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COMMUNICATIONS

RETINITIS NEPHRITICA OR ALBUMINURICA

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(Continued)

In my opinion, both the kidney tests and the clinical examination are one whole, one picture, and it would mean destroying the latter by eliminating one part. The same applies to the ophthalmoscopic examination; but it remains one of the most important of renal tests, because the bio-chemistry of the tissues in the living body is incomparable in delicacy to any test that can be carried out in the laboratory; and after all, roughly speaking, the increase of urea in blood simply means an incomplete oxidation by the tissue cells.

Some important theoretical and practical applications of the foregoing conclusion assert themselves; de Schweinitz states that "In general terms it is likely that the renal disease must be present for some months before retinal lesions appear." In my opinion, hyperpiesis appears first of all—de Schweinitz does not mention this at all—most probably pre-renal in origin, and, early with it, retinal changes and the *onus probandi* that the renal lesion precedes the retinitis lies with de Schweinitz. It is quite probable that they are simultaneous in their course and origin. The above criticism also applies to Swanzy's statement that "The retinal changes are the result of the renal disease."

In diabetes the ophthalmoscope may reveal three conditions: the hyperpietic fundus with sclerosis of the vessels, the same fundus with sclerotic retinal exudation, and true albuminuric retinitis. The first is fully described and discussed in my two previous papers. To the second and third varieties everything that was said in connection with benign and malignant nephrosclerosis fully applies, oedema of the retina in combination with raised blood pressure being a true sign of true interstitial nephritis as distinct from the advanced renal sclerosis of hyperpiesis. Viewed from this stand-point, diabetes, pre-eminently complicated by vascular disease, may give rise to true interstitial nephritis, but not so often as is generally believed. McDonagh, for instance, considers that "In most cases of diabetes there is a varying degree of interstitial nephritis" (p. 147, Vol. II). Instead of interstitial nephritis, I should say vascular disease, nephro-sclerosis, and moreover usually of a benign hyperpietic type. As previously explained, I cannot agree with Leber's opinion, expressed on page 915, that, with a large degree of probability we can take it for granted that the disease of the retina starts only when the disorder of the renal function has already caused a rise of blood pressure. Nor can I agree with Duke-Elder who writes on page 191: "We have seen that the basis of pathology of all three (arterio-sclerotic, diabetic, and renal retinitis) is in all probability the same." Arterio-sclerosis—if he means involutionary arterio-sclerosis—is a disease of the retina quite apart, causing "retinitis" by atheroma of the arterioles and capillaries; hyperpietic retinitis is quite different from renal, and the diabetic form assumes an intermediate position between the two.

There is one pathological condition in which we can decide with a great degree of precision all the above controversies—toxaemia of pregnancy. If we examine the patient regularly and carefully we can exclude the question of a previously existent renal lesion. Kahn, for instance, believes that the kidney is an important *aetiological* factor in toxaemia of pregnancy and that it has usually been previously affected in childhood, puberty or adolescence. De Condia considers that the kidney is impaired in pregnancy. By means of the phenolphthalein test and the estimation of diastase in the serum and urine, he found that in the later months of a normal pregnancy there is a definite diminution of renal function, most marked at term, definitely increased during labour, and quickly returning to normal within the first days of the puerperium. If the pregnancy had been complicated by nephritis, the impairment of function persisted after labour and pointed to a permanent damage of the kidney. But have we any right to presume a hidden nephritis during a pregnancy, when blood pressure is normal, the fundi and urine are normal and the personal history negative?

Yet, quite suddenly, albumin appears in the urine and the blood pressure rises. (In one of my cases within three weeks the blood pressure increased from 130/90 to 190/105, commencing fundus changes were observable, and traces of albumin in the urine were present). The condition grows worse, but completely regresses, if the pregnancy is interrupted early, with a return to "normal." If allowed to go too far, permanent pathological changes will be left in the cardio-vascular system.

Is the kidney the cause of all the mischief? Certainly not; it is the pregnancy, whether it be the placenta, the foetus or a kind of intolerance to the foreign protein of the male, etc. The renal lesion can be demonstrated in the majority of cases. Of course, it can, but so can the vascular, hepatic and retinal changes also. And again we see the appearance of retinitis albuminurica only in combination with hyperpiesis. Since we know the aetiology here, we can cure the patient by removing the evil. In the case of essential hyperpiesis we know but little, and some help is possible, even to a considerable degree if the case be taken in hand early, but the outlook of true interstitial nephritis is hopeless at the present time, because we do not know anything at all about the causative agent. Moreover, the course of the disease is deceptive. A patient may have hyperpiesis with little albuminuria and satisfactory renal tests, and yet he may be a case of true interstitial nephritis, or malignant nephro-sclerosis; it is the condition of the retina that will tell the true state of affairs, but sometimes not immediately. Careful and repeated examinations are necessary. If this be so, then a new field entirely unexplored is opened up in the case of surgery generally and of genito-urinary surgery especially. A greater call is made on the efficiency of the kidney at some time or other, such as immediately after an operation. The patient has to recover from the effects of the anaesthetic—possibly he is in a state of acidosis, of shock, or of excessive reabsorption of his own proteins, and it is quite probable that many a bad recovery or even death is the result of relative kidney insufficiency, unsuspected before the operation. A routine ophthalmoscopic examination, which is quicker, less disturbing and less expensive than the elaborate kidney tests, would help considerably in eliminating this danger. In genito-urinary surgery the renal tests are always performed and the ophthalmoscopic examination is omitted. If the kidney efficiency tests are unsatisfactory, the operation is postponed or altogether rejected. Often enough a careful clinical examination of a prostatic case is made before his operation—past history and physical condition, state of kidneys are investigated, a search is made for foci of infection, renal tests are performed, and complete blood, X-ray and cystoscope examinations made. But the fundus is not often examined.

Yet the ophthalmoscope can supply useful information as is illustrated by three cases in my own limited experience.

K. M., aged 65 years. Enlarged prostate. Blood pressure 205/120. Blood urea: 34 per cent. B. E. translucency lost, copper wire colour, light reflex dotted. Some of the arteries are of "non-true" silver-wire colour. Change in the lumen of the small arteries. The veins are very crushed by the arteries, "hour-glass" contracted, centrifugally deflected. R. E. The sup. temp. vein is very tortuous. A large flat haemorrhage over the artery. Afterwards the blood pressure came down to 170/90, blood urea being 24 per cent. L. E. Idem. Judging by the fundi he was not a suitable subject for operation, but he was not operated upon, as there was a strong suspicion of malignancy of the prostate.

T. M., aged 66 years. 1927.

Blood pressure 220/120. Enlargement of the prostate. Heart enlarged. Urine normal, sp. gr. 1002. B. E. translucency lost; copper wire colour, light reflex dotted. White lines on the crossings. Veins are sometimes crushed. Change in the lumen of some veins. Then gradually the blood pressure became 185/95, 180/80. A few days afterwards he disappeared. In the meantime he had an attack of "uraemia" and was admitted to hospital. There he was diagnosed as a case of "enlarged prostate and secondary renal inefficiency." Blood urea was 72 per cent. and the urea concentration test: second hour—1.05 per cent.; third hour—1.11 per cent. Phenosulphopht. test: first hour—a faint trace in 140 c.c. of urine; second hour—a faint trace in 150 c.c., third hour—a faint trace in 150 c.c. Three days afterwards he was discharged or discharged himself. A month later he was taken bad again, and, on my insistence, was operated upon (a two-stage operation); the blood urea being 97 per cent. before the cystostomy and 54 per cent. soon afterwards. He made a good recovery and is now in a good state of health, the blood pressure being 160/90; fundi status idem.

K. M., aged 83 years.

Was admitted for a few weeks in a hospital for operation of enlarged prostate. After being there for two weeks he was discharged, the surgeon deciding not to operate as the kidney tests were below normal. When I first saw him, he shewed definite signs of catheter-cystitis and was in great pain. Blood pressure 120/75. B. E. fundi only mildly sclerotic without any oedema or retinitis. His cystitis quickly improved under appropriate treatment. Then I advised removal of his prostate, which was carried out at another hospital, in a two-stage operation with a satisfactory result. He lived for three and a half years and died of uraemia without any rise of blood pressure or any changes in his retinae.

Aetiology

The aetiology of hyperpiesis, which is so closely connected with both forms of nephro-sclerosis, is still undecided. The most widespread theory is the mechanical one, the original view of Allbutt. It implies that there is always some obstruction or narrowing in the peripheral vascular system which makes the heart work more intensive to raise the blood pressure and overcome the difficulty. Cushing argued that with sclerosis of the vessel wall the blood pressure must be increased for the same amount of fluid to be filtered off from the vessel into the tissues. In Price's text-book the same explanation is applied to sclerosis of the glomeruli and even of the renal vessels generally: "Sometimes the renal vessels suffer first, necessitating a rise of blood pressure to force sufficient blood through them." The same opinion with slight variations will be found in the majority of text books. Even Elwyn believes that the hypertrophy of the heart is the result of raised blood pressure. The exponents of the colloid theory are not agreed between themselves as to the pathogenesis. Fisher believes that vascular disease is the cause of hyperpiesis, and not *vice versa*. He is very much against the theory of the renal origin of hyperpiesis: "Blood vessel disease, high blood pressure and cardiac hypertrophy are not secondary to the loss of kidney function" (pp. 24-27). The loss of elasticity by the vessels and their contraction increase the work of the heart; to this may be added the greater viscosity of the blood, when its acidity is increased, especially after strenuous muscular work. The cause of vascular disease for Fisher is always the same—patchy sclerosis of the vasa vasorum, produced by microbic infection (Rosenow's experiments). On the other hand he mentions that a high protein diet will considerably raise the blood pressure in experimental animals and that oedema of the brain, especially of the medulla oblongata, will produce an enormous hyperpiesis; but he does not seem to pay much attention to this. McDonagh, like Fisher, deplores the introduction into the nomenclature of the term "hyperpiesis." It is not even a condition *sui generis* (p. 211). There is no essential difference between arterio-sclerosis and hyperpiesia: "Arterio-sclerosis denotes fibrosis of the vessel wall and hyperpiesia—that the protein particles are undergoing dehydration in the fibroid vessel" (p. 210). Hence, the cause of raised blood pressure is interstitial nephritis" (p. 218). "The destruction of the glomeruli by narrowing the richest capillary network in the body causes a rise in the blood pressure." (p. 249). "Hyperpiesis is due to lysis and not to a rise in the viscosity of the blood" (p. 15, Vol. I), and, even more, "Lysis is accompanied by a low viscosity . . . aggravate the hyperpiesis" (Ibidem). I understand that McDonagh teaches us that the viscosity of the

blood has nothing to do with hyperpiesis and that in his hyperpietic cases, successfully treated by insulin, the viscosity was diminished and the blood pressure lowered at the same time. Hyperpiësis precedes chronic nephritis and is, therefore, "independent of the renal changes" (p. 196, Vol. I). Rather a surprising sentence, to my mind, in view of his own opinions as stated previously. The agents that produce lysis will quickly make the blood pressure rise. "Therefore, the hyperpiesis is due neither to renal sclerosis nor to capillary fibrosis. These two conditions are merely the result of the continued action of the agent which causes the lysis" (p. 198). An affirmation contradictory to his own theory that two factors are necessary for hyperpiesis—dehydration and contraction of an end-capillary network. "The hyperpiesis is due to the protein particles undergoing lysis, which, at the same time, occasions the vascular changes. It is not the hyperpiesis which is responsible for the vascular changes, or the vascular changes for the hyperpiesis" (p. 203, Vol. I); an opinion diametrically opposed to that of Fisher. It seems to me, to put it briefly, that in his first volume McDonagh advocates the chemical theory and in the second volume a combined chemico-mechanical theory of hyperpiesia. Fine as all this mechanical explanation of hyperpiesia appears it does not stand a detailed criticism.

The uniform explanation of all forms of sclerosis by Fisher or McDonagh does not correspond to facts. Rosenow, a first-class authority, produced atheroma or athero-sclerosis (Marchand) by injecting various microbes, cultivated from the tonsils, gall-bladder, etc., into animals and shewed that there exists a certain affinity to definite organs. That atheroma is produced in the large arteries by disease of the vasa vasorum is a well-known fact in the case of mesaortitis syphilitica. Rosenow thus explained the patchy character that is so characteristic of atheroma. And for primary atheroma this explanation is most probably correct. Sir James Barr (*Brit. Med. Jl.*, Sept., 1928), writes that longitudinal straining of the arteries, when the pulse-wave is passing, especially in the case of the greater curvature of the aorta, leads to atheroma through degeneration of the deep layers of the intima. Important only is the difference between the systolic and diastolic pressures (evidently not their actual heights). Hence the calcareous degeneration of the mouths of the coronary arteries and of the large vessels arising from the greater curvature of the aorta. But, in my opinion, if only the pulse pressure is important, then in aortic incompetence it is one of the greatest, and in my limited experience cases of coronary thrombosis usually have either a normal blood pressure or they are hyperpietic with not an excessive pulse pressure. On the other hand I have been able to

shew longitudinal straining of the arteries in the retinae of many normal persons, and yet by itself it does not cause atheroma, at least in the retinal vessels. The mechanical moment of longitudinal straining perhaps plays a rôle, but a subordinate one. Atheroma in the retinal vessels is of the same character as elsewhere in the body—patchy. But it may often be secondary to hyperpiesis, the continuous high pressure wearing down the intima—what I call “secondary” atheroma. Investigations of Hertel, Ralhman, etc., and my own have shewn that involutionary (senile) sclerosis is not patchy, but represents a widespread disease of the vessels, approximately of the same degree everywhere. In essential hyperpiesis I was able to demonstrate, at least in the case of the retina, that the disease is not so patchy as in atheroma and, moreover, that it is chiefly a sclerosis of the perivascular sheath. In retinitis albuminurica degeneration of the vascular wall itself is much more prominent (“non-true” silver-wire artery) and it is also not patchy; usually the whole wall of the vessel is involved and not only a part of the circumference, as it may be in the case of atheroma. And so far as my knowledge goes there is not sufficient clinical, pathological, or anatomical evidence to postulate the unity of all vascular disease except on purely theoretical considerations.

Loss of elasticity of the arteries is habitually seen in the aged. At some time—Osler puts it at 40 years—in every person may be detected a widespread degeneration of the arteries in a mild degree. It progresses with time and (say) after the age of 70 we can reckon that the elasticity is completely lost. However, the blood pressure is normal and the heart is not hypertrophied in the aged. Thomson and Todd have examined 102 patients, aged 75–90; in 49, the systolic blood pressure was below 150, in 10 over 190, in 43 between 150–190. The diastolic was below 90 in 79 cases. Dumas, Chevasau and Labry examined 50 persons over the age of 70, 20 of them being over 80. The diastolic pressure exceeded 90 mm. in only five persons. Am I not therefore justified in believing that loss of elasticity cannot be the cause of hyperpiesis or of cardiac hypertrophy?

The viscosity of the blood is, according to Fisher, increased with acidity. In diabetic coma the acidity is certainly increased; therefore, the viscosity ought to be higher and the blood pressure raised. However, the blood pressure is always low. Moreover, we have the opinion of McDonagh that the viscosity of the blood and hyperpiesis are quite independent and may even be antagonistic to one another. It is difficult then to believe that increased viscosity of the blood is the cause of hyperpiesis. See also the opinion of Parkes Weber in “Splénomégalic Polycythaemia with high blood pressure.”

If a sufficient capillary area is cut off from the general circulation, the blood pressure will rise and the heart will work harder. As the kidneys have the most extensive and most important capillary network in the body, sclerosis of a sufficient number of glomeruli (and their arteries) will produce hyperpiesis. This as previously mentioned is the most generally accepted theory. If it be true, then the removal of one kidney and a considerable part of the other in experimental animals ought to produce hyperpiesis. But it does not, and removing both kidneys does not produce it either. These experimental facts alone are sufficient to disprove the above theory. Moreover, clinical evidence of analogous character may be found in Batty Shaw's book on hyperpiesis. "In hyperpiesia the kidneys may be normal" (p. 127). I cannot agree with him when he says, on page 169, "Hyperpiesia may exist and yet the patient during life may reveal little or no symptoms of the disorder . . . no signs or symptoms of cardiac hypertrophy and none of vascular changes." It has been my endeavour in my two previous papers to shew that the retinal vessels are attacked early and present definite changes in the great majority of hyperpietics. Osler points out that "Exceptionally typical contracted kidney has been found unaccompanied by arterio-sclerosis or cardiac hypertrophy. Roth reports six such cases. Death was generally from uraemia. In most of the cases the blood pressure had been low" (p. 899). J. Spence in a notable paper on chronic nephritis in childhood quotes cases of renal dwarfism with severe pathological changes throughout the body and early death from uraemia, but with normal blood pressure. This in my opinion is a clinical analogy to the experimental animal in which both kidneys were extirpated without producing hyperpiesis.

Moreover, there is one pathological condition where we can verify all this theorizing—endarteritis obliterans or thrombo-angeitis. Here we have all the mechanical factors that are supposed to be the cause of hyperpiesis throughout a considerable portion of the vascular tree. Throughout the whole extent of one or even both femoral arteries the elasticity is lost and the lumen is narrowed, and, as the change takes place gradually not suddenly, it gives the heart an ample opportunity to raise the blood pressure and to hypertrophy. The condition usually occurs in the case of a man of middle age, with a good and strong heart. The disease usually develops without any signs of cardiac debility. The blood pressure in my cases was always normal, although sometimes the oscillometric index taken on the thigh near the saphenous opening was 0.25 or 0.5, instead of three or four as on the other leg. The only objection to quoting endarteritis obliterans may be the fact that not all the vascular system is involved, but only a part of it.

But through one femoral artery there is surely more blood flowing than through even both kidneys. If so, why is a satisfactorily functioning kidney, as tested by ordinary clinical examination, renal efficiency tests and the ophthalmoscope, probably the cause of hyperpiesis, whilst a femoral artery with its lumen reduced to half its normal, is not? This concludes my brief criticism of the main points of the mechanical theory.

The only way out of the impasse in our state of imperfect knowledge is offered by the chemical theory. At this point of the discussion, it is not important to determine the nature and origin of the toxin, whether it be an endocrine disturbance, an exogenous influence, or an auto-intoxication. Supposing we have a chemical agent that will exclusively excite to their maximum contraction the muscles of the vascular system. This would explain, first of all, the factor of inheritance, "the vital rubber" of the arteries (Osler), the vascular system being of peculiar composition in certain individuals and, therefore, more open to attack. Then it would explain the contraction of the peripheral arteries and the hypertrophy of the heart, produced simultaneously by the same cause, the heart muscle responding by increased work which will cause hypertrophy. Exactly the same would happen to the arteries. After years of toxic contraction they will become hypertrophied, the "myo-hypertrophy" of Pal. In involutionary senile sclerosis the elasticity of the arteries is lost; therefore the heart has no help in propelling the blood; the peripheral arteries are simply distended by the systolic wave and this additional burden does not cause the hypertrophy of the heart. It fares quite differently with a hyperpietic artery; being in a state of toxic contraction, it does not distend under the pressure of the systolic wave—more than that—it actually opposes such a distension. Therefore the heart not only loses the co-operation of the arteries in maintaining the circulation, as in a case of senile sclerosis, it meets an actual and very effective opposition from a hyperpietic artery. Moreover, a vicious circle will be established and another powerful factor will be brought into play. With narrowing of the arteries and toxic contraction of their muscles the supply of blood to the tissues will be lessened. A partial oxygen starvation and increased irritability of the medulla oblongata will immediately arouse to action the respiratory and vasomotor centres. The contraction of the arteries will be increased by the vasomotor influence to force as much blood as possible to the head. The heart will also be subjected to the same influence and will therefore be under a double burden. The presence of vasomotor nerves in the cerebral and retinal vessels is still debatable, some denying (L. Brown) and others affirming the fact (Aubaret et Sedan). The brain will be kept well supplied with blood and, consequently, with

the toxic agent too. Therefore, the cerebral and retinal vessels will suffer more and shew the disease earlier than any other part of the body, excepting perhaps, the kidneys, a fact that is well known in the clinical history of hyperpiesia and which, I hope, I have succeeded in proving in the case of the retina in my two previous papers. Moreover, Bordley and Bexer have published the results of completed and detailed post-mortems in 24 patients. A large proportion of their cases shewed renal changes of the chronic type, some of the patients having had, when alive, normal blood pressure, others having been hyperpietics. Of the latter some shewed very slight renal changes or none at all, but in all of them was present a well-marked sclerosis of the vessels of the medulla oblongata. Of the cases with normal blood pressure not one shewed the presence of medullary sclerosis. In this country Parkes Weber holds a similar view. This is the angiospastic theory, one of the best exponents of which is Pal. If the tonicity of the muscle increases, it becomes hard, even very hard. In the artery this means a hardening of the muscularis media and it may become permanent. That is why even the artery which is hardest on palpation is not so frequently found to be in a state of organic disease, as is commonly believed. Pal goes so far as to say that the hardness of the radial artery is usually not due to calcification. Hypertonia is a hardness of the arterial wall caused by increased tension of its muscle, a functional condition that may lead in time to histological changes. The latter are: myohypertrophy or, occasionally, fibrosis. Hypertonia is usually associated with high blood pressure, but it may exist even with low blood pressure, as in cases of cardiac weakness or loss of blood; that is to say, the blood pressure may suddenly fall, but it does not cause the disappearance of the tonic contraction of the arterial wall. On the other hand, in cases of cerebral haemorrhage, by palpation we may find the arteries on one side of the body (possibly the paralysed side) soft, and on the other side hard. But the blood pressure is, nevertheless, the same throughout the body. Hence it is obvious, according to Pal, that there is a special tonic centre (corpus striatum). Several very illustrative cases of transient hemiplegia have been recently published by Williamson, who ascribes this condition to arterial spasm which has not existed long enough to produce softening in the affected cerebral area. The combination of increased hardness of the arterial wall and raised blood pressure is a most alarming and serious symptom. It is important to realize that permanent hypertension is not due to vascular spasm, but to the inclination of the muscles of the arterial wall to hypertonus. The presumption of the existence of permanent vascular spasm is untenable, but abrupt increases of pressure alone (pressor vascular crises) are of an

angiospastic nature and frequently occur in cases of permanent hypertonus; that is to say, tonus and tension are two different conditions. Many authorities ascribe to the corpus striatum the control of complex motor activities. Kinnier Wilson disagrees with this view and does not think that the corpus striatum controls any complex movements, but that its function is much simpler, namely, that of controlling muscle tone and of inhibiting a neuro-muscular rhythm (*Brit. Med. J.*, Nov., 1928, "Neurological problems of to-day.") Diminution of the capability of dilatation of the small arteries due to hypertonia of their muscular coat is enough to produce high pressure without a considerable reduction of their transverse diameter and without renal changes. Furthermore, arterio-sclerosis and hypertonia are, of course, not identical. Moreover, in chronic high blood pressure we have not only narrowed arteries, but also dilated, hypertonic ones. The peculiar cerebral symptoms in certain persons suffering from hypertonia are due to the fact that cerebral arteries with their weak muscular coats cannot resist the pressure and, consequently, a passive arterial hyperaemia develops in the brain. Primary hypertonia is evidently a neurosis of the angiotonic nervous apparatus, the source of which is in the central nervous system.

My own experience has led me to believe that this theory of Pal explains more or less satisfactorily the known facts, with some exceptions. The toxins attack, most probably, the arterial walls throughout the body, and the central nervous apparatus comes into play later. I have tried to shew that loss of translucency of the retinal vessels is the first and an early sign of essential hyperpiesis. I very seldom, if ever, saw spasm of the retinal arteries, but have often observed contracted and narrowed retinal vessels; I have but little experience of passive arterial hyperaemia of the retina. The loss of translucency is due not to spasm, but to actual and very fine histological changes, which may be so delicate as to be seen only *in vivo* and not to be detected even by means of a microscope. Apart from the unknown cause of essential hyperpiesis, blood infection, such as measles, diphtheria, etc., may cause it. After hyperpiesis has existed for some time definite changes are seen in the retinal vessels, but these are chiefly due to sclerosis or fibrosis of the perivascular sheath, the arterial wall itself playing sometimes only a secondary rôle. That is why I cannot accept so readily the present-day teaching that in the kidney the interstitial tissue plays only a passive rôle, as it is a very active one indeed in sclerosis of the retinal vessels. Only recently have we begun to realize the importance of the reticulo-endothelial system in the general defensive mechanism of the body. Therefore the change is an actual histological one, and not merely an angiospasm. Commencing as an angiospastic disorder

essential hyperpiesis readily causes sclerotic changes in the perivascular sheath and arterial wall, the intima being involved the last (secondary atheroma of hyperpiesis). This pre-eminence of perivascular changes gives us an important clue as to the nature and origin of the toxin. It originally circulates in the lymph and not in the blood. The lymph is composed of the contents of the thoracic duct and the waste products of the local tissue cells. Furthermore, my experience of retinal changes has led me to the conviction that in the fundus abnormal products of the retinal metabolism plus abnormal contents of the thoracic duct mean an oedema of the retina, which is concurrent with true interstitial nephritis. In the purely vascular disease—essential hyperpiesis—the metabolism of the retinal cells is, in my opinion, not abnormal, although they may suffer as the result of insufficient oxygen supply. Therefore, perivasculitis is caused by the lymph from the thoracic duct; the lymph is chiefly the result of chemical processes inside the alimentary canal and in its wall.

To understand better how such a toxin may be produced we must go back to the work of Vaughan. His experimental results have been especially gratifying to me, as I have for a long time felt that the theory of infection is overdone in the modern tendency to explain by it nearly every pathological process, although I am fully aware of the importance of microbic invasion. By injecting animals with different products of protein hydrolysis, Vaughan has demonstrated that microbes are chiefly particulate, specific proteins, and that all true proteins or their split products contain a poisonous group, which is *physiologically* identical with microbic toxins. "At first we found that the cellular substance of certain pathogenic bacteria could be split up with liberation of a poisonous substance; then we tested non-pathogenic bacteria and then animal and vegetable proteins, all with the same result. Not only do all of these contain a poison, but so far as its gross effects on the higher animals have been studied, the same poison. We have held that when we know more about these poisonous bodies, obtained from all proteins, it will be found that chemically they are not identical, but physiologically they are so closely similar that up to the present time we have not been able to distinguish one from another by the symptoms induced." Vaughan found a complete physiological identity between the toxins of the typhoid bacillus, egg-white and edestine of hemp-seed; they all "kill animals in the same doses, with the same symptoms and with the same lesions." "This is striking evidence of the similarity in the structure of the protein molecule, whether it be of bacterial, animal or vegetable origin . . . The theory unfolds itself, all proteins are constructed on the same basis and contain a chemical nucleus, archon, or key-stone. This is the poisonous group and

is practically the same in all proteins. One protein differs from all others in its secondary and possibly its tertiary groups. In these lies the specificity of proteins. Living proteins function through their secondary and tertiary groups. When the primary group is detached from its own subsidiary and specific groups, it manifests its poisonous action through the avidity which it has for secondary groups of other proteins. These are thus detached from their normal positions and consequently the living protein is deprived of its capability of functioning normally." It follows from this theory that any foreign protein which grows and multiplies in the body of an animal may poison its host by interfering with the normal cycle of protein metabolism. "It is the protein poison and it is physiologically the same whatever its source, whether it comes from cocci, bacteria, spirilla, or protozoa. The specificity which characterizes the infectious disease is not due to the poison formed, but to the protein cause and the specific ferment produced." Vaughan believes that it is a biological law that when a living cell is invaded by a foreign protein it destroys the latter by elaborating a special ferment. He has succeeded in dividing the split products of protein and bacterial lavage into poisonous and non-poisonous parts. By injecting the non-poisonous part into an animal he developed in it a high state of immunity against the corresponding living bacteria; by injecting the poisonous part an immunity against their toxin. A new aspect in our knowledge of chronic infection has been opened up by these researches. It is not necessary for the invading microbe to be always present and to attack repeatedly and periodically the host. It is quite possible for it to upset to such an extent the local metabolism of the protein cells in one or even several organs of the body, that a vicious circle is formed. The protein of the cells being deprived of its secondary or tertiary groups will rob the neighbouring cells, and so begins the vicious circle. The split products of this imperfect protein metabolism will continuously maintain the body in a state of chronic intoxication. "Scalds and burns that are going to do badly develop, before any symptoms are noticed, a severe acidosis, and apparently the toxæmia is due not to a bacterial infection, but to the breaking down of the damaged tissue, *i.e.*, it is due to histamine poisoning, or to something akin to it. Later on, undoubtedly, microbes may cause complications" (Kirk, *Brit. Med. Jl.*, Aug. 1928). "The amount of oxygen given was largely determined by the amount of acid in the urine. An interesting point in connection with anoxæmia is that symptoms do not appear until late, and once they appear the patient goes downhill very rapidly, almost as if he had fallen over a precipice." (*Ibid.*) The problem is the more attractive, if Fisher is correct when he points out the extreme chemical affinity of many

amido-acids and fatty acids, the formula for the first being

$$\begin{array}{c} \text{X} - \text{COOH} \\ | \end{array}$$

and for the second $\text{X} - \text{COOH}$, X being the NH_3

unknown nucleus, identical for both. A very good illustration of the correctness of Vaughan's views is presented by the Wassermann reaction, when it remains persistently positive, although all the syphilitic virus has left the body. Not being, as is well known, a specific reaction, it depends in cases of syphilis most probably on a special chemical *status quo* of the plasma, resulting from the mutual reaction of the body of the host and the invading spirochaete. "Protein particles once altered may perpetuate the change, although the agent producing it in the first instance vanishes and there is always a tendency for the physico-chemical changes the protein particles undergo to be cyclical, independent of the activity or otherwise of the syphilitic micro-organisms" (McDonagh). "The persistence of nephritis does not appear to depend on the continued presence of septic focus or of an infectious organism" (Acute Nephritis, *Brit. Med. J.*, Sept. 1928).

Up till now everything mentioned above applies equally to hyperpiesis and to interstitial nephritis. But here the path divides. In hyperpiesis the origin of the condition is in my opinion probably situated in the delicate chemistry of the gastro-intestinal tract, as evidenced by the predominating influence of perivascularitis in the hyperpietic retinal vessels (as previously explained), and by the daily experience of clinical medicine that rest and restricted diet may considerably lower for a time the raised blood pressure. For interstitial nephritis no such evidence exists and in my opinion it is more probable that the latter is caused by a parenteral invasion of the pathogenic agent, as in my case mentioned below, where the skin-wound was most probably the site of entry. The aetiology of the toxæmia of pregnancy, when the latter is accompanied by renal retinitis, may also furnish the cause of interstitial nephritis. It would be purely guesswork in our present state of knowledge to discuss whether the pathological lesion is situated in the kidney itself or somewhere else in the body, or even throughout the organism. Only a very careful and complete clinical examination of every patient with an injury or a septic focus, when first seen, as compared with subsequent events in his clinical history (so far as the migratory habits of patients will allow), can clear up this important medical problem.

A new and promising field of investigation was recently opened up by the researches on vitamins by Rowland, Cramer, and others. They found that if animals were fed on foods deficient in some vitamin the mucosa of the gastro-intestinal tract began to slough

and the bacillus coli and other intestinal flora not only invaded the lymphatic spaces of the intestinal wall, but penetrated outside it and were found even in the liver and other important organs. Larimore treated five cases of chronic ulcerative colitis by means of a diet rich in vitamins. All the cases shewed a good healing reaction of the intestinal mucous membrane, and the X-rays indicated progressive improvement. On the other hand Arnold by experimenting on animals came to the conclusion that the injection of large quantities of protein or sudden alkalinization of the upper part of the intestine in the presence of foreign protein interfered with the normal bacteria-killing power of the intestine. As a result of this diminished resistance the bacteria were able, in certain instances, to pass through the intestinal wall and reach the blood stream. Cruickshank made a survey of all these problems. "Nothing has emerged from the experimental, chemical or bacteriological work which has given any support to the view that as a result of bacterial action in the intestine poisonous substances are elaborated, which by their entry into the tissues of the body produce chronic intoxication." Brook, Murphy, Maxwell, Dragsted and others have proved that in acute intestinal obstruction the close loop of intestine contains some split products of bacterial action, which are extremely poisonous for the body when introduced parenterally, but the intestinal mucosa presents an efficient barrier, provided its *vascular supply* is not damaged. An animal may for a time remain in good health, but as soon as distension of the isolated intestinal loop interferes with the vascularization of the mucosa, toxic absorption rapidly occurs, or perforation results. (These recent experiments are the most brilliant proof of the theory of vascular crises and of Oertel's clinical facts about gangrene of the intestine when spasm attacks the mesenteric artery). The above evidence furnishes another possibility of how the body of a hyperpietic may be invaded by intestinal flora. Cruickshank believes that poisonous action depends not only on bacterial toxæmia, but on the split products of food stuffs, and that "the results of even complete obstruction in the lower levels of the intestinal tract, in so far as they are due to bacterial poisons, are extremely slow to appear."

Another fresh discovery is the fact that a patient's blood may inhibit the growth of intestinal flora obtained from a focus in his own body. This obviously depends on the reaction of the body as a whole against the bacteria that can penetrate the intestinal wall even in health. In spite of the local immunity of the intestinal mucosa, the existence and importance of which has been so ably demonstrated by Besredka, Cronin Lowe in a very important paper "Pathogen-selective cultures as an aid to the diagnosis of infective foci" points out that the above method enables one to detect

pathogenic bacteria not only of the intestinal flora, but even of other foci of infection (nasal accessory sinuses, tonsils, etc.) A negative result would shew that the particular focus is not the cause of the disease, or that it is a purely local infection without any influence on the body as a whole.

The oldest of my patients with albuminuric retinitis was 50 years of age. Is it a coincidence? Or is it a general experience that retinitis albuminurica is not seen, *e.g.*, after 60 years? If it be so, it would signify, in my opinion, that with progressing age the body becomes immune to the particular form of infection that causes true interstitial nephritis.

Let us suppose, for argument's sake, that the kidney really possesses those anatomical peculiarities in its structure which I propose on purely theoretical grounds; that the tubular cells are situated on a strong mesodermic membrane which plays the rôle of a barrier not easily penetrated, like the perivascular sheath of the retina; external to this membrane would be a perivascular lymphatic channel and on the thin outer wall of the latter an arterial capillary or a capillary network, the separating wall being very thin and chiefly represented by the intima of the capillary. A tubular cell is busy at its inner end in contact with the urine coming from above from the glomerulus in absorbing and excreting certain constituents of the urine, and for the purpose of our present discussion it is not necessary to know the exact nature of these constituents. At its basal or outer end the cell is in contact with the mesodermic membrane and so far as the latter allows with the perivascular lymphatic space. It would absorb the necessary elements from the lymphatic fluid and it would discharge therein the products, if any, of its metabolism. Blood, then, would reach the tubular cell only after passing through many membranes and after mingling with lymph in a common vessel. The *vis a tergo* being completely or practically exhausted, it would be left to the local activity of the tubular cell to take what is necessary for its life and function from this mixture of fresh nutritious material and waste products of its own metabolism. If a toxin with a special affinity attacks the tubular cells alone their disturbed activity will make the urine decidedly and characteristically pathological. It is quite possible, however, that the mesodermic membrane will prevent a reabsorption of the products of this pathological metabolism into the perivascular canal and so into the general circulation. The capillary network will not be involved, as the toxin has a special affinity for the tubular cells; in short, the condition will be clinically "nephrosis." If the vascular system only be affected, including the glomerulus, the afferent arteries and the capillary network, the tubular cell will escape and will carry on a normal function

although half-strangled by the insufficient oxygen supply and ready to break down if too much burden be suddenly added to its ordinary work; clinically it will be nephro-sclerosis. If a toxin attacks both the tubular cells and the vascular system, we shall witness a disturbance of metabolism of the former, aggravated and complicated out of all proportion by the additional affection of the latter. For the whole body the result will be similar; all the tissues will suffer from the vascular disease and from insufficient oxygenation brought about by the retention of waste products usually eliminated by the kidney, plus intoxication from the perverted metabolism of the tubular cells themselves. If in nephrosis the tubular cells discharge their poisons into the urine and the body escapes, in true interstitial nephritis they eject them into the general circulation and the urine is relatively normal. My supposition that interstitial nephritis is probably caused, at least in the beginning, by a parenteral bacterial infection, finds some support in the "patchiness" of the kidney affection, which is also characteristic of atheroma of the vessels, as shewn by the experiments of Rozenow. Of course, this imaginary outline of renal disease leaves many questions still unanswered especially from the physiological point of view. It seems to me, however, that it corresponds better to the facts supplied by clinical examination of the patient and especially of his *réтинаe*.

S., male, aged 45 years. First examined early in 1922. A perfectly healthy man. Fundi normal. Blood pressure 140/85. In 1924 met with an accident and had a large wound of the right calf. It took a few months for the wound to heal up. He was examined many times then and nothing abnormal was found. Was working all the time, feeling "fine." Three years later I was called to see him because of palpitation and dyspnoea. Blood pressure 310/150, enlarged heart, systolic murmur at the apex. B. E. Fully developed retinitis albuminurica with neuro-papillitis. Blood urea 126 per cent., phenol-sulpho-phthalein in two hours, 6.2 per cent. Urea concentration test 1.3 per cent. Urine albumin +; granular and hyaline casts, erythrocytes; sp. gr., 1012. Just over a month later he was dead.

By "nephro-sclerosis" I understand a vascular disease of the interlobular and afferent arteries, the glomerulus, the efferent artery and the capillary network formed by the latter on the outer side of the tubule. Supposing all this system is affected to a moderate degree and a sufficient supply of blood is secured for the glomerulus and tubular cells, the latter being intact and healthy, or perhaps only one part of this miniature vascular tree is involved but still allows a considerable flow of blood, the kidney will function satisfactorily for many years, even decades. The condition is exactly the same fine and beautiful process of vascular

sclerosis that I discussed and described in the case of the retina and that is most probably identical in all parts of the body. There may be some differences which are dependent on anatomical peculiarities at present still unknown. It is the benign form of nephro-sclerosis, as clinically it is benign in the retina and in the case of all the body, where as a rule the intact parenchyma may function satisfactorily for years in spite of even advanced vascular sclerosis. This means that in the kidney this benign form of sclerosis may be diffuse, attacking all the nephrons but in a moderate degree, and bringing into constant work a larger number of nephrons than are usually in active work in a normal kidney during a definite period of time. The reserve power of the kidney is enormous and this means that a kidney that is able to carry on will pass successfully any chemical tests; only the sphygmomanometer and the ophthalmoscope will be able to give warning. This is the type of case which with care may live for decades, but such a patient will stand a severe shock badly, whether it be an injury or a surgical operation—the “vascular type” of patient. The vascular disease may attack one part of the nephron’s vascular tree and interrupt the blood supply either to a great extent or completely. The nephron will work slower and slower and in time will collapse altogether. The other nephrons, however, are still left sufficiently intact and sufficiently alive to keep the patient clinically in the category of benign nephro-sclerosis. Such a process will be “patchy” and, moreover, I have seen it in the retina and have described it, quoting also an anatomical paper of Morax to the effect that in the same miniature vessel one part of the circumference is considerably diseased and the remainder is intact. For argument’s sake, let us suppose that an interlobular artery be completely blocked in the same way as I have described in the case of the retina under “Change of the lumen of the vessels,” then all the dependent nephrons will perish in time. If the same were to happen to the afferent artery, the corresponding glomerulus and tubules would be affected. If the efferent artery is blocked, the glomerulus will function satisfactorily but the tubules will be affected. By analogy with what we see occurring in the retina we can be practically certain that exactly the same process, *caeteris paribus*, is going on in the kidney or in any other part of the body, and here again appears the tremendous scientific importance of ophthalmoscopic examinations. And as the retina can function satisfactorily in spite of having a few tiny atrophic sclerotic spots of perished retinal tissue, likewise the kidney in a similar condition will be for us normal or “healthy,” because no bio-chemical test up to the present can reveal such miniature anatomical lesions in the kidney as may be observed by means of the ophthalmoscope in the eye. But for the patient’s body, vitally

concerned in this matter, such a kidney will not be "healthy," and the former will furnish evidence of the hidden disease which can be detected by means of the ophthalmoscope and sphygmomanometer.

It may go differently with the patient. The vascular disease may progress quickly and cause severe damage. It may incapacitate so many nephrons that the kidney efficiency will be badly impaired, and in a few years' time the patient will die. This type is "malignant nephro-sclerosis." Here again we can see the same complete analogy with what is observed in the retina, when the eye becomes blind as the result of venous or arterial thrombosis of the central vessels or of their branches, or even as the result of widespread obstruction of the arterioles and capillaries *without* serious involvement of the larger vessels, as in the above described case. If such a malignant vascular disease affects the brain, it will mean cerebral thrombosis or haemorrhage, and if it involves the kidney it will mean a badly functioning organ. The tubular cells themselves intact but deprived of oxygen, will be in the position of an animal with a constriction in its trachea; if more air be allowed through, it will survive and even live a long time, although crippled; if the vascular supply of the nephrons be even partly restored, they will regain some of their efficiency. That this is quite probable may be verified in the case of the retina, which usually becomes transparent and "normal" again following a thrombosis of the central artery. Of course, it cannot work any more and the patient remains blind, because a few hours of complete anaemia mean a severe degeneration of the retinal cells; but *ophthalmoscopically* oedema of the retina is absent, and the observer will not see any other abnormalities, if the blood pressure be normal the whole time, as in my case of atheroma. ("Sclerotic retinal vessels"). The disc, of course, will be atrophic and the vessels narrowed. I mean that in the great majority of cases the blood circulation ceases only for a short time and then commences again, although in a lesser degree, and in tissues with end arteries, like the brain, kidney and retina, there is no exception to this rule. If the tissue be a highly specialized one, like the retina and cerebrum, it dies and does not revive. Whether the nephron behaves similarly or revives, it is impossible to say in our present state of knowledge. The body will be in a highly diseased state and it will suffer first of all in its oxygenation. This means an insufficient oxygenation of the retina: oedema and, therefore, retinitis. It will be combined with arterial hypertension, because the latter was a pre-existing and causative factor of the disease. In this stage the bio-chemical tests may still be normal (as in the case described earlier on). But *so far as the organism is concerned retinitis albuminurica always means a badly excreting kidney, its*

inefficiency having been brought about by a combination of vascular disease and tubular incompetence. The whole of the body is affected with a severe form of vascular disease, but matters are aggravated by the additional burden of the renal inefficiency. Therefore, it will not be logical to call such a case "interstitial nephritis"; it is still a case of "nephro-sclerosis" of the malignant variety, the kidney not being the cause but the victim of the disease. As in the case of a suffocated animal, if more oxygen be available, the kidney may partially recover and the body become freed from the additional burden of the badly excreting nephrons; the amelioration of the vascular disease will at the same time be beneficial for the whole of the body to the same extent as for the kidney. The result will be that the retinitis albuminurica will commence to regress, but in the retina, where we can actually see them, *vascular changes* will remain as a permanent and silent witness of a pathological past. Those are the cases, in my opinion, where in the post-mortem room the typical "interstitial kidney" will be found, the cases described in text-books as surviving for 10—20 years. There is every reason to believe that even anatomically, *caeteris paribus*, the kidney process is analogous to that of the retina, or of any other part of the body. If to this schematic description of both forms of "nephro-sclerosis" the reader will add the powerful influence of vasomotors, causing a general or local disability of the vascular wall leading to temporary complete interruptions of the vascularization and the influence of bacterial invasion and of auto-intoxication, he will easily realize how protean in character can be this vascular disease of the kidney, and all this with the presumption that the tubular cells of the nephron, ectodermic in origin, are still "healthy" and their own metabolism is not perverted. Compare this with nephrons where the tubular cells are diseased and the mesodermic elements of the nephrons are healthy, cases where the blood pressure is normal and retinal changes are absent. Suppose now that the vascular system of the nephron and the latter itself are attacked by the same toxin that has a predilection for both of them. We shall then have the dread and mysterious disease against which we are still helpless and which I prefer to call "true interstitial nephritis." By analogy with the *ophthalmoscopic* appearance of the retina the vascular tree of the nephron will sometimes be badly diseased, sometimes only slightly affected. This applies equally to isolated parts of the same nephron and to individual nephrons as well. The vessels may be affected in all their parts: atheromatous degeneration and hypertrophy of the intima, even leading to an obstruction of the lumen of the vessels; hyaline degeneration of the media; oedema and fibrosis of the adventitia. The interstitial tissue will play a double rôle: its swollen and degenerated peri-

vascular sheaths will compress and cripple the already pathological vessels and the inflammatory and oedematous tissue as a whole tightly surrounds and compresses the vascular tree. The more so since the renal capsule is not very elastic and therefore does not allow of much expansion to the kidney. The tubular cells, themselves diseased, will suffer the most from an insufficient supply of blood in addition to being charged with badly oxidized products of the metabolism of the whole body. Urea, being after all one of the products of insufficient oxidation, will increase in the blood with the diminution of the oxidation process in the tissues. Therefore, the tubular cells will break down in their function early and badly, exactly like the retinal cells when losing their function, as witnessed by the well-known subjective and objective symptoms of albuminuric retinitis. The hypothetical lymphatic space between the tubular cells and the capillary network will become a veritable cesspool.

It is impossible, in my opinion, to differentiate by any of our present methods of examination, perhaps even including a post-mortem, between true interstitial nephritis and the terminal stage of malignant nephro-sclerosis. However, a considerable difference exists. Malignant nephro-sclerosis has a longer history of five to six years' duration. It commences with hypertension, cardiac hypertrophy and sclerosis of the retinal vessels. Anoxaemia and its clinical sequel retinitis albuminurica appear later, one to two years before the end. Although a very small chance, there is still a possibility for the vascular disease to become milder and for the patient to survive, although in poor health. True interstitial nephritis begins more or less suddenly, as in my patient or the above mentioned case of J. Burnford. It invades the body severely and quickly; retinitis albuminurica appearing early, and the span of the patient's life being two to three years. All the victims perish and no treatment is of any avail, as the cause of the trouble is not in the vascular disease alone, but in the affected tubular cells as well. The difference may be found only in a complete and early examination of the patient. Here again the ophthalmoscope is predominant. If, as in my patient, the blood pressures are normal, the urine and the fundi also normal, and there are no signs of vascular sclerosis; for what reason does one affirm that he is suffering from a pre-existing renal lesion simply because three years afterwards he dies from true interstitial nephritis? In my opinion, it would be purely guesswork.

Again, in pregnancy we find a complete verification of these theoretical deductions. Many a woman with vascular disease becomes pregnant and passes through it safely, exactly as many "vascular" patients live a strenuous life and undergo surgical operations without necessarily succumbing to them. On the other

hand it may happen that a healthy woman becomes pregnant, suddenly develops arterial hypertension, albuminuria and retinal changes, and partially recovers after interruption of the pregnancy. I say "partially" because in several cases that I have seen I could always recognize some abnormalities in the retinal vessels. If I had not seen and examined such a patient early, when even the translucency of the retinal vessels was preserved, it could be argued later that she was suffering from a pre-existing kidney lesion. The solution of the kidney problem lies in the wise counsel of Sir J. MacKenzie, to examine early and thoroughly.

It is my pleasant duty to express my sincere thanks to Mr. A. H. Levy for his usual benevolent criticism, advice and help in revising and editing this paper and to the Staff of the London Jewish Hospital for their kind co-operation and to Dr. M. Cohen for his valuable help with the manuscript.

Summary

(1) Oedema of the retinal anterior layers is seen ophthalmoscopically as a fine veil covering the larger vessels. If fully developed, it leads to "wool-patches" in the retina.

(2) Retinal oedema is usually combined with renal insufficiency of varying degree and decreases or increases approximately *pari passu* with the latter, but an invariably co-existing condition is the presence of arterial hypertension. With a normal blood pressure retinal oedema means only a deficient oxygenation of the retinal cells.

(3) Albuminuric retinitis is ophthalmoscopically different from the sclerotic form. The appearance of this retinal oedema will therefore mean for the patient all the difference between benign and malignant nephro-sclerosis; and its degree and protean character will be analogous to what has been previously said about nephro-sclerosis in general. From the point of view of the organism retinitis albuminurica always means an inefficient kidney, although bio-chemical tests may be satisfactory.

(4) It is not yet proven that retinitis albuminurica is caused by disease of the kidney; both organs may suffer from the same toxin. But the retina is more readily affected than any other part of the central nervous system, and this may be explained by the peculiarities of its histological structure. If the histology of the kidney were to prove similar in this respect to that of the retina, it would explain the affinity of both organs when diseased.

(5) The kidney affection that is characterized by fully developed retinitis albuminurica is clinically different from any other form

of nephritis, and to it alone the term of "true interstitial nephritis" ought to be applied. It is a fatal disease of comparatively short duration, probably connected with a parenteral infection of the body, and its prognosis is bad in spite of possibly good results from bio-chemical tests. *Quod ab initio viciosum est tractu temporis convalescere non potest.*

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INTRA-CAPSULAR CATARACT EXTRACTION AT MOGA, PUNJAB

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GALWAY

IN discussing ways and means of cataract extraction, there is a tendency, perhaps more marked in this country than abroad, to be slightly shocked at any apparently radical operative procedure. This is surely contrary to the spirit of modern surgical progress. Reasoning by analogy, we must congratulate throat surgery on the passing of the defunct tonsillotomy operation. The days of that futile operation are no more, when patients paid an annual visit to have another piece snipped off the tonsil. One makes a mental comparison of the convincing nature of an operation like excision of the gall-bladder, as against a mere drainage of its contents. Remembering that the cataractous lens is a degenerated if not