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 THE ARTIFICIALLY PRODUCED RETINAL PULSE

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It is a well-known phenomenon that if the disc and its neighbourhood are observed while external pressure is applied on any part of the globe, at a given moment the arteries in the disc start to pulsate. The artery empties and blanches corresponding to diastole, and fills up with blood corresponding to systole, or as Duke-Elder well puts it: "The blood column flashes across the disc." It is possible by greatly increased pressure to abolish completely the pulsation of the arteries on the disc—a procedure very unpleasant to the patient and rather dangerous. The veins on the disc are behaving in a peculiar way. When the first arterial pulsations appear on the disc, the venous pulse is accentuated, but usually does not change its character. With increased pressure on the eye, just before the arterial pulsation is completely abolished, the veins gradually become narrower, exhibit first a trickle of blood and then this trickle becomes beaded; finally the vein blanches completely. This is a summary of the general description of the artificially-produced arterial retinal pulse as given by such distinguished authorities as Baillart and Duke-Elder.

Some important details may now be added. Baillart (p. 28, ed., 1923) wrote that normally the retinal arteries look immobile. He mentions the opinions of Kummel and of Dr. Steyer, that they personally saw the pulsation of the light reflex (the retinal pulse), but not that of the central artery. I described some years ago that in red-free light one can easily see the pulse wave in nearly all primary and in some secondary branches of the central artery in a normal person, if the waves formed by those branches

are sufficiently prominent. The same applies to the rare but beautiful cases, where a primary branch of the artery forms a partial ring on the disc. This kind of pulse is easily explained by haemodynamics, and will not be considered in this paper. But if one applies very gradual pressure to the globe, one will first see a peculiar trembling of the arteries, difficult to describe, but very beautiful to behold. This trembling will be rhythmically intermittent and will correspond to the pulse wave. This is the moment, in my opinion, when the applied pressure plus the intra-ocular tension will be equal to the diastolic pressure of the central artery. The wall of the central artery will be unsupported; the diastolic pressure from inside will be equal to that from the outside, and the wall will contain that additional stimulus to extra contraction that is usually brought on by increased internal pressure, *i.e.*, this trembling is due to the vibration of the wall of the artery under the impact of the systolic wave. Let us increase our pressure a little, and the artery will start to collapse. This will start first deep in the disc on the proximal part of the primary branches, and it will spread to the periphery of the disc only with further increase of the pressure. The veins will become slightly narrowed, but their pulse will be accentuated. This is the level of the mean pressure. Baillart thinks that this is the level of the diastolic pressure; and that what we see is the vibration of the wall of the vessels. In my opinion we see the actual collapse of the artery.

Let us now increase the pressure still more, and watch the disc very closely. This flashing column of blood will reach the periphery of the disc and will be seen in the primary branches as well, not far from the disc, but with one great difference—the arteries on the disc are empty and, during the diastole, collapsed, to fill up in the systole; the same arteries on the retina will never collapse, although the arrival of the systolic wave can be actually seen. Why should it be so?

It is thus a strange fact that one can never collapse the retinal arteries, starting from outside the disc, even if the pressure is much higher than the systolic, and that the arteries on the disc will be completely empty and all pulsation will cease. These facts are not mentioned or described by anyone, except Baillart. He is emphatic about this—p. 39—“ Cette observation (des vaisseaux) doit porter sur les artères sur le disc de la papille et non au delà.” On page 51 he returns to that, and tries to explain it *en passant* “ nous avons vus que les artères retiniennes ne battant que sur le disque papillaire; cela tient sans doute à ce qu’au delà du nerf optique elles sont incluses dans la tissue retinienne. . . . Quoi qu’en soit. . . . ”

There is a definite law of hydraulics, that if pressure from outside is applied on any part of a ball full of liquid, inside this ball the pressure will be equal in all directions. The eye is such a ball, with a not very elastic outside membrane—the sclera—with a scarcely compressible vitreous gel. Therefore the retina and the vessels will be under equal pressure everywhere, *mutatis mutandis*. Is the turgor of this tiny and fragile membrane such that, caught between the sclera and vitreous, even with the cushion effect of the uvea behind it, it will be able to stand *per se* a pressure higher than the systolic retinal pressure, even though the blood-vessels run only in one or two layers of the retina? It is hardly conceivable, however, and it is contradicted by the fact that the patient will notice a darkening of the field of vision—the best proof that the circulation is at a standstill. The possible explanation of this strange inability of the observer to collapse the retinal arteries, so much narrower on the retina than on the disc—and there they are collapsed—might be looked for, in my opinion, in a different direction.

If the pressure inside the eye is equal, then why should the collapse of the artery be seen first in the centre of the disc, not simultaneously on the whole of the disc? Because here the vessels are nearest to the acute angle formed by them on the disc. The greatest vascular obstacle to the pulse-wave will be this 90° angle—this is why we see normally the pulsation of the vessels on the retina if the waves are sufficiently prominent—and what we observe is the stoppage of the pulse in the main artery of the optic nerve, when the pulse-wave has to overcome the difficulty of penetrating the arteries on the disc passing through this acute angle.

If my reasoning is correct, then what we measure is not the diastolic pressure of the central artery at all, but that of the ophthalmic artery. So thinks also Duke-Elder (Vol. I, p. 707). Let us now see what is going on under the cuff of the sphygmomanometer when applied on the brachial artery. When inflation of the cuff is started the air, as is usual with gases, will try to form a kind of round ball in the middle of the rubber, and from then will spread gradually to the sides, so that the artery will be first compressed in the middle, and from then to its extremities up and down, leaving some small corners at the upper end, where the artery will not be completely compressed. Even before the level of the diastolic pressure is reached in the cuff (say 70 or 80 mm. of mercury) the venous pressure (17 cm. of water) will be overcome. Sainsbury argued (on proper theoretical grounds, that the colloids are incompressible and blood is a colloidal mixture), that really the artery under the cuff remains permanently patent.

It would be so, if the blood from the artery could not escape. But it does, and it goes not back toward the heart, but forwards to the periphery. The artery does therefore completely collapse, as it is seen to be doing on the disc in the eye. The venous stasis therefore starts in the arm before the level of the diastolic pressure is reached. With increased pressure in the cuff a stream of blood will reach the brachial artery from the subclavian, and an increased outflow from the brachial artery will go towards the periphery. It will be accommodated by opening up more capillaries in the periphery and their increased effort to expel the extra blood into the venous system. The veins will swell more, the cyanosis of the lower portion of the arm up to the lower edge of the cuff will increase, and the venous pressure will rise enormously and quickly, being nearly equal to the systolic pressure in the arteries. The patient will complain of severe discomfort. Take now a large-bore needle, and pierce the ante-cubital vein—the blood will pour in a strong stream from the needle; it will start to pulsate later on, and the discomfort of the patient will diminish. The whole circle of blood circulation is engaged, the capillary bed is widely open, the pressure in all sectors is nearly equal. The capillary bed—the great buffer, and very active in its own right as well—is the deciding factor in what was discussed. This will explain what we see in the eye.

When the arteries on the disc start to collapse and fill up, as mentioned before, the incoming pulse-wave with still more increased pressure can be followed, say a disc-diameter, in the primary branches, but these branches never collapse. Why do they collapse on the disc? First, because the blood column is still able to force itself a short distance to the periphery within the primary branches, as can be seen by red-free light. Secondly, because I believe that in the disc the vessels are under increased pressure, being already caught between the substance of the disc and the special membrane covering the disc and described by Krückman. But further to the periphery the blood column cannot flow quickly, as it has to meet and to overcome the great regulator-buffer of the retinal capillary bed. We see in the eye what we only imagined going on under the cuff of the sphygmomanometer, but with one difference. There is always an escape from the venous blood through the veins on the disc. That is why the venous pulse is intensified in our experiments; the veins become narrower and then collapse completely, while the entrance of the blood into the arteries is stopped by our pressure. With increased resistance the pressure in the ophthalmic artery rises (a well-known phenomenon of local angiospasm—a defensive reaction), the increased force of the pulse wave is transferred from the arteries

to capillaries and from them—partly passively but chiefly actively—to the veins. It is this constant pressure on the inside of the capillaries—a very active force—that prevents the retinal arteries from collapsing, no matter how great is the external pressure. Theoretically, if a large-calibre cannula could be introduced into the veins in the disc, so that venous blood could flow out independently of the intra-ocular tension, the arteries on the retina would probably collapse.

In the retina we have to deal with a specially constructed pressure system with one narrow entrance and one narrow exit. Why do not the veins become blackish and tortuous during this experiment? Tortuous or flattened, as in glaucoma, they could become, but the time is too short, and they are not diseased, as they may be in glaucoma. They cannot become blackish, because there is no permanent obstruction, as in thrombosis, neither is the venous pressure raised in the ophthalmic vein, as in asthma, etc.

When the artery collapses on the disc, but still remains full of blood on the retina, just outside the disc, what prevents the blood from trickling back to the disc during the diastole, and so prevents the artery from collapsing? Even immediately after death it does not happen, but the blood eventually trickles back towards the heart later on. In a boy who died from advanced aortic insufficiency, I saw it happen within a minute or two of his death. The arterial system is full of blood, and the *vis a tergo* is a combination of the heart action and that of the muscular wall of the artery, distended by the blood column and the pulse waves, always tonically contracted and prepared to close the artery completely, if opportunity presents itself. It is therefore the contraction of the muscular coat of the primary branches that moves the blood ever forward away from the heart, and does not allow any back-flow.

Summary

A full description of the artificially-produced retinal pulse is given, and the impossibility is noted of collapsing the retinal arteries in contrast with those on the disc. This remarkable fact, hitherto observed only by Baillart, but not explained by him, is analysed and explained by the active buffer-action of the retinal capillary bed.

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