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THE BLOOD-PRESSURE IN THE EYE AND ITS RELATION TO THE CHAMBER PRESSURE

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THIS paper is a belated sequel to three others which appeared in this journal under the same title in 1917 and 1918. The first dealt with principles governing the pressure of fluids moving through tubes,⁽¹⁾ the second with measurements of blood-pressure in general,⁽²⁾ the third with the blood-pressure in the retina.⁽³⁾ The present article re-examines the methods by which the pressures in the central artery and vein have been estimated; it deals particularly with the effects of external pressure on the eye, and with the significance of the "venous pulse."

The use of certain terms. The term "chamber-pressure," occurring so frequently in these pages, stands for "intraocular pressure": it saves three syllables every time, is not less expressive I think, and avoids any possibility of confusing the pressure in the intraocular blood-vessels with that in the chambers. It signifies, of course, the pressure exerted by the fluid in the aqueous and vitreous chambers against the containing walls—a pressure not measurably different in the two regions.

The condition of the eye which we test with two fingers or the tonometer is universally called the "tension," and the term, though somewhat vague when so used, is far too convenient to be dropped; but is there any good reason for using this word, in otherwise careful writing, as though it were synonymous with "pressure"? One reads of "intraocular tension," and of the "tension" inside

and outside a blood-vessel. I venture to urge, as I have done before, that such expressions rob the word of its distinctive meaning; they obscure the difference between the pressure of a fluid and the tension of the membrane that contains it.

Properly used the words mean different things: *pressure* implies push, *tension* implies pull. The pressure of the fluid in the chambers keeps the cornea and sclera in a state of tension; the pressure of the blood gives tension to the wall of the vessel. The tension in these cases is due to the pressure, but it is neither identical with it nor always proportional to it. The sclera and the cornea bear equal internal pressure per sq. mm., but the sclera is tenser than the cornea because of its flatter curvature. With equal chamber-pressures a large eye has a tenser wall than a small one. With equal blood-pressure in both, an aneurysmal sac is tenser than the artery that feeds it; if it bursts it does so not only because its wall is weakened, but because it is stretched more forcibly. In short the tension of a membranous envelope is determined not merely by the internal pressure per unit area, but by the size of the cavity and the curvature of the wall in the region in question.

This principle has long been known and stated mathematically (see Leber,⁽⁴⁾ Parsons,⁽⁵⁾ and for fuller exposition and references Cranston Walker⁽⁶⁾). Objective proof is open to anyone who cares to repeat a simple but striking experiment shown some years ago by Rayner Batten.⁽⁷⁾ Slightly modified it is easily made as shown in Fig. 1:—

FIG. 1.

Connect a rubber toy balloon with a small hand-bellows. Before inflating it slip over it a small curtain- or finger-ring, placing this rather nearer to one end of the balloon than to the other. Now inflate. The balloon forms two communicating chambers, one larger than the other, and while the larger expands rapidly, its wall becoming thin and tense to the touch of the finger, the smaller enlarges slowly and remains comparatively slack. To prove that this is not due to any initial inequality in the membrane, deflate the balloon and shift the ring to the other side of the middle; then re-inflate. The two parts are now reversed in size; the larger again expands and grows tense—and perhaps bursts—while the smaller remains much less tense. Yet from first to last the pressure per sq. inch is the same in both. Caution:—When the parts differ much in size the larger may drag the smaller through the ring and shoot the ring off with force.

In this experiment the relation of the tension to the pressure alters greatly because the membrane is highly extensible. Under slight inflation the wall of the larger chamber becomes tenser than that of the smaller, and, therefore, suffers more expansion; this increases the inequality of tension and leads to further inequality of expansion, and so on. Fibrous envelopes are comparatively inextensible, but their tension in relation to size and to pressure from within is subject to the same principle.

Blood-pressure in the central artery and vein

Entrance - pressure. The pressure in the central artery has been measured by raising the chamber-pressure artificially, and noting its height when it just suffices to overcome the blood-pressure and close the vessel. Intermittent closure shows that the diastolic pressure is overcome, persistent closure that the systolic pressure is overcome. The behaviour of the artery is watched with the ophthalmoscope.

Clearly this method can be trusted only if the chamber-pressure at the stages mentioned can be correctly measured. In the laboratory it can be raised by known amounts by means of a canula connecting the vitreous with a manometer (von Schultén⁽⁸⁾). In the clinic, the canula being inadmissible, a different method has been employed. The chamber-pressure is raised by pressing on the eye externally with an instrument which, by means of a spring and pointer, indicates the amount of force exerted, and from this indication the chamber-pressure is inferred (Thomson Henderson,⁽⁹⁾ P. Bailliar⁽¹⁰⁾). The clinical method, apart from its manual difficulty, has certain drawbacks. The instrument at best can only measure the *increment* of pressure it induces in the eye, whereas it is the *resulting total*, i.e., the previous pressure plus the increment, that we want to know; its reading, therefore, must be supplemented by an estimate of the previous pressure. Moreover, the increment is not measured with certainty, for it varies not only with the amount of force exerted, but with the manner of applying it and with the character and condition of the eye.

To what extent do these drawbacks impair its value? The following experiments were undertaken with the aim of answering that question. In considering them the reader will kindly bear in mind that the static force exerted by a spring in any condition of extension or compression can be represented by a weight; also that the pressure of a liquid, though conveniently expressed by the height of a column of water or mercury, is defined more fully in terms of weight and area: a pressure of 20 cm. H₂O means 20 grammes per sq. cm., for 1 cc. of water weighs 1 gramme.

FIG. 2.

Fig 2 shows the nature of the apparatus. The chief tests were made on excised eyes of freshly killed oxen, sheep, and pigs; some on a brass drum covered by a membrane; a few on small rubber balloons.

External pressure was applied by a series of 20-gramme weights placed on a carrier weighing 20 grammes. Two interchangeable carriers were used; one terminated in a flat disc of greater diameter than the area of contact with the eye, the other in a spherical knob 8 mm. in diameter.

The internal pressure was controlled by a water-column, but the readings are here converted for convenience into mm. Hg, the nearest whole number being given; thus the initial pressure of 20 cm. H_2O is given as 15 mm. Hg instead of 14.7. The canula lay in the anterior chamber, where blockage is more easily avoided than in the vitreous, the weight being applied, like the dynamometer in the clinic, over the sclera.

The tubes were freed from air by opening the outlet. The necessary bubble was brought into the horizontal tube by momentarily raising the syphon from the reservoir while the outlet was open. This bubble (von Schultén's device) is essential. To measure the rise of chamber-pressure induced by a given weight one must have the tap open; but an open tap allows fluid to escape through the canula, and this, unless rectified, vitiates the measurement, for there is no such escape in an unpunctured eye. The bubble meets the difficulty. Before the weight is added, the tap being open and the height of the reservoir registered, the position of the bubble is marked. On addition of the weight the bubble moves towards the reservoir, but is immediately driven back to its previous position by raising the reservoir. The eye now contains the same quantity of fluid as before—except for the small amount that has escaped meanwhile through the natural channels—and the height of the reservoir shows how much the chamber-pressure has risen under the added weight.

The results of these experiments may be summarised as follows:—

The rise of chamber-pressure under a given weight varies with the form and extent of the surface bearing on the eye: the smaller the surface the greater the rise. Table I shows the pressures that were found under the disc and knob respectively when they were

equally and progressively weighted. The initial chamber-pressure in every case was 15 mm. Hg. Notice that it always rose more under the knob than under the disc; that the difference increased as the weight increased; and that it was greatest in the smallest eye. In the pig's eye weighted with 80 grammes the rise was 21 mm. Hg greater under the knob than under the disc (average of four eyes tested).

The reason that the disc and the knob raise the chamber-pressure unequally is that they deform the eye unequally. The knob finds a smaller supporting area than the disc, and a smaller supporting area for a given weight implies a higher internal pressure per sq. mm.

FIG. 3.

Let Fig. 3 represent the disc and knob bearing on two equal membranous spheres filled with water and having no outlet. The disc flattens the membrane down until the flattened part meets with as much pressure from the water below it as from the weight above; then the flattened part supports the weight. The supporting area in this case is the area of contact: (diameter a b). The knob, on the other hand, pits the membrane and sinks into it until the upward pressure against the pitted area equals the weight. Here the supporting area is not the area of contact, but that of the depression as measured on an imaginary plane subtending it (diameter a' b').

In each case fluid is displaced, and the greater the volume displaced the more is the whole membrane stretched, and the more is the pressure of the fluid raised. The figure shows that when the disc and knob are so weighted as to find equal supporting areas ($a b = a' b'$) the knob displaces more fluid than the disc: it abolishes not only the space above the line ($a b$), but also some of that below it; in other words, in order to give *equal areas* the knob must be weighted more heavily than the disc. It follows that when equally weighted the knob will give a *smaller area* than the disc, and as already shown, a smaller area for a given weight implies a greater pressure per sq. mm. to uphold it. In each case, according to a well-known principle, the internal pressure per unit area (P) is found by dividing the weight (W) by the number of such units in the supporting area (A), thus $P = \frac{W}{A}$.

TABLE I.

Weight in grammes ...	...	...	...	...	0	20	40	60	80
Chamber-pressure in mm. Hg.	ox	{	disc ...	...	15	18	23	28	34
			knob ...	...	15	20	29	37	43
			difference			2	6	9	9
	sheep	{	disc ...	...	15	21	29	36	43
			knob ...	...	15	25	37	47	55
			difference			4	8	11	12
	pig	{	disc ...	...	15	22	31	39	47
			knob ...	...	15	29	44	56	68
			difference			7	13	17	21

A given instrument raises the chamber-pressure less when placed on the eyelid than when placed directly on the eye, and the smaller the terminal the greater the difference. A pig's eye was tested, first bare then covered by two layers of wet chamois leather—a very supple artificial eyelid 1.5 mm. thick. Under the knob the differences were as shown in Table II; under the disc they were smaller still but definite. We tried the same thing with the real eyelid of a pig, but it was so mobile that the knob slipped sideways and the results were worthless. Unquestionably an interposed pad lessens the rise of pressure by enlarging the supporting area, and its effect is proportionally greater as the terminal is smaller, but if the eyelid is at all comparable with the substitute we used, its effect in this way must be small.

TABLE II.

Weight in grammes ...	...	...	...	...	0	20	40	60	80
Chamber-pressure in mm. Hg.	{	eye bare ...	...	...	14.7	32.7	47.5	61.0	73.5
		eye covered ...	...	...	14.7	31.5	46.0	58.6	69.8
		difference ...				1.2	1.5	2.4	3.7

The rise of chamber-pressure under a given weight varies with the size of the eye: the smaller the eye the greater the rise. This is seen in Table I, if we compare the effects of any given weight on the eye of the ox, the sheep, and the pig respectively. Under the heavier weights the differences are large: under 80 grammes the knob raised the pressure to 43 mm. Hg in the ox's eye, to 55 in the sheep's, and to 68 in the pig's. The reason, in part at least, is easily given: other things being equal, a given weight finds a smaller supporting area on a small eye than on a large one, as shown in Fig 4.

But these eyes differed greatly in size: they were chosen for that reason to demonstrate the principle. Healthy human eyes differ much less. Except in young children and high myopes, the smallest diameter is seldom less than 22 mm., the largest seldom more than 26 mm.,⁽¹¹⁾—a ratio of 1 to 1.18, whereas in the case of the pig and ox the average diameters are about as 1 to 1.68. The errors likely to arise through difference of size in human eyes are, therefore, much smaller than the figures in Table I might suggest, but are probably not quite negligible.

FIG. 4.

Fig. 4 was drawn by measurement from an ox's eye and a pig's eye, each under a flat weight of 80 grammes. An initial chamber-pressure of 25 mm. Hg. was established in each, the tap was closed and the weight applied. The supporting area printed itself on the surface of the weight by means of colour smeared on the sclera. The smaller eye, because of its sharper curvature and lesser expansibility, gave a smaller supporting area than the other, and, therefore, suffered a greater rise of chamber-pressure.

The rise of chamber-pressure under a given weight varies with the character of the eye wall: the less extensible the tissue the greater the rise. An ox's eye was compared with a rubber balloon filled with water, these being about equal in size when their internal pressure was 15 mm. Hg. Under a weight of 100 grammes the chamber-pressure rose to 52 mm. Hg in the eye, and to 23 in the balloon—a rise of 37 against 8 mm. Hg—the wide difference being due, of course, to the high extensibility of the rubber as compared with the fibrous tissue. We also used the drum (see Fig. 1) covered first with sheet-rubber 0.7 mm. thick, and then with the same supplemented externally by very thin silk. The silk limited the extension of the rubber and thereby increased the rise of pressure under a given weight.

But the principle hardly needed demonstration: it is self-evident; the question is whether human eyes differ in this respect enough to affect the findings materially. Schiötz's experiments in tonometry,⁽¹²⁾ and later my own,⁽¹³⁾ showed that, of eyes having the same chamber-pressure, some are more impressible than others, but they did not show how far this may depend on difference of tissue rather than of size. The only positive evidence on that point relates to the eyes of children. Under continued excess of

chamber-pressure the child's eye, unlike that of the adult, enlarges considerably in all diameters, although by reason of its smaller size its envelope is normally less tense than that of the adult eye (see p. 450); evidently its tissue is more extensible—as one would expect.

Equal increments of weight may induce unequal increments of chamber-pressure in one and the same eye. Having considered the effects of difference in the instruments used, in the manner of using them, and in the eyes examined, we have still to note the effect of a given increment of force at different pressure levels in a given eye. Table III shows the same series of observations as Table I, but gives the *increments* of chamber-pressure instead of the *totals* induced by successive weights of 20 grammes.

TABLE III.

Weight in grammes	...	...	...	...	0	20	40	60	80
Increments of chamber-pressure in mm. Hg.	ox	...	{	disc	...	3	5	5	6
				knob	...	5	9	8	6
	sheep	{	disc	...	6	8	7	7	7
				knob	...	10	12	10	8
	pig	...	{	disc	...	7	9	8	8
				knob	...	14	15	12	12

In every case the increment was smaller under the first than under the second weight. This was to be expected, for the extensibility of the eye diminishes as the chamber-pressure rises: with subnormal chamber-pressure it is considerable, about the normal level it diminishes rapidly, at high levels it is very small (see measurements in dog, rabbit, and man by von Schultén⁽⁸⁾ and Koster⁽¹⁴⁾). Since the initial pressure was subnormal in our experiments, the first weight would displace fluid more easily than the second, and would, therefore, raise the pressure less.

But under the third and fourth weights the increments, instead of growing larger as one might expect from the lessened extensibility of the eye, fell off again. This can hardly have been due to faulty observation for the tendency was always in the same direction; moreover, it is visible also in Bailliar's charts. Possibly it was due to the altered shape of the loaded eye and a consequent effect on the size of the supporting area; or it may have arisen through increased escape of fluid through the natural channels under increased pressure. Whatever the cause, one sees that under equal additions of force the chamber-pressure may rise by unequal steps.

The foregoing observations show that the clinical method in question cannot be expected to measure the pressure in the central artery with precision. It has, in fact, given some very discrepant

results, but it is not on that account to be hastily condemned, for much of the discrepancy is explained when one compares the instruments employed.

Thomson Henderson's "pressure-gauge"⁽⁹⁾ was the first in the field, and through Dr. Henderson's courtesy I have been able to examine the original instrument. Its foot presents a circular flat bearing-surface 3 mm. in diameter. Bailliar's dynamometer,⁽¹⁰⁾ designed later but without knowledge of Henderson's, ends in a button 7 mm. in diameter with a "slightly convex" bearing surface. Applied with equal force to the same eye, these instruments would raise the chamber unequally, Henderson's giving the greater rise because of its smaller foot. Their findings, therefore, are not directly comparable. A *uniform foot* of fully specified and easily checked dimensions for all such instruments (as also for tonometers), would save much uncertainty.

They differ also in their *system of graduation*. The pressure-gauge is marked in mm. Hg, the intention being to show the rise of chamber-pressure induced by the force applied. 'Such a scale would be invaluable if trustworthy but, as shown above, it cannot be correct for all eyes, and even for the average eye the desired values are difficult to obtain. They can only be found by elaborate trial on many human eyes; no drum or other artificial substitute can give them. The instrument kindly lent me for examination was found, when tested against a balance, to indicate a rise of 3 mm. Hg for each 10 grammes of force exerted—except near to zero. It will be shown below that this value is too small for the human eye and would lead to a considerable underestimate of the effect produced on the chamber-pressure.

Bailliar's dynamometer is marked in grammes and shows the force exerted. This scale has no difficulty for the instrument maker, and can be tested at any time by the user, but it does not show the rise of chamber-pressure. For clinical purposes Bailliar holds this to be unnecessary: he is content to measure the applied force under which the artery closes in the individual case, and to compare it with an ascertained normal average. But he has not left the matter there. With infinite pains he has used a Schiötz tonometer together with his dynamometer on many healthy eyes, in order to learn the rise of chamber-pressure occurring under known increments of force. Taking from his table⁽¹⁰⁾ the trials up to 100 grammes, and allowing for the known loss of force when the dynamometer is used horizontally, one finds an average rise of 6.6 mm. Hg for each 10 grammes of force, and a little may be added because the mercury-values in the Schiötz chart are somewhat too low.

Further, Bailliar (with Magitot⁽¹⁵⁾) used the dynamometer together with an injection-manometer on a cat under anaesthesia.

The average rise per 10 grammes works out at 8.3 mm. Hg—a result according well with the foregoing, as the cat's eye is smaller than the man's. Certain observations of my own⁽¹³⁾ on human eyes after 'death, with an injection-manometer and an applanation tonometer weighing 10 grammes, are also in agreement; with four different initial chamber-pressures, the average rise under the 10-gramme weight was respectively 7.1, 7.2, 7.3, 7.3 mm. Hg. These findings together with those from the pig, sheep, and ox are placed seriatim in Table IV; they must not be taken as strictly correct, and they are obviously not exactly comparable with each other, but it is significant that the figures found for the human eye place it among the others in accordance with its size and shape. We may be sure, I think, that under an instrument such as Bailliant's the chamber-pressure of normal human eyes rises on the average by something like 7 mm. Hg per 10 grammes of force, and that under one with smaller bearing-surface it would rise more.

TABLE IV.

Average rise of chamber-ressure per 10 grammes of force applied.

Ox's eye (as described, with flat compressor)	...	...	...	2.4 mm. Hg.
" " " knob "	...	...	...	3.5 "
Sheep's eye " " flat "	...	...	...	3.5 "
" " " knob "	...	...	...	5.0 "
Pig's eye " " flat "	...	...	...	4.0 "
" " " knob "	...	...	...	6.6 "
Human eye, living, chamber-pressure measured by Schiötz tonometer (from Bailliant's table; <i>Annales d'Oculistique</i> , p. 658, 1917)	...	...	...	6.6 "
Human eye, post-mortem, chamber-pressures measured by manometer and applanation tonometer (Priestley Smith)	...	...	...	7.3 "
Cat's eye, living, anaesthesia, chamber-pressures measured by manometer (Bailliant and Magitot)	...	...	...	8.3 "

So far then, one would expect Bailliant's method to afford valuable though not precise information as to the normal entrance-pressure in the human eye, and in some hands it has, in fact, given nearly uniform results. Bailliant estimates the diastolic pressure in the central artery to be a little over 30 mm. Hg, the systolic a little over 70 mm. Hg; Velter⁽¹⁶⁾ puts them at 35 and 65; Nunes at 30 and 62. On the other hand, Duverger and Barré,⁽¹⁷⁾ apparently following the same method, found higher pressures: diastolic 50 to 60; systolic 80 to 100; no reason for the divergence is apparent. And there is another difficulty.

von Schultén, using a more direct method, found much higher pressures. He raised the chamber-pressure by means of an

injection-manometer, and watched the artery under high magnification (see *Brit. Jl. of Ophthalm.*, 1918, p. 258); result: diastolic pressure at least 90, systolic 100 to 120 mm. Hg. True, this was in rabbits' not in human eyes, but mean blood-pressure in mammals is said to differ little and to be independent of size. Whence then the discrepancy? Possibly the following considerations will explain it.

The injection-apparatus makes pressure on the arteries inside the eye, but not on those behind it; the dynamometer makes pressure on both. von Schultén's method raises the pressure in the afferent arteries to some extent, and perhaps considerably, for since the carotid is not far distant from the ocular capillaries, the natural fall between the two must be steep, and the steeper the natural fall the greater the rise above an obstruction (see *Brit. Jl. of Ophthalm.*, 1917, p. 658). Bailliart's method includes an additional and conflicting factor: the eyeball, hardening as its chamber-pressure rises, is pressed against the parts behind it. This double influence on the arteries suggests that of two fingers side by side on the radial at the wrist: the force of the stream under the lower finger is at the mercy of the upper. The conditions in the orbit are highly complex; one cannot compute the net result; but it seems possible that the backward pressure of the eye—by slightly bending the arteries at points between the optic foramen and the papilla, as well as by lessening their calibre—may lower their blood-pressure before they reach the eye. In any case the two methods must affect the blood-pressure at the papilla unequally in the act of measuring it.

It is clear that only a rough estimate of the normal pressure in the central artery can be made. The figure, 90 mm. Hg, given as the mean in my earlier paper was based on the findings of von Schultén and Leonard Hill, and was no doubt too high. The following figures are probably nearer to the reality. *Normal entrance-pressure: diastolic, 40 to 50 mm. Hg; systolic, 70 to 90 mm. Hg.*

Exit-pressure. The central vein like the artery is subject to the chamber-pressure and must support it or collapse. In many eyes the so-called "venous pulse" shows that the resistance of the vein near to its exit is alternately greater and less than the chamber-pressure, for each in turn overcomes the other; and since this resistance depends chiefly on the lateral pressure of the blood-stream, we have only to measure the chamber-pressure to learn approximately the exit-pressure of the blood.

But what is the exit-pressure in the many eyes that show no venous pulse? Clearly it is high enough to hold the vein continuously open in spite of the oscillating chamber-pressure, and

we have indirect proof that it is usually not much higher. The lightest touch of the finger on the eyelid narrows the vein at its exit (Thomson Henderson⁽⁹⁾), in other words, the wall of the vein yields to a very small rise of chamber-pressure; before the touch, therefore, it must have been only just holding its own.

Further, we know that when the chamber-pressure rises above its normal level the venous pressure rises with it, for the finger even when firmly applied does not close the vein. As the vein narrows, the *vis a tergo* of the blood-stream drives up the venous pressure until it supports the increased load, and the outflow though diminished is not arrested. Only by exerting force enough to close the central artery can the finger permanently close the vein, for so long as there is inflow there must be outflow.

An experiment described in a previous paper illustrates the principle (*Brit. Jl. of Ophthalm.*, 1917, p. 661). A piece of pigeon's gut carries a stream of water through a glass chamber filled with water; the pressures in gut and chamber are controlled. A pressure of 15 cm. being established in the gut, that in the chamber is gradually raised from below that level. When it just exceeds 15 cm. it affects the stream through the gut: the distal end of the gut is compressed, the outflow is diminished, the pressure of the stream above the point of compression rises, below that point it falls. *The hindrance begins simultaneously with the narrowing at the exit.*

Again, we have evidence that when the venous pressure is raised by disturbance of the circulation outside the eye, the chamber-pressure rises also. Leonard Hill⁽¹⁸⁾ found (in animals) that a rise of blood-pressure in the vena cava induced an equal rise in the torcular Herophili, and in later experiments he and Thomson Henderson⁽¹⁹⁾ found that the rise in the torcular was associated, after a little delay, with an equal rise in the chambers of the eye.

It appears then that the venous exit-pressure and the chamber-pressure are usually nearly equal, and that when either of them rises or falls the other does the same—a safeguard against oedema and haemorrhage in the eye. Yet they are not quite equal: the varying behaviour of the central vein shows that their relation to each other differs in different eyes, and that it often changes in the same eye. The question is how and to what extent this happens? Please observe that we are not inquiring whether the venous pressure at the papilla can rise much above its normal level—we know that it does so—but whether it can much surpass the chamber-pressure.

There are conditions under which it certainly does so. During free leakage from a corneal wound, the chamber-pressure may sink almost to zero, while the venous exit-pressure cannot fall below that in the orbital veins; there is then an abnormal difference between the two with consequent risk of haemorrhage. In thrombosis of the central vein the venous pressure rises almost to the level of that in the central artery, while the chamber-pressure may not rise at all, and haemorrhage is inevitable; the

glaucoma occurring later results indirectly from the haemorrhage (Inouye, G. Coats⁽³⁰⁾). But these conditions are exceptional; we are considering what may happen in normal eyes.

M. Bailliart,⁽¹⁵⁾ after much study of the subject, reaches some surprising conclusions. He places all eyes that show no spontaneous venous pulse in two groups: (1) those in which the exit-pressure is continuously higher than the chamber-pressure; (2) those in which it is continuously lower, and he distinguishes them as follows:—In the former group finger-pressure induces the venous pulse by raising the chamber-pressure to the level of the blood-pressure; in the latter it does not, because the chamber-pressure is already the higher of the two. Further, he holds that excess of exit-pressure over chamber-pressure can be measured in the following way: if the venous pulse is absent, he ascertains by means of the dynamometer how much the chamber-pressure must be raised in order to induce it; if it is spontaneously present, he ascertains the rise that abolishes it. Venous hypertony in the eye is to be detected in this way.

Unless I am mistaken there are weak points here. Such measurements would be instructive if the dynamometer could raise the chamber-pressure to the level of the venous pressure without altering the latter, but if it raises both it cannot determine their previous difference. Undoubtedly the venous pulse, by its coming and going, gives very delicate indications of changes in the mutual relation of these two pressures, but it seems to be at the mercy of influences so numerous and so small that it can hardly, I think, have any diagnostic value. Its causation has been discussed by many writers since it was first observed more than 60 years ago—notably by Donders in the first volume of *Arch. f. Ophthal.*—but not quite conclusively, I think, and to justify the objection raised above I shall venture to reopen the question. In gathering the evidence now available I have been much helped by Bailliart's writings, and by Elliot's recent article in this journal (*Brit. Jl. of Ophthal.*, 1921, p. 481). I will first quote certain clinical observations from the latter:—

Of 200 healthy eyes systematically examined in this respect by Elliot, nearly one half showed the "pulse" spontaneously, the slightest rhythmic change of colour being reckoned as such; about half of the others showed it under light finger-pressure; the remainder not at all. When present spontaneously it was usually increased by light finger-pressure, but sometimes abolished. Firmer pressure usually abolished it, but not in every case. Some persons showed it in one eye only; some at times but not at other times. Bailliart found it to occur spontaneously in a smaller percentage of normal persons, Lang and Barrett⁽²¹⁾, (Ballantyne also according to a letter lately received from him), in a larger

percentage, but its precise frequency is not important here; the problem is the seemingly capricious conduct of the vein. What are the influences that promote and oppose its pulsatile collapse?

(a) *Arterial pulsation inside and outside the eye.* The collapse occurs almost simultaneously with the beat of the carotid in the neck, and, therefore, with the arterial pulse in the eye, the orbit, and the cranium, and it is easy to see that the pulse-wave in these several regions tells on the collapse in different ways.

Inside the eye under normal conditions an arterial pulse is not visible by ordinary methods, but the Gullstrand ophthalmoscope shows it even in the finer branches: the pulse-wave expands them and enlarges their curves (Kuemmell,⁽²²⁾ de Speyr⁽²³⁾). The Schiötz tonometer shows that with each expansion of the arteries the chamber-pressure rises by about 1 or 2 mm. Hg, and it is under this rise that in many eyes the vein on the papilla suffers more or less collapse. The collapse implies displacement of blood from the collapsing part and the volume displaced must be received elsewhere. It cannot pass upstream, for here the vein is subject to the same momentary increase of external pressure and cannot expand to receive it. It must pass down stream, expanding the vein in the nerve-trunk or quickening the outflow through it, and any influence which opposes this expansion or quickening of the flow outside the eye opposes the collapse on the papilla. *On and round the papilla* branches of the artery often cross the veins and perhaps compress them as they pulsate. Such direct compression, however slight, would tend to check the venous stream above the crossing and lower its pressure below it, so favouring collapse at the exit. I have published examples which seemed to exhibit this effect (*Brit. Jl. of Ophthal.*, 1918, p. 265), but as Elliot could find little evidence of it they were probably exceptional.

In the optic nerve the artery and vein lie for some distance side by side, sometimes in separate connective-tissue sheaths, but usually in a common sheath (Leber⁽⁴⁾), a position in which the vein is likely to be compressed by each pulsatile expansion of the artery, or at least to be prevented from itself expanding at that moment. The arterial pulse in the nerve, in so far as it tells on its companion vein, opposes the venous collapse at the papilla.

In the orbit the veins on their way to the cavernous sinus are subject to pulsatile compression by the neighbouring arteries, the impulse being transmitted by the other contents of the cavity as in the eye, though probably with more loss because the containing space is less completely walled in. Here again the arterial pulse opposes the expulsion from the eye.

In the skull, as in the eye, the arterial wave raises the pressure of the contents and quickens the escape at the outlets; thus, in

the torcular Herophili (in dogs) Leonard Hill⁽¹⁸⁾ found a pulsatile oscillation of the venous stream, and in the internal jugular vein near to its emergence from the skull (in dogs and calves) von Bergmann and Cramer, in 27 out of 31 experiments, found a similar small oscillation (see Helfreich⁽²⁴⁾); in each case *the rise of the venous pressure synchronized with the beat of the carotid*. In some degree, therefore, the arterial pulse in the skull supplements that in the orbit in its opposition to the expulsion from the eye.

The influence of arterial pulsation over neighbouring veins is, of course, not peculiar to the region of the eye; it appears to be widely spread. Bailliant⁽²⁵⁾ quotes Gley as follows: "Les battements des artères exercent une influence sur les veines voisines . . . on a constaté, en effet, que toute dilatation artérielle donne lieu à une ondulation veineuse." In some organs, indeed, it seems to be essential to a normal capillary blood-flow, for when an artificial circulation is set up it is soon obstructed by oedema unless the driving force be made intermittent in imitation of the heart's action (Leonard Hill). But arterial pulsation is not the only influence that requires consideration here.

(b) *Venous pulsation from the right auricle*. The internal jugular vein, just above the level of the clavicle, pulsates under the influence of the right auricle, and tracings taken at this spot show that the pressure is lowest in the vein just when it is highest in the carotid artery (Sir James Mackenzie,⁽²⁶⁾ p. 102, and Fig. 44). The fall in the jugular synchronizes, therefore, with the collapse at the papilla, and if we imagine it travelling back to the eye we may well suppose, with Bailliant and Elliot, that it takes part in causing the collapse. But can it travel so far? No doubt it has *some* effect at the papilla, and even further back, but a fall that is real and considerable low down in the neck may become merely virtual—a minute lessening of a rise—higher up the stream; and apparently this is what actually happens before the eye is reached. Even at the upper end of the jugular the venous pressure (in animals—see above) already rises a little with each arterial pulse, and in the orbit it is further subjected to the same influence. One can hardly doubt, I think, that the fall due to the auricular diastole is converted into a rise before it reaches the eye, and that at the papilla the venous pressure rises and falls not alternately with, but together with, the arterial pressure.

If this be true, it follows that under the influence of arterial pulsation *the venous pressure and the chamber-pressure rise and fall together*—but not necessarily to the same extent. When the venous pulse is present we may be sure that with each beat of the carotid the pressure in the chambers is rising more than that in the vein; when we see no venous pulse we may reasonably

suppose that their oscillations are more nearly equal, though a different explanation may be the true one—the basic level of the venous pressure and not its pulsatile oscillation may be raised (see p. 466 and Fig. 5).

Imagine on the other hand that these pressures oscillate *against* each other, the one rising as the other falls, and consider what this implies for those cases where, according to M. Bailliart, the venous pulse is absent *because the venous pressure is continuously lower than the chamber-pressure* (see p. 461). At the moment when the chamber-pressure is at its lowest the venous pressure will be at its highest, but still lower than its opponent—they will be nearly equal; at the next moment, the one having risen and the other fallen, they will differ by the sum of this rise and fall, and a trifle more—yet the vein will show no collapse. Under finger-pressure the oscillation of the chamber-pressure will increase [see below, *d* (2)], and the difference will be greater still, and still there will be no collapse!

The wall of the vein could not remain steady under such an oscillation, for when the inside and outside pressures were equal its tension would be nil, and in that condition it would be displaced by a very small change in their relation to each other.

It may be objected that as the venous collapse usually lasts longer than the arterial wave, it cannot depend entirely on the latter. But the sharp “stabbing” pulse of the central artery seen under finger-pressure is abnormal; the normal pulse, if visible, would show a somewhat different character. Moreover, the pointer of the tonometer shows, I think, that the time during which the chamber-pressure is raised is long enough to account for the effect on the vein; one would expect the refilling of the vein to be a little less prompt than the subsidence of the arterial wave.

The influences so far considered are pulsatile, but there are others which are not so. The venous pulse when spontaneously present can usually be abolished by raising the venous blood-pressure, and when absent it can often be induced by raising the chamber-pressure. It is probable, however, that under either of these changes the difference between the two pressures is still quite small.

(c) *Disturbance of the circulation.* When the arm is raised, the head lowered, or the neck compressed, or when a forced expiration is made, a venous pulse previously present will often disappear, and at the same time the vein may be seen to swell (for references see Helfreich⁽²⁴⁾). Under such circumstances the venous pressure rises and acquires a continuous instead of an intermittent mastery over the chamber-pressure. We know, however, that when the one rises the other rises also, and that when the pressures inside and outside a vein are equal a very small addition to the inside pressure will expand the wall freely; a small excess of the venous pressure over the chamber-pressure, therefore, will account for the swelling of the vein. It is significant, I think, that healthy eyes showing no venous pulse exhibit, as a rule, no such swelling, *i.e.*, no suggestion of even a small excess of venous pressure.

Changes of arterial blood-pressure also affect the behaviour of the vein at the papilla. Thus, raising the aortic pressure (in animals) may induce a venous pulse; ligature of the common carotid has been seen to banish it on the operated side, and to induce or amplify it on the other; nitrite of amyl (in animals and man) will often induce it.⁽²⁴⁾ These disturbances act no doubt both on the chamber-pressure and on the force of the arterial pulse.

(d) *Finger-pressure on the eye* influences the central vein in more ways than one :—

(1) By deforming the eye it raises the chamber-pressure, adding thereby to the burden on the vein and favouring its collapse. But by narrowing the vein at its exit it leads to a rise of the venous blood-pressure and so opposes the collapse (see p. 460).

(2) By raising the chamber-pressure it makes the tunic of the eye tenser, and therefore less extensible under the arterial wave; it thereby augments the pulsatile rise of the chamber-pressure. We have good evidence of this. The pointer of the Schiötz tonometer shows that in normal eyes the average oscillation is about 1.5 mm. Hg; in glaucomatous eyes about 6 mm. Hg (Ballantyne⁽²⁷⁾). More conclusive still, it is greater under a heavy tonometer-weight than under a light one in the self same eye. The augmented oscillation of the chamber-pressure favours the collapse of the vein.

(3) By pressing the eyeball against the other contents of the orbit, especially if the pressure be firm, the collapse is opposed, for pressure on the veins behind the eye hinders the outflow through them and pressure on the arteries here probably lessens the force of their pulsation inside the eye (see p. 459).

(4) By continuance it tends to deplete the vessels and the chambers (though exceptionally it causes turgescence of the veins—see Elliot) so that when it is suddenly relaxed, both venous pressure and chamber-pressure drop to an abnormally low level. Thereupon the veins fill up quickly and become over-full, while the chambers refill more slowly, and normal conditions are gradually re-established. The behaviour of the vein, as described by Elliot, is in accord with these changes. In a few cases the venous pulse puts in a brief appearance immediately the finger is withdrawn, showing that the two pressures though lowered are for the moment about equal; but it soon vanishes, for the venous pressure rises more quickly than its opponent. In most cases there is no venous pulse at this moment, for the fall leaves the venous pressure the higher of the two—as may be proved by light re-application of the finger; this raises the chamber-pressure to the level of the venous pressure and the pulse appears.

Figure 5 is an attempt to indicate these varying relations in a diagrammatic way. Let CP (the thicker line) stand for the chamber-pressure, and RV (the thinner line) for the resistance of the vein at the papilla, *i.e.*, its blood-pressure supplemented by its structural rigidity. Whenever CP rises above RV the vein suffers more or less collapse, and the greater the excess of CP over RV the greater the collapse. CP and RV both oscillate, sometimes equally, sometimes unequally, under the influence of arterial pulsation.

PART I shows RV in three different relations to CP, involving respectively a big collapse, a small collapse, and no collapse of the vein. The difference lies, not in the basic level of RV—this is the same in all—but in its oscillation, *i.e.*, on the local effect of the arterial pulse-wave. Imagine the pulsatile expansion of the

FIG. 5.

arteries to affect the venous stream more powerfully inside than outside the eye, and we have the conditions for a venous pulse at the papilla; imagine it acting equally in the two regions, and we have the conditions for a steady venous stream, for with each beat of the arteries RV will rise equally with CP. Such equality, or something near it, is probably true, I think, of most of the normal eyes that show no spontaneous venous pulse.

PART II shows RV again in three different relations to CP, but here it is the basic level of RV and not its oscillation that differs. Supposing CP to oscillate to the extent of 2 mm. Hg, then a change of 1 mm. Hg in the mutual relation of the two pressures may cause the venous pulse to appear or disappear. Slight disturbances of the circulation will change the relation of RV to CP to this extent, and will explain the varying behaviour of the central vein in the selfsame person.

PART III purports to show what commonly happens when the finger (or the dynamometer) makes gradually increasing pressure

on an eye that shows no spontaneous venous pulse. Until the finger is applied, RV and CP oscillate nearly to the same extent and there is no visible collapse of the vein. Under the finger, they rise together but not quite uniformly; their mutual relation changes, and changes back again; the venous pulse appears, increases, decreases and disappears. The changes are not alike in all cases, for they depend on numerous and varying factors. The diagram stands for the average case and the likeliest explanation of the changes seems to be this:—*Light finger-pressure induces the venous pulse chiefly by augmenting the oscillation of the chamber-pressure; firm pressure banishes it chiefly by compressing the veins behind the eye* (see p. 465 (2) and (3)).

This explanation is perhaps incomplete even for the average case. Firm pressure stretches the vessels inside the eye. We know that violent compression of the eye from in front often stretches the membranes sufficiently to rupture the choroid concentrically with the papilla, and it is not unlikely that firm finger-pressure may stretch the central vein enough to add a further hindrance to its blood-flow, and so oppose its collapse. Glaucoma stretches and displaces the vessels at the papilla, but makes no pressure on those behind the eye; its average effect on the venous pulse, if definitely known, would throw light on the question, but the available evidence is insufficient. Bailliart saw the venous pulse in only 7 out of 57 glaucomatous eyes—a much smaller proportion than that for healthy eyes. Fuchs⁽²⁹⁾ says that it is very often seen; Elliot says that a strong venous pulse is not uncommon, and is often banished by relief of tension. More observations on the point would be helpful.

In almost every eye that shows the venous pulse, one sees proof that a very small factor is enough to turn the scale in its favour or against it. While certain branches of the vein on the papilla pulsate, others do not. These branches can only differ slightly in their structural rigidity, and they can hardly differ at all in their blood-pressure for they are on the point of uniting in a single trunk.

Summary

1. *The arterial entrance-pressure* cannot be measured with precision. The injection-manometer is only applicable in the laboratory; moreover, though it measures correctly, it raises the arterial blood-pressure before it measures it. The dynamometer lacks precision in measuring the chamber-pressure, and perhaps lowers the arterial blood-pressure by compressing the arteries behind the eye. The following estimate is probably near to the truth, but is given with reserve: Diastolic pressure, 40 to 50; systolic, 70 to 90 mm. Hg.

2. *The venous exit-pressure*, under all ordinary circumstances, is nearly in equilibrium with the chamber-pressure. When it falls *below* the chamber-pressure, by more than the small amount which the vein can support by its own rigidity, it is immediately driven up by the *vis a tergo* until the equilibrium is re-established.

When it rises *above* the chamber-pressure, the latter rises also, and equilibrium is approximately re-established, though less promptly and perhaps less completely than in the former case. Any abnormal excess of the venous pressure over the chamber-pressure causes the vein to swell.

3. The self-adjusting equilibrium of pressure inside and outside the vein safeguards the retina against oedema and haemorrhage; when it fails, as during free leakage from the chambers, blockage of the vein, or sudden compression of veins outside the eye, disaster is apt to occur.

4. The veins in the eye are indirectly compressed by each pulsatile expansion of the intraocular arteries; those in the orbit are similarly compressed by the intraorbital arteries. When the effect is equal, or nearly so, inside and outside the eye, there is no visible interruption of the venous stream; when it is greater within than without, blood is expelled from the eye suddenly, and the vein collapses on the papilla—we see the so-called “venous pulse.”

5. *The venous pulse*, though essentially dependent on arterial pulsation, is at the mercy of many influences. It is opposed by any condition or change that prevents sudden expulsion at the papilla, and particularly by overfilling or compression of veins outside the eye. It is favoured by all influences that promote such expulsion. The venous pulsation that proceeds from the right auricle tends to favour it, but not in discoverable degree.

6. *External pressure by finger or dynamometer* raises both the chamber-pressure and the venous exit-pressure. It commonly alters their relation to each other, and thereby causes the venous pulse to appear and disappear, but it does not abolish their approximate equilibrium—except when suddenly withdrawn. The amount of artificial pressure which suffices to induce or to banish the venous pulse is not a measure of the difference between the venous pressure and the chamber-pressure.

[Sept. 4.—Since sending this article to press, I have received through the kind courtesy of M. Bailliant, a copy of his new work: “*La Circulation Rétinienne à l'Etat Normal et Pathologique*” (Paris: Octave Doin, 1923), a volume of 400 pages with a preface by Professor H. Vaquez. In the first part the author summarizes, and in some respects elaborates, his previous studies of the retinal circulation in the normal eye, while in the second and third he discusses its functional disturbances and the various diseases of the retinal vessels. Nothing less than a long and careful notice could do justice to this important work. Here I can only say that my reasons for dissenting from certain of M. Bailliant's conclusions are not affected by it. With much

respect and interest, I submit them as they stand to his consideration, and to that of others who may care to follow up these somewhat subtle questions.—P.S.]

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THE PECTINATE LIGAMENT IN ITS RELATION TO CHRONIC GLAUCOMA

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THE greater part of this report was read at the last Ophthalmological Congress at Oxford, in recognition of Professor Arthur Thomson's well known work on the same subject.

The results are given here of a recent research upon over thirty eyes. The starting point of the investigation was the obtaining of an eye in so early a stage of chronic simple glaucoma that a rare opportunity was afforded of looking for predisposing anatomical causes of the disease, uninfluenced by the secondary effects of high tension. The plus tension began while the