ERASMUS WILSON LECTURES

ON THE PATHOLOGY OF

GLAUCOMA.

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LECTURE II.

INCREASE OF THE INTRA-OCULAR PRESSURE.

IN my first lecture I spoke of the normal intra-ocular pressure, and the processes by which it is maintained. I have now to bring to your notice the conditions under which this pressure becomes abnormally increased. It will be well to approach the subject from the physical side, for though we may be unable at present to explain all the phenomena of secretion and excretion by physical laws, we cannot be too careful to build our knowledge of them upon a foundation of this kind.

This apparatus (Fig. 7) will serve to illustrate in an elementary

Fig. 7.

fashion the chief causes of an increase of pressure within the eye. The glass globe represents the eye. The red rubber tube (A), conveying the water from the reservoir, represents the arteries which bring blood to the ciliary processes. The black tube (v) represents the efferent veins. The short piece (c) which joins them inside the globe has permeable walls, and permits fluid to escape into the chamber; this represents the capillaries of the ciliary processes. The other tube (o) represents the outlets through which the intra-ocular fluid escapes from the eye, of which the angle of the anterior chamber is much the most important. The vertical glass tube serves to indicate the pressure in the chamber. A continuous stream of fluid passes in through the artery and out through the vein; as it passes the capillaries it gives off a certain quantity into the chamber, and a corresponding amount escapes through the outlet of the chamber. Now the pressure in the chamber may be increased by the action of various causes.

In the first place, it might be temporarily increased by a simple encroachment on the cavity of the chamber. Please to imagine that the vessel has a flexible wall, and that I indent it with my finger. For a moment the internal pressure will be raised, the inflow checked and the outflow quickened until compensation is made; then the pressure will fall to its normal level, and the inflow and the outflow will proceed as before: or, if one could cause the wall of the vessel to contract upon its contents,

or could intrude some foreign substance into it, there would, in like manner, be a temporary rise of pressure, lasting only until compensation was made for the encroachment. Just in the same way the pressure in the eye may be increased by external pressure or by internal hæmorrhage, but the excess will soon disappear, unless there be some permanent disturbance of the secretion or excretion processes.

In the second place, the pressure may be raised by various causes directly affecting the circulation and filtration of the fluids.

1. If we raise the reservoir to a higher level, the pressure is increased and the flow is quickened throughout the whole apparatus. This may be taken to represent a stronger impulse from the heart.

2. Or, if we increase the calibre of the afferent vessel close to the chamber, we get a similar result, although the initial pressure from the reservoir remains unchanged. This illustrates the effect of a local arterial hyperæmia.

3. If it were possible to enlarge that portion of the tube which represents the capillaries, or to diminish its resistance to filtration, we could, in that way also, increase the pressure in the chamber.

4. On the other hand, if we diminish the calibre of the efferent vein, we get, as before, an increased pressure in the capillaries, an increased pressure in the chamber, and a more rapid escape through the outlet of the chamber.

5. Once more, if we leave untouched the tubes which represent the vascular system, and diminish the outflow from the chamber itself, again the pressure in the chamber rises. This represents an obstruction at the filtration angle.

6. Finally, we may cause any two or more of these disturbances to act at the same time. For instance, we may increase the secretion-pressure either by accelerating the arterial, or by retarding the venous, stream, and, at the same time, we may diminish the outflow from the chamber. Under these circumstances, the fluid secreted into the chamber will be lessened in quantity but greatly increased in pressure. This would represent hyperæmia of the ciliary processes in association with an obstructed filtration angle.

Here I must call attention to an important point in which my apparatus fails to represent the living eye. These rubber tubes are sufficiently incompressible to bear with impunity the pressure which falls upon them as they pass through the chamber. The choroidal veins, on the other hand, have thin walls which offer little resistance to external pressure, and it is evident that they would be in danger of occlusion if the pressure in the vitreous chamber were at any time to exceed the blood-pressure in their interior. Many years ago Körner, of Vienna, and more recently Birnbacher and Czermak, made a number of experiments bearing upon this point.¹ They showed that if a current of fluid be conducted through a closed chamber in a tube of thin and pervious membrane (Fig. 8), such as the intestine of a small animal,

Fig. 8.

the pressure in the chamber gradually arrests the circulation through the tube; the tube becomes dilated near the point (A) where it enters the chamber, compressed near to its point of exit (B). The cause of the phenomenon is simple. According to a well-known physical law, fluid moving along a horizontal tube of uniform diameter exerts a pressure against the wall of the tube, which decreases as the distance from the reservoir increases. In the case of the membranous tube (A B), which permits filtration along its whole length, the fluid exudes at higher pressure at A than at B; the pressure in the chamber rises until it is nearly equal to the highest pressure in the tube; the further end of the tube is then necessarily compressed. The movement of the fluid through the tube can only be maintained by providing the chamber with an outlet sufficiently large to keep the pressure in the chamber lower than that in the tube.

Birnbacher and Czermak apply this observation to the case of

¹ Von Graefe's Archiv, vol. xxxii, part iv, p. 1.

the eye, and maintain that, if the escape of the intra-ocular fluids from the chambers be arrested, the choroidal circulation must be arrested also. Now, this is not necessarily true. The law which I have stated refers only to tubes of uniform calibre. In tubes of varying calibre the pressure at any given point is modified by the diameter at that point, being increased by a larger diameter, diminished by a smaller. For example, in a tube having the form shown in Fig. 9, the pressure may be greater at B than at A.

Fig. 9.

We have, therefore, no right to assume that the pressure in a vein is necessarily lower than that in the corresponding capillaries. It is possible that the aggregate sectional area of the choroidal veins may be so proportioned to that of the capillaries that the pressure in the former may at all times be greater than that in the latter, in which case the choroidal circulation can never be absolutely arrested by the pressure in the chambers. We know that it is often seriously embarrassed, but there is no evidence that it is ever arrested. As a matter of fact, the veins of the choroid are remarkably large and numerous where they have to bear the intra-ocular pressure, while at the points where they perforate the sclera and escape from the pressure they show a sudden diminution in calibre.² This looks like a special adaptation

Fig. 10.—Filtration angle closed—no glaucoma. Fluid has probably found abnormal outlets.

to the requirement in question.

Now, the pressure changes which I have attempted to illustrate represent the action of well-known physical laws; they could be foretold from a mere inspection of the apparatus. In the living eye the conditions are, of course, infinitely more complex. There we have to deal not with a mere filtration of fluid, but with a more subtle process. The secretion which enters the chambers differs from the fluid circulating in the vessels, and under certain conditions its own constitution changes and its filtration power is altered. Here, again, is a possible cause of pressure change. But, in spite of their greater complexity, the processes in the living eye are unquestionably governed by the same physical laws, and even this rough analysis may serve to illustrate the directions in which we should seek the causes of an abnormal intra-ocular pressure.

Passing now to physiological experiment, we find that pressure changes do actually occur in the living eye from such causes as I have described. Experiments on the lower animals have established the following facts:—

An increased flow of blood to the head produced by compression of the descending aorta raises the intra-ocular pressure in a marked degree.

A local dilatation of the ophthalmic artery induced by irritation

of the fifth nerve within the skull raises it in a still more decided manner, and if this nerve be irritated during compression of the aorta, an additional rise occurs.³

Venous obstruction in the neighbourhood of the eye produces a similar effect. Ligature of the vortex veins outside the eye may raise the pressure to more than twice its normal height. Obstructions at a greater distance from the eye have much less effect; for instance, compression of the large veins of the neck and even ligature of the superior vena cava raise the intra-ocular pressure but little.³

Obstruction of the outlets through which the intra-ocular fluid escapes can hardly be made the subject of satisfactory experiment in the living eye. We have seen that the chief outlet is at the angle of the anterior chamber, and that a very much smaller escape occurs at the papilla. With regard to the papilla, the results of experiment are negative. Ligature of the nerve produces no noteworthy change in the intra-ocular pressure, and, as we know that papillitis, with swelling of the disc, constriction of the vessels, and dropsy of the sheath, is not commonly associated with any discoverable rise of tension, we may safely conclude that the posterior outlet cannot have much influence on the intra-ocular pressure. With regard to the filtration angle, my own experiments on the freshly-excised eyes of animals, described in the previous lecture, have shown that artificial closure of this outlet almost entirely stops all filtration from the chambers; and this fact has been confirmed by Leplat's more recent experiments on living animals.⁴

Fig. 11.—Filtration angle closed—no glaucoma. Secretion of aqueous and vitreous fluids suppressed.

Changes in the constitution of the intra-ocular fluids as a cause of increased pressure are also almost beyond the reach of satisfactory experiment, but it is easy to prove that albuminous fluids escape from the eye much less easily than the normal intra-ocular fluid. Using the apparatus exhibited in my former lecture, and experimenting with sheep's eyes immediately after death, I found that a limpid albuminous fluid drawn from the abdomen in a case of dropsy escaped from the anterior chamber much less rapidly than a normal salt solution.

From these observations, physical and physiological, we may safely conclude that the chief conditions which are capable of causing high pressure in the chambers of the eye are the following: (a) hypersecretion by the ciliary processes, (b) obstruction at the filtration angle, (c) serosity of the fluids. Moreover, we can comprehend how, in the presence of such disturbances, the inflow and the outflow act and react upon each other. It is obvious that, except when the eye is actually altering in size, the inflow and the outflow must be equal under all degrees of pressure, high or low. Now comes the question, What evidence have we of such disturbances in the various forms of glaucoma?

What evidence have we of hypersecretion? We should expect to meet with it in connection with irritation of the fifth nerve and hyperæmia of the ciliary vessels. Attacks of glaucoma are often accompanied and sometimes preceded by pain in the region of the fifth nerve. Other attacks are associated with injuries

² Fuchs, *Von Graefe's Archiv*, vol. xxx, part iv, p. 34.

³ Graefe-Saemisch, *Handbook*, vol. ii, p. 371.

⁴ *Annales d'Oculistique*, January-February, 1889.

which presumably involve nerve irritation in the eye itself. In such cases, though the evidence is not conclusive, there is, at first sight, reason for attributing the glaucomatous attack to hypersecretion. On the other hand, we see that trigeminal neuralgia, even when severe, and when accompanied by injection and watering of the eye, is not usually productive of glaucoma. In dental caries we have a very severe peripheral irritation of the fifth nerve, which certainly sometimes causes reflex disturbance in the eye; but we have not, as a rule, any discoverable increase of the intra-ocular pressure. Hermann Schmidt,⁵ finding that paroxysms of toothache appeared to diminish the range of accommodation, assumed that this was due to an increase of pressure within the chambers. In pursuit of this idea I made careful measurements with the tonometer in sixteen patients at the Birmingham Dental Hospital.⁶ I found a complete absence of pressure change. More-

Fig. 12.—Filtration angle open—glaucoma. History of serous iritis. Fluid albuminous. (Mr. McHardy's case).

over, in very many case of glaucoma, there is no evidence whatever of an antecedent nerve disturbance or active hyperæmia.

While, therefore, there is good reason to believe that an increased afflux of blood is sometimes the exciting cause of an attack, the hypothesis that the glaucoma process is the expression of a persistent hypersecretion remains a hypothesis and nothing more; and, as Adolph Weber has pointed out,⁷ it is in-

Fig. 13.—Iritis. Annular posterior synechia. Secondary glaucoma.

sufficient even as a hypothesis. A rise of pressure, due simply to hypersecretion, would tend to correct itself by a gradual dilatation of the outlets, whereas the glaucoma process tends to in-

tensify it. And more important still, the idea of a persistent hypersecretion is negated by the presence in glaucomatous eyes of certain changes which render hypersecretion a physical impossibility.

What evidence have we of obstruction at the filtration angle? In the year 1876 Max Knies⁸ and Adolph Weber,⁹ following the clue given three years earlier by Leber, and working independently of each other, pointed to a well-marked obstructive change at the angle of the anterior chamber. This discovery marks an epoch. In my opinion, the most important addition to our knowledge of the subject since the time of Von Graefe is to be found in Weber's work, *Die Ursache des Glaucoms*. Hundreds of eyes have now been examined, and in the large majority this obstruction of the filtration angle has been found. I have myself examined more than seventy eyes, representing glaucoma of many varieties, and, with the exception of two or three, of which I will speak immediately, I have found the filtration angle compressed or closed in every instance.

Fig. 14.—Wound of cornea. Permanent abolition of anterior chamber. Secondary glaucoma.

To emphasise the importance of this pathological change, I may remind you of the experiments described in my first lecture, which proved that the escape from the anterior chamber varies greatly in amount according as the filtration angle is more or less widely open, and that when this outlet is closed the escape of fluid from the whole eye is almost completely arrested. It must be noted also that Leber has proved by injection experiments that fluid escapes with great difficulty from eyes which have been blinded by glaucoma.¹⁰

Now this obstruction of the filtration angle is sometimes found in eyes which have no history and present no sign of glaucoma, and on this ground some writers have urged that it cannot be an essential factor in that disease; but in such eyes we can always find, I think, other changes which explain the absence of the glaucoma. I have many specimens of the kind in my collection. In some the fluid appears to have found an abnormal outlet. Fig. 10 was drawn from an eye in which the cornea had been partially destroyed by suppuration and replaced by new tissue, lined throughout by the remnant of the iris. Here the filtration angle is absolutely abolished, and yet there has certainly been no

Fig. 15.—Central perforating ulcer of cornea. Secondary glaucoma.

glaucomatous tension, for the disc shows no trace of excavation. The secretion of the intra-ocular fluid has not been suppressed, for the retina is not detached, and the soft pseudo-cornea has

⁵ Von Graefe's Archiv, vol. xiv, Part i, p. 107.

⁶ Glaucoma, its Causes, etc., 1879, p. 11.

⁷ Von Graefe's Archiv, vol. xxiii, Part i, p. 1.

⁸ Von Graefe's Archiv, vol. xxii, Part iii, p. 163.

⁹ Von Graefe's Archiv, vol. xxiii, Part i, p. 1.

¹⁰ Report of Seventeenth Meeting of Ophth. Soc. of Heidelberg, p. 131.

been distended by pressure from within. I suspect that in such cases the intra-ocular fluid finds exit through the pseudo-cornea. Vessels permeate the new tissue; the endothelium which, as Leber has shown, renders the healthy cornea impervious, is here destroyed; and the lining of iris tissue is extremely attenuated.

In other specimens presenting closure of the filtration angle without glaucoma the secretion process has evidently been arrested. When the ciliary processes are damaged by inflammatory changes the vitreous shrinks, the lens degenerates, vicarious exudations fill the spaces which are left, and the eye becomes too soft rather than too hard. Fig. 11 was taken from an eye which was blinded by cyclitis and infiltration into the vitreous, after fever, six years before excision. The vitreous body has disappeared, the lens is a white degenerated mass, the aqueous chamber is abolished—the normal secretion process has been totally suppressed. In such an eye glaucoma is impossible, unless, indeed, a blood vessel ruptures; then blood pours into the closed eyeball, and the tension may be very high for a considerable time, for the outlets, long disused, have become useless.

Fig. 16.—Spontaneous dislocation of lens into anterior chamber. Secondary glaucoma.

Finally, what evidence have we of serosity of the intra-ocular fluids as a cause of high pressure? I have already mentioned an experiment which shows that albuminous fluid escapes from the anterior chamber much less easily than a normal salt solution. Now, in all forms of iritis there is probably more or less albuminous exudation into the aqueous chamber, but so long as the inflammation is limited to the iris, there is usually little alteration in the tension of the eye, for the aqueous humour comes, as we have seen, almost exclusively from the ciliary processes, and is secreted so copiously that a slight morbid admixture from the iris does not greatly affect its filtration power; but if the processes are involved changes of tension are sure to follow. If the inflammation be intense, with plastic or suppurative exudation, the secreting organ is rapidly impaired and minus tension soon sets

Fig. 17.—From same eye as Fig. 16.

in; if, on the other hand, it is comparatively mild, with serous exudation, then we get the cloudy aqueous, the punctate deposits on the cornea, the dilated pupil, and the high tension which characterise the so-called serous iritis, a condition which should, I think, be called serous cyclitis. Fig. 12 was drawn from a typical specimen kindly given to me some years ago by Mr. McHardy. The filtration angle, far from being closed, is widely distended; the disc is deeply cupped. This is a glaucoma which owes its high tension to the serous nature of the fluid, and also perhaps to

tissue changes around the filtration angle. In many other forms of glaucoma serosity of the fluids is probably a supplementary cause of the high tension, for whenever the circulation of the blood is obstructed serum is apt to escape from the capillaries.

It appears, then, that hypersecretion is sometimes concerned in the onset of glaucoma; that serosity of the fluids plays an important part in those forms which present a deep anterior chamber and a wide filtration angle, and perhaps in other forms as well; and that obstruction at the filtration angle is a part of the glaucoma process in the great majority of cases.

Fig. 18.—Lateral dislocation of lens by a blow. Secondary glaucoma. B Diagrammatic.

Now, it is not yet universally admitted that the high pressure of glaucoma is directly connected with the latter change. We are told by some writers that the closure of the filtration angle occurs at a late stage of the disease; that it is a consequence, rather than a cause, of the high pressure; that the essential cause of the high pressure still remains to be discovered. Laying aside generalities, let us, so far as it may be possible, examine each variety of glaucoma in turn, and ascertain its mode of onset.

Here it will be convenient to divide all forms of glaucoma into two groups, the primary and the secondary, and a very simple definition will suffice to distinguish them: the primary forms are those in which we cannot see any previous disease in the eye which accounts for the glaucoma, the secondary are those in which we can see such a disease. Beginning with causes which are visible rather than with those which are invisible, I shall endeavour, in the first place, to sketch the sequence of events in various forms of secondary glaucoma, and I am able to lay before you specimens and drawings of nearly all the forms described.

Fig. 19.—From same eye as Fig. 18.

Annular Posterior Synechia.—After repeated attacks of iritis, the pupil margin remains widely adherent to the lens. We propose iridectomy, but the patient refuses, for the tension is normal and there is no pain. Some months later the patient returns, the eye now being painful, injected, and hard, and the iris bulging forwards against the cornea. An iridectomy is at once performed, fluid escapes freely from behind the iris; the iris returns to its

normal plane, and the glaucoma is gone; or, if the eye be already blind, we remove it and find the iris-base widely in contact with the cornea, as shown in Fig. 13. Here the cause of the glaucoma

Fig. 20.—Needle operation for cataract. Secondary glaucoma.

is obvious. The passage from the posterior to the anterior aqueous chamber becomes closed or insufficient; the iris is pushed forwards by the fluid imprisoned behind it; the filtration angle is closed, and with that closure high tension sets in. A small aperture in the iris suffices to liberate the imprisoned fluid, reopen the filtration angle, and to banish the high tension. In a recent case of my own I merely punctured the iris through the margin of the cornea with a keratome. The patient being restless, I was unable to seize or excise any portion of it; the bulging disappeared, however, and the high tension did not return. In some of these cases the tension remains permanently sub-normal after the operation, showing that the ciliary processes are permanently damaged. While the outlet is closed, their secretory power suffices to cause high tension, but when it reopens the impoverishment of the supply becomes manifest.

Perforating Wounds and Ulcers of the Cornea.—The anterior chamber is opened by a large wound or ulcer, the aqueous escapes, the iris applies itself to the cornea and becomes adherent. So long as the aqueous drains away, the eye remains soft; when the aperture closes it becomes hard, for the filtration angle is closed throughout a large part of the circle. Fig. 14 was drawn from an eye excised six weeks after receiving a jagged wound in the cornea. The tension was very high. The anterior chamber had not been re-established; the filtration angle remained closed. Fig. 15 was drawn from an eye blinded by glaucoma, following central ulcer of the cornea. Here the lens had become adherent to the corneal cicatrix and the pupillary margin of the inflamed iris had adhered to the lens. When the leakage stopped, the lens retired a little from its advanced position, the adhesions becoming stretched, and the iris being doubled upon itself and pushed forwards by the imprisoned fluid. Here again the filtration angle is occluded.

Dislocation of the Lens into the Anterior Chamber.—Both lenses have been imperfectly suspended and oscillating from birth, but the patient has never had pain or inflammation in the eyes. One day while stooping over the wash-hand basin she suddenly feels something wrong in one eye. Within an hour an intense glaucoma is established. The surgeon sees that the lens is in the

anterior chamber; that outside the margin of the lens, the iris is in close contact with the cornea, and that the rest of the iris bends backwards over the posterior surface of the lens. An operation is proposed, but before it can be carried out, the lens slips back through the pupil, and the glaucoma is gone. The dislocation recurs several times, and each time with glaucoma. Ultimately the lens is extracted and the glaucoma appears no more.¹¹ In another case of spontaneous dislocation, followed by intense glaucoma, the eye having been blind before the accident, is excised.¹² The conditions seen on bisecting the frozen globe, are shown in Figs. 16 and 17. Here again the filtration angle is closed in a manner which cannot be misunderstood. Under normal conditions the intra-ocular fluid meets with no resistance in passing forwards through the pupil, but no sooner is the relation of the iris and lens reversed, than the stream which previously kept the passage open closes it with a constantly increasing pressure. The iris is firmly applied to the posterior surface of the lens; its periphery, where not supported by the lens, is driven forwards against the cornea. In some cases, as we have seen, the high pressure effects its own cure by driving the iris forwards so strongly that the distended and paralysed sphincter offers no resistance to the return of the lens when the patient lies upon the back. The great force exercised by the fluid in such cases is

Fig. 21.—Tumour of choroid. Secondary glaucoma. (Mr. Hodges's case).

the pupil.

Lateral Dislocation of the Lens.—The patient receives a severe blow upon the eye. When the swelling of the lids has subsided so far as to permit thorough examination, we find the tension increased and the vision much impaired. The pupil is dilated, but less so at one side of the circle than elsewhere, and at the less dilated side the iris lies very near to the cornea, or in contact with it. Looking into the eye very obliquely with the ophthalmoscope we see the edge of the lens behind the more dilated side of the pupil; it is dislocated laterally, and presses at the one side against the ciliary body. Possibly we are able to reduce the tension by means of eserine and cocaine, and a useful eye is retained for years; or in view of irreducible tension, we attempt to remove the lens, and perhaps succeed. Possibly the patient declines operation until excision of the hard and painful eye becomes inevitable.

¹¹ *Glaucoma, its Causes, etc.*, pp. 262, 263.

¹² *Ophth. Rev.*, 1882, p. 211.

¹³ J. J. Minor, *New York Medical Journal* 1881, p. 194.

I have been able to examine two specimens of this kind. Figs. 18 and 19 were taken from one of these.¹⁴ The lens was pressed against the processes and iris at the one side of the circle, and very firmly pressed, for it was actually indented by the processes. It was wedged in, so to speak, between the ciliary body in front and a consistent vitreous behind. The filtration angle was closed, not only where it was compressed by the lens, but more or less throughout the whole circle. I believe that the vitreous body, displaced at the one side by the intrusion of the lens, was driven forwards the other, in such a way as to effect this closure.

Injury of the Lens.—In performing preliminary iridectomy in a case of senile cataract, the surgeon accidentally touches the lens with the point of the keratome; twenty-four hours later the eye is very hard; the lens is swollen and manifestly presses the iris against the cornea throughout a large part of the circle. The cataract is extracted without delay, and the eye recovers perfectly with normal tension and a good anterior chamber.¹⁵ In the same way a sudden swelling of the lens after a needle operation sometimes causes compression of the filtration angle and high tension; and in the same way evacuation of the lens matter, or even the expulsion of a large portion from within the capsule into the anterior chamber relieves it. Again, traumatic cataract from accidental injury often leads to glaucoma; though here, the manner of its occurrence may be less easy to distinguish, for the swelling of the lens may be complicated with synechia or prolapse of the iris, or with inflammatory exudations into the chambers.

Removal of the Lens.—Glaucoma sometimes occurs in an eye from which the lens has been removed. The manner of its occurrence is not always the same.

In some cases, an annular posterior synechia locks up the posterior chamber, and causes bulging of the iris with closure of the filtration angle, just as when the lens is present. In an infant, four months old, I needled both lenses for lamellar cataract. Both pupils cleared centrally, but remained small through posterior synechia. Seven years later, I saw the child again with glaucoma in one eye. The pupil was small, undilatable, and deeply seated; the rest of the iris bulged forwards against the cornea. A small iridectomy gave exit to the imprisoned fluid; the iris retired from the cornea; the eye recovered with normal tension and useful vision. Fig. 20 was drawn from an eye blinded by this form of secondary glaucoma. Two or more needle operations, for congenital cataract, were performed by a well-known surgeon, when the child was nine months old; the result appears to have been a clear, but adherent pupil and a persistently increased tension. The blind and distended eye was removed by myself seven years later. The pupillary margin is adherent throughout to the remains of the lens periphery, and the iris is bulged forwards in the manner already described. The pupil is not blocked by any visible false membrane, but the vitreous body appears to have opposed the free escape of the fluid from behind the iris. The iris adhered to the cornea in two places where the needle punctures had been made.

Fig. 22.—Tumour of choroid. Secondary glaucoma.

In other cases the iris, the capsule, and the hyaloid are coated and united by inflammatory exudation, so as to form an impermeable or insufficiently permeable diaphragm between the vitreous and aqueous chambers; an excess of fluid becomes imprisoned behind it, and the filtration angle is more or less compressed. This may happen, although a good iridectomy has been made. In a case of this kind, on the eighth day after the extraction, and in the presence of acute iritis and very high tension, I tore through and excised a part of the partition. The eye recovered, with normal tension and good vision.¹⁶

In other cases, again, the iris adheres to the cicatrix of the extraction wound, and the filtration angle is closed throughout the corresponding part of the circle; this complication is more likely to occur when no iridectomy has been made, but is not confined to such cases.

In a few cases glaucoma occurs after what appears to be a faultless extraction and a normal healing process. It seems probable that either through a fault in the membranes which separate the two chambers, or in the vitreous fluid, there is an insufficient filtration from the vitreous to the aqueous chamber. This is not mere conjecture. In an elderly woman I performed a preliminary iridectomy, and later an extraction, apparently with complete success. A few months later an insidious glaucoma began, which at first yielded to eserine, but later became persistent. The field contracted, the disc became cupped. Sclerotomy with a Graefe knife was performed in the region of the extraction wound. On the withdrawal of the knife, hardly any fluid escaped, and the iris applied itself closely to the cornea, showing that there was no ready filtration from the vitreous chamber. The point of the knife was then passed in again through the same wound, and through the coloboma into the vitreous; a gush of fluid escaped, the iris retired from the cornea, and the globe became slack. The eye recovered with normal tension. Curiously enough, I operated later on the fellow eye of the same patient, and encountered almost exactly the same sequence of events. In some cases of this kind the high tension may be permanently banished by passing a cutting needle through the area of the pupil, so as to divide the posterior capsule and anterior part of the vitreous.

Intra-ocular Tumours.—Eyes which contain tumours usually become glaucomatous if excision be long delayed. If the eye be excised while the tension is still normal, the filtration angle will be found open; if it be excised after high tension has come on, the filtration angle will be found closed. I have met with no exception to this rule. Clinical observation shows that the onset of the high tension corresponds in point of time with a displacement of the lens and iris towards the cornea. This displacement is not caused by direct pressure of the tumour against the lens or ciliary body, for it occurs when the tumour is limited to the posterior hemisphere (see Fig. 21). It is caused by a general overfulness of the vitreous chamber. This overfulness may, perhaps, sometimes be due simply to the intruding bulk of the new growth, but this is not the usual cause, for, as the tumour enlarges, the

¹⁴ *Ophth. Rev.*, 1883, p. 257.

¹⁵ *Ophth. Rev.*, 1884, p. 264.

¹⁶ *Glaucoma, its Causes, etc.*, p. 273.

Fig. 23.—Tumour of retina. Secondary glaucoma.

Fig. 24.—Tumour of choroid. Filtration angle open. No glaucoma. (Mr. McHardy's case.)

vitreous commonly shrinks in even greater degree, and the new growth may reach a large size before the tension rises. The chief factor, as Fuchs has pointed out,¹⁷ is probably the compression of the large choroidal veins, and the consequent outpouring of serum between retina and choroid which the tumour occasions. The overfulness of the vitreous chamber closes the filtration angle, and with this closure the glaucoma begins.

Fig. 22 was drawn from an eye in which a tumour of the choroid, with detachment of the retina, was diagnosed several months before the patient would consent to excision. While the eye was still painless and free from obvious disfigurement, the young lady went to a ball. The same night an agonising glaucoma began. Three days later I found the eye stony hard, the aqueous chamber shallow and turbid, and the periphery of the iris visibly in contact with the cornea. Here, at the onset of the attack, the vitreous chamber was distended; the lens, processes, and iris were driven forwards, and the filtration angle was tightly closed. Later, perhaps after the excision, the lens and processes returned to their normal position.

Fig. 23 was drawn from an eye excised on account of a retinal glioma. In this case, also, the anterior chamber became shallow, and the tension increased only a few days before the excision.

Fig. 24, drawn from an eye given to me by Mr. McHardy, shows a large sarcoma which appears to have sprung from the ciliary region. Here there was no glaucoma, and the filtration angle remains open.

Fig. 25 exhibits a wide-spreading infiltration of the ciliary body and iris, probably of syphilitic nature. This specimen also was given to me by Mr. McHardy. Here the filtration angle is completely blocked, and the glaucoma was intense.¹⁸

Fig. 26 was drawn from a specimen given to me by Mr. Solomon. In the iris is a melanotic tumour from which a number of free cells have been carried by the current of the aqueous humour into the meshes of the filtration angle, where they have been caught and have proliferated. They were found throughout the whole circle; they did not, however, seriously impede the escape of the aqueous humour, for the tension of the eye remained normal up to the time of the excision.¹⁹

Intra-ocular hæmorrhage, in a previously healthy eye, is sometimes followed by glaucoma; in other cases it causes no discoverable rise of tension. The difference probably depends partly on the amount of blood extravasated, partly upon the ease with which the eye makes compensation for the intrusion. If the overfilling of the vitreous chamber is readily compensated by an overflow into the aqueous chamber, the tension rapidly returns to its normal level; if not, the filtration angle is likely to be compressed, and the glaucoma-process established. Intra-ocular hæmorrhage is a frequent cause of sudden high tension in eyes previously blinded by irido-cyclitis—eyes with a closed pupil, a shrunken vitreous, a detached retina, and, until the moment of the hæmorrhage, a subnormal tension. In such eyes the outlets have long been closed, and compensation is made with great difficulty.

In all these various forms of secondary glaucoma the onset of the high tension is manifestly connected with a

¹⁷ *Das Sarcom des Uveal Tractus*, Vienna, 1882, p. 210.

¹⁸ *Transact. Ophth. Soc.*, vol. viii, p. 67.

¹⁹ *Transact. Ophth. Soc.*, vol. iii, p. 257.

disturbance in the eye which impedes the escape of the intra-ocular fluid. The last two varieties, namely, those connected with intra-ocular tumours and with intra-ocular hæmorrhage,

Fig. 25.—Tumour of iris. Secondary glaucoma. (Mr. McHardy's case).

Fig. 26.—Tumour of iris. Filtration angle open. No glaucoma. (Mr. Solomon's case).

bear superficially a close resemblance to the primary form, the causes of which will be considered in the concluding lecture.

A CASE OF PELVIC TUMOUR TREATED BY GALVANO-PUNCTURE: CURED.

By J. INGLIS-PARSONS, M.D.

No one who has had practical experience of galvano-puncture will have the hardihood to deny its power of destroying almost any tissue with which it comes in contact. It also accomplishes this in a way that no other agent is capable of doing; and, when surgical procedure is quite inadmissible, it is a direct means of saving life, as the notes of the following case show.

Mrs. W., aged 44, married; family none; one miscarriage eighteen years ago. Menstruation always been regular, loss slight, until October 13th, 1887, when she had a severe attack of hæmorrhage, lasting several days.

October 28th. Admitted into Chelsea Hospital for Women, under Dr. Edis.

November 1st. Examination: A mass as large as a foetal head can be felt between the bladder and uterus, projecting above the pubes, and pressing down the anterior vaginal wall. The cervix can be felt high up and far back; the anterior lip is enlarged. Sound passes upwards backwards, and shows uterus to be separate from the tumour.

November 7th. The patient complains of great pelvic pain, with occasional vomiting, and pain on micturition. Ether was administered, and a consultation held. It was agreed that the tumour was either a fibroid or sarcoma; and that, from its position and

close attachment to the bladder and surrounding organs, no surgical procedure was admissible beyond puncture with a finetroc. Somewhat serious bleeding followed this procedure, and was checked in the usual way.

November 25th. As the tumour rapidly increased, Dr. Edis passed the case on to me for galvano-puncture. The first application was on November 25th, 1887, and the last on February 6th, 1888. In all, twenty-eight were made between and inclusive of these dates. The duration of the applications varied from twenty minutes to half an hour; intensity of current from 100 to 250 milliampères, average 150. Two fine insulated platinum needles were used. These were driven through the anterior vaginal wall into the tumour to a depth of one inch, and connected to the positive pole. A large copper electrode on a felt pad, placed over the abdomen, formed the negative pole. No anæsthetic was used, nor was it required, as the introduction of the needles caused no pain. The temperature rose only once on December 13th, 1887, to 102° F., but in a few days it was normal again. A depression could be felt in forty-eight hours after each application of the current, corresponding to the site of puncture, and large enough to admit the index finger. After each application, the vagina was well douched with a carbolic injection (1 in 60); an iodoform pessary was passed morning and evening. At the end of the treatment, it was impossible to introduce a needle without causing pain, showing that very little of the tumour was left. Bimanually nothing could be detected by the hand on the abdomen; *per vaginam* a hard board-like mass could be felt, spread out between the uterus and bladder, and in front of the anterior vaginal wall. She has no pain now; no vomiting. Two-thirds of the tumour have disappeared.

April 6th, 1888. The patient came up to town; has been in excellent health. On examination, the local condition remains unaltered.

October, 1888. One year after the commencement of her illness, she was in very good health; no sign of recurrence. The tumour is still one-third of its original size.

REMARKS.—Hitherto considerable diffidence has been felt in resorting to this method, on account of the difficulty experienced in insulating the needles with any material capable of adhering to them when they are driven into the tissues, and of standing the action of acids and alkalies which form at the respective poles. For if puncture be made through the vaginal wall by a non-insulated needle, the current acts on the vagina as well as on the tumour, and a sinus communicating with the tumour is the result. Whereas, if the needle be insulated so that only the point be left exposed, the current acts on the tumour alone and the vaginal wall remains intact, except for the small opening made by the passage of the needle, and which soon closes up. By this means the chances of septic infection are reduced to zero.

Galvano-puncture has often been compared by those ignorant of its use to the actual cautery. The difference, however, is very wide. With the latter, whatever tissue comes in contact with it is charred by the heat and destroyed, and it is utterly impossible to apply it to a tumour covered by healthy tissues, such as the vaginal wall, without first destroying them, and so producing a sinus with all its attendant risks. Whereas, with galvano-puncture by insulated needles, there is no damage done to the healthy tissues, which have to be pierced first in order to reach the tumour; the insulation protects them from the current, while the bare point acts on the tumour. Nor is there any development of heat sufficient to produce an appreciable effect. The reason is this: Nearly two-thirds of the electrical force are expended in electrolysis. As the current enters the body it spreads out, and finally converges on the other pole. Any development of heat, caused by the resistance of the needles and the body, is spread over a large area, and is quite insignificant. Ciniselli has given up trying to insulate needles. Apostoli, Onimus, and Legros use a movable sheath. But if this is driven into the tissues and makes too large an opening, it is apt to set up a sinus. If it is not so used, the current will come off at the point of entry and produce the same result.

The insulating material used is gutta-percha, put on in fine layers, compressed and tapered down towards the point, so as to have no shoulder. The inert mass left behind in this case is, no doubt, the fibrous tissue of the tumour, all the rest being destroyed.

BEQUESTS.—Under the will of Miss Agnes Sarah Adams, the Hospital for Diseases of the Chest, Brompton, receives £500; the Cancer Hospital, Brompton, £500; St. Thomas's Hospital, £250.