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#### THE MECHANISM OF THE INTRA-OCULAR PRESSURE IN MAMMALIA

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THE simplest procedure by which the nature of the intra-ocular pressure can be studied is by the ordinary method of ophthalmoscopic examination<sup>1</sup>. All that is necessary is to observe the most proximal portion of the central retinal vein at the disc, particularly if there is a physiological cupping, and note the constriction which takes place the instant one touches the eyelid with the lightest of light touches. The pressure of the merest touch on the skin, transmitted through the lid to the intra-ocular contents, is sufficient to cause a narrowing of the lumen of the proximal end of the retinal vein at the disc, which disappears the moment the finger is raised. It is an extremely pretty and instructive phenomenon and one which, incidentally, is much more conclusive than any experiment that human ingenuity can devise. It is, in short, the key to the understanding of the problem of the mechanism of the intra-ocular pressure.

The prompt response of the retinal veins to the slightest touch on the lids, proves how exquisitely balanced are the intra-ocular pressure and the venous pressure at the points of venous exit. Although only the exit of the central retinal veins is visible and

open to inspection, it is obvious that a similar pressure response takes place simultaneously at every other point of venous exit, including the venae vorticosae and Schlemm's canal. Incidentally Schlemm's canal is composed of merely a single layer of endothelium and thus considerably thinner than the retinal veins or venae vorticosae. By the use of our staple instrument, the ophthalmoscope, the question of the nature of the intra-ocular pressure and its equilibrium with the venous exit pressure in the eye is placed beyond discussion and above discordant laboratory findings. Further, the absence of any venous pressure response in accommodation or convergence demonstrates that, contrary to accepted views, action of the intra- or extra-ocular muscles produces no increase of intra-ocular pressure.

The extreme lightness of the touch which will produce the venous response gives an important indication of the level of the intra-ocular pressure. It supplies the requisite clinical proof that the intra-ocular pressure is in equipoise with the pressure within the veins as they pass out of the globe. This is exactly what Leonard Hill<sup>2</sup> found in the brain when he showed that the venous pressure in the torcular Herophili, a point of venous exit in the brain, is the same, a matter of 10 mm. Hg or 130 mm. H<sub>2</sub>O, as the intra-cranial pressure. The two pressures always move together so that variations in the one always produce equal and corresponding changes in the other. The venous pressure response at the disc is a practical demonstration that the same level of pressure and the same mechanism exist in the eye as in the brain. Remembering the unity in Nature's workings, this is after all only what one would expect. When Nature has evolved a mechanism, she may and does alter the details, but the working principles concerned are always constant. The fact that the eye is a special sense organ is no adequate reason for endowing the tissues that enter into its structure with a special physiology at variance with the general principles that apply to similar tissues elsewhere in the body. The nerve elements subserving vision, the optic nerve and retina, constitute, embryologically, a lobe of the brain, yet at the lamina cribrosa, they are conceived as coming under physiological conditions differing materially, both in nature and level of pressure, from those existing in the brain.

### **The Experimental Intra-ocular Pressure**

The practice of lumbar puncture has verified Leonard Hill's findings and the intra-cranial pressure is recognised as being about 10 mm. Hg or 130 mm. H<sub>2</sub>O. Intra-ocular pressure as measured manometrically by numerous observers has been found to be in the neighbourhood of 25 mm. Hg or 330 mm. H<sub>2</sub>O. This figure,

no less than two and a half times greater than the intra-cranial pressure, has long been generally accepted, but the circumstances responsible for such high readings, though visible and open to inspection by ophthalmoscopic examination, have never been taken into account. No thought has been given to what happens to the circulation inside the eye when a not too sharp canula is driven into the anterior chamber.

The effect of the localised application of pressure on the globe is similar to that produced in the hydraulic press, every square millimetre of the internal surface of the globe is subjected to the same pressure, sufficient to check the circulation and exsanguinate the eye temporarily. As soon as the canula has entered the anterior chamber, the circulation is restored with a bound. The action and reaction is felt by every cell in the eye, tissue pressure is affected, secretory and circulatory pressure raised. Under such conditions it is only to be expected that the manometric findings average 25 mm. Hg. Such readings are, however, but the reaction of the intra-ocular contents and circulation to the sudden pressure they have been subjected to. If one were to waylay a man, slap him on the face and then forcibly take his blood pressure, one would hardly expect such readings to be exactly physiological, regardless of the number of individuals on whom this experiment was made. Similarly the experimental finding of an intra-ocular pressure of 25 mm. Hg is the expression of the eye's reaction to the assault it has sustained. One admits the manometric findings but the conditions under which these high figures have been obtained must be taken into consideration.

If the experimental figure of 25 mm. Hg is to be accepted as the physiological level of the intra-ocular pressure, then, if correct, there should be no difficulty in co-ordinating it with other relevant conditions. After all correlation is fundamental to the correct interpretation and understanding of all physiological processes, but as soon as one attempts to do so in the eye, one encounters all sorts of involved and anomalous situations whose existence has been overlooked.

### **Problems arising from the alleged High Physiological Level of the Intra-ocular Pressure**

The first problem is the very natural one, why the eye should require such a high level of pressure. This, it is explained, is necessary to keep the eyeball distended and maintain its form as an optical instrument. In other words the globe is not rigid enough, but like a toy balloon, it must be kept inflated to maintain a definite shape. A study of the corneo-sclera in mammalia shows that this fibrous envelope is, however, more than sufficiently thick

to maintain the shape of the eye, without the necessity of a high intra-ocular pressure. The maintenance of the form of the mammalian eye depends not on the pressure exerted from within, but on the rigidity of its covering. We have in our daily work, ample evidence of this when performing an intra-ocular operation. If the aqueous exerted a pressure equal to 13 inches of water on the inner surface of the cornea, it would be impossible to make one's incision for extraction of cataract without fluid squirting out instead of merely oozing away, while at the same time the globe, being released from its internal pressure, should collapse.

The conditions at the disc present a whole series of problems which are quite incompatible with a high level of intra-ocular pressure (Fig. 1). To begin with the lamina cribrosa interposed between the intra-ocular and intra-cranial components of vision, is the weakest area of the corneo-scleral envelope. As its name implies, its construction is eminently unfitted to withstand any

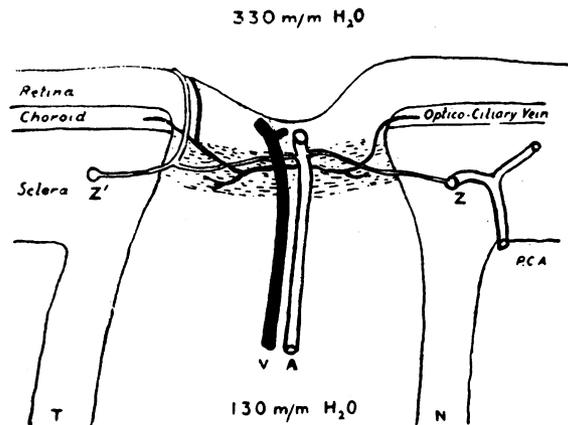


FIG. 1. Diagrammatic Section of the Optic Nerve Head.

The lamina cribrosa marks the anatomical boundary between the intra-ocular contents with alleged pressure of 330 mm. H<sub>2</sub>O and the optic nerve with pressure of 130 mm. H<sub>2</sub>O. Central retinal artery (A) and vein (V). On nasal side (N), the posterior ciliary artery (PCA) gives a branch to form the arterial circle of Zinn (Z), which anastomoses with the central retinal artery. On the temporal side (T) the circle of Zinn (Z) gives off the cilio-retinal artery. The cilio-retinal vein joins the central retinal vein (V). On the nasal side an optico-ciliary vein from the choroid drains into the retinal vein.

pressure, yet such is taken for granted, but does not make it any more possible.

The question of the venous return at the disc is even more peculiar. The anastomosing branches of the arterial circle of Zinn (Fig. 1, Z) are associated with a corresponding capillary venous network which joins the central retinal vein and a passing thought

should be spared as to how they carry on between the upper millstone of the intra-ocular pressure and the nether of the intra-neural.

Similarly the unobtrusive existence in the lamina cribrosa of the optico-ciliary veins (Fig. 1) that pass from the choroid to the central retinal vein or one of its ramifications, is somewhat of an enigma. Fluids always flow in the direction of least resistance, yet the choroidal venous return though linked with the central retinal vein, makes no use of this route to short-circuit the venae vorticosae and escape out of the high intra-ocular pressure. One can only ask why and obtain an unresponsive echo.

From these unconsidered trifles one must pass to the consideration of the central retinal vein itself (Fig. 1, V). The pressure phenomenon already detailed, shows how responsive the venous wall is to the very slightest variations in the intra-ocular pressure. If, in the globe, the venous wall is subjected to a pressure of 330 mm. H<sub>2</sub>O, what happens when the vein has passed through the lamina cribrosa and lies in the optic nerve with a tissue pressure of only 130 mm. H<sub>2</sub>O? There is no anatomical evidence that the vein distends or that its walls are in any way altered to meet the internal pressure they are now suddenly called upon to withstand. Veins are not in the habit of withstanding long continued high pressure, yet that is exactly what the central vein in its course through the optic nerve is expected to do. Incidentally too, the same question arises in the case of the venae vorticosae, for having passed through their oblique course in the sclera, how come these veins to resist such high internal pressure without support? The problem has only to be stated to stand out as contrary to all known physiological conditions.

From the clinical aspect the alleged physiological disparity of pressure on either side of the lamina cribrosa shows up even more incongruously in papilloedema. The manifestations of venous engorgement and oedema present at the disc in this condition, are the direct product of a raised intra-cranial pressure<sup>3</sup>. To produce either venous congestion or oedema it is necessary that the intra-cranial pressure rise above the intra-ocular, *i.e.*, above 330 mm. H<sub>2</sub>O.

It is an established clinical fact from lumbar puncture, that an intra-cranial pressure approaching 330 mm. H<sub>2</sub>O is a very high one, yet marked papilloedema is present with intra-cranial pressure very much lower than the supposed physiological intra-ocular pressure. How can oedema and venous engorgement appear at the disc when the pressure on the cranial side of the lamina is still below that of the ocular? (Fig. 1). Papilloedema should be a physical impossibility except in those cases where the intra-cranial pressure has risen above 330 mm. H<sub>2</sub>O. One has not exhausted

the list of similar paradoxes, but will conclude with a final clinical anachronism.

Thanks to the pioneer work of Lagrange, every surgeon has produced "fistulisation" of the corneo-sclera with a visible opening between the anterior chamber and the sub-conjunctival tissue. The resulting "filtering cicatrix" raises the question of how the sub-conjunctival tissue can support the intra-ocular pressure. Tonometric readings, for what they are worth, may indicate "subnormal tension," but then under such conditions, how comes the eye to function as an optical instrument? A high physiological level of pressure is regarded as essential to keep the globe a rigid optical apparatus, but on the other hand, an eye that has been successfully trephined demonstrates that such is not at all necessary. One cannot expect to have it both ways, and furthermore Nature is not in the habit of working at higher levels than absolutely necessary. This must be particularly the case when conformity with associated physiological conditions cannot be established.

### **The Physiological Equilibrium between the Intra-ocular and Intra-cranial Pressures**

If the above objections are valid, then I submit that my published experimental findings with Leonard Hill<sup>4</sup> are not only more likely to be correct than those generally accepted, but in addition, as they are supported by clinical proof, that they present a complete and simple explanation of the mechanism of the intra-ocular pressure in mammalia.

We found that simultaneous measurement not only gave the same level of pressure as in the torcular Herophili, about 10 mm. Hg, in both brain and eye, but these pressures rose and fell together under all circumstances. This physiological conformity is after all only what one would expect from a recognition of the following conditions:

(1) Fluids are incompressible, transmit pressure equally in all directions and invariably lie at the lowest attainable hydrostatic level.

(2) Within the closed coverings of the brain and eye, the blood stream flows through an elastic, and not a rigid, system of tubes, and the pressure within the veins as they pass out of the spheres, is the lowest circulatory pressure.

(3) In the eye, as in the cranium, the fluids lie at venous exit level of pressure because this is the lowest circulatory pressure; hence the retinal venous pressure phenomenon at the disc.

Neither the brain nor the eye possesses any extraneous mechanism to protect them from changes in the general venous or general

arterial pressures. A rise in vena cava pressure makes itself felt at once by backward pressure and the pressure within the brain and eye rises millimetre for millimetre with that of the vena cava. On the other hand a rise of arterial pressure in the aorta only produces a proportional rise of pressure, as between the aorta and venous exits in brain and eye, lie the unknown and varying resistance of the arterioles.

In other words the intra-cranial and intra-ocular pressures, while varying with venous exit pressure and influenced directly by that in the vena cava, can, on the arterial side, be regarded as representing the pumping force of the heart left over after the blood has been driven through and has overcome the resistance of the arterioles.

Leonard Hill had to lay stress on the rigid nature of the cranium to emphasise that it was circulatory pressure and not volume that is responsible for the intra-cranial pressure. The principle of the circulatory nature of the intra-cranial pressure having been once established, the nature of the covering is immaterial so long as the contents are physiologically enclosed, and this applies equally to the infantile cranium with its open fontanelles and variable volume, as to the eyeball. The material fact is that it is the general circulatory pressure on the venous side which governs the physiological level of the intra-cranial and intra-ocular pressures and maintains them in the neighbourhood of 130 mm. H<sub>2</sub>O. With such a mechanism and equilibrium the problems enumerated above cease to exist.

### The Intra-ocular Tension

If the manifestation of the venous pressure response is carried a stage further and the disc kept under observation while the globe is palpated with the finger of the disengaged hand, the sequence of events which give rise to the appreciation of the intra-ocular tension can be followed.

As the fingers are placed in position one will first perceive the immediate constriction of the retinal termination of the venous exit. As the globe is palpated one will note, most readily in children, that a retinal arterial "pressure pulse" is produced, similar to that in glaucoma. This pressure or collapsing pulse is created in consequence of the applied pressure being greater than the diastolic pressure, so that only the systolic arterial wave can pass into the eye.

This clinical observation shows that the feeling of tension imparted to the fingers, when palpating the globe, is actually due to the pressure which the fingers are exerting on the eye. The intra-ocular pressure is thereby raised temporarily from the low

level of venous exit pressure, manifested by venous pressure response, to the higher level of diastolic arterial pressure, as shown by the collapsing arterial pulse.

The intra-ocular tension as tested clinically, is nothing more than the feeling of the range or depth of resilience of the globe, when, by palpation, its internal pressure is raised from its normal venous exit level towards the arterial side of the intra-ocular circulation. In glaucoma the intra-ocular and venous exit pressures being correspondingly high the range of resilience of the globe is proportionally less and in consequence the eye feels hard.

### Summary

(1) A series of problems, clinical and physiological, are enumerated which are inexplicable, if as alleged the intra-ocular pressure is two and a half times greater than the intra-cranial pressure. They cease to exist with the realisation that the same mechanism and the same level of pressure, about 10 mm. Hg, obtain in the eye as in the brain.

(2) The key to the understanding of the mechanism of the intra-ocular pressure is the reaction of the central retinal vein at the disc to the slightest touch on the lid, supplying clinical proof of the equilibrium between the intra-ocular pressure and venous exit pressure.

(3) The intra-ocular pressure is not dependent on volume but, as in the brain, it is maintained and varies with venous exit pressure because this is the lowest circulatory pressure in the closed sphere of the globe.

(4) It is the general circulatory pressure on the venous side which governs the physiological level of the intra-cranial and intra-ocular pressures and maintains them at 10 mm. Hg. Every variation in vena cava pressure is reflected backward millimetre for millimetre in both brain and eye. Variations in arterial pressure only produce a proportional change, as between the aorta and the venous exits in brain and eye the unknown and varying resistance of the arterioles has to be overcome.

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