**ARTERIAL HYPERTENSION AND RETINAL
CHANGES**

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IN offering to the attention of the reader the result of my investigations of the correlation between arterial hypertension and retinal changes, I feel that it is necessary to answer the possible question : What else can be added to this particular subject, after the brilliant work and investigation published by such authorities as the late Marcus Gunn, or Foster Moore, or Maitland Ramsay, especially if the investigator be a general practitioner, even if he have some experience in ophthalmology? But the specialists themselves are partially responsible for this: they so often appeal to the general practitioner, asking him to investigate or observe and to make deductions, that I was tempted. On the other hand, the patients who are seen in hospital or consulting practice are for the most part more definite, more or less advanced types of the disease. I had the opportunity of examining patients, many of whom were my panel patients, who came to consult me about disorders which had nothing to do with their visual organs nor with hypertension, and I tried to look for arterio-sclerosis of the retinal vessels in the different stages of this pathological process. I could not carry out such complete investigation of my cases, as is possible with hospital in-patients. From this point of view my observations are very incomplete indeed. As to kidneys I only examined the urine for sugar and albumen. I took the specific gravity and called the urine normal if albumen and sugar were absent and the specific gravity was normal. I never determined the quantity of urine in twenty-four hours, I did not perform kidney efficiency tests and did not examine the sediment microscopically. The Wassermann reaction was not performed. When examining the eye, I only noted the objective signs and changes as shown by the electric ophthalmoscope. I did not measure the visual acuity, nor did I examine the field of vision nor sense of colour. These are all very serious defects from the scientific point of view. I tried to carry out my investigations in the simplest possible way, because of the impossibility of performing all these highly technical tests, and because it is my profound conviction that simple observation and careful attention of the examining medical man will always bring forth some valuable result. I have seldom used a mydriatic. When examining the blood pressure the patient sat with the elbow approximately on the same level as the

apex of the heart. The method was Korotkoff's, and the maximum systolic pressure was verified by palpation. Each patient was examined at least twice at different times. I found that usually by auscultation the systolic blood pressure was a few mm. (10 or so) higher than by palpation. I had to rely entirely on the electric ophthalmoscope, sphygmomanometer and routine examination of the urine, such as can be easily performed in the daily work of a general practitioner. In a few selected cases I had the opportunity of investigating my cases more fully, thanks to the kind co-operation of a few consulting physicians. I consider it my pleasant duty to express my gratitude to Mr. A. H. Levy, for discussing and clearing up many doubtful points, to the Honorary Staff of the London Jewish Hospital and to Dr. Lewis Smith, Dr. J. Parkinson, Dr. A. Rowlands, and Dr. O. Leyton of the London Hospital, for giving me full particulars of their investigations of a few of my cases.

The reader will note also that I very seldom palpated the arteries to investigate their hardness. It is not because I under-estimate the value of palpation, but because I think it very difficult to recognize by palpation, in cases of arterial hypertension, what is the cause of the hardness: real arterio-sclerotic changes or tonic contraction of the muscular tunica media. The arterio-sclerosis of the clinician is something different from the arterio-sclerosis of the pathological anatomist. The latter means by this term sclerosis of the tunica intima; it corresponds to the "athero-sclerosis" of Marchand, angio-sclerosis of English authors, and the ultimate form is atheromatosis. Athero-sclerosis chiefly attacks the type of artery where the elastic fibres are predominant—aorta and large arteries. But it is this type of sclerosis, even if the radial artery itself be involved, that cannot be detected by palpation; and patients with an atheroma of the aorta may have soft radial arteries. In the eye, too, this athero-sclerosis, although it can be seen—embolism and thrombosis of the central retinal artery, thrombosis of the central retinal vein—is secondary in frequency to the other type—the arterio-sclerosis of the clinician. In this type of arterio-sclerosis the seat of the pathological process is chiefly in the tunica media. And of the state of the media we can judge by palpation. The hardness of the media may be caused either by degenerative changes in it, brought on by proliferation of its elastic and connective tissue fibres and by diminution of the muscular fibres, which is followed by calcification—the arterio-sclerosis of all persons of advanced age, caused by the wear and tear of life—"chacun a l'âge de ses artères"—or by a tonic contraction of the tunica media—hypertension—which may exist in a vascular system entirely free from the above-mentioned arterio-sclerotic changes. The study of cases of hyperpiesis has confirmed

what is well known, namely, that hyperpiesis may exist and yet the patient, during life, may show few or no symptoms of the disorder. Even repeated and detailed clinical examination has failed to show any of the signs, other than hyperpiesis—no signs or symptoms of cardiac hypertrophy and none of vascular changes (Batty Shaw⁽¹⁴⁾).

These are the two kinds of clinical arterio-sclerosis which are more often seen by a general practitioner, and which show changes in the retinal vessels. The correlation between these retinal changes and the results of the sphygmomanometric examination is the subject of this paper. I was chiefly interested in the question: Why do we so rarely see definite and advanced arterio-sclerotic changes in the retinal vessels of aged people who clinically present definite and advanced sclerosis of the general arterial system, and who often show signs of arterio-sclerosis of the central nervous system? And how far can you go in the prognosis of your patient when you know his blood pressure and have examined his urine and his fundi?

Normal Fundus

The retinal arteries and arterioles, which can be seen by the ophthalmoscope, possess a relatively weakly developed arterial wall, especially of the muscular part. The connective tissue of the tunica adventitia is sharply differentiated from the surrounding nervous tissue (Salzmann⁽¹⁵⁾). The arteries are weakly supported by the connective tissue round them because this tissue—Müller's fibres and the neuroglia—is very delicate in the retina. Those two peculiarities make the vessels, especially the arteries, translucent. More often the arteries cross the veins, but sometimes *vice versa*. Sometimes in the same patient in one eye the arteries cross the veins, and in the other eye the veins cross the arteries. As far as my experience goes, even in young and healthy persons of the age of 7-25 years it is difficult to decide if the calibre of the vessels be normal or not. The calibre of the vessels varies considerably in different subjects and therefore it is difficult to define how far any vessel is wider than a normal one; or this widening or narrowing of the vessel may be so definite that it is easily distinguishable. Therefore I very seldom describe the calibre of the retinal vessels. But if the same vessel shows differences of calibre in various parts of its length, then this will be a serious pathological feature, as is justly pointed out by Foster Moore. It is true that I have never seen this phenomenon present alone—usually you can find other signs of profound pathological changes in the same eye.

Quite a different significance is attached to the translucency of the retinal vessels. Here we have an important and certain basis

for forming an opinion about the state of the wall of the vessel. The larger retinal vessels lie chiefly in the nerve fibre layer and partly in the layer of ganglion cells and they project very slightly, if at all, above the surface of the retina. At the points where they cross one another it is easy to determine their translucency. In a young and normal eye the artery or vein allows one to see dimly the underlying vessel—with one exception—if the superior or inferior main branch crosses the corresponding large vein, or *vice versa*, the blood column is so thick that the underlying vessel cannot be seen at all. This is the case even in children. Therefore the translucency of the vessels can only be stated to be normal in the medium and small arterial or venous branches, but not in the whole length of the vessel up to the point where it joins the disc. Marcus Gunn states: "that normally in consequence of the darkness of the venous blood stream, an artery is invisible through a vein." I could easily see an artery through a vein in all medium and small branches in a normal eye, although I agree that the translucency of the artery, because of the smaller blood column, is greater than that of the vein. I think that the use of the electric ophthalmoscope allows one to pour into the eye a luminous beam of such intensity that the translucency of the vessel becomes much higher. This opinion of Gunn may possibly be explained by the fact that he used an ordinary reflecting ophthalmoscope. The translucency of the vessels, in my opinion, is a fact of primary importance in determining whether they are normal or not. My investigations have led me to the conclusion that if this translucency of the vessels is gone, it is a certain sign of arterio-sclerotic changes in the wall. Even if the tunica media is in a state of tonic contraction, as in cases of pure hypertension, it should still be translucent. The reader will remember, as stated previously, that the middle coat is very weakly developed in the retinal arteries. But if this coat be the seat, not of muscular contraction, but of proliferation of its elastic and fibrous tissue, with a proportional diminution of pure muscular tissue, or of true calcification, then, of course, its optical qualities will be changed and the translucency will disappear first. It is of some interest to find that the translucency in some cases is lost in all vessels, except one or two of the smallest ones. The oldest of my patients, who still preserved the translucency of his retinal vessels is No. 13, aged 65 years. My investigations also showed that I have never found translucency completely absent in one eye and still well preserved in the other. Except the "dotting" of the light reflex and "copper-wire" colour of the arteries, all other arterio-sclerotic changes may be present in one eye and completely absent in the other (deviation of the veins, change of the lumen, etc.), but the translucency of the vessels disappears simultaneously in both eyes. In group No. 1.

where I put all cases with a normal eye-fundus, the translucency is present in 100 per cent. Special attention is drawn to case No. 12.

Two other signs are strictly connected, as will be easily understood, with the translucency of the vessel wall. These are the light reflex and the colour of the arteries. But their definition is much less reliable than the translucency. Light reflex may be only stronger, but not dotted. Therefore I preferred to put all the cases where I was not certain of the "dotting" of the light reflex, in the "dotted" category. Even more difficult sometimes is the definition of the colour of the arteries. Is it only slightly changed or already of "copper-wire" colour? Here too, when in doubt, I called them "copper-wire." My statistics show that in group 1a, with moderate arterial hypertension, the translucency is gone in 97 per cent., but the light reflex is dotted only in 43 per cent. and "copper-wire" colour of the arteries is found in 41 per cent. These changes occur in very much the same proportion but are not nearly as frequent as loss of translucency and hence the latter is of much greater importance. In cases of severe arterial hypertension, No. 1c, as it would be only logical to expect, the translucency is gone in 100 per cent., light reflex is dotted in 94 per cent. and arteries of copper-wire colour are found in 88 per cent. Thus, in my opinion, the analysis of the tables shows that my previous theoretical reasoning is more or less correct. Group No. 1b shows 88 per cent. of translucency gone and 100 per cent. copper-wire colour and "dotting" of the light reflex, but they are all cases of diabetes mellitus. As a résumé I must say that in all my cases, I have never seen one where there was good translucency of all, or even of a considerable part of the vessels in combination with other signs of arterio-sclerotic changes. The translucency of the vascular wall is one of its most delicate qualities and it diminishes or disappears first of all while other signs are only beginning to show. Therefore I divided all my cases into four groups, guided by this principle of translucency. In group No. 1, I collected all the cases where the fundus is normal as well as both blood pressures. In group No. 1a, I put together all the cases where the fundus already showed arterio-sclerotic changes, but the systolic blood pressure was not higher than 200, and the diastolic not more than 90-95; group No. 1c, where the respective readings were 200 or over and 100 or over; group No. 1b, all the diabetic cases; and consider separately the cases of D. and R. as of exceptional interest.

Apart from the translucency of the vascular wall, another detail may be seen at the point of crossing. The artery does not interfere with the vein and does not crush it, but only depresses it. The calibre or shape of the vein is not changed, but the portions just before and after the crossing, and the crossed part itself are not

seen as clearly as the whole vessel. The difference is a very small one, about 0.5D. or so, and altogether this phenomenon is not very often seen (11 per cent.). One can compare this to the depression of a piece of string, if another piece be lying on top of it. The vessels of the retina are fixed by surrounding connective tissue. They are as if they were bound together at the point of crossing.

In his excellent book "Medical Ophthalmology," where the designs of different forms of vessels are most surprising, Foster Moore points out, as an important pathological change, that the veins are crushed by the arteries; they may also be bent or deflected in the direction of the artery. This, of course, is correct. But I have seen this deflection of the vein twice in group No. 1 (8 per cent. in 26 cases). In the first case (No. 10) the fundus is normal in every respect, but the systolic, and especially the diastolic, pressure is certainly high for his age. In the second case, No. 21, the translucency is much diminished; the diastolic pressure is decidedly pathological, being 105. Is the deflection of the vein here caused by early sclerosis of the artery, or is it a congenital condition, aggravated possibly by increased blood pressure?

Arterio-Sclerotic Fundus.

In case No. 13 the fundus is normal, in spite of the age and definitely marked sclerosis of the general vascular system, bad pyorrhoea and some deafness (sclerotic?). The only suspicious sign is the white lines accompanying the artery at the point of crossing. But his blood pressure is only 130/75. In case No. 12 the translucency of the arteries is gone, but not of the veins. His blood pressure is 190/90. In case No. 5a the translucency only is gone; the outlines of the discs are not clear. Otherwise her fundi are normal, the blood pressure being 130/80. She is an intelligent woman with a knowledge of her medical history. She had a severe puerperal sepsis ten years ago, which kept her life in danger for six months and involved both her kidneys. She has even now many signs of old phlebitis in both legs. In case No. 28 the fundi are normal. He has an arterio-sclerosis of the second degree of the brachial and radial arteries, and his blood pressure is 125/70, with a history of a chronic gastric ulcer. Case No. 3a was under my care for five years. His fundi would be normal, but that the translucency is gone. His blood pressure is now 130/85, but in the past was 160/100. Last summer he had a periostitis of the upper edge of the right orbit on the temporal side, which was caused by a chronic purulent infection of his right frontal sinus, a septic ethmoiditis and septic tonsils. He was

operated on for all these last year. His periostitis disappeared but later on he developed some signs of irritation of the sympathetic system, possibly connected with intestinal intoxication. Case No. 14 is also worthy of attention. Case No. 1a is a good demonstration of the well-known rule that even generalized arterio-sclerosis may not be accompanied by arterial hypertension. Evidently there must be some special toxin which causes arterial hypertension. I will discuss this matter later on. The same applies to cases Nos. 23a, 37a, 38a.

Case No. 42a is worthy of special attention. I was called to see the man—I never examined him before—early one morning, when he was in agony from cardio-vascular pain. Diagnosis—angina pectoris(?). Two days later he walked into my surgery telling me that he was feeling better. I found his blood pressure 110/90, pulse 120, arterio-sclerosis of the second degree of the brachial arteries and arterio-sclerotic changes in his fundus. Fearing that he might be a case of coronary thrombosis, I rushed him to a hospital where he was kept in. The physician in charge of the case kindly informed me later that it was a case of coronary thrombosis with a negative Wassermann. The patient is improving now. He has, consequently, a generalized arterio-sclerosis (with athero-sclerosis?) everywhere including the fundi, but his blood pressure is low. Is the cause of this an impairment of his heart, as O'Hare and Walker think, or has he a persistently low blood pressure? It is difficult to decide now. He is obviously not a case of the involutionary sclerosis of Allbutt, whose general scheme is so very helpful.

Cases Nos. 11a, 14a, 20a, 16a, 31a I consider to be types of this involutionary arterio-sclerosis with involvement of the retinal vessels and low blood pressure. In case No. 36a, I was not certain of the softness of the arteries as shown by palpation, but the diastolic pressure is decidedly raised, especially in view of the low systolic pressure. In case No. 32a the unusually low blood pressure may be explained by the far advanced pulmonary tuberculosis. In case No. 22a the systolic blood pressure is very high for his age, although the diastolic is still normal. May I suggest that the toxin is already attacking the retinal vessels, but has not yet produced sufficient irritation or change in the peripheral system to raise the diastolic blood pressure? From the analysis of the tables No. 1 and No. 1a, I draw the conclusions that (1) in involutionary arterio-sclerosis the retinal vessels may be involved in cases without arterial hypertension, but they may escape this till a very late age (65 years) if the arterial tension continues to be low and there is no history of toxæmia in the past; (2) hyperpiesis will always bring with it sclerosis of the retinal vessels, the diastolic pressure being especially important.

Group.	Average Age.	Average Systolic Pressure.	Average Diastolic Pressure.
1	30	133	82
1a	51	153	93½
1b	54	189	88
1c	59	225	123

I did not differentiate between the sexes as this is of no importance from our point of view. Foster Moore, when discussing renal retinitis, says: "It is very unusual to meet with retinitis except in the presence of a very considerably increased blood pressure, but retinitis is often absent though the blood pressure is greatly raised." As regards retinitis, this is correct, but in my cases, where the kidneys were not involved or only secondarily involved, sclerosis of the retinal vessels was always present, although in a different degree. If the hypertension is of recent origin, sclerosis of the retinal vessels may not have yet developed completely; some time must elapse before the unknown toxin of hyperpiesis will cause degeneration of the retinal vessels. They may escape when the general system is already degenerated. In some cases the toxin disappears, the blood pressure becomes normal again, but the changes in the retinal vessels are nevertheless permanent. I will discuss this matter more fully later on. And now I will analyze some arterio-sclerotic retinal changes in my cases, group No. 1a and No. 1c.

In my opinion the only changes one can consider to be pathological are the white lines which accompany the vessels at the points of crossing or on the retina. If they are present only on the disc they may be congenital. If I may be allowed to draw up a scheme in the order of appearances of these changes I would put as first the white lines at the point of crossing or on the disc, as second on the retina near the disc, and as third those on the periphery of the retina. In most cases in the third group these white lines are uninterrupted, but sometimes one part of the vessel between the disc and the periphery may be free from them. These white lines represent in my opinion, a thickened adventitia and sclerosis of the surrounding supporting tissue. As the adventitia is developed principally in the large vessels and less developed in the smaller ones, therefore, these changes must be seen earlier on the disc, where the neuroglia is also more plentiful. The vessels lie between the bundles of the nerve fibres, *i.e.*, where the glia is also lying. The quantity of these nerve fibres in the sagittal meridian of the retina increases in proximity to the disc and reaches

the maximum on the disc, still preserving their septa gliosa (Salzmann). And as in ophthalmoscopic examination we see the retina in this very direction, therefore, it is that these white lines are seen more often on the disc, even in normal eyes. I must mention here that Römer says that in arterio-sclerosis "the walls of the vessels become opaque because of the thickness. On the arteries appears a white line." Fuchs says that "in the later stages white streaks along the arteries and veins (perivasculitis) and haemorrhages." Marcus Gunn states "changes of the coat of the artery . . . narrowed the blood stream are rarely here accompanied by so much fibrous thickening as to lead to the appearance of the white lines along the vessels." This is quite correct for the later stages of the disease, but not for the early stage. Evidently the white lines will first be seen at the crossing points, where one thickened adventitia lies over another one, which is also thickened, or on the disc, if the reader will remember the above-mentioned anatomical particulars. Accordingly in group No. 1 they are 4 per cent. and 8 per cent., in group No. 1a, 15 per cent., 27 per cent., and 5 per cent. But in group No. 1c, with far gone degeneration, they are 18 per cent., 23 per cent., and 35 per cent. If I am right, then another conclusion may be drawn, that in the *medium cases* Müller's fibres remain free from degeneration for a longer time than the neuroglia. If they are limited to the *disc* they are very often congenital, and it is impossible by ophthalmoscopic examination to differentiate these from the pathological ones in the absence of other signs.

Change in the lumen of the vessel I have seen in one case, *i.e.*, 4 per cent. in group No. 1. And I am still in doubt whether it was an actual change or only a tonic muscular contraction of the media. It was 15 per cent. in group No. 1a and 47 per cent. in group No. 1c. Very often the change of the lumen of the vessels is a sign of "athero-sclerosis" of the intima. I am under the impression that it is to be seen more frequently in the small cilio-retinal arteries than in the branches of the arteria centralis retinae.

Tortuosity of the vessels may be a normal feature, as all authors agree. It is present in 30 per cent. of the cases in group No. 1, 39 per cent. in group No. 1a, and 53 per cent. in group No. 1c.

The last and very important sign to discuss is the state of the veins when crossed by the arteries. In the normal eye the vein may only be depressed by the artery (11 per cent. in group No. 1), although de Schweinitz states that "ordinarily, as an artery crosses a vein, there is no sign of pressure." With increasing arterial hypertension the frequency of depression of the vein increases, 30 per cent. in group No. 1a, but decreases sharply in group No. 1c: most of the veins are not depressed, but already crushed.

As a further stage the veins are crushed by the arteries. But

there is not always an actual interference with the blood stream, as shown by interruption of the venous pulse or ampulliform dilatation of the distal end of the vein. It is 0 per cent. for the group No. 1, 27 per cent. for group No. 1a, and 41 per cent. for group No. 1c.

Crushing of the veins, accompanied by interference with the blood stream, is 0 per cent. for group No. 1, 15 per cent. for group No. 1a, and 30 per cent. for group No. 1c. In nearly all cases the *distal* end of the vein is dilated, as is only to be expected. But in case No. 43a, in the left eye the venous pulse may be seen on the disc. It is interrupted by the crossing artery with an enlargement of the *proximal* end of the vein. Can this be explained by a further obstruction of the blood stream higher up in the trunk of the vena centralis, as was anatomically demonstrated by von Michel? This crushing may even be a local condition of the artery. Two crossings in the same eye (No. 3c) may be quite different in this respect. Foster Moore, of course, is right in explaining this crushing by the sclerosis of the artery. He explains in the same way the deflection of the vein. The *centrifugal* (from the disc towards the periphery or following the direction of the artery) deflection of the vein I have seen in 8 per cent. of group No. 1, as stated previously, in 9 per cent. in group No. 1a, and in 6 per cent. in group No. 1c. But *centripetal* deflection I have seen in 3 per cent. in group No. 1a, and in 6 per cent. in group No. 1c.

In a few cases (Nos. 25a, 30a, 40a) the vein is deflected, but not crushed, by the artery. Possibly some significance must be attached to the fixation of the vessels at the point of crossing and to the sclerosis of the fibrous tissue.

The Clinical Value of Arterial Hypertension.

What is the normal blood pressure in man? For systolic pressure the table in Green's "Medical Diagnosis" is, in my opinion, correct. The French authors (Lyon) tried to construct a formula to deduce the normal diastolic pressure from the systolic, but as far as my knowledge goes, other authorities did not find this satisfactory. And the diastolic pressure is of the utmost importance, possibly even more so than the systolic one, as showing the permanent pressure under which the vascular system works. Personally, I am always alarmed if the diastolic pressure rises with a more or less constant systolic pressure. In one of my cases, to take an extreme one, the reading was 250/120, 260/150, 295/175.

In my opinion the maximum normal diastolic pressure at the ages of 50-60 years is 90 mm. Everything higher than that should be regarded as pathological. Green says (page 487): "*as a matter*

of practical application a decided and persistent hypertension with a high, or relatively high, diastolic level means chronic nephritis, until another cause is proved or rendered a reasonable assumption (italics of Green). This statement holds good regardless of the absence of the classical urinary findings at the time that hypertension is becoming manifest. In other words chronic Bright's disease, with or without albumen and casts, but seldom without evidences of impaired renal permeability, is so commonly the cause as to place all other claimants among the 'possibilities' only."

Lewandowsky states: "Ein Blutdruck von 180-200 mm. Hg und höher beweist aber mit fast völliger Sicherheit eine diffuse vasculäre Nephritis, am häufigsten in besonderen eine arterio-sclerotische Schrumpfnieren, auch dann wenn der Eiweiss in der Urine zurzeit nicht nachzuweisen ist" (p. 305). The problem is even more complicated than this: "Most authorities state that abnormal tension follows, rather than precedes, definite urinary findings, but the author believes this to be an exact reversal of the true sequence" (Green, page 262). Other authorities, like Batty Shaw or Langdon Brown in this country, agree that the arterial hypertension may for a long time precede actual anatomical changes in the vessels or in the kidney. "In hyperpiesis the kidneys may be normal" (Batty Shaw, p. 177). The ordinary examination of the urine—as regards specific gravity, sugar, and albumen, will not help much, as the reader can see for himself from my tables (groups No. 1a and No. 1c). Of course the kidney efficiency test will clear up the diagnosis in most cases, but not in all.

Ramsay says: "The toxin being kept back in the blood still eludes the most diligent search. The organic chemist has not tests sufficiently sensitive to discover many of the subtle toxic products of Nature's laboratory. Any tests that have been proposed can be passed successfully by a kidney which, to clinical observation, is manifestly unable to cope with the demands being made upon it." The diagnosis of essential hyperpiesis and interstitial nephritis or, in other words, of secondary and primary kidney, is a very fine and difficult one, and here the ophthalmoscope helps us considerably.

I should not state the case in this form "either the kidney efficiency test or the results of the ophthalmoscopic examination." If a parallel can be drawn, I would compare the kidney efficiency test with the Wassermann reaction: if it be positive, it shows that the kidney is impaired—there can be no discussion, it is settled. But if the kidney efficiency test is doubtful or fairly good, but the ophthalmoscopic examination results in very grave suspicion of interstitial nephritis, I dare to say that I would be more inclined to rely upon the latter than upon the former. Look at the difference

of the retinal changes in the cases Nos. 3c, 4c, 12c, 13c on the one hand, and No. 17c on the other. Possibly the later history of this highly interesting case will clear up the diagnosis, if the migratory character of the patient does not deprive me of the opportunity.

Batty Shaw states that "both (hyperpiesis and eclampsia) may show changes in the retina which are generally accepted as indicative of grave renal disease, but which, as post-mortem examination shows, should merely be accepted as indicative of a grave toxæmia, not necessarily of grave renal disease. The retinal changes may occur without an association with chronic disease of the kidney."⁽¹⁴⁾ What then is the cause of the ophthalmoscopic picture in such cases? Is it primary vascular degeneration or is it inflammation of the retina? The answer is of some importance. If the vascular changes be primary—then in the eye we see the process which is probably going on in the same degree all over the body. But if the retinitis be primary—then we have before us a picture of perverted metabolism in a highly developed and sensitive part of the body, which may be the first to indicate a retention of toxins usually eliminated by the kidneys. Even more, the possibility of some intimate relation between the retinal vessels and the excretory (endocrine?) activity of the kidneys may exist. Take the retinitis of pregnancy: why of all sense organs are the eyes chiefly affected? Truly, we cannot examine the state of the vessels of the ear, as we examine those of the retina. But from the classical symptoms of inflammation—*tumor, rubor, calor, dolor, et functio laesa*—the last one is easily demonstrated. Vision is always very considerably impaired in the retinitis of pregnancy. Why not the hearing or the taste, etc.?

Römer and von Michel, to quote from German authors, consider that in retinitis albuminurica "the whole picture of the disease is the result of a hyaline degeneration of the vessels."⁽¹³⁾ Ramsay⁽¹²⁾ says that "vascular degeneration always precedes permanent structural changes, either in the kidney or in the retina, and consequently the most typical cases of albuminuric retinitis occur when an acute or subacute attack supervenes on previously existing chronic nephritis."

Other authorities think that in certain cases the changes in the retina come first and the vascular changes are only secondary to them. To quote from Poulard⁽¹¹⁾: "En somme les alterations vasculaires" (in retinitis albuminurica) "ne sont pas très marquées, elles n'ont ni la constance absolue, ni l'intensité, ni l'extension qu'on doit exiger des lésions considérés comme génératrice de toutes les autres" (Rochon Duvigneaud).

The last word is with the pathological anatomist. Only the microscope can settle the dispute. Ginsberg⁽⁴⁾, when discussing

the histology of retinitis albuminurica, says: "*Die Blutgefäße sind meist, aber nicht immer verändert Doch können die Gefäße ziemlich normal sein* : Treitel fand nur an einzelnen Venen geringe Verdickung der Adventitia mit spärlicher Einlagerung von Körnchenzellen Jedenfalls dürfen die Netzhaut Veränderungen im allgemeinen aber wohl als entzündlich bezeichnet werden, nicht als einfach degenerativ."

Therefore, if the vascular changes are the cause of the retinitis, they would always be present. But if the vessels can be "fairly normal," evidently they are secondary in time and in importance, especially if we remember that in nephritis death does not occur quickly, but that the toxins have ample time to act upon the retina, before the eye comes to the pathological laboratory.

Therefore I think that we may have in retinitis a real pathological metabolism of the retinal cells; the tissue is highly organized, consists chiefly of nervous elements which cannot proliferate, but only degenerate, and this easily too, and the capillary net is very close and fine.

There is a parallel in arterial hypertension, as discussed previously: Is it the result of interstitial nephritis or is it a pre-existing sign? The same unknown toxin—will it produce first essential hyperpiesis and later interstitial nephritis, or is there one toxin for hyperpiesis and another for interstitial nephritis?

I did not have a definite case of albuminuric retinitis in my series, possibly I was not lucky enough, or more probably such cases are more frequently seen in hospital practice. But in three cases: Nos. 17*c*, 5*b*, and 4*b*, I saw a very fine oedema of the retina. It looked to me like a delicate veil covering the vessels. These can still be seen but dimly. Sometimes it appears as a wide band along some major branch of the central retinal artery, but in my experience it never leads to definite changes in the retinal structure. Sometimes it lasts a week or two, sometimes only a few days. As these cases have generally very markedly degenerated fundi the phenomenon is not easily seen, unless the eye is examined often or the examiner is lucky enough just to happen on it. In case No. 5*b* I proved to be right. Cases Nos. 4*b* and 17*c* are, in my opinion, under grave suspicion, although in No. 17*c* the kidney efficiency test is fairly good.

As a résumé, if a general practitioner has under his care a case of arterial hypertension, he can fairly well judge the question of interstitial nephritis, relying upon frequent urine tests and ophthalmoscopic examination. The kidney efficiency tests are, of course, desirable. But, unfortunately, the general practitioner is seldom in a position to carry them out on his patients, even with the benevolent help of a hospital.

Diabetes Mellitus

The importance to the general practitioner of ophthalmoscopic examination in diabetes mellitus lies, in my opinion, in a different direction. If the urine be sugar-free, with or without insulin, is the patient really safe? In a medical paper, published two or three years ago, an article appeared stating that such was the case and that the blood-sugar estimation is practically unnecessary with insulin injections and frequent and careful testing of the urine. I cannot agree with this opinion. Case No. 4b may be the tragic result of such an idea. Of course the estimation of sugar in the blood will settle the matter. But again, as with kidney efficiency tests, can it be easily performed in general practice? It is expensive; it must be performed often. And the hospitals, busy with their own work, cannot help the general practitioner much in this matter. But if the retina be only arterio-sclerotic in a mild degree, without haemorrhages, which occur so often, or without retinitis, and if the blood pressure be not high, he can feel more or less safe. If he sees retinal haemorrhages, he must be on guard, and the question will arise whether the dose of insulin is sufficient, especially if the blood pressure be rising. But if, in spite of proper anti-diabetic treatment the blood pressure continues to rise, especially the diastolic pressure, and the state of the retina grows worse, haemorrhages becoming larger, I would be very alarmed lest interstitial nephritis is not coming into play. I know how difficult it is to differentiate between the various forms of retinitis, and how closely they may resemble each other, but I think that even a general practitioner, knowing the clinical facts, as seen and easily determined in routine surgery work, may have a fair and just idea of what is happening to his patient, if he will combine all his investigations.

Prognosis

"Involutionary arterio-sclerosis results rather in the contraction of the spheres of mental and bodily activity than, as with hyperpiesis, in the imminence of the fell sergeant, Death—death by apoplexy, by cardiac defeat or by intercurrent acute pneumonia." (Allbutt's words, quoted by Langdon Brown.) "Pressures of 190-200+ convey a threat and those of 220+ mean that the sword overhead is suspended by a hair." (Green, page 486.) In the opinion of Marcus Gunn: "the prognosis in cases with advanced arterio-sclerotic changes is unfavourable." And he states that all his cases were fatal. This is, of course, the case; the prognosis is very serious indeed. Therefore the following case I consider as worthy of special attention. Mr. R., male, aged 63 years, was

seen at the out-patient department of the London Jewish Hospital in 1923, with retinal haemorrhages in the right eye. The preliminary diagnosis was thrombosis of the central vein? Still he did not develop the picture of venous thrombosis. I followed him up for a few months: fine haemorrhages in the central part and in the periphery of the retina (both eyes), urine 1010, no sugar nor albumen. The systolic pressure, by accident, was not recorded, but I remember that it was well over 200, near to 250. Diastolic pressure, written down in his history, was 140. According to the rule the man must die, and in a relatively short time. But, neglecting the rules, he discharged himself from hospital. In the course of a few months the haemorrhages disappeared. The old man lives near me and is puzzling me as he is behaving in a quite normal and healthy manner. I examined him a few weeks ago. His age now is 66 years. His systolic blood pressure is 150, diastolic 90. He is carrying on his work as a tailor and he tells me that he feels better than he did three years ago. *Fundi*: translucency of the vessels gone; small vessels are tortuous. In the left eye a small artery, going to the macula, is nearly obliterated and the corresponding vein also. No signs of retinal haemorrhages now. In my opinion this case shows that Batty Shaw is right; there is a special toxin (or a combination of them?) peculiar to essential hyperpiesis, and it is pre-renal in its origin. The toxin disappeared, very luckily for the patient, and the body has gone back to normal. But some retinal changes are left, and these are permanent. Of course there is no question of primary kidney disease in this case.

O'Hare and Walker say: "The finding of retinal arterio-sclerosis in patients with a normal or low blood pressure indicates the existence of a previous hypertension with a *subsequent myocardial weakening or loss of vascular tone*" (Foster Moore⁽¹⁰⁾). The case of R. and many others, as analyzed previously, contradict this. In many cases the explanation is more simple: the toxin is gone, either as the result of rational treatment or spontaneously, the hyperpiesis disappears, leaving permanent changes in the retinal vessels, but the heart and vascular system are quite good, from a clinical point of view, in a better state than when hyperpiesis was present. What can be said of the nature of this toxin?

I mentioned before that, in my opinion, the case of R. does show that Batty Shaw is right in thinking that the toxin is of pre-renal origin. But, to quote Foster Moore again: "Batty Shaw believes that the difference between the arterio-sclerotic retinitis and renal retinitis is one of degree only; he believes that the haemorrhages, the white patches and slight changes in the disc, met with in arterio-sclerotic retinitis, differ only from those met with in renal

retinitis, in their being called into existence by the slower operation of the blood-poisons, or by a decreased local tissue sensitiveness, or by the smaller amount of such poison arriving at the scene of action in the former case, as compared with the latter."

I think that the following case is unique and will clearly contradict this view of Batty Shaw: My panel patient, D., male, aged 42 years, always enjoyed good health and had always led an abstemious life. On March 28, 1926, he had a severe haematuria without any pain. Being afraid that the haematuria might be of malignant nature, I sent him with a letter to a hospital. I got a reply that most probably it was a case of hypernephroma. On May 26 he came to see me after a successful operation for removal of the left kidney. On July 6 I saw him again after he returned from the Convalescent Home. On July 18 I examined his eyes: translucency of the vessels gone, light reflex dotted, copper-wire colour of the arteries. Wide white stripes accompany the arteries and especially the veins over the disc and adjacent retina. *R.E.*—Change in the calibre of the inferior retinal artery in one spot and very tortuous small vessels. Large veins are crushed by the arteries, the medium ones only depressed. The macula is pigmented, is of yellow-white colour and flattened. Vision= $4/4.5$. *L.E.*—The same as the right; vena retinalis inferior is deflected *centripetally* by the crossing artery. Commencing maculitis; vision= $4/6$, patient told me that he always had bad vision in this eye. General health good, weight 11 stone. Arterio-sclerosis of the first degree of the brachial arteries. Blood pressure 150/90, 145/95, 140/85: urine normal, specific gravity, 1018-1020: Wassermann negative. His renal efficiency for the right kidney was normal (report from the hospital).

Of course it is my fault that I examined his eyes so late. But I did it only by chance, when coming to the end of my present investigations. Well, I think we can call his fundus an advanced clinical arterio-sclerosis of the retinal vessels (not athero-sclerosis), especially in view of the macular changes. But he never showed any oedema or exudation in the retina and his vision is good. His blood pressure is not high; arterio-sclerosis of the first degree one would expect at his age. Evidently the hypernephroma so upset the endocrine activity of the supra-renal gland (and kidney too?) that the retinal vessels have undergone serious and permanent changes in a short period of time without involvement, in the same degree, of the peripheral vascular system. Of course it is quite possible that he has an atheroma of the aorta or an arterio-sclerosis of the cerebral vessels, as it is impossible to investigate them. But as far as the peripheral vessels are concerned there is no reason to think that they are diseased, as they are soft enough to palpation and the blood pressure is not high.

If we compare his case with experiments on animals, atheroma is much less probable than arterio-sclerosis. One would expect in this case, if Batty Shaw be right, a picture of renal retinitis, not of arterio-sclerotic retinitis. On the other hand, it seems to me, this case does illustrate that there is an intimate (hormonic?) connection between the activity of the kidneys and retinal vessels (or cerebral vessels too?).

The only explanation that I can give of this case is the following: the hyperplasia of the supra-renals may lead to an excessive secretion of adrenaline; this excess of adrenaline can be neutralized in the blood or not. If not, then it can favour the precipitation of ionic calcium. Experimentally arterio-sclerosis can be produced in animals by injections of adrenaline. "Comme action sur le métabolisme signalons encore l'infiltration calcaire de la tunique *moyenne* des artères (arterio-sclérose expérimentale) consécutive aux injections répétées d'adrenaline" (Gley⁽⁵⁾). Unfortunately I never took his blood pressure before the operation nor was this done at the hospital.

Conclusions

(1) Sclerosis of the retinal vessels is recognized first of all by the loss of translucency of the vascular wall; other symptoms develop later. In a normal and healthy person this sclerosis may begin very late indeed, and may not be present even in advanced age, when the vessels of the other parts of the body are already stricken with arterio-sclerosis, if the blood pressure be normal.

(2) The same toxin which is the cause of essential hyperpiesis quickly develops arterio-sclerotic changes in the retinal vessels, even at a young age, if this arterial hypertension continues long enough. Its action may cease, and *clinically* the general vascular system may recover completely, but the arterio-sclerotic changes in the retinal vessels remain permanently.

(3) There is some reason to believe that the toxin of essential hyperpiesis is pre-renal in origin, but renal retinitis and arterio-sclerotic retinitis are probably caused by different toxins. It is probable that there is some intimate connection between the state of the retina and activity of the kidney (endocrine?).

In conclusion it is my pleasant duty to express my thanks to Mr. A. H. Levy for permission to use many of his cases and for his valuable assistance in the preparation of this paper.

ABBREVIATIONS USED.

A.	-	-	-	-	-	Arteries.
A. R. INF.	-	-	-	-	-	Arteria retinalis inferior.
A. R. S.	-	-	-	-	-	Arteria retinalis superior.
B. E.	-	-	-	-	-	Both eyes.
C. W.	-	-	-	-	-	Copper wire.
DITTO	-	-	-	-	-	Idem.
F. N.	-	-	-	-	-	Fundus perfectly normal.
L. R.	-	-	-	-	-	Light reflex.
L. V.	-	-	-	-	-	Large vessels.
L.	-	-	-	-	-	Left eye.
N.	-	-	-	-	-	Normal.
R.	-	-	-	-	-	Right eye.
TR. DIM.	-	-	-	-	-	Translucency diminished.
TR. PR.	-	-	-	-	-	Translucency present.
TR.	-	-	-	-	-	Translucency of the vessels.
TR. AB.	-	-	-	-	-	Translucency absent.
V.	-	-	-	-	-	Veins.
V. D.	-	-	-	-	-	Veins depressed.
V. D. CF.	-	-	-	-	-	Vein deviated centrifugally.
V. CR.	-	-	-	-	-	Veins crushed.
W. INT. BL.	-	-	-	-	-	Without interruption of the blood stream.
W. S.	-	-	-	-	-	White stripes.
♂	-	-	-	-	-	Male.
♀	-	-	-	-	-	Female.

ANALYTIC TABLE.

	Normal (29 cases).		Moderate hyper- piesis (56 cases).		Diab. mell. (8 cases).		Advanced hyper- piesis (14 cases).	
	No.	%	No.	%	No.	%	No.	%
Tran. present	26	100%	—	—	—	—	—	—
Tran. partly pres.	—	—	2	3%	1	12%	—	—
Tran. absent	—	—	54	97%	7	88%	17	100%
L. ref. dotted	—	—	24	43%	8	100%	16	94%
Copper w. art.	—	—	23	41%	8	100%	15	88%
Silver w. art.	—	—	—	—	—	—	3	18%
Lumen of the vess. changed ...	1	4%	8	15%	—	—	8	47%
Tortuosity of the vessels... ..	8	30%	22	39%	3	37%	9	53%
Veins depressed	3	11%	17	30%	—	—	1	6%
Veins depressed and deflected ...	2	8%	5	9%	1 (centri- petally)	12%	—	—
Veins cr. w. int. bl.	—	—	15	27%	2	25%	7	41%
Veins crushed with int. bl. ...	—	—	8	15%	1	12%	5	30%
V. cr. and deviated centrifugally	—	—	5	9%	—	—	1	6%
V. cr. and deviated centripetally	—	—	2	3%	1	12%	1	6%
W.s. accompany the vessels on the crossing points only ...	1	4%	8	15%	1	12%	3	18%
W.s. accompany the vessels over the disc	2	8%	15	27%	1	12%	4	23%
W.s. accompany the vessels on the retina... ..	—	—	3	5%	—	—	6	35%
Gener. narrow. of the vessels ...	—	—	5	9%	—	—	6	35%
Discs not sharply outlined ...	—	—	2	3%	—	—	2	12%
Oedema of the retina	—	—	—	—	1	12%	1	6%
Old ret. haemorrh. (pigment) ...	—	—	—	—	—	—	2	12%
Fresh ret. haemorrh.	—	—	1	1½%	2	25%	4	23%
Exud. in the macula	—	—	—	—	—	—	2	12%

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
1	16	♂	100	60	B.e. f.n. No tr. of the l.v. In the medium and small vessels tr. pr.	N. 1015
2	17	♀	105	55	Ditto	N. 1012
3	14	♂	—	—	Ditto	Not examined
4	25	♂	130	90	Ditto	N. 1025
5	19	♂	—	—	Ditto	Not examined
6	7	♀	—	—	Ditto	Not examined
7	18	♂	—	—	Ditto	Not examined
8	22	♂	110	85	Ditto	Not examined
9	13	♀	—	—	R. A large branch of the a.r.s. is crossing twice the corresponding vein. V.d. at the first crossing	Not examined
10	18	♂	130-140	80-95	Father suffers from a high degree of essential hyperpiesis; he is insane now. B.e. tr. pr. in medium vessels. V. prominent. One medium branch of the a.r.s. depresses the vein on crossing it; v.d. cf., disc but tr. pr. in the artery. No w.s. at the point of crossing	—
11	26	♀	140	95	R. One small artery above macula is tortuous (spastic?) Tr. ab. on one medium branch of a.r.inf. Veins are not depressed. L.r. normal. B.e. Retina very sensitive to light	N. 1015
12	58	♂	190	90	B.e. Tr. ab. on the arteries, but still pr. in the veins (especially the r.e.) L.r. altered (?) Slight tortuosity of the small vessels	N. 1015
13	65	♂	130	75	Bad pyorrhoea; deaf; hard arteries (arterio-sclerosis II degree). B.e. Tr. pr. in most of the vessels; when crossed by an artery, the vein is not crushed and its shape is not changed; only a w.s. accompanies the art. on the point of crossing. Slight tortuosity, if any, of medium arteries and arterioles	N. 1015
14	60	♂	115	60	Arterio-sclerosis II degree. Spontaneous haemorrhages (right leg intramuscular; r. episcleral). Urticaria. Carbunculus of the neck. B.e. tr. pr.; r. one large v. is accompanied by w.s. when crossing the disc. Changed calibre of one small (macular) artery	N. 1018
15	26	♀	130	80	B.e. tr. pr.	Not examined
16	28	♂	145	90	Haematuria suddenly 7/12/24; no surgical cause revealed in a hospital. Since then in good health. B.e. tr. pr.	N. 1020, persistent albuminuria
17	39	♂	140	90	F.n.	N. 1020

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
18	33	♂	155	100	Vertigo. B.e. tr. dim. on the arteries. Veins are very full, the smaller v. are sometimes tortuous. L.r. is very strong	N. 1020
19	54	♂	160	90	Vertigo; heavy smoker. No central col. scotoma present. B.e. tr. pr.; small arteries are tortuous	N. 1015
20	51	♂	150-160	100	B.e. tr. pr.; small arteries are tortuous, L. W.s. accompany the vessels, espec. the v. when crossing the disc	Traces of albumen, N. 1012
21	29	♂	150	105	B.e. tr. ab. from the larger arteries, nearly absent from the smaller; all arteries are tortuous in the L. V.d. by crossing arteries, but not crushed. L.r. very strong. R., V. superior is crossed by the artery and v.d. cf. on the retina and near the disc; L., the same on the disc	—
22	37	♀	110	85	Advanced and partly uncompensated valvular heart disease, cyanosis, enlarged liver, extra systoles. Auricular fibrillation in the past. B.e. tr. pr.	Not examined
23	24	♂	115	60	B.e. tr. pr. v.d. by crossing arteries	Not examined
24	37	♂	140	90	B.e. tr. pr.	Not examined
25	24	♂	125	80	B.e. tr. pr. v.d. by crossing arteries	Not examined
26	23	♂	140	75	B.e. tr. pr.	N. 1015
27	30	♂	105	60	Advanced tbc. of the left lung. B.e. enlarged pupils, tr. pr.	N. 1016
28	45	♂	125	70	General weakness. Pyorrhoea. Gastric ulcer (?) Arterio-sclerosis II degree. B.e. tr. pr. Slight tortuosity of the macular arterioles (?)	—
29	42	♂	125	90	Soft arteries, b.e. tr. pr.	N. 1015
1a	45	♂	130	75	Arterio-sclerosis II degree. B.e. tr. ab.; arteries very tortuous, especially the cilio-retinalis R. L.r. dotted. C.w. arteries. R. V. retinalis inf. crushed by the artery. W.s. accompany the artery, when crossing the vein and over the disc	N. 1012
2a	61	♂	160	95	B.e. tr. ab.; w.s. accompany the arteries over the disc. L.r. dotted. C.w. arteries. Tortuosity of the vessels	N. 1015
3a	63	♂	160-130	100-85	Septic nose, septic tonsils. Orbital periostitis of nasal origin. Signs of Graves's disease (intestinal auto-intoxication) (?). B.e. tr. ab.; slight tortuosity of the small vessels (?). Otherwise normal	N. 1014
4a	56	♂	175	100	B.e. tr. ab.; w.s. accompany the vessels over the disc; v.d., but not crushed by the arteries. R. One cilio-retinal artery, going to macula, is very tortuous	N. 1020

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
5a	35	♀	130	80	Chronic phlebitis of both legs. Puerperal sepsis in the past. Perihepatitis now. Wassermann negative. B.e. The outline of the disc cannot be seen clearly. Tr. ab. in most vessels. Otherwise normal	N. 1015
6a	34	♂	140	100	3 years ago Wassermann posit. Right pupil enlarged with a slight reflex on light, but good reflex on accommodation. B.e. "choked" (?) discs in the past; now their outline is not clear; tr. ab.; tortuosity of the small macular vessels; w.s. accompany the vessels over the disc	N. 1012
7a	48	♂	150	100	Vertigo; b.e. tr. ab.; tortuosity of the small arteries. Retina very sensit. to light. L. One of the smaller branches of the ar. ret. inf. is crushing the v. ret. inf. with an ampulliform dilatation of its distal end. Arterial and venous pulse. Tension normal	N. 1015
8a	55	♀	170	100	B.e. tr. ab.; arteries are very tortuous, especially the small ones; their lumen is changeable. The veins are rough; they are not crushed by crossing arteries. C.w. arteries. L.r. dotted	N. 1010
9a	29	♂	145	70	Active tbc. of the right apex, b.e. tr. ab.; v.d., but not crushed, with some enlargement of the v. on the spot of crossing; L. w.s. accompany the lower branch of ar. ret. near and over the disc; R. A small spot of old chorio-retinitis. B.e. Some of the small arteries are spastic	Not examined
10a	52	♂	155	100	B.e. tr. ab.; arteries are slightly tortuous, c.w. colour. L.r. dotted. V. cr. by crossing arteries. The lower branch of the a. ret., near the disc, is crushing the corresponding large vein, so that the venous pulse can be seen only from the disc till the crossing point. W.s. accompany only one or two large arterial branches, on the R. also one medium macular branch	N. 1012
11a	65	♂	160	70	Bronchial asthma, extra systoles of the heart. B.e. tr. ab.; tortuosity of the small arteries. C.w. colour. L.r. dotted. V. cr. by the arteries, without interruption of the bl. stream	N. 1003

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
12a	47	♀	170	100	Sterility; lost her menses 14 years ago. B.e. tr. ab.; v. cr. by the arteries, without interruption of the venous pulse. Medium tortuosity of the vessels. W.s. accompany the arteries over the disc. L. a cilio-retinal artery, going to the macula, shows a change in its lumen	N. 1010
13a	42	♂	170	110	Severe headache; b.e. tr. ab.; v. cr. by the arteries w. int. bl. Slight tortuosity of the arterioles. W.s. accompany the arteries over the disc	N. 1025
14a	69	♂	150	90	Attacks of minor uraemia(?) B.e. tr. ab.; v.d. by the arteries, but not crushed. W.s. accompany the arteries over the disc. Very slight tortuosity of the vessels	Traces of albumen. N. 1007
15a	66	♂	165-180	85-95	Emphysema, slight cyanosis R. Tr. ab.; v. cr. by the arteries, with an enlargement of the distal end of the v.; slight tortuosity of the arteries. A spot of old chorio-retinitis under the macula. L. One small branch-artery shows a change of the lumen; the same with a vein. V. cr. more than in the R. White dots in the central part of the retina Tr. ab.	N. 1012
16a	60	♂	150-160	85-90	R. Cataract immature. L. Tr. ab.; the vessels are rather narrow. Otherwise normal	N. 1015
17a	50	♀	170	100	Tinnitus; systolic murmur of the heart. B.e. tr. ab.; tortuosity of the small vessels. W.s. along the vessels over the disc, especially along the arteries; sometimes on the periphery too, but not on the whole length of the vessels. C.w. arteries. L.r. dotted	Traces of albumen. N. 1018
18a	60	♀	160	85	A huge fibromyoma uteri; hard of hearing. A profuse and dangerous haemopt. few weeks ago. Probable cause arterio-sclerosis (X-ray of the chest), b.e. tr. ab.; slight tortuosity of the vessels. Otherwise normal	Traces of albumen. N. 1015
19a	70	♀	(?)	(?)	Paralysis agitans, reading of the bl. pres. very difficult and uncertain. Tinnitus. B.e. tr. ab.; medium tortuosity of the small and medium arteries. L.r. dotted. Otherwise normal	Not examined
20a	55	♀	160	85	Gall-stones; systolic murmur of the heart. B.e. tr. ab. of the arteries, but not of the veins. (L.) Otherwise normal	N. 1015

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
21a	56	♂	155	95	Bronchial asthma. B.e. myopia 6.0 D.; myopic crescents; very narrow vessels. Tr. ab.; slight myopic changes in the macula	N. 1012
22a	27	♂	150	75	Soft brachial and radial arteries. B.e. tr. ab.; v.d., but not crushed. Otherwise normal	N. 1016
23a	50	♀	140-155	70-90	Gall-stones: giddiness. Arterio-sclerosis I degree. B.e. tr. ab.; v. are not depressed. Increased tortuosity of the arterioles. Otherwise normal. L. A small flame-shaped fresh haemorrhage from a small arterial (?) branch near the disc	N. 1012
24a	72	♀	160	110	B.e. tr. ab.; the vessels are narrow, not tortuous, c.w. colour. L.r. dotted. V. cr. by the arteries. Increased pigmentation and red-yellow colour of the fundus	Traces of albumen. N. 1002
25a	51	♀	150	100	Arterio-sclerosis II degree (?). B.e. tr. ab.; v. cr. and v.d. cf., but not dilated. W.s. accompany the vessels over the disc. Tortuosity and change of lumen of the cilio-retinal arteries. L.r. dotted	N. 1015
26a	62	♂	170	100	Carcinoma ventriculi (?). B.e. tr. ab.: v.d. (crushed ?). L.r. dotted. W.s. accompany the arteries over the disc	N. 1015
27a	48	♂	<div>3 years ago</div> <div>175-180 90-100</div> <div>now</div> <div>190 120</div>		B.e. myopic 10 D.; the arteries are very narrow, c.w. colour; the v. are larger. Tr. ab.; v. are not depressed	N. 1015
28a	55	♀	170-190	90-95	Graves's disease 3 years ago, improved now. B.e. tr. ab.; L.r. dotted; c.w. colour. Lens opacities. L. A small branch of the a.r.s., joining the main trunk on the disc, is crossing the v.r.s., considerably crushing it with partial interference of the circulation. R. The temp. branch of a.r.s. is twice going round the corresponding vein, and the vein is not crushed	N. 1015
30a	57	♂	145	95	Syphilis 2 years ago. Arterio-sclerosis I degree. R. lens opacities; tr. ab.; c.w. arteries, l.r. dotted. L. tr. ab.; c.w. arteries, l.r. dotted; v.d. only, not crushed by the artery, but d. cf.; v., but not the a., are changed in their lumen	N. 1012
31a	46	♂	140	80	Arterio-sclerosis I degree; tr. ab.; v.d. slightly; otherwise normal	N. 1008

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
32a	49	♂	110	80	Tbc. of the lungs; laryngitis tbc'osa. Soft arteries. B.e. tr. ab.; c.w. arteries; the lumen of the small v. is changed. V. are not crushed. R. an area of old chorio-retinitis in the lower part of the fundus. L. small vessels are crushed, but not deflected	—
33a	60	♂	155	85	B.e. tr. ab.; c.w. arteries; no tortuosity of the vessels; v. cr. R. ampulliform dilated. W.s. accompany the a. over the disc and on the points of crossings. L.r. dotted	N. 1020
34a	64	♂	160	90	Paralysis agitans of the right arm. B.e. tr. ab.; l.r. dotted; narrow arteries and v., but their lumen is not changed; v. cr., but not displaced	N. 1010
35a	45	♀	170	95	Sterility; lost her menses, 3 years ago. B.e. tr. ab.; l.r. dotted; v. cr. and d. cf.; w.s. accompany the a. over the disc	N. 1007
36a	49	♂	135	95	Severe headache; soft arteries (?) B.e. tr. ab.; v. cr. w. int. bl. W.s. accompany the a. on the crossings and over the disc. L.r. dotted. R. One v.d. slightly cf.	N. 1005
37a	55	♂	135	95	Arterio-sclerosis I degree; b.e. tr. ab.; v. cr. by the a., but w. int. bl.; l.r. dotted (?)	N. 1015
38a	50	♀	120	75-90	I knew her for 4 years, always with low syst. pressure. Arterio-sclerosis II degree. Septic tonsils, enteroptosis. B.e. tr. ab.; l.r. dotted (?). Otherwise fundus normal. L. Part of the v. cr., even slightly d. cf.	N. 1010
39a	60	♂	170	100	B.e. tr. ab.; large v. on the disc, cr. by the a., w. int. bl.; and without deflection. L.r. dotted; w.s. accompany the a. on the crossing points. R. Tortuosity of the arterioles in the area of the macula. L. Tortuosity of two small cilio-retinal arteries	N. 1012
40a	55	♀	190-210	90-110	B.e. tr. ab.; v.d. sometimes slightly d. cf. R., but not cr. L.r. dotted. Otherwise normal	N. 1008
41a	50	♂	140	100	Angina pectoris (?). Arterio-sclerosis I degree. B.e. tr. ab., congenital tortuosity of the vessels, chiefly of the large and medium; l.r. dotted; c.w. colour. Slight change in the lumen of some arteries. (R. cilio-retinal artery.) V.d. slightly. L. but not cr. and not d.	N. 1015

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
42a	47	♂	110	90	Arterio-sclerosis II degree. Coronary thrombosis b.e. tr. ab.; c.w. colour. W.s. accompany the vessels over the disc. Lumen of the small cilio-retinal a.r. changed. Large v. tortuous, v. cr. d. cf., w. int. bl. L.r. dotted	N. 1022
43a	53	♀	145-160	95	B.e. tr. ab.; c.w. colour. L.r. dotted, w.s. accompany the a. on the crossings. V. cr., without deflection, but sometimes with interference with the blood circulation. (L. Venous pulse on the disc, interrupted by the crossing; ampulliform enlargement of the <i>proximal</i> end of the vein)	N. 1015
44a	43	♂	150	100	B.e. tr. ab.; v.d. but not cr. Otherwise normal	Not examined
45a	33	♀	150	100	Hard hearing (sclerotic). Arterio-sclerosis II degree. B.e. tr. ab.; v. cr., but w. int. bl. W.s. accompany the arteries on the crossings. Otherwise normal	N. 1002
46a	72	♂	155	90	Arterio-sclerosis II degree. B.e. tr. ab.; c.w. arteries. L.r. dotted (?). V. cr. w. int. bl.; w.s. accompany the a. on the crossings. Venous pulse. R. Surprisingly slight tortuosity of the vessels. L. Venous pulse on the v.r. inf., can be seen very far away from the disc	N. 1005
47a	50	♂	155	95	B.e. tr. ab.; c.w. arteries, l.r. dotted. Posterior choroiditis (slight myopia). Tortuosity of the larger veins. Sometimes the small arterioles are tortuous too. V. cr., w. int. bl.; L. V. ref. super. tempor. is cr. and d. <i>centripetally</i>	N. 1015
48a	52	♂	170	120	B.e. tr. ab.; c.w. arteries, l.r. dotted; v.d., but not crushed. R. A small spot of old chorio-retinitis	—
49a	49	♂	130	85	Heavy smoker, arterio-sclerosis I degree. B.e. tr. ab.; normal colour of the arteries, l.r. normal. V.d. slightly. W.s. accompany the a. on the crossings	—
50a	54	♂	165	95	B.e. tr. ab.; c.w. colour (?), l.r. dotted (?). V.d., but not crushed. L. V. ret. inf. d. centrifugally	N. 1020

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
51a	52	♀	165	100	B.e. tr. ab.; c.w. colour; l.r. dotted. V. cr. w. int. bl. and sometimes d. (L. One vein d. <i>centripetally</i> .) R. W.s. accompany the artery on the periphery, but not over the disc. A small vein coming from above and independently crossing the disc, shows venous pulse and forms a loop, which is lying much deeper than the rest of the vessels. L. a spot of old chorio-retinitis near the disc	N. 1010
52a	40	♂	155	95	B.e. tr. ab.; c.w. colour (?), l.r. dotted (?). V.d. only slightly, or not changed at all. Otherwise normal	—
53a	56	♂	155	95	Bronchial asthma, myocardial weakness. B.e. tr. ab.; c.w. colour (?). L.r. dotted. V.d.	N. 1008
54a	29	♂	180	105	B.e. myopia, over 10.0 D., a. narrow, v. much larger. Tr. ab., v. cr. slightly, w. int. bl. Posterior choroiditis	N. 1020
55a	48	♀	160	95	Tinnitus, b.e. tr. ab.; l.r. dotted; c.w. colour (?). V.d. and sometimes slightly deflected, w. int. bl. No tortuosity of the vessels	N. 1015
56a	36	♀	160-165	95-100	2 last children stillborn. When first seen, P-126, bl. pr. 140-60; slight oedema of both ankles, enlarged liver. Wassermann neg. Now P-60, bl. pr. 160-165, 95-100. R. tr. ab.; except in one small art. branch, where the vein is considerably deflected centrifugally, but still can be dimly seen. L.v. are crossing the a. B.e. otherwise normal	—
1b	51	♂	170-140	100-90	Diabetes mellitus discovered 4 months ago. B.e. tr. ab.; but not of the veins. R. A medium art. branch is covered by a medium vein and can be seen comparatively well. L. A branch of the a.r. sup. is going under the vein, then twice over it and then again under it, and can be seen through the vein in the last crossing. V.d. but not cr. c.w. colour (?). L.r. dotted (?). W.s. accompany the vessels only on crossing points. L. A vein is deflected <i>centripetally</i>	N. 1012

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
2b	61	♀	190, 160, 160, 150	90, 70 90, 85	Tinnitus. B.e. tr. ab.; tortuosity of the large and medium arteries. L.r. dotted (?). C.w. colour (?)	Traces of albumen. N. 1020
3b	56	♀	170, 210, 175	95 100 95	Diab. mell. for the last few years. Sugar free, if careful in diet. B.e. tr. ab.; slight tortuosity of the small vessels. C.w. colour (?). L.r. dotted (?). One would pass her fundus as normal for her age	Traces of albumen. N. 1012
4b	58	♀	210	100	Suffered from diab. mell. for years. 3 years ago was put on insulin, when being in hospital, and remained under medical supervision. Her eyes were never examined all the time. I first examined her eyes in February, 1926. B.e. old and fresh retinal haemorrh., large ones; tr. ab.; c.w. colour. L.r. dotted. White exudation in the macula, with haemorrhages in it. V. cr. w. int. bl. Very fine oedema of the retina, discs not quite clear, and not sharply outlined. I was under the impression that she was going to develop interstitial nephritis. 2 months later her bl. pr. was $\frac{164}{70}$ B.e. practically the same, except that the fine oedema of the retina was gone and the discs were of normal outline. Her blood urea was 0.25 mgrm. %; her bl. sugar 0.143. With 30 units insulin a day bl. sugar 0.092. She developed signs of hypoglycaemia; insulin was stopped and her bl. sugar rose to 0.225. With 10 units daily 0.216. On the 30/6/26 she turned comatose with sugar and traces of acetone in her urine. 80 units of insulin same day; urine free from sugar and acetone. Died in a comatose state 48 hours later. 24 hours before death sudden clonic fits of all limbs and twitching of the face. Hemiplegia of the right side. No. P.M. performed. Probable cause of death cerebral haemorrhage or uraemia	—
5b	57	♂	190	100	Diab. mell. discovered first time now; b.e. tr. ab.; c.w. colour. L.r. dotted. V. cr. w. int. bl. L. W.s. accompany the v. ret. inf. over the disc. R. The temporal branch of a.r. sup. is crushing the vein; the vein is deflected <i>centripetally</i>	—

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
6b	50	♀	200-220	100	Three years ago first seen with a severe neuritis. Glycosuria. Consultation. Insulin injections started. Bl. pr. was $\frac{150-160}{90}$. In spring 1925 the eyes ex. for the first time; many fresh haemorrh. were seen, old chorio-retinitis. Fine oedema of the retina. Bl. pr. $\frac{220}{100}$. Specific gravity of the urine 1005, suggested interstitial nephritis; different opinion of a consultant. Few weeks later he agreed that she had interstitial nephritis. Now bl. pr. $\frac{200-210}{100}$, last May $\frac{240}{130}$. B.e. no oedema of the retina. Fresh and extensive retinal haemorrh., chiefly the round ones, old chorio-retinitis. (Autumn 1925.) B.e. tr. ab.; c.w. colour sometimes silvery arteries, l.r. dotted. R. Veins are crossing the a. L. V. cr. def. cf.; change of the lumen of one artery, when crossing a focus of exudation. The macular part of the disc is blurred. B.e. retin. haemorrh., old and fresh one, extensive, round and flame-shaped; foci of fresh and old exudation all over the retina, chiefly in the central part, with complete atrophy of the retina (autumn 1926)	—
7b	45	♂	150	100	Diab. mell. 2 years now. B.e. tr. ab.; l.r. dotted. C.w. arteries. V. are not deflected and not crushed by the arteries. Very slight tortuosity of the vessels	N. 1010
8b	60	♀	240	120	Diab. mell. many years. B.e. tr. ab.; c.w. colour; l.r. dotted. V. depressed, but not cr. No tortuosity	Sugar + 1015
1c	70	♂	200	100	Retrosternal pain; cannot walk. B.e. tr. ab.; c.w. colour; l.r. dotted. Surprisingly slight tortuosity of the vessels. W.s. accompany all vessels, including the cilio-retinal arteries, on the crossing points, and over the disc. A. on the periphery are rather narrow. V. cr. with interference with the bl. stream	N. 1015

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
2c	57	♀	270-210	140-125	B.e. tr. ab.; c.w. colour; l.r. dotted. Tortuosity of the small and medium a. W.s. accompany the arteries on and near the disc. R. A small striated haemorrh. near the disc. L. Change in the lumen of a few small arteries, independently going upwards from the disc.	N. 1002
3c	48	♀	235	120	B.e. tr. ab.; c.w. colour; l.r. dotted. Tortuosity of the medium and small arteries (macular area!) is marked in the highest degree ("cork-screw" of de Schweinitz). W.s. accompany the large arteries. V. cr. w. int. bl. R. A small branch of the a.r. sup. is crushing the v. ret. sup. Blood urea proved to be 76 mgm.	Traces of albumen. N. 1007
4c	46	♂	220-295	120-145	Weight 15 stone; tall, heavy man. When first seen he had galloping rhythm of the heart. Sent, after eye examination, to see a heart specialist, who agreed with the diagnosis of essential hyperpiesis. B.e. tr. ab.; a. are narrow, c.w. colour; l.r. dotted. Medium branches are tortuous with a change of lumen. The discs are not sharply outlined. R. The lower vein is covering the artery without crushing it; w.s. accompany it over the disc. L. The lower vein is nearly completely crushed by the artery, which crosses it in an oblique direction, venous pulse being seen only from the disc till the point of crossing, and the distal portion of the vein is enlarged; the upper artery is crossing the vein in two places, but does not crush it completely, so that the venous pulse, although feeble, can be seen in the periphery	Traces of albumen. N. 1012
5c	50	♀	220	130	Pendulous belly; murmur cordis (systolic?). B.e. tr. ab.; c.w. colour; l.r. dotted. W.s. accompany the vessels on the points of crossings. Crossings are very few indeed, but the v. are not depressed even. The patient is very difficult to examine	—
6c	70	♂	200-210	90-100	B.e. tr. ab.; c.w. colour of the a.; l.r. dotted. Venous pulse. V. cr. w. int. bl. Very slight tortuosity of the vessels. V.d. centrifugally	N. 1002

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
7c	52	♂	200-225	110-120	B.e. tr. ab.; the a. are tortuous, especially the macular ones; w.s. accompany some of the ar. branches. Tortuosity of the a. increased in the L. so that the difference with the R. is striking. Change in the lumen of the arteries. C.w. colour; l.r. dotted. V. are full; v. cr. w. int. bl.	N. 1015
8c	47	♂	160-210	110-130	B.e. tr. ab.; l.r. dotted; c.w. arteries. R. Small cilio-retinal arteries are tortuous, their lumen changed. All a. are narrow. V. cr. w. int. bl., but not deflected. L. A vein. is cr. and d. <i>centripetally</i> . W.s. accompany the a. on crossing points	Traces of albumen. N. 1022
9c	54	♀	220-270	100-120	Seen in consultation a few weeks ago. Retinal haemorrhage (?). Admitted to the hospital; bl. urea normal. On the third day developed symptoms of thrombosis of the frontal cerebral lobe. Was discharged in a comatose state with her right leg paralyzed. Now incontinence of the faeces and urine gone; limb movements restored. Mentality—very much confused, very slow. R. Myopia 4.0 D.; tr. ab.; c.w. arteries, l.r. dotted; the a. are narrow, with change of the lumen; v. more normal. In some of the cilio-retinal arteries at places the blood-column is completely interrupted; they are of silvery colour. Old haemorrhage (pigmentation) near the disc, in the direction of macula. A white exudate in the macula. W.s. accompany the vessels on the disc and some distance on the retina. V. cr. very much, with interference with the blood stream. L. Myopia 17.0 D, narrow vessels, faintly seen arteries. V. cross the arteries and are not deflected. Change in the lumen of the veins. Posterior choroiditis. Died 5 months later from cerebral thrombosis	—
10c	68	♂	210	105	B.e. tr. ab.; c.w. colour; l.r. dotted. V. cr. w. int. bl., but not d. Slight tortuosity of the vessels. R. A huge block of pigment in the macula with a fresh haemorrhage in the centre of it	Not examined

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
11c	54	♀	200	110	Sterility; gout; b.e. myopia 13.0 D.; post. choroiditis. Tr. ab.; c.w. colour; l.r. dotted, narrow arteries, large veins. V.d. by crossings. Opacities of the lens	N. 1005
12c	56	♀	210	110	Known to me for 5 years. Started with attack of minor uraemia (head-ache, epigastric pain, sickness, "air-hunger," fainting). B.e. tr. ab.; c.w. colour; l.r. dotted. V. cr.; but without interference with the blood-circulation. L. A striking change in the lumen of one vein All v. are more crushed than in the right eye. R. The small a. are tortuous	Traces of albumen. N. 1000-1005
			200	90		
			210	100		
			190	100		
			190	90		
			180	90		
			240	110		
13c	50	♀	220	110	Known to me for 4 years. First stroke 3 years ago with the involvement of the n. facialis dexter, only. The second stroke 2 years ago, with hemiplegia sinistra. The majority of the movements restored by now. Last year's kidney efficiency test—good result. B.e. tr. ab.; c.w. colour; l.r. dotted. Slight tortuosity of the a. V. cr. moderately, w. int. bl. L. W.s. accompany one artery in the periphery. A small spot of chorio-retinitis near the macula	—
			260	125		
			290	140		
			300	150		
			310	160		
			290	170		
			260	150		
14c	50	♂	210	100	Acute nephritis with oedema of the legs 3 years ago. B.e. tr. ab.; colour of the a. is silvery; l.r. dotted. Change in the lumen of the a. W.s. accompany one small branch of the a. ret. sup.; a large white track accompanies the a. ret. infer.	—
15c	65	♀	190-200	110	For the last 2 years suffered from attack of tachycardia. Hysteria. B.e. lens opacities; tr. ab.; c.w. colour; l.r. dotted. A. are tortuous. V. cr. w. int. bl.	N. 1005
16c	67	♀	210-220	110-115	B.e. tr. ab.; c.w. colour; l.r. dotted. Change in the lumen of the a. V. cr. w. int. bl. W.s. accompany the a. on the crossing points. R. A small haemorrhage over the disc from a small artery?. L. Old arterio-sclerotic maculitis—pigmentation, white exudation, flattening of the macula	Traces of albumen. N. 1010

No.	Age	Sex	Blood pressure		Ophthal. appear.	Urine
			Syst.	Diast.		
17c	48	♀	1925-7 { 250 210 300 280 1926-7 { 250 310 240 250	110 130 150 150 140 160 140 150	<p>In spring 1925 the diagnosis of essential hyperpiesis was made by a consultant. Later on, when under my care, was seen with retinal haemorrhages, arterio - sclerotic (far advanced) fundus, occasional oedema of the retina. I suggested interstitial nephritis. Admitted to hospital August. 1925. Report, "specific gravity of the urine 1020, traces of albumen, few epithelial and granular casts. Bl. urea on admission 0.3% and renal efficiency test were fairly good. Her arteries were extremely hard, and fundi oculorum showed silver-wired arteries, haemorrhages and exudation into the discs." This spring she had again her kidneys tested in another hospital. Report, "suffering from gross cardio-vascular changes. Her kidneys are comparatively good. Her bl. urea is normal and her urea concentration test quite satisfactory." Tested again the 3rd time. On the 16/7/26 in the morning, urea gr. xxx given at 10.30 a.m. 1st specimen taken 11.30; 2nd at 12.30; 3rd at 1.30. Total quantity of urine for 24 hours nearly 2000 gram., specific gravity 1005. Pathological report:—No. 1 urea, 0.4%; No. 2, 0.7%; No. 3, 0.8%. Blood urea (blood taken at 2.30 p.m.) 17 mgrm. %. <i>General health.</i>—Except the first attack 18 months ago with sudden loss of speech for a few days, with full consciousness, is fairly good. Headache, palpitation of the heart. Was a very fat woman; by now her weight is 13 stone. B.e. transluc. gone; arteries are of silvery colour, especially the smaller ones. The vessels are very tortuous. The veins are crushed. Large haemorrhages (1/3 or 1/4 disc) are seen from time to time, chiefly between the disc and the macula, and smaller one in the periphery. They usually disappear without leaving any trace, and do not appear again for weeks or months. The vision is impaired. Characteristic is the oedema of the retina. Sometimes, at intervals of weeks, the disc and the retina round it, with the vessels, are seen covered with a very fine veil; the discs are then a little swollen, their outline not clear. The oedema may persist for a week or so, and may disappear completely in two or three days</p>	—

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ANNOTATION

Miners' Nystagmus

There have recently been many communications published on the subject of miners' nystagmus. Dr. Freeland Fergus, whose practical experience of the disease entitles his opinion to considerable respect, states his views at some length in the *British Medical Journal*, June 18, 1927 (an abstract of the paper will be found on page 525 of this number). He rejects the findings of the Nystagmus Committee respecting the defective light theory or at least regards them as not proven. He points out that the introduction of electric light has not always been followed by any improvement in the nystagmus statistics, and even in some cases nystagmus seems to have increased. He says: "No doubt we may be told that it is because the best type of electric lamp has not yet been procured and the coal owners may be called upon to face further great expense on that head. I think that before any such demand is made of them, there should be a much more thorough investigation into the cause of the disease, which investigation would include a well organized collective investigation throughout the entire coal fields of the United Kingdom. Such a research ought unquestionably to be in the hands of persons competent to deal with vital statistics and with public health." We trust he does not desire another Commission. Professor Haldane and Mr. Lister Llewellyn reply to this criticism of the work for which they were largely responsible in a very temperate letter (*Brit. Med. Jl.*, July 2, 1927). They point out that the evidence that the essential cause of the disease