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THESIS FOR M.D. DEGREE.

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ON THE IMPORTANCE OF
ARTERIAL CHANGES IN
THE CAUSATION OF
GLAUCOMA.

No apology is offered for selecting Glaucoma as the subject of this Thesis for the degree of M.D. This dreadful disease is in many ways the most mysterious in the whole domain of Ophthalmology, and the consideration of any side-light on its possible pathology, especially if this should have some influence on its treatment, must be a source of deep interest to such of us as have elected to devote our energies to this
particular branch of the healing art. From the title of the Thesis it will be seen that it is not asserted that Glaucoma is always due to vascular disease, but it is considered that proper attention has not in the past been given to vessel changes, and that ophthalmologists have concentrated their energies perhaps too much in the direction of local conditions. This is true of nearly all observers since the real discovery of the disease by V. Graafe, in 1854, down to the present day. Such a narrowing down of one's point of view is perhaps the besetting sin of all specialists, and although it is not the purpose of this Thesis to detract from, or in any way to minimise such work as that of Priestley Smith on the growth of the lens, or the more recent theory of sclerosis of the pectinate ligament enunciated
by Thomson Henderson, it is submitted that ophthalmologists should not forget that they were once general physicians and surgeons, and ought never for that reason to omit a close inquiry into the general condition of their patients. In no eye condition is this more important than Glaucoma, for here we have usually a bilateral condition, in which the disease in the second eye in no way depends upon that in its fellow, and it will readily be admitted that under such circumstances, departures from the general health may possibly have an important bearing on its causation. In point of fact, this Thesis is written with the object of showing that in the majority of cases of Glaucoma, general disturbances in the vascular system do exist, and that their recognition may modify the treatment adopted. The scanty mention in most of our Text
Books of anything that is not local in the causation of Glaucoma, not less than the various new methods of local treatment (such as Herbert's Wedge operation, Lagrange's sclerectomy, Heine's Cyclodialysis) only confirm the contention that the vast importance of vessel changes is not as much appreciated as it might be. Local treatment, of course, must be employed, and the old iridectomy is still the sheet anchor with most practitioners; but what I do insist upon is, that the use of such means as we have for the control and, if possible, the prevention of arterial Hypertension must favourably modify the ultimate results which in some cases in spite of perfect technique, are not everything one would desire.

In the Regulations for M.D. Thesis, it is stated that no Thesis will be approved which does
not contain the results of original observations, or else a full digest and criticism of the researches of others on the subject". The idea which is expressed in the title was quite original, being based on reflection upon three cases occurring in my practice during 1908. These cases will presently be cited. In making this statement I am aware that I lay myself open to very just criticism, for although in receipt of several monthly Ophthalmic Journals, I regret that only those papers which are of present interest are perused, the volumes being then put away for reference. It came somewhat as a shock to find, on looking up the literature, that so much had been written which bears directly or indirectly on the subject. I will, however, attempt to do full justice to the latter part of the Regulation and to
discuss, as fully as is necessary, my views on the conclusions of other authors.

CASE 1.

F.J.A. Iron Founder. First seen Nov. 11th, 1904, then aged 64, consulted me in reference to his glasses. I found the following vision.

R.V. 6/9 with + 2.50 sph + 1 cyl. ax. 20
L.V. 6/6 with + 2.50 sph + 1 cyl. ax. 160

Right eye has always been weaker than left. Right disc noted paler than left. ? Slight cupping. He was told to return in three months but I did not see him again until July 9th, 1906. R.V. was then only 6/24 with glasses, disc deeply cupped, Right Field contracted to within ten degrees of fixation point on nasal side. Right
iridectomy performed July 16th, 1906. Operation unfortunately took away fixation point and his vision became reduced to Fingers at 2 metres, occasionally 6/60 when looking sideways. This vision he has retained. When I saw the patient on July 9th, 1906, his left vision was full with glasses and his field was quite full; in fact, there was nothing to indicate Glaucoma in the left eye. The patient was seen at short intervals but until April, 1908, I couldn't discover the least suspicion of Glaucoma in the left eye. Just before that date, however, one of the patient's business assistants embezzled a large sum of money, and this caused great worry and anxiety to F.J.A. He began to feel generally unwell and on April 9th, 1908, I noted his left vision as 6/9 with glasses, very slight cupping of disc, field slightly regularly contracted on nasal
side. He merely complained of fogginess of vision. As the iridectomy in the right eye, though perfect technically had not been very successful in its result I advised him to see Mr. Priestley Smith who recommended iridectomy but gave a very guarded prognosis. He noted a marked rise of blood pressure in a sphygmographic tracing which he took at the time. For this reason he was advised to leave off all work, and take small doses of Bromide for a month. At the end of that time, May 21st, I operated, and he recovered well. His vision on June 29th, 1908, was 6/8 with +1 sph. combined with +4 cyl. ax. 180. This vision still remains unchanged and there has been no further limitation of the field, but the cupping seems more marked. The blood pressure has
been examined and was found to be 160 m.m. (Riva Rocci). The patient has been carefully in-
structed as to diet etc. in the hope that as the rise of arterial pressure was evidently only temporary his present vision may be preserved.

CASE 2.

T.C.K. aet. 44. Publican. Left eye in-
jured 31 years ago in a railway accident, and was excised twelve years ago by Mr. Hartridge on account of sympathetic changes in the right eye. At any rate, vitreous opacities have been present in this eye ever since. For nearly seven years there have been recurrent attacks of dimness with rainbow vision in the right eye, and these attacks have latterly been more frequent. His history was that four days
previously the sight became misty, and he saw haloes of colours around lights, the symptoms passing off at intervals only to return again in a few hours. At the same time his face became flushed and he felt "thick in the head".

On examination I found his cornea covered with small vesicles, his anterior chamber rather shallow, episcleral veins widely dilated, tension plus, vision only hand movements. I concluded he was suffering from a mild attack of acute Glaucoma and ordered pilocarpin drops. April 27th, corneal vesicles disappearing, tension still full, but eye generally much quieter. R.V. 12. April 29th, no vesicles, eye quiet, disc difficult to see but apparently not cupped, vitreous full of dense membranous opacities, field full. He had another
attack Oct. 29th, 1908, which passed off in the same way. Patient is very averse to operation and I have not pressed this very strongly as the eye is not one of the best to operate on, and, moreover, he seems up to now to recover so completely from the attacks. The important point in this case is to be found in a statement by the patient that he has always been able to cure these attacks by taking a strong purgative, sufficient to insure a free watery evacuation of the bowels. The blood pressure was estimated and although in the intervals between the attacks it was only 140 m.m., it rose during the attack to 160 m.m.

CASE 3.

W.M. aet. 42. Publican. First seen in the
afternoon of Oct. 28th, 1908. He complained that the right eye had been misty ten days, the mistiness passing off in the afternoon but returning again next morning. R.V. $\frac{6}{6}$ ? slight cupping of disc, field full. Has smoked 50 cigarettes a day for many years. Alcohol immoderate. The left eye had no type vision, having been injured 30 years ago. Fundus showed old retinal atrophy in the region of the macula and some secondary optic atrophy. ? result of old haemorrhage. I wrote to his doctor and asked him to watch the patient. Tobacco and alcohol were discontinued entirely and he was placed on a mixture of potassium iodide and nux vomica. He was next seen Nov. 18th, 1909, in the morning and I then noted marked vesicular affection of both corneas,
deep ciliary injection, tension full, anterior chambers not markedly shallow. He was at once admitted into hospital and watched. It was found that both eyes presented the appearance of acute Glaucoma in the mornings but cleared up well towards evening. He was kept on eserine three or four times a day. As his vision was gradually deteriorating, even when the eye cleared up I advised him to have an iridectomy in each eye. This was done Nov. 26th, (left eye) and Dec. 3rd, (right eye). Both eyes soon quieted down and before his discharge from hospital just before Christmas his vision in the right eye was $\frac{6}{6}$ with glasses, field full. Blood pressure estimated later was 150 m.m.

The notes of these three cases are somewhat
incomplete, inasmuch as I have not considered it necessary to send copies of the fields of vision, as I do not think this is relevant to the question at issue. What struck me was, that though the causes are apparently very different, they can be explained entirely by increase of blood pressure, though this may only have been temporary. Thus, in Case 1, acute worry and anxiety over business matters at once seemed to bring on the symptoms which had been carefully looked for in vain for two years.

A physician friend of mine who is at present attending a case of arteriosclerosis, finds that a little worry sends up the manometer readings very quickly and, of course, business worry if long continued is a well recognised cause of this
disease.

In Case 2 the sudden disappearance of all symptoms which follows a free watery evacuation of the bowels suggests that the effect of the purgative is to lower general blood pressure, and that this should be followed by a diminution of intraocular pressure is what one might expect.

In Case 3 the peculiar symptoms, which appear to me distinctly glaucomatous, might reasonably be said to be the result of over indulgence in tobacco and alcohol. Russell (1) in his recent work on Hypertonus singles out tobacco as a very common cause of hypertonic contraction and alcohol probably has a similar effect, though this is denied by Oliver (2) who says that whiskey and gin are vasodilator. I presume, however, that this
does not apply to excessive use.

The use of the sphygmomanometer by physicians in cases of increased blood pressure suggested to me that an examination of cases of Glaucoma by this instrument might be fruitful of results. I have, therefore, applied this test to several cases, the details of which will be found at the end of the Thesis. The pressure has generally been taken at about 11-30 a.m. when the normal digestive hypertonus would be at a minimum and also after the patient has rested an hour or two. Where possible, more than one examination has been made and the mean pressure stated. Unfortunately my department at Northampton is chiefly an out-patient one, composed largely of refractions and cases of external disease and I
have therefore not had the opportunity in the time at my disposal to examine a large number of cases. There are, however, several papers on the subject in the literature and my numbers must necessarily be supplemented by the work of others in the same direction.

ABSTRACTS OF LITERATURE WITH CRITICISMS:

By far the most important contribution to this aspect of the pathogenesis of Glaucoma has been supplied by Troncoso (3). In several papers he has endeavoured to prove that it is a vessel wall disease, basing his assertions on numerous experiments on the filtration of intraocular fluids. He considers that the conditions are analogous to those present in chronic interstitial...
nephritis, and that the increased quantity of albumin in the aqueous in Glaucoma is due to the changes in the intraocular blood vessels allowing an abnormal transudation of this substance into the eye. It is generally admitted that the pathological changes in the vessels in Glaucoma are similar to those seen in senile arteriosclerosis and I contend that the latter is present in almost all cases at least of chronic Glaucoma. Troncoso affirms that the difficulty with which albuminous fluid passes through the canal of Schlemm is the primary cause of Glaucoma. He asserts that the vitreous becomes progressively increased in volume owing to excessive transudation and oedema, and that this presses forward the iris, and so makes the escape of the albumin-burdened aqueous
through Schlemm's canal still more difficult.

After this has been going on for some time, a little vasomotor dilatation such as might be produced by emotion or shock, is sufficient to produce the acute attack. Troncoso finds that all the tissues bathed by the albuminous aqueous undergo fibrous sclerosis, and in this way I suppose he would explain the sclerosis of the pectinate ligament which plays so important a part in Thomson Henderson's theory.

Experimnetally Troncoso finds that injection of albumin into the aqueous in high enough proportion produces plus tension. His experiments with Leber's apparatus prove that albuminous fluids filter through the eye badly as compared with saline solutions, and if the experiment
is continued long enough filtration ceases altogether on account of the accumulation of albumin in the anterior chamber. If the vitreous pressure is raised, the filtration through the anterior chamber is diminished and ceases altogether when the pressure in the vitreous is 3 to 5 m.m. above that of anterior chamber, but is restored again by taking away the vitreous pressure. Leber (4) objected to Troncoso's view that increased amount of albumin in the aqueous causes Glaucoma, and states that he has found more albumin in eyes associated with minus tension; moreover, that the eye is a single elastic capsule filled with fluid, and that, therefore, there can be no difference between the pressures in the aqueous and vitreous.
21.

These objections were refuted quite successfully by Troncoso. In the first place, he said that the above mentioned minus tension diseases had a period of plus tension, but then the exudation organises and less aqueous is produced on account of the condition of the ciliary body - pressure then sinks because of diminution of eye contents.

In the second place, Troncoso believes that the eye is divided into two elastic compartments by the iridocrystalline diaphragm, and that therefore there may be differences between the pressures in the two compartments. Leber admitted the latter contention but gave a different explanation viz. that it was a question of elasticity of tunics, which he admits may be diminished in old age and pathological conditions.
Personally, I consider Troncoso's work very valuable in that it affords strong experimental evidence of the way in which arteriosclerosis might act in the production of Glaucoma. That this condition of vessels is present in nearly all cases at any rate of chronic Glaucoma is a clinical fact, and I don't think anyone would question that temporary rises of blood pressure such as might be caused by worry, emotion, fright, etc., are very frequently if not always present in the history of our cases of acute Glaucoma.

Apropos of Troncoso's theory, Knapp (5) has recently published the result of an examination of an eye affected with acute Primary Glaucoma. The posterior chamber was filled with a semiopaque gelatinous fluid which took on the Eosine stain.
There was a coagulated mass in the anterior part of the vitreous, which appeared to be an exudate, with no cellular elements, coming from the ciliary vessels. The increased content of the vitreous chamber pushed forward the iris which was adherent to the cornea. The aqueous was highly albuminous so as to make osmosis difficult. Schlemm's canal was noted patent. This case seems to me to confirm Troncoso's theory.

Glaucoma occurred in the other eye two years later, and a year afterwards the patient died of apoplexy - I presume from this that the patient suffered from arteriosclerosis.

Brown Pusey (6) also inclines to the belief that intracocular tension is a question of osmosis and that the cause of Glaucoma will probably be found in the osmotic condition of the aqueous and
vitreous. He asserts like most other observers that closure of the filtration angle is a result and not a cause of the disease, and he thinks that while swelling of the lens and ciliary body are important they could not cause Glaucoma.

There has been no difficulty in finding in ophthalmic literature a large number of papers in which incidentally or otherwise the close connection between Glaucoma and increased arterial pressure is demonstrated.

Frenkel (7) examined 15 cases of Glaucoma and found increased blood pressure (by means of the Riva Rocci instrument) in no less than 14. He concludes that Glaucoma is due to increased arterial tension or to toxic agents. In the
light of recent work by physicians there is no doubt that the toxic agents produce the increased arterial tension so that the two causes given by Frenkel are associated.

Joseph (8) found arteriosclerosis in 18 cases of Glaucoma which he examined and he considers high arterial tension the most important factor in the production of the disease.

Horstmann (9) publishes a paper on Glaucoma Simplex in which he relates 13 cases in almost all of which arteriosclerosis was present and suggests that perhaps this disease plays a part in the etiology.

Dunn (10) more recently in a paper on the value of the sphygmomanometer in ophthalmic practice relates 5 cases of Glaucoma in which the
blood pressure was definitely increased. He insists in the use of the instrument in all cases of Glaucoma as an important aid in prognosis and declares that when the pressure is high the results are poor after iridectomy in spite of the most perfect technique. In one of his cases though the patient was 70 years old the pressure was only 140 m.m. and operation checked the disease for 2 years. I must say that my experience leads me to concur entirely with Dunn, for often in the past I have done iridectomies which have healed well but have been unable to stop the progress of the disease. One such case came under my care recently (Mrs. B). In this case the operation was entirely unsuccessful and when the blood pressure was examined later it was
Terson (11) records a case in which haemorrhagic Glaucoma supervened in a case of renal retinitis. It will not be denied that the blood pressure is always increased in chronic renal disease and this case therefore confirms the main argument of this Thesis. I agree with Dunn (10) that operation should be avoided when albuminuria is present and in this connection it is useful to note that in Terson's case eserin failed to relieve the tension, but $2\frac{1}{2}$% of dionin was successful in reducing it. In another paper Terson maintains that acute Glaucoma is an acute idiopathic oedema as seen in the lungs and that some toxaemia is generally present, such as, erysipelas, influenza, gout, etc. The physicians
tell us (Russell) that arteriosclerosis is largely due to toxic substances in the blood, therefore it seems natural to me that when this toxaemia is present, Glaucoma may occur at any time as a complication.

Beltman (12) records a case of chronic Glaucoma associated with telangiectasis of face and external blood vessels of eye, in a young woman. The Glaucoma was more pronounced just before menstrual period, when blood pressure is highest. He assumes that the telangiectasis extended to the choroid and caused increased exudation of lymph, and absorption of fluids would be hindered by increased pressure in the veins of the sclera and the plexus in the angle of the anterior chamber. This case speaks for
itself, the Glaucoma present being more pronounced when the blood pressure was at its highest point.

Bartels (13) finds that there is more or less arteriosclerosis in all cases of Glaucoma. Experimentally, he finds that by obstructing the venous return from the anterior part of the eye he can produce Glaucoma and he concludes that this obstruction is present in man.

Green (14) records a case of Glaucoma Simplex in a woman aged 30 which began about puberty. Myotics did no good but the treatment of gastric and intestinal myasthenia improved the vision. To me it seems clear that in this case there was toxic absorption from the intestinal tract causing hypertonus and that the relief of the latter by the proper treatment of the former
naturally had a beneficial effect on the Glaucoma.

Coats (15) discusses the relation between Glaucoma and vascular diseases of the eye such as thrombosis of the retinal vein and embolism of the artery. He holds that these diseases are not both due to the angiosclerosis, first, because the thrombotic eye alone becomes glaucomatous, and secondly, because the conditions are sometimes present in children with no angiosclerosis. Coats therefore thinks that the Glaucoma in these cases is due to the haemorrhagic retinitis, which alters the lymph circulation in the vitreous, in such a way that this becomes more colloidal and less easily drained. Whether this conclusion is correct or not, the admission is distinctly made that changes in the intraocular fluids may cause
Glaucoma, and as these fluids have been proved by Troncoso to contain more than a normal amount of albumin in Glaucoma, it would appear that here again we have a confirmation of Troncoso's theory.

Apropos of Coats' paper I published brief notes of four cases of the kind in the British Medical Journal of Jan. 18th, 1902. The radial arteries were thickened in all four cases, and when I say that Glaucoma supervened not only in the thrombotic eye, but also in its fellow in two of the cases it will be seen that I cannot admit Coats first objection why these cases are not due to arteriosclerosis. In fact, I firmly believe that both thrombosis and Glaucoma were merely the results of the vessel conditions.
Harm's (16) publishes a record of 90 cases of thrombosis etc. in 42 of which Glaucoma supervened.

Sala (17) publishes a case of bilateral Glaucoma after needling for congenital cataract and 2 cases after contusion of eyeball. In all his cases there was a great excess of albumin in the aqueous and he considers that this caused the Glaucoma. Here again we have Troncoso's views confirmed.

Peters (18) records 4 cases of Glaucoma after contusion and comes to the same conclusion as Sala as to the cause.

Zimmermann (19) thinks the primary cause of Glaucoma is a difference between general blood tension and that of the eye. Contrary to most
other writers he states that there is a lowering of blood pressure because of general disease affecting the heart, and that venous stasis in the eye is a result, causing increased transudation into the vitreous. In this way he accounts for the occurrence of Glaucoma after debilitating diseases, and after disturbed circulation in the eye, such as thrombosis. He admits that Glaucoma is generally associated with arteriosclerosis but recommends such remedies as strophanthus and adonis vernalis. In my experience I have only seen one case in which the blood pressure was lowered (vide notes) and in that case the Glaucoma was subacute and the tissues of the eye were in an atrophic state.

Starling and Henderson (20) as the result of
Experimental investigation found that:

I. Intracocular pressure equals the pressure at which the rate of formation of intraocular fluid is exactly balanced by its absorption.

II. The production of intraocular fluid is exactly proportional to the difference in pressure in the capillaries of the eyeball and the intraocular fluid.

III. Intracocular fluid is the result of transudation pure and simple.

IV. Increased proteid content of intraocular fluid slows its rate of absorption.

V. Filtration at high pressure is favoured by contracted pupil and hindered by dilated pupil.
It is interesting to note that though these observations were made presumably on normal eyes the fourth point bears out Troncoso's contention exactly.

Thomson Henderson (21). A thesis on the pathogenesis of Glaucoma written at this period would be incomplete without some reference to the recent theory of T. Henderson. He considers that the primary cause of Glaucoma is sclerosis and closure of the pectinate ligament. This change progresses with age, and when complete, the filtration of the aqueous takes place through the crypts of the iris. If from any cause these crypts are not available, as when the pupil is dilated either by drugs or by some nervous condition, acute Glaucoma may be the result, the iris crypts in fact
forming a regulating mechanism. Normally the intracocular pressure is equal to that of the venous capillaries in the eye and the venous sinus of Schlemm's canal, the pressure in the latter being just less than that in the iris veins with which the crypts are connected, so that the exit through Schlemm's canal is in the direction of least resistance. When the intracocular tension rises above that in Schlemm's canal, the hydrostatic pressure of the aqueous by pressing the sinus wall and reducing its lumen raises the venous pressure in the sinus. As soon as the sinus pressure equals that in the iris veins the aqueous will drain through either the iris veins or the cribriform ligament and Schlemm's canal. The resulting increased outflow through the iris veins
reduces the pressure in the aqueous and so the pressure on the sinus is removed and the pressure in the sinus falls to normal. In normal eyes any increase in intracocular pressure is thus soon compensated by increased outflow through the iris veins, but this is not so in Glaucoma, on account of the diminished range of potential outlet. In an eye predisposed to Glaucoma by sclerosis of the pectinate ligament, a congestive attack is produced.

I. By dilatation of pupil, closing the iris crypts.

II. By rise of arterial pressure, which always causes increased intracocular tension, but which in Glaucoma cannot be compensated on account of obstruction to outflow. The veins
are compressed and the congestion which follows increases the tension.

III. By rise in venous pressure, the venous congestion of the uvea upsetting the balance of aqueous outflow.

My comment on this theory is that, granting all these conditions to be present in Glaucoma, the overloading of the aqueous with albumin which has been proved by Troncoso must be an important added factor in the production of the disease, for it will be admitted for instance that the passage of albuminous fluid through the iris crypts must be much more difficult than in the case of normal aqueous.

An interesting sidelight on the influence of changes of blood pressure in the production of
Glaucoma is furnished by Steindorff (22) who finds that the disease is much more prevalent in the cold winter weather than in summer. This is the experience of most ophthalmic surgeons. The physicians tell us that if we wish to control supernormal blood pressure it is advisable to send our patient away to warm climates during the winter. The bearing of this on Glaucoma is evident.

Fuchs (23) in his Textbook tells us that genuine Glaucoma appears in an eye which is predisposed to it by

I. Smallness of eye.
II. Shallow anterior chamber.
III. Large ciliary processes.
IV. Disproportionate size of lens, as in all hypermetropic eyes of the aged.
He gives as exciting causes disturbed circulation and dilatation of the pupil. He also states that rigidity of the vessel walls and constipation predispose. Russell would say that these latter are one and the same thing viz. that the rigidity is due to the constipation. Like all other authors of Textbooks Fuchs omits to mention that arteriosclerosis is found in nearly all cases of Glaucoma.

Parsons (24) in his "Pathology" states that the lymph in the eye is not a secretion but a filtrate and that therefore the intraocular pressure is directly dependent upon the blood pressure, any loss or increase being compensated. This may be so in health but it does not follow that this compensation can be carried out where the
vessels are diseased and where perhaps as Thomson Henderson asserts the normal outlet is greatly diminished.

A volume recently published by William Russell (1) on hypertonus etc. is most interesting as instructive, and a brief mention of some of his points will make these references to the literature more complete. He describes normal arterial tension as a sustained measure of contraction of the individual fibres of the artery, controlled by the vasomotor fibres of the Sympathetic, and when this is cut, soon restored by the blood pressure. This normal tonus is exaggerated during digestion and physical effort (physiological hypertonus). If this exceeds the normal we get hypertonic contraction. If this again be continued for a long time the
changes in the vessels known as arteriosclerosis result. Clinically these may be:

I. Hypermyotrophy.

II. " " with thickened intima.

III. " " " " " " and externa.

This change is general, affecting all the vessels in their entire length. In discussing the causes of hypertonus, Russell lays great stress on the composition of the blood. Certain substances when in the blood are known to cause hypertonic contraction such as digitalis etc., but perhaps more important for clinicians it is also produced by nitrogenous waste and the products of imperfect metabolism and also by tobacco and alcohol. In
another chapter Russell points out that toxic absorption from the intestinal canal plays an important part in the causation of hypertonus, and I have been struck with the number of cases of Glaucoma which on inquiry have suffered from constipation most of their lives. Russell thinks that the high sphygmomanometer readings (over 200 m.m.) are partly due to increased blood pressure and partly to thickened vessel wall, but the latter contention has been lately denied by Leonard Hill (25). In chronic interstitial nephritis for instance Russell states that the pressure seldom comes below 200 m.m. but rises to 230 m.m. or more when complications such as uraemia are pending. So in bad cases of Glaucoma, I believe the pressure is generally above normal, but a marked rise is
generally coincident with an acute attack. For clinical purposes, absorption of toxic products from the intestinal canal always applies to decomposition of albuminous material in animal foods. This sort of patient may have enfeebled digestion and chronic constipation, and slowly develops arteriosclerosis. Another important point is that as age advances these toxins seem to have a more pronounced effect. Free living is often not harmful if there is also free evacuation of bowels. The ideal conditions for health are found in a moderate proteid dietary with free bowel evacuation daily.

The occurrence of similar circumstances in the histories of cases of arteriosclerosis and cases of Glaucoma suggests strongly to my mind
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The occurrence of similar circumstances in the histories of cases of arteriosclerosis and cases of Glaucoma suggests strongly to my mind
that the connection between these two diseases is no mere coincidence, and I therefore maintain that it is the duty of every ophthalmic surgeon to keep in touch with the work of physicians on arterial disease, not only in regard to pathology, but perhaps especially in regard to treatment. In this connection, it will be well to mention the usual methods employed by physicians for controlling supernormal blood pressure. An excellent article was written on this subject by Oliver (2) in the Lancet. The diet should be such as to be least stimulating to the circulation, the bulk of every meal should be reduced, salts, extractives, and alcohol, which are directly absorbed and which stimulate the heart and capillaries, should be taken sparingly. It is best to have meats boiled
and served without gravy, red and white meats if roasted being equally harmful. The indication is to diminish animal and to increase vegetable food. The bulk of meals should be reduced by reducing the amount of fluid taken with them. A tumblerful of water an hour before meals and also night and morning should be ordered, the water being hot and also soft and non-aerated. Hard water is always best avoided by elderly persons - a slice of lemon added to hard water improves it. Tea and coffee are permissible in strict moderation and only when not followed by palpitation or wakefulness. Alcohol is best dispensed with, but small quantities are not injurious if we exclude wine and beer. Oliver considers that whiskey and gin in small doses are vasodilator. In bad cases a salt-free
diet is given but only for short periods. Milk diet is also very good. A lacto-vegetarian diet is best (bread, rice, green vegetables, and fruit) especially when combined with cream, cheese, and eggs. The best form of fish is fresh water fish such as trout. Condiments are admissible in low salt diet. When the blood pressure is above 220 m.m. salt-free diet is very good, especially where there is albuminuria. It must be given only when chlorides are not properly eliminated by the kidneys. It is important therefore to estimate the chlorides in the urine and also the urea and xanthin bases. (Cantonnet (26) has employed this salt-free diet with advantage in Glaucoma but does not in my opinion lay enough stress on the importance of first estimating the chlorides in the urine).
Tobacco is harmful especially cigars and cigarettes because a certain amount of chewing is inevitable, and it should therefore be given up where there is high pressure. In very high pressure cases absolute rest is essential e.g. a day once a week, or for longer periods according to the case; the assumption of exercise after the rest to be gradual. In some cases, the patient becomes lethargic and when this is so, regular moderate exercise (walking) must be ordered. Straining at stool must be avoided and in this connection I happen to know that a celebrated ophthalmic surgeon who became blind from Glaucoma had habitually strained all his life. Warm massage douching (Aix les bains) is better than ordinary warm baths even with chlorides and carbonic acid. D'Arsonval high frequency current is recommended for 20 minutes daily.
49.

True (27) found this treatment valuable in Glaucoma as a reducer of tension. The patient should be clad in warm woollen clothing and where possible should live in warm climate in Spring and Winter (Vide Steindorff on the effect of climate in Glaucoma). Turkish baths or the lamp bath (home Turkish Bath) are useful once or twice a week, followed by strong rubbing to remove effete epithelium. This relieves the plasma and the tissues of excess of sodium chloride and metabolic residues. Blood letting is indicated where dangerous symptoms are threatened. If constipation is present, purgatives should be given regularly, e.g. sodium sulphate, or magnesium sulphate in hot water in the mornings fasting, and blue pill at bedtime once or twice a week. Intestinal antiseptics such
as Benzonaphthol, Salol etc., are very useful. In bad cases nitrites and potassium iodide must be used. Oliver states that probably 90% of pressures above 200 m.m. are due to chronic renal disease or arteriosclerosis. Pressures ranging from 145 to 160 m.m. are doubtful as to whether they are normal or not, but they should always be treated.

To sum up my argument, I maintain that, as vascular conditions producing a definite though sometimes temporary rise of blood pressure are necessary concomitants of acute Glaucoma, and as arteriosclerosis is more or less always present when the Glaucoma has been in existence some time e.g. in Glaucoma Simplex and chronic inflammatory Glaucoma, the treatment of Glaucoma can never be regarded as complete unless means are employed to remedy the increased blood pressure. The theories put forward to account for Glaucoma are
various. Looking at the disease as primarily vascular, it seems to me that Troncoso's views are convincing and if we bear these views in mind, and combine them with other more local theories (of which I consider Thomson Henderson's the best) the latter become more adequate to explain a larger number of cases. I do not deny for instance that progressive sclerosis of the pectinate ligament after middle age is an established fact, but may it not be part of a general sclerosis which also affects the blood vessels? There is no doubt that arteriosclerosis is practically always present in Glaucoma and we should as physicians be prepared to treat it. It would be well for general practitioners to know this as they could often be of great assistance to the oculist who is called in to treat a case of Glaucoma. For
instance, when the doctor diagnoses acute Glaucoma, he generally instils eserin once or twice pending the arrival of the oculist, and will then consider he has done his duty well. In my opinion, he would improve the prognosis by giving a brisk purge, applying leeches to the temple, ordering complete rest in bed, and instilling eserin (best in oil gr. 2 to the ounce) once every hour. This of course applies to patients residing in the country at some distance from the nearest ophthalmic surgeon.

The sphygmomanometer should form part of the armamentarium of every oculist and should invariably be employed before any treatment is adopted, as the result of such an examination may materially affect the prognosis.
In three cases which came under my care the result of operation was disastrous. I have since examined the blood pressure in these cases and have found it to be 200 m.m. or over. In an acute case in which the pressure is high it is advisable to do a scleral puncture first and this may be followed either at the time or next day by iridectomy. As a matter of fact this procedure is a matter of routine with some surgeons, notably Mr. Priestley Smith. If this were always done I feel sure there would be fewer cases of extruded lenses after operation. I must say, however, that I shall not be inclined to operate at all if the pressure reading be over 200 m.m.

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The more important bearing of these arguments on the treatment will be found in cases of chronic Glaucoma and here again the assistance of the
practitioner will be very valuable. How often have we seen iridectomies carefully done exercising no material influence on the progress of this dreadful disease? In these cases I venture to hope that the control of the abnormally high blood pressure will yield good results and put off further and further the evil day of blindness. Moreover, where one eye has been operated upon and there are as yet no signs of Glaucoma in the other the patient should be carefully dieted and treated in such a way that his hypertonic contraction would be kept at a minimum or even made to disappear. Iridectomy is still our most formidable weapon against this disease, but we must not forget to insist on our patient living such a life as will be less conducive to vessel changes.
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The following brief notes of cases that have occurred in my practice during the last two years will serve to illustrate the fact that increased blood pressure is the rule in cases of Glaucoma, especially in the chronic form. The pressure was estimated by Martin's modification of the Riva Rocci sphygmanometer, and where possible more than one reading was taken, and the mean pressure given. As previously stated the readings were taken at about 11-30 a.m. after the patient had rested an hour or more. The urine was examined in all cases, but only abnormal results are here stated. I have purposely left these notes of cases until the end of the Thesis (even after
Bibliography) in the hope that more cases might be seen at the last moment.

CASE 1. vide page 6 of this Thesis.

CASE 2. vide page 9 of this Thesis.

CASE 3. vide page 11 of this Thesis.

CASE 4. Mrs. Betts, aged 65, nurse. Has had bronchitis every winter for last 13 years.

On the 4th August, 1908 she had a kind of stroke, the right arm becoming flexed and stiff, the right leg also stiff, with pins and needles all up that side - no actual paralysis, could move arm and leg with difficulty. At the same time she had severe headache. There was no affection of facial muscles and no loss of consciousness. She remained in this condition for nearly six weeks and then gradually got quite well again. On the 22nd Dec. 1908, she began to have severe pain in the
right eye with almost incessant vomiting. She however did not send for her doctor until Jan. 7th, 1909. Acute Glaucoma was diagnosed and she was sent into hospital at once. The tension was plus 3, the pupil was widely dilated and there was no perception of light. Iridectomy was performed the same day, the wound gaping rather widely after operation. The next day, the lens was found in the dressing, and there was a large hyphaema. Patient said she had coughed a little during the night. The final result in this case was nil. The patient was kept in hospital for several weeks owing to her extreme weakness and when the blood pressure was examined on the 23rd February, it was found to be no less than 240 m.m.* There was no albuminuria. A year previously she had been tested for glasses. The following was her refraction:

* normal pressure being 120 m.m.
R.V. $\frac{6}{24}$ with -13 sph.

L.V. $\frac{6}{24}$ with -14 sph. and -2 cyl. ax. 165

Myopic choroidal lesions each eye.

The association of myopia with Glaucoma is very infrequent, but I consider that the high blood pressure in this case bears a definite causal relation to the Glaucoma.

CASE 5.

T. Rainbow, aged 75. Farm labourer. Admitted April 25, 1906. Chronic inflammatory Glaucoma each eye. R.V. $\frac{6}{18}$, field contracted very much on nasal side. L.V. perception of light only.

Iridectomy was performed in each eye and although technically the operations were perfect, the vision in the right eye fell almost at once to perception of light only. Pressure estimated
April 1909, equals 210 m.m.

CASE 6.


L.E. Absolute Glaucoma, no perception of light, blind some months. History of Chronic constipation. Blood pressure 180 m.m. Iridectomy was performed in the right eye and the above vision has been retained.

Case 7.

B. Lovell aet. 64. Shoe worker. Admitted February, 1908, with Glaucoma Simplex in each eye.

R.V. Hand movements only.

L.V. 18, field contracted nasal side. Iridectomy each eye, both healing well. April, 1909, blood
pressure 180 m.m. L.V. 6 with some difficulty with +1 sph. - 2 cyl. ax. 90.

CASE 8.

H. Ringrose, aet. 59. Farm labourer.

First admitted for Right Chronic Glaucoma March, 1908. R.V. 6/50 only, field much contracted. Right Iridectomy March 5, 1908, healed well. At that time there was no affection of left eye. Early in May, however, L.V. was noted 6/12 very slight limitation of nasal field, disc pale. Left Iridectomy May 8th, 1908. In April, 1909, R.V. was found to be Fingers only. L.V. 6/12 without glass, the astigmatism following iridectomy having worn off. Field nearly full, blood pressure 180 m.m.
CASE 9.

Mary Lillyman, aet 50. Admitted as outpatient Feb. 1908. R.V. \( \frac{5}{6} \) disc slightly cupped, vessels displaced to nasal side, field slightly contracted regularly. L.V. No perception of light, deep cup (Glaucoma Simplex). Both eyes were operated upon during March, 1908. April, 1909, L.V. \( \frac{6}{9} \) with +6.50 sph. +1 cyl.ax. 180. Blood pressure 160 m.m. History chronic constipation and great family worry.

CASE 10.

W. Allum. aet. 66, shoe worker. Admitted February, 1908, R.V. \( \frac{6}{12} \) with +3.50 sph. field full, no cupping. Left eye, pale cupped disc, very contracted field, L.V. \( \frac{6}{18} \) Iridectomy performed, April, 1909, L.V. \( \frac{6}{24} \) with glass. Pressure 170 m.m.
CASE 11.

Mrs. Rainbow, aet. 67. Admitted Nov, 14th, 1908. Nearly absolute Glaucoma in each eye, with great pain. Vision only perception of light in each eye. Right Iridectomy performed to relieve pain, wound remained open and five days later became septic, necessitating evisceration. Pressure 170 m.m. Albuminuria present.

CASE 12.

Ellen Dixon, aet. 49. Admitted July 5th, 1908, with right Glaucoma Simplex R.V. 60. Left eye normal. Right Iridectomy performed. Pressure only 140 m.m. History of chronic constipation and prostrating headaches.

CASE 13.

M.A. Green, aet. 65. Admitted September, 1907, with Subacute Glaucoma in each eye of several weeks
duration. Vision was only perception of light in each eye. Iridectomy was performed but no improvement in sight followed. Pressure estimated April, 1909, was only 145 m.m. although there was distinct thickening of the radial arteries. She has been very feeble for many years.

CASE 14.

M. Dunkley. aet 70. Admitted August, 1906. Absolute Glaucoma each eye. Right had been previously removed on account of pain. Left iridectomy attempted, but in this case also the wound refused to heal and the lens was extruded and the eye gradually shrank. Blood pressure estimated April, 1909, 180 m.m.

CASE 15.

Mrs. Stokes. aet. 70. Seen March 26th, 1909, found to be suffering from acute Glaucoma in the left eye, coming on after an attack of Influenza.
Her heart was dilated and an anaesthetic could not be given. Iridectomy was performed the next day under cocaine. The iris was atrophied, and the patient had no pain during or after the operation. The blood pressure estimated at the time was only 120 m.m. This patient suffered from extreme debility. In these low pressure cases I think it is possible that the great peripheral resistance of the sclerosed vessels has caused a dilatation of the heart and aorta, and the pressure becomes lowered on account of aortic regurgitation.

CASE 16. Eliz. Hayes, aet. 46. Publican, Admitted February 18, 1909, with chronic inflammatory Glaucoma in right eye. R.V. Fingers only at $\frac{1}{2}$ metre. History of subacute attack in Sep. 1907,
since then the eye has failed. February 22nd, R. iridectomy with scleral puncture. Mar. 6th, pressure 160 m.m. Mar. 16th, R.V. perception only.

CASE 17. Mary Humpage. aet. 60. Admitted Dec. 16th, 1908, with chronic inflammatory Glaucoma in each eye. History of bilious attack last fortnight with pains in eyes. R.V. \( \frac{6}{4}\) with +4.50. L.V. less than \( \frac{6}{60}\) with +4.50.

Dec. 16th, right scleral puncture.

Jan. 2nd, 1909, right iridectomy. Feb. 7th, left iridectomy. Blood pressure Feb. 7th. 174 m.m. March 2nd, R.V. \( \frac{6}{36}\) with glasses. L.V. as above.

Fields examined Dec. 16th, 1908, were only slightly contracted but on March 2nd, 1909, there was practically no nasal field left in either eye.
CASE 18. Ellen Johnson, aet. 35. Admitted Feb. 19th, 1909. Three days previously she had a foreign body in left eye. Since then eye very painful. On admission, left cornea hazy, tension plus 2. Feb. 25th, left iridectomy with scleral puncture. Feb. 26th, right cornea hazy, subacute Glaucoma. Right scleral puncture same day.

March 8th, right iridectomy. March 6th, pressure was 170 m.m. March 22nd, R.V. 18 with glass, L.V. 24 with glass.


After writing this Thesis, it occurred to me that the opinion of an authority on Glaucoma such as Mr. Priestley Smith might add strength to the arguments contained therein. I therefore wrote to him and asked him for his experience in this direction, and received in answer the following letter:

Dear Dr. Harries-Jones,

I am glad to answer your question, but I can only give my experience in a general way for I have not yet tabulated my results as regards blood pressure. I have tested the radial systolic pressure with Hill's Pocket Sphygmometer made by J. J. Hicks, of Hatton Garden, London, in many cases of chronic (simple) primary Glaucoma. In many, I think in the large majority, the blood pressure has been above 150 m.m., and in some much above. More
significant than this, I found variations in the blood pressure apparently associated with variations in the condition of the eye in the same individual. I have little doubt that an excess of blood pressure bears some relationship to chronic Glaucoma. It does not offer a complete explanation of the disease, for there are some cases of chronic Glaucoma in which there is no excess of blood pressure, and there are elderly people with very high pressure blood pressure, leading to retinal haemorrhage etc., who have no sign of Glaucoma. Perhaps one might draw a parallel between renal insufficiency and the tendency to Glaucoma. In each case the organ remains competent so long as little stress falls on it, but has no margin of safety. In treating chronic Glaucoma, both before and after operation, I do my utmost to get rid of
73.

habits tending to high blood pressure - by dieting and by insisting on the abandonment of over-strenuous ways of life. Excessive mental effort continued over many years seems to be a frequent cause. All this is quite compatible with the retention theory of Glaucoma, which in my opinion stands quite unshaken. Please make any use you like of this letter.

With kind regards,

Yours very truly,

Priestley Smith.

I have not measured blood pressure in any case of acute Glaucoma.