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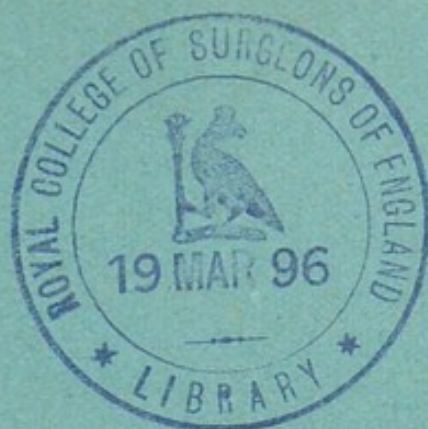
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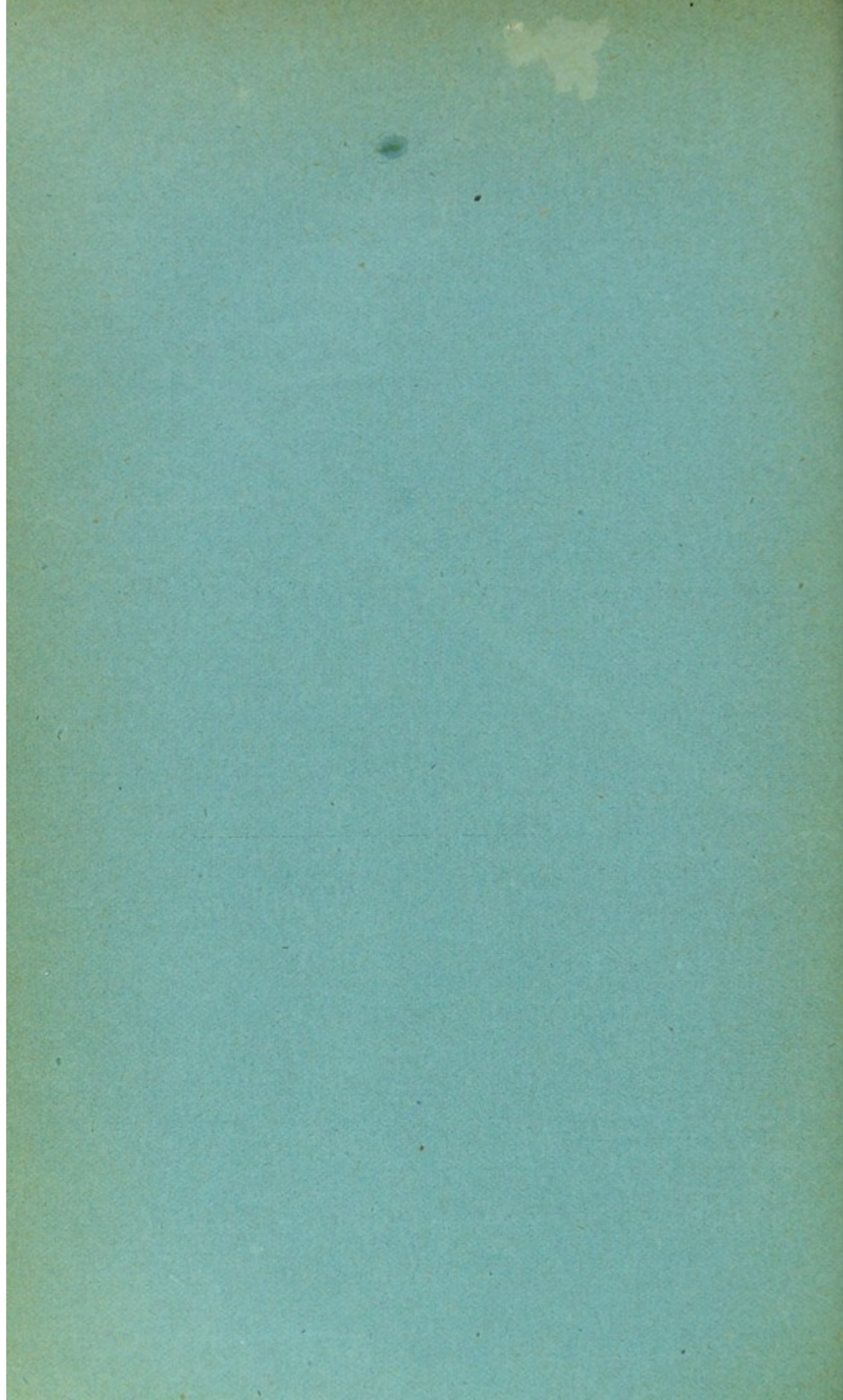


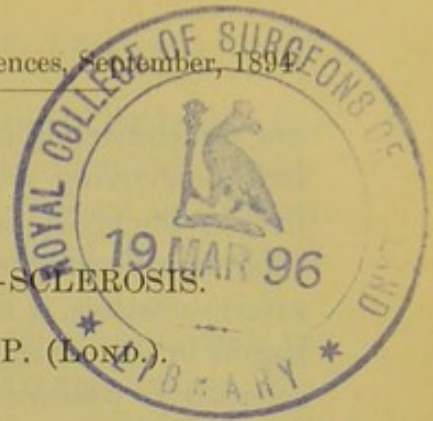
On the Pathology of Arterio-Sclerosis.

BY

F. PARKES WEBER, M.A., M.D., M.R.C.P. (LOND.).

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ON THE PATHOLOGY OF ARTERIO-SCLEROSIS.

BY F. PARKES WEBER, M.A., M.D., M.R.C.P. (LOND.).

ARTERIO-SCLEROSIS was first described by Sir William Gull and Dr. Sutton in 1872. In their paper before the Medical and Chirurgical Society of London,¹ they suggested the name of arterio-capillary fibrosis for the morbid change they described.

The term arterio-sclerosis is merely a Greek rendering of Gull and Sutton's term, but has perhaps the advantage of a more international character.

The essential change, according to Gull and Sutton, was a thickening of the walls of the arterioles and capillaries, more or less in all parts of the body, and more recent observations have not made much advance on this. The hyaline appearance of the fibroid change, as described by Gull and Sutton, was shown to be due to the acidulated glycerin in which their microscopical specimens² were prepared. It is also true that in the arterioles Gull and Sutton considered the change to be situated chiefly in the tunica externa, whereas most recent observers have considered the primary change to be rather an accumulation of cells in the interna.

This latter view appears to me not proven and the difference not a matter of the greatest importance. Upholders of the latter view have maintained that the essential change in arterio-sclerosis may be termed an endarteritis obliterans affecting the smaller arterioles. To this term I should prefer the original one of Carl Friedländer, namely, arteritis, or arteritis obliterans.³ It may certainly not always be easy to see whether a process (even when certainly inflammatory) is primary in the tunica externa or interna, for, as Friedländer points out, cell accumulation in the one is often accompanied by a corresponding process in the other. A double process like this may be the method by which vessels

¹ Transactions of Medico-Chirurgical Society, vol. lv., 1872, p. 273 et seq. Dr. Walshé had in 1849 endeavored to show that "Bright's disease" was of constitutional origin (see *Lancet*, 1849, vol. ii., p. 29: "Bright's Disease Not Essentially a Renal Disease, but Essentially and Primarily a Blood Disease.") He went, however, in a direction quite different to "arterio-sclerosis," and concluded that "Bright's disease" is a blood disease, *ab initio*, essentially tending to chronicity, though (like cancer and phthisis) sometimes running an acute course.

² Fagge's Principles and Practice of Medicine, third edition, vol. ii., p. 464.

³ Dr. Carl Friedländer, "Ueber Arteritis Obliterans," in the *Centralbl. für med. Wissenschaft*, January 22, 1876. In this paper of Friedländer the process is discussed with no special reference to Bright's disease.

are sometimes obliterated in a tuberculous lung-area, the perivascular irritation giving rise to a sort of endarteritis obliterans; so also at the base of the brain, perhaps a perivascular¹ syphilitic process may sometimes be the cause of an endarteritis obliterans. At any rate, as regards arterio-sclerosis, it may still be considered doubtful whether the process begins in the tunica externa or interna, and therefore, as far as arterio-sclerosis is concerned, the term arteritis obliterans seems better than endarteritis obliterans.

There is, however, another objection to either of the terms endarteritis or arteritis obliterans being spoken of as the primary lesion of arterio-sclerosis; this is that the termination "itis" implies that the process is necessarily one of inflammation. For although undoubtedly the two are very frequently associated, it remains doubtful whether inflammation is a *necessary* factor in arterio-sclerosis. As regards this point I have been obliged to look for a modern definition of inflammation. The "rubor, tumor, calor, et dolor" of Celsus can no longer be considered satisfactory except from a purely clinical point of view, in which case it is certainly convenient. The "functio læsa" may be a more constant accompaniment of inflammation, but does not help us in the present case. For the present purpose it is perhaps sufficient to regard inflammation² as the series of tissue changes caused by an injury or by the presence of some harmful irritant, and usually leading to an accumulation of cells in the part affected.

This includes the cell accumulation resulting from proliferation of the fixed cells as well as that resulting from the migration of leucocytes. Such a modification of modern definitions of inflammation includes tuberculous affections, and will include cancer and sarcoma if ultimately the presence of coccidia should be proved to be necessary to their development. The presence of necrobiosis, secondary to inflammation or due to the same original cause, will not confound the definition. If then we accept such a definition, does arterio-sclerosis fall under the head of inflammation?

The cell accumulation is certainly present, but the irritant cause is what I consider not proven. It is better to speak of the essential change which the microscope discloses in arterio-sclerosis as a thickening of the external and internal coats of the arterioles, and not to apply to it any particular name implying the presence of inflammation.

This naturally brings one to the *etiology* of arterio-sclerosis. Gull and Sutton left this a doubtful point, and a doubtful point it remains in common with the etiology of many other chronic diseases. All that can be supported by fact is that arterio-sclerosis has some relation to age,

¹ But either of these processes may also give rise to miliary aneurisms.

² Compare the definition used by Prof. Burdon-Sanderson in the "Lumleian Lectures on Inflammation," 1882.

syphilis, arthritism, and the uric acid diathesis (if the latter be not included under arthritism); probably only through the diathesis is there any relation with actual uricæmia; furthermore, it must be allowed that an hereditary tendency to arterio-sclerosis, allied to inherited arthritism, may exist.

How uncertain are the alleged causes of arterio-sclerosis may be gathered from the fact that of two well-known French writers, Huchard¹ includes alcohol, tobacco, saturnism, inheritance, acute and chronic diseases, such as typhoid, smallpox, syphilis, malaria, etc.; whereas Lancereau maintains that arterio-sclerosis always owes its origin to nervous causes. It will be here impossible to do more than allude briefly to the most important of the alleged causes, but in examining them one is struck by the uncertain nature of their claims. Sir George Johnson² considered that the kidney disease is primary, that in consequence of this the blood contains urinary excreta, and is otherwise altered, that the minute arteries throughout the body resist the passage of this abnormal blood, and in consequence of this the muscular walls of the arteries and left ventricle become hypertrophied, as seen at autopsies. Johnson's theory was combated by Gull and Sutton; they pointed out that the vascular changes could precede the kidney disease, or, as Bamberger has stated, appear in its earlier stages; moreover, they stated that extreme degeneration of the kidneys (either large white or contracted³) may be accompanied by œdema and uræmic symptoms, which indicate doubtless a noxious state of the blood, and yet the characteristic cardio-vascular changes may be absent. Neither does it appear proved that the high-pressure pulse of arterio-sclerosis is due usually to an abnormal contraction of the arterioles, though doubtless this may occasionally occur. If it were so, one would expect to find the muscular coat of the arterioles hypertrophied, as Sir George Johnson described them to be; but, according to most observers, when this thickening exists it is due to increase of fibrous tissue. Certainly in some cases of chronic Bright's disease the arterial tension can be lowered by nitrite of amyl,⁴ but this does not affect the point, because arterio-sclerosis, although a generalized disease, by no means affects all parts of the body equally. The mechanical arterio-capillary fibrosis may be the cause of increased arterial pressure, and yet the arterioles over large

¹ *Maladies du Cœur*, 2d ed., Paris, 1893, pp. 119 et seq.

² See *Brit. Med. Journal*, April 16, 1870.

³ *Med.-Chir. Trans.*, vol. lv. p. 294. So also it has been stated that chronic interstitial nephritis can be caused through obstruction to the ureters by uterine cancer, without any hypertrophy of the heart resulting. On the other hand, it is stated that high arterial pressure and hypertrophy of the heart may follow hydro-nephrosis, say from impacted calculus, in a few weeks. (See *Brit. Med. Journal*, November 4, 1893, p. 998.) Gull and Sutton referred to cases of contracted kidneys in young subjects, not over twenty years of age, and believed that such cases were of quite a different nature to ordinary cases of contracted kidney.

⁴ *Vide Fagge*, op. cit., 3d ed., vol. II. p. 467.

areas of the body may still be insufficiently affected to prevent them from dilating under nitrite of amyl, and thereby a fall in the general arterial pressure would ensue on the administration of nitrite of amyl.

Let us now consider some other alleged causes of arterio-sclerosis—namely, acute and chronic infectious diseases. Of these I will confine myself to syphilis, as it is the most often adduced.

Certainly many patients with marked arterio-sclerosis and chronic interstitial nephritis have had syphilis, but the question is whether the proportion in arterio-sclerosis exceeds that in other diseases; moreover, how many patients have had syphilis and even tertiary manifestations, and yet do not suffer especially from arterio-sclerosis! It seems curious that syphilis, which has such characteristic tertiary lesions of its own, should also have been represented as playing so great a rôle in the etiology of many chronic diseases, which certainly are not exclusively¹ dependent on it. I need only mention “arterio-sclerosis,” “tabes dorsalis,” and “general paralysis of the insane.” With reference to the frequent antecedence of syphilis in these diseases, one must remember, first, the great frequency of syphilis as a disease, and, secondly, the cases are to be considered where true syphilis may simulate one of these diseases. Thus, a spurious general paralysis has been described, which is due to actual syphilis, and is really benefited by antisypilitic treatment; Charcot points out that true syphilis may simulate the symptoms of tabes dorsalis; in like manner true syphilitic lesions may cause (by occlusion of vessels or rupture of miliary aneurisms of syphilitic origin) brain symptoms which are frequent in arterio-sclerosis.

In short, the claims of syphilis do not appear quite established as regards the etiology of arterio-sclerosis.

There certainly are some remarkable cases which appear to support the view that syphilis may give rise to arterio-sclerosis. Such a case is that narrated by A. Fraenkel at the Berlin Medical Society (*Berliner klin. Wochenschrift*, 1894, No. 12).

The patient was a woman, aged thirty-six, who died in an attack of angina pectoris. At the necropsy on this case the right coronary artery was found to be obliterated at its orifice by a process resembling arterio-sclerosis, and to confirm the view that this apparent arterio-sclerosis was really due to syphilis, a gumma was found in the septum ventriculorum. Might not, however, in this case and in similar cases, the stenosis of the artery be due to an actual tertiary syphilitic process, and, though closely resembling a process of precocious arterio-sclerosis, be really caused by the irritation of the specific poison or microbe of syphilis, if such a specific microbe or poison be admitted as the cause of tertiary syphilitic lesions? If, then, the claim of syphilis still appears doubtful, the

¹ Unless, in the case of tabes, we accept the view of those who make syphilis a *necessary* antecedent of tabes dorsalis.

claim of other infectious diseases to be regarded as causes of arterio-sclerosis appears more doubtful.

Other alleged causes are the continued presence of abnormal substances in the blood or tissues, due to indulgence in alcoholic drinks, tobacco-smoking, saturnism, or uricæmia. Statistics have not yet settled these questions.

If we suppose that these substances cause arterio-sclerosis by inducing spasm of the arterioles we admit that the high arterial pressure is the cause and not the effect of arterio-sclerosis; but, as I have just pointed out, the increased arterial pressure may be the effect and not the cause. If, on the other hand, we suppose that these substances act by setting up a chronic inflammation in the walls of the arterioles, we admit that the essential lesion in arterio-sclerosis is one of chronic inflammation, and I pointed out (when speaking of endarteritis obliterans) that this assumption is not justified. That chronic lead-poisoning may induce a chronic interstitial nephritis is admitted; but some observers deny that in these cases the cardio-vascular changes of arterio-sclerosis¹ are a necessary accompaniment. As regards uricæmia it seems more probable that any relation which arterio-sclerosis may have with it is rather a relation with the diathesis than with the actual excess of uric acid in the blood.

This brings us to the most important group of alleged causes of arterio-sclerosis, namely, inheritance, age, and the arthritic and uric-acid diatheses. Rather than adduce old age as a cause, its relation is, perhaps, better put by stating merely that arterio-sclerosis is a disease of the second half of life, and very rarely attacks those younger. The arthritic and uric-acid diatheses are probably also rather associated conditions than standing to arterio-sclerosis in any direct relation of cause and effect.

One fact seems certain, namely, that there is often an hereditary tendency to arterio-sclerosis, and this is especially the case where there is an "arthritic" family history.

One might suppose, indeed, as has been suggested to me, that in families which possess sufficient vitality and energy to adapt themselves to their surroundings, and which in consequence survive for many generations, there may, side by side with the resistance to tuberculosis and other diseases² which exterminate families who are physically too weak to adapt themselves to their surroundings; side by side with this resistance there may grow up a tendency to gout, arterio-sclerosis, and "degenerative" diseases, which may themselves, if unchecked, lead to

¹ If, however, any influence of lead in the production of arterio-sclerosis be granted, it can still be maintained that this action is brought about through the medium of the nervous system, on which the action of lead is so well known.

² See the Milroy Lectures, by Dr. J. Berry Haycraft, in the *Lancet*, 1894, vol. i. Nos. 7-9

the ultimate extinction of the family. This view does not, however, bring one to an end of the difficulties, and even in the hereditary cases of arterio-sclerosis alternatives present themselves; the hereditary tendency may be a tendency to a primary dystrophic process in the arterioles, or, as Lancereaux maintains, the faulty inheritance may lie entirely in the nervous system, the nutrition of the tissues being only indirectly affected. In like manner a nervous origin for gout and rheumatoid arthritis is being seriously entertained, and it would indeed simplify matters if we could rest here; but a history of mental strain and overwork, or rather, as Sir Dyce Duckworth would say, "irregular work," is certainly not necessarily present in cases of arterio-sclerosis, nor are the symptoms of any nerve trouble necessarily present.

If it be permissible to venture a somewhat vague suggestion as to what is one of the most probable causes of arterio-sclerosis, it is that what may be termed "excess" in its sometimes necessary and often long-continued forms seems, in the present state of our knowledge, to have the greatest share in the production of arterio-sclerosis. By such "excess" I mean merely a "strained" manner of living, which can by no means always be associated with any expression of reproach. It may be excess in physical labor; it may be excess and irregularity in mental work, including anxiety and worry; it may be the habitual taking of too much or too little food, from over-indulgence or insufficient means, both of which are so often accompanied by mental worry and anxiety. Such agents—or, rather, combinations of them—seem to be likely factors in the production of arterio-sclerosis; they can act on a single individual or cumulatively, through several generations, on a family.

On the whole, however, although such hypotheses appear very tempting, it must be admitted that the etiology of arterio-sclerosis remains as yet uncertain; but the condition must still be regarded as a primary one in pathology, though in some way allied to some of its alleged causes.