"Every theory, my friend, ages; eternally blossoms only the golden tree of life."  

Goethe.

The student of ancient Greek history is always struck by the thought of how near the philosophy of that clear-minded, light-hearted people, was to the modern conception of the universe, divided as they were into numberless miniature states, engaged in continual warfare with one another, and in perpetual danger from within and from without. Everything flows, παντα ρει: these two words of Heraclides formulated more than two thousand years ago form one of the most striking discoveries in human knowledge, the dynamic state of the universe. There is no end and no beginning; all beings and all events form one continuous chain, each one supplementing the other. The limited capacity of the human mind cannot easily grasp this great truth. Man must break the chain, must examine it bit by bit, must stop for a moment so that he may concentrate his analytic spirit to try and discover the mystery of mysteries—the secret and source of life. And very often in doing so the explorer loses the perspective of the whole picture, thinking that in his drop of water is truly and entirely reflected the magnificent sum of the world. This is equally true of the human microcosm. The luxurious and brilliant growth of
modern bacteriology and biochemistry lessened in the mind of the medical man the importance of purely clinical investigation and diagnosis based on the result of personal experience, knowledge, and sometimes even intuition. This tendency to find a firm basis for his knowledge is overdone; he underestimates his part in the diagnosis, where both himself and his patient are living beings and medicine is still an art and not only a science. One enthusiast tests the blood for nitrogenous products, another for chlorides, a third for sugar, and each claims to have in his hands the solution of a particularly important pathological problem, ignoring the clinical picture and often even supplanting it. We forget that the patient’s body is the finest and best equipped laboratory in the world. In this connection I cannot help feeling that the wonderful properties of the ophthalmoscope and sphygmomanometer, especially when they are combined together, are undervalued; and how rarely the ophthalmoscope is used in a proper way, even by the consulting part of the profession, which has the knowledge, the time and the opportunity! A case of hypertension was demonstrated recently at the Royal Society of Medicine. Every possible test was performed on the patient and discussed by the participants of the meeting. But not a word was mentioned by any of them regarding what they saw in the fundus, and I am under the impression—sit venia verbo—that this part of the clinical examination was simply omitted. At most the fundus is examined by the physician himself, often with an undilated pupil and not in a dark room. This is not the proper way to examine the details of the retina, and a fundus, often decidedly pathological for a trained observer, will be passed as normal.

Everybody theoretically bows before the ophthalmoscope; nobody dares to dispute its significance and opportunities; and enveloped by this general piety, the instrument is rarely used or not used in a proper way, by the medical profession, outside the small number of ophthalmic surgeons. And the latter use only the ophthalmoscope and not the sphygmomanometer, two instruments intimately connected and inseparable in the clinical examination of the reno-cardio-vascular system. Though the sphygmomanometer has its limitations, yet it gives an arithmetical reading of a very variable clinical detail, it supplies an important and timely warning, which no palpating finger can ever furnish. It is of great value, but it is not everything. It is impossible to make any deductions relying only or even chiefly on the sphygmomanometer, for the purely mechanical interpretation of arterial hypertension is utterly misleading. This is where the ophthalmoscope becomes important. And if the feeble voice of this paper will help in the slightest to move medical opinion in the direction of more detailed clinical examination, not apart, but together with
laboratory examinations, my modest effort will be amply rewarded. Even among ophthalmologists the examination of living retinal vessels did not attract much attention. All forms of retinitis, exudations, haemorrhages and gross changes have been examined, classified and described so fully that even the modern attainments of the slit-lamp do not lead us much further. But in the field of the examination of the retinal vessels much is still undone. The pioneer and best investigator, giving us the most of our small knowledge, was the late Marcus Gunn. And I must state here that the more I work and think in this direction, the more I admire him for the accuracy of his observations, and his clarity of thought and expression. He formulated rules and types from the midst of an enormous conglomeration. His deductions, on the whole, are correct, and the minor differences can easily be explained by his use of the ordinary ophthalmoscope instead of the self-luminous one. His papers read to the Ophthalmological Society of the United Kingdom and his portion of the Medical Ophthalmology of Gowers, in which one immediately recognises the spirit of this precise thinker and able investigator, especially in the first 28 pages, are still, as far as I am aware, the most abundant and best documents on the subject. Foster Moore’s excellent book must next be mentioned. In the prodigious volumes on the retina and optic papilla of the Graefes Saemisch Handbuch, I found only half a page dealing with sclerosis of the retinal vessels and giving chiefly a compact review of Gunn’s opinion on the most interesting and enigmatic phenomenon of the sclerotic fundus, arterio-venous compression; the opinion of Röhlmann is also quoted. Among French authors some remarks will be found in Poulard’s Ophthalmology. Bailliart in his Retinal Circulation writes of it, but really, sine ira et studio, he is not as unbiased in his views as Gunn. Moreover, in his book, many pages of which are written with the brilliancy of l’esprit gaulois, he makes many insufficiently proved statements, some of which are not correct or may be accepted only cum grano salis. De Schweinitz added some valuable information with his “corkscrew” vessels. Why, then, has so little been done on this subject? We see with the ophthalmoscope the circulation in a highly organised nervous tissue. It is quite probable that there is a close analogy between the retinal and cerebral vessels. The retinal vessels are small, about 1/100 of an inch in diameter. Therefore, they will show early any pathological process in their walls. But the examination is extremely difficult. The change may be so small and so fine that it may be easily overlooked. Although gross retinal changes are easily detected, for the vascular changes one has to look carefully and to keep in mind the possibility of their presence. Every vessel has to be scrutinised with
close attention, even the smallest ones. It is necessary to differentiate between pathological changes and congenital peculiarities. It is essential to use the ordinary and red-free or Hallauer's light to see the fine, especially the perivascular changes, and the intensity of the beam of light must be increased or decreased for the exploration of difficult points. It is not always necessary to dilate the pupil, or, better said, a trained observer will soon decide if such a dilatation is necessary. But the darkened room is essential. I advocate also a full and detailed description of the fundus. We see the retina only as it is at the time of the examination; it changes as everything else in nature. And, again, the changes are so minute that they will certainly be overlooked, if another ophthalmic surgeon, or even the same one, examines the patient after some time has elapsed. It usually takes me two sittings to examine completely a markedly sclerotic fundus. This kind of retinal examination is really of the nature of microscopical work, and I would ask for the same fullness of detail in description and explanation as is usually allowed to a histological preparation. The drawings were made to illustrate my dissertation and help make a living picture of the fundus from the dry description of details; and indeed it would have been worth while to prepare many more of them. To be as impartial as possible, I asked Mr. Hamblin's permission to look through his rich collection of drawings. I chose a few of those most suitable from the point of view of this paper, and approached the various ophthalmic surgeons for permission to use their drawings as illustrations for this paper. I feel it my pleasant duty to express to them my gratitude for their kindness. My especial gratitude is due to Mr. A. H. Levy for his kind advice, for discussing and verifying some of the cases, and for invaluable help by revising and editing this paper; and also to Dr. M. Cohen for correcting the manuscript.

In my first paper (Brit. Jl. of Ophthal., Vol. XI, p. 489) I described and discussed normal, mildly hyperplastic, advanced hyperplastic, and diabetic fundi. In this paper I will study only the sclerotic fundus (involutionary arterio-sclerosis, hyperpiesis, and diabetes). Not one case of interstitial nephritis is discussed or described here. My cases of sclerotic fundi numbered 464, i.e., over 900 fundi. Many phenomena as, for instance, the centripetal deflection of veins and aneurysms by crushing, have not been previously described anywhere, so far as I am aware. Many facts regarding arterio-venous compression, as often observed by me, simply do not fit in with existing theories. The gradual development of this condition, so to speak, under my eyes and under my continual supervision, led me to certain conclusions, each of which may be proved by some case of mine. On the other hand, in talking of essential hyperpiesis and even in-
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Evolutionary arterio-sclerosis, I tread on difficult ground, badly explored, full of uncertainties, about which still rages a storm of controversy by authorities greater than myself. An explosion may bring to the surface new facts which will shatter my theories. Let us be consoled by the wise words of Goethe, which I have taken for the motto of this paper. Let us hope that the friendly critic in his review will be able to see in the facts which I have collected and ascertained, a few small leaves from the golden tree of vascular life. *Sapienti sat.*

The Normal Fundus

Disc.—Our anatomical knowledge of the retinal vessels and their supporting tissue, of the disc and its minute structure, is still incomplete. I can refer here to the opinion of Prof. Krückmann, that the technical difficulties in histological preparations of the retina are enormous and are still not overcome. More amazing is the fact that clinically the minute structure, especially of the disc, was not sufficiently studied, although constantly seen by numberless ophthalmic surgeons. *Habent sua fata fundi!* Many discs have been faithfully reproduced in drawings, especially in pathological conditions, with a full description of details appended, but certain particulars were not explained, for instance, why veins crushed by the arteries on the disc are not changed at all by them when crossed on the retina. Or, you can see reproduced arterio-venous compression and no explanation given. In Frost’s excellent atlas, I found a few examples of this, e.g., in Drawings 14, 74, 75, the fixation of the vessels at the crossing points, with venous compression, is faithfully reproduced, but no description or explanation of the phenomenon is given, although usually Frost’s description is very full and detailed. In the commentary on drawing No. 80 he describes the phenomenon of arterio-venous compression, but does not explain it. In Fig. 84 he simply points to a gap in the vein where the artery passes; he faithfully reproduces the white lines which accompany the sup. temp. vein, especially where it crosses the artery, but he does not explain or even mention them in his description. In Fig. 86, the sup. temp. vein is crossed by the artery, and is centripetally deflected without any mention in his description of this remarkable phenomenon. In Fig. 87 is reproduced the perivasculitis of the sup. temp. vein, but not mentioned in the text. Of modern text-books, I shall mention De Schweinitz. In plate No. IV—albuminuric retinitis—the sup. temp. vein is depressed, crushed and “banked” by the artery at the three crossings, but the author does not describe or explain this important
phenomenon. It means only that Frost and De Schweinitz either forgot to describe this or else overlooked it. But the drawing is a true reproduction of what they repeatedly saw in the fundus and needs explanation. Further on, the reader will see that in discussing these phenomena I have come to some conclusions, only indirectly connected with the purpose of this paper, but pointing to a solution of some undecided anatomical questions.

The vessels of a normal papilla, such as that of a child, show some particulars worth describing. They are very important for the proper understanding of pathological conditions—the basis of pathology being anatomy and physiology. In the trunk of the optic nerve both central vessels travel together in the middle of the nerve, surrounded by fibrous tissue, which forms a sheath around them (Bridgett). As Weigert pointed out, and as I shall ask the reader to keep in mind during the rest of this paper, the vessels are after all foreign bodies in the central nervous system. And, as in the case of a foreign body, they are surrounded and separated from the proper nervous system by connective tissue. The lymphatic vessels run along the arteries and veins, and their walls, if they exist as a separate entity, blend together with the adventitia and form what has been called, and what I shall also call, a "perivascularis." The sheath of the optic nerve must have elastic walls to allow of the enlargement and partial collapse of the vessels during their pulsation. The lamina cribrosa embraces the vessels more closely. This is evident from the fact that the vein is often enlarged on the papilla just before entering the lamina—it looks like a leech—although the subject is a young person without any sign of sclerosis. Frost mentions this fact on p. 119. Pathology also points to the fact that this distension of the vein means an actual impediment to the venous outflow. It is the customary place for a venous thrombus. It sometimes happens that with the ophthalmoscope one can follow the central vein deep down into the papilla. It is, of course, impossible to decide clinically what kind of a lamina exists in such cases, but it is probable that it is an unusually thin one. If the vein happens to be pulsating, or if it be made to pulsate by finger pressure, you can see its sheath definitely segregated by white lines from the surrounding tissue of the disc. The sheath fills up with the interruption of the venous outflow, and partially, but not entirely, collapses with the emptying of the vein in which a tiny stream of venous blood is still visible. It means that this sheath is very elastic. Its contour is often not a straight one, but of a zigzag form.

On the disc the vessels are very often accompanied by white lines. They are very beautiful, if strongly marked, especially on the rosy discs of children. Sometimes they are very fine and can
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be seen better with red-free light. These white lines were noticed and described long ago as, for instance, Gowers, Römer, etc., and in text-books the reader is warned not to confuse them with pathological conditions; but authors are rather reticent in their explanation of the nature of these white lines. Some simply mention them; others speak of their probable structure. Juler, on p. 255 of his text-book, says that they are "due to a prolongation of the connective tissue, which supports the vessels while in the substance of the optic nerve, beyond the lamina cribrosa. The tissue does not extend beyond the margin of the disc." Let me state immediately that it does sometimes extend beyond the disc, usually as a "tongue" accompanying the large vessels and finishing rather abruptly a short distance from the disc. This may be seen in many normal fundi and Fig. 6 is an illustration of this particular point. The vessels do not show any particular sclerotic changes, except perhaps a small banking of the sup. temp. vein between the first and second crossings, but the white lines are strongly represented; they accompany the vessels on the disc and on the retina near the disc. Although there is some disease of the macula these lines are physiological. As a contrast, plate 8 shows pathological white lines. On the living fundus the difference is more striking. The physiological white lines are usually whitish or greyish and finer; the pathological ones are distinctly white, glittering and clearly defined. They usually accompany the vessels from the disc far along the retina, and are not absent at crossings. There can be no doubt or difference of opinion as to their structure as shown in plate 8. They are the result of perivasculitis, an inflammatory process along the vessels.

I saw in one patient the physiological white lines on the disc and, in addition, a narrowing of the vein when entering the lamina. The sup. cent. vein was composed of three veins, all joining together on the disc, the third vein entering the common vessel from behind. The vein was in the form of a large pear, the narrow end of which was seen deep in the lamina, very constricted. Sometimes the white lines are much stronger in one particular part of the disc, and then they present a very striking picture indeed. What do these white lines represent?

I quoted above the opinion of Juler, but in Graefe-Saemisch, Vol. VII, p. 22, one finds a most surprising sentence: "With the lesser degrees of loss of translucency of the vessel-wall the blood column is seen accompanied on both sides with whitish lines, sometimes only on the papilla, or more seldom, in the immediate neighbourhood of their appearance on the disc, sometimes far away in the retina." In view of this statement it was worth while describing the physiological white lines, which have nothing to do with loss of translucency and are often seen in perfectly normal
fundii. True enough, after confusing different phenomena, Prof. Leber farther on makes a remark, that it is possible that the white lines at the point where the vessels emerge from the disc are not pathological; they represent not the wall of the vessel, but only the surrounding tissue. I shall state here briefly that, in my opinion, in the early stage of retinal sclerosis the white lines appear first of all at the crossing points of the vessels, in 44 per cent. of the first group and 65 per cent. of the second, more advanced cases (vide analytical table). The white lines on the retina are very seldom seen (in 2 per cent. of the first and 4 per cent. of the second). White lines on the disc are found in about the same proportion in both groups, 46 per cent. and 40 per cent. The best proof that they are physiological and have nothing to do with sclerosis is that they do not increase in the second group, and in this respect, they stand out alone from all other signs of sclerosis. The difference between Leber's opinion and mine is therefore considerable.

Salzmann does not describe these white lines as a separate item. He states only that the retinal part of the optic nerve does not contain any mesodermic elements, except the vessels with their adventitia (p. 106). He is entirely correct. A priori, the opinion of Juler must be wrong. The lamina cribrosa affords so little room that the nerve fibres lose their myelin sheaths to enable them to pass through it. The vein is distinctly narrowed. The artery alone is not affected. It would be logical to think that the vessels passing through the lamina are denuded also ad maximum, or at least deprived of the rich connective tissue which surrounds them at the margin of the optic nerve. Römer (p. 290) thinks that "the whitish lines around the retinal vessels may be a congenital anomaly. These white brilliant lines, accompanying the vessels, ought to be explained as a prolongation of the connective tissue of the lamina cribrosa." In Fox's and Poulaud's text-books I did not find a word on this subject, or possibly I overlooked it. Fuchs's opinion is very similar to that of Römer. It is surprising that Baillart never mentions or describes them in his book, but Gunn, in Gowers' Medical Ophthalmology, describes and tries to explain them. On p. 14, "Such an appearance may often be seen near the centre of the disc, where the amount of such tissue varies much, apart from any disease. . . . It has been thought that this tissue is sometimes a result of chronic congestion of the disc, but the condition is so common that its presence alone is of little significance. When a vessel curves over the edge of a hollow central cup and is seen foreshortened, the white tissue of the wall often appears as a ring around the blood column." The reader can appreciate that Gunn understood that it is the wall of the vessel we see.
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I am inclined to think, however, that the explanation may be a different one. The retinal vessels lie among the nerve fibres, being surrounded by their adventitia and neuroglia. Approaching the disc, the largest of the nerve fibres are thicker in the sagittal diameter. They all converge fan-like from the periphery to the centre to pass through the disc and lamina cribrosa. According to Salzmann they do not lose their neuroglia, and so they overlap one another. The optical result will be that the adventitia with thickened neuroglia will be more refractory to light, which means it will be more visible. The adventitia itself is naturally thickened from the periphery to the centre, and it is thickest at the disc. The adventitia is rich enough even in the veins, their one point in common with the arteries; otherwise the vein is easily collapsible, because it is deprived of the tunica muscularis. Add to these two factors, the optical contrast of a rosy or whitish disc, and they will be even more marked. I mentioned previously that sometimes you can follow the canal of the central vein deep into the papilla and you can see its wall distinctly segregated from the disc proper. In a few cases I could see the wall of this canal becoming white lines around the primary venous branches on the disc, or better said, blending completely with them. I mean that those white lines are the point where the adventitia (perivascular) of the vessels is separated from the nervous tissue of the disc. Remember the statement of Weigert! It is a fact long known that the retinal arterioles and arteries are surrounded by a sheath of glia and thus isolated from the nerve fibres. That this is a very complex structure was shown by Krückmann, and will be fully discussed when speaking of the normal retina. Here it is important only to realise that, fine and thin on the arterioles, this segregating membrane naturally increases with the size of the vessels, reaching its maximum on the disc; and I cannot decide yet, and, as far as I am aware, it is not yet definitely known, whether it ceases when reaching the lamina cribrosa, or, transformed in the "connective tissue meniscus" of Kuhnt, passes through the lamina surrounding the central vessels to be blended and transformed in the fasciculus which surrounds them in the trunk of the optic nerve, and which was recently closely examined and described by Bridgett. It means that the vessels are running on the disc through separate channels, formed by the neuroglia and their own "perivascularis," and that these channels are often, but not always, visible. These channels are elastic ones and so allow the pulsation of the vessels. But still they exercise a definite pressure on them. The artery with its strong muscular wall can withstand this pressure easily and is not affected by it at all, at least, ophthalmoscopically, just as it is not affected by the pressure of the lamina cribrosa. The vein with
its weak and collapsible wall will show this pressure when entering the lamina cribrosa. It is distended on the disc where it goes through a narrow opening in the lamina. It is larger in the normal fundus just on the border of the disc, i.e., on the retina, before entering the disc. It is such a usual phenomenon and so clearly physiological that I did not consider it in my classification. In children and healthy young people, if you follow the vein carefully from the periphery towards the disc, you will often see the veins or vein distinctly narrowed as soon as they reach the disc, and remaining so narrowed, or even more so, until they reach the lamina. It is not the abrupt "banking" of arterio-venous compression; it increases gradually, and it is very well demonstrated by the nasal superior branch on plate No. 25, and by both vena inferior and superior on plate No. 2, and by the sup. temp. vein on plate No. 3. Especially noteworthy is plate No. 4—one can see this gradual narrowing of the inf. cent. vein which is formed by two branches, and which is in the middle of the disc narrower than either of its constituents. The sup. nasal vein evidently would be the same, but it is crushed by an artery, deflected centrifugally and "banked." The reader can see immediately the difference between the gradual narrowing of the lower vein and the abrupt difference in the upper one before and after the crossing. Sometimes all the veins are narrowed, sometimes only one or two, and the rest are not affected. The pressure on the vein is exercised from the sides in the horizontal meridian; if it were in the sagittal direction the vein would be flattened, as sometimes happens in cases of glaucoma or even in normal eyes on the disc itself, as will be described immediately. It is possible, of course, that the veins are narrowed, but become deeper, i.e., they change their larger diameter from the horizontal meridian to the sagittal one; but it is impossible to decide this point with the ophthalmoscope.

The importance of realising the existence of these strong dermic channels will be easily grasped if we turn for a moment from the normal fundus to the pathological one. On the disc the vessels are crowded together; they are the largest; they press against one another; they have to squeeze through the lamina; the pressure of the blood from within on their walls is higher than in the vessels on the retina, because immediately behind lies the lamina, a strong obstruction for the blood-flow, as described previously. Therefore, all conditions for a haemorrhage are more favourable on the disc than on the retina. Gunn, in Gowers' book, thinks that a haemorrhage from the retinal vessels on the disc is so rarely seen because they are so well supported by the intraocular pressure. There is some truth in this idea, but it cannot explain the well-known fact that haemorrhages on the disc are very rare.
Sclerotic changes in comparison with retinal ones. The unfavourable conditions of the disc affect the inside of the vessels, and the lamina is the usual spot for venous thrombosis. On their outside the vessels are so well supported by their meningeal channels that a haemorrhage per rhesis is practically impossible, and per diapedesis is also very difficult. One of the common haemorrhagic conditions in the fundus is undoubtedly thrombosis of the central vein or its branches. "Most of the haemorrhages are on the retina, especially immediately near the papilla, and the disc itself is comparatively free. The haemorrhages are constant. They occupy nearly exclusively the peripapillary area," Poulard p. 1043. The same cause, strong support for their walls, ought to make aneurysms on the disc more rare, although the conditions for their occurrence are more favourable here than on the retina. In my cases I had only two aneurysms on the disc and four on the retina, too small a number to show anything. Most of my aneurysms were caused by the crushing of one vessel by another, as is shown by plates No. 30, 31, 32. The vessels, especially the veins, are much more crushed on the disc than on the retina; therefore, one would also expect more aneurysms on the disc; but this is not true so far as my cases are concerned. Of course, aneurysms of another type, secondary to arterial obliteration, will be seen more often on the disc than on the retina. I am under the impression that in Gowers' book Gunn also thinks that miliary aneurysms on the disc are extremely rare.

When the vessels emerge from the lamina cribrosa they may be subjected to the same pressure as in the retina, or to a pressure which is higher than that in the retina, but lower than that of the lamina. The narrowing of the vein as described above is in itself a proof that on the disc they are under greater pressure than on the retina. This pressure must be less than that of the lamina, otherwise we should not see the venous pulse most clearly (though not exclusively) on the disc. We have much clinical evidence that on the disc the vessels are under considerable pressure from above, in the sagittal meridian, squeezed so to speak, between the lamina cribrosa behind and something else in front. In children and young people alike, in many instances, in a perfectly normal fundus, the arteries cross the veins on the disc and crush them severely. The same vessels cross again on the retina, or sometimes in the immediate neighbourhood of the disc, without any change in their calibre and without any white lines accompanying them at the crossings. Evidently then something else besides the intraocular pressure presses the arteries on the underlying veins in a normal eye. Usually in such cases the white lines are also more strongly marked. On the other hand, although the veins are severely crushed on the disc they are very seldom displaced by
the arteries. It is a striking feature in many advanced sclerotic fundi to see the veins at the retinal crossings severely deflected and crushed, but on the disc only crushed. On the other hand, even in children in a normal fundus a deflection of the vein on the disc, usually with crushing, may sometimes be seen. I mean to say that such a deflection of the vein by an artery on the disc is usually congenital. It is not a sign of sclerosis, it cannot be produced by sclerosis, but may be aggravated by it. The lateral channels fix the veins firmly and do not easily allow any displacement, although they do allow the crushing, i.e., pressure from above. In the sclerotic eye, on the other hand, one would logically expect the crushing of the vein to be stronger on the disc than on the retina. The smallest artery, nearly invisible, may crush severely a large vein on the disc, whereas on the retina a large artery does not affect it. Such is the case in plate No. 5, where the sup. temp. vein on the disc is crossed by a thin reddish streak (arteriole) which is so thin that it can only be seen at the point of crossing—it shows an hour-glass contraction, is definitely "banked," but not deflected, although on the retina it is crossed by the large a. temp. sup. without any change. Severely crushed veins on the disc of a sclerotic eye can also be seen in plate No. 23. In plate No. 4 two superior veins are crushed and banked by the artery. It is well-shown in plate No. 16. The patient (of Mr. Batten) was a woman aged 70 years; blood pressure 210/130. The retinal crossings of the vessels are drawn as normal, except that their translucency is lost; but on the disc the v. cent. inferior is severely crushed, banked by the artery and sharply deflected centripetally against the direction of the arterial blood-flow. With these illustrations in mind, the reader will better be able to understand the description of my cases. In a few of them I had direct evidence, in my opinion, that all this havoc with the veins is wrought not so much by the arteries as by their "perivascularis." The vein and the artery are running close to one another on the disc. One can see them on the same level—they are only touching, the artery not crossing or covering the vein, and still this portion of the vein running close to the artery is narrowed. That this means an actual impediment to the venous blood-stream is proved by the fact that the peripheral end of the vein is enlarged and "banked" just before the point where the vessels are approaching each other. The artery may even go under the vein on the disc, just in the centre, and then push the vein away. In one case both veins crossed in front of the arteries and had ampulliform dilatations. It was difficult to decide whether they were aneurysmal because the entrance in the lamina was too narrow, or whether they were flattened by the underlying arteries. The patient was a healthy boy aged 10 years. In another case the sup. temp. vein
was sharply deflected centripetally by the artery on the disc; farther down, on the retina, the artery again crossed the vein, and the vein the artery, without any change at all, but on the disc all the vessels and their crossings were accompanied by white lines. Such cases prove that the "perivascularis" and not the size of the artery is the cause of the crushing. A small arterial branch (1/8 of the vein approximately) crushes the inf. cent. vein causing it to have an ampulliform dilatation from this point up to the border of the disc, and a large artery crosses the sup. temp. vein also on the disc without any change in the vein. On the disc the deflection of the vein by the arteries is congenital and not sclerotic, because in spite of being well deflected by the same artery, the vein is not crushed and the venous pulse is not interrupted. If it were a sclerotic crossing, the vein would be crushed and the venous pulse interrupted. But the supreme test is furnished by the fact that sometimes the vein on the disc, when crossing an artery, is definitely flattened. One can see the calibre of the vein increased at the crossing and decreased immediately after. Moreover, in most cases the artery can be seen clearly or dimly through the vein. It is especially striking when it occurs in the sclerotic eye where all other vessels have lost their translucency at the crossings, even where a small artery crosses a large vein, or a tiny vein a large artery. This is a very striking appearance, but it is very difficult to draw. On plate No. 34 in a highly sclerotic eye, an attempt has been made to show it; the branch of the sup. temp. vein crosses an artery on the disc, the vein is flattened and the artery can be seen dimly through it. The vein is crossed further up by an artery at the border of the disc and is crushed and "banked." The explanation is a simple one. The blood-stream of the vein is so thinned by the flattening that it allows the artery to be seen through it. I cannot find any other explanation of this remarkable phenomenon of the flattening of the veins on the disc only, except by admitting that they are squeezed between the lamina cribrosa from behind and some structure in front. This explanation is proved, in my opinion, by one of my cases of sclerotic fundus, where a vein crossed the artery three times, both vessels being of equal size, one of these crossings being on the disc where the vein was distinctly flattened and the artery clearly seen through it, and then twice on the retina, without any sight of the artery. I saw this flattening in 40 cases out of 464. If this flattening of the vein occurs just at the point where the vein crosses the artery on the border of the central cup, before dipping into the centre of the excavation, then it may also be explained by the collapse of the venous wall when bending at a sharp angle over a rigid artery. No other explanation except pressure from above is available, in my opinion, when this flattening occurs anywhere
else on the disc. Twice I have seen a medium sized artery crossing a vein larger than itself on the disc. The vein was crushed, flattened and had strong white lines on both sides of the crossing. They were sclerotic eyes, with a loss of translucency of all other vessels, but this artery allowed the vein to be clearly seen through it. It was a beautiful picture of three portions of the vein, like a flattened violet band.

A very curious sight may be seen if the vein be dilated like a leech before dipping into the lamina cribrosa. If a small artery crosses the vein nearer the periphery, the head of the leech will be distinctly cut off from the body by the white lines at the crossing.

In one sclerotic eye, the vein appeared to form two varicosities. The venous pulse was present. I collapsed the vein by pressure, and came to the conclusion that this appearance was simply due to a promontory of disc-tissue, which squeezed itself on the lumen of the vein and was, of course, congenital. What can be the nature of this mysterious something in front of the disc? We can immediately exclude the membrana limitans retinae interna and the corpus vitreum, as their pressure is the same everywhere, and I did not see any of the above-described phenomena on the retina. Moreover, in three sclerotic fundi, in the retina I saw the vein distinctly elevated when crossing the artery, a condition which I have never seen on the disc, and which is very seldom seen on the retina; only three times in more than 900 fundi.

In one case I saw a vein crossing the junction of two arteries and the arteries themselves; the vein was distinctly elevated and perhaps also flattened. As the vein was stretched over two arteries, probably this flattened it. Otherwise the explanation is very simple. Exactly the same would happen to a soft, elastic, collapsible tube full of liquid, if it lay over a hard, rigid, non-collapsible tube also full of liquid and approximately of the same size. If the pressure from without were increased, the collapsible tube would be flattened against the rigid one. This is exactly what happens on the disc, and is the best proof that the vessels on the disc are under the same pressure as on the retina, plus something else. The nerve fibres cannot exercise this additional pressure. They are thickest at the junction of the retina and disc and thinnest towards the centre of the disc, so that the phenomena under discussion ought to be seen not on the disc but on the retina near the disc. They are not seen there, however, and their maximum occurrence is nearer the centre of the disc. Besides, in most cases of myelinated fibres, which are much thicker, of course, than the non-myelinated ones, the retinal vessels (even the usual sufferer, the vein) emerge from them without any change in their volume. The reader can see this for himself in the drawings of
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Frost, Nos. 35, 36, 37, 38, in Fox's Ophthalmology, p. 359, in Foster Moore's Medical Ophthalmology, p. 281. Well, then, if not the nerve fibres, perhaps the neuroglia? But the neuroglia accompanies the nerve fibres and does not reach the centre of the disc. In a few cases, I saw a very peculiar phenomenon. Some of the vessels on the disc can be seen covered by a thin and narrow white bridge, connecting the lower part of the disc with the upper, in the same sector. I never saw this in the centre of the disc; it was always near the periphery of the disc. The vessels, when crossed by those fibres, were not affected by them in their lumen, nor in their direction. I think that those fibres are anomalous, congenital bands of neuroglia. In one of my cases a "tongue" of whitish tissues starting from the centre of the disc is probably neuroglia, in which the artery and vein run without any change in their lumen. Gradually, as they passed farther down in the retina, they lost this whitish tissue on their outer side, but still preserved it between them. I can only explain this as a visible proof of those hypothetical channels formed from neuroglia and perivascularis, through which the vessels run on the disc. Only once have I seen those white fibres slightly affect the vein. Altogether I have seen that in eight cases out of more than 900 fundi. Therefore, we are left with one conclusion only, that there is a special membrane, transparent and strong, covering the disc on its inner retinal surface and pressing upon the vessels in the sagittal meridian. In six cases out of more than 900 fundi, I saw a thick membrane, connected with the lamina cribrosa, always arising from the centre of the disc and covering a more or less considerable part of the disc with its vessels and part of the adjacent retina. It was always of a whitish colour, thin and translucent, and irregular in contour. The vessels were not affected by it when passing through it. Gunn, in Gowers' Medical Ophthalmology, page 6, states that "peculiar white films sometimes lay in front of the vessels on the disc, looking like fragments of tissue paper or white gauze, and allowing the vessels behind to be dimly seen. They are usually congenital, the relay of the tissue at the back of the vitreous." He also gives a picture showing the retinal vessels not altered by this membrane. The difference between this description and my cases is chiefly that I never saw the whole disc covered.

When I came to the conclusion that there existed a special membrane on the disc, I also formulated at the same time a special theory for the explanation of arterio-venous compression. I did not feel at all at ease as I could not find any anatomical evidence in the text-books. Frost does not even mention the possibility of such a membrane; it is the same with Parsons, Fuchs, Römer, Fox. Salzmann states distinctly that the vessels are covered on the papilla only with a thin sheath of glia, which is stronger at
the bottom of the physiological excavation; but, in his opinion, there is not even a proper membrana limitans interna present, as this is the spot, where there were in embryonic life the arteria hyaloidea and the canalis hyaloideus. The pathology of retinitis proliferae also shows that there does not exist here a special demarcation from the corpus vitreum. An opinion rather damaging to my theory of a specially strong membrane!

Therefore, it was a great relief to me to find a few encouraging remarks, after my own work was accomplished, in Ginsberg's book, published in 1911, and as far as it is possible at present, a complete anatomical confirmation of my deductions in an important paper by Prof. Krückmann, published during the war. This was all the more important as Krückmann studied the subject as a purely anatomical one, and his work was far removed from any connection with the sclerotic fundus.

Ginsberg points out the fact that if the connective tissue and the glia are less developed at one part of the papilla, the excavation will be larger at this spot. The surface of the papilla is covered, according to Elschög, by a membrane which is sometimes very thin and sometimes much thicker, and which seems to arise from the central fasciculus of connective tissue. The number of nuclei diminish rapidly near the border of the disc where this membrane joins the membrana limitans interna. The reader is referred for more particulars to the original book of Ginsberg.

Of the utmost importance is the paper of Prof. Krückmann, where he gives photograph plates of his microscopic preparations, which struck me with their exquisite beauty and remarkable clearness and precision. By the kindness of Prof. Krückmann I am able to reproduce a few of these plates and they will be found as Figs. 1, 1a, 1b. While holding the chair of ophthalmology at Königsberg he had the opportunity of injecting Zenker-Spuler's fluid into the head of a young criminal immediately after his execution. In this way he obtained excellent anatomical evidence of the distribution and structure of the supporting tissue in the papilla and retina. Krückmann states that Bergmann was the first to see, in 1857, a special membrane on the surface of the cerebellum; Retzius described it on the surface of the cerebrum, and Held found present everywhere on the surface of the central nervous system a constant glious membrane, membrana limitans gliae superficialis, which exists also on the surface of the optic nerve as an unbroken membrane. We have known a long time of the existence of a similar membrane on the retina—membrana limitans interna; but also on the outer surface of the vessels of the cerebrum and spinal cord we encounter a homologous formation of the glia, membrana limitans gliae perivascularis. Does the reader remember the sentence of Weigert? This membrane was first discovered by
Held and again described later by Krückmann himself. It is extremely difficult to discover, because one has to use especially fine methods. Probably these technical difficulties are the reason why, according to Krückmann, many authorities doubt, or simply deny, its existence. When successfully treated, the membrana limitans gliæ perivascularis may be recognised anywhere on the optic nerve and the retina. The limitans gliæ perivascularis is very thin and easily torn. The point where the membrana limitans gliæ perivascularis meets with the membrana gliæ superficialis is called by Held, glia marginalis. All these three membranes are also present on the optic nerve.

The first layer—a mesodermic one—is limitans perivascularis. It forms a barrier between the connective tissue in which the vessels run and the nerve layer, and is called by Krückmann, "border-skin," "Grenzhaut." The second layer is the "border-leaf," "Grenzschicht," with fibres and threads of the reticulum of the glia, and corresponds to the membrana basalis retinae. The third layer, which, according to Held, is not always present, consists chiefly of fibres of the glia—"Gliamantel." This is, briefly, according to Krückmann, the structure of the surface of the papilla. The reader is referred for further particulars to the original article. The structure is a complicated one and through it the vessels run, enveloped on all sides and in addition covered over from above.

Let me recapitulate. In my opinion, the vessels of a normal papilla run through a perivascularis which is in itself a very complicated and strong structure. It probably contains lymphatic channels and may be reinforced by neuroglia. The same difficulty which the optic nerve experiences when passing through the lamina cribrosa is experienced by the retinal vessels when passing through the structure of the disc, squeezed, as they are, laterally and sagittally. The veins adapt themselves by becoming narrower than on the retina. The white lines, which accompany the vessels on the disc, are the points where their "perivascularis" meets the tissue of the disc proper, and where the connective tissue is at its maximum. It is probable that these white lines may be more easily seen in the sclerotic eye, which, logically, is only to be expected, but in this case the sclerosis is only aggravating a pre-existing condition and is therefore making it more markedly evident—an important thesis, which often lies at the root of many phenomena of retinal vascular sclerosis. Therefore, all the various vascular changes seen on the disc such as white lines accompanying the vessels or at their crossings, crushing of the veins by the arteries, deflection of the veins by the arteries, flattening of the veins when crossing the arteries, and their collapse or banking, although easily explained by mechanical causes, cannot
be regarded as pathological even in a highly sclerotic fundus, as it is impossible to differentiate them from congenital ones. These considerations may be verified by a glance at the analytical table. In Group A, where the blood-pressure was normal—average 128/84, I saw white lines on the disc in 46 per cent. In Group B, where the blood pressure was moderately high, 200/110, they were present in 40 per cent., i.e., in nearly the same number. Even in diabetes—a disease notorious for its sclerotic vessels—I saw them only in 41 per cent. These percentages may be a coincidence; another observer may in his cases find white lines more or less frequently, but the important fact is that they do not increase in frequency with the progress of sclerosis, whilst all other signs do. How important it is to make all the foregoing distinctions I have already shown when criticising the opinion of Prof. Leber. Gunn, on p. 198, when describing the perivascularis in Bright's disease, says: "Ultimately the thickening becomes great and may cause a white line to be seen on each side of the vessel. This appearance, however, is also seen in cases of a natural excess of connective tissue about the vessels on and near the disc." In my opinion, these appearances have nothing in common; the first is pathological, the second chiefly physiological.

The Normal Retinal Vessels

The anatomy of the retinal vessels is not quite clear at present. They lie chiefly in the nerve fibre layer, sometimes being above the common level of the retina, in which case they form little grooves. The arteries are composed of intima, muscularis, which varies in amount with their size, and an abundant adventitia. The veins have no muscular coat. A common and remarkable feature of all retinal vessels is the fact that the connective tissue and elastic elements of their walls increase with age, from childhood onwards. The veins, lacking the muscular coat are rich in elastic fibres; they possess a well-developed adventitia. The existence of a "perivascularis" on the retinal veins has been known for a long time; it was already known to Frost. Ginsberg says that "the veins and capillaries of the retina have perivascular sheaths, formed of endothelial cells (as in the brain) and which form with the wall of the vessels a perivascular chamber; therefore, the capillaries and smallest veins look like tubes of endothelium which go through a larger endothelial pipe," (p. 303). The presence of the perivascularis and especially the rôle of the perivascular chamber or channel were not always so clearly described and the opinions of the authorities were divergent. According to Bailliart,
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Rochon-Duvigneaud did not think that these perivascular channels were lymphatic spaces. Prof. Krückmann's work, which has been quoted above, decided the issue. The capillaries of the retina are covered with endothelium on their inner surface (e—in the illustration). The endothelial cells are fixed to the membrane which corresponds to the intima of the larger arteries and which is the wall of the capillary. The outer surface of this wall serves as the inner wall of the lymphatic channel. The outer wall of this lymphatic channel is formed by a strong membrane, composed of membrana limitans gliae perivascularis (P.) and membrana piae accessoriarum (P.). The space between the outer and inner walls, which corresponds approximately to a quarter of the lumen of the capillary itself, is filled by loose connective-tissue fibres. This space also possesses its own endothelial cells (C.). Over the external wall of this lymphatic channel, which corresponds to the outer wall of the adventitia and which is formed, as mentioned above, by two glial membranes, comes the third membrane—membrana basalis gliae—which is formed not by Müller's fibres, but by broad and prominent neuroglial cells. An interesting feature is that these glial cells are very abundant in the angles between the divisions of the vessels; they are more numerous there than anywhere else. Krückmann shows that the perivascular channels are undoubtedly lymphatic vessels. Such are the brief particulars of this noteworthy paper, where the fine microscopic preparations leave no room for doubt as to their correctness. He could not, however, prove the existence of these lymphatic vessels in the walls of the retinal arteries and veins. The vessel walls and their surrounding connective tissue were always so compressed that, histologically, no space could be demonstrated by Krückmann. Logically they ought to exist there also. Their presence has been proved in the case of the capillaries and optic nerve. Can we imagine that the flow of lymph commences around the capillaries, disappears around the arteries and veins, and appears again on the disc? Frost thinks that the existence of a perivascular space around the veins is proved. I am inclined to think that, although histologically not proved, the "perivascularis" of the retinal arteries is no less a complicated structure than that of the capillaries. The arteries possess a well-developed adventitia with abundant elastic fibres, which varies directly with the size of the vessel. The fibrillar connective tissue also exists in the arteries of the retina, which possess, as mentioned above, a definitely developed, but not very strong, muscular coat. "The smaller the artery, the less muscle does it possess; in the larger branches the muscular coat consists of 2-3 circular layers with some longitudinal fibres, in the smaller it is reduced to one layer only, and in the smallest to a few muscle cells. The amount of connective tissue increases
correspondingly." (Ginsberg.) It is a point of some interest that in Gowers' book, p. 200, we find an anatomical picture of the pathological process in the retina, where the author remarks: "The wall of the artery itself appears to be blending with the connective tissue of the adjacent retina. . . . An even more remarkable example of the effects of the thickening of the arterial wall is seen in Plate X, Fig. 1, in which the increased tissue of the wall is apparently in the outer wall and ceases abruptly." This is, in my opinion, the best proof of the keen eye of Marcus Gunn, who caught sight of a phenomenon and registered it correctly, although he was unable to explain it and wondered what it could be. Parsons, on p. 1286, states that the central retinal artery consists of an endothelial lining, elastica interna, a relatively thick muscular coat with scanty elastic fibres and a connective-tissue adventitia, fading indefinitely into the surrounding connective tissue and containing an abundance of irregularly disposed fine elastic fibres.

The vessels of the normal retina usually form waves, mostly in a lateral direction. That is why I came to the conclusion that it was difficult to decide whether in any given case the tortuosity of the vessels was increased or not. If only one vessel showed tortuosity, or many, especially smaller vessels, were "cork-screw," then I regarded the tortuosity as a pathological one. On the other hand, these lateral waves are a pitfall when describing and explaining arterio-venous compression of the sclerotic fundus. An artery may happen to cross a vein just over such a wave, and the deflection of the vein in such a case is not pathological, but congenital. At most, as on the disc, the sclerosis in such cases will aggravate a pre-existing condition, but will not create a new one. A good illustration of this can be seen in plate No. 24. The end of the sup. temp. artery crosses the vein twice, the vein being deflected the first time centrifugally, and the second time centripetally. The symmetry of both waves suggests that the vein would be of the same shape in any case, independent of the arterial crossing, and that the artery simply meets the vein in two opposite directions because of the change in the course of the artery itself. Therefore, I consider as pathological such deflections of the vein as exist only at crossing points, but nowhere else; or if they are so exaggerated as to be put out of all proportion to other waves on the same vein. Besides the lateral waves, the vessels may be tortuous in the antero-posterior meridian. Prof. Leber and Gunn, to quote only two, think that such a wave is always a pathological one, caused by retinitis or exudation covering a part of the vessel. In Leber's opinion, in physiological antero-posterior waves the vessel remains on the same level. I beg to differ. In a normal fundus one may see, though not often, an antero-posterior wave.
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Part of a vessel may be observed to dip down in the retina, and to see it clearly one has to change the refraction, when the vessel will be seen distinctly, sharply outlined, like other parts of the same vessel. In pathological cases the contour of the vessel is oedematous, not sharp, as if covered with a veil or a thin layer of water. In one sclerotic fundus, in the right eye, I saw a small vein, coming from above and crossing the disc independently. It formed a loop which was situated much deeper than the rest of the vessels—the venous pulse was present in it. In another case an artery runs on the disc, in the vertical meridian, which is also not on the same level as the other vessels. These are extreme cases, and they are very rare—two cases out of over 900 sclerotic fundi; but small waves on the retina are more common. These waves may lead to another mistake, especially if near the disc, where the arteries are thick; they catch the beam of light from the ophthalmoscope at one angle and reflect it to another. The result will be that this part of the vessel will be seen as a "silvery wire" and taken to be sclerotic. Careful moving of the ophthalmoscope and the fact that the fundus does not show any sclerotic changes, will help to clear up the doubtful point.

In a boy aged 10 years, healthy and with a blood pressure of 100/50, I saw the large sup. temp. vein crossed by an artery and centripetally deflected. There is always some difference between a congenital and pathological deflection, the latter being sharper and more limited; the former is of a longer radius and involves a larger part of the vein.

All retinal vessels have normally a light reflex, a brilliant white streak, along the centre of the vessel, gradually diminishing with the size of the vessel, and much stronger on the arteries than on the veins. Gunn thought that this reflex was the result of the refraction of the light by the anterior curve of the blood-column. Bailliart, p. 180, thinks that this light reflex is more probably the result of the reflection of the light by the walls of the vessels. "It is generally more marked on the arteries than on the veins, undoubtedly because the veins, flatter and less contracted, present a larger reflecting surface." Bailliart's explanation is more correct than Gunn's, but is still incomplete. Of course, the primary venous branches are larger than the corresponding arterial ones, but the secondary ones are of equal size to the primary arterial ones. Then, if Bailliart were correct, the light reflex would be in the latter case of the same intensity for both the veins and these arteries. However, it is notoriously not so, being especially weak on the secondary venous branches, although their reflecting surface would be the same as that of the primary arterial branches. That is why I have arrived at the conclusion that the light reflex is caused chiefly, though not exclusively, by the con-
nective-tissue fibres and muscular coat, which are so strongly developed in the primary arterial branches, diminishing as mentioned previously with their size, and are poorly represented in the veins.

There is much more to be said about the normal fundus, but I intend to discuss here only points which are specially important for the proper understanding of the sclerotic one. I shall often have to criticise some orthodox views and many living authorities. I trust that this will not be taken in a spirit of animosity. I have full respect for their knowledge and experience against which my own is very small. I simply think that we see facts differently—

amicus mihi Plato, sed major amicus veritas.

Another characteristic feature of the fundus in the young is the beautiful glare. Its origin is still unknown. Personally, I am inclined to think that it is an expression of the full turgidity of the whole retinal tissue, chiefly perhaps of the glial membranes, as it is especially brilliantly reflected around the vessels. It disappears with age alone and is not essentially affected by sclerosis of the vessels. This is proved by the fact that in all cases of patients aged 20-25 years with increased blood pressure and early signs of retinal sclerosis, this glare was still present. On the other hand, in the elderly, say aged 50-60 years, with a perfect fundus and normal blood pressure, this glare is absent. Therefore, it does not play, in my opinion, any part at all in the diagnosis of retinal sclerosis.

The translucency of the retinal vessels is perfect in a normal eye. Gunn and Frost think that by careful focusing, especially at the crossing points, one may sometimes see the walls of the vessels as very fine whitish lines. I beg to differ from them. I saw the latter only in cases where translucency was already lost, or nearly lost, in all the vessels, except perhaps one or two. Then, as an early sign of sclerosis and next to the loss of translucency, these white lines appear on the crossings. Only in one case out of over 400 in a man aged 48 years, with blood pressure 110/65, and a preserved translucency of the vessels, did I see these white lines present on the disc and only on the retinal crossings of the large vessels. Will it be audacious if I suggest that in this case they are a congenital anomaly? Bailliart is also of the opinion that the walls of the retinal vessels in the normal state are transparent and invisible to us" (p. 180).

A point of the utmost importance is the fact that normally on the retina the vessels cross one another without any change. All their fantastic combinations, except when the vein twines round the artery a few times and is slightly distended, perhaps due to its own gravity and collapsibility—all differences in size, even when the artery is twenty times larger than a venule, or vice versa.
—all this is seen on the retina without the vessels being crushed, deflected or even depressed. As far as I am aware, the opinions of all writers are unanimous on this cardinal point, and I have not met with a dissenting voice. "Ordinarily, when an artery crosses a vein there is no sign of pressure" (de Schweinitz). Here is the key to the problem of retinal sclerosis, the correct basis of diagnosis. As described previously, conditions are quite different on the disc, where the crushing of the veins and their "banking" is physiological.

'Bearing on the discussion of the arterial pulse in the sclerotic fundus, I should like to say a few words about the venous pulse. The latter may be seen, contrary to the opinion of Bailliart and most writers, not only on the disc. I have seen it most clearly on the disc, but with careful examination it may often be discovered very far away from the papilla, 6-8 discs away, and this not so infrequently. Usually it shows itself, as a dilatation and collapse of the trunk of the vein. On the disc, where the vein dips down into the excavation, one can see the blood-column advance and stop. There is, however, a finer method of detecting the presence of the venous and arterial pulses. When using a good illuminating ophthalmoscope, I prefer Dr. Woolfe's, one can often see a fine glare some distance away from the vessels—say 0.25 or 0.5 D.D.—which is the reflex of the beam of light from the wall of the vessel. It is not very easy to obtain it, and one has to train oneself by carefully changing the position of the ophthalmoscope and watching the vessel all the time to see it clearly. Then this glare will be seen pulsating and the pulsation for the arteries will coincide with the radial pulse of the patient. This local glare is of a different appearance from, and must not be confused with, the glare of the whole retina in young persons, which was mentioned previously. It would take us very far from our theme to discuss the cause and nature of the venous pulse. It is important only to note that the blood-stream is usually not interrupted on the disc when the veins are crushed physiologically, as in a normal eye. Duke-Elder in "Recent Advances in Ophthalmology," p. 131, explains the cause of the venous pulse as follows: (1) The arterial pulse is communicated through the capillaries to the veins; and (2) The veins in the eye are compressed by the intraocular pressure increased by the arterial pulse; at the exit—passing through the lamina cribrosa—a sudden expulsion of the venous fluid is possible. As many secondary factors, he mentions the rigidity of the sclerotic, sclerosis of the arteries, proximity of the vein to the arteria centralis in the trunk of the optic nerve, and communication with the sinus cavernosus. I should reverse all of this. In my opinion, the last one, the communication with the sinus cavernosus and through it with the systole and diastole
of the auriculum cordis, assisted possibly by the respiratory movements of the chest and by the weight of the column of venous blood, is the chief cause, and all the others are secondary—helping or eclipsing the primary cause, as the case may be. In many cases, where there were actual complete stoppages of the pulse-wave, as shown by the oscillometer, and when the venous pulse was also present I saw the venous pulse on the disc stop completely, alternating with the radial pulse, and then start again—a beautiful picture, indeed! In one case, that of an aged woman, it was one stoppage for every 4-5 beats, and not once did the venous pulse fail to stop as well. In one case, I had a rare opportunity of verifying this. The man, aged 57 years, was suffering from angina pectoris with hyperpiesis. He walked into my surgery on January 1, 1928, at 8.30 p.m. with, what seemed to be, an attack of asthma (cardiac or bronchial?). He was advised to go to bed, and was able to walk back to his home, a few hundred yards away. At 9.30 p.m., I was urgently called to see him. I found him very pale, frightened and fully conscious. He was standing in the middle of the room, holding both his hands to his heart, and expectorating blood and sputum. Râles were audible all over his chest. I did all I could for him. His systolic blood pressure fell from 170 to 110 and then to 80, the pulse being quick, but regular. I performed a large venesection. For this he preferred to lie down on the couch. He died under my hands within the next minute or two. Ten or fifteen minutes after his death, when there were no respiratory movements and the flow of venous blood from the wound had ceased, I examined his fundi, which I knew very well, as I had examined them many times previously. The disc was unusually pale and the excavation very deep; the retina was rosy, the arteries were very rosy and very much contracted. The veins were blacker than usual, and deep in the lamina, where both the veins met, feeble, flickering, like the flame of a candle before it goes out, very irregular, but still distinctly to be seen, I saw both veins pulsating. The same veins on the disc were already motionless. I carried out artificial respiration for five minutes. At the end of that time, when I again examined the fundus, the venous pulse was gone, the arteries more contracted, the veins collapsed and in the inf. cent. vein, the blood-column broken in two, the point of division being the spot where the vein was centripetally deflected and closely connected with the artery, everything being still very clearly visible. I can explain what I saw in this case only by the presumption that, when life had apparently ceased, the auricle was still beating and contracting for a few minutes and caused some movements in the venous column, without actual propulsion of the blood-stream.
Sclerotic Fundus

It is with some purpose that I call my cases, not arterio-sclerotic, but sclerotic. The term arterio-sclerosis is very misleading. It is used for many conditions which have nothing in common. That is why many writers now use the terms of primary arterio-sclerosis and secondary arterio-sclerosis. Classification of the different forms of arterio-sclerosis varies considerably according to the personal views of the authors; compare that of T. Thompson in Price’s Text-book of Medicine with that of Evans, as given in Halls Dally’s “High Blood Pressure.” The source of all the trouble is the difference between the points of view of the pathologist and the clinician. In spite of a complicated morphology, the histology of the retinal vessels, as of all other vessels, is relatively simple. They consist of endothelium, muscular and connective tissues. In the attacks by the innumerable toxins which afflict the human body the artery—first to be assailed in general toxæmias—will react in a simple way—broadly speaking by atheroma or sclerosis. In the former, the endothelium shows a pathological thickening, sometimes nearly blocking the lumen of the vessels and, growing outwards through the tunica elastica, involving the muscularis as a secondary process. In sclerosis the chief changes are situated either in the muscular coat (this corresponds to Allbutt’s involutionary arterio-sclerosis—a common feature in the aged, the result of the wear and tear of life, and perhaps most truly explained by Metchnikoff in his “Etudes de l’Optimisme”) or it is the severe hyaline degeneration and thickening of the artery with atheromatous blocking of its lumen in nephritis. Syphilis, which is supposed to be the greatest dangerer of the vascular system, though I hope to show later on that in the case of the retinal vessels it is not, sometimes produces a change sui generis, and sometimes not. If the dead artery is a meagre food for the student, the living one is infinitely more varied and changeable; and one of the most fascinating problems in modern medicine, in my opinion, is the study of the actions of different toxins on the various parts of the vascular tree, or even, as will be seen later, on the minutest parts of the tiniest vessel.

Atheroma usually attacks the large vessels of the body, where elastic fibres predominate (aorta, carotids, etc.). It is one of the most insidious diseases and extremely dangerous because of its localisation in the coronary arteries or in the aorta at the point where they arise. It may also attack the cerebral vessels, but I am of opinion that this is rare and when it does occur, it is the result of essential hyperpiesis or nephritis. Pure atheroma is very rare in the retinal vessels (thrombosis of the central artery or its branches). It more commonly occurs in the form of “beaded” silver-wire arteries, especially in the small independent arteries
around the disc (I prefer to call them independent, as often one cannot be sure whether they are real cilio-retinal arteries or branches of the central artery). The change in the lumen of the larger arteries may be of varied origin, but these "beaded" independent arteries, in my experience, were invariably connected with raised arterial tension. This is why I think that in the cerebral vessels atheroma prevails as a complication of the arterial hypertension of essential hyperpiesis and of interstitial nephritis. I am well aware of the fact that cerebral vascular sclerosis may be present without any accompanying retinal vascular change, but, I think, this is exceptional. The vast majority of cases will show some fine retinal changes, and, if changes are found in the fundus, one can be absolutely certain that the former is present also. Palpation of the peripheral arteries, although important, does not help to clear up the diagnosis; the radial artery may feel quite soft even though its intima is already atheromatous. The blood pressure, systolic and diastolic, is usually normal. This sclerosis of the intima corresponds to Marchand's atherosclerosis; its ultimate result is atheroma and its chief characteristic is its patchy distribution in various parts of the vascular tree, as described above.

The hard, "thickened," or "whipcord-like" artery may be the sign of a true degeneration or of a contracted muscularis, which ultimately leads to some histological changes. The true degeneration, Allbutt's involutionary arterio-sclerosis, is more or less equally distributed all over the body. Calcification usually accompanies it in the later stages. Its rôle is still uncertain and it is possible that this is Nature's way of repairing the damaged wall and maintaining the circulation at its optimum. There are some indications that the calcium content of the blood of such arterio-sclerotics is lower than normal. Palpation cannot help us, as it is impossible to decide on the exact state of affairs by it alone whether we are dealing with the contracted, hard muscularis of a hyperpietic or the degenerated and hard muscularis of involutionary arterio-sclerosis. The blood pressure is usually normal, or perhaps the systolic pressure is slightly raised. This sclerosis, according to many authorities, is present in every person of a certain age, say after 50 years, but, of course, varies considerably with individuals. If the rise of blood pressure were the result of this form of sclerosis, then arterial hypertension ought to increase with age, and be a constant sign in every person say over 70, but luckily such is not the case. This kind of sclerosis has a very favourable prognosis; its chief effect is a limitation of the activities of the body. It is generally the case that the parenchyma of the organs can function satisfactorily for many years, although vascular degeneration is present and even advanced.
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The hard artery of a hyperpietic starts as a tonic contraction of the muscularis media. Some time must elapse before this tonic contraction will become evident and, therefore, it is possible to find a soft radial artery along with raised arterial tension. However, this is only a transitory phase and can be omitted for the purposes of this paper. This purely functional tonic contraction of the media may persist for years without any histological changes. Then it leads to hypertrophy of the muscularis media ("myohypertrophy") or to fibrosis. A frequent and dangerous complication of hyperpiesis is atheroma. The cause of this disease is unknown. Some point out its dependence on local sclerosis of the cerebral arteries. For instance, sclerosis of the vessels of the fourth ventricle will affect the respiratory and vaso-motor centres. The latter (concerned with the vascular system) will maintain at any rate, a good circulation in the circle of Willis, and will do so by all means, including permanent tonic contraction of all the arteries. In some cases of apoplexy I found the arteries of the affected side to be much softer than those on the opposite side. Prof. Pal explains this by the involvement of the tonic centre, which he refers, on theoretical grounds, to the corpus striatum. On the other hand, there is definite evidence that there are toxins of unknown origin whose presence in the fluids of the body—I am not sure that it is always the blood only—will contract the arteries. It is an extremely difficult matter to discover them, but some of them are evidently known. Docent Westphal (Münch. Med. Wochenschr., No. 29, 1926) reports that he succeeded in producing considerable and lasting hypertension in rabbits by feeding them with choleserin. On isolated strips of an artery, choleserin acts as a sensitizer for adrenaline and other vasoconstrictors. He goes so far as to say that in 75 per cent. of cases of essential hyperpiesis there is a definite hypercholesterinaemia. Choleserin is a substance which most effectually inhibits the permeability of vessels and of animal membranes, so that its damaging effect is increased. I cannot help thinking that there is some truth in his statements. On the other hand, hypercholesterinaemia is often associated with endarteritis obliterans, where the blood pressure may be normal. We know very little, if anything, about the endocrine secretion of the liver. That essential hyperpiesis may depend on some endocrine disturbance is proved by its frequent occurrence in climacteric women. A case is published where the woman had a systolic blood pressure of 220 to 240 mm., and the heart's action was becoming impaired. Disease of the gall-bladder was diagnosed and operation advised. The blood pressure thereafter fell to 150 mm. and the patient is now quite well (three years after the operation), the blood pressure keeping steady. Be this as it may, the fact is that essential hyperpiesis
is a response of the vascular system to the action of certain bodies—auto-secretions, bacterial toxins or products of faulty metabolism. Let them disappear, and the blood pressure may come down to normal. I have described an illustrative case in my previous paper, to which the reader is referred. The foregoing is the chemical explanation of the problem. Formerly, a mechanical theory was advanced, in order to explain the accompanying hypertrophy of the heart. The heart has to work under raised diastolic pressure, the pulse-wave has to be propelled against increased opposition at the periphery. Therefore, the heart hypertrophies. This theory is now discarded as far as the heart is concerned, but this mechanical explanation is still given by different writers. In Price’s Text-book, in the chapter on nephritis, the reader will find the opinion that, with the sclerosis of the glomeruli in the kidney, the arterial pressure must be raised so as to maintain the circulation. If this explanation be correct, one would expect the same in cases of pure endarteritis obliterans. I have had under my care, such a case for a few years. It is that of a young man aged 33 years, in whom the circulation is nearly completely blocked in the right femoral artery. One cannot feel the pulse in it, and the oscillometer shows a movement of only 0.5. In the left leg, the movement is 6. His blood pressure is 150/75. I have on one occasion seen a man aged 47 years, six weeks after a sympathectomy had been performed on his femoral artery. His blood pressure was 110/60. Now, the amount of blood running through a leg in a unit of time is much greater than that of both kidneys; if sclerosis of the glomeruli is sufficient to raise the blood pressure mechanically, then a gradual blocking of the whole femoral artery and its branches ought to do likewise; but it does not do so, as shown in the above cases.

Based chiefly on the reading of the blood pressure, one could distinguish two forms of essential hyperpiesis. If I am not mistaken, Volhardt was the first to call attention to this. If the ordinates of the systolic and the diastolic pressure remain high, but keep parallel and do not show any inclination to meet, then the prognosis is more favourable (hyperpiesis benigna). If, on the other hand, they show an inclination to meet, either by a fall of the systolic or a rise of the diastolic pressure, or by a combination of both, grave danger may be anticipated, cardiac failure being imminent. There is much truth in this. As long as the heart is able to carry on, it responds nobly to the needs of the body, and the systolic pressure therefore is an indication of the maximum of the cardiac action. This, of itself may be so high as to carry with it a danger to the body, for instance, the rupture of an artery. Such a vascular accident is comparable to the last drop which makes an already filled cup overflow. A decrease of systolic
pressure, if the pathological cause be still present, means therefore a myocardium which is giving way, while a high systolic pressure, good or bad as it may be, presupposes a strong myocardium. That is why one might be misled by relying only upon the systolic pressure. I had a case where a woman, when first seen, had a blood pressure of 310/130. Five weeks later, I saw her with a blood pressure of 240/110, and three days after she had a cerebral haemorrhage. She died seven days after the cerebral haemorrhage. A purely mechanical prognosis based on the systolic pressure only would therefore be very wrong. Two patients with a systolic blood pressure of 210 may be in quite different states of health. The systolic blood pressure, although an invaluable part of the clinical picture, can be appreciated correctly only in conjunction with other clinical data.

Diastolic pressure bears quite a different significance. It shows the continued pressure and, therefore, the permanent damage to the arterial wall. A continuously raised or, even worse, rising diastolic pressure means a serious prognosis. It does not fluctuate as widely as the systolic pressure; one can rely more upon it. Two men having a diastolic pressure of 130 are both under the sword of Damocles, although they may seem quite well and clinical investigation does not show signs of damage to the kidney. The retina, of course, will show in most cases the true state of affairs.

The rôle of the kidney is too important a matter to be dealt with briefly and it does not directly concern our argument. However, if an involutional arterio-sclerosis is present everywhere, why should the kidney escape? Here is a good example of the sclerotic kidney, and what was stated previously about involutionary sclerosis—that it merely involves a limitation of function—applies to it also. Prof. Schlayer (Münch. Med. Wochenschr., No. 21, 1926) published a very interesting article on "contracted kidney," as seen in practice. In his opinion, the primary contracted kidney (nephro-sclerosis) is a constituent symptom of a general arterial sclerosis; the secondary contracted kidney is the result of some inflammation, toxic or bacterial, of the kidney, and is clinically identical with terminal chronic glomerulo-nephritis (interstitial nephritis, Bright's disease). Young hyperpietics under 40 years of age, especially with a history of syphilis or other vascular poison, acquired or hereditary, are particularly apt to develop nephro-sclerosis with albumen, casts, and from time to time erythrocytes in the urine, and retinitis. Death supervenes in three to four years—malignant nephro-sclerosis—and treatment can only retard the inevitable end for a limited period.

Benign nephro-sclerosis is, according to Schlayer, twice as frequent as the malignant type. The age is usually over 50 years, and there is some albumen and casts in the urine; constant high
blood pressure and cerebral sclerosis (evidenced by retinal sclerosis). Such cases may remain stationary for many years, even for decades. They do not die of renal disease but of apoplexy, angina pectoris, or cardiac failure. The last condition is frequently referred to as "a gross cardio-vascular lesion." If the reader will remember that in both forms the renal efficiency tests may be normal—in malignant nephro-sclerosis in the early stage only, and in the benign type till the end of life, he will realise how many difficulties the problem of essential hyperpiesis presents and how many gaps still exist in our knowledge. The purpose of this paper is to examine the question from the point of view of retinal changes, and perhaps in the sclerotic fundus will be found some indication of the solution of this fascinating and important problem.

This scheme only represents on general lines the essence of all that has been discussed previously as far as the blood pressure is concerned.

(To be continued.)
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depends on the circumstances of place and time.” If fresh retinal haemorrhages appear in spite of a lowering of the blood pressure in essential hyperpiesis, the danger is great; if they appear in diabetes and if in spite of insulin the blood pressure is rising and the specific gravity of the urine is decreasing then the danger is great.

But, we can occasionally hear it said “A retinal haemorrhage is so small! And often nothing happens to the patient for years!” If we compare the amount of blood running through the eye-ball with that running through the lung, the smallest retinal haemorrhage will amount approximately to half a teaspoonful of blood in the sputum. And surely such a haemoptysis will move a physician to undertake a thorough investigation of the pulmonary system, in spite of the fact that often nothing happens to his patient for years after he has coughed up blood. The retinal haemorrhage is always a serious and temporary warning, although it does not always mean immediate danger. And I earnestly wish that its significance should be grasped and should lead to the same thorough investigation of the reno-cardio-vascular system, as a haemoptysis usually does for the lungs.

Summary.—As far as my investigations have shown, in spite of the lack of direct anatomical evidence, the vessels in the normal eye run on the disc and on the retina through channels of perivascularis. The disc is covered by a transparent and strong membrane of mesodermic origin. The vessels on the disc are under pressure in the sagittal and horizontal diameters, which is greater than that on the retina. When they become sclerotic they first lose their translucency, then change their colour, and “dot” their light reflex, then the sclerotic perivascularis appears, first as white lines at the crossing points, and later on it takes a leading part in arterio-venous compression. This thickened perivascularis plays an important part in every pathological vascular process in the sclerotic eye, but its action is chiefly a local one affecting separate parts of the vessel, especially at crossing points. Essential hyperpiesis greatly increases and aggravates all the sclerotic signs of the retinal vessels. It is quite probable that there is a special predilection for the retinal vessels and that these are affected more early and more severely than other parts of the vascular tree, except the cerebral vessels and, perhaps, those of the kidneys.

(To be continued.)

CORRIGENDUM

In Dr. Pines’s article in our last number, on p. 97, in the penultimate line, for “sum” read sun. On p. 112, line 30, for “Figs. 1, 1a, 1b,” read Figs. 1, 1a, 1b, 1c. On p. 115, line 14, for “endothelial cells (C),” read endothelial cells (B).