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THE RETINAL PULSE

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IT is extraordinary how little is known on the subject of the retinal pulse. The textbook statements on the subject are meagre, and much that has been written is inaccurate. Bailliant's writings have recently stimulated an interest in the subject, especially in France. Under the spell of this work, the writer has recently endeavoured to study the subject more closely than he had done before, and to strive to obtain some accurate and reliable information on points which have hitherto been nebulous and vague.

The Arterial Pulse

There would appear to be not a few ophthalmologists who have never observed this pulse, and yet, as a phenomenon it is so striking and characteristic that no surgeon who has once seen it can ever mistake it afterwards. When dealing with the venous pulse, all gradations are seen between a well-marked beat on the one hand and an almost imperceptible flicker on the other. Indeed, it might well happen in the latter category that several men examining the same case would differ in their opinions as to whether pulsation were present or absent. This would be impossible with the arterial pulse: it flashes out over the whole breadth of the disc, affecting every branch of the artery thereon and even beyond it. It is, however, best seen on the disc itself, for "in the retinal part of the optic nerve, the vessels lie wholly superficial; i.e., they are not

covered by nerve fibres" ("Anatomy and Histology of the Human Eye," Salzmann, Chicago, 1912, p. 89). The pulse is so instantaneous as to suggest the quick flash of a lighthouse stabbing the darkness of night, and passing away as quickly as it came. It has, therefore, nothing in common with the localized, gradual, slowly-developing and slowly-passing venous pulse.

It is seen whenever the intraocular pressure passes the pressure in the arteries during the period of diastole, but is lower than the latter during the systolic period. As Bailliant has so pertinently pointed out, it is an indication that the circulation through the eye is being maintained only during a part of the cardiac cycle. It has therefore very little in common with the ordinary arterial pulse, which is a physiological phenomenon, while this is pathological. Bailliant has observed that, if we use a magnifying ophthalmoscope, such as Gullstrand's, we can see the physiological pulse in the retinal arteries under normal conditions. This is on all fours with the observation that, by the use of suitable apparatus, we can detect the pulse even in the digital arteries; indeed, with care, and granted a delicate sense of touch, we can do this in many patients, without the aid of any form of apparatus. We should not, however, think of confusing this faint digital pulsation with the pulse of aortic insufficiency, which arises as the result of changed pressure conditions within the vessels. In the same way, it would not be reasonable to confuse the normal retinal pulse, as seen under magnification with the ophthalmoscope, with the flash pulse, which we commonly speak of as "the diastolic pulse." Bailliant thinks it is better to reserve the term retinal pulse for the appearance of this phenomenon under abnormal conditions, and probably most of those who have studied the subject will, for convenience sake, agree with him in this.

The diastolic pulse can be very easily seen in most normal eyes: Get the patient in a comfortable, easily reachable position, and examine the fundus with an electric ophthalmoscope by the direct method; while doing so, press with the tip of the index finger on the outer commissure of the eyelids, beginning quite gently, and gradually increasing the force used; as this is done, the pulse will suddenly flash into sight, and will continue so long as the pressure is continued. It is of interest to note that at the lower level of pressure, when the intraocular pressure just exceeds the diastolic arterial pressure, the period during which the artery is emptied is very short as compared with that during which it is filled. As the pressure is increased and the systolic level is approached the filled phase of the artery gets shorter and shorter and the empty phase correspondingly longer. Bailliant and others advise the continuance of this pressure until the pulse disappears, at the point when the intraocular pressure has been raised above the level of the maximum,

or systolic, pressure in the retinal artery. However interesting this may be from the point of view of the examiner, it must be borne in mind that it is a proceeding, which, at least under morbid conditions, may not be wholly free from risk. That this is true, is evident from the fact that on two occasions Bailliart has observed the onset of syncope in the course of the routine performance of this manoeuvre. It may be countered that the darkness which precedes syncope, whether from haemorrhage or otherwise, and which has been noticed in those in the algid stage of cholera and in the dying, is practically of the same nature, and yet that in the syncopic cases, and possibly in the choleraic ones, there is no evidence of any permanent damage being done. To this we may reply that it is not easy to be satisfied that no harm is done to the sight in syncopic cases, especially when due to haemorrhage; nor could we unreservedly subscribe to a similar contention in the case of cholera. The question raised is a thorny and difficult one, but certain important points may here be mentioned: (1) When pressure is being applied to the eye we are *provoking a rise* in pressure in the venous channels at their exit, and back through the capillaries to the arterioles. Is it quite safe in the elderly to risk the possibility of a haemorrhage in this way? The condition in syncope is quite different, for there the pressure *falls* throughout the vascular system of the eye. (2) The surgeon, as distinguished from the physiologist, has much to learn from the amount of pressure, which will induce the appearance of the diastolic pulse; has he an equal amount to gain from the abolition of the pulse at the point where systolic pressure is reached and passed? If not, the possible increase of dangers, as the higher level is approached, would make it advisable to exercise caution. Be it understood that these questions are merely meant to be suggestive, and that the writer makes no attempt to answer them, because he feels that at present it would be difficult to do so. His desire is that those who employ these interesting and fascinating methods of study, should do so with their eyes open.

The Venous Pulse

This differs widely, and in many respects, from the arterial pulse: (1) It may be so well marked that a student would easily recognize it, or so feeble as to require the closest scrutiny for its detection; indeed, in some cases, the only available evidence is a rhythmic change in the depth of colour at a point where a vein makes a sharp bend on its course, no actual movement being detectable; any intermediate condition may be found between the two extremes above described. (2) It is best seen where a vein is either passing into the structures of the optic disc, or is making a sharp bend during its course. It may sometimes be observed on one or other side of

an artery which crosses the vessel, a matter to which we shall return later. (3) It may effect only one branch of the central retinal vein, or may be seen in a number of branches. It is probable that anatomical considerations have much to do with this peculiarity. (4) If very carefully studied in chosen cases, the pulse will be seen to resolve itself into a double movement: (I) An alternating increase and decrease in the breadth of the vein, and in the depth of colour of the blood column within it, and (II) a slow, emptying and refilling of a segment of the vessel—the changes in colour run along its length, away from the heart, as the vessel empties, and back toward it, as it refills. This movement can be particularly well studied in cases in which gentle pressure increases the venous pulse; as the force applied is increased, the length of vein affected is seen to increase with it, until quite half or even the whole of the part on the disc may be involved. One can never detect it beyond the papillary limit; this may be accounted for in three ways: (a) The vessels and their movements show up best on the optic disc; (b) in the retina they are embedded in the structures of that coat, whilst on the disc they lie comparatively unhampered (Bailliant); (c) the pulse has probably spent itself within the limits of the disc. Another impression may be given of the appearances presented by this phenomenon: The vein first seems to flatten to some extent along an appreciable length of its course, and then to empty itself along a part of this length. The emptying seems indeed to be merely an exaggeration of the flattening, and to affect the part where the pressure is lowest, travelling all the while in the direction of the higher pressure. (5) In a number of eyes the venous pulse is comparatively short. This is presumably due to the venous pressure being here relatively so high that the flow in the vessels is only interrupted during the height of systole, and even then only for a brief period. If, however, a gradually increasing pressure is exerted on the eye from without, the pulse, at first, often becomes more and more marked. The principal point that one then notices is that the duration of the phase of emptiness of the vein is markedly lengthened. This is obviously due to the ocular pressure having been so far raised by the pressure applied, that it is above the venous level for a comparatively long period during each cardiac cycle. The onflow of venous blood, at least in full current, then only takes place during the comparatively short period, when the venous pressure is at its highest, simultaneously with the arterial being at its lowest. (6) When, under pressure on the eyeball, a diastolic arterial pulse is produced side by side with a venous pulse, it will be noticed that these alternate; the vein fills as the artery empties, and *vice versa*. We shall return to this subject later. For the moment we may content ourselves with saying that this phenomenon is best observed in those eyes in which under digital

pressure a pulsating vein and artery run parallel with each other for some distance, immediately before their disappearance into the substance of the nerve head.

The anatomical and physiological factors in the causation of the pulse in the retinal arteries and veins

Much of the confusion that has clouded our understanding of this subject in the past has been due to the want of a careful study of the various factors concerned in the production of the pulse in the arteries and veins, and especially of the time-factors.

It is hoped that the diagram here given will materially help the student to arrive at a clear comprehension of the subject. It may look complicated, but a very short study of it will, it is hoped, prove that it is not so, and will serve to straighten out the tangle existing in many of our minds. The data have been mainly derived from Starling's "Principles of Human Physiology," and from Mackenzie's "Diseases of the Heart."

The vertical lines show tenths of a second, and the whole period of a heart beat is taken at 8/10ths sec. The columns should be read horizontally from left to right, starting with No. 1, which deals with the successive events that occur during a cardiac cycle.

No. 1. Here we have in turn the entry of blood into the auricles and ventricles from the veins for 0.4 sec., followed by the auricular systole, 0.1 sec., and that again by the ventricular systole 0.3 sec. During a fraction (shown in the diagram by the cross-hatching) of the first tenth of these 3 tenths of a second, the ventricle is closed in all directions, then the aortic valves open and the blood leaps out into the aorta.

No 2 shows the contrast between cardiac systole and diastole, as looked at from the point of view of the physiologist. It will be seen that the heart is at rest (in diastole) for 0.4 sec., and in systole for a similar period. This is, however, a very different thing from systole and diastole, as ophthalmologists look at the matter, when they talk about the systolic and diastolic pressures present within the eye during the course of a cardiac cycle. A confusion of these two different uses of the same terms does much to prevent us from obtaining a clear conception of many of the problems involved. We can see this when we pass on to consider the next column.

No. 3 confines itself to a consideration of the variations in intra-ocular pressure, so far as they are produced, or influenced by the pulse-beat in the retinal arteries. Each time that the heart beats it pumps an additional quantity of blood into the retinal artery and its branches; the beat is an expression of the fact that the arteries become fuller for a brief period and then return to their previous calibre; the entry of an additional quantity of fluid into the eye—be the source of that fluid what it may—will have the effect of

raising the intraocular pressure in a measure proportional to the volume of the fluid so added; similarly the escape of a quantity of fluid from the eye—whether by the veins, as in the case we are considering, or in any other way—will effect a corresponding reduction in the intraocular pressure. This is the true story of the variations in intraocular pressure, which are brought about by changes in the blood-pressure conditions within the eye. Admittedly, it is not the whole story, but with that we are not concerned at present. On the other hand, to envisage intraocular pressure as being determined and ruled by intraocular blood pressure, is, save with very important reservations, unsound in theory and contrary to the findings of research. With these remarks in mind, let us turn again to column 3, and note that the increase of intraocular pressure, due to ventricular systole, only prevails for about 0.26 sec. The exact fraction of a second lost, owing to the time it takes ventricular pressure to force the barrier of the aortic valves and to commence to distend the systemic arteries, is differently stated by different authorities. It is not important; what is important is that we should note that the reign of systolic pressure within the eye, is over in about 0.26 sec., as compared with the period of 0.4 sec., when systole is considered from the point of view of the contraction of the heart as a whole. The importance of this observation will be clear later on.

We can now pass on to mark in columns 4 and 5 the points in the time-schedule of the cardiac cycle, at which the aortic, and auriculo-ventricular valves close, and open, for the important events which are thus signified have an obvious bearing on our subject.

No. 4. The first feature of interest is emphasized in this column, and is one to which we have already drawn attention in connection with No. 1, namely, the delay in the opening of the aortic valves, after the commencement of ventricular systole, and the consequent shortening of the systolic period in the arteries. We shall advert to this again in connection with a leading feature shown in column 7.

No. 5. The point of outstanding interest here is the delay in the closure of the auriculo-ventricular valves after the commencement of ventricular systole. The period of delay is, for obvious reasons, not so great as that which precedes the opening of the aortic valves, but it is none the less an appreciable one.

No. 6 crystallizes in a practical form the lessons we have learned in the previous columns. It shows the varying pressures within the auricles during the cardiac cycle, with special reference to the events on the right side of the heart. It may be taken to be approximately representative of what is happening throughout the systemic veins, since presumably, every change in the auricular pressure will influence the column of blood right away back to the capillaries. The sharp contraction of the right auricle causes a steep

rise (a) in the auricular pressure, and at the same time checks the onward rush of blood from the veins, and, since no valves are found at the orifices where the great veins debouch into the auricles, some reflux of blood must occur, even though this is limited, by the contraction of the muscular rings round the veins, which accompanies auricular systole. Clear evidence of this reflux wave is to be found in venous pulse tracings from the jugular vein, and indeed, there is reason to believe that it is transmitted right back to the retinal veins, and that it there forms a factor in the production of the venous pulse. The sharp closure of the tricuspid valve causes a steep, if limited, rise in auricular pressure (c) immediately following the very sharp fall (b) which is due to the relaxation of the auricles, and which follows their contraction. Then a steady rise (d) takes place in auricular pressure, as the auricle fills with blood; this rise is momentarily checked (e) by the opening of the auriculo-ventricular valves, and the consequent rush of fluid into the now dilating ventricle; and so we come back to auricular systole with its sudden, sharp exacerbation (a) of the hitherto steady rise in pressure. It is usually accepted that every feature of these changes is transmitted from the auricles right along the columns of venous blood behind them. Thus we see that venous pressure is at its lowest during the systole of the ventricles, and that a steady rise takes place up to the ventricular presystolic moment of auricular systole.

No. 7 must now be considered. It shows, primarily, the variations in pressure level within the retinal arteries, and secondarily the influence of those variations on the intraocular pressure. Like the previous column it is merely diagrammatic, but it presents in graphic form certain large features of the prevailing conditions. It will be noticed that during cardiac diastole, auricular systole and the presphygmic period (that which precedes the opening of the aortic valves), the arterial pressure within the eye is steadily falling. Coincident with, and consequent upon that fall, there is a corresponding, steady, though slight, decline (f) in intraocular pressure. The moment the aortic valve opens, and the sphygmic period begins, there is a sudden rise (s) in the arterial pressure within the eye. The slight delay in the arrival of the pulse at the periphery may be for the moment left out of account. In consequence of the volume of blood thrown into the eye by this pulse, a slight, but perceptible rise of intraocular pressure takes place, only to pass away again during diastole, as the arteries once again empty themselves. We have thus alternating phases of low and high pressure introduced into the eye as a result of the pulse in the arteries. Be it clearly understood that neither the arterial pressure, nor the variations in it, are to be held solely responsible for the maintenance of the level of intraocular pressure. Our point is that the arteries introduce a pulse into that pressure level,

and that the timing of that pulse can be clearly seen in the diagram.

A comparative study of columns 6 and 7 has several lessons to teach. Before attempting to learn them, it is essential that we should remember that the variations in intraocular pressure introduced by the beat of the arterial pulse, and those in the venous pressure, whose causation we have already discussed, are merely factors superadded to the main problem of the maintenance of a comparatively steady intraocular pressure. The latter is established at a mean level by the amount of total fluid within the eye (both aqueous and vascular) and depends upon the rates of secretion and excretion within the eye, and upon the nervous control of the great choroidal reservoir of blood. For the moment we are not concerned with this main factor of the problem. Our interest lies with the cycle of changes met with as a result of periodic variations in the arterial and venous circulation of the retina. Having thus cleared the ground, we may return to consider these two columns.

(1) *The cause of the venous pulse* is diagrammatically demonstrated to us, for we notice that when the venous pressure level is lowest, the arterial is highest and *vice versa*. Thus we have coincidentally a maximum of pressure on the outside of the veins with a minimum of pressure within them in the seventh tenth of a second; whilst, on the other hand, in the fifth tenth of a second we have a maximum pressure within the veins, and a minimum pressure on their surface. These rapid alternations of pressure conditions at once explain the existence of a venous pulse.

(2) *The varying characters of the venous pulse* are likewise explained: When the pressure within the vein only exceeds that outside for the period of the sharp systole of the auricle, we find during the fifth tenth of a second, that brief flickering pulse, which shows itself only as a change in colour at the point where a vessel takes its bend. The long, slow, venous pulse, which we see in other cases, finds its explanation in the steady fall of the arterial pressure and in the steady rise of the venous pressure, culminating in a reversal of these conditions in the sixth tenth of a second. When the period, during which the pressure *within* the vein is above that *outside*, is a long one, the pulse is slow, and the area of vein affected is extensive; when this period shortens, the duration of the venous pulse and the extent of vein involved likewise diminish. The whole sequence of events can be grasped by a study of these two columns.

(3) *The appearance of the diastolic pulse and its variations under varying conditions of pressure* can be studied from column 7 alone. In order to present the matter as graphically as possible it may be permissible to carry the diagram a stage further, always with

the proviso that the demonstration is diagrammatic, and that mathematical accuracy is not in any way claimed: (a) Imagine that the base line which marks the seconds represents the normal level of intraocular pressure; (b) That the dotted line represents an increase in intraocular pressure (digital or otherwise) just sufficient to produce a distinct diastolic pulse; and (c) that the broken line represents the level of pressure at which the systolic pulse is all but extinguished. It will be clear that when the pressure reaches the second of these two levels, the pulse produced will be a very short one; it will represent a very brief interruption in the even flow of blood through the arteries, at the period when the intraocular arterial pressure is at its lowest, and, as shown in the diagram, it will be met with during the fifth tenth of a second, and in a fraction of the following tenth. The result will be that the diastolic pulse produced will have the quick stabbing appearance with which all who practise digital pressure for the purpose are familiar. Such a pulse may take only a fifth of the cardiac cycle, or even less, for its manifestation, that is a period of rather less than $1/6$ th of a second. When, however, the pressure reaches the third level, the conditions are reversed, for now the period, during which blood is passing, will only be during the seventh and eighth tenths of a second; during the whole of the rest of the period, the flow will be arrested. Once again, the pulse will be a very brief one, but this time the short period will be that when the artery flushes red with blood, and the long one that in which it blanches. At any period intermediate between these two, an intermediate condition of the pulse will be encountered, so that if the pressure is suitably graduated, each pulse period may be made equal to the other, or the one can be increased at the expense of the other. With these considerations before us, we are able to appreciate the phenomena seen as we increase or decrease digital pressure on the eye.

The Relative Frequency of Occurrence of the Venous Pulse

This was studied by Bailliart in a series of 66 cases. The writer has examined a hundred consecutive patients, and has somewhat extended the observations made by the French scientist. The conditions found fall naturally into a number of classes: (1) Those in which a spontaneous pulse is present; (2) those in which no spontaneous pulse can be detected, but one appears under the influence of pressure on the eye; (3) those in which no pulse is detected either with or without the influence of pressure.

STATISTICS DERIVED FROM THE EXAMINATION OF 200 EYES.

A careful ophthalmoscopic examination was made of the fundus of both eyes in 100 consecutive subjects, in whom no disease was

present. The great majority of them consulted the writer for errors of refraction. Any patients with definite signs of disease were excluded; so also were those who were intractable or nervous, and those, who by reason of high errors of refraction or from any other cause, did not lend themselves to accurate observation.

(1) We shall deal first with the 100 patients considered as such:

(a)	A spontaneous pulse was present in both eyes in	46 per cent.
(b)	Do. do. absent in both eyes in	40 per cent.
(c)	Do. do. present in one eye only in	14 per cent.

Total 100 per cent.

These figures require a little further consideration:

(a) Of these 46 per cent.—

The pulse was even in both eyes	...	...	in 28 per cent.
Do. uneven in the two eyes	...	...	in 18 per cent.

Total 46 per cent.

(b) Of these 40 per cent. in which a spontaneous pulse was absent—

A pulse was elicited by pressure in both eyes	...	in 12 per cent.
Both eyes showed no pulse even on pressure	...	in 20 per cent.
Pressure elicited a pulse in one eye only	...	in 18 per cent.

Total 50 per cent.

(2) We shall now deal with the 200 eyes themselves regardless of the patients they belong to:

(a) A spontaneous pulse was present in	49 per cent.
(b) Do. do. absent in	51 per cent.

Total 100 per cent.

This classification is capable of a further subdivision in accordance with the different effects of pressure applied to the eye on the pulsation.

(a) Of the 49 per cent. of cases which fall under this heading—

Pressure increased the pulse in	35.5 per cent.
„ stopped or diminished the pulse in	7.0 per cent.
„ had no influence on the pulse in	2.0 per cent.
This point escaped notice in	4.5 per cent.

Total 49 per cent.

The above observations are based on the application of light pressure only. The cases which evaded full observation were among the early ones of the series.

(b) The 51 per cent. of cases in which a spontaneous pulse was absent, are capable of a further subdivision into two equal groups in accordance with the influence of pressure, since when this was applied—

a pulse was elicited in	...	...	...	...	25.5 per cent.
and none was elicited in	...	...	...	...	25.5 per cent.

The whole of the figures given above are somewhat further complicated by the observation that, in a certain number of cases, an eye which shows no spontaneous pulsation on one occasion, may do so on another. An observation of similar bearing is that the force of the pulse can sometimes be observed to differ quite distinctly on different occasions, being sometimes stronger and sometimes weaker. This is in keeping with Bailliant's observations; indeed he went so far as to establish a separate category in his classification for the eyes in which the venous pulse came and went from time to time his figure for these was no less than 21 per cent. of the total examined.

Having to some extent cleared the ground by detailing the results of the observations above recorded, the occasion appears opportune for a statement in general terms of some of the lessons which may be learnt therefrom. A certain amount of repetition seems to be unavoidable, and for this no further apology need be offered.

As has already been said, it is a very difficult thing to form a clear idea of statistics relating to the venous pulse. In some subjects such a pulse is present in both eyes; it may then be so weak as to be almost imperceptible, requiring the closest attention for its detection, or so strong, that even a careless observer could not easily miss it; between these all grades exist. Then again, it may be present in every single one of the venous trunks on the disc, or it may be absent in one or more of these; it does not even follow that when present in all it is of equal strength in all. Indeed, this is very far from being the case, for where there are three trunks, it may be strong in one, moderate in another, and weak or absent in a third. We shall return at a later date to consider the influence on this pulse of pressure exerted on the veins by arteries which cross them obliquely or at right angles.

In other subjects, we find the total absence of a pulse in the veins of both eyes, or again, we may find a pulse in one eye and not in the other.

When we come to study the influence of pressure applied with the finger to the eyeball, the complications increase. It must be understood that in doing so, a definite line of conduct must be observed, if a comparison of the results obtained is to be of any value. The writer, throughout this series of experiments, applied digital pressure through the lower eyelid, or through the external

commissure, increasing it steadily until the flashing diastolic pulse was seen, and then stopping it, or at least being careful to go very little beyond this point, and then only in carefully selected and healthy eyes. It is of special importance that the early pressure should be light and gradually increased, and that the effects of light pressure and of the later heavier pressure should be carefully differentiated.

In a number of cases the application of light pressure will increase a pulse already present, or will elicit one not previously observed. Rarely, it will speedily abolish a pulse previously present. The continued increase of pressure will usually result in a speedy abolition of all venous pulse, but rarely an actual increase of the pulse may be observed, and this may go on right up to the point where a diastolic arterial pulse is produced. In such a case a very interesting phenomenon may be observed, if by good fortune the vein and artery lie parallel to each other and not too far separated for easy observation. The writer has been fortunate enough to get this combination in several cases. Under these circumstances, it can be clearly seen that the diastolic arterial pulse alternates with the venous pulse; when the artery is at its fullest, the vein is emptiest and *vice versa*. This is only what might have been expected when we remember the conditions prevalent in the eye at the time of the appearance of the diastolic arterial pulse. During the short period that the artery is stretched to its maximum, the intraocular pressure is appreciably increased, while the venous pressure is at its lowest; on the other hand, during diastole, the artery flattens and the intraocular pressure falls at the very moment when the venous pressure is at its highest; hence the appearance of the alternation of the pulses above recorded. This matter has already been considered when we were discussing the diagram illustrating the anatomical and physiological factors which cause the pulse in the retinal vessels.

Next, a word must be said of those eyes in which not only is no spontaneous pulse present, but none can be produced even by the application of digital pressure sufficient to elicit the flash pulse in the retinal arteries. That this is by no means a rare event can be judged from the fact that the author's statistics of 200 eyes show it in 25.5 per cent. This is not very different from Bailliant's estimate of 30 per cent. for the same phenomenon. That writer's explanation of the absence of a pulse, even under pressure, is as courageous as it is interesting and original. He believes that the venous exit pressure in such cases lies steadily below the intraocular pressure, and that the circulation is maintained through the flattened wall of the vessel, kept sufficiently open for the purpose by its own rigidity. He supports his contention by the following evidence: If we lower the pressure of the globe in these cases by pressure or massage, or

if we raise the venous pressure by making the subject hold his breath, after a strong expiration, the venous pulse will appear. To these observations of Bailliant's the writer would add a corroborative item: In a certain number of cases in which the venous pulse is absent, and remains so in spite of digital pressure on the globe, it may be caused to appear, for a short period, by complete relaxation of the applied pressure. This phenomenon can only be observed in a small minority of cases, but its appearance in them is unmistakable. The obvious explanation would appear to be that we have been able to raise the venous pressure for the moment above that of the intra-ocular pressure during the ocular diastolic phase, though it still falls below it in the systolic phase. After a very short delay the pulse so induced disappears, presumably through the fall of the venous pressure to its previous low level once again.

The present would appear to be a favourable opportunity to discuss certain other phenomena met with in the eye, when the veins fill with blood in the relaxation-phase which follows the application of digital pressure. We shall take these in turn.

(A) In one case, in which there was no spontaneous pulse, no pulse under pressure, and none after relaxation of the pressure, a pulse was at once produced when light pressure was reapplied to the eye, whilst the veins were still engorged with blood. Here it would seem as if the rise in venous pressure overshot the stage of near balance between venous and ocular pressure, and established, for the time being, a condition in which an increase of intraocular pressure was necessary before the pulse could be produced. This will be better understood by reference to the next class of cases.

(B) It is frequently observed that when a pulse is either spontaneously present, or is elicited by digital pressure, but stopped after relaxation of firm pressure, it can be easily made to reappear by a further application of light pressure. Obviously we have to do with a transient rise in venous pressure, or with a fall in ocular pressure, or with both. In any case the result is a disturbance of the balance between the venous and ocular pressures; the former is raised relatively to such a level that it is above the latter throughout the cardiac cycle. The application of light digital pressure once again brings the ocular pressure up so close to the venous pressure that it is now above it during the ocular systolic phase of the cardiac cycle, and below it during the diastolic phase.

(C) The following phenomena are very difficult to explain. They are possibly of all the more interest for that reason. In the right eye of a certain patient there was no spontaneous pulse, whilst the left eye showed a distinct pulse. On applying digital pressure, a pulse appeared in the veins of the right eye, and was strengthened in those of the left eye. Firm pressure was applied, and the retinal veins became engorged with blood; no pulse could now be observed

in them, nor could one be elicited by again applying pressure to the eyeball.

The Phenomena observed on Relaxation of Digital Pressure

If, after applying firm digital pressure to an eye, up to the point of producing the diastolic arterial pulse, the finger is removed whilst the disc is steadily observed, certain phenomena appear, which, so far as the writer is aware, have hitherto escaped record. The whole disc blushes pink, or even darkish red, the veins are markedly distended, and the venous pulse is abolished for the time being. After a variable period of from seconds to a minute or longer, the disc resumes its normal colour, the veins go back to their usual calibre, and the pulse returns. It is quite clear that we have here to do with an artificially induced rise in the venous pressure, which is sufficient to keep that pressure above the intraocular level throughout the phases of the cardiac cycle. It is a much more difficult matter to decide whether this change is brought about by a fall in the general intraocular pressure, or by a rise in the venous pressure due to a damming up of the blood in the vessels of the eye as a result of obstruction to outflow, or by both. It is quite likely, indeed it is practically certain, that firm pressure on the eyeball empties, to some extent, the great choroidal reservoir, whose importance in maintaining the level of intraocular pressure has been insisted on by Magitot. The effect of a partial emptying of this reservoir would be a proportionate lowering of the intraocular pressure, a state of affairs which would soon be righted with the re-establishment of the normal vascular conditions in the choroidal vessels. On the other hand, the obstruction, partial or complete, of the venous outflow would result in the raising of the pressure in the vessels of the eye right back to the arteries, and even in the arteries themselves; this might be expected to be attended by an increased storage of blood in the arteries and capillaries, which storage, on the moment of relaxation of the pressure, would lead to a flushing of the veins with blood. An observation of the fundus whilst pressure is being applied, would certainly not lead one to believe that blood was being stored up in the capillary circulation; such a view is contradicted by the pallor presented. On the other hand, it seems certain that the pallor of the central ends of the veins does not really indicate their absolute closure and the cessation of the flow of blood through them, as it has been assumed by some to do. We have several reasons for such a view: (1) There is our knowledge of the outstanding fact that an obstruction to a flow of this kind leads to a rise in the pressure behind, and that cessation of flow is only reached when the pressure throughout the organ is equal to or above that of the maximum systolic pressure; (2) these phenomena of flushings and palings can be produced by light pressure,

insufficient to provoke a diastolic arterial pulse, or even to stop the venous pulse, and sufficient only to increase the latter; this is clear evidence that the circulation through the capillaries has not really been stopped, for the capillary pressure must be higher than the venous; (3) a venous pulse may sometimes be seen right up to the production of a diastolic arterial pulse, becoming stronger and stronger, until the two pulses beat side by side. It would be difficult to explain such a phenomenon, if the venous exit flow had really ceased.

Looking at the question all round, the probable explanation of the fundus appearances observed on relaxation of digital pressure, is a dual one: (1) Intraocular pressure falls temporarily as a result of the emptying of the choroidal reservoir by the force applied, and (2) the arteries become over distended, as a result of the obstruction to the free onflow of the blood through the capillaries and veins, and empty themselves the moment an opportunity offers of doing so.

A series of eyes was examined with a view to study *the possible influence of the pressure of the arteries upon the veins on the production of a venous pulse*. Such an influence was suggested by Priestley Smith (this journal, Vol. II, pp. 264-5), as accounting for a certain number of cases of the kind, and the writer was therefore led to study the subject in some detail. This could best be done by selecting those cases in which one or both eyes showed a difference in the manner in which the arteries crossed the different veins. For the moment we may forego any discussion of the grounds for assuming that an artery crossing a vein necessarily does compress it. It is obvious that for the purposes which we have in view, the conditions required are that the different branches of the retinal vein in the neighbourhood of the optic disc should show widely different relations to the branches of the retinal artery. Where every venous branch is crossed under nearly similar conditions by an arterial branch, or when not one of them is so crossed, no materials for comparison exist. All such cases were excluded from the series, only those eyes being selected in which one or more vessels were distinctly crossed by arterial branches, whilst one or more of them showed no such relation to the arteries. In all, 35 patients were examined before it seemed certain that definite results had been obtained, and in these 35, 56 eyes were found to provide material for consideration and study.

It was quite frequently observed that when one vein was markedly crossed by an artery, and another was free from such crossing, the pulse was considerably more marked in the latter than in the former. Sometimes, indeed, the most markedly crossed vessel was the only one which showed no pulse. At other times the crossing or non-crossing of the veins appeared to have no relation whatever to the presence or absence of a venous pulse. Much less commonly, the

compressed vessel showed the most marked venous pulse, or the only such pulse recognizable. This result was so unexpected as to be a little staggering, and the actual figures may therefore be of interest. Of 56 eyes only 6 showed an increased pulsation in the cardiac segment of veins crossed on their surface by arteries, as compared with those not so crossed, while 22 showed a diminished pulsation. In 28 no possible difference could be observed in the pulse noted in the various veins, despite the great difference which existed in these eyes in this matter of the crossing of the veins by the arteries. In close connection with the last statement, a further important observation deserves to be recorded, viz., that in a number of eyes the pulse was found to vary greatly in different venous branches, being sometimes present in one, absent in another, and weak in a third, or showing similar variations of the same kind, despite the fact that the degree of arterial crossing was alike in all three. The same thing was also seen when not one of them was crossed at all.

These are but a few broad deductions from a mass of material, the main lesson running through the whole of which appears to be either that there is no connection whatever between the manner in which the artery is crossed by a vein and the venous pulse therein, or else that other factors enter into the case which, in a large number of instances, entirely obscure such action as arterial pressure may tend to exert.

It is of interest to consider for a moment the grounds for supposing that when an artery crosses over a vein pressure is necessarily, or even probably, transmitted to the vein walls in consequence of the anatomical relations between the two. *A priori*, it does not seem very likely that any such pressure would be exerted, nor does clinical observation lead us to believe that it actually is so exerted, always provided the vessels are healthy. The indentation of veins by thickened arteries must be admitted to be in a different category. This raised the suggestion that the increase of venous pulse, when present would probably be found to be correlated with a definite thickening of the arterial wall of the superjacent vessel. No evidence in support of this could be obtained, though a number of eyes in which retinal arterial sclerosis was present were carefully observed. This is perhaps not very surprising, since the venous pulse, presumed to be produced by an overlying artery, must depend to some extent on the amplitude of the variations in the calibre of the vessel, and we believe that such amplitude is at its lowest in vessels attacked by arterial sclerosis. (R. Foster Moore, *R.L.O.H. Rep.*, Vol. XX, 1915.)

Another point of interest—The venous pulse, seen in apparent connection with the crossing of a vein by an artery, is usually noted on the cardiac side of the line of compression; it may,

however, be most obvious on the capillary side of this line. The explanation afforded would be essentially the same in either case. The argument is, that as the artery expands and contracts, it alternately dams back and permits the flow of the venous blood in the subjacent vessel. Above the line of constriction every wave of arterial dilatation will give rise to a banking up of blood in the veins, while below it the segment of vessel on the cardiac side is permitted to empty itself. Again, with every constriction of the artery the blood that has collected behind the line of obstruction is again permitted to flow onwards, and with that the previously comparatively empty segment refills with blood. It would not, therefore, be difficult to explain a pulse on either side of the line of obstruction, provided that we met with such at all constantly. The question is, do we? Such work as the writer has been able to do on the subject causes him to doubt whether we do.

The Relationship of the Venous Pulse to Glaucoma

It has been generally believed that in glaucoma, we get an increase in the venous pulse. Another belief has gone hand in hand with this, viz., that a similar increase is met with after the use of mydriatic drugs. Bailliant has attacked the first of these dogmata and has contended that the view is entirely due to a confusion between the conditions which produce an arterial pulse and those which bring about venous pulsation. Briefly put, his argument is this: There is in the normal eye a large and definite difference between systolic and diastolic arterial pressure on the one hand and between diastolic arterial pressure and intraocular pressure on the other. When intraocular pressure rises to the level of diastolic arterial pressure, the arterial circulation suffers an interruption during a greater or less part of the cardiac cycle; the appearance of the diastolic pulse is therefore an evidence of the rupture of the normal relationship between the retinal arterial pressure and the intraocular pressure. On the other hand, the appearance of the venous pulse is merely an evidence that the venous exit pressure and the intraocular pressure are so closely balanced, that the small volume of blood thrown into the eye at each systole suffices for the moment to place intraocular pressure above venous pressure, whilst its escape during the arterial diastolic period suffices to lower the intraocular pressure below the level of the venous pressure. This, Bailliant argues, may occur equally under conditions of high, common, or normal pressure. However attractive such a thesis may be from a scientific point of view, it may safely be said that it is in opposition to a large volume of ophthalmological opinion, which bases itself upon clinical experience. The writer was among those who believed unhesitatingly that a marked venous pulse is a significant feature in glaucoma cases, and

he turned to his records confidently expecting a confirmation of this view, only to find that, in a number of his cases, the degree of venous pulsation had not been noted. This defect in his observations has since been remedied, but it will take some time before the records are sufficiently numerous to enable him to speak on the subject with the confidence and certainty that are desirable. If in a series of a hundred glaucoma cases the records of venous pulsation were available for comparison with those of the hundred normal cases dealt with at an earlier part of this paper, it should not be difficult to decide on the value to be attached to venous pulsation in the diagnosis of the disease. Although the available data are not sufficient to decide the question definitely, there are certain points which seem comparatively clear: (1) A strong venous pulsation is a not uncommon feature of the glaucomatous fundus. It is, of course, difficult, when speaking from limited statistics, to be sure that we have here to do with cause and effect, but the broad impression gained is that such is really the case. (2) In glaucomatous eyes in which marked venous pulsation has been observed before operation, later examinations following the successful relief of tension have shown its disappearance or its very marked diminution in a number of instances. (3) Notwithstanding all this, marked venous pulsation is certainly not an invariable feature of the glaucomatous eye; indeed, there may be a total absence of venous pulse in a globe whose tension is undoubtedly pathologically high.

Closely associated with the question we have just been discussing is the widely prevalent belief that venous pulsation is increased in normal eyes under the influence of mydriatics. A careful examination of a number of patients before and after mydriasis, has not lent uniform support to this suggestion. The origin of the idea is obvious: If we raise the pressure of an eye by indenting it lightly with the finger, we observe that in a number of cases the venous pulse, if already present, is increased, or, if not present, is induced. We are not unnaturally led to conclude that the same thing is likely to happen if we raise the pressure of the eye in any other way, as we are prone to assume, perhaps not always correctly, that we do when we bring about mydriasis. In any case the facts are of interest. The instillation of a mydriatic will sometimes cause a very marked increase in venous pulsation, while in others, the influence of the drug in this direction seems negligible. This observation gains added interest when we remember that it is in conformity with what we observe when we increase the intraocular pressure by pressing on the eyeball with the finger. If the writer has been correct in the suggestion put forward above that a pathological rise of intraocular pressure sometimes causes an increase in venous pulsation, and at others fails to do so, the interest deepens still farther. Running

through all these observations of the eyeball in health, in disease, and under the influence of drugs, there is on the one hand, a broad tendency towards obedience to definite physical and physiological laws, and on the other hand, an elf-like erratic element, which is extraordinarily puzzling, and which leaves on the mind the impression that there is some very important factor which we have hitherto been unable to identify or comprehend. May it be that this has to do with the anatomical and physical conditions which govern the escape of blood through the ocular tunics, or with the hydrodynamic conditions prevailing in the reservoirs into which the blood flows on its exit from the eye? If this question could be satisfactorily answered, it is possible that we should be nearer than we have ever hitherto been to the elucidation of yet another problem, viz., that of the elemental difference between simple and congestive glaucoma.

THE HALOS OF GLAUCOMA

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AN endeavour to obtain precise information as to the nature and character of the halos seen by glaucomatous eyes, and under certain other conditions, speedily reveals the fact that there is but little accurate information to be obtained from the existing medical writings on the subject. Apparently few have thought it worthy of careful study, and the very great majority of ophthalmic surgeons are content to record the observation of coloured rings by a patient, without troubling to investigate the matter any further. The writer feels that this attitude is a mistaken one, and believes that valuable results may be obtained from a systematic study of these halos. In order to clear the ground it must first be stated that the coloured rings may be seen subjectively by an eye under a number of different conditions: (1) In the presence of the corneal oedema that accompanies a rise in ocular tension (the true glaucomatous halos). (2) The coloured rings seen by an eye which has been treated with African arrow-head poison or with silver nitrate are probably due to a transitory corneal oedema induced by the drugs, and therefore may be assumed to be closely comparable physically with those of true glaucoma. (3) When the surface of the eye is covered with thick mucus, faint coloured rings may be seen round a flame; these disappear at once on washing the eyes and are not of serious import. The writer believes them to be due to the presence in the mucus of vast numbers of tiny air bubbles. (4) When the pupils of certain patients are dilated by a mydriatic, somewhat faint,