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THE OCULAR CIRCULATION: ITS NORMAL PRESSURE RELATIONSHIPS AND THEIR PHYSIOLOGICAL SIGNIFICANCE

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THERE is no question in the whole of ophthalmology more important and fundamental than the vascular pressures of the eye. Not only must these form the only logical basis of a rational understanding of the pathology of many ocular diseases, but, inasmuch as the mechanism of the formation and absorption of the intraocular fluids depends primarily upon their magnitude and upon their relation to the intraocular pressure, their accurate determination is of paramount importance in the physiology of this organ. There is no question in the whole of ophthalmology which has excited more unnecessary controversy and received more varying answers. Estimates of the systolic arterial pressure vary from 60 to 120 mm. of mercury, of the diastolic, from a few mm. of mercury above the intraocular pressure to 70 mm. above it; within the last few years the pressure of the exit veins has been stated by one observer to be 10 mm. of mercury below the chamber pressure, by another to be 48 mm. above it. Consequently it is stated by one that an outflow of aqueous is brought about by hydrostatic forces, by another that its absorption is effected by osmotic energy; one affirms that the pressure gradient from capillary to aqueous is sufficient to allow the formation of the latter by a process of simple transudation, another, that the pressure relations between the two necessitate the intervention of a special secretory mechanism, involving an active expenditure of energy by the cells of the ciliary body. The unwieldy literature that has accumulated upon the subject is swollen with wearisomely

protracted and often acrimonious discussions of unnecessary hypotheses, with defective and confused experiments based on unsound physiological reasoning, with a multitude of theorizings and a paucity of facts. The present paper is an endeavour to point out the principles through whose neglect this disagreement has arisen, and to suggest what may approach more nearly the solution of a problem which forms the obvious preliminary to an inquiry into the nature of the intraocular fluids, and of the mechanism of their pressure changes.

I. The Ocular Circulation

It will simplify the subsequent discussion to consider at the outset two questions which are of importance throughout the following pages: the comparison of the vascular supply of the eye in man and in the lower animals, and the comparison of the uveal and the retinal circulations.

While in man the blood supply to the eye is derived wholly from the internal carotid artery, in the lower animals it arises from the external carotid. In the lower mammals, such as are used for experimental purposes (dog, rabbit, etc.), the transition stage between the two types occurs, and, although variations are frequent, as a general rule two ophthalmic arteries are present, one derived from either source, with a free anastomosis between them (Henderson, 1903; Parsons, 1903; etc.). The corresponding blood pressures in the various organs of the higher animals are generally accepted as comparable with those of man, but in considering the ocular circulation there may be some legitimate hesitancy in transferring experimental findings obtained in laboratory animals to the case of the human eye owing to the anatomical differences between the two. While it is very probable that they are approximately the same and in every way comparable, it would seem that the difference, if any, will be in the direction of a higher pressure obtaining in the ocular arteries of man. In him the ophthalmic artery is a direct branch of the internal carotid, virtually of the circle of Willis, a vessel which may be considered as the master vessel of the body inasmuch as the whole vasomotor mechanism is adjusted to maintain it continuously at a high pressure level. Immediately after this branch is given off the internal carotid constricts: its diameter proximal to the branching off of the ophthalmic artery averages 5.4 mm., immediately beyond this point it narrows to 3.8 mm., while the diameter of the ophthalmic artery averages only 1.5 mm. (Whitnall, 1921). The narrowing is therefore out of proportion to the cross section of the vessels, a provision which, by reducing the calibre of the main vessel and damming up the blood stream, will favour the passage of blood down this important branch, and ensure a high pressure in it.

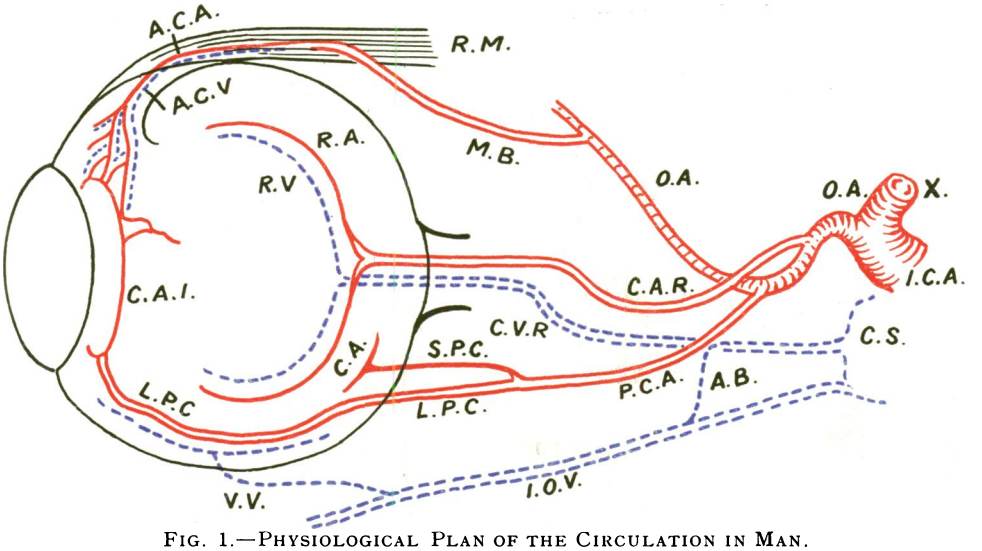

FIG. 1.—PHYSIOLOGICAL PLAN OF THE CIRCULATION IN MAN.

A diagrammatic plan of the circulation in the eye of man to emphasize the physiological principles dealt with in the text.

(I.C.A.). The internal carotid, which after giving off the ophthalmic artery (O.A.), bends round and narrows its lumen at (X). Diam. I.C.A — 5.4 mm.; diam. at X —3.8 mm.; diam. O.A. — 1.5 mm.

The branches of the ophthalmic artery involved are:

I. (C.A.R.), the central artery of the retina, breaking up into the retinal arteries (R.A.). These are accompanied by the retinal veins (R.V.), leading into the central vein of the retina (C.V.R), which enters (usually) the cavernous sinus (C.S.) directly. The central vein of the retina has always at least one anastomosing branch (A.B.), usually with the superior ophthalmic vein, here shown with the inferior. These may be considered to form, physiologically, a separate circulation.

II. The ciliary arteries comprising—

i. (P.C.A.) the posterior ciliary arteries, consisting of several short (S.P.C.) and two long (L.P.C.); the former supply the choroid, the latter terminate directly in the circulus arteriosus iridis major (C.A.I.).

ii. (A.C.A.) the anterior ciliary arteries derived from the muscular branches (M.B.) of the ophthalmic to the recti muscles (R.M.), also anastomosing with the circulus arteriosus iridis.

These two groups are to be considered as one system physiologically, the blood from which is drained away by the anterior ciliary (A.C.V.) and vortex veins (V.V.). For purposes of physiological comparison the long posterior ciliary arteries may be said to correspond to the central artery of the retina; the sub-divisions occurring in the anterior ciliary arteries prior to their entering the circulus arteriosus iridis are to be noted.

Physiologically, the vessels supplying the eye may be considered as forming two separate systems. The anterior and posterior ciliary arteries anastomose freely to form the uveal circulation, while normally the retinal circulation is anatomically apart except for a few and occasional anastomoses, which, being of little more than capillary dimensions (Leber, 1865), are of no importance from the physiological point of view: anomalous anastomoses, although they do occur, are rare (see Weizenblatt, 1926). Pathologically also, although there is some intercommunication (Kugel, 1863), a considerable amount of independence is manifested in the degree to which the two systems of vessels are involved (Coats, 1913). At the same time there is a large amount of evidence to show that, although they are anatomically distinct, in these two circulations the entrance and exit pressures are similar, that in both the same pressure gradient exists although it may be differently apportioned, and that they exhibit the same pressure variations under changing conditions.

In the arterial system, other things being equal, the pressure falls proportionately with the number of branchings and the size of the lumen of the vessels, and therefore, the central artery of the retina and the posterior ciliary arteries being both direct branches of the ophthalmic, both entering the eye directly, being both of the same order of size (Schwalbe, 1874; Henle, 1876), and being both subjected to the same external conditions along their course, we would expect them both to have approximately the same pressures. This anatomical deduction is substantiated by their physiological behaviour. Jacobi (1876) first observed the occurrence of an arterial pulse in the uveal vessels in a case of choroidal atrophy and noted that it appeared and disappeared under the same external pressure applied to the globe as did a synchronous pulse in the retinal vessels, an observation verified considerably later by Bailliart and Magitot (1925) under more exact conditions. Laqueur (1877) similarly noted the parallel behaviour of pulsation in the vortex veins and the central retinal vein, and Ulrich (1880) produced a pulsation in the former veins by finger pressure on the eye, correlating them in their behaviour with the similar pulsation produced in the central vein of the retina initially by Coccius (1853). Leber (1903) called attention to the parallelism in the pressure conditions in the choroidal arteries and those upon the disc, and Weiss (1911) brought forward evidence of the identity of the venous pressures in the two systems. Taking advantage of a case of vascularized pupillary membrane, Vossius (1921) correlated the behaviour of the vessels therein on increasing the intraocular pressure with that of those of the retina, and Bleidung (1924), compressing the globe with a pressure chamber through which ophthalmoscopic examination was possible, concluded that the pressures in the retinal and choroidal systems were the same

and varied together. The same relationship obtains in animals. Pulsation in the choroidal vessels has been known and studied in albino rabbits from the time of Waller (1856) and Liebreich (1858). Moreover, in most laboratory animals the circulus arteriosus major, instead of being placed inaccessibly at the base of the iris as in man, is situated in the substance of the iris, and is plainly visible under the magnification of the binocular loupe. The appearance and disappearance of pulsation in this vessel on applying pressure to the globe was first studied by Wegner (1866) in rabbits, and by Weber (1868) in dogs. The question was revived by Lepat (1920) who showed that in the dog the pressure changes in this vessel took place *pari passu* with similar variations in the retinal vessels; a similar relationship was noted by Bonnefon (1921) in the rabbit, and by Magitot and Bailliart (1921) in the cat. While the pressures obtained by these methods are not to be taken as correct absolutely, as will be seen later, the physiological and dynamical errors of the technique employed are common to the two cases, and do not invalidate their comparative value. We may therefore take it that the vascular pressures in the eyes of laboratory animals are comparable with those of man, and that in both, the entrance and exit pressures in the two systems of vessels—retinal and uveal— are of the same order of magnitude.

II. The Arterial Pressures of the Eye

Although several observers have claimed to have measured the arterial pressures in the eye, no one has yet succeeded in doing so. All the methods employed, with one exception, depend on raising the intraocular pressure, and observing at the same time the behaviour of the arteries of the eye and their pulsations. Before discussing these methods, therefore, it will be well to consider briefly the mechanics of the retinal arterial pulse and the conditions which determine its appearance and disappearance.

THE RETINAL ARTERIAL PULSE

Since the pulse in the retinal artery was first noted by Jäger (1854) an unnecessary amount of discussion has taken place over the mechanism of its causation. There would seem to be no reason for assuming that the pulse in the arteries in the eye is in any way different from that in the arteries in the rest of the body, or that its behaviour with reference to the intraocular pressure involves principles other than those which apply to the vessels generally when they are subjected to external pressure applied by a sphygmomanometer.

In all the arteries of the body a pulse is always present, which extends throughout the entire length of the arterial system down to the capillaries with progressively lessening amplitude. In the retina a spontaneous pulse was early noted occurring in normal

eyes by Donders (1855) and Becker (1872); it is seen with the ophthalmoscope, according to Ballantyne (1913) in 36 per cent. of normal individuals, and it is often accompanied by locomotion of the vessels. It has been noted, also occurring spontaneously, in the choroid (Galezowski, 1916). With sufficient magnification it is always seen, either in the capillaries entoptoscopically (Onishi, 1913; Scheerer, 1924; Fortin, 1926), or extending down the arteries through to the veins, by the Gullstrand ophthalmoscope (Speyr, 1914; Kümmell, 1915).

Neglecting the disturbing factor of the rigidity of the arterial wall (Thoma, 1889) it is more easily seen, and can often be traced with the ordinary ophthalmoscope to the finest arterial ramifications if the arterial pulse is of large amplitude, as occurs in aortic insufficiency (Quincke, 1868; Mackenzie, 1875; Holloway, 1917; Wolff, 1917; Zentmayer, 1921), or in aortic aneurysm (Becker, 1872; Helfreich, 1882; Raehlmann, 1885), or in Graves's disease (Becker, 1873). In some of these cases it may be seen in the capillaries upon the disc (Quincke, 1868) as an alternate deepening and lightening of the normal tint. It becomes very marked if the intraocular pressure and the arterial pressure approximate; and when the diastolic pressure is reached it changes its character, and the "expansile" pulse becomes a "pressure" pulse at the disc, the arteries emptying and filling alternately, and the blood column flashing out of sight across the disc and filling up again suddenly. This occurs either when the intraocular pressure is high, as in glaucoma (v. Graefe, 1855; Jacobi, 1876; Ballantyne, 1913; Krämer, 1920; Bailliart, 1922), or on compressing the globe (v. Graefe, 1854; and others *vide infra*), or when the arterial pressure is low —either the general arterial pressure, as in debilitating diseases as anaemia (Pflüger, 1878; Becker, 1880; Raehlmann, 1885; Schmall, 1888; and others), or cholera (v. Graefe, 1866), or in syncope (Wordsworth, 1863; Ballantyne, 1913), or the local arterial pressure, as in embolism of the central retinal artery (Elschnig, 1892), or in the presence of retrobulbar orbital tumours (v. Graefe, 1864).

This pressure pulse is communicated by the incompressible ocular contents to the elastic sclera, where it becomes a volume pulse. In the normal eye this latter structure is so poorly distensible that the volume pulse is damped down beyond visible limits, but in conditions where the distensibility of the envelope of the eye is increased, such as in high myopia with a posterior staphyloma (Kyrieleis, 1925) or in keratoconus, or nearly perforating corneal ulcer, the pulse is readily seen (Javal, 1884; Gullstrand, 1891; Collies, 1891; Wagenmann, 1898; and others). When magnified by the lever of a tonometer, however, this volume pulse is always seen in the normal eye (Schiötz, 1905; Foster Moore, 1917; etc.), and again, if the continuity of the corneo-sclera is broken, and the contents of the eye are put into direct communication with an open manometer, the arterial pulse is imparted plainly and undiminished to the fluid in the latter. This was first observed by Weber (1850); and Bellarminoff (1886), registering the excursion of the fluid photographically, demonstrated its amplitude to be 1 to 2 mm. Hg, a finding confirmed by many subsequent observers.

This volume pulse is due to the intraocular arteries and is not communicated from the orbital vessels, since, on inducing local endocular hyperaemia (by subconjunctival saline injections, etc., Wessely, 1908) the ocular pulse increases proportionately, while a simultaneous carotid tracing remains unchanged. Inasmuch

as the choroidal vessels form eight-tenths of the entire circulation of the eye, the pulse will be largely due to their influence. This is demonstrated by the fact that clinically, in cases of embolism of the central artery of the retina, when this vessel is occluded, the pulsations of the eyeball as shown by the lever of a tonometer proceed as usual. As has been noted, the volume pulse communicated from the uveal vessels behaves in every way parallel with the pressure pulse seen in the retinal circulation.

THE MEASUREMENT OF THE ARTERIAL PRESSURE

In the normal eye therefore the arterial pulsation is very small, but on raising the intraocular pressure it becomes progressively larger until the diastolic pressure is reached; at this point the arteries are completely collapsed during a portion of the cardiac cycle, and the pulsation is at a maximum. On increasing the pressure still further, the amplitude of pulsation will progressively diminish until, when the systolic pressure is overcome, the blood flow ceases, pulsation stops, and the artery collapses.

In this way, it is to be noted, the eye is converted into a natural sphygmomanometer, and when the vessels are compressed the column of blood contained in them is partially or completely immobilized. The pressure thus registered is therefore not that of the vessels in the eye at all, but the lateral pressure at the most proximal arterial branching, that is, of the ophthalmic artery. Just in the same manner as a manometer in the carotid measures the lateral pressure in the arch of the aorta and not that in the carotid artery itself, or a sphygmomanometer over the brachial artery registers the pressure in the subclavian, so the lateral pressure in the ophthalmic artery is measured through the central artery of the retina by observing the retinal pulse, or through the posterior ciliary arteries by measuring the volume pulse of the globe.

In order to get a strictly correct interpretation from the pressures measured, there should be deducted from them the force necessary to overcome the resistance of the artery wall, a factor which varies with its rigidity and the degree of sclerosis. That such a consideration is not inconsiderable in many cases is seen in the observations of Foster Moore (1916) on the retinal arteries. A further correction factor should be applied depending on the length of the immobilized column of blood, on the acuteness of the angle at which the branch vessel leaves the parent stem (since, if it does not leave at right angles, in addition to the lateral pressure of the feeding artery, there is added to the pressure measured some of the kinetic energy of the forward moving stream), and depending further on the relative capacity of the occluded channels and the remaining channels (since, if the former be of any size, the pressure in the latter is increased correspondingly by engorgement), while in addition, if the vessels are large, the purposive action of the heart in attempting to overcome the obstruction thus caused must be allowed for. In the present case, however, these factors may be neglected for all practical purposes, and the pressures thus measured may be considered as representing the lateral pressure in the ophthalmic artery.

The intraocular pressure has been raised by two methods:

(1) *The Manometric Method.*—A manometer is inserted into the eye and the pressure therein raised by forcing in saline. Coincidently the behaviour of the retinal arteries has been studied,

the points of maximum oscillation and cessation of oscillation being taken as the diastolic and systolic pressures respectively. These points have been determined in two ways:

(*a*) By the Oscillatory Method.—By recording the points of maximum oscillation and cessation of oscillation as conducted to the mercury column of the manometer. The amplitude of these oscillations, first noted by Weber (1850), was correlated with variations in the intraocular pressure by Hippel and Grünhagen (1869), whose observations were substantiated by Hoelzke (1883), Bellarminoff (1886), Stocker (1887), and Koster (1895). Wessely (1908), recording the oscillations graphically with a membrane manometer, obtained the first reliable results. Weiss (1911), and more recently, Lullies and Gulkowitsch (1924) used the same method, or a modification of it. Their results are as in Table I.

TABLE I

	mm. Hg		
	Diastolic	Systolic	
Wessely (1908)	70	—	Rabbit
Weiss (1911)	50-70	—	,,
Lullies and Gulkowitsch (1924) ...	54-70	92-108	,,

(*b*) By the Ophthalmoscopic Method.—By observing the pulsations of the retinal artery with the ophthalmoscope. This method, adopted first by Schöler (1879), was carried out with a more refined technique by v. Schultén (1884).

TABLE II

	mm. Hg		
	Diastolic	Systolic	
Schöler (1879)	mean pr. 70		Rabbit
v. Schultén (1884)	90-120	100-130	,,

(2) *The Tonometric Method.*—The pressure in the eye is raised by external pressure applied to the intact globe. As in the first case, observations have been made by two methods:

(*a*) By the Oscillatory Method.—By observing the amplitude of the excursions of the tonometer lever on increments of pressure. The variations of the excursions with different degrees of pressure were first noted by Schiötz (1905); Bailliart (1919) registered the movements of the lever photographically. These movements, however, are so small, and their variations so inconsequential, that

this method cannot lay claim to any accuracy. The inherent fallacies which it involves are in large part common to the ophthalmoscopic method of Bailliart, in which connection they will be discussed in detail. To these is to be added the greater influence of the marked diminution of the distensibility of the sclerotic with increasing tension (v. Schultén, 1884; Koster, 1895; Wessely, 1908; Greeves, 1913). The amplitude of the ocular volume pulse varies with the volume capacity of the globe, a factor which varies with the elasticity of the sclera; on each pressure increment a condition of greater tension prevails in the eye, and therefore, in the higher pressure levels a proportionately smaller excursion is registered for the same increase of pressure. On raising the pressure, therefore, the amplitude of the oscillation not only varies with the increasing pressure in the eye, but is a function also of the decreasing distensibility of the sclerotic. To obtain readings which will correctly bear comparison, the continuity of the corneoscleral envelope must be broken, and the incompressible fluids of the eye put into direct communication with the fluid in a manometer.

(*b*) By the Ophthalmoscopic Method. — The pulsatory behaviour of the retinal artery as seen ophthalmoscopically on the application of pressure to the globe was first correlated by v. Graefe (1854) with the relation of the raised intraocular pressure to the arterial pressure. Bailliart (1909) and Black (1910) ascribed to the phenomena thus observed a clinical value, and Bajardi (1910) and Rubino (1911) attempted its assessment. So also did T. Henderson (1914), Bailliart (1917 *et seq.*), and Bleidung (1924). T. Henderson (1914), applying over the upper lid a Geneva lens measure, calibrated in mm. Hg instead of dioptres, noted that the arteries at the disc showed a pressure pulse at 15 to 25 mm. Hg (as recorded by his instrument) above the intraocular pressure. This, he claimed, represented the height of the diastolic pressure in the retinal arteries over the chamber pressure. Bailliart (1917) developed a more accurate technique, and since the procedure he has elaborated has been popularized clinically by its author and by Magitot (1922 *et seq.*), and has received a considerable amount of attention during the last few years, while the principles on which it is based have been generally accepted without question, I shall consider it in some detail.

The technique adopted by Bailliart is to apply to the sclerotic a piston working against a standardized spring ("dynamometer"), with which the tension of the eye is raised. While doing so the points of commencement and cessation of pulsation of the retinal artery are noted. A Schiötz tonometer is then applied to the eye, and the pressures as registered on the tonometer at these two points are taken respectively as the diastolic and systolic pressures of the branches of the retinal artery. The results obtained thus

by different observers vary by more than 100 per cent., the majority of them being low. They are as given in Table III.

TABLE III

Author	Pressure. Interpreted as mm. Hg Diastolic	Systolic	Subject
Bailliart (1917)	25-30	50-70	Man
Velter (1920)	35	60	Man
Duverger and Barré (1920)	60	80-100	Man
Vossius (1921)	—	70	Man
Salvati (1922)	32-50	60-70	Man
Magitot (1922)	30-35	70-80	Man
Leplat (1920)	50-65	80-90	Dog
Magitot and Bailliart (1921)	45	130	Dog
Do. (1921)	45	100	Cat
Stasińska (1925)	30-35	67-70	Man
Lebensohn (1925)	30-35	65-75	Man
Vita (1925)	30-35	70-75	Man
Verway (1925)	=radial pr.	—	Man
Bauermann (1926)	54.9	—	Man

The method of Bailliart, apart from registering, as has been pointed out, not the pressure in the branches of the central artery of the retina, as its author claims, but that of the ophthalmic artery, involves several fallacies in addition to incidental sources of error depending on the construction of the dynamometer, on the amount of force used, and the rate and manner of exerting it, on the nature and condition of the eye and the arteries, and on the very limited value of the tonometer as a means of recording pressures of any kind.

This last is a very important factor: see Priestley Smith (1917), and others. In the laboratory I have found that tonometric readings of the tension differ from manometric readings of the pressure of the same eye in living cats, dogs, and rabbits, in the most disconcerting manner, sometimes by as much as 10 mm. Hg, the variation being quite inconstant, rarely above, more frequently below. The same has been established in human eyes: compare Wessely (1916), MacLean (1919), Seidel (1922,b). The variation I find is more marked with Bailliart's instrument (1923) than with either Schiötz's or MacLean's, and the discrepancies occur with the first even in rabbits, on whose eyes the tonometer was calibrated. *The use of a tonometer as an instrument of accuracy is confined to the comparison of the two eyes of the same individual at the same time, or of the same eye at different times, provided that their condition is not widely different in the first case, or does not greatly alter in the second.*

The tension is not equal to the pressure, nor is it proportional to it in its variations. Inaccuracies are therefore involved in the method, and these to a certain extent Bailliart himself admits (1924). They are, however, greatly accentuated in the conditions of high tension which the manipulations involve, and would seem to be accentuated beyond all reason by the simultaneous use of two instruments both pressing heavily upon the eye at the same time, since the force exerted by the dynamometer must considerably

deform the curvature of the globe, upon which the tonometer largely depends for any accuracy it may have.

Further, in the application of the pressure, the eye is rendered tense and hard, and is pushed back into the orbit, kinking in doing so the central artery of the retina, and compressing the ophthalmic artery, thus partially occluding the blood flow in both. This probably accounts for the lowness of the results obtained, and would seem to provide an explanation for the much higher readings which Bailliart and Magitot (1921) obtained in animals than in man. It has been already suggested that everything points to the reverse relation holding in fact. In these animals, however, with their double and anastomosing arterial supply, it is possible for the intraocular circulation to be maintained in the absence of one or the other of the ophthalmic arteries. On compression, therefore, it is probable that this error will be largely eliminated, and that the higher results obtained in these animals will indicate a closer approximation to the correct measurement than the readings obtained in the case of man.

The explanation offered, for example by Gaudissart (1921), to explain this discrepancy found by Bailliart and Magitot—namely, that the pressure in man is less than in animals because of the erect attitude and the greater length of the arteries—will not bear examination. The effect of attitude upon the blood pressure of animals which habitually adopt the erect posture is very efficiently compensated (L. Hill, 1895; Hill and Barnard, 1897), or even over-compensated (Barach, 1913) by reflex action. Further the length of the artery involves practically no fall in pressure. This last decreases proportionately with the diminution in the size of the lumen of the vessels, and the number of branchings. In point of fact the work of Bazett (1924) seems to show that normally, both in the dog and in man, the femoral pressure is higher than the brachial by an amount, according to Burdick (1925) varying from 20 to 40 mm. Hg; while L. Hill (1909) and others have demonstrated that the systolic pressure in the peripheral arteries may under certain conditions be higher than that in the aorta. The phenomenon doubtless depends among other things upon the transference of the kinetic energy of a fluid in rapid motion into stress when the flow meets with resistance (Bazett, 1924), upon the formation of "breakers" in the transmission of the pulse wave (Bramwell and A. V. Hill, 1925), and upon the "conductance" of the arterial wall (L. Hill, 1912), but such considerations serve to illustrate the inadequacy of the explanation here offered.

Further, according to Bailliart, the diastolic pressure is only some 10 to 15 mm. Hg above the intraocular pressure: this is important from the sweeping deductions some writers have drawn from this low value regarding the impossibility of the formation of the aqueous without the intervention of a special secretory mechanism. Reasoning that whenever the pressure on the external side of the artery is equal to the internal pressure, the arterial wall so placed in equilibrium begins to vibrate, he takes (if I understand him aright) the first clearly visible pulsation in the retinal artery as the diastolic pressure.

Thus Bailliart (1917, p. 265): "l'apparition du premier battement artériel, facilement constatable à l'ophtalmoscope"; (ibid., p. 265): "pour provoquer le premier battement artériel, c'est à dire, pour équilibrer la pression diastolique." The pressure is taken (ibid., p. 656): "au moment où le premier battement artériel à été noté."

But we have already seen that under the influence of the pulse pressure the arteries all over the body vibrate, and that the same

thing is always seen in the eye under sufficient magnification. This pulse increases under increments of external pressure until it reaches a maximum when the diastolic pressure is overcome. The first appearance of a pulse "which is easily made out by the ophthalmoscope" is therefore not an index of the diastolic pressure but of the magnifying power of the instrument used to observe it. The point at which the lumen of the artery is obliterated, that is, the point of maximum pulsation, is the diastolic pressure.

An illustration from Bailliart and Magitot's own measurements (1921) is illuminating. They record the diastolic-systolic pressures in the aorta of a dog as 170-195 mm. Hg, and the diastolic-systolic pressures in the retinal arteries of the same animal as 45-130 mm. Hg. Throughout the whole arterial system the tendency is for the intermittent flow in the larger arteries to be converted into a more constant flow peripherally under the influence of the elasticity of the arterial walls, an effect which in the present case is further increased by the damping influence of the elasticity of the sclerotic and the intraocular pressure. On travelling to the periphery, therefore, the pulse pressure progressively decreases, until in the smaller vessels the difference between the diastolic and systolic pressures becomes very limited; it is difficult to conceive how a pulse pressure of 25 mm. Hg in the aorta becomes converted into one of 85 mm. Hg in the eye.

A further illustration of the fallacies of the method is seen in the conclusion of Bailliart that the retinal arterial pressure ascends and descends parallel with the intraocular tension and this without variation in the general arterial pressure. Thus he states that the two retinal arteries of the same individual of whom one eye is glaucomatous differ largely in their pressure (1921); in a case of unilateral traumatic glaucoma the entrance pressure in the retinal artery rises in the injured eye only; and on the relief of a glaucoma by an iridectomy it falls coincidently with the intraocular pressure: thus: I. O. P., 50 mm. Hg, D./S. pr., 50/80 mm. Hg; subsequently, I. O. P., 10, D./S. pr., 26/40; later I. O. P., 35, D./S. pr., 39/65. While it is admitted that the venous pressure in the eye varies with the intraocular it is difficult to imagine what influence would make the pressures in the entering arteries vary up to 100 per cent. with it, and when it is remembered that the pressure actually measured is not the pressure in the arteries in the eye, but is an incorrect reading of the pressure in the ophthalmic artery, it is impossible to believe that any changes in the eye would exert an influence of this extent. The obvious deduction is that the method involves the introduction of a relative and indeterminable error, which is a function of the intraocular pressure and the state of tension of the sclerotic.

It would appear, therefore, that the technique elaborated by Bailliart is a fallacious method of estimating the lateral pressure in the ophthalmic artery; and that, whatever clinical value its results may be interpreted to have if considered in this light, merely as figures with a comparative and no absolute significance, they can give no indication whatever of the pressure in the branches of the retinal artery, nor of the relation between the blood pressures in the eye and the intraocular pressure.

Bleidung (1924) employed an air chamber connected with a manometer as a means of compression. This he fitted hermetically over the eye, and as the pressure in it was raised, the ocular circulation was observed ophthalmoscopically through a window in front of the cornea. His results average D./S. pr., 64 – 75/96 – 117 mm. Hg in man. Although herein some of the mechanical errors of Bailliart's technique are avoided, others are introduced: the air pressure in the chamber is not equal to the tension, still less to the pressure in the compressed eye. Here again many of

the same criticisms as I have already considered equally apply; and here again the pressure registered depends on the pressure in the ophthalmic artery, and is not, as Bleidung claims, that in the retinal and choroidal vessels.

The Pressure in the Anterior Ciliary Arteries has been measured by Seidel (1924,a) and Hiroishi (1924) by applying a small pressure chamber over them connected to a manometer. The pressures appeared as diastolic systolic: 35-45/65-75 mm. Hg.*

Seidel argues that since these vessels supply the ciliary body, the arterial pressure therein, that is, the arterial pressure associated with the formation of aqueous, must be less than this, and that consequently the aqueous is a secretion. He, however, compressed the arteries just at their entrance into the globe; the pressure registered was therefore that at the most proximal branching, that is, almost immediately at the limbus. Here these vessels are very minute, and have undergone several sub-branchings since leaving the ophthalmic artery; their pressure will consequently be considerably less than that of the long posterior ciliary arteries which run to the ciliary body directly from the ophthalmic artery (see Fig. 1).

That the anterior ciliary arteries are of secondary importance, and that the uveal circulation is determined largely by the posterior vessels is seen in the fact that the former are absent in some animals; when they are present, their obliteration would seem to lead to little or no deleterious effects, while ligation of the posterior arteries leads to hypotony and widespread degenerative changes extending even to structures as near the distribution of the anterior arteries as the cornea. This was demonstrated experimentally by Wagenmann (1890) and Siegrist (1900), and has been noted clinically after their operative obliteration in an optico-ciliary resection by Knapp (1874) and Löhlein (1910), or after their pathological obliteration (Coats, 1913). Further, an observation by Serr (1926) is interesting: measuring the pressures in these vessels by Seidel's technique, he finds that in glaucoma the intraocular pressure is frequently higher than the systolic pressure in them. Thus I. O. P.=61 mm. Hg, D./S. pr., ant. cil. art.=40/57 mm. Hg; I. O. P.=74, D./S. pr., ant. cil. art.=45/61, etc. In these cases the intraocular circulation was still proceeding and therefore the capillary pressure in the eye must have been still higher than the intraocular. Obviously, as Seidel's technique itself reveals, the pressure in these vessels is, or at any rate, can be, less than

* I gather from an abstract that Bauermann (*Versam. d. Nordwestdeutsch. u. d. Neidersachs. Augenärzte-Verein.*, May, 1926) has recently repeated Seidel's experiments, using the same technique. His results appear to be S./D pr.—53.7/80.1 mm. Hg. He also finds the diastolic pressure by Bailliart's method to be 54.9 mm. Hg. While it is impossible to assess adequately experimental results from an abstract, and dangerous to attempt to do so, these values appear to me to be more reasonable than those obtained by Seidel.

the general blood pressure in the vessels of the eye, and it is obviously wrong to assume that its measurement is indicative of the pressure in the latter.

Incidentally, it may be noted in passing that T. Henderson (1910) claims that the anterior ciliary arteries are veins: this is merely mentioned because it seems to have passed without being directly contradicted hitherto. This decision was arrived at after the study of "literally hundreds of sections" (ibid., p. 73), and provides an excellent example of the futility and danger of basing physiological conclusions as to function upon purely anatomical or histological data, another example of which is seen in the functions sometimes ascribed to the ciliary epithelium. To keep to the present case in the meantime, these vessels are directly given off from the muscular branches of the ophthalmic artery, they contain blood at a pressure of 75 mm. Hg, while their companion veins have a pressure of 10 to 15 mm. Hg, and the blood in them flows in a centrifugal direction from the heart to the eye, as can be seen by compressing them in man and watching the direction from which they refill, or by dividing them in animals and watching the blood flow from the proximal end. This, the physiological method of inquiry, was that which enabled Harvey to establish the fundamentals whereupon our knowledge of the circulation rests: see his MSS. lectures to the Royal College of Physicians (1616)—"WH . . . constat per ligaturem transitum sanguinis ab arteriis ad venas. . ." It is a method of inquiry a little less time-consuming and a great deal more conclusive than that adopted by Henderson.

EXPERIMENTAL TECHNIQUE

(1) *The Pressure in the Branches of the Retinal Artery.*—The pressure in the branches of the retinal artery was measured directly by the insertion into their lumen of a micro-pipette as used by Barber in his bacteriological work (1914). This is made by drawing out capillary glass tubing over a micro-burner into a needle with a rapidly tapering point, and converting the needle into a pipette with a sharp point by jamming its tip against a cover-slip until it breaks off. In this way it is possible to make pipettes with an orifice of a few micra in diameter, and relatively easy to make one of such a size as will enter the branches of the retinal artery (diam. at disc, 0.1 mm.: Hess, 1919), and at the same time allow the circulation therein to proceed unimpeded. So long as the tip of such a pipette is surrounded by air, the pressure of the internal liquid is kept in check by capillarity, but on immersing the tip in blood this error is eliminated. A theoretical error remains depending on the difference between the surface tension of the two fluids, but its magnitude is so small that it can be neglected. The pipette was controlled by a micro-manipulator designed after the type elaborated by Chambers (1922) for the injection and dissection of single cells. It was provided with adjusting devices worked in three planes, controlled by finely-threaded screws opposed by springs, so that accurate and continuous control of the pipette was obtained in every direction, and its tip could be maintained in any desired position.

The animals used in the investigation were cats. In the dog the retinal vessels are ensheathed and to a large extent obscured by neuroglia, only fine branches being visible upon the optic disc. The fundus of the rabbit is traversed by opaque nerve fibres

running in two large sheaves out from the disc. But the retina of the cat closely resembles that of man, and in it the retinal vessels are easily differentiated against a bright, glistening background, and their relation to variations in the pressure conditions are readily observed: usually three, and sometimes four, large arteries run out from the disc, each flanked by a vein.

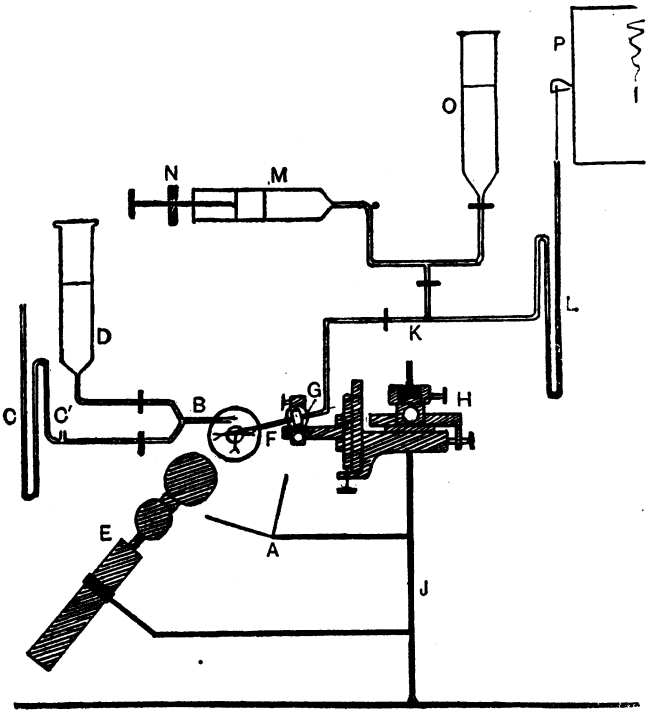

FIG. 2.
(Reproduced from the *Journal of Physiology*.)

I have elsewhere (1926,c) described the technique employed. Anaesthesia was induced with ether, and maintained by intravenous chloralose. The lower lid was reflected along with the soft tissues, the periosteum elevated, a V-shaped piece of bone removed from the lower orbital margin, and the under surface of the globe exposed. The animal's head was then securely clamped (A, Fig. 2) in a suitable position. A hollow needle (B) of large bore to prevent any valvular action, and provided with a side-arm, was inserted into the vitreous, and connected to a compensated mercury manometer (C) with an attached reservoir (C') by means of a capillary tube with an air bubble to serve as an index of equilibrium. The side-arm of the needle was connected to a second reservoir filled with normal saline (D). The normal intra-

ocular pressure was then taken as described in a previous paper (1926,a) by means of the compensated manometer; the reservoir (D) was then raised to a corresponding height and its connection with the eye opened—by this means the intraocular pressure was maintained at its normal level throughout all the subsequent manipulations.

A self-illuminating ophthalmoscope (E) was then adjusted so as to obtain a good view of the fundus by the direct method, and was clamped into position. The sclerotic was then pierced post-equatorially by a fine metal needle, and the micro-pipette (F), carrying a collar (G), was inserted immediately on the withdrawal of the needle through the hole thus made. Under the guidance of the ophthalmoscope the tip was then made to approximate closely the branch of the artery on the side of the disc opposite to that at which the pipette was inserted, and when in this position, the micro-manipulator (H) was brought into position, and the collar securely fixed by a screw. The animal's head, the manipulator, and the ophthalmoscope were all clamped to the same support (J) in order to secure rigidity of adjustment. Under observation through the ophthalmoscope the tip of the pipette was then made to enter the lumen of the artery by adjusting the screws on the manipulator. The micro-pipette was connected by tubing (K) to a mercury manometer (L), the whole system being filled with a solution of methylene blue in physiological saline. Any desired fluid pressure was made to act on the pipette tip by means of a syringe (M), whose movements were accurately controlled by a milled screw adjustment (N) on the piston, the pressure being simultaneously recorded on the manometer (L); while a constant supply of solution was maintained by re-charging the syringe from a reservoir (O) and suitably adjusting the stop-cocks.

While the pipette was being manipulated into the artery the system was kept at the normal intraocular pressure, so that none of the methylene blue escaped into the eye and obscured the field. When it was introduced, blood was seen to flow up into it from the artery. The pressure was then raised by means of the syringe until the methylene blue flowed continuously into the artery: the flow was easily seen ophthalmoscopically. At this point the pressure in the system, registered by the manometer, was higher than the blood pressure at its highest point in the cardiac cycle. The pressure was then lowered until a slight stoppage of this flow occurred periodically, when a small amount of blood tended to enter the tip of the pipette: this point marks the pressure required to equilibrate the highest pressure level reached in the artery in the cardiac cycle, that is, the crest of the systolic pressure. On lowering the pressure further the dye and the blood fluctuated in the tube of the pipette, and no continuous flow took place, until a point was reached when an almost steady flow of blood into the

pipette occurred, with a periodic stoppage at diastole, when a small spurt of the dye entered the vessel. These two pressure levels were recorded by marking their height on the kymograph (P). Several readings were taken in each experiment, the mean of which is given in Table IV, a correction factor being added to allow for the influence of a column of saline equal to the difference in level between the manometer and the eye.

TABLE IV

No. of Cat	Intraocular Pressure mm. Hg	Pr. Retinal Artery mm. Hg		
		Systolic	Diastolic	Mean, interpreted as S. + D./2
1	22	91	65	78
2	25	88	59	73.5
3	29	94	69	81.5
4	20	86	65	75.5
5	23	83	63	73
Average	24	88.5	64	76

(2) *The Pressure in the Ophthalmic Artery.*—The pressure in the ophthalmic artery was measured by a method modified from that adopted by previous workers, by making use of the eye as a natural sphygmomanometer. In the criticism of their technique, it was pointed out that any interference with the circulation behind the globe by pressure applied to the intact eye entirely vitiated the results. Further, we have seen that unless the continuity of the corneo-sclera is broken and the incompressible fluids of the eye put into free communication with a rigid fluid system, owing to the progressive damping of the pulse by the decreasing distensibility of the sclerotic with increasing tension, any method which purports to compare the variations in the amplitude of the ocular pulse becomes progressively more inaccurate as the tension rises. A manometric method of raising the pressure was therefore adopted; and the pulse was both observed by the ophthalmoscope, and recorded by the oscillatory method.

A manometer needle (A, Fig. 3), of large bore (1 mm.) to ensure free communication of the oscillations, and provided with a side-arm, was inserted through the cornea, the point of the needle being kept in the periphery of the anterior chamber to allow ophthalmoscopic examination of the fundus. From the side-arm a tube (B) was connected with a compensated mercury manometer (C) with an attached reservoir (C′), and communicated with a syringe (D) whose piston was controlled by a screw adjustment (E). The whole was filled with saline, a constant supply of which

was obtained from a reservoir (F). The straight end of the manometer led directly into a very fine capillary tube (G) lying horizontally, to which was attached a scale graduated arbitrarily. The capillary was connected by stout pressure-tubing to a glass tube (H) which served as a reservoir filled with saline, and which could be raised and lowered by a pulley. It was made as small as was convenient in order to reduce the inertia of the fluid in the capillary (G), and thus make the oscillations as large as possible. When

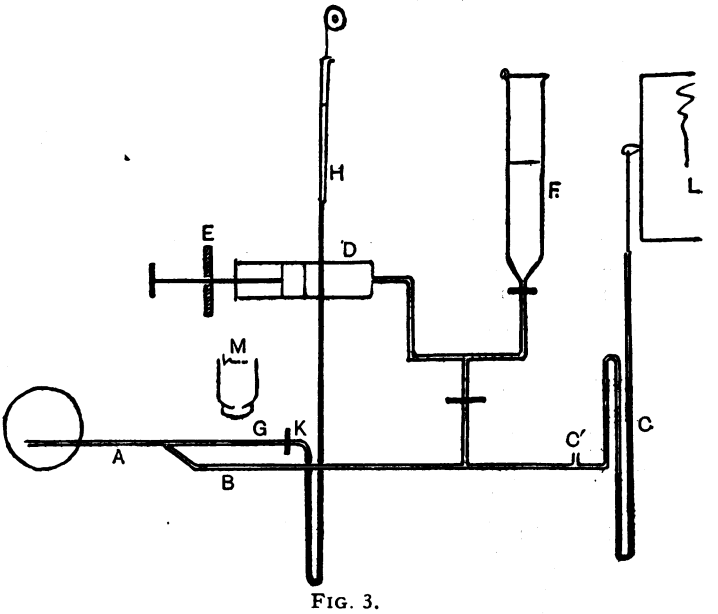

FIG. 3.

(Reproduced from the *Journal of Physiology*.)

the manometer was inserted, the reservoir (H) was shut off by a stop-cock (K); an air-bubble was then introduced into the tube (B) by a fine hypodermic syringe, and the pressure in the system adjusted by raising and lowering (C′) until the bubble was stationary, the normal intraocular pressure being thus recorded on the manometer. Communication with the reservoir (C′) was then closed, and, after the height of (H) had been adjusted to correspond with that registered on the manometer, communication between it and the eye was established by opening the stop-cock (K). The pressure in the syringe was then slightly increased, while the level in the reservoir (H) was kept unaltered, so that the air-bubble travelled slowly down the tube (B) and along the capillary (G). Having arrived here, it was kept constantly in the same place by raising or lowering (H) coincidently with any subsequent manipulations of the syringe. When the pressure in the eye was varied by means of the syringe, the behaviour of the

retinal artery was observed ophthalmoscopically, and the excursion of the air-bubble in the capillary was noted simultaneously through a microscope (M). The pressures at the points of maximum oscillation and cessation of oscillation were marked on the kymograph (L), and were subsequently measured, corrections being applied to compensate for a column of saline equal to the difference in level between the eye and the manometer.

With the ophthalmoscope it was not always easy to observe the retinal circulation sufficiently accurately to get end-points of satisfactory exactitude. If the manometer was inserted into the vitreous, this substance was found to block the point of the needle by valve action when the pressure was made to vary and this tended to complicate the readings. When the needle was immersed in the aqueous the corneal astigmatism brought about by its insertion tended to blur the view of the fundus, and this effect was further complicated by the raised pressure tending to displace and alter the dioptric properties of the lens. The end-points, moreover, were not sharp. In the previous series of experiments it was quite easy to distinguish between the methylene blue and the red blood; but here, while there was no pulse visible ophthalmoscopically at normal pressures, this was seen to appear as the pressure was raised, and to increase until pressures of from 70 to 80 mm. Hg were reached, about which region the maximum occurred. This point, the maximum, and not the first appearance of the pulse, was taken as the diastolic pressure, but its precise determination was of too subjective a nature to be of great value. Similarly at 100 to 120 mm. Hg the blood-flow in the vessels was seen to stop, and at 5 to 10 mm. Hg above this the arteries became collapsed and flattened out; but here again the end-point was indefinite, and did not lend itself to objective exactitude. Greater reliance was therefore placed upon the oscillatory method.

There follows the protocol of a typical experiment (Table V); the figures expressing the amount of oscillation refer to divisions of the scale, and are therefore arbitrary and of purely comparative significance.

Taking the point of maximum oscillation as the diastolic, and that of cessation of oscillation as the systolic pressure, the results of a series of six experiments are given in Table VI.

(3) *The Pressure in the Aorta.*—The lateral pressure in the aorta was registered by a mercury manometer connected with a cannula inserted into the lumen of the carotid artery. To obtain an idea of the pulse pressure in the carotid the technique used in the estimation of the pressure in the retinal arteries was attempted, by which a pipette was inserted into its lumen. The systolic pressure is easily obtained in this way as the point at which blood just starts to come up from the artery into the pipette; but the determination of the diastolic pressure proved

to be more difficult. The pulse pressure in this artery is too large and the oscillations too free to allow accurate readings to be made by this method; and with a large artery with opaque walls making it is impossible to see the dye inside the

TABLE V

Intraocular Pressure mm. Hg	Ophthalmoscopic Appearances	Amplitude of Oscillation (Scale Divisions)
26	Vessels normal	4 - 5
28	Venous pulsation at disc; arteries normal ...	4 - 5
40	Veins engorged; arteries pulsating slightly ...	6 - 7
60	Veins beginning to become constricted; ... arteries pulsating.	8 - 10
70	Arterial pulsation increasing	11 - 12
75	Do.	14 - 15
78	Arterial pulsation maximal	18 - 20
80	Arterial pulsation marked	14 - 17
90	Arterial pulsation less	9 - 12
110	Veins constricted; arteries filling only at systole	6 - 7
120	Blood stream segmented; arterial pulsation ceased.	0
125	Arteries barely visible	0

Mean aortic pressure - 114 mm. Hg

TABLE VI

No. of Cat	Intraocular Pressure mm. Hg.	Pressure Ophthalmic Artery mm. Hg			Mean Aortic Pressure mm. Hg
		Systolic	Diastolic	Mean, interpreted as S. + D./2	
1	26	78	120	99	108
2	29	85	129	107	118
3	26	74	106	90	96
4	23	81	110	95.5	100
5	25	73	116	94.5	108
6	20	80	109	94.5	104
Average...	25	78.5	115	97	106

vessel an end-point is not easy to decide upon. A mean aortic pressure of 110 mm. Hg corresponds to a systolic crest of 150 mm. Hg in the carotid, and the diastolic level is probably in the region of 70 mm.

Only in one case were the four measurements taken in the same animal; as a general rule the length of time occupied by the

manipulations and the consequent change in the animal's condition, rendered comparative values of little account. In this case the measurements are given in Table VII; and of these, the pressure taken in the carotid, being taken last, is probably somewhat too low.

TABLE VII

	Pressures in mm. Hg		
	Systolic	Diastolic	Mean
Pressure in carotid (aortic) (Rt. side)	—	—	104
Pressure in ophthalmic artery (Rt.)...	119	80	99.5
Pressure in retinal arteries (Lt.) ...	86	65	75.5
Intraocular pressure	—	—	20

It is seen that while the systolic crest has fallen very largely, the mean pressure in the ophthalmic artery is only about 10 mm. Hg below that measured in the carotid, that is, below the mean aortic pressure; and that in the first branching in the eye a larger fall of about 25 mm. Hg, or approximately 30 per cent. of the total pressure, has occurred. Between this and the veins a fall of 54 mm. Hg takes place. This distribution of the pressure gradient is in conformity with that encountered in other parts of the body, where the arterial pressure is maintained at a fairly constant high level throughout the larger arteries until the smaller vessels are reached. This has been recognized since the time of Poiseuille (1828). It is well illustrated in the pressure gradient in the arm: Müller and Blauel (1907) showed that the pressure in the volar digital arteries was only 8 per cent. less than that in the brachial, and Oliver (1916) found no appreciable fall in the arterial pressure down to the forearm, and only a fall of 5 to 15 mm. Hg to the forefinger. Tigerstedt (1919) places the commencement of a marked fall in pressure about the region where the diameter of the artery becomes less than approximately 2 mm. The diameter of the ophthalmic artery averages 1.5 mm. (Whitnall, 1921) to 2 mm. (Merkel and Kallius, 1910); we would therefore expect its pressure to be little less than that of the aorta. The central artery of the retina has a diameter at its commencement of 0.28 mm. (Henle, 1876) and at its termination in the eye of 0.23 mm. (Schwalbe, 1874); a fall of pressure will therefore take place here. The branches of this artery at the disc are about 0.1 mm. in diameter (Hess, 1919); and here again a further fall will occur.

III. The Venous Pressure

HISTORICAL SURVEY

In the estimation of the venous pressure of the eye five different methods have been employed. The inadequacy of the various

forms of technique which have been adopted to meet the requirements of what is admittedly a difficult problem is seen in the complete lack of agreement in the results which they have produced. Not only do their findings differ from each other, but some of the measurements obtained by the same observers using the same method involve a variation in themselves of over 100 per cent.

These experimental procedures may be divided into three categories according as they ascribe to the venous pressure a value higher than, or equal to, or less than the intraocular pressure.

1. *Methods showing a venous pressure higher than the intraocular pressure*

The direct manometric method.—Weiss (1920), experimenting on rabbits, inserted a cannula directly into the superior vortex vein as it comes into relation with the superior rectus muscle, and thus measured the pressure directly. He obtained results varying from 33 to 63 mm. Hg, and he found the venous pressure invariably considerably higher than the intraocular, the ratio between them varying from 12 :10 to 19 :10. Lullies (1923), using the same technique in dogs, came to the same conclusion. His results vary from 21 to 39 mm. Hg : in two cases the venous and the intraocular pressures were approximately equal, in the remaining four the venous pressures were considerably higher.

The blockage of a vortex vein, however, by a manometer involves the production of a hyperaemia with which the collateral venous channels are unable to cope, and the normal pressure equilibrium is thus completely disturbed. In the rabbit the vortex veins are easily ligated without further disturbance by passing a ligature subconjunctivally round the tendinous insertion of the corresponding rectus muscle. A ligature passed in this way round one of the vortex veins thus stopping the circulation in the same manner as an end-on manometer, involves a rise of tension of about 20 mm. Hg, a result which occurred with great consistency in my experiments. The inadequacy of the anastomosing channels can be easily seen in the albino in the marked hyperaemia which comes on immediately thereafter, limited to the segment of the iris drained by the vein in question. If the operation is repeated on the inferior aspect of the eye large tensions up to 70 and 80 mm. Hg are obtained, while at the same time the anterior chamber becomes shallow, the pupil dilated, the iris becomes hyperaemic, and the vessels of the fundus engorged and swollen; later the cornea becomes opaque, and further examination is impossible.

In the dog the same reaction occurs, although it is less pronounced : ligature of the superior veins induces a rise of tension from 23 to 30 mm. Hg, of the two sets, to 50 mm. Hg. In the cat a somewhat intermediate result is obtained, the tension

rising from 25 mm. Hg to 40 in the first case, and 60 in the second. It is to be noted that, correspondingly, Weiss working on the rabbit got higher figures than Lullies on the dog. The difference is due to a difference in the efficiency of the venous anastomoses in these animals. In the dog there is a direct anastomosis between the anterior ciliary venous system and the vortex veins, in the rabbit there is not; on obliterating one efferent channel in the dog, therefore, more anastomotic avenues are available for carrying on the circulation than in the rabbit, and the reactionary hyperaemia is less marked. The results of both observers thus involve a large error; Lullies's measurements are more nearly correct than those of Weiss, and of these the lowest are the nearest to the truth.

2. *Methods showing a venous pressure lower than the intraocular*

(*a*) *The compression method.*—Seidel (1923,b) and Hiroishi (1924) estimated the pressure in the episcleral veins as they are seen under the conjunctiva near the corneo-scleral junction by applying over them a pressure chamber connected with a manometer, and observing the point at which they were obliterated. Their results were:—

7 to 11 mm. Hg in the rabbit.
11 to 18 mm. Hg in the dog,
10 to 14 mm. Hg in man,

the intraocular tension in each case being about 25 mm. Hg (Schiötz). The measurement here made, however, is that of the pressure of the veins outside the eye—virtually in the orbit, and to assume that it represents the intraocular venous pressure, as Seidel does, is quite unjustified.

(*b*) *The method of injection.*—A very large number of experimenters from the time of Schwalbe (1868) and Leber (1873) have drawn conclusions as to the mechanism of the exit of the intraocular fluids and the relative levels of the pressures in the eye and in the veins from experiments involving the injection of dyes. The great diversity of the conclusions arrived at is alone sufficient for the condemnation of this method of approaching the question. Most of the earlier experimental work was complicated by the fact that the injection material used was of such nature as to bring into play such disturbing factors as the forces of diffusion and osmosis and the phagocytic activity of the endothelial cells lining the anterior chamber, but the method has lately been taken up with greater care by Seidel (1922,a) and Hiroishi (1924). These investigators made use of non-diffusible dyes made up in solutions isotonic with and hypertonic to blood, and injected them into the eye under "normal" and "sub-normal" pressures. Since the dye was seen in the episcleral veins in a very short time, its appearance

there was taken as necessarily indicating a fall in hydrostatic pressure between these two points.

The very large amount of work which has been done on the intraocular injection of dyes lends itself readily to criticism, and the physiological deductions that have been drawn from it are only to be accepted with reserve. The introduction of a needle into the eye, and the injection thereinto of any material profoundly alters the pressure equilibrium, and completely changes the circulatory conditions. No matter how sharp the needle employed, a considerable amount of force is required to transfix the tough cornea, and the immediate result of this impounding force will be to raise the intraocular pressure considerably.

The great pressure disturbance which follows the application of a small amount of external force to the globe is well seen in the effects produced by the contractions of the orbital musculature. (See the work of Parsons, 1903; Halbern, 1909; Lederer, 1912; Levinsohn, 1916; Wessely, 1916).

The pressure will be further raised temporarily once the needle is introduced into the globe by the additional volume required to accommodate the needle—a small volume in itself, but, when translated into terms of pressure, its influence becomes by no means inconsiderable when the very small distensibility of the sclerotic is borne in mind. The effect of the temporary rise of pressure conditioned by these two factors will be to compress and force out some of the fluid contents of the globe; the readiest exit will be found by the blood in the exit veins, and this will be supplemented by a considerable part of the blood in the turgescent venous reservoir of the choroid which they immediately drain. When the needle has entered, pressure equilibrium will rapidly tend to re-establish itself, and the elasticity of the sclerotic will tend to make the eye resume its normal state. At this point there will be a very much lower pressure than normally in the depleted exit veins, and fluid will tend to be drawn into them by hydrostatic forces from all available sources, including the aqueous with any dye-stuffs injected into it. Further the transfixion of the cornea with the needle is a severe trauma to a highly innervated structure, and is a procedure which causes a considerable reflex vasomotor disturbance, with a further disturbing effect on the intraocular pressure. This effect, which forms part of the reflex noci-ceptive syndrome (Bayliss, 1923), can be produced by the mere pricking of the cornea with the point of a needle (Magitot, 1923).

The complete dislocation of the circulation thus brought about by the summation of these three influences—an immediate anaemia followed by a marked hyperaemia—can be easily observed by introducing a needle into the eye of an albino rabbit, the behaviour of whose vessels can be readily seen. Any immediate results of injections, therefore, must be neglected as far as physiological conclusions regarding the normal conditions are concerned, and

after the initial disturbance has subsided, if any of the injection material at all flows into the eye, the pressure in the feeding reservoir is obviously higher than the intraocular pressure: to speak of injecting fluid under "normal" pressure into the anterior chamber is a contradiction in terms. Seidel further claims that a flow occurs from the anterior chamber into the veins with a pressure head as low as 10 mm. Hg below the normal intraocular pressure, *i.e.*, at 15 mm. Hg; this, therefore, he argues, represents the venous pressure. This result was obtained by withdrawing a small quantity of aqueous from the eye, and then observing the flow of dye-stuff from the reservoir adjusted to a height of 15 mm. Hg. But the withdrawal of a small quantity of aqueous reduces the intraocular pressure to the atmospheric level, and as will be shown later, when the chamber pressure falls, the venous pressure falls likewise; it will certainly fall below 15 mm. Hg. The flow of fluid as observed by Seidel will then necessarily take place, but it is a flow occurring in greatly reduced pressure conditions, and it can give no information at all about the pressure relationships which normally obtain.

The following experiment of my own, which has been repeated several times under conditions approximating as closely as possible those described by Seidel, is of interest. Two needles were introduced into the anterior chamber each of which was connected with a reservoir, one containing saline, and the other a solution of indigo-carmine, the former communicating with the eye by means of a tube containing an air-bubble. Both reservoirs were kept at the level of the normal intraocular pressure for some time after the initial disturbance subsequent to the insertion of the needle had subsided. That this state existed was made clear by the fact that no fluid either entered or left the eye as was indicated by there being no movement of the air-bubble. The bulb containing the coloured solution was than raised 1.0. cm., and the other lowered 0.3 cm., thus allowing a slow circulation of coloured fluid from the first, through the eye, into the second, governed by a pressure head of 1.3 cm. water (1 mm. Hg). The episcleral veins were illuminated by a powerful beam of direct light and observed through a dissecting microscope, but in no case was there any sign of this discoloration, even after some time. If, as Seidel claims, there is a pressure gradient of 10 mm. Hg between the aqueous and the veins, it would seem to follow that some of the fluid would follow this path, rather than flow down a gradient of only 1 mm. Hg.

It is evident, therefore, that it is impossible to deduce any conclusions of physiological value from the method of injection. The most cogent commentary that can be made upon it is to quote the results of three investigators who have used it. In order to get filtration, Hamburger (1914) considered he required a pressure of 30 to 40 mm. Hg, Leber (1903) a value "a little above" the intraocular pressure, and Seidel (1922) 10 mm. Hg below it. All three experimented upon living rabbits' eyes, all three used indigo-carmine, and all three took as the index of flow the discoloration of the espiscleral veins.

(*c*) *The formation of a fistula.*—With a view to determining the relative pressures of the aqueous and the venous exits, Wegefarth (1914) introduced a fine needle into an episcleral vein in the dog, pushed it down through the sclerotic into the anterior chamber, and then withdrew it and ligated the vein. Since no

blood flowed into the eye, he concluded that the chamber pressure was slightly greater than the venous pressure. The fistula was, however, made when the normal pressure relations were completely dislocated by the introduction of the needle, it was made through raw tissues, and immediately thereafter the vein was ligated, thus stopping the blood flow and providing every opportunity for the formation of clots in the eddy thus formed. Wegefarth satisfied himself of the patency of the channel so formed after 48 hours by demonstrating a flow of blood from the fistula on paracentesis, and by tracing an injection mass injected into the anterior chamber directly up the track. Neither of these tests is conclusive. On paracentesis in the normal eye, owing to the withdrawal of the supporting pressure of the aqueous a capillary engorgement is induced, so great as to lead sometimes to minute haemorrhages, and always to considerable haemorrhage if a small stimulus, as pressing on the abdomen, is added (L. Hill, 1912); if normal vessels are ruptured under these conditions, a clot recently deposited at the time of fistulization will be readily dislodged. Conversely, we would expect the same result to follow the injection of any quantity of fluid into the anterior chamber.

A similar argument has been employed at various times derived from the behaviour of the operative fistula left after an iridectomy. Fuchs (1896) pointed out that after cutting the iris tissue no cicatrization took place, and that after the operation of iridectomy, the lumen of the iridic veins appears histologically to be potentially open; Fuchs's observations were later corroborated by T. Henderson (1907) and McBurney (1914). Since such an operation can be conducted without any haemorrhage it is therefore argued that the blood pressure in the veins may be slightly less than, but is more probably equal to the intraocular pressure. The argument exposes itself to a *reductio ad absurdum*: when the wound is made the eye is open and therefore at atmospheric pressure, and for some considerable time afterwards the intraocular pressure is subnormal; the arteries as well as the veins are cut across; since there is no bleeding it follows that the arterial pressure as well as the venous must be equal to the atmospheric pressure. Clearly the explanation is to be sought, not on any lines of pressure difference, but in the fact of the spongy tissue of the iris allowing the vessel walls to retract immediately on section, a process aided by the temporary anaemia brought on by the mechanical traction upon the iris, and the vaso-constriction induced by the manipulation of a structure so highly innervated. Moreover, at a later date, when normal pressure conditions have been restored, a considerable amount of haemorrhage may occur from such a wound; an occurrence which usually takes place in infective cases, when presumably a thrombus formed at the time of operation becomes softened. This brings about the formation of a fistula under conditions of pressure more nearly approaching the normal, and an opposite result is demonstrated.

3. *Methods showing the venous pressure sometimes greater than and sometimes less than the intraocular pressure*

The dynamometric method of Bailliart.—With a technique analogous to that which he elaborated for the measurement of the pressure in the retinal arteries, Bailliart (1918,b) has made use of his dynamometer to estimate the pressure of the retinal veins relative to the intraocular pressure. On compressing the eye, the veins at the optic disc are observed simultaneously. In a certain proportion of eyes there is a spontaneous pulse in the veins just at their exit at

the optic nerve; the increase of pressure on the globe necessary to abolish this pulse is a measure of the height of the venous pressure over the intraocular. In others which show no spontaneous pulse, if the globe is compressed, a pulse is sometimes induced: again the pressure increment represented by the amount of force required to induce the pulse gives the value of the venous pressure over the intraocular. In the remainder, in whom no pulse can be elicited on pressure, the venous pressure is constantly below the intraocular. These deductions Bailliart formed on very little evidence. I shall suggest presently that I have obtained experimental proof that they are wrong. In the meantime, they lay themselves open to criticism, for many factors other than mere pressure differences enter into the mechanics of the venous pulse of the eye.

The Venous Pulse.—The venous pulse in the eye is in every way comparable with the arterial pulse, of which it is essentially a direct continuation. The essential factor in its mechanism would appear to be a centrifugal pulsatory wave from the arteries which is continued through the capillaries into the veins. This was early noted ophthalmoscopically by Wadsworth and Putman (1878), and verified by Haab (1897) and Türk (1899); with the magnification afforded by the Gullstrand ophthalmoscope, it is apparently constantly seen in normal eyes as a widening and narrowing of the retinal reflexes, which may be traced from the finest venous ramifications up to the disc, occurring post-systolically (Kümmell, 1915). In pathological conditions, as in aortic insufficiency, where the arterial pulse is exaggerated, this "progressive peripheral venous pulse" may be very apparent (Quincke, 1868; Raynaud, 1874; Helfreich, 1882; Rählmann, 1885; Osten-Sacken, 1890), and in those cases of cardiac lesions where the contractions of the right auricle and left ventricle are dissociated, Kümmell (1925) found that the venous pulse in the eye follows the ventricular rhythm, thus demonstrating its dependence on the arterial pulse, as well as its non-dependence on the activity of the right auricle as transmitted up the jugulars. The continuation of this "expansile" pulse into the venous stream depends on two factors: first, the physiological constriction of the veins at or near their exits (Holtz, 1889), and secondly, the fact of the incompressible ocular contents enclosed under tension by the elastic sclera tending to make the circulatory system react to pressure variations more after the manner of a series of rigid tubes than occurs elsewhere in the body (Türk, 1899).

At the exit of the veins from the eye a further factor comes into play. The pressure wave of the arteries at systole is conducted directly by the intraocular contents and rhythmically compresses the veins (Donders, 1855). Elsewhere in the eye one part of a vein cannot dilate to allow another to collapse owing to the increased intraocular pressure acting equally, but at the exits a

sudden expulsion of blood is possible, for here the vein is passing from a region of high pressure (the eye) to a region of low pressure (the optic nerve and orbit) (v. Graefe, 1854). A "pressure" pulse may therefore occur at the exits synchronous with arterial systole (Comberg, 1924). At the disc the occurrence of this pulse is probably aided by the greater distensibility of this part of the wall of the globe (the lamina cribrosa) under pressure (Jacobi, 1876), the influence of which factor is seen in the occurrence of a spontaneous "pressure" pulse at a distance from the venous exits in cases of posterior staphyloma (Thorner, 1902).

Since this pressure pulse was first noted by van Trigt (1853) and Coccius (1853) it has been remarked and discussed by numerous observers. It occurs at the disc in a large proportion of normal eyes—70 to 80 per cent., Lang and Barrett (1888), 58 per cent., Bailliart (1918), 46 per cent., Elliot (1921). It occurs similarly in animals: sheep and pigs (v. Graefe, 1854; Helfreich, 1882), dogs (Michel, 1881), and cats (Howe, 1885). On increasing the intraocular pressure slightly by the application of external pressure to the globe its amplitude is usually increased, or a non-spontaneous pulse may be elicited, as was first noted by v. Graefe (1854) in the retinal veins, and by Laqueur (1877) and Ulrich (1880) in the vortex veins. Sometimes a non-spontaneous pulse is not thus elicited—in the retinal vein of man in 30 per cent. of cases (Bailliart, 1918), or in 25.5 per cent. (Elliot, 1921); or in the vortex veins, both in man (Jacobi, 1876) and in animals (Becker, 1872; Helfreich, 1882). It would seem, however, unjustifiable to assume, as Bailliart and many others do, that the behaviour of this pulsation is indicative only of the pressure relations between the eye and the veins. (Compare Priestley Smith, 1917; Bauermann, 1925.)

In the first place a pulsatile flow at the exit does not necessarily imply an equality of pressure on both sides of the venous wall: if a rhythmic force is acting on the vein at the exit, a rhythmic outflow of blood will be produced even if the external force (the intraocular pressure) is less than the internal (venous) pressure, particularly if its action be enhanced by a centripetal pulse derived from the arteries. Such a phenomenon can readily be demonstrated on a simple mechanical model.

In the second place, although depending primarily on pressure differences, many other factors must be considered in the occurrence of the pulse, and in the variation of its occurrence. In the eye itself, the state of rigidity of the sclerotic is a source of variation; the less distensible this structure is, the less "give" before the arterial pulsation, and the more likelihood of the production of a venous pulse. Similarly the state of the arteries is a determining factor, depending on the degree of sclerosis of their walls; with extreme sclerosis there is often no demonstrable ocular pulse at

all (Foster Moore, 1917). Further, the arrangement of the arteries exerts an influence: the compression of a vein near the disc by an artery favours the collapse of the former distal to its crossing, the pressure here being lowered since the stream is dammed back (Priestley Smith, 1891; Marcus Gunn, 1892). A high blood pressure without accompanying sclerosis may involve a more ready and marked pulsation (Basch, 1876), so also do all the conditions which lead to an increased amplitude in the arterial pulse, as well as any factor which leads to the freer communication of this pulse through to the veins, as for example, capillary dilatation (Lieger and Forster, 1881).

Further, since pulsation implies the displacement of fluid, the amount of pulsation and its incidence will be influenced by the ease of the exit afforded the fluid—a very variable condition.

Having left the eye the retinal vein runs along the optic nerve alongside the retinal artery, often sharing a common sheath with it; the pulsation of the artery in systole will oppose the free expansion of the vein as it simultaneously seeks to accommodate the pulsating out-flow of blood (Jäger, 1854), and the proximity of the two will introduce a variable factor. Further, the retinal vein usually runs directly into the cavernous sinus: here there is a positive pulse, also at systole—the cerebral venous pulse—which will oppose the ocular venous pulse, and the ready exit of blood from the eye (Helfreich, 1882). That the influence of such back-pressure is not negligible is seen in the congestion which occurs in the frontal and ophthalmic veins on laughing, or coughing, or in conditions of cerebral pressure. A further variation depends on the mode of termination of the vein: if the retinal vein does not communicate directly with the cavernous sinus, it joins the superior, or more rarely, the inferior ophthalmic vein; in this case the opposing pulse from the cavernous sinus will be less markedly felt; and a further variation in the conduction of the pulse will depend on whether the superior ophthalmic vein is dilated into an ampulla or constricted at its junction with the cavernous sinus. Again, at systole, blood is driven both from the eye and the cerebral sinuses, and room is found for it in the anastomosing orbital veins; at the end of systole this excess of blood will tend to return to the eye, and this helps to form the ocular pulse by distending the veins near their exit at the commencement of diastole (Haab, 1897); a further variable depends on the efficiency of these anastomoses which are by no means constant. All these variables are further complicated when it is remembered that the pulse is induced by pressing the eye backwards into the orbit, thus obstructing the venous outflow by a variable amount.

Whenever the outflow from the veins is sufficiently obstructed the venous pulse will not occur, no matter what the pressure relations are. This is seen experimentally in the abolition of a pulse in any condition of venous engorgement, as by prolonged expiration (Trigt, 1853; Donders, 1855), on raising the arms above the head (Manz, 1874; Laqueur, 1877), on compressing the jugulars or the thorax (Helfreich, 1882), or on kinking the vein by rotating the eyes to the side (Graves, 1922).

The occurrence or non-occurrence of a venous pulse is therefore no indication whatever of the relative pressures in the eye and the veins; nor is the artificial pressure necessary to be applied to the eye to induce a non-spontaneous pulse any accurate measure of the normal pressure difference between the two, for since, as will be shown, the venous pressure varies with the intraocular, the latter cannot be approximated to the former without altering it

also, thus leaving the initial difference between them quite unknown. *In any technique for the measurement of the venous pressures of the eye it is absolutely essential that the normal intra-ocular pressure be undisturbed, and that the normal circulation be in no way interfered with.*

EXPERIMENTAL TECHNIQUE

In the investigation of the venous pressure dogs were employed, since they are the most convenient laboratory animal with tolerably large veins, and in them the anastomoses of the venous channels,

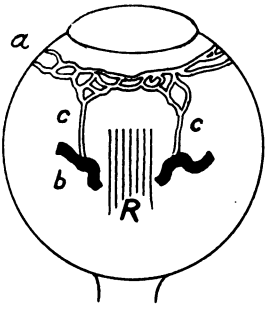

FIG. 4.—THE SCLERAL VEINS OF THE DOG.

The scleral veins of the dog from an injected specimen:
a. The circle of Hovius.
b. The vortex veins leaving the eye post-equatorially in relation to the rectus muscle R.
c.c. The anastomosing veins between the two.
a. and *c*.—drawn in open lines—lie within the scleral tissues.
b.—drawn black—come outside of the sclera.

as we have seen, are very efficient. In them the venous blood from the choroid is drained by two systems (Fig. 4): (*a*) from the anterior part of the choroid, the ciliary body, and the iris by a complicated, inter-anastomosing ring plexus in the substance of the sclerotic which takes the place of the canal of Schlemm, emptying into a second ring plexus running round the corneo-scleral junction, near the surface of the sclerotic but still in its substance, being covered by a thin layer of scleral tissue through which it is clearly visible; (*b*) from the main body and posterior part of the choroid by four or five vortex veins which leave the eye post-equatorially, and are carried away *via* the recti muscles. The anterior plexus—the circle of Hovius—is drained by two veins or groups of veins running forwards to join the orbital veins, and by intra-scleral vessels, usually one above and two below, running backwards to anastomose with the vortex veins,

lying, like the circle of Hovius itself, in the outer layers of the sclera, and visible through its outermost fibres.

(1) *The Pressure in the Intra-scleral Veins.*—The technique employed in the measurement of the pressure in the intra-scleral veins was a modification of the micro-pipette method already

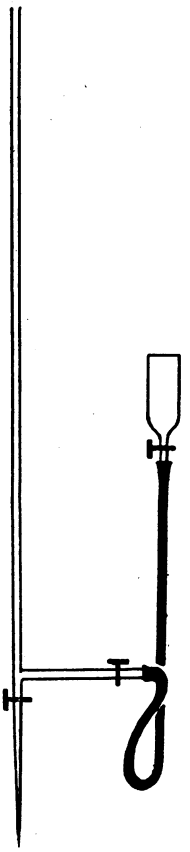

FIG. 5.—PRESSURE IN THE INTRA-SCLERAL VEINS.

described in the measurement of the arterial pressure. Anaesthesia was induced by chloroform-ether, and maintained by intra-venous chloralose. The scleral veins were reached by slitting the lids back to the orbital margin in the mid-line, and keeping them retroverted by stitches. The conjunctiva was then opened behind the limbus and dissected back until the veins were exposed. A dissecting microscope was then focussed on the exposed sclera, direct illumination being obtained from a strong light concentrated by a lens system.

A piece of glass tubing (Fig. 5) 4 mm. in diameter was drawn to a fine point at the end, the process being repeated several times so as to obtain a short and sharp tip. A side arm near the point was connected by a rubber tube to a reservoir filled with physiological citrate solution; as this was raised and lowered the water column in the tube followed suit, and a pressure was therefore exerted at the capillary tip equal to the height of this column. When the pipette is inserted into the lumen of a vein blood will flow into the tube if the venous pressure is higher than the pressure of the citrate column, if the venous pressure is lower, citrate will flow into the vein. By raising and lowering the reservoir and observing the capillary point through the microscope, a very exact end-point can be reached. The scleral veins of the circle of Hovius and the anastomosing veins just described are ideal for the method, since under the microscope they appear large enough for the point of the capillary pipette to be introduced into them without hesitation and for it to be held in their lumen for some time; meantime the investing scleral tissue, by keeping the vessel patent, ensures the ready entrance of the point, and at the same time allows perfect freedom for the continuance of the circulation, thus preventing any hyperaemia or damming up of the stream. The lateral venous pressure is thus measured, while retaining a free and undisturbed circulation and leaving the intraocular pressure conditions wholly unaffected.

Precautions were taken to record the intraocular pressure accurately with as little disturbance as possible. In the first experiment it was taken by a compensated saline manometer, using an air-bubble inserted into a horizontal capillary tube as an index of equilibrium, and no measurements were taken until 30 minutes after the pressure reaction following its introduction into the eye had settled down. In the second experiment the tensions in the two eyes were taken by a tonometer, and were found to be equal; although as we have seen this cannot be considered as accurate for absolute measurements construed as pressures, especially as applied to the eye of a dog, under the conditions of the experiment they may be accepted as comparative measurements, and the pressures in the two eyes may be taken as identical. The pressure was then taken in the second eye by the compensated manometer, and the reading transposed to the first. In the third experiment the tension was taken by the tonometer, and the corresponding manometric value determined in the other eye. The venous pressure in the first eye was then measured; the manometer was then inserted, and after equilibrium had been established at the original intraocular pressure for some time, the venous pressure was again taken, and readings similar to the first were obtained.

The results of three experiments are given in Table VIII.

TABLE VIII

No. of Dog	Intraocular pressure	Venous pressure	Difference V.P — I.O.P.
1	22 mm. Hg (300 mm. saline)	23.5 mm. Hg (320 mm. saline)	1.5 mm. Hg
2	26 mm. Hg (350 mm. saline)	28 mm. Hg (380 mm. saline)	2 mm. Hg
3	25 mm. Hg (340 mm. saline)	26 mm. Hg (350 mm. saline)	1 mm. Hg

The venous pressure in the intra-scleral veins is therefore slightly above the intraocular pressure, the pressure difference averaging 1.5 mm. Hg.

(2) *The Pressure in the Extra-scleral Veins.*—In the measurement of the extra-scleral venous pressure, the compression method of v. Recklinghausen (1906) and Hooker (1911) was used, as had been adopted by Seidel (1923,*b*). The pressure was determined by placing over the vein a glass cylinder (*a*, Fig. 6) whose base was formed by a membrane of softened cellophane, and which was placed in communication with a manometer tube (*b*) and reservoir (*c*) containing warmed saline. The intraocular pressure was taken as in the previous experiments both by manometer and tonometer. The estimation was made in the subconjunctival veins near the corneo-scleral junction, that point when the vein was first definitely obliterated being taken as standard of measurement: this precaution is necessary in order to eliminate the factor of hyperaemia, since if the pressure be maintained on the vein for any length of time, the vessel, originally emptied of blood, refills and begins to pulsate, and a vein which originally required 15 mm. Hg to obliterate it, now requires a pressure of 20 mm.

Over a series of twelve experiments in dogs results comparable to those of Seidel were obtained, although they were on the average slightly higher. The pressure in the episcleral veins soon after their exit from the eye varies from 5 to 8 mm. Hg below the intraocular pressure, the average being 7.2 mm. It may be repeated, however, that the interpretation put upon these results by this observer is not admitted—that this represents the intraocular venous pressure.

The rapid drop in magnitude is only to be expected after leaving the eye, the pressure level falling quickly to that obtaining in the veins of the orbit and head. This, even in man in the upright position, is always a small positive pressure, although above the level of the heart—a physical consequence of the flow of liquids through collapsible tubes. The venous pressure is determined by the hydrostatic difference of the point under con-

sideration and the thoracic cavity, and the frictional resistance, which is governed by the cross-section of the veins and the rate of flow. On any excess of pressure from outside the readily yielding veins collapse, and this entails a diminished sectional area and an increased frictional resistance. The negative hydrostatic pressure which would obtain in a rigid tube above the level

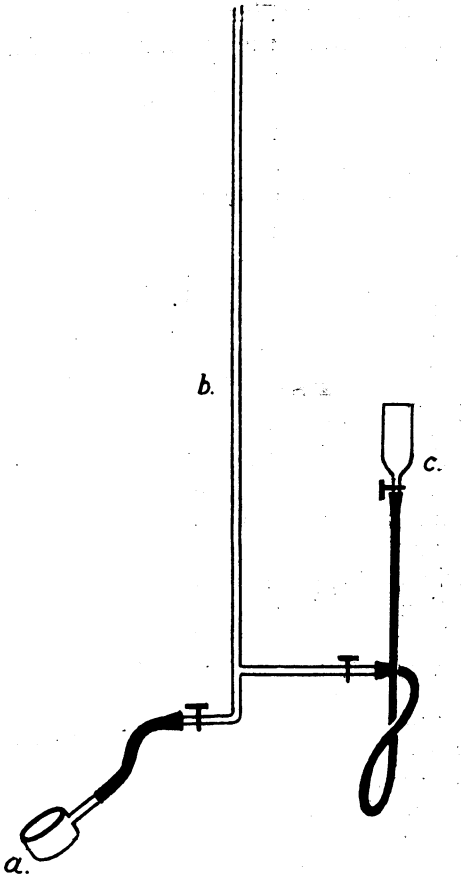

FIG. 6.—PRESSURE IN THE EXTRA-SCLERAL VEINS.

of the heart is thus automatically compensated for, and negative pressures are registered only when the veins are kept permanently open by their fascial investments as in the lower part of the neck.

(3) *The Pressure in the Intraocular Veins.*—The pressure in the intraocular veins was studied in the veins of the retina. Evidence has already been brought forward to point to the fact that the exit pressures in the uveal and retinal circulations are identical and vary together; and the vessels of the latter lend themselves to investigation more readily, since they are seen by

the ophthalmoscope at a sufficient magnification to permit easy observation, and they are less under the influence of a delicate vasomotor mechanism which reacts to any intraocular manipulation by an immediate response. On pricking the iris large pressure variations invariably follow which would completely vitiate any result obtained by experimental procedures involving this structure.

The relation of the intraocular pressure to the venous pressure in the eye was determined by the establishment of a fistula between a vein on the optic disc and the orbital contents. The outer canthus was slit up, and a fine needle with a knife point inserted through the sclerotic behind the ciliary body and lens into the vitreous. Guided by the ophthalmoscope, the point of the needle was approximated to a vein, and there carefully supported for some time until the pressure reaction due to its introduction had subsided. That such a reaction is very small provided the needle employed is fine enough and sharp enough was demonstrated by repeated controls carried out with a manometer inserted into the eye. Still under direct vision through the ophthalmoscope, the slightest movement of the needle now suffices to pierce the vein with the point. When this is done a fine jet of blood flows out slowly, and forms, initially, a cone in the vitreous in the track of the needle. This happened invariably in dogs, in cats, and in rabbits over the series of 25 animals experimented upon. Since the blood flows out of the vein, the venous pressure must be higher than the intraocular, and any deviation from the normal pressure conditions occasioned by the slight movement of the needle will act in the direction of raising the intraocular pressure, and so confirm rather than vitiate the result.

This relation was further confirmed by the introduction of a micro-pipette into a vein at the disc by a technique similar to that already described in the measurement of the pressure in the retinal arteries. The intraocular pressure was kept at the normal level by a manometer (C, Fig. 7) and reservoir (D) containing saline as in the previous experiments, and the pipette was introduced into the eye while connected with a second reservoir (M) consisting of an upright tube of 4 mm. bore adjusted to the same level as (D) by a levelling bulb (N). When the reaction subsequent to the insertion of the two instruments had died down, the pipette was made to enter the vein with the aid of the micro-manipulator. Again, in every case, blood flowed from the vein up the pipette, and the saline column in the reservoir in connection with it rose by an amount varying from 2 to 3 cm. It is not claimed that this measurement gives an accurate determination of the normal difference between the two levels, since the intraocular pressure, and with it the venous pressure, must have been upset to some degree by the necessary manipulations. In the case of the entrance

arteries this small error will be of no consequence, but it cannot be safely neglected in measurements so nearly alike and so intimately varying as the chamber pressure and the venous pressure. The only deduction that can justifiably be made is that the venous pressure is higher than the chamber pressure, and that the difference between them is not large—a deduction borne out by the fact of the occurrence of a spontaneous venous pulse, and

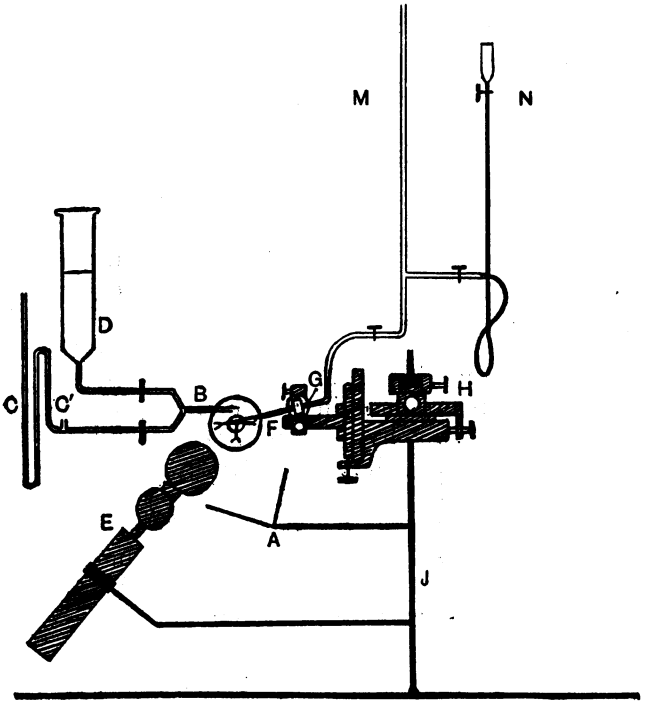

FIG. 7.—THE INTRAOCULAR VENOUS PRESSURE.

the constriction that occurs at the veins at the disc on the application of a small amount of pressure to the lids (T. Henderson, 1914).

As mentioned above, Bailliart (1918,b), and Magitot (1922) consider that those cases wherein no venous pulsation can be elicited on pressure should be interpreted as having a venous pressure lower than the intraocular. We have seen that such a phenomenon occurs also in animals; it is particularly well seen in some cases in cats. In these the establishment of a fistula and the insertion of a micro-pipette has given, in my experiments, the invariable demonstration of a higher pressure in the vein than in the eye. When the delicate nature of the venous walls is kept in mind, it is difficult to imagine how they could resist any such external pressure as Bailliart postulates without collapsing.

Even the large superficial veins of the arm, protected as they are by the skin, collapse under an external pressure of about 0.8 mm. Hg (Moritz and Tabora, 1910). To maintain the circulation, therefore, it would seem to be necessary that the venous pressure should be always higher than the intraocular; and my experimental findings point to the fact that this is constantly so. In any case they definitely show that Bailliart's contention is unjustified, and that the failure of increased pressure on the globe to induce the appearance of a non-spontaneous pulse cannot be accepted as evidence that the venous pressure is lower than the intraocular.

THE VARIATION OF THE VENOUS PRESSURE WITH THE INTRAOCULAR PRESSURE

It has been established in a general way that the intraocular pressure varies very intimately with the venous pressure.

On increasing the venous pressure by tying the vortex veins as they issue from the eye very large intraocular pressures up to 80 and 90 mm. Hg are registered, the amount varying with the species of animal and the efficiency of the anterior ciliary veins and their anastomotic connections with the vortices. Ligature of a single vein produces the same effect to a lesser degree, as we have seen in discussing the experiments of Weiss and Lullies; ligature of all the veins produces in a short time a shallow anterior chamber, a dilated pupil, a hyperaemic iris, and engorgement of all the vessels throughout the eye—later the cornea becomes opaque, the tension remains stony hard, and further ophthalmoscopic examination becomes impossible. After some time, when collateral venous channels have opened up, the glaucomatous state tends to subside. These effects find ample confirmation in the observations of several workers, the majority of whom were investigating the effect of the venous circulation in glaucoma: Adamük (1867), Leber (1873), Weber (1877), Schöler (1879), Alt (1884), v. Schultén (1884), Ulrich (1889), Koster (1895), van Genus (1899), Bartels (1905), and Magitot (1923). A similar rise occurs on ligating the veins at the back of the orbit (Hiroishi, 1924), or on obstructing them with irritant injections (Birch Hirschfeld, 1909). The somewhat equivocal results that have been obtained on ligating the jugular veins are readily understood when the very free anastomoses are taken into consideration; thus while Memorski (1865) and E. E. Henderson and Starling (1904) got no constant changes, Adamük (1867), Graser (1883), v. Schultén (1884), and Parsons (1903) produced only slight rises in pressure. When, however, the channels are all simultaneously impeded, the result is more marked, as on passing a ligature round the neck (Bonnefon, 1922), or on compressing the thorax (Mazzei, 1920) or the abdomen (Comberg and Stoewer, 1925), or on obstructing the vena cava (L. Hill, 1912).

A large amount of clinical evidence, moreover, is available on the production of glaucoma by the obstruction of the efferent veins by traumatic, thrombotic, or inflammatory processes. The literature of such cases with their pathological findings include these: Roser (1859), Birnbacher and Czermak (1886), Wagenmann (1892), Stirling (1893), Koster (1895), Römer (1895), Zirm (1895), Bartels (1905), Stähli (1911), Christel (1912), Ischreyt (1912), Rönne (1913), Magitot (1917), Thomsen (1918), Haussen (1918), Seefelder (1924), Guist (1925), Magitot and Bailliart (1925), and Larsen (1926).

Conversely, on decreasing the local venous pressure by venesection the intraocular pressure falls (Schneller, 1857; Bornacini, 1909; Leplat, 1923): the fall is not constant (Pergens, 1899), and its regular occurrence appears to be complicated by vasomotor reflexes. Clinically, phlebotomy has been used in one form or other (by scarification, leeches, etc.) since the time of the Hippocratic writings (*περὶ τόπων τῶν κατ' ἀνθρωπον*, § 13; *περὶ ἕλκων*, § 26,27). Further, in a previous paper (1926,a), I have shown that the intraocular pressure tends to follow the variations in both directions of the systemic venous pressure, sometimes even more closely than it does the arterial pressure.

Similarly when the intraocular pressure is raised, the venous pressure is found to rise coincidently with it. If a mercury manometer with a reservoir is connected with the eye of a cat, and the behaviour of the retinal vessels watched as the pressure is raised, these vessels show no compression, apart from an occasional slight disturbance just at their exit, until a pressure of from 60 to 70 mm. Hg is reached; when the intraocular pressure is raised above this level, ophthalmoscopic examination reveals that the veins, which in the lower stages of pressure increment had been engorged, have now been reduced to mere streaks, the contained blood taking up a granular appearance as the circulation ceases. This experiment finds its analogue in the procedure adopted by L. Hill (1912), who, on opening one of the vortex veins and allowing it to bleed, was able to stop the haemorrhage only when he had raised the intraocular pressure almost to the level of the carotid pressure; and its clinical demonstration is seen in the engorged ciliary veins often so apparent subconjunctivally in cases of glaucoma.

In order to test the relative values more exactly, the venous pressure was estimated in the intra-scleral veins by the capillary manometer already described, while the intraocular pressure was raised by means of a mercury manometer inserted into the eye. On raising the intraocular pressure to a considerable height, the pressure in the exit veins was observed to rise coincidently, but to remain at a level slightly below that obtaining in the eye: thus with an intraocular pressure of 40 mm. Hg the venous exit pressure was found to be 39 mm. At the same time ophthalmoscopic examination showed that the intraocular retinal veins were engorged. A needle was then inserted into the posterior part of the eye as in the previous series of experiments, and the same

result was obtained as formerly on the establishment of a fistula —a haemorrhage into the eye, demonstrating that the intraocular venous pressure was still higher than the chamber pressure. A further reading of the intra-scleral venous pressure was then made, the results of which confirmed the previous findings—that it was slightly less than the artificial pressure maintained in the eye. This would seem to have an important bearing on the physiology of the intraocular pressure, and the mechanism called into action to conserve normality in its variations.

The experimental results of this investigation suggest that the venous pressure in the eye is always higher than the intraocular pressure, and that, while there is a fall of pressure in the veins as they pass through the thickness of the scleral coat, normally the pressure at the exit is still in excess of that in the eye. The eye thus falls into line with the rest of the body, wherein the venous pressure is slightly higher than the tissue pressure, the tissue pressure in this case being represented by the intraocular pressure. Owing to the delicate and pliable nature of the venous walls it is unlikely that the pressure in these vessels can normally be greatly in excess of the chamber pressure, a consideration borne out by the readiness with which venous engorgement is noted pathologically. Conversely, and for the same reason, it is difficult to understand how they could withstand any degree of pressure from outside without collapsing: it is an essential postulate for the maintenance of a continued circulation that the arterial pressure should be higher than the capillary, the capillary higher than the venous, and the venous higher than the intraocular pressure. When the chamber pressure is raised the circulatory system is compressed. That part with the lowest lateral pressure will give way first, that is, the veins at their point of exit will tend to become obliterated. As soon as this occurs the blood flow will be checked, the *vis a tergo* from the arteries will pile up pressure, the constriction will be forced open, and the circulation will proceed at a higher level, the venous pressure rising with the intraocular. This process will repeat itself in a cumulative manner until the available force from the arteries is exhausted, that is, until the pressure in the ophthalmic artery has been reached, at which point the entire circulation will cease and the vessels will be obliterated.

The venous pressure in any part of the body behaves in an exactly similar manner in similar circumstances. Thus experimentally, by compressing the arm by a sphygmomanometer the pressure of the veins in the limb can be raised to 120 mm. Hg (Frank, 1913); and the same phenomenon is met with clinically in states of venous obstruction from any cause—mediastinal tumour (Eyster and Middleton, 1924), a gravid uterus (Runge, 1924), phlebo-scleritis (Hooker and Eyster, 1908), etc.

While this is going on the pressure in the exit veins just inside the eye has approximated the chamber pressure, the pressure

decrement in the vessels traversing the sclera still obtains since their lumen is always kept open by the investing scleral tissue, and therefore the pressure in the exit veins just outside the sclera will now fall below the intraocular pressure. Thus while it is not possible normally, under conditions of raised intraocular pressure, a hydrostatic outflow may be set up, draining off aqueous, acting as a safety valve, and tending to restore the normal pressure conditions of the eye. Such a flow may in some degree be aided through the activity of the ciliary muscle by the pump-action of the scleral spur of A. Thomson (1910).

IV. The Capillary Pressure

Inasmuch as all vital processes take place through the capillary walls the determination of the capillary pressure is of the greatest importance and interest from the physiological point of view: the arteries and veins are merely conducting tubes and the heart a pump, the capillaries are the essential part of the circulation. No one has yet devised a method to measure its value in the eye. Its determination would seem to present many difficulties, since any intraocular manipulation or external pressure applied to the eye at once affects the intraocular pressure, through it the venous pressure, and therefore the capillary pressure, the three tending to rise coincidently but not in parallel. The influence of the venous pressure generally on the capillary pressure (although the reaction is not invariable, Boas and Doonieff, 1924) is usually very readily felt (Bayliss and Starling, 1894; Danzer and Hooker, 1920; Liebesny, 1923); further, on raising the intraocular pressure any effect thereby produced on the capillary circulation is referable not to the capillaries themselves but to the feeding vessels, that is, ultimately to the ophthalmic artery. For this reason all the estimates that have hitherto been made are valueless.

Two such methods have been employed:

(1) *By the stoppage of the formation of aqueous by raising the intraocular pressure.*—This method postulates that the intraocular fluids are a simple transudate of the blood plasma, and that therefore, when the chamber pressure is raised to that in the capillaries, the transudation of fluid will stop. Niesnamoff (1896) thus determined that at 50 mm. Hg the outflow of fluid into the eye from a reservoir was the same for the dead and the living eye; this point he presumed to be the capillary pressure, since in neither case was there any formation of fluid. This observer, however, as far as one can gather, based his conclusions on a single experiment; Grönholm (1900) by the same method found that the outflow was not the same in the two cases at this pressure; and E. E. Henderson and Starling (1904), repeating the experiments, failed to find any agreement. The argument is full of fallacies. We have already

seen that a rise of intraocular pressure necessitates a coincident rise in capillary pressure; in addition, by stretching the globe in the act of raising the pressure, fresh channels of outflow are opened out at the higher pressure levels; and in any circumstances the conditions of the dead and the living eye are in no way comparable.

(2) *By raising the intraocular pressure and observing the capillary flow.*—Bleidung (1924) estimated the external pressure which had to be applied to the globe in order to induce a capillary pulse at the disc, and claimed that this represented the pressure in the small arterioles feeding the capillaries (77 to 91 mm. Hg in man). Deiter (1925) has claimed that the pressure at which the cessation of the capillary flow occurs as it is seen entoptoscopically at the macular region represents the capillary pressure (51 mm. Hg in man). Any such method as these, however, measures neither the pressure in the capillaries nor in the arterioles supplying them, but inasmuch as the whole eye is compressed and the entire circulation in it affected, it measures, with the large and unknown error already pointed out, a quantity depending on the pressure in the ophthalmic artery. Consequently, each measuring the same thing with the same error, Deiter's figures for the capillary pressure are the same as the value given by Bailliart (1923,b, p.50) for the systolic pressure in the retinal artery.

As a result of their investigations both observers have come to the most far-reaching conclusions. Thus Deiter (1925), who finds that the capillary pressure is always in the region of 33 mm. Hg above the chamber pressure, concludes, since this figure represents the partial osmotic pressure of the blood colloids, that the intraocular pressure is determined in all conditions—normally, in glaucoma, and after trephining—by the simple formula: I. O. P. *equals* capillary pressure *minus* osmotic pressure of blood proteins. Upon this foundation he has built up a theory of glaucoma. Since, however, the first two of these measurements are mutually interdependent as cause and effect, by thus manipulating variables without reference to any constant, and dealing in circles with conclusions without a premise, it would seem that with enough ingenuity he could have come to any conclusion he cared.

A direct method of measuring thus being denied us—in the meantime, at any rate—we must resort to an indirect method of estimation, and, by considering first what is known about the pressure conditions in the capillary circulation generally, and seeing how far the results thus obtained can be applied to the eye, base our estimation upon the arterial and venous pressures therein which are now definitely known quantities.

The Capillary Circulation.—The very many attempts that have been made to estimate the capillary pressure in the body have produced an assortment of results so discordant and bewildering as to represent this value as anything from 1 to 70 mm. Hg. Most of the methods which have been employed lend themselves to a considerable amount of criticism, and the various criteria which have been adopted by different observers as a standard of measurement have led to much confusion in the interpretation of the results they have obtained.

The later measurements taken within the last few years are these: the majority of them were obtained by methods involving the compression of the skin and the observation of the blood flow under the compression: Landerer (1913), 17-25 mm. Hg; Goldmann (1914), 5-8 mm. Hg; Krauss (1914), 6-9; Briscoe (1918), 20; Danzer and Hooker (1920), 22; Leonard Hill (1920), 2-4; Basler (1921), 7; Boas and Frant (1922), 18-22; and up to 60 pathologically; Kylin (1923), 8-14, and up to 50 pathologically; Liebesny (1923), 15-40; White (1924), 3-14 mm. Hg. Carrier and Rehberg (1923), using the less equivocal method of measuring directly by a pipette, found in the human skin, 3-6 mm. Hg; and Landis (1926), by the same method in the frog's mesentery, 10 mm. Hg.

The tendency of the later results is to put the pressure at a comparatively low value. The great majority of them, however, have been made upon the skin and none have been taken in the internal organs of the higher animals. There seems little justification for applying the figures thus obtained to the general circulation, and none at all for applying them to the specialized pressure conditions in the eye. Moreover, it is a characteristic feature of the vascular supply of the skin that in it there are few arterial capillaries (Spalteholtz, 1893; Krogh, 1922), the exchange of substances taking place largely through the venules which are extremely thin, a condition no doubt associated with the fact that the metabolic level of this tissue is low and not very variable. These values may then be taken to refer mainly to the venous-capillary pressure (Liebesny, 1923). The only deduction one can justifiably draw from them is the fact of the great variability of the capillary pressure—a variation which persists even into the veins: thus Bergolmez (1911) found that the pressure in the venules of 0.2 mm. diameter in the rabbit's ear, as measured by a needle cannula, varied from 4 to 23 mm. Hg.

A more correct picture of the conditions in the capillary circulation can be visualized from the recent work of Krogh (1922) and his co-workers. In the light of their researches the capillaries are not to be thought of as a static part of the circulation, and the blood pressure in them as a constant and measurable quantity, but they are to be considered as the most active, purposive, and dynamic part of the vascular system, with a pressure continually varying from individual to individual, from organ to organ, and in both of these, from time to time. The circulation is not regulated by the heart alone, but the capillaries still retain much of their primitive nature when the entire circulatory mechanism depended on the contraction of the peripheral vessels.

The capillary pressure is not therefore wholly dependent upon the arteriolar pressure (Oinuma, 1924) or regulated entirely by the arteriomotor nerves, and large variations in the venous pressure

may have no influence upon it (Boas and Doonieff, 1923). The tone of the capillaries is an independent property, controlled in part by a special capillariomotor nervous mechanism, and in part by complex physico-chemical influences, some of them locally determined, some of them hormonal, acting on the contractile elements of the capillary wall over which they exercise a balanced control. A similar though lesser control persists apparently through to the veins, whose tonus is regulated in part by an independent venomotor nervous mechanism (Hooker, 1918; Bancroft, 1898; etc.), and in part directly by chemical agencies (Y. Henderson, 1916; Ebbecke, 1923). This active tonicity suggests that the peripheral resistance is not confined to the arteriolar region, but that no inconsiderable part of it is located in the capillary region of the circulation, and that therefore a large part of the fall of pressure in the arterial system occurs normally here, and that on occasion it may to some degree be transferred through to the venules. To the influence of this variable tonicity in deciding the point of pressure fall must be added the factor of the resistance offered by the passage through the capillaries of the blood corpuscles (Krogh, 1922), which appear to be normally much deformed as they traverse these vessels; and also the factor of the tortuosity of the path with its increasing multiplication of passages in this region, since the average diameter-ratio of the stem to the branch entails the consequence that the smaller subdivisions offer a higher resistance than the larger (Priestley Smith, 1917), a process which goes on progressively to the mid-capillary region.

These deductions have received striking confirmation in the recent work of Dale and Richards (1918) and Burn and Dale (1926), who, by dissociating the arteriomotor and capillariomotor effects, have brought forward a large amount of evidence to show that the peripheral resistance is not limited to the arterioles, and that the common assumption is unwarranted that their exists an abrupt fall in pressure in this part of the circulation. Such a conception—that the fall of pressure is evenly distributed without any sharp line of demarcation—would seem to have been recently substantiated by the work of Landis (1926), who, using a micro-injection technique, such as is followed in this paper, found that in the peripheral vessels of the frog's mesentery the fall of pressure does not cease abruptly in the arterioles, but while a large part of it occurs in the capillary bed, it continues evenly through to the venous capillaries before flattening.

Defining capillaries, therefore, as simple endothelial tubes through which fluid interchange can take place, there is probably no sharp dividing line—either anatomically or physiologically, in structure or in function—between them and the arterioles on the one hand and the venules on the other. With their continuously

changing conditions and wide range of variation, it seems unreasonable to speak of a capillary pressure at all, and if we do it must be in elastic terms as comprising a large part of the pressure gradient from the arteries to the veins.

Such a conception of a dynamically active capillary bed and an ever changing capillary pressure shows the inadequacy of estimating the capillary pressure by any schema based on calculations of the sectional area, or on the rate of flow, or any hard and fast mechanical considerations derived from the study of the flow of fluids through tubes, as has been elaborated by Priestley Smith (1918: capillary pressure, 40 mm. Hg in the eye). The assumption that such mechanical considerations, or that the calculations on which they are based hold for the same capillary for any length of time or for any two capillaries at the same time, can lead to no quantitatively correct results (see Dale, 1926).

It also effectively answers the argument stressed by T. Henderson (1910), Flack (1913), and others in discussing the "secretion" of the aqueous, that the capillary walls, being films of fluid protoplasm, would be unable to bear any such strain as a filtration theory demanded. Apart from the support afforded them by the Rouget cells, such an argument takes no cognizance of a world of physical forces. In the human skin where the normal pressure is undoubtedly relatively low, Lewis (1924) estimated their contractile power as being capable of expelling fluid against a pressure of 50 to 60 mm. Hg, and when contracted, of resisting the entry of fluid up to 90 to 100 mm. Hg.

The Capillary Circulation in the Eye.—It has been said on so many occasions that the capillary pressure of the eye is governed by the diastolic arterial pressure and must be less than it, between it and the intraocular pressure, that it may be worth while pointing out that the statement is quite wrong.

Thus during this year, Seidel (1926, p. 547), who finds the diastolic pressure in the anterior ciliary arteries to be 30 mm. Hg, insists that the capillary pressure in the eye lies between this and 25 mm. Hg—the intraocular pressure. And Serr (1926, p. 699): ". . . . der diastolische Druck 30-45 mm. Hg beträgt. Der intraokulare Capillardruck muss aber unbedingt niedriger sein als dieser extrabulbär gemessene Wert. . . ." Hence, they argue, the aqueous is a secretion.

On the contrary it may almost be said that the capillary pressure has nothing to do with the diastolic arterial pressure; certainly it need not be less than it. If we neglect the ocular capillary pulse, provided there were no friction, the capillary pressure would equal the arterial mean pressure, and its actual value is determined by the amount deducted from this mean pressure by friction. The capillary pressure throughout the body is governed by the aortic pressure; at the beginning of the aorta the diastolic pressure is nil, and in a case of regurgitation it is a negative quantity; yet the capillary pressure exists as a positive quantity. In the eye

we have already seen that it is no uncommon thing for the arteries to be completely obliterated at diastole when a spontaneous "pressure pulse" occurs, that is, the diastolic pressure is less than the intraocular; in the same eye the veins are full, their pressure must therefore be higher than the intraocular pressure; the circulation goes on indefinitely, and therefore the capillary pressure must be still higher than this, that is, than the diastolic pressure of the entering arteries. We will admit that in normal circumstances the capillary pressure is less than the diastolic, but it need not be, it often is not, and under no circumstances should its value be deduced from it.

We have seen that the mean pressure of the arteries entering the eye is about 75 mm. Hg, and that the pressure in the veins leaving the eye is from 1 to 2 mm. Hg above the intraocular pressure. There is therefore a fall of from 50 to 55 mm. Hg in the vascular system. We have seen further that a large part of this fall occurs in the capillaries. But in addition to the fact that the "tissue pressure" in the eye is 20 to 25 mm. Hg, instead of 1 to 2 mm. as obtains throughout the body generally, there are several indications that the capillary pressure in this organ will be higher proportionately than in any other organ in a state of rest. The ciliary arteries seem to be anatomically peculiar in that they break off almost at once into a rich net-work of wide capillaries (Fusita, 1919), which appear to be capable of such extreme distension in places as to allow of the passage of ten corpuscles at a time; in these it will be possible for the energy component represented by the lateral pressure to rise to a very considerable height, theoretically higher than that in the arterioles. Again the veins of the eye are physiologically constricted at their exits, and the whole circulation is confined under considerable tension in a feebly distensible and elastic case under whose influence the whole system preserves a pulsatile flow. This will tend to throw the point of the fall of pressure further towards the veins, and make the vascular system approximate in its behaviour to a series of rigid tubes.

It is very probable, therefore, that a variation of from 30 to 35 mm. Hg occurs in the capillary region of the eye, and that, while definite figures are apt to give a wrong idea of what is an indefinite and ever changing quantity, at the arterial end of the capillary bed the pressure may be in the region of 50 to 55 mm. Hg, and that in the venous capillaries it may be a few mm. Hg above the intraocular pressure.

If it be permitted to transpose the measurements obtained in the dog and apply them comparatively to the cat, we therefore arrive at the following scheme of the blood pressures in the eye (Table IX).

TABLE IX

		Pressures mm. Hg		
		Diastolic	Systolic	Mean
Cat... ...	Pressure measured in carotid ...	70	150	108
	Pressure in ophthalmic artery...	78	115	97
	Pressure in retinal arteries, *i.e.*, first arterial branchings in eye	64	88	76
	Estimated capillary pressure ...	—	—	55–28
Dog ...	Pressure of venous exits ...	—	—	21.5
Cat and Dog	Intraocular pressure	—	—	20
Dog ...	Pressure in episcleral veins ...	—	—	12.8
	(Facial vein: dog — Burton-Opitz, 1909)	—	—	5.1
	(Jugular ,, ,, ,, ,,)	—	—	0.5
	Sup. vena cava ,, ,, ,,)	—	—	−1.4 – −2.9

V. The Physiological Significance of the Vascular Pressures

From the physiological standpoint the main interest in these vascular pressures is their relation to the formation and absorption of the aqueous humour. The formation of the aqueous is associated mainly with the vessels of the ciliary body and iris, and from a theoretical point of view it would have been preferable to have obtained measurements of the arterial pressures here. In the cat, as has been noted, the circulus arteriosus iridis major, which supplies the ciliary processes and iris directly, instead of lying inaccessibly at the base of the iris as in man, lies in the iris itself, and with sufficient magnification is readily seen particularly on the nasal side. To introduce a pipette into this vessel, however, was found to be difficult or impossible on account of the mobility of the supporting structures, while the immediate and high rise in the intraocular pressure which invariably followed penetration of the iris—due presumably to nociceptive vasomotor reflexes from this richly innervated structure—seemed to render any such attempt useless. This vessel, however, is directly formed from the long posterior ciliary arteries. Since, other things being equal, arterial pressure falls proportionately to the number of branchings and the size of the lumen of the vessels, and since the long posterior ciliary arteries and the central artery of the retina are both direct branches of the ophthalmic, and are of the same order of size, it would seem probable that the pressure in this arterial circle would approximate that in the branches of the central artery of the retina. Moreover, the close relationship between the pressures in the two circulations—uveal and retinal—under physiological variations has already been pointed out. It is probable, therefore, that we can assume with a fair degree of certainty that the pressures measured in the branches of the central

artery of the retina are not far removed from those in the ciliary body.

It may thus be assumed that in the ciliary body the arterial mean pressure is about 75 mm. Hg, and that the venous pressure is about 21 mm. Hg when the intraocular pressure is about 20 mm. Hg. There is thus a pressure fall in the ciliary vascular system of from 50 to 55 mm. Hg, and a capillary pressure may be suggested varying from about 22 to 55 mm. Hg.

When it is remembered that the intraocular pressure is 20 to 25 mm. Hg, it would seem that the arterial and venous pressures in the eye bear a relation to the chamber pressure similar to that which the vascular pressures do to the tissue pressures throughout the body. If the aqueous is formed by a simple physical mechanism as are the tissue fluids generally (Starling, 1896; Bayliss, 1920) without the intervention of a special "secretory" force exercised by the cells of the ciliary endothelium, the hydrostatic pressure in the capillaries must be capable of exceeding the intraocular pressure by the difference between the osmotic pressures of the aqueous and the plasma. I have elsewhere shown (1926,d) that in their estimates of the osmotic pressure of the aqueous, previous observers were largely in error, and that it is less than that of the plasma by an amount depending on the electrostatic forces conditioned by the stresses called into being by the almost complete impermeability of the capillaries of the eye to colloidal micelles; the difference between the osmotic pressures of the two therefore varies with the protein content of the blood—in the rabbit it is about 20 mm. Hg, in man about 30 mm. Hg. The tissue fluids generally contain about one-half the quantity of blood proteins, and therefore (in man) a difference in hydrostatic pressure between aqueous and capillary of about 30 mm. Hg must exist in the eye, instead of about 15 mm. Hg between tissue fluid and capillary elsewhere in the body. We have already seen that the specialized conditions in the eye provide every reason to believe that this is the case. The capillaries in various parts of the body vary very considerably in their degree of permeability to colloids (Krogh, 1922, etc.), and it may be suggested that the great impermeability of those in the eye is a biological adaptation to keep the intraocular fluids optically homogeneous, and that the specialized circulatory conditions which obtain here are a further adaptation to compensate for the higher pressure differences which are thus rendered necessary.

E. E. Henderson and Starling (1906), in an investigation to test whether it was possible that the capillary pressure was capable of forming the aqueous by a process of filtration, compared the aortic and intraocular pressures over a large series of animals. The average difference was found to be 84.4 mm. Hg, and the conclusion arrived at by them was that the pressure fall rendered

such a view possible. The present investigation takes the matter a step further and amply corroborates their findings. There is every reason to believe that the capillary pressure in the eye rises to 50 or 55 mm. Hg. Around this level it will be constantly fluctuating, at one moment above it, at another below it; and in any one capillary the balancing hydrostatic and osmotic forces will render a flow of fluid possible outwards at one moment, inwards at another throughout its length, or in both directions contemporaneously in different parts of its length, the amount of fluid interchange being a function of the pressure head as the intensity factor and the length of the path over which flow takes place as the capacity factor. While such fluid interchange in both directions is possible throughout the eye as a whole the greater amount of dialysation outwards will take place, that is, the larger part of the aqueous will be formed, from the vessels in the ciliary body and iris. This we would expect from the direct vascular supply of this region from the uninterrupted long posterior ciliary arteries. The results of innumerable injection experiments amply confirm this deduction, as also do the experiments of Wagenmann (1890), who found that on ligating the short posterior ciliary arteries or the anterior arteries little or no change resulted, but that on obliterating the long posterior ciliaries hypotony and widespread degenerative changes took place throughout the eye.

Thus while they do not of themselves prove anything, the vascular pressures of the eye show nothing inconsistent with the hypothesis that the intraocular fluids are formed by dialysation from the blood. It is hoped in further researches to be published in the near future to offer evidence of another kind and of a more conclusive nature that this is the case.

VI. Summary and Conclusions

(1) The methods which have been adopted in the estimation of the vascular pressures of the eye are detailed and criticized. It is shown that no one has yet succeeded in measuring the pressure in the intraocular arteries, that the technique employed hitherto in measuring the venous pressure has been inadequate in that it has involved in every case a wide departure from the normal conditions, and that no method has been suggested which can claim to have measured the normal pressure in the capillaries.

(2) The mechanism of the arterial and venous pulses in the eye are discussed: the arterial pulse is shown to be dependent upon the excursion of the pulse pressure, and upon the difference between the lowest point of this cyclical variation and the intraocular pressure. The venous pulse is dependent primarily on the arterial pulse, and in addition to the factor of pressure relations, its occurrence or non-occurrence is influenced by other factors, being largely determined by the ease with which the blood finds an exit

from the eye; any method of estimating the venous pressure from its occurrence is therefore fallacious.

(3) Experimental technique is described which has established the first measurements which have been made of the arterial pressure in the eye; and which has provided measurements of the venous pressure for which are claimed a closer approximation to the normal conditions than that involved by the methods employed hitherto.

(4) The mean pressure in the ophthalmic artery is not greatly below the mean aortic pressure (about 100 mm. Hg in the cat.)

The pressure in the retinal arteries is about 25 per cent. below that of the ophthalmic artery (75 mm. Hg in the cat).

The pressure in the intraocular veins is in all circumstances greater than the intraocular pressure.

The pressure in the venous exits is normally slightly above the intraocular pressure (1.5 mm. Hg in the dog).

There is a rapid fall of pressure as soon as the veins leave the eye; the pressure in the episcleral veins in the dog is about 7.2 mm. Hg below the intraocular pressure.

(5) The venous pressure and the intraocular pressure vary together very intimately.

(6) Under conditions of raised intraocular pressure the pressure in the venous exits may fall below the chamber pressure.

(7) The nature of the capillary circulation is discussed, and the variability of its pressure pointed out. It is suggested that the capillary pressure in the eye varies from about a few mm. Hg above the intraocular pressure to a height of about 50 or 55 mm. Hg.

(8) It is shown that the vascular pressures and their relation to the intraocular pressure are compatible with the theory of the formation of the aqueous by a process of dialysis from the blood, and do not necessitate the postulate of any "secretory" energy.

(9) The circulatory conditions lead to the expectation that the dialysation of the intraocular fluids would occur from the blood stream mainly, but not entirely, through the vessels of the ciliary body and iris, and to the blood stream mainly, but not entirely, through the canal of Schlemm.

(10) Inasmuch as the venous pressure in the eye is normally higher than the intraocular pressure, a hydrostatic outflow of the aqueous is impossible. Osmotic re-absorption into the blood stream is possible throughout the eye generally, but with its favourable position down the venous pressure gradient, and with its endothelial wall resembling that of a capillary, it is probable that a great part of the process takes place in the canal of Schlemm. Further, under conditions of raised intraocular pressure, the equilibrium is so altered that a hydrostatic outflow may occur here temporarily, the canal of Schlemm under these conditions acting as a safety-valve mechanism to aid in the maintenance of the intraocular pressure at its normal level.

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ABSTRACTS

I.—DISEASE OF UVEA

(1) Fuchs, A. (Vienna).—Anatomy of inversion and recession of iris. *Amer. Jl. of Ophthal.*, August, 1925.

(1) **Fuchs** states that in inversion the iris lies completely back upon the ciliary body, whereas in recession the iris is folded and incarcerated between the lens and ciliary body. Either condition may occur after cataract operations, when there has been prolapse of vitreous which fills the anterior chamber and pushes the iris backwards. In the region of the wound this displacement is particularly marked, the iris disappearing from view and giving the appearance of a coloboma whose two borders run backwards into the depth of the eye. The condition occurs frequently when attempting iridectomy in cases of traumatic dislocation of the lens. The anterior chamber is filled with vitreous and after making the incision the iris drops so far backwards that it cannot be grasped with forceps. During a curette evacuation, after needling, some vitreous escaped with the lens matter. A black hole appeared in the iris in the region of the corneal incision, there was no prolapse and the condition was due to retroversion. The phenomenon is also seen after perforating injuries, in which vitreous enters the anterior chamber and presses the iris backwards, and the same may occur with haemorrhage. It is found as a general rule, that if the traumatic perforation is in front of the scleral spur, the iris inversion or recession is on the opposite side of the eye, and *vice versa*, if the injury is behind the spur. In the latter case the foreign body indents the sclera to a considerable degree before perforating it, thus raising the intraocular pressure. After the perforation the whole contents of the eye are moved towards the opening in the sclera. The aqueous in attempting to reach the wound can depress the iris backwards or invert it on this side. The lens may be luxated towards the wound or entirely