Summary

(1) The cocaineized eye tolerates subconjunctival injections of crystalline penicillin in doses of 100,000 units at 12 hourly intervals and doses ranging from 200,000 to 500,000 units at 24 hourly intervals.

(2) With doses of 500,000 units in 0.5 ml. of adrenalin solution 1:1,000, at 24 hourly intervals, preliminary results in hypopyon ulcer are distinctly promising, both as to a satisfactory end result and rapidity of cure.

(3) Though theoretically less efficient, retrobulbar injection of such doses appears to have clinical possibilities.

REFERENCE

SORSBY, A. and REED, H. (1948).—*Brit. Jl. Ophthal.*, Vol. XXXI, p. 528.

ON SYMPTOMS OF THE PULSE IN THE CENTRAL RETINAL ARTERY

BY

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My "Observations on the pulse and retinal arterial pressure" appeared in the May issue of the *British Journal of Ophthalmology*. The paper was translated from Polish into English in London and printed without my having read the proofs. Unfortunately the translation contains a great number of errors and in consequence parts of the original text have been misinterpreted. Taking advantage of the courtesy of the Editors of the *British Journal of Ophthalmology*, I am presenting here again the main points of my considerations on the problem in question. As my first paper was completed two years ago, and as in the meantime I have had the opportunity to make further observations which induced me to modify to a certain extent my former opinion, especially with regard to the measurements of arterial pressure by Bailliart's method, I consider it essential to take these modifications into account in the present paper.

On direct examination of the fundus oculi we notice two kinds of pulse: one of them I would call the physiological, the other the artificial pulse resulting from pressure on the eyeball. The physiological pulse may be perceived in a certain percentage of eyes examined. The first to describe this pulse was O. Becker¹ in 1872. Limited space does not allow me to submit here a critical review of Becker's observations; suffice it to say that they do not in all instances correspond with the real state of affairs. Ballantyne observed the physiological pulse in all cases which he examined with the aid of Gullstrand's ophthalmoscope. This pulse is based on the following phenomenon: when observing

the S-shaped sections of the off-branchings of the central retinal artery, we notice in them movements which I have called "rebounding movements." These movements occur simultaneously with cardiac systole and consist in a shifting of the bends of the artery in opposite directions. The greater the arterial bends, the more distinct will be these movements which disappear entirely towards the periphery. We do not perceive the phenomenon of dilatation and contraction of the artery which is regarded as an essential feature of the pulse. What then induces me to consider these rebounding movements as the pulse? In order to answer this question it is first of all necessary to explain the mechanism which produces these movements.

Rebounding movements are found in all arteries running an S-shaped course or showing peripheral contraction. Their occurrence can be explained in the following way: at the moment of cardiac systole the blood stream receives an impetus which causes an increase in pressure distributed almost simultaneously throughout the whole arterial system—according to Pascal's law—at an average velocity of 15 m. per second. This pressure disappears in the periphery owing to resistance offered by the viscosity of the blood. In contrast to the great speed with which the pressure is spread, the velocity of the blood stream is very considerably smaller, being only about $1/4$ m. per second. The increase in pressure produced by cardiac systole subsides in cardiac diastole. Thus rhythmic changes of the pressure in the arterial system occur: this is the pulse. Outside, the pulse manifests itself by movements of the artery which are visible on its S-shaped bends. In straight running arteries these movements are imperceptible. The changes in pressure which follow the course of the blood stream are manifest only by striking against the bent wall of the artery pushing it thus out of its course, as may be seen distinctly in the arteries of the fundus oculi. When speaking of Pascal's law, I mentioned that pressure is distributed throughout the whole arterial system; this would lead to the conclusion that the lumen of the artery should also dilate under the impetus of this pressure. But such dilatation, which according to the law of physics undoubtedly takes place, is absolutely imperceptible. Neither can it be peristaltic because this dilatation would have to be almost simultaneous throughout the whole arterial system, which is inconsistent with the idea of peristalsis.

In my former paper I quoted publications concerning this problem, therefore I shall not repeat them here. I would only like to mention A. Horst's study on haemodynamics², in which the author reviews the entire problem and refers to the literature on the subject.

In short, rebounding arterial movements in the fundus oculi are manifestations of pressure-changes in the blood stream and therefore also of the pulse which reveals itself in the same manner throughout the whole arterial system.

I now proceed to discuss the origin of the pressure pulse. The explanation for this origin given by v. Graefe a hundred years ago is still accepted. Graefe maintained that, on pressure on the eyeball, at a given moment this pressure will be higher than that in the central artery. At the turning-point of both these pressures the blood wave flowing into the artery will still have enough force to overcome this obstacle and to push through towards the periphery. This moment has been regarded as manifesting diastolic pressure in this artery. There is an analogy here with the examination of pressure in the brachial artery. This explanation of Graefe would be correct if the observation of the pressure pulse in the central retinal artery were accurate—but *it is not*. The phenomenon manifests itself differently. If Graefe's statement were indeed true, then the first movement perceived when pressing on the artery would be a dilatation of that artery, whereas the first movement consists in a sagging of its walls. This sagging of the artery is synchronous with cardiac systole, *i.e.*, with increased pressure in the arterial system. However, this sign of sagging of the artery simultaneously with cardiac systole is not the only one which we observe in these conditions. If we increase pressure on the eyeball we observe another phenomenon, namely, the rhythm of the arterial pulse will cease to synchronize with cardiac systole and become alternate, *i.e.*, there will be a dilatation of the artery in the fundus oculi simultaneously with cardiac systole and a contraction of that artery with the diastole. This therefore is the symptom which should be interpreted according to Graefe's theory. It is by no means easy to distinguish these arterial movements or the intervals of this pulsation when exerting pressure on the eyeball. To make this experiment it is absolutely essential that another person should feel the pulse, counting aloud. The experiment is best observed in persons whose pulse is slow, about some 60 beats per minute. When the pulse is quicker, the particular phases overlap and it is difficult to distinguish the moment of their origin.

To sum up, I wish to emphasize once more that in order to understand the symptoms of pressure pulse, it is indispensable to feel simultaneously the pulse of any other artery with the assistance of another person who counts the beats of that pulse.

I discovered this phenomenon of dual phases after the completion of my first article. As it throws new light on the whole

problem I have been compelled to reconsider my opinion published in that article.

What are the conclusions to be drawn from the above-described phenomena? If the first sign appears in the form of sagging of the artery as a result of pressure on the eyeball, then it is certain that this symptom was not produced by the blood stream which overcomes outside pressure exerted on the artery. Nor can it be manifested as an active contraction of the artery, or as a systole, because the artery is incapable of contracting or dilating rhythmically, as assumed by former authors, *viz.*, Graefe and Becker. It is true that the arteries can contract under the influence of the sympathetic nerve, but this contraction will not have the character of a rhythmical movement. The sagging of the artery may be due to an external force, the tension of which changes rhythmically. Such a force is the pulsation in the eyeball due to a rhythmic inflow of blood into the eyeball following cardiac systole. The sagging of the artery therefore would originate in the same manner as the so-called venous pulse. This theory is supported by the symptom of pressure pulsation in a blocked artery, that is to say, in conditions where the flow of blood into the artery is checked.

To summarize, the first sign which becomes manifest when slight pressure is exercised on the eyeball and occurs synchronously with cardiac systole, is produced by an external force, namely, the pulsation of the eyeball. The second symptom, on the other hand, which does not synchronize with cardiac systole when stronger pressure is exerted on the eyeball, occurs in the way described by v. Graefe.

In view of the above, what is to be said of Bailliant's method of measuring central arterial pressure? According to this method, the pressure pulse is the indicator which, however, deviates considerably from the physiological conditions of blood circulation in the arteries of the eyeball, and therefore cannot answer the question concerning the actual systolic and diastolic pressure in the retinal arterial system. Such an answer could only be obtained by a direct examination of the pressure; this experiment was made by Duke-Elder on cats' eyes with the aid of a micromanipulator. Nevertheless, on the basis of Bailliant's method we are able to form an opinion on the pressure in the retinal arterial system, although the results of this method are only of relative value, as are also the results of examining pressure in the brachial artery by way of well-known clinical methods. The indicator for defining the minimal pressure cannot be the first movement of the artery, as Bailliant suggested, but the second which appears alternately with cardiac systole.

Thus when applying Bailliar's method, the pulse of any other artery must be examined simultaneously, and the moment of reversal of the rhythm is to be regarded as the indication of minimal pressure. The difference between systolic and diastolic pressure in the central retinal artery will then be much smaller and will no doubt correspond with the actual state of affairs. Taking into account that in the off-branchings of the central artery rebounding movements are usually accentuated only very slightly, we may assume that the differences in pressure in these arteries will also be very small. It is of no special importance to which artery the measurements by Bailliar's method are applied, as the results can be only relative, although their clinical value will be indisputable.

I believe that the application of this modification of Bailliar's method in determining the indicator will help to eliminate to a considerable extent the disparities in the results obtained by various authors.

BIBLIOGRAPHY

- BECKER, O.—*Graefe's Arch. f. Ophthal.*, Vol. XVIII, 1872.
HORST, A.—Problems of Haemodynamics, *Bulletin de la Société des Amis des Sciences de Poznan*, Série B, Livraison VIII, 1947.

A CASE OF PIGMENTED LEIOMYOMA OF THE IRIS

BY

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MRS. S. consulted me on August 15, 1944, with regard to something wrong with her left eye. This proved to be a tumour, involving apparently the whole breadth of the iris and extending over the lower and inner third of its circumference. The growth was encroaching on the pupil, rendering the pupillary margin convex instead of concave in the part involved. In shape the tumour was like a bean and in colour purplish black. The surface was smooth and the margins clearly defined. The growth was in contact with the cornea for about a quarter of its extent and some secondary change in the cornea could be made out.

The patient complained that this mass was increasing in size and becoming dark in colour, that her sight was not so good and that the eye was diverging. This change had been noticed during the past year. The patient's own story leaves one in no doubt