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ON

THE ESSENTIAL SIGNS OF ARTERIO-SCLEROTIC DISEASE.*

(With Plate in Colours.)

BY

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NOMENCLATURE.

It will make the ultimate exposition of the subject clearer if I begin with a few explanatory remarks on the nomenclature of arterial disease. "Arterio-sclerosis" is a term which includes both forms of arterial disease of uncertain etiology as well as those so lacking in identity that there is no agreement on their names. The term "arterio-sclerosis" may, indeed, be compared to a crèche for children who have not known their parents, or who have so little individuality that they cannot be distinguished apart. Thus when the etiology of some form of arterio-sclerosis becomes known, the crèche loses one of its children, an event which took place with the recognition of tuberculous endarteritis and syphilitic endarteritis and mesaortitis. Again, although the etiology may be unknown, there are forms of arterial disease, such as thrombo-angiitis obliterans and periarteritis nodosa, which are so distinct pathologically and clinically that they cannot pass the test of nonentity that admits them to the crèche.

CLASSIFICATION OF ARTERIO-SCLEROSIS.

If the children of the crèche are examined with care they lend themselves to classification on alternative lines. On the one hand, in terms of etiology, there are some with a presumed parentage, though they are lacking in distinction, such as the arterio-scleroses caused by infections, of which typhoid fever is an example; on the other hand, in terms of morphology, there are forms of arterio-sclerosis that appear sufficiently distinct to deserve group names, and examples of these are nodular, diffuse hyperplastic, senile, and Mönckeberg's sclerosis. The distinction between these several groups is a morphological one, and it is based on variation in the form of the lesion; the distinction must not be pressed to too fine a point, because, just as groups of individuals, as families, lose some of their identity by marriage, so these groups of arterio-scleroses lose some of their distinction by coincidence and overlap. So long as the etiology of arterio-sclerosis remains veiled in relative obscurity, the latter of these two lines of classification is the more convenient, and, being based on variations in structure, it is in accord with the proper use of the term "arterio-sclerosis."

THE SIGNIFICANCE OF ARTERIO-SCLEROSIS.

Arterio-sclerosis is first and foremost an anatomical as opposed to a clinical conception. To the morbid anatomist arterio-sclerosis has a definite significance. It is a lesion that he can recognize and describe, and though he more

often collects his material for examination from the *post-mortem* room than the operating theatre, the lesion has for him a living interest, for in it he sees the fierce struggle between the forces of destruction and the forces of resistance and repair. Contrast this vital interest of the morbid anatomist with the commonplace opinion of the clinician, who is inclined to see in arterio-sclerosis mere evidence of senility and arterial decay. It cannot be only the failure of therapeutics that gives the clinician this hopeless outlook; it is due rather to an imperfect understanding of vascular function and vascular disease. Thus it is not long since arteries were regarded merely as pipes, whose chief function was to withstand the pressure of the blood they contain, and conduct it from the heart to the periphery; as a result, most changes in arterial structure were attributed either to the continued effect of this pressure or to the use of bad material in making the pipes. These simple mechanistic views are being superseded as we recognize variations in tone of the whole vascular system, including the capillaries, and a power of rhythmical contraction of the arteries—activities which bring the physiology of arteries into line with that of the heart.

An incomplete appreciation of the pathology of arterial disease also lent support to the inanimate clinical view of arterio-sclerosis. Thus, much of the thickening and hardening of arteries is due to calcification and fibrosis—the end-result of processes of necrosis and repair; the fatty degeneration so constantly present in arterio-sclerosis is evidence of mere arterial decay; even the cellular proliferation, which is an integral part of the lesion, is open to explanation in terms of replacement fibrosis and repair. If this were the whole explanation of the changes in vascular structure that make arterio-sclerosis, the lesion would be simply evidence of a process of degeneration or repair. It would provide a natural explanation of the fact that the majority of persons with arterio-sclerosis do not suffer from it, and the palpation of a thickened radial artery or the inspection of retinal arterio-sclerosis in the fundus oculi would approach in its clinical significance to the signs of a healed tuberculous lesion in the apex of a lung.

ACTIVITY OR QUIESCENCE OF THE LESION.

To a certain extent we are, indeed, right in regarding the presence of arterio-sclerosis as evidence of recovery from disease, but it is only right within strict limitations, because arterio-sclerosis tends to be a progressive or an active lesion and one that often leads to incapacity and not infrequently to death.

Whenever the diagnosis of arterio-sclerosis is made, we want to know if the person is suffering from it; the first step in diagnosis is the determination of its presence, the second immediate step is the determination of its activity or quiescence.

The importance of this distinction is more clearly realized by comparison with the same problem as it is presented by valvular disease of the heart. The first step in diagnosis is the recognition of the presence of an organic change in the valves; the second step is the distinction between an active and an old lesion. When the lesion is active we may speak of the person suffering from *heart disease*, and similarly, if we can define the position in which we are able to make the diagnosis of active arterio-sclerosis, we shall be able to speak of *arterio sclerotic disease*.

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The term "arterio-sclerotic disease" is synonymous with "an active arterio-sclerotic lesion" or "active arterio-sclerosis." But whereas the term "arterio-sclerosis" has a morphological significance, arterio-sclerotic disease has a clinical significance, because the diagnosis of active arterial disease raises the presumption that the person affected is suffering from arterio-sclerosis or will suffer from it unless its activity is arrested. Further, in using the term "arterio-sclerotic disease," there is no implication of a disease *sui generis*, for arterio-sclerosis, like endocarditis, is a part of the picture of many clinical entities and the product of many different causes.

THE BASIS FOR THE DIAGNOSIS OF ARTERIO-SCLEROTIC DISEASE.

My ideas of arterio-sclerotic disease rest on observations of the morbid anatomy of arterio-sclerosis. My studies of arterio-sclerosis have been chiefly concerned with the form which is known as arterio-capillary fibrosis, or diffuse hyperplastic sclerosis. The striking fact of this form of arterio-sclerosis is the cellular proliferation which, both in priority of appearance and degree of development, dominates the histological picture of diffuse hyperplastic sclerosis.

At any rate, in this form of arterio-sclerosis there is no doubt as to the activity of the lesion while the form of the lesion is that of an active inflammatory process, followed by degenerative changes at a later stage.

Histology of Diffuse Hyperplastic Sclerosis.

The main incidence of the lesion is on the finer ramifications of the vascular tree. Some organs tend to be affected more than others, the kidneys and spleen being especially affected. In other organs, such as the heart and skeletal muscles, the lesion is rarely found. At any one time, in an organ such as the kidney or spleen, the lesion is to be found in all stages of development. The earliest stage is one of endothelial proliferation visible in the terminal arterioles, and this, indeed, may be preceded by capillary changes. At a later stage the swollen and proliferated endothelial cells undergo fatty degeneration, and occlusion of the lumen results. The arteriole change varies in the length of the vessel; there is a varying swelling of the vessel wall, giving it a moniliform appearance, and a variable degree of fatty degeneration. At this stage there are obvious capillary changes in the glomerular tufts, such as are seen in Fig. II. While these changes are proceeding, the proliferation of the intima of the parent vessels, the intralobular arteries in the kidney, is taking place. In these vessels also there is, I think, endothelial cell proliferation in addition to proliferation of the other cells of the intima; at the same time there is formation of new layers of elastic tissue, which is perhaps a secretion by the newly formed endothelial cells. These vessels are in contrast to the terminal arterioles in that there is little or no fatty degeneration in the intima, even when the lesion is advanced. Fig. III shows the complete lesion as it affects a terminal arteriole, and the parent vessel from which it springs.

In addition to the vascular changes already described, there may be hypertrophy of the media, and there is occasionally a suggestion of thickening of the adventitia. The larger vessels are relatively free from change; on the other hand, they may be affected by nodular or other form of arterio-sclerosis.

The important fact is that cell multiplication precedes

degeneration, and from our knowledge of the function of endothelium and its behaviour under pathological conditions it is not, I think, open to question that the endothelial proliferation is evidence of inflammation, and further, as it precedes degenerative change, it must be a primary response to irritation and not the secondary effect of degeneration or necrosis of cells in its immediate environment.

There is further evidence of the activity of the lesion in the frequent presence of areas of intense small-cell infiltration in the kidneys of patients who have died with arterio-sclerotic disease. Fig. I shows a patch of such infiltration, and similar foci of inflammatory reaction were scattered through the renal cortex, which, in its main extent, was relatively normal. The signs of activity of the lesion are, indeed, so obtrusive that one is led to expect coincident symptoms and physical signs in a person whose vessels are affected by this lesion.

SYMPTOMATOLOGY OF ARTERIO-SCLEROTIC DISEASE.

In the attempt to identify the signs and symptoms due to activity of the arterio-sclerotic lesion, the best cases to examine will be those in which arterio-sclerotic disease is so severe in its incidence and rapid in its course that it overcomes resistance and forestalls repair. The two outstanding examples of this disease are cerebral haemorrhage and angina pectoris. These two conditions provide the basic symptomatology of arterio-sclerotic disease; the cardinal sign is haemorrhage, the cardinal symptom is pain.

Haemorrhage.

The occurrence of haemorrhage in arterio-sclerosis is common experience, and cerebral haemorrhage is its outstanding illustration. The significance of the haemorrhage has not, however, been adequately recognized. It is either quickly dismissed as a complication of the vascular disease, without pause to consider the process responsible for its occurrence; or the sequence of thought is disturbed by consideration of the etiology of the lesion. These matters are of such speculative interest that an important link in the chain of causation receives no adequate consideration, and the actual change in structure of the vessel wall which allows the escape of blood is hardly considered. Thus high blood pressure, even when considerable and lasting over a long period of time, or even when suddenly increased above its normal level, is not responsible for haemorrhage unless the vessel wall is so damaged by disease as to permit the escape of blood. The number of persons living with high blood pressure bears witness to this. *Per contra*, cerebral haemorrhage is not uncommon in arterio-sclerosis with a normal blood pressure, and any series of cases of cerebral haemorrhage includes a fair proportion without cardiac hypertrophy *post mortem*, or raised blood pressure during life.

The determining factor in cerebral haemorrhage is disease of the vessel wall, and the vascular lesion is as directly and primarily responsible for the haemorrhage in arterio-sclerosis as it is in the cases of gross haemorrhage sometimes seen in encephalitis lethargica.

The importance of vascular disease in distinction from blood pressure in the causation of cerebral haemorrhage is brought out by what seems to me a fair comparison of cerebral haemorrhage with perforation of the stomach in gastric ulcer. We are told that the immediate cause of

DESCRIPTION OF COLOURED PLATE.

FIG. I.—This drawing is reproduced to show intense small-cell infiltration of the renal parenchyma in a case of arterio-sclerotic disease that died of cerebral haemorrhage, without evidence of kidney disease. Similar sharply defined patches were scattered throughout the kidney, intervening areas of the cortex being relatively normal. It is from the same organ as Fig. II ($\times 80$).

FIG. II.—This figure shows hyaline swelling and fatty degeneration of certain capillary loops in a glomerulus, the rest of the glomerulus being practically normal. At the left-hand corner there is some proliferation of the epithelial cells of the capsule. It is an early stage of the condition seen in Fig. III ($\times 200$).

FIG. III.—To the right of the centre is an intralobular artery in cross-section. Its lumen is greatly reduced by thickening of the intima. The faint outlines of proliferated endothelial cells with clearly defined oval nuclei are seen towards the inner surface. Nearer the media the cell outlines are indistinct. There is hyaline swelling and in places slight fatty degeneration. The internal elastic lamina is duplicated in part of the circumference. The glomerular artery cut longitudinally in its whole length is seen arising from its parent vessel. The extreme fatty degeneration of its intima is in marked contrast with the relative integrity of the intimal cells of the parent vessel. The faint outline of degenerate endothelial cells is still visible, and at either end of the vessel the original lumen can be seen in longitudinal section. The typical moniliform appearance of the arteriole is well shown; the capillary nexus in which the vessel ends has undergone degeneration. This section shows all the histological features of diffuse hyperplastic sclerosis at a glance. [Section prepared from kidney embedded in gelatin; stained with haemalum and Sudan III ($\times 400$). Camera lucida drawing by J. R. Ford.]

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FIG. 1.

FIG. II.

FIG. III.

the perforation of the stomach is separation of a slough or the spread of ulceration, but that it may occur from over-distension of the stomach or as the result of sudden strain. Mechanical influences in the causation of perforation of the stomach are of minor importance in comparison with the pathological changes that result from activity of the ulcer. Similarly in arterial disease, I believe that as a cause of haemorrhage the height of the blood pressure or its variation from time to time is of minor importance in determining rupture of the vessel, compared with increase or activity of the process of arterial disease.

In short, I suggest that haemorrhage in arterio-sclerosis is evidence of activity of the vascular lesion; that, in other words, it is a sign of arterio-sclerotic disease.

If the first haemorrhage is fatal, there is little clinical advantage in appreciating this significance of haemorrhage in a given case; but it frequently happens that the final and fatal haemorrhage is preceded at an interval of months or years by smaller haemorrhages, from which recovery occurs. There is, indeed, a group of cases in which the palsy or other cerebral disturbance is so frequently repeated, so slight in its manifestations or short in its duration, especially so completely recovered from, that it is regarded as due to vascular spasm. This opinion is difficult to disprove; but the fact that in such cases death often occurs finally from a gross haemorrhage is evidence in favour of the view that the slight antecedent pareses are due to minute haemorrhages. I certainly incline to the opinion that there is a definite lesion in the majority of such cases, because my own experience is that brains which appear normal on careful naked-eye examination frequently show numerous minute areas of capillary haemorrhage on microscopical examination. It is not uncommon to find the pons, for instance, peppered with microscopical haemorrhages and spots of pigmentation, some of which appear to be due to old extravasations of blood, though there was no visible sign of the lesions on naked-eye examination of serial sections made in the *post-mortem* room. This, and many other controversial matters which I refer to this evening, are fully discussed by Sir Clifford Allbutt in his great work *Diseases of the Arteries, including Angina Pectoris*.¹

Our great need as clinicians, and if possible from the point of view of advising curative or prophylactic treatment, and at any rate from the point of view of prognosis, is to be in a position to anticipate cerebral haemorrhage. If haemorrhage is accepted as evidence of activity of arterio-sclerosis, if it is accepted as a cardinal sign of arterio-sclerotic disease, haemorrhages elsewhere in the body acquire a new significance. This is already realized in the case of recurrent epistaxis, which is sometimes antecedent to cerebral haemorrhage. A similar significance attaches to haemorrhage from the lungs, kidneys, stomach, intestines, uterus, and other situations in the body where haemorrhage may occur. Not only should gross haemorrhages be considered from this point of view, but whenever arterio-sclerotic disease is suspected a careful search for minute haemorrhages should be conducted. The fundus oculi is of first importance in this connexion, and the work of Foster Moore² provides one of the links between retinal and cerebral haemorrhage.

Another important site of haemorrhage in arterio-sclerotic disease—in fact, perhaps the most important situation in which haemorrhage occurs—is in the kidneys; a careful examination of urinary deposits will often reveal the presence of red blood corpuscles in the urine, when there is a trace of albumin present, although the guaiacum test for blood is negative. There are, of course, very numerous causes for haematuria, which require exclusion before the presence of red blood corpuscles in the urine is accepted as evidence of arterio-sclerotic disease. It is, however, unnecessary to make a differential diagnosis between chronic interstitial nephritis and arterio-sclerotic disease, because the former includes the latter.

It is thus seen that the discovery of haemorrhage in the kidneys, eyes, brain, and elsewhere in the body may in the first place lead the way to the diagnosis of arterio-sclerotic disease in a patient whose symptomatology does not of itself suggest this disease; in this way it may be the crucial

sign in the differential diagnosis between neurasthenia and arterio-sclerotic disease. Again, in a patient who has the physical signs of arterio-sclerosis, whether it be in the appearance of the retinal vessels, the thickening of his arteries, the condition of the urine, or the level of the blood pressure, the finding of haemorrhage will make the differential diagnosis between healed, or quiescent, and active disease.

But there is another side to the picture. If haemorrhage is realized as the critical sign of arterio-sclerotic disease, local lesions will not be so readily accepted as the cause of haemorrhage until the presence of arterio-sclerotic disease has been reasonably excluded. In the somewhat obscure cases of haemorrhage that are not uncommon in practice the possibility of arterio-sclerotic disease requires the closest consideration; it may be responsible for such gross haemorrhage as to necessitate the removal of the organ from which the bleeding occurs. Thus it is the explanation of some cases of renal epistaxis and a not uncommon explanation of some cases of excessive uterine haemorrhage; in one year I saw four women in whom hysterectomy had been performed on this account, and though the removal of the uterus was probably necessary, the fact of its being due to arterio-sclerotic disease was not recognized, and it was on account of the exacerbation of the symptoms of this disease that further medical advice was sought after hysterectomy had been performed.

In concluding this part of the subject reference must be made to the controversial distinction between haemorrhage and thrombosis. It is quite possible that haemorrhage is preceded by thrombosis in many instances. I think, however, that it is immaterial to my thesis, because the chief factor in determining thrombosis is disease of the vessel wall, and the occurrence of thrombosis is probably as much evidence of activity of this disease as is haemorrhage.

Pain.

The question of pain as a symptom of arterio-sclerotic disease is more difficult than that of haemorrhage, because it is often difficult to distinguish between pain due to arterial disease and pain due to the effects of arterial disease. This distinction may be chiefly of academic importance; it needs, nevertheless, to be borne in mind.

There can be no doubt that painful sensation may be provoked by vascular disease. The local pain of arterial embolism is on occasion sudden and excruciating; there is often marked tenderness in phlebitis—as, for instance, tenderness on pressure over the affected vein in femoral thrombosis. Although arterio-sclerosis is in general a painless disease, the superficial arteries are occasionally tender on light palpation. Thus I have on occasion found the temporal artery tender, and I was once consulted by a patient simply on account of pain in this vessel. Recently I saw a man with arterio-sclerotic disease whose transverse cervical artery was tender on palpation; he said it felt like a pin-prick when it was touched. On the other hand, the pain of intermittent claudication is an example of pain provoked by deficient blood supply, a secondary effect of vascular disease. The recent researches of Professor MacWilliam³ in this country have provided a rational explanation of the production of this pain. One of the most striking symptoms in medicine, both owing to its severity, its peculiar nature, and its occasional fatal association, is the pain of angina pectoris. Whether the pain has its origin in the aorta or in the heart, there is a consensus of opinion that it is primarily determined by vascular disease—that is, by disease affecting the base of the aorta or the coronary arteries. Angina pectoris points to disease of the root of the aorta, and though the disease is more often associated with syphilis than arterio-sclerosis, true angina pectoris may result from arterio-sclerosis of the aorta. There is no other pain than that of angina which points with such deadly directness to arterio-sclerotic disease, but there are other pains which are suggestive of it. There are pains, for instance, in the neck and occipital region which are closely associated with arterio-sclerotic disease; there are also pains in the shoulders and hips, and in the arms and legs, which are directly due to arterial disease,

or which have their origin in fibrous tissues, joints, or nerves irritated by the poison or poisons responsible for the coincident arterio-sclerotic disease.

After haemorrhage and thrombosis on the one hand, and pain on the other hand, which form the basis of the symptomatology of arterio-sclerotic disease, there follow a large group of signs and symptoms dependent on disturbance of vascular function. It is, of course, appreciated that functional disturbance may occur without structural defect; nevertheless, functional disturbance is so frequently associated with structural defect that its presence suggests a lesion. Common symptoms in arterio-sclerotic disease are flushings and blotchiness of the skin; in more marked cases there are Raynaud-like symptoms, and other circulatory disturbances. The physical sign which has received most attention is an alteration in the blood pressure. It is beyond the scope of this communication to discuss the significance of raised blood pressure in relation to arterio-sclerotic disease. In general I think that a raised blood pressure is significant of arterio-sclerotic disease; but it may be that it has no such significance in those cases in which the blood pressure though raised maintains a steady level, little affected by exercise or by medical treatment.

THE OCCURRENCE OF ARTERIO-SCLEROTIC DISEASE.

In the previous paragraphs I have attempted to present arterio-sclerotic disease as the active phase of the lesion arterio-sclerosis, and recognizable by certain signs and symptoms due to activity of the lesion. It will give the condition greater distinctness if one considers the clinical entities of which it may form part.

On the basis of its histology, diffuse hyperplastic sclerosis is the most active or acute form of arterio-sclerosis, and it is therefore in this form that one would expect the signs and symptoms of arterio-sclerotic disease to be most evident. In previous communications⁴ I have given reasons for the opinion that persistent hypertension is in itself evidence of diffuse hyperplastic sclerosis; and it is, therefore, in those clinical conditions in which persistent hypertension is present that one would expect to find arterio-sclerotic disease. This in fact is the case, and the best examples of arterio-sclerotic disease are to be found in hyperpiesia, chronic interstitial nephritis, and chronic nephritis.

There is reason to believe that the more chronic forms of arterio-sclerosis also have their active phases which may be capable of clinical recognition. In such cases, though there may be no alteration in the blood pressure, evidence of activity of the arterial lesion—that is, evidence of arterio-sclerotic disease—may be present in retinal haemorrhage and exudates, and in the presence of haematuria, either gross or only discoverable on careful microscopical examination of the urinary deposit. In one such case, that of an old man with massive retinal exudates, there have been changes in the form of the electrocardiograms taken from time to time—at one time indicating a right bundle-branch lesion, and at another time showing its recovery; it is possible that these electrocardiographic changes are the result of vascular disturbance, or it may be that they result from the action of the poison responsible for the vascular disease. It is in this direction at any rate—in the most careful examination of the cardio-vasculo-renal system, both in regard to its structure and function—that the signs and symptoms of arterio-sclerotic disease are to be sought.

TYPES OF ARTERIO-SCLEROTIC DISEASE.

In order to give the conception of arterio-sclerotic disease clearer definition, it may be usefully compared with diseases of the joints; thus, in a recent textbook of medicine Sir William Willcox divides disease of joints into two classes: those of known causation and those of obscure causation. In the first class are grouped (1) traumatic arthritis, to which there is a parallel in arterial disease in arterio-venous aneurysm; (2) specific infective arthritis, of which a number of examples are known in arthritis, and of which tuberculous and syphilitic endarteritis are the best examples in arterial disease. Under the same heading is included gout, and its parallel in vascular nomenclature, by reason of its being a

definite entity but of unknown causation, is thrombo-angitis obliterans or periarteritis nodosa. Again, there is the neuropathic type of arthritis, and with this may be compared the endarteritis present in the late stages, at any rate, of Raynaud's disease.

The second main class—arthritis of obscure causation—has its parallel in arterio-sclerosis, and I would suggest that diffuse hyperplastic sclerosis in the most general way is comparable to the more acute forms of polyarthritis, known by such names as rheumatoid arthritis, subacute or chronic infective arthritis; while the senile type of arterio-sclerosis is again in a most general way comparable to the osteo-arthritis type of joint lesion.

To my mind the subject of arterio-sclerosis is simplified by this comparison, and I think that some of the obscurity which has surrounded the subject in the past has been due to a failure to recognize the part that the process of inflammation plays in the production of the arterio-sclerotic lesion. It has been due in part to the overemphasis of trauma as a cause of arterial disease, to the exaggerated importance attached to high blood pressure as an etiological factor. Senility has also been overemphasized, and it probably plays a more important part in determining the form of the lesion than in determining its onset.

At the present time the etiology of arterio-sclerosis is veiled in obscurity, but the opportunities of discovering its causation will be multiplied when we are in a position to recognize its onset and follow its course clinically. To do this we must study the signs and symptoms of activity of arterio-sclerosis; we must, in fact, learn the symptomatology of arterio-sclerotic disease.

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A British Medical Association Lecture.

NOTES ON MEDICAL PRACTICE.

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On Prognosis.

PROGNOSIS is one of the legs of the tripod when we are dealing with disease, the other legs being diagnosis and treatment. The patient asks, "Am I going to get well?"

I have always been interested in the dogmatism of youth, which knows and seldom hesitates to give its opinion; of course the patient likes this. Yet surely as a rule it is experience of cases, and constantly seeing them for years, that enable the doctor to give sound and wise advice in this matter of prognosis.

Increasing age has its advantages and disadvantages to the doctor. As regards active surgery there is a limit beyond which it would be foolish for a man to go. Yet, providing that a man keeps up his reading and his powers of observation, also allowing at the same time that he is seeing cases, then age is a great advantage in prognosing the particular disease under his care. A man who has watched carefully his work for forty or even fifty years ought to be, and must be, in a better position to give a sound prognosis than a man who has been in practice for a comparatively short period. There is the exception—that one man may in a few years see and observe more than another man who has been longer at work. Still, the rule I have laid down is a wise and sound one.

On Diagnosis.

I am inclined to the view that diagnosis is not thought of paramount importance, or perhaps it is more just to say that not sufficient time in thought, word, and deed is given to diagnosis. There is a tendency of late, I have noticed,