Morbid Anatomy of Glaucoma,
and its relations to
The Theories of Causation.

The subject of Glaucoma is among eye Diseases the most serious, and one of the commonest conditions which one sees in an Ophthalmic Institution. Moreover, it is beyond a doubt, the most puzzling, and from a Scientific point of view the most interesting. These facts were in themselves sufficient to lead one to a closer study of the subject as a whole, but more particularly to the minute changes in the process, which have been the basis of so many interesting Theories.

At the same time, I cannot omit to mention the very great impulse given me to this study by my present teacher and friend, Mr. Teacher Gollino, who has himself contributed a great deal to our knowledge of this self-same subject.

I must mention that I am greatly indebted to the Authorities at Moorfields...
Eye Hospital, for allowing me every facility in this work, for the past seventeen months. My special indebtedness to Mr. Herbert Parsons, the Curator and Librarian, who was very kind in placing at my disposal the use of the appliances of the Pathological Laboratory, besides supplying me with several specimens.

It is my pleasant task now, to lay before the Senate of my Alma Mater, the record of the Pathological conditions which I have found in the systematic and minute examination of a total of 113 cases of the disease.

A great deal has been written, of the morbid changes, by several very able observers. From the time that MacKenzie first pointed out the overfullness of the chambers in 1830; much valuable contributions to the subject have been made, by Dr. Graefe, Max Kries, Sehler, Czermak, Bradley Priestley Smith and many others.

As replete as have been their observations, and as complete as have been their records, so keenly has followed a scientific
controversy; either, in the interpretation of those observations on the one hand; or, in the acceptance of what is more "antecedent" to that which is "cause".

I have, from the very outset, tried to be uninfuenced and unbiased in the examination of my specimens; noting everything, independently of any particular theory; viewing the several changes as only part of a great whole; in elucidating individual facts, and in eliminating others in turn, I thus hope to be reduced to that minimum which may be ranked as invariable antecedents; in these, there is to found the cause or causes, provided this be sufficient sure to distinguish true observation, from that which is only inference from observation.

There has always been a very abundant supply of materials at Moorfields. During the year 1962 alone, the number of Tridestrogy Oper. performed for Glaucoma was 174; in that same period, total N. of Excision of Globe 232, with 9 more in which artificial eyes were put in. A good many specimens have
been given me, by my friends, Mr. G. W. Thompson and Mr. Ruston, surgeons of the Western Ophthalmic Hospital, Melbourne.

The method I have employed, in preparing the specimens for examination, is, in its essential particulars, the same taught and employed at Moorfields.

In many cases, the eye was placed immediately after excision, in Müller's fluid, and kept there for a varying period, according to the part particularly desired for examination; then frozen, and divided in the required directions.

The colloidin method has been largely employed, where, any change in contour seemed prejudicial to a correct estimation of the relation of the parts. For, in the ordinary method of freezing, or cutting sections there is, of necessity, an alteration of the internal parts which leads to fallacy.

In a few cases, shreds of this, removed by trephine, were examined; and, where necessary, the pigmented epithelium was examined after treatment by Mr. Tracer's method.
as modified by Mr. Griffith, which essentially consist in the alternate immersion of specimens into chlorine water and water slightly acidulated with hydrochloric acid; the specimens having been previously embedded in gelatin. This is an excellent method for depigmenting epithelium and allow of a clear examination.

It is, perhaps, unnecessary to dwell upon the minute details of the processes employed in the preparation of the specimens.

In presenting the results of the pathological findings, I have to content myself in this thesis, with recording minutely the lesions in these parts which are most commonly the sites of the greatest morbid change.

In recording a large number of cases with the constant examination of the same parts in most, it is most difficult to avoid a considerable amount of repetition.

I am therefore compelled to use a little discretion in giving minute details in some cases, and more abstract in others; using as many abbreviations as are necessary, and which admit of no ambiguity.
In what order can I best record the cases examined? This is a question not without much difficulty. What seemed to me the ideal pathological report of such an examination, should, I think, commence with the cases of the shortest duration; for in these are to be found the initial lesion or lesions; at the same time, these are less likely to present the exhaustive lesions or complications, which may exist, in the cases which are most advanced. I mean that the cases of longer duration are likely to show other pathological lesions, which are either further progressive changes in the same process, or else more consequences, of a general pathological nature and thus entirely masking the initial lesion. This method, in many cases is difficult, from the fact that the cases of undoubted short duration, which would be the most useful, are not so easily obtainable. In the earliest stage of the disease, the patients are hardly ever ready to consent to such a radical measure, as excision, straightway;
The surgeon himself is bound to give every opportunity to less radical measures, employing Plidectomy or Hyperic as the nature of the case demands, or mere palliative measures, even though they be not the slightest chance of any usefulness in the eye.

Further, this method is not wholly reliable. The duration of the disease can only be roughly estimated from the history, and this may lead one into errors. So a correct history depends, so much, on the intelligence of the patient, and, even if intelligent, the onset of the disease may be so insidious as to entirely escape even the keenest observations. What then appears to us as a case of short duration is really one of long duration, with an acute interruption, or sudden exacerbation.

Another method, which suggested itself to me, is to divide the cases up into Groups of Acute cases and Chronic cases, treating the Sub-acute and Intermittent types, as Chronic cases varying in intensity and rapidity.

This method, though very important
clinically, cannot have such favour in a Pathological Record of this nature. The very basis of the division, may rest on some additional unknown morbid agent, which manifest itself in the degree of obvious inflammatory changes. Acute Glaucoma is often spoken of as an inflammatory disease; true are, of course, changes of an inflammatory nature; true is, however, no evidence to show that the inflammation originates Glaucoma. The evidence which contradicts such a supposition is too thoroughly opposed to such a view. The most potent being the suddenness of the attack; if it were inflammatory, how could Iridectomy, so thoroughly relieve it. It is not out of place here to quote the words of Sir W. Bowman "Glaucoma is in its essence not an inflammation, and, when inflammatory, only so as it were by accident or complication." Why should a drop of Atrophic suddenly cause an attack of inflammation? Mr. George Ritchelt regarded it as a surgeon today regard 'Strangulated Hernia.'
Yet another method is the division into Primary and Secondary. The meaning of the words being the same as generally employed in all text-books, based on the analogy of malignant growths. Secondary, implying that some morbid condition pre-existed, which is assumed to give rise to the Increased Tension and its sequela.

This is a very useful division from the clinical point of view, in influencing more especially the mode of treatment.

To make such a division for a Pathological purpose, the first thing to be settled is, whether the Increased Tension and its sequela, from a demonstrable cause, is identical with the obscure cases of the same condition, obscure only in so far that there is no demonstrable pre-existing condition.

Most are agreed that the issue of Glaucoma is directly traceable to the Increased Tension, which, saw of itself, explains all the other phenomena, Pain, Progressive loss of Vision, Crippling of Disc, etc.
The obscurity of the causes of Primary Glaucoma
then resolving itself into the obscurity
of the cause or causes of High Tension.
In an investigation of this sort, one cannot
neglect the consideration of Secondary Glaucoma.
For to do so, would be to suppress most
valuable information, which could be obtained
from a close study of the process in
Secondary Glaucoma and comparing it
with the findings in cases of Primary
Glaucoma. The great difficulty would be,
to fully appreciate, and be able to dis-
 criminate, those changes which are due
to the pre-existing condition, and those
which are due to the subsequent Increased
Pressure.
By a combined method of Agreement and
Difference, the inference drawn from the comparison of the
Pathological changes in Primary and Secondary
Glaucomas, must afford some certain
knowledge.
One cannot overlook the fact, that in
a certain degree, every case of Primary
Glaucoma, could, as soon as we knew
its cause, be placed in the category of
Secondary Glaucomas. In the usual acceptance of the terms, however, we mean that some demonstrable, obvious, antecedent disease condition forms the distinction. In the selection of a few cases of secondary Glaucomas for examination, those only, affected to our wherein this antecedent diseased condition was of such a nature, as least calculated to present many anomalous morbid changes.

Yet a fourth method would be to record the cases in groups, the basis of the grouping being the special part showing the most marked changes. A comparison of the History of cases in any group, and the comparison of the History in different groups, would be useful in many respects. It is clear that if some particular part was always affected, when any factor in the History was always prominent; then the former could be anticipated, if the latter was known to be present.

This method of recording the cases cannot however be adopted; for the basis of grouping will be found to be too
too narrowly restricted. Many cases presenting changes in several parts, and thus, the groups would overlap each other.

A few other methods, I have thought of, but they are unsuited and do not merit any share.

Why, after all, should so much importance be placed on the selection of any method for recording the cases? What advantages are to be derived?

The chief advantage is, to readily enable one to summarize the individual facts; to see at a glance, if possible, the relation of cause and effect; and, at the same time, to avoid or to facilitate useless repetitions.

As no one method seemed to meet all the requirements, I have decided to choose a medium one, by a sort of combination, namely, cases into Primary and Secondary. The Primary further grouped into cases of Short Duration and Long Duration. The cases of doubtful Short Duration coming under Long Duration.
Primary Short Duration.

1. History

Mary P., age 63: Glaucoma of right eye of 4 months duration; farm servant, hard drops put in by family doctor, only temporary relief could count fingers up to 3 months ago; now P. L.; O. E. somewhat shallow, pupil inactive, pain worse or less continuous. T + 2.

Specimens showing:
(i) Biliary Region by meridional sections upper transplant.
(ii) Optic Disc with adjacent Ch. and Ret.
(iii) Vertical section through sclerotic, Ch. and Ret. near periphery to contain long biliary nerve and posterior long biliary artery in its course forwards.
(iv) Trans. Sect. of Optic Nerve, little posterior to entrance into Globe.

(v) Several equatorial fine sections to show pigment epithelium and eff. vessels of ciliary region.

Necrotic Changes:

Biliary Region

Biliary muscle reduced in bulk, circular fibres smooth, atrophied, a delicate fibrous tissue intervening between the muscular remnants.
longitudinal fibres of the external part of the muscle, show little change of this nature. The connecting fibres between these, the radial fibres, become more distinctly visible; the bundles are prominent between the newly infiltrating tissue, in which occur some nuclear tissue and some blood vessels.

Biliary body on its internal border occurs a thin layer of newly formed nuclear bodies, the epithelium is disintegrated in parts, especially towards the apex near the stroma. The non-pigmented layer of epithelium is interrupted showing the structureless deeply pigmented internal layer of pigment cells. Here there appears vacuoles between fibres of Scone of Ziem and eil body.

Blood vessels dilated and varicose, lining endothelium of the larger ones, show slight proliferation, while in others the wall is thickened. The eil. folds are altered in fulness, somewhat swollen, the clefts between the folds are much narrowed. the anti. folds are very close to, but not in contact with, post. surface of the iris base. The space between the eil. folds
and lens (circumetal space) shows great reduction, though no part of the swollen folds actually touches the lens.

Iris

It is thinned and attenuated, pupillary portion and a greater part of the cili. zone of the iris lies quite free, the remaining portion of cili. zone (about 1/3) being closely applied to ligamentum pectinatum and post. surface of cornea.

The contraction grooves on the anti. surface are obliterated. Posterior layer of pigment cells seems to have undergone some disintegration. The pupillary margin presents a tuft of this heaped up pig. epith. There is little trace of the bundles which represent the sphincter pupillae.

In the body of the iris there is dense looking connective tissue with few or blood vessels, some of these are nerve. remains.

The pupillaty process bulges forwards and outwards, the anti. surface is in immediate contact with the rim of the anti. chamber of the aqueous, and posterior...
surface of the cornea for a short distance. Ligamentum Recitimation.

This is denser in appearance, its meshes representing the spaces of Fontana are completely obliterated and show a few faintly staining nuclear bodies.

The peel, portion which corresponds to its attachment, seems dragged upon it being pulled backwards & outwards. Vocal of Schlemm's.

This is separated by a more thornt, the walls being in apposition, and is represented by a more linear piece of denser connective in which two nuclear bodies are seen. Its position, in consequence of the alteration in position of iris, seems placed much further back than in the normal.

Lens and Suspensory Lig.

Lens was in an early cataractous state, its capsule showed a slight degree of thickening by an apparent delicate plastic looking material. Susp. Lig. somewhat more dense and
Shallow, incroached upon by the anterior part of lens. Appears shorter by obliteration of angle. Aqueous very opaque.

Very greatly edematous; the most superficial layer of transitional epithelium being studded with vacuoles containing a homogeneous translucent material; the anterior surface is very irregular, so many cells have been loosened. The nuclei stain very deeply, the outline of the cells, under a higher power seems to be intensified except in those cells immediately adjacent to Bowman's membrane. B's membrane seems somewhat swollen, and in some parts, are indications of slight solution of continuity; though no break could be traced through its entire thickness.

The stroma on the other hand shows changes of an opposite character, appearing narrower, the lamellae seem more closely applied to each other. There are fewer
corneal corpuscles. Many of the so-called lymph spaces in which they lie being destitute of any cells and show no sign of nuclei.

The posterior limiting membrane (Descemet's) can be traced without difficulty and is small, though many of the epithelial cells lining its internal surface have desquamated and thus a sort of irregular beaded appearance is presented.

Ophie Disc.

Moderately deep cup, extending however only over the nasal half, with a gradual slope of the other half. There is a striking back-wards displacement of the Lamina Cribrosa; it seems very much denser in the deeper part of the excavation, it moreover lies more posterior than the sclerotic coat.

On the surface of the cup there is some adhesion of the nerve fibres and between the fibres some delicate connective tissue. The Ophic Nerve in trans. sect. shows somewhat similar changes; the nerve fibres are much narrowed and also,
compressed by the addition of trabeculae of connective tissue between them. The blood vessels stand out very prominently in this section, and can be best studied here.

The greater number, both large and small, being surrounded by a layer of loose fibrous tissue, varying in thickness, concentrically arranged, the nuclei staining very darkly.

This fibrous tissue merges into and can be traced on into connective with that which lie between the nerve bundles.

The lumen of the vessels in many cases show slight proliferation of the lining endothelial, while the lumen in others is entirely obliterated.

The nerve bundles have lost their usual rounded appearance, being somewhat irregular instead of rounded. Even the nerve sheath is not unaffected, as there seems to be the same deposition of newly formed connective tissue here.
Blood Vessels.

In all the vessels, almost without exception, there is some definite change; most characteristic changes are seen in the ciliary region. In most the dilatation is pronounced, the wall is thinned, and the endothelium here and there proliferating. The circleus arteriosus iridis major in meridional section was specially examined. Further back the venae vorticosae show the same change; although the dilatation was not so pronounced in proportion to their calibre.

In the optic nerve region, changes described before (optic nerve), the arteries seem to be more greatly affected than the venous trunks; their walls instead of being thinned, however, are rather thickened; and not a few of the smaller ones, by pressure is obliterated entirely or nearly so. The long internal or posterior ciliary artery is affected like the venae vorticosae; in having its walls thinned and
humer dilated. It is not always easy
to recognize this vessel unless the section
happily strikes the sclerotic coat where
the vessels pierce it.

Choroid. This does not show any marked
change except sharing in the change
of the vessels generally though to a less
extent.

Briefly reviewing from within outwards: the
pigment layer is of a more delicate
appearance, as though the pigment were partially
washed out: it is however not sufficiently
depigmented as to show any definite cellular
elements.

The layer of capillaries (chorio-capillaris) a
membrane of Brugschi, shows an alteration
as though it were unsupported; their
lumen dilated, and the interfaces between
the loops of capillaries absent. The
homogenous membrane lying between these
two (lamina vilica), becomes distinctly
visible in contrast with the pigmented
layer of epithelium, which is itself
closely applied, and with difficulty differentiated
from the adjacent Retina.

The layers of "medium-sized" and that of "large vessels" (Kaller's) are greatly reduced in thickness, their lumens smaller.

The intervening supporting structures thicken; the fibrous sheaths around the arteries will marked. In the sheaths of this layer the pigment cells are not so apparent. The pigment seems rarified.

The supra choroid and sclerotic seem more intimately blended together; the former only discernible by the well marked pigment; the pigment here has apparently suffered less rarefaction than in the other layers.

Retina

The changes were in general conformity with those described under Blood vessels. Many vessels were filled with red blood corpuscles, the walls of the arteries were thinned, especially in the region of the optic Papilla.

The Retina showed slight detachments from the choroid; leaving pigment layer attached to the latter. The detachments
appearing as mere rhincks or linear spaces.
A minute examination from without inward.

External limiting membrane cannot be recognized in its whole extent; some parts broken up; in other parts, altogether wanting.
The layer of Rods and cones have lost its
their regularity; many cells only showing
the remains of nuclei; others are lying
in a direction parallel to surface
of the limiting membrane as though
lying flat upon it.
The outer granular + outer reticular layers
cannot be differentiated they show however,
an excess of pigment cells and pigment
masses.

The Inner Granular + Inner Reticular layers
can be distinguished from the above
but not easily from each other.
The cell bodies are almost lost, the
nuclei alone being seen.
In the inner reticular layer minute
clusters of red blood corpuscles are
seen lying apparently free; other
clusters are represented by a lightly
coloured mass of pigment.
The coarse meshed network in the
 succeeding layer shows few ganglion cells,
 but remains of the nerve fibres are seen.
The supporting tissue is very attenuated
 except in the immediate vicinity of the
 larger vessels where it is denser.
The smaller vessels have somewhat hyaline
 looking walls.
The so-called lymph spaces here cannot be
 made out.

Vitreous

The only noticeable change was
 a slight detachment on the nasal side
 near the optic bulb.
A few irregular white opacities were
 scattered in it.
The hyaloid membrane was somewhat
 thickened in its anterior segment.

Remarks

Five months after extirpation the woman
 returned to hospital complaining that for
 the past 6 weeks she had noticed that
 the left eye is getting bad; everything seems
 foggy for several hours, then she see
quite well.
She was taking medicines for Neuralgia which first came on since the operation. For the past fortnight she has been having pain on the left side of forehead.
On examination - slight episcleitis. Pupils doubtful + Pupil semi-dilated, acting sluggish, anterior chamber fairly deep. Cornea shows slight haziness, media quite transparent, no cupping but well marked palatation.
Iridecetomy was performed a week later.

2. History.
Jonathan R. aged 37. Suddenly seized one night after work, with severe pain in R. Eye. Pain lasted about 3 hours, was quite well for 2 months after that, when he had another attack of pain accompanied by photophobia & lacrimation, the pain was not as severe as in the first attack, but it lasted longer. He at this time objected to operation. Emin's treatment for 1½ months, when he ceased coming to hospital.
Eye was finally excised 7 months later when prodromal symptoms began in the left.
The right now still a little painful. Completely lost P.C. T+2, aqueous opaque, pupil fixed, A. C. shallow, cornea dull and without retraction.

Irideotomy was performed on left, in which already there was a slight contraction of field on nasal side, with a large scotoma on temporal side.

Optic disc constricted, pulsation not spontaneous but elicited on very slight pressure.

Congestion of the episcleral vessels:

Specimens:

1. Bilian Region
2. Optic Disc
3. Vertical section through S. O., Ch. and Ret.

Bilian Region.

This shows the most pronounced changes.

Bilian muscle. All the division of family, good size and shape. Much proliferated.

The change is very much like that in the brain. Atrophy of circular fibres is not so marked.

On external border, newly formed homogeneous layer shedded with a few nuclei. Blood vessels dilated various to a greater
After much attenuation. Position completely altered. Its base seeming to originate directly from the corneoscleral junction.

Its post. surface is much shrivelled and very irregular; in a small part in contact with the inner fold.

Ant. surface. It is free at its base for a very short distance the rest of its base is closely applied to post. surface of the cornea its periphery is quite free.

Pupillary margin does not show the eversion of pigment as in no.1.

Scleral Pupil visible as an elongated thin band.
The blood vessels, dilatation of circular, act. iris. maj. prof. of nuclei around vessels but not much within the lumen.

Lig. Post. here represented by a well marked thin almost fibrous band the greater part is free from the amp. of the iris.

Ligament of Schlemm.

Does not seem entirely blocked though very small some sign of a lumen remains, there are several deeply staining nuclei around it and a few in the lumen. On section it is elliptical.

Lius and suspensory ligament.

Lius shows great disproportion to size of globe, this led to actual measurements which were:

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It was occupying a slightly advanced position.

Suspensory Ligament.

False; capsule showed several opacities, no change in lens substance.
Anterior Chamber

This is represented by an elongated channel, with an isolated part in the region of the ciliary. Its walls here have a very fine homogeneous whitish coating.

Aqueous

Opaque and have several very dense loose flocculi.

Iris, C.

Greatly oedematous, with disorganization on the ante. surface marked proliferation of the pigment epithelium. Minute flakes, which in colour adhering to its post. surface.

Optic Disc

Very shallow central pit, lamina crib. pushed back to a slight extent. Slight atrophic change in nerve fibres on disc.

Optic Nerve, shows little change except a sort of vascularization, vessels distended with blood corpuscles, their adventitia show an early sclerosis. Many nuclei being scattered around but not a definite formation of connective tissue. Some vessels show slight proliferation of lining epithelium.
Blood Vessels

These all afford some proof of vascular disturbance. The ciliary region showing the most pronounced change of dilatation and profusion of endothelium. Same change is admirably seen in the large vessels, vein, vasa vasorum, central artery, and art. irid. major.

Choroid

Compressed and slight atrophic change: it does not present signs of any infarction. Characteristic though there is much disintegration and disorganization of the pigmentary elements, in some parts the pigment is entirely wanting.

Retina — chiefly shows vascular engorgement: humor filled with red blood corpuscles; minute extravasations, shriveling of the nervous elements at the expense of a nearly formed delicate cor. tissue. The retina was not detached anywhere from the choroid.

The rods and cones show the same condition as in case 1, being for the most part disintegrated, but not in any degree as in the preceding case.
Vitreous.

Several thick dense masses, many uniform in shape, hyaloid membrane shows thickening all round.

3. Georgiana B., age 49: domestic. For the last 3 months she has been suffering from severe neuralgia on the left side. She attended General Hospital for same, a week after she began taking the medicine, she thought the neuralgia was getting worse, but more now than before the pain was more severe at the back of the eye and over left side forehead; her eyes began to water and became very red. She was then sent from General Hoop. There was a great deal of injection of the episcleral vessels, the pupil was dilated and irregular, large dark-purple venous knobs were seen swelling on the scleral media, not transparent. She could not count fingers at a couple feet but perceived shadow. Sutectomy was performed.

9 months later when at the seaside she had another attack in the same
eye it lasted 2 days - now she has returned complaining of a constant pain. Eye excised.

Specimens

1. Bilary Region several specimens.
2. Optic Disc, nerve etc.
3. Vertical section through fifth, eth.

Biliary Region

The change here was of an interesting character: biliary muscle showed a moderate degree of atrophy in both the circular and longitudinal fibres. The internal border was covered with caudalis with minute flakes projecting from it. The vessels were engorged and a marked thinning of their walls was present; in the upper part, there were several varicose dilatations.

Between the muscle bundles, there was a great multiplication of nuclei, also a translucent material.

The biliary folds were separated from.

The loops lies margin by an unusual
large space

Iris — was very much flattened and applied to the inner, neat, and not the surface of the cornea, at least throughout its entire extent; a very small portion of the extreme peripheral peripheral portion alone lying free in the aqueous. The pigmented epithelium on the posterior surface was very much scattered, by which it seemed to be less pigmented than usual. In the region of the collumna, there was still the base of the iris firmly adherent to the ligamentum. The irises were apparently a little in front of the annulus. Attachment of the latter, the cut end of the iris base was adherent to the cicatricial tissue which was shown by a small indentation and the pig under the iris formed the floor of this indentation.

Lig. Posterior — much thickened, large number of nuclei scattered about in its immediate neighborhood.
Panel of Schlemm... closed much fibrous tissue around it of a fairly dense character.
Lens and Sph. Lig.

The lens was very much advanced.
and almost abolished the aqueous chamber.
Sph. Lig. moderately stretched, much
elastic material adhering to it.
Ant. Chamber

Very much reduced by encroachment
of the Lens. dense opacities of a membraneous character.

Cornea - changes as in (1) and (ii)
Optic Disc - slight central depression,
margin a little swollen.

Optic Nerve showed changes so similar
in nature to those in case 1. that it
is almost useless to repeat; the same
maybe said with regard to the choroid
and retina.

Vitrous presented several minute threads
and flaky particles, more especially
abundant in the region of the vitreous body.
In commencing this my Thesis, my original intention was to give a record of all the cases I had examined and the morbid changes which they presented. I must confess that I did not fully appreciate the amount of repetition that such a record would entail; and what is equally opposed to such a record is the amount of space required to record these 113 cases. So far I have only recorded 3 cases and the repetition is already assuming proportions. To record 110 more, with scarcely any new additional morbid change to add, would be unbearable long before I reached the middle of my record; and I am sure the reader would also find it so to a greater extent. If I could think that this Thesis would not receive its full consideration, because I have suppressed perhaps more than two hundred pages of what is unbearable, then I should most willingly proceed to the record
of all the other cases in the same manner
as I have done in the two preceding
ones.
I should not hesitate to so record them if they
were calculated to throw more light on, or
more clearly indicate the merid
changes, with which the first part of
my thesis is concerned.
In view of having to deal with the relation
of these merid changes to the theories
of causation; this, being by far the more
important consideration, will have to be
reviewed at some length. It is therefore
necessary for me to adopt some other method
in order to keep my thesis within a reasonable
length and save the necessity of too monotonous
a repetition.
As I have indicated the changes pretty minutely
in Nos. 1 and 2; and as these coincide with the
changes in the remaining cases, I shall only
point to them by the use of broader terms in
the most terse manner.
St. Julian A. age 47.

History - 15 months. Glaucoma.

Morbidity changes.

Scl. muscles: Of fairly good size, moderate inflamed.
slight vessel:

Iris - Peripherally inflamed, applied to cornea.

Lumen of Schlemm - Lumen closed, protuberance of nuclei around lig. pect.

Blood vessels:

Orb. Region - Enlarged, footlift, endothelium.

Optic Disc - Shallow pit, uniform, margin swollen.

Pitire nerve - Moderate vascularization, slight edema.

Retina - Venous diluted with red bl. esp.

adventitia thin, protein layer.

Choroid - slight vascular dilation.

Protein of pigment.

Aqueous humor - slight opalescence.

Lens ligament - slightly advanced.
History. John W. age 63.

11 months drunk.

Car. Pupil. Little inflamed.

Moderate atrophy. Slight sclerotic plaque.

Iris - Periphery of iris was not adherent to the iris, a small canal separated it from the ciliary body to which it was not applied. This is one of the cases of my whole series that the iris was entirely free.

Iris of Schlemm's - Very much inflamed, hazy, almost obliterated.

Phacocornea - Dilatd. & Proptosis around.

Optic Disc - Slight central depression, marquis margins.

Optic Nerve - Much vascularized, /& protuberant.

Retina - Vascular changes.

Aqueous - Alburnus of 70%, small blood clot.

Remarks.

Much epithelitis.
6. John J. age 69
   History 14 months, Tosst

7. Ethel J. age 51
   History 13 months

8. Jim J. age 49
   History 17 months

These cases all agreed as well as the changes
that they may be summarized.

And. Macul. - Very thin, much lift, round
areas of vitreous. Infarct.

Disc - in all, adherent, periphery grey, blow-out.

Ret.

Optic Disc - Fairly deep cup.

Optic Nerve - Slight, infarct of shrunken
Vessels not much enlarged.

No autopathology.

Agueous Viscera - no opacities, but capsule
thickened.

Lens: all three enlarged beyond what
they should be at their respective ages
according to Mr. Presbyte Smith's table.
In the cases of short duration I have never included any with a history of over 18 months. The number was 33 in all, the changes are so precisely the same in nature that they do not call for any special description. In one more however of this series the skin was not adherent but in it there was a considerable alteration in position & contour of the angle. I shall receive its description and consider it with two others cases, which closely resembled it in discussing Thoein's relation to Palsy or chemical alteration of intraocular fluids.

I have classified 11 other cases as of doubtful duration, insomuch as the history was unreliable. These cases presented the very same changes as most of those of short duration. In 3 however the degree of atrophy was so pronounced in the region of the optic papilla and the cupping so extensive, that they more closely resembled there cases existed as of long duration.

In all 11 cases the size of the lens was very much increased above that
increase which would correspond to their respective ages.

Long Duration.

This was the largest number of cases that was obtained as it numbered 39 in all. These cases varied much in the respect of length of disease. The minimum being 18 months, the case of longest duration I had was of an intermittent character 18 years. There were 11 cases of over 10 years duration, the greater number lasted between 3 and 9 years. I shall briefly describe a typical case:

Andrew W., age 62.

Right eye failing slightly for 5½ years painful 5 months ago, Silicone in left. R. now has no P.D.

Scleral Body:

T. Processes, somewhat thickened, pointed and muscle much thickly, dense connective tissue between muscular bundle.

Blood vessels tightly adherent, no protein.

Resorbein.
Sir. Periphery of iris firmly adherent to retinal surface. \textit{Gonioscopy} much reduced in bulk.

\textit{Canal of Schlemm} & \textit{Lig. Ret.}

Canal completely closed. Annulated by well dense fibrous tissue.

\textit{Lig. Ret.} will marked moderately thick fibrous band represents it.

\textit{Bloodvessels} not much enlarged; dense chronic concentric arrangement.

\textit{Optic Disc} deep cup, sides very much excavated, papilla nerve fibers very much atrophied.

\textit{Lamina cribroa} below level of the sclerotic coat.

\textit{Optic Nerve} moderate degree of atrophy, moderate atrophy of the inner. Increase some translucent substance in the peri vascular space.

\textit{Retina} rods & cones completely disintegrated

\textit{Nearly recognizable}: arteries have somewhat hypertrophied walls, smaller ones have their lumen standing out prominent, others obliterated, there is a
hyaline-looking appearance in the Some
of the small vessels. Large nuclei are seen
there. The choroid + retina, both atrophied,
an adherent.

Blood vessel.

Some have hydrosphyd walls, some
small ones are hyaline, oil region, some
show more cont. around

Choroid - Atrophy is marked. Very
rupt disintegration of pigment, vessels change as above

Nuclei - few speciality of a membranous

Character:

Nuclei not thickened.

The most characteristic change of the whole
of this series is the invariable presence
of a very well marked "cup", which though
it differs much in the atrophic change its
depth, yet its nature is always well seen
being an expression of the result of mechanical
pressure and not of a pure atrophic
change.
Secondary Glaucoma.

The remaining three cases belong to this series.

In 4 of these the condition was exactly the same. All had a history of repeated attacks of Iritis.

The pupil margin remained well adherent to the lens capsule: the cause of the Glaucoma was Anular Posterior Synechiae;

The essential change that may be added was complete exclusion of the pupil. Ant. chamber represented by a small triangular space completely isolated from the posterior part.

The filtration angle closed.

Iris for a large part applied to cornea. Small accumulation of fluid behind it. Lens somewhat advanced.

In one of these 4 there was also a detachment of the Retina.

In 5 cases -

There was anterior synechiae, from corneal plexus. In all 3 - the Iris was adherent at its base.
and for a large part of its extent.
In 3 cases of these the lens was very
much advanced.
My next case was a blow caused by a
clioleation of the lens by a piece
slow on the eye some months previous.
The iris was jammed against the
cornea and kept there by the dilated
diaphragm.
There was nothing of an essentially different
morbid condition in these cases to call
for any detail account.
These cases all have this in common
with the cases of Primary Glaucoma
that the Iris is completely altered in
its relations.
I have now to pass on to
the more important consideration of the
nature of Glaucoma.
The morbid changes are well known
and well understood but the
origin of these changes are still in
dispute.
It is now established, beyond a doubt, both by Physiology, General Pathology and Physical laws, that all the changes which have been observed in connection with Glaucoma can be attributed as consequences of an Increased Pressure within the Globe.

A diagnosis of Glaucoma is only arrived at when there exist marked signs of such pressure; either present at the time of examination, or which can be proved to have existed some time before.

The term Glaucoma has undergone considerable limitation in the course of time. Originally employed to denote merely any condition represented by a greenish or greenish-blue colour of the Pupil. Such a colour replacing the normal black Pupil may arise from any superficial affection in connection with the Lens or adjacent parts.

With the advance of knowledge such superficial affections have been more clearly defined, and aptly removed from the category of Glaucoma, to that of Lenticular Opacities. The term
Glaucoma has now come to be restricted, not to such superficial appearances but to definite disease affecting generally the whole eye.

The pioneer of this great achievement has been MacKenzie, who in 1830, first pointed out the overfullness of the chambers in Glaucoma, and made the first attempt to relieve the same by puncturing the coats of the eye.

A quarter of a century after this, von Graefe called attention to the pitting and circulation of the extremity of the optic nerve; he assumed that along with this condition of the nerve, there must be such a pressure on the whole retina as might be expected to impede or destroy its function. Further, he gave it as his opinion that the increased fulness was no mere accident or complication, but the essential cause of the remarkable train of symptoms.

Through this conviction he was able to achieve one of the greatest triumphs in surgery, by giving to the art his great discovery of the
Beneficial action of Sudeck's paracentesis.

Though, from the earliest date of the history of some definite knowledge of the disease, when high tension or increased fulness was pointed out as a feature of it, working on this assumption, most beneficial results have been obtained. Nonetheless, this was outside the majority who regard increased tension as almost synonymous with Glaucoma, not a few extremists (if we may call them so) who take advantage of the absence of obvious increased tension in some cases of Glaucoma, to assert and support their views that what is generally called Glaucoma is not materially connected with high tension.

Such views are only maintained on the ground that there exists cases with a contracted field of vision, marked cupping of the disc, etc., in which there has been no discoverable increased fulness.

By virtue of this they maintain that Glaucoma is the manifestation of some unknown agent, which though
usually excavating the disc and raising the tension at the same time, not infrequently excavates the disc without raising the tension.

These cases might be considered as exceptions to a general rule. But here we are not dealing with mere Exceptions or mere General Rules. Every exception to a Physiological truth confirmed by Pathological evidence and supported by Physical Laws deserves an explanation on the same grounds.

It is impossible to explain the typical Glaucoma cup by any Physical, Physiological or Pathological agent which does not embody as one of its factors Pressure. The typical cup can be formed by Pressure alone; or by Pressure and Atrophy together. Pressure, of itself, is a cause of Atrophy, as is well known in general Pathology. Atrophy alone does not form the true glaucoma cup.

Is this agent then one that grinds away the tissue; producing results analogous to the appearances of those
wanes, with undermined edges which are met with in the intestines and elsewhere. The analogy is only a superficial one. For the glaucoma cup has too close a relation to the degree and particularly to the duration of the pressure. The pathological evidence supports this, while it offers not the slightest indication of any erosive action.

Cases on 1 and 2 of short duration, are good examples of the early microscopic appearances. These give indications of the earliest change namely, a pushing back of the lamina cribrosa. Compare these two with several of long duration, where the cupping is very far advanced, the lamina cribrosa in many cases still be traced, the atrophy is marked and cannot be mistaken for erosion of tissue.

Experiments have been performed on the healthy excised eye, by the application of artificial pressure and the same displacement can be noticed. How they can we harmonise.
the apparently anomalous cases of Normal Tension in Glaucoma?

Every science and every question in science is first a matter of fact only then a matter of quantity, and by degrees it becomes more and more precisely quantitative.

By this method of Induction, our first question then becomes: Is it a "fact" that there are such anomalous cases, which is recognized as, and eventually produces that pathological change commonly summarized as Glaucoma, with a Normal Tension Throughout the disease?

The opponents to this theory have set themselves a most difficult problem to establish this "fact." The task seems unnecessary. It is easy to see how such an assumption is unsubstantial because (1) the excess of pressure is sometimes too slight to be appreciated and still less to be estimated either by finger test or the more unreliable mechanical instruments—onometers, manometers which only furnish...
correct results within certain limits.

(ii) The more important source of fallacy however is that the excess of pressure is often intermittent. High tension alternating with normal tension.

Thus a case of true Glaucoma may for weeks or even months present a normal tension, such a case has been fully recorded.

There is no reason then to depart from the ample evidence that the True Glaucomatous Process is the result of Increased Pressure. The variability of this process being dependent upon so is an indication of: (i) the degree and duration of the pressure.

(ii) the initial cause or causes which gave rise to and maintains this increased pressure.

The possibility of this view being incorrect is conceivable. If however the anomalous cases are proved correct then they merit an entirely new nomenclature.

In this way, admitting that Glaucoma is the expression of High Tension, we
next proceed to the consideration of High Tension.

What conditions can cause an increased hardness in a closed chamber? obviously:
1. Diminution of Capacity of Chamber
2. Increase in the contents.

In a chamber, such as the Eye, where we have to deal with an imperfectly closed chamber. What conditions can cause an increased pressure? clearly:
1. Inflow must exceed outflow — capacity remaining the same.
2. Diminution of Capacity — inflow and outflow remaining the same.

How can the inflow exceed the outflow?
Either by the inflow being interfered with or the outflow being interfered with.

How can it? the inflow be interfered with? In two different ways (a) from within — Hypersecretion — (b) from without — Increased Blood flow —

How can it? the outflow be interfered with? In two different ways (a) from within — alteration in contents, Pressure — (b) from without — Impedes outflow, Obstruction —
A genealogical tree may be constructed to show these connections.

```
  Eye
    Wall     Contents
      Shrinkage of Tissues       Inflow       Outflow
                        Local or General Hypertension
                          Vascular Distortion
                  Porosity  Obstruction
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Each one of these factors acting separately is capable of giving rise to an increased pressure. More readily, then will a combination of them— if of the same intensity— give rise to and exaggerate the effect.

Each one has had the merit of being regarded as the originating circumstance of Glaucomatous attack. Theories have been framed having one of these factors as the fundamental or underlying cause of Glaucoma. In this sense, the so-called theories are
still merely hypotheses.
I have now to review to what extent
the pathological changes confirm or
contradict these various theories.
The first I shall treat of that known as
the Retention Theory. It is by far the most
important, the most recent and the one
which is the most generally accepted.
This theory is more than any other well
supported by the evidence which the
Pathological study furnishes.
Retention Theory.

The authors of this theory
were Max Kries and Adolf Weber. These
observers, in 1876, working quite independently,
ascertained that in Glaucomatous eyes
the periphery of the Iris lies in
contact with the periphery of the cornea
in the region of the Canal of Schlemm,
venous plexus, and Ligamentum Post-
iridium.

Such an observation—however strikingly
abnormal—could hardly be of much
practical use, nor could it furnish
any material for correct inference.
Unless the significance of such a change was known.
Consequently much of the credit is due to Leber whose original experiments and observations led to our knowledge of the course of the effete intra-ocular fluids.
Leber's observations were made some years previous to this theory; which may be regarded as the direct outcome of his researches.
I fully appreciate the value of the observations of Leber, one must have a clear notion of the intra-ocular pressure and its relations to the channels of entrance and those of exit of the intra-ocular fluids.
A brief sketch then, of these three items is necessary to the full comprehension of the Retention Theory. Even apart from the theory, to fully detect the abnormal one must have a clear understanding of the normal intra-ocular pressure, and such variations of it as are outside the pathological limit.
I shall first treat the intra-ocular...
pressure, then the channels of entrance and exit, as truly as possible.

So one subject has perhaps been more the region of investigations than the study in connection with the estimation, variations of the intra-ocular pressure, chiefly of an experimental nature.

For its exact measurement a manometer has been employed, one arm of which is connected with a cannula, the other being introduced into the eye. It has been found that the normal intra-ocular pressure in a healthy human eye is equal to 26 millimetres of mercury, or rather equals that of a column of Hg. 26 m.m. high. This method, however, is not generally applicable because dangerous.

Tonometers have therefore been devised one of the most useful is that of Priestley Smith. The principle of all is the same. The amount of hardness is estimated by impinging the instrument against the eye. The area of impression or depth of impression being noted.

Besides the Exp., above by Wahlforss
on the human eye. Several others have been performed on cats, dogs, rabbits. They all have this in common that most observers have found very close agreement of the results.

They may be summarised:

The Pressure 20 to 30 millimetres of mercury.
The intraocular Tension rises with any rise in the general blood pressure, or with any active local dilatation.

It also varies with Respiration and Pulse.

Several things also cause it to vary. Chloroform, Atropine, A.C.E.

Contraction of both extrinsic and intrinsic ocular muscle cause a rise.

Ligation of carotid causes a fall.

Ligation of vessel posterior causes a rise.

What is still more important to the Retention Theory is the relation of Pressure in the two different chambers of the eye, namely the Anterior Chamber and Posterior Chamber.

The lens and suspensory ligament regarded as a diaphragm forming the division between these chambers.

Under normal conditions we know
that this diaphragm does not prevent or offer hindrance to an equalisation of pressure in both chambers. Experiments have shown that the pressure in the anterior chamber is equal to that in the posterior chamber.

In some cases the difference is so slight that it can be disregarded altogether.

The next question is: In what way does a rise in one chamber affect the other?

Priestley, Smith and others, among the most recent, Harding and Parsons (Intra-ocular Circulation) have shown that by the use of a double manometer that any increase within the vitreous chamber causes a corresponding increase in the anterior chamber. The first effect however, is that as the eye is not a perfect sphere and the sclera has elastic fibres, it becomes more and more a perfect sphere.

Any further increase of pressure about 5 mm. causes a change in
the position of the lens and iris, and an excess of 10 millimetres and over, almost entirely abolished the anterior chamber.*

The results of i and ii are reversed when the excess is artificially applied to the Aqueous Chamber; the result however is not so satisfactory in this exp. because of the nature of the vitreous fluid.

Von Hessel and Gruenhagen have recorded, the highest artificial pressure namely 200 millimetres, this was done by compression of the Aorta and simultaneous irritation of the Fifth nerve. *

Wahlfors has recorded y. on. on. in a case of Chronic Glaucoma, after there had been a slight reduction of the pressure by an unsuccessful iridectomy.

The next important consideration is that which pertains to the channels, which regulate by their flow, the amount of intraocular pressure. It chiefly concerns the lymph flow (exit channels). So a brief description of the blood vessels suffices.
to dismiss those which are the entrance channels.
These vessels are divided into three systems:
1. Conjunctival.
2. Retinal.
3. Ciliary or uveal system.
We are chiefly concerned with (iii).
The ciliary system consists mainly of two sets:
(a) Posterior ciliary vessels.
(b) Anterior ciliary vessels.
The Posterior ciliary arteries are branches of the Ophthalmic artery; they consist of two "long," and several "short." The long ciliary arteries perforate Schwalbe's coat, travel between the Choroid and Sclerotic, as far forwards as the ciliary muscle where each divides into branches. Here the branches anastomose and this annulus anastomosis is termed the Circulus Arteriosus Iridis Major. This circle gives branches which run radially in the iris and near its pupillary margin form a second circle—the Circulus Arteriosus Iridis Minor.
The Anterior ciliary arteries come from in front arising from the arteries in the four recti muscles. These perforate the
Sclera near corneal margin, and assist in the formation of the ciliary iris of iris major.
The arrangement of the veins is entirely different. The capillaries of the choroido-ciliary capsule empty into veins which keep uniting to form larger and larger trunks. A number of these trunks converge towards a common centre or vortex. These vortices or vortices of or more in number give rise to the venae vorticosae. Hence almost all the venous blood of the area empties into these venae vorticosae.
A portion of the veins coming from the ciliary muscle, however, take another course as they pass out through the sclera directly, and come into view under the conjunctiva near the margin of the cornea. There are: the anterior ciliary veins, and their course corresponds to that of the anterior ciliary arteries. In some cases of glaucoma, these vessels stand out prominently because of the stasis. These veins anastomose with
the conjunctival veins and also with Schlemm's canal.
This last, which plays such an important part in the Theorie of Glaucoma
is merely a venous sinus running along the corneo-sclerotic junction.

Lymph Channels and Leren's Observations.

Leren experimented with coloured
injections into the tissues of the dead and
also the living eye of dogs, pigs and cats.
In this manner he was able to observe
the course the coloured fluid took.
His results are: In the anterior section of the
eye, the lymph is collected into two large
spaces, the anterior and posterior chambers
which communicate by means of the pupil.
The outflow of lymph is from the
posterior to the anterior chamber, passing
through the pupil, never through the
iris, substance, from there it filters
through the meshwork of the Lig. Post.
into the subjacent canal of Schlemm;
therefore onwards into anterior ciliary veins
which are in direct communication
with the canal of Schlemm. This Leren,
ascertained by injections of solutions of diffusible coloured substances into Ant's chamber. The solution passed readily into Schlemm's canal, and the veins of the iris, causing injection of the episcleral venous plexus, and conjunctival veins; also to a less extent into the venous plexus posteriorly—venae corticae.

In his exp. with non-diffusible substances in solution, this did not cause any injection of the vessels, as colloid substances do not pass through animal membranes.

The force of this argument is important inasmuch as it indicates the absence of any direct channels of exit from the aqueous. The only means of escape for the effete fluids being by a process of filtration or osmosis into the veins of the iris and through the Lig. Post. into Schlemm's canal.

Leber also employed a crucial test by employing a mixture of a diffusible and a non-diffusible substance—Barium
and Prussian Blue, in solution. The results were again confirmed. The
amine readily filtering through the
membranes and causing a pink
injection of the vessels. The Prussian
Blue was left in the Act. Chamber
and microscopic particles was found
embedded in the oozing of the
Lig. Rect.
The Posterior Lymph Passages.
There are 3 in number:

1. The hyaloid canal in the vitreous
equities into the lymph spaces of Optic Nerve
2. Perichoroidal space, between Choroid and
Sclera, continued along vessels forming
the Sclera-Veins of vitreous - which communicate
with ii.

(iii) Tenon's space between Sclera and
Tenon's capsule.

Finally I, ii, & iii; all empty into lymph
spaces along optic nerve, namely the
Interangular and Supra-angular spaces.
But does the effecy matter from the
vitreous chamber take any of these
courses? The knowledge of the course
of the internal channels of the vitreous is still a matter of different opinions. One the less some notion can be had from the interesting expts of Schwabik, Leplat and Giffard. The last mentioned made expts with a solution of cinabar into the vitreous of rabbits. The result showed that some amount of the particles collected at the Papilla. The expt were varied and injections were made further back in the vitreous, two to three days were allowed to lapse. Exami microscopically the particles were found along the hollow of the papilla, and along the lymph spaces around the central vessels, apparently leaving via nerve trunk, though they did not get into the nerve sheath. These results were definite and constant.

None the less, other observers have not entirely coincided with these views, among them Scherer, Meloffs and Priestly. Smith whose expts gave no discoloration
beyond the most superficial layers of the Retina.

The most definite results were obtained with expts. on the eyes of recently killed sheep.

These results have been amply confirmed by Leplat. They tend to show that the amount of fluid escaping at the papilla is very small as compared with that escaping into the ant. chamber and thence via Schlemm's canal. Further that the current is very slow.

So long as there is slight excess of pressure in vitreous, and the hyaloid and Zonula remain permeable, and the fluid be diffusible the tendency is for the excess of pressure in the vitreous to relieve itself by flowing into ant. chamber.
This Theory then from a review of the cases I have examined microscopically, receives full confirmation and is supported almost in its entirety by the total number of cases in presenting that morbid change in the position of the iris which has been made the basis of the Theory.

In two, however, of my cases; this closure of the filtration angle is not present. The number is certainly very small, but it is however sufficient as an objection to the theory; because of this it deserves consideration and some explanation.

It is perfectly clear that the first step must be a mere application of the iris base to the Ligament, Pectinatum and corneal surface—without any regard to the way in which it is brought about— if without any inflammatory addition at first without any true adherence.

Sooner or later adhesion will be formed provided the pressure maintains this application of the iris in the abnormal situation. More readily will the
adhesions form if there be any plastic material or exudate. With the degree and duration of the increased pressure, further changes must occur as one would expect it. The iris will suffer more and more from the pressure, it becomes thin and attenuated, because the longer the pressure is maintained—provided it be not too great—the more atrophy will it cause.

In the cases then of very old Glaucoma, one ought to find a mere thread of tissue with some dark pigment, representing the iris in the region of its adherence. The cases show this admirably.

Sudden and very intense pressure does not cause atrophy. But what is its effect? It produces venous congestion, strangulation of the tissues which may be beyond repair—gangrene—but if the vitality still survives & the conditions favourable, then a process of repair begins, which is really closely allied to the inflammatory process. In the case of the
iris, if the pressure be sudden and intense, why should we not expect it to share the same fate as all other tissues of the body? On the pathological evidence it does; though it never shows the extreme condition of that beyond repair—perhaps its anatomical relations is its only preservative; in that it can always get sufficient nutrition from the aqueous which bathes it.

Does any of the cases show any process in the iris which point to a repair manifestation? Certainly a very great number do. And as one would expect it must occur in the cases of short duration, for in them the pressure has very frequently a history of "suddeness"; and if the symptoms be any indication of the intensity, then, it is always reasonable to suppose, that an attack of Acute Glaucoma is related closely to a very sudden and abnormally high pressure. The treatment purely justifies this view, for by removing the cause—High Pressure— the intensity of the pressure being lessened
we would expect the symptoms to abate. This is really what does occur in the operative treatment.

Further the pathological changes harmonize with this view for in the cases of short duration, in the ciliary region is always to be found some change of a slight inflammatory character, dilatation of vessels—but this may be due to the stasis alone. New formed connective tissue, proliferation of nuclei, etc. With these changes then, an adherence of the iris must be greatly favoured during the repair process.

The degree of firmness of that adherence must be allowed some latitude so that the slips may be regarded as mere application, very slight adhesion, slight adhesion in parts, firm adhesion in parts, through the whole extent of the iris and finally a complete blending together of iris and bowene.

We may in this way offer an explanation for those few cases which do not show the abnormal position, by saying that it is possible that there was only an
application, or a very slight adhesion at the time of excision.
The excision, of itself, would be sufficient to restore the iris to its position or at any rate remove it from immediate contact with the cornea, as during the operation there is loss of a certain amount of blood and fluid, which, however small is yet sufficient to cause some reduction in the intra-ocular pressure.
On experimental grounds we know that the pressure within the eye is distributed equally on every part of the wall. In this way, the excess which maintained the iris base in application, being removed, leads readily to an alteration in the position of the diaphragm and with it the iris base. The elasticity of the structures forming the diaphragm may help to drag the iris base backwards in an excised eye.
On the same change may have occurred, if not at excision, during the process of the preparation of the specimen, for if the adhesion
were of a slight nature, in cutting the section unless special precautions were taken, it is very likely to occur at this stage.

In the absence of evidence of direct application of the iris to the cornea, a careful examination of the parts always shows some disturbance of the relations, in the degree of proximity of the parts concerned. The iris being found very much closer to the cornea than in the normal eye, and what is still more evident is the complete alteration in the shape of the filtration angle.

(a) The inner angle cannot be made out in any of the cases, being flattened in one case - in the other two it seems to be replaced by an elevation.

(b) The outer angle which is usually rounded and more nearly a right angle, in all these cases becomes very obtuse.

(c) The posterior angle which is usually acute becomes very much more obtuse in all these cases.

(d) The outer border of the angle of filtration, that border which lies between (b) and (c) is usually nearly straight but in these cases it is
represented by an almost acute angle. In the nomenclature of these angles, the
terms are adopted from Gerlach.

May not these changes be regarded as a sort of indirect evidence that actual opposition of the parts may have been present? The degree of proximity, and the alteration in contact forming an index of such an indirect inference.

I have no case in which there is no alteration in contact, and the iris far removed from the cornea. A good many cases of this nature have been recorded.

Cases of true Glaucoma with these changes absent, wherein the basis of the Retention Theory lies, seem fatal to the theory at first sight.

But an explanation of these cases is easily afforded and at once convincing. For mechanical obstruction is not the only cause of impaired outflow. The other branch of Outflow, beside Obstruction, is Permeability, which deals with changes in the intra-ocular fluids themselves, whereby they become more colloidal in nature.
Many cases of Serosus Iritis present all the glaucomatosus changes; except this, that there is little or no alteration in the region of the filtration angle. In these cases the chemical analysis shows a considerable increase of the serum albumen. Normally the Aqueous contains only 0.12% of Proteid, made up of Serum Albumen, Serum Globulin and Fibrinogen. It is really only a modified form of lymph and thus filtrates readily, but in serous iritis the great increase in the amount of albumen is the cause of the non-filtration, as this substance does not pass through animal membrane. Perhaps the viscosity of the fluid plays an important part in helping to maintain an abnormal pressure which has been maintained sufficiently long to allow of the transudation of much lymph; for whenever the circulation is obstructed, there is a rise of blood pressure, with this rise of pressure there follows a transudation of serum from the capillaries provided the blood pressure was raised sufficiently long.
In a good many of the cases examined there is much evidence of this transudation, both into the Aqueous and Vitreous, also very fine exudates on the surfaces of the lining membrane of the Vitreous.

A Theory of Glaucoma then, if it were called "Theory of Obstruction" (in its widest application) would seem to meet all the requirements to explain the Glaucomata process. For such a Theory being based on experimental Physiological evidence, and confirmed by mostly all the Pathological examinations could not meet with any serious objections.

The Theory of Retention is more restricted however, inasmuch as it asserts the region of obstruction—the filtration angle—supplying only a mechanical obstruction.

Most observers are so satisfied with the stability of this Theory, and of its general application, that they have taken it as the basis, for more extensive research. These investigators accept:

Glaucoma is the result of high tension.
High tension is the result of Obstruction.
Obstruction is the result of (?).
This difficulty is still unsolved. The point at issue is—what are the factors which cause the iris to assume this abnormal position?

As Weber was the prime author of the Retention Theory, I shall consider his views first. He holds that it is the result simply of swelling of the ciliary processes, the swollen processes press upon and push the papillary of the iris forwards, and thus gives the starting point for glaucoma.

Any condition which tends to increase the amount of blood within the vessels, especially the venous trunks of the head and neck, must at the same time cause an overfullness in the vessels vessels, consequently turgescence of the ciliary folds.

Such a condition of itself seems incapable of explaining all the facts in the pathology of the disease, more especially age incidence.

In many cases this turgid condition of the ciliary folds holds good to explain those cases in which glaucoma comes on suddenly after severe muscular exercise,
in such maladies as are cardiac, vascular, and pulmonary, and which induce venous congestion. But these diseases in their most severe forms are rarely associated with glaucoma.

There must be some additional and more potent factor than mere swelling of the processes.

Brailsby, to a certain extent, adopts this view of Weber, but he attributes it to an inflammatory hypersecretion in the first instance. He holds that there is always inflammation of ciliary body, iris and ophic nerve, as one of the earliest condition in glaucoma; being developed, even before the increased tension.

The iris periphery is nearly always applied to the cornea, and this advanced position is caused, in the first instance, by an enlargement of the ciliary folds due to their vascular turgescence; afterwards its application is rendered more close and more extensive by the pressure of fluid behind it.

This pressure he attributes to an inflam-
matory hypersecretion of fluid from ciliary body and iris.
Most of my cases point greatly in this direction — namely that of a chronic inflam.

The cases of short duration all indicate that in the region of the ciliary body there has been a degree of irritation and inflammation; always more or less moderate increase of nuclei, vascular engorgement, proliferation of cells within and around the vessels, increase of the connective tissue stroma. In the more advanced cases this connective tissue is greatly increased, the ciliary muscle in a markedly atrophic state.

The iris in most of my specimens do not show these changes in such an advanced stage; but in the moderate stages it is a constant feature.

That there is this slow inflammatory condition seems an irresistible conclusion in all the cases examined; but that it exists before the high tension is purely a matter of speculation and most difficult to prove.
Mr. Knies holds the same view, regarding glaucoma, in its essence, an irido-cyclitis. The different clinical varieties owe their nature to the varying intensity of the irido-cyclitis.

Priestly-Smith adopts the Retention Theory, but maintains an entirely different view as to the initial cause of the abnormal position of the Iris.

Not without considerable labour, he has in a series of articles, detailed his observations, which go to show that the initial cause must be sought in such a direction, as to readily harmonize with the known aetiological facts of the disease.

He holds that the main predisposing condition is an insufficiency of the space between the lens margin and the structure surrounding it (circumcylindrical space).

How can a narrowing of the circumcylindrical space cause an application of the Iris base to the cornea & ciliary feet? It does so as follows:—

An increased amount of blood in theveal tract must be compensated by the expulsion of some other fluid from
from the chamber of the eye: the aqueous humor then filters more rapidly than normal at the angle of the ant. chamber. With this diminution of the contents here the iris and lens move slightly forwards towards the cornea. In the normal eye this compensation takes place without any morbid complication; because in these the lens is small, the circumcircular space sufficiently large, and the ant. chamber of sufficient depth.

When however the lens and ciliary processes are already in close relation to each other and the ant. chamber shallow, then any increased fulness of the uveal tract menaces the angle of the chamber. The tension in the ciliary processes have not, under these conditions, sufficient space to expand; they are thus carried forwards together with the lens and thus by pressing upon the base of the iris lead to a closure of the angle of the ant. chamber. Further escape of fluid is now almost impossible.
and the pressure thereafter increases more than before.
This explanation suffices for acute cases.
How then can one possibly get a
similar chronic condition? Since the initial
pressure continues increasing.
Priest-Smith thinks that in these cases
the vascular dilatation being gradual
and slight, the vessels have time to
adapt themselves to the slowly in-
creasing pressure; the angle of the
Aqu. Chamber is more or less compressed
but not tightly closed.
Does the narrowing of the circumocular space
actually occur? This the author has con-
clusively proved as a natural change
with the advance of age—so far, at
any rate, as the narrowing can be
produced by the increase in size of
the lens.
The statistics of his measurements show
that the lens increases in size with
every successive period of life—During the
45-years between the ages of 20 and 65—
it adds about 1/3 to its volume, 1/3 to
its weight and one tenth to its diameter.

The specific gravity shows no change with the advance of life. It further proves that small eyeballs are especially liable to primary glaucoma, a fact which is often demonstrated by the small size of the cornea in the eyes affected.

The all-important question of liability of the disease in proportion as age advances has been most carefully worked up by Kiiick and his conclusions from 100 Primary cases furnish strong evidence in support of his theory.

In many cases I examined, actual measurements were taken of the lens, more especially in those where some disproportion could be made out with the naked eyes. The increase in diameters (working on Priest's tables for age, periods, and size of normal lens) 12 in 20 out of 37 cases measured. Of these cases that did not come up to the normal measurement for the age period, but showed no increase in diameters, 7 of these how.
ever had this in common that the disproportion of size of lens to globe was very obvious, thus rendering the ciliary space a little below the normal. This view of Priestly Smith, however, is not generally accepted, in spite of the volume of evidence he supports it with.

This view of Dr. Smith is admirably supplemented by Snellen, who attributes the forward displacement of the lens to an abnormal slackness of thezonula. He holds that in advanced life when the elasticity of the lens is lost, thezonula must be renderedslack when the ciliary muscle contracts strongly. The lens then moves forwards carrying theiris forwards to the cornea. In the youthful eye this does not occur, because during accommodation the diameter of the lens diminishes with that of thecontracting ciliary muscle, and thezonula remains tense.

This same abnormal slackness can arise, according to him, from degenerative
changes. Thus an explanation is offered, for Glaucomatous attacks in certain cases of senile cataract.

Max Kries maintained that the originating focus was to be found in a circumscribed inflammation of the Ligamentum Postcruatum and the adjacent tissues. This I omitted to mention when discussing his view "Iride-eyelids"—the arguments and evidence of pathological changes which support Bailey's view apply equally to Max Kries' view.

Dr. Brubacher and Egermatt at one time also maintained this view with Max Kries, though they went still further and asserted that the closure of the filtration angle by pressure from the ciliary processes can be put entirely out of consideration. This, however, seems too rash a conclusion, for on physiological ground it is possible; experiments prove its occurrence; why should it not occur if the pathological condition
were to favour it. Further, the eyes examined
by them were opened without freezing, and
in no case without hardening: this, of

course, is sufficient to justify any worse
statement.

Among theories of obstruction that are
left, the most important is that of
Lagerny* who believes that the change

primarily involves the sclerotic.

This must become the seat of an
inflammatory change of a gradual
nature, which renders it less elastic

and thus altogether rigid. Such a
change produces an obstruction of

the posterior channels of exit of the
intra-ocular lymphatics, namely those

which pass around and along the

4 venae vorticose.

There is no doubt that if such a change
were invariably found in the sclerotic, the
theory would receive more attention.

But the pathological evidence is only
supported in 5 of any cases, and that

only so far as it concerns the thickening

and slight rigidity, but no actual
proof of the blockage of the lymphatics. Further these posterior channels take very little share in the removal of the effete matter as noted before under lymph channels; even if they were blocked, I think tend to show that it could easily be compensated by the anterior channels of exit.

The anatomical relation of the vessels is one great point in favour of very passagable great liability to compression. One fatal objection to this theory is—If such were really the cause of glaucoma, what effect would iridectomy have or excise? Iridectomy may conceivably improve it by suddenly reducing the pressure and allowing the channels to open up under the diminished pressure. But could it be a permanent cure?

Other theories have from time to time been brought forwards, important as they have been at some date; they are at present only of historical interest. They however differ greatly from all those I have before considered in that they deal with "Inflow".
That there are two possible ways in which the
inflow can cause an increased tension has
been pointed, namely, vascular disturbances
and hypersecretion.

On very close study however they are not
essentially different, for hypersecretion is
directly the result of local vascular dilatation
as the latter causes transudation of serum.
But this is not the sense in which
hypersecretion is here employed in this
context, as it does not imply a mere
transudation, but an active glandular
secretion. And yet another sort of relation
exists as such an active glandular secretion
can not be increased without an increase
of the vascular supply.

What evidence is there that local vascular
dilatation can cause an increased pressure?
Several exp. cited before show this. But
can such local vas. dilatation produce increased
pressure within pathological limits so as to
establish the glaucomatomic state?

Whether or not the mere local dilatation
is the cause, yet there are certain cases
in which the glaucomatous attack seems very
closely related to such a condition. We may expect it as the consequence of some irritation of the fifth nerve. More or less pain is usually present in this nerve during such attacks. Occasionally the Neuralgia is distinctly antecedent to the outbreak of an attack of Glaucoma. Such a case has been recorded by Hutchinson.*

Many cases have had their starting point in some injury which presumably caused a cerebral or a nervous lesion. Many premonitory symptoms are often only congestion of some part, and still more striking proof is that many an attack can be ward off by rest in bed, purgatives, or other palliative measures. Any disturbance which flushes the face, or causes congestion of the head is apt to cause a recurrence.

In these instances one can perceive the cause of attack - if not wholly, at any rate in part, to nervous and vascular disturbance; remembering that it cannot be a very material part, since then
are hundreds of other cases with the same degree of congestion of the head, with injected ciliary vessels, who never get Glaucoma.

Moreover there is sometimes no sign of any nerve irritation or active hyperaemia throughout the disease.

The pain and injection of acute Gl.

seem more consequences than causes of the High Pressure within the Globe, since they outside where the excess of fluid is allowed to escape.

Among those who supported the above theory is Von Graefe* himself, for he believed that a pernous choroiditis lay at the root of the Disease. This condition, he thought, gave rise to an exudation of serous fluid into the vitreous humor.

There is not the slightest doubt that a choroiditis can originate an attack of Glaucoma, this it does by raising the pressure in the vitreous chamber, thus driving the lens and ciliary processes forwards and eventually closing the filtration angle. The vitreous
The chamber can only be enlarged at the expense of aqueous chambers as we have seen. Changes, in the hyaloid membrane, in the stroma of the vitreous, or in the constitution of the vitreous fluid, are possible causes of high tension incurred as these changes cause a diminished filtration.

In a good many specimens the vitreous is found more distinctly membranous, its septa being thickened; in many cases the septa and hyaloid are coated by some albuminous exudate or coagula in some cases the fluid collects not in the vitreous itself but between the vitreous sub. and its hyaloid sheath causing a separation of the latter.

These opacities are very often detected with the ophthalmoscope.

As the theory is such an old one it is no disrespect to the author to classify such cases as Secondary Glaucoma, if the case allow of a proper diagnosis. Often such a diagnosis is hard to make or even impossible, and
to the last it may be considered one of Primary Glaucoma.

Donalds and others hold that irritation of the fifth pair of nerves, which govern the secretion of the intra-ocular fluids gave rise to hypersecretion of these fluids. What has been said with regard to local vascular dilatation applies to this hypersecretory theory.

It is certainly possible that hypersecretion of the intra-ocular fluids may occur through some nerve irritation or vascular change, but this can be only a hypothesis and not a hypothesis.

For clearly any rise of pressure due simply to hypersecretion must tend to correct itself by a gradual widening of the channels of exit. This is the objection that Weber raises.

Are there any pathological evidences to contradict this hypothesis? In order to bring forward these path evidences it is first necessary to ascertain the source of the secretion. So it is not
surprising that the source is found to lie in the ciliary processes; as all the other structures have well defined functions to perform.

Fraser-Collins' observations amply confirm by Nicoli establish the fact that there are glands in the ciliary processes which are concerned in the elaboration of the aqueous humour and nutrient fluid of the vitreous. Whether these glands are the sole providers is still unsettled for these glands were absent in some of the cases examined by Fraser-Collins, he confesses that he is at a loss to explain their absence in some eyes and presence in others.

Even admitting that these are glands, does not the pathological condition itself indicate that hypersecretion under such abnormal pressure become a physical impossibility?

Having in this manner treated the several factors which build up my genealogical tree—with that great branch of "Contents" this last theory ends—
The only other factor left in that tree is the "knot."

As one should expect, so we find a theory was built having this factor as its basis. The theory asserted that changes in the sclerotic, rendering it rigid and leading to some shrinking of it, caused the increased intraocular tension.

From all that I have said before, more especially when discussing Lague's theory, and from the knowledge we have of the normal intraocular tension, and its physiological variations, this theory becomes so obviously untenable that it needs no further comment.

With the end of this theory comes the end of my Thesis.

Under the various theories I have taken the opportunity of discussing the degree of confirmation each theory receives from the pathological evidence of my specimens. I hoped by this means to save the reader his patience, and myself the task, which a summary of these changes in this
final page would cause. I should
now be compelled, had I not adopted
this plan, to recapitulate those
pathological changes and the several
theories.
In drawing attention to the changes under
each theory, I fear I have been
in some cases too brief and dealt-
too lightly with most important changes.
I may, at the same time, omitted
some special feature in the morbid
change, passing over it, as it were
unnoticed. Such errors of brevity and
omission may, perhaps, be in another
way useful, for in some parts I
fear that I have been most tiring
with irrelevant details and most
elementary facts; not that I wished
them to be so in the least; if I
have made myself too tiring in this
way, my only excuse is that I erred
in a good cause, for in trying to
make myself understood, I overstepped
the limit and became too elementary.
If I have treated the subject
in too lengthy a manner, the cause lies not so much with me as with the subject itself. Far is there another subject in the whole of medicine or surgery which can compare with Glaucoma in extent! Does not a discussion of Glaucoma mean the discussion of the Anatomy, Physiology and Pathology of the whole Eye? My difficulty, in so extensive a study, has been to thoroughly eliminate what is not pertaining to this Thesis. The literature on its Pathology alone would form a few separate volumes, yet I have tried to embody it all in these few pages. Were I an authority, I could only claim consideration here; but being a novice I expect condemnation, though I shall hope for sympathy in this effort.

But, does not my own confession now seem to indicate an empty conceit, in this feeble attempt of so complex a subject?

Perhaps it is. But were I to ask myself, what has really prompted me to choose so complex a
difficult a subject? My only answer could be, that I do so; because I feel convinced, that the futile efforts we employ, in trying to understand the most complex subjects, are not really futile. These are the means by which we unconsciously learn to solve subjects of a more simple character.