane, except quite anteriorly, where senile fibrosis tends to be rather marked. And it is impossible, on the other side, to trace any continuity between this inter-fascicular tissue and the fibrous centres of the pectinate ligament.

(3) There remain the elastic fibres found in the muscle fasciculi, that certainly must support a portion of the zonular tension.

It is in the minute anatomy of the *hyaline-free space between the muscle and the limitans externa* of the corona, the elevated portion of the ciliary body, as commonly seen in middle and old age, that the old descriptions are most wanting. And the chief interest centres in the elastic tissue found here, supported by loose, collagenous fibres. The general plan of the tissue is expressed fundamentally in the statement that the space is bridged across by numerous elastic fibres. They are most obvious in the posterior two-thirds of the corona, where their general direction roughly

corresponds with that of the radial muscle bundles, when the latter are at all defined. And in the greater part of this posterior two-thirds or more, the course of the fibres is broken by two lines of connection, forming the boundaries of the vessel layer. The inner connecting line is the forward continuation of the elastic

Fig. 17. $\times 67$.

lamina of the orbiculus. The space is thus divided into three compartments, the inner of which always remains quite narrow, as measured in the depths of the valleys between the ciliary processes.

Posteriorly, the fibres are in series with the "tufts" of elastic fibres (Salzmann⁽²³⁾) in which the meridional muscle fasciculi end in the suprachoroidea. And they are here very definitely collected into bundles, coming from the outer radial muscle fasciculi, well seen in Fig. 17. It is difficult or impossible to trace any fibres from

these bundles inwards beyond the elastic lamina; and the collagenous tissue to the inner side of the lamina (the forward continuation of the "interlamellar" tissue of the orbiculus) is very light, and also loosely attached. There is evidently a fairly free range of movement possible here, between the elastic lamina and the limiting membrane, in accommodative changes of the eye.

Slightly further forwards, owing to the fore and aft spread of the muscle to form the inner plate, the elastic fibres in the outer

Inner muscle plate

FIG. 18. $\times 67$.

Nos. 1, 2, 3, 4 placed in the vessel layer opposite small processes of muscle, drawn inwards from the inner muscle plate.

(broadest) of the three divisions of the space are usually fairly evenly spread out. Where, however, there has been much intrusion of solid fibrous tissue from the muscle area into the space, the elastic fibres may rarely be crowded secondarily together into bundles. In Fig. 18 such bundles of elastic fibres have drawn out little processes of muscle from the inner muscle plate (1, 2, 3, 4) emphasizing the almost exclusively muscular connections of the fibres on the muscle side of the space.

Here, the fibres of the middle compartment of the space—the vessel layer—tend to form a network, more or less supporting the veins.

The fibres of the narrow inner compartment of the space, in the valleys between the ciliary processes, still remain extremely fine; but those passing into the processes are strong, as dimly seen in Fig. 11.

Anteriorly, owing to the paucity of veins, most of the fibres pass uninterruptedly from muscle to limiting membrane (see Figs. 17 and 19). Here, they are relatively few in number, isolated in the fibrous tissue. They are seen fairly definitely in Fig. 19 to be in series with the radiations from the dilatator pupillae. Their direction here may therefore differ by an angle approaching 180° from that of the most posterior fibres seen in Fig. 17.

FIG. 19. $\times 67$.

The statement made in Part I needs substantiating, that the total bulk of elastic tissue crossing the hyaline-free space is definitely greater than that to be found in and around the muscle fasciculi that converge towards the pectinate ligament.

There can be no doubt that the amount of elastic tissue in the "tufts," in which the meridional muscle fasciculi end, is much greater than that found in the fasciculi.

A rough examination of the spindle-shaped sections of the muscle "stars" in which the meridional muscle fasciculi end, shows (under lower magnification) a corresponding finding to that given by Barfurth for individual fibres⁽²⁴⁾. As the muscle spindle narrows, the elastic tissue on each side of it thickens progressively to form Salzmann's tuft. Barfurth stated that the attachment of the plain muscle cells took place by means of elastic fibres, which bifurcated at the end of the muscle cell. The two branches extended along each side of this, and were firmly attached to it.

Eyes such as that of Fig. 17 show precisely similar endings of the outer radial muscle fasciculi as given for the individual fibres, in the thickened bundles of elastic fibres that pass to the elastic lamina. And doubtless the same proportionate increase of elastic tissue would be equally obvious further forwards in the fibres that cross the space to the outer limiting membrane, if the muscle fasciculi were not spread out to form the inner net.

The Elastic Ring. The collection of elastic tissue that has been referred to repeatedly under this designation in the foregoing pages, was first described by Stutzer⁽²⁵⁾ in 1898 as a ring of oblique and cross-cut elastic fibres on the mesial side of the angle of the anterior chamber. Hence the suggested name. It is too definite a structure in many eyes to remain without some designation. Prokopenko⁽²⁶⁾ noted that the fibres did not lie in the iris.

In its fullest development, as in Figs. 9 and 11, it is mainly confined to the eyes of middle-aged and old people, with peripherally placed, and often rather poorly developed, ciliary muscle, the anterior surface of which is curved by retraction of the radial muscle heads. The tissue lies immediately in front of the muscle, in the hollow of the cup, but dips down also between the muscle bundles.

It is mainly or entirely a ciliary body structure, *i.e.*, it lies beyond the terminals of the pectinate ligament. And it serves as the chief connecting link between the latter on the outer side, and the peripheral radiations of the dilatator pupillae (Figs. 9 and 16), together with some diffused (Fig. 19) or collected (Fig. 9, *b*) elastic fibres reaching to the limitans externa on the inner side.

Its site of development is seen (for example, in Fig. 19) in eyes with the myopic type of ciliary muscle—backward-sloping and parallel-bundled—that escape marked retraction of the radial muscle heads. It is formed largely between the muscle heads, and, to a varying degree, by thickening of the bases of the elastic fibres that cross the space between the muscle and the limitans externa. In some of these eyes the elastic tissue tends to be spread out along the sloping border of the muscle. A little of this tendency is seen in Fig. 4 of this paper.

Other ciliary muscles that escape marked retraction of the muscle heads are of the powerful, bulky hypermetropic type. In such eyes the development of the elastic tissue remains poor, and it may be more or less broken up between the muscle heads.

The exceptional development seen in some old people, as in Fig. 11, does not indicate exceptional elasticity. The fibres are swollen, degenerate; and some of them may be broken down into droplets.

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AN ENQUIRY INTO THE OCCURRENCE AND EFFECTS OF VOMITING AFTER CATARACT EXTRACTION

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It would seem at first sight difficult to assemble any concrete facts concerning the causes and effects of vomiting after cataract extraction, but it is here my intention to endeavour to indicate the main factors operating in the causation of this phenomenon—one which may at best be described as an ever-present nuisance, and at worst may be a catastrophe spoiling an operation otherwise perfect in all respects—and by following up closely the relevant cases, to gauge the extent of the deleterious effects caused by it.

In the first place it would be appropriate to give details of the routine treatment of cataract patients at Moorfields Eye Hospital. These patients are admitted two days before operation, and a conjunctival swab is taken for bacteriological examination; an aperient is given on the day of admission, and on the following day, if necessary. The eye is irrigated with boric acid lotion twice a day; the