ARTERIO-SCLEROSIS:

ITS CLINICAL SIGNIFICANCE, MODE OF ORIGIN,
STAGES, AND CONTROL.

BY

WILLIAM RUSSELL, M.D., LL.D.,
F.R.C.P.Ed.,EMERITUS PROFESSOR OF CLINICAL MEDICINE, UNIVERSITY OF EDINBURGH;
CONSULTING PHYSICIAN TO THE ROYAL INFIRMARY.

INTRODUCTORY.

THERE still exists widespread doubt both regarding the mode of production and the clinical significance and importance of arterio-sclerosis, even when it is recognized as present. Owing to this both the individual and the community suffer. The questions which surround its mode of production have much and urgent significance, when the fully developed condition is present, as it is only by understanding the former that indications are provided for the management of the latter. The first clinical step is the recognition of the fact that the arteries used for clinical examination contract and relax—that is, that they become smaller and larger—irrespective of the pulse wave travelling along them. This variation in size indicates the degree of myotonia of the arterial wall. It is appreciable to the finger on the radial artery, but it is often more easily recognized in the brachial artery. It is, of course, the physiological sequence of the fibres of the muscular coat being placed transversely and not longitudinally. As in all hollow viscera, it varies, and there is a hypertonia and a hypotonia. The degree of myotonia is determined by two factors: (1) the nervous mechanism of the arterial wall, and (2) blood composition or blood content acting directly on the vessel wall and independently of central nervous control.

The next step is the biological and pathological fact that involuntary muscle hypertrophies under conditions such as prevail in arterial hypertonia. Persistent or recurring hypertonia leads to hypertrophy of the middle coat; and it is this thickening of the middle coat which constitutes the *first stage* of arterio-sclerosis. That this increase in thickness, and consequent hardness (sclerosis), of the arterial wall adds much to sphygmomanometer readings seems still to be questioned, or ignored, or tacitly denied in certain quarters. When this attitude is adopted it is hopeless to expect that contraction and relaxation of the thickened wall will be noted, or the fact that such contraction raises sphygmometer readings, while readings fall with relaxation. Unless the great clinical fact of this play of the arterial wall, even when sclerosed, is accepted, observed, and acted upon there can be no widely diffused understanding of the origin of arterio-sclerosis, or, when developed, of the significance of its clinical manifestations.

The persistence of this attitude to the matter under consideration is, I think, due in the main to thinking as well as speaking and writing of sphygmometer readings as "blood pressure." Another unfortunate term is "hypertension." The terms "high tension" and "low tension" were applied to the pulse before the sphygmometer was used in clinical medicine, but even then the question was asked what was meant. Was it the pressure of the blood on the arterial wall, or the condition of the wall itself? The same questions may be repeated to-day, for the terms "hypertension" and "blood pressure" are applied to sphygmometer readings. Warfield ignores the arterial wall as taking any part in this, but both terms are used in America, and whichever may be used it always means instrumental readings. Truncceek of Prague, in an interesting communication, writes of *hypertension vasculaire*; but it is quite clear that he means increased blood pressure in the aorta and its branches, which possess an elastic and not a muscular coat, for he enlarges upon the physical signs of dilated aorta and dilated subclavian artery. He does not mention the sphygmometer, but he recognizes constriction of the arteries, with diminution of their contained blood as leading to aortic hypertension.

THE CLINICAL SIGNIFICANCE OF ARTERIO-SCLEROSIS.

The view which is taken of the clinical significance of arterio-sclerosis seems to depend upon the pathological conception formed of the condition. As long ago as 1901 I showed that the pathological change in the arteries used for clinical examination, and to which the term "sclerosis" was applied, was thickening of the tunica media, due in the first place to hypertrophy of its muscle fibres, and that it was totally different from atheroma. Many observations since that date have confirmed my earlier observations. Recently I have examined a further series of arteries by a newer staining method to demonstrate the amount of fibrous tissue which had replaced the muscle tissue. In all of them the middle coat was much thickened; and, although the arteries were taken from old persons, they all showed the persistence of normal muscle fibres, especially in the outer part of the coat. The amount of fibrous tissue varied in inverse proportion to the amount of muscle tissue present. This partial fibrous transformation constitutes the *second stage* of arterio-sclerosis.

In this series of cases there were several in which the radial arteries were recognized during life as calcareous, and they were examined with a view to determine the situation of the calcareous deposit. In all the calcareous change was confined to the thickened middle coat. Even in these muscle fibres persisted. The middle coat was in no instance entirely infiltrated. Infiltration occurred irregularly in the inner segment of the media, while external to it there might be two or more layers of normal muscle cells. This calcareous deposit in the thickened and devitalized parts of the tunica media constitutes the *third stage* of arterio-sclerosis, and it is totally different from atheroma.

While those are the changes in the arteries used for clinical examination, the arterial change in the branches of the renal artery inside the kidney, and possibly the arteries inside other organs, is an obliterative endarteritis with sometimes thickening, at other times atrophy, of the tunica media.

The Effect of these Changes on Sphygmomanometer Readings.

The effect on instrumental readings of the diffuse and uniform thickening and hardening of the artery wall can be illustrated by two cases recently under observation. The first was a lady about 82 years of age. The mental faculties had been slowly deteriorating for a few years; she spent much time in bed, and latterly was entirely confined to bed. The radial pulse was regular and of moderate power; the artery wall was thickened. When the armlet was put on and blown up a pressure of 300 mm., which was the upper limit of my mercurial manometer, had no effect whatever on the radial pulse. On palpation with the finger the whole brachial artery was felt so hard, rigid, and pulseless that I came to the conclusion it was an example of complete calcification. In due time I got the artery, but it was not calcareous. Sections showed the thickened media to consist of mixed fibrous and muscle tissue. This is, of course, an extreme example of the resistance a sclerosed arterial wall offers to the armlet; but the greater includes the lesser.

The second case was a man, a metal worker, aged about 80 years, who, while convalescent from a severe acute pneumonia, was transferred from the Royal Infirmary to the men's sick-room in Queensberry House. When I saw him the morning after admission he was listless, apathetic, and had no pulse at the wrist. The radial artery was partly calcareous. At the bend of the elbow the brachial artery was superficial, large, thickened, and tortuous, and the artery higher up the arm was in the same condition. The tap of the pulse could be felt; but it was a mere tap. When the armlet was applied the pulse at the bend of the elbow was stopped at a pressure of 110 mm. With the stethoscope over the artery no sound was audible as the armlet was tightened. The heart sounds were inaudible. Under treatment he revived sufficiently to enable the radial pulse to be felt and counted. The armlet then gave a pressure of 125 mm., both with the finger at the wrist and at the elbow. In this case the circulation was so feeble that the pulse wave felt at the elbow did not reach the wrist, yet the armlet gave 110 mm.; next day, when the heart had

recovered sufficiently to drive the pulse wave to the wrist, the armlet gave only an extra 15 mm. Every clinician will realize the conclusion to which the facts in these two cases inevitably lead.

AGE AND ARTERIES.

That a man is as old as his arteries has long been a medical aphorism; and it has so biased medical thought that at the present time arterio-sclerosis is, apparently, in many quarters regarded as the inevitable result of advancing years. It has followed from this that the thickening artery, with its rising sphygmomanometer reading, is regarded as normal, and has led to the enunciation of the doctrine that "blood pressure (sphygmomanometer reading) rises 1 mm. for every year of life, beginning with 100 or 120 mm."

At the time of the inception of the above aphorism, and for long afterwards, it was probably not far off absolute truth, for the feeding and drinking habits of past generations fostered arterio-sclerosis and shortened life. The aphorism must not, however, be construed to-day as warranting the doctrine that age and thickened arteries are necessarily and naturally associated. Every doctor of experience must know that there are many persons in the later decades of life who have soft arteries and sphygmomanometer pressures of, say, 110 to 130 mm. I have seen many such. From the medical reports now coming into a life assurance office this statement receives confirmation. Further confirmation is available from America. I quote from an address delivered to the Association of Life Insurance Presidents in December, 1923, by Mr. Arthur Hunter, chief actuary, New York Life Insurance Company, on "Blood pressure; what affects it?" This definitely means sphygmomanometer readings, and is defined as the pressure the blood exerts on the arterial wall. The "average blood pressure" in a quarter of a million male lives "accepted for insurance at the regular rate of premium" according to age is given as follows:

Age.	Systolic Pressure.	Age.	Systolic Pressure.
20	120 mm.	50	130 mm.
30	123 "	60	135 "
40	126 "		

These figures are taken as the standard of normal lives. He then gives the figures of risks accepted, with an additional premium, by his company, and the extra mortality as follows:

No. of mm. over average for age.	Extra Mortality.
Plus 10 to 25 mm.	46%
" 26 to 35 "	88%
" 36 to 50 "	148%

These figures ought finally to dispose of the doctrine that arterio-sclerosis with its increased resistance to pressure is to be accepted as normal and inevitable. In America the "blood pressure" of every proposer is as much a necessary part of examination as the examination of the urine, but as British offices have not adopted this procedure no statistics are available here.

The big fact is that the thick-walled, sclerosed artery, with its high resistance to the pressure of the armlet of the sphygmomanometer, is no necessary accompaniment of advancing age. It is, on the contrary, the pathological product of a long course of disordered or overtaxed physiological processes.

There are, however, many old persons with markedly sclerosed arteries and sphygmomanometer pressures of 200 mm. or more; and it is probably this class of person which is responsible for the belief that these phenomena are merely a manifestation of old age, and of negligible clinical significance. I have always under observation individuals showing these arterial changes, 60, 70, or 80 years of age, but they are persons who have led a life of manual and physical labour. They are all broken down, unfit for work, and require supervision and care. Some are confined to bed and present various symptoms referable to their cardiovascular condition. They ultimately develop further manifestations attributable to that condition and die. Some of these old people show marked signs of a slowly failing myocardium, and when they do so a pressure of 200 or more

gradually falls, but the sphygmomanometer registers 100 or more so long as there is a pulse to feel. Corresponding cases occur in very different social conditions, of which the lady with the incompressible artery referred to earlier is an example.

MODE OF PRODUCTION OF ARTERIO-SCLEROSIS AND ITS CONTROL.

From the foregoing it will be seen that arterio-sclerosis with its high sphygmomanometer readings is no necessary accompaniment of advancing years; that, on the contrary, it must be regarded as the product of pathological processes. The first step is *hypermyotonia*, appreciable to the finger applied to the radial and the brachial arteries. This is the stage of *pre-sclerosis*. Continued hypertonia is caused by, and is proof of, blood impurity, using the word in the special sense of something in the blood which has an irritant or stimulant influence on the arterial wall. This irritant has two sources—it is absorbed from the alimentary tract, or it results from the retention of the waste products of cell metabolism which ought to be excreted. Another factor has now to be added—namely, increased activity on the part of endocrine glands whose internal secretions act as vaso-constrictors. The discussion of these various factors can be only briefly entered upon here. On the threshold of it stands the fact of differences in the chemico-vital constitution, or activities, or responses of individuals. I have seen and known of individuals under 30 years of age with sphygmomanometer pressures of 170 and 200 mm., lowered to normal by abstention from red flesh. In insurance practice I have had to deal with persons of this age with pressures of 160-170-180 mm., apparently in perfect health otherwise; and yet, in my judgement, unsuitable for life insurance. They belong to the small group of persons who die between 40 and 50 years of age with advanced arterial thickening, combined with cardiac and renal results of a persistent blood impurity, acting as a vaso-constrictor or as an irritant. These persons seem to possess a vessel sensitiveness to certain common articles of food: it is a food idiosyncrasy, manifesting itself by its action on the arterial wall. This is fortunately the extreme type, but the extreme example is valuable as establishing a law suggested by less pronounced types. Passing from idiosyncrasy to the big protein feeder—the person who eats red flesh three times a day—various examples are suggested, but this is a widely recognized type. I am informed that arterio-sclerosis is alarmingly common in Australia and that it is attributed to excessive flesh eating. The sheep-shearers are paid by the hundred fleeces; they work at great speed for hours, they consume three pounds of flesh daily, and they all have sclerosed arteries in early life. This is again the extreme example, but every medical man of experience must know this type, although in lesser degree. When the large eater also possesses an appreciative palate for wines or spirits the results become more readily apparent.

The gross type does not form a large group and need not be enlarged upon. There are, however, very many persons who illustrate various phases and degrees of corresponding effects, although in a minor measure. Of these the following are examples, and illustrate how the constriction may be controlled.

M., a retired medical man, aged 65, came to me in May, 1920, as his life had been declined for life insurance because his "blood pressure" was too high. Some time before he had had a left hemiparesis which was practically recovered from when I saw him. On examination I found the pressure 210 mm.; the arteries were thickened and to my finger hypertonic. He had a gouty inheritance. The urine was normal. He complained of inability for slight effort and of mental apathy. His past history was medically uneventful. He was not a big eater, but took a little whisky and enjoyed a glass of port after dinner. He had been troubled with constipation. The port was stopped, red flesh was reduced to a minimum, he was given a laxative to relieve the colon without purgation, and 5 grains of sodium iodide thrice daily. Under this treatment the sphygmomanometer readings came down to 150 mm. in thirteen days. The hypertonia of the arteries was checked but the arteries remained somewhat thickened. He regained mental interest and took considerable physical exertion without fatigue. In August he went for a fishing holiday and gave up taking the iodide salt. The result was that the pulse resistance rose to 185 mm. It fell with resumption of the iodide, and has remained stabilized between 150 and 160 mm. down to the present time; and within that period he has weathered a smart attack of pneumonia and a surgical operation of some magnitude.

Miss D., aged two or three years over 70, had sclerosed arteries and a mitral and aortic systolic murmur. She was having recurring headache, attacks of disordered vision which made her temporarily unable to read, and passing attacks of aphasia and of unilateral disturbances of sensation. The pulse resistance measured from 200 to 210 and 220 mm. By means of rest, careful dieting, and suitable medicinal management, aided by the understanding watchfulness of the lady's local doctor and her own intelligent co-operation, the pulse resistance was stabilized to round about 170 mm. But it took ten months to effect this.

Mr. S., aged 68, came to me in November last saying that he had "high blood pressure." He complained of listlessness and somnolence so great that whenever he began to read he fell asleep. The arteries were sclerosed and tortuous, and the pulse resistance to the armlet 210 mm. The heart apex was one and a half inches beyond the mammary line. The aortic second sound was accentuated. The urine was normal. He had always been of costive habit and took a daily laxative. He was not a big eater, but he drank burgundy, and suffered somewhat from gastric hyperacidity. Wine was stopped, the acidity was checked, and the laxative modified. The result was that the somnolence passed off, mental activity returned and he felt quite well, and the pulse resistance fell to 180 mm. He has continued to follow out instructions and the arterial condition remains stable.

It will be noted that in these three cases a stable pressure of 160 to 170 to 180 respectively—which was a lowering of 50 to 30 mm.—removed symptoms, and the patient kept well so long as instructions were followed. The arteries being sclerosed no attempt was made to lower the pressures further. Treatment was directed to removing and checking the hypertonia. The structural resistance of the thickened arterial wall could not be removed.

The foregoing cases illustrate the limitations of treatment when dealing with sclerosed vessels. The following case illustrates the progressive character of sclerosis when its early manifestations are not understood.

Mr. B. was seen by me in 1913 for digestive disorders. He was then 44 years of age. His pulse resistance to the armlet was 170 mm., the artery was thickened, the heart and urine normal. I did not see him or hear of him again until the end of February, 1924, when he was again sent to me. He was by this time 54 years of age. He complained of apathy and drowsiness; he could not remember what he had read in the morning newspaper half an hour after he had read it. The radial artery was small and calcareous. The pulse wave was moderate. The brachial artery was hard and tortuous, the pulse resistance was 240 mm. The sounds of the heart were faint. There was a trace of albumin in the urine. Such was the advance of the arterial changes, with their accompaniments, in the ten years between the ages of 44 and 54. The immediate prognosis, of course, was very bad.

CONCLUSION.

I have mentioned Trunczek's definition of vascular hypertension. He recognizes, as I have long preached, that the raising of aortic pressure results from constriction of the arterial wall and consequent diminution of the content of blood in the constricted arteries. He further recognizes that the true blood pressure is determined by the response of the left heart to the increased aortic pressure and that the left heart may become embarrassed and commence to fail, so that the arteries may be constricted without elevation of the blood pressure. The arterial constriction he attributes to blood composition, regarding it, as I have suggested elsewhere, as due to acidosis, traceable to "la dyspnée cellulaire." I frequently find that the application of the doctrine of acidosis is effective in some of the clinical manifestations of arterial hypertonia, both when the arteries are sclerosed and when they are normal. Whatever the precise nature of the blood impurity, it is more than a vaso-constrictor, for it leads to obliterative endarteritis in the kidneys and to capillary occlusion.

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BIBLIOGRAPHY.

- Warfield: *Arterio-sclerosis and Hypertension*. London, Henry Kimpton, 1920.
 Trunczek: *La dyspnée cellulaire comme origine de l'hypertension vasculaire*, *Bruzelles Médical*, 17 et 22 février, 1923.
 Russell: *Arterial Hypertonus and Arterio-sclerosis: Their Relations and Significance*, *Lancet*, June 1st, 1901; *Arterial Hypertonus, Sclerosis, and Blood Pressure*, 1907; *The Sphygmometer*, Baillière, Tindall and Cox, 1921.
 Hunter: *Blood Pressure; What Affects It?* New York, December 6th, 1923.
 Rolleston: *Reflections on High Blood Pressure*, *Lancet*, March 10th, 1923.
 Evans: *The Nature of Arterio-sclerosis*, *BRITISH MEDICAL JOURNAL*, March 17th, 24th, and 31st, 1923.
 Brockbank: *High Blood Pressure*, *Lancet*, October 20th, 1923.
 MacWilliam: *Blood Pressure and Heart Action in Sleep and Dreams*, *BRITISH MEDICAL JOURNAL*, December 22nd, 1923.
 Shaw: *Hyperpiesia and Arterio-sclerosis*, *BRITISH MEDICAL JOURNAL*, March 22nd, 1924.

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ACUTE APPENDICITIS AND INTESTINAL OBSTRUCTION.

BY

H. H. RAYNER, M.B., CH.B.MANCH., F.R.C.S.ENG.,
 CONSULTING SURGEON, MANCHESTER CHILDREN'S HOSPITAL;
 SENIOR ASSISTANT SURGEON, MANCHESTER
 ROYAL INFIRMARY.

THE occasional occurrence of intestinal obstruction as a complication of acute appendicitis has long been recognized, and there must be very few abdominal surgeons who have not had to face this sinister association many times. It is difficult to estimate with any accuracy the frequency of the complication: if we examine the terminal causes of death in fatal cases of acute appendicitis, as revealed in the published lists of individual surgeons, we cannot fail to notice a considerable discrepancy in the incidence of ileus and general peritonitis, though the mortality rates in these lists differ but slightly; nor is it possible for us to obtain any clear notion as to the type of ileus, whether mechanical or paralytic, in many of these cases. The same uncertainty in regard to ileus is encountered when we examine the non-fatal complications of acute appendicitis.

In the light of my own experience I believe ileus, and that frequently of a mechanical type, is associated with acute appendicitis much more frequently than the literature would lead us to believe, and that some, if not many, of the deaths ascribed to general peritonitis or to intestinal paralysis are due primarily to mechanical ileus. McWilliams,¹ in describing a series of 687 cases of acute appendicitis (1906-9), states that ileus, mechanical or paralytic, developed to such a degree in 18 cases as to require secondary operation, and of these 14 died. On the other hand, Beekman,² in describing a more recent series of 500 cases of acute appendicitis with 34 deaths, ascribes the cause of death to ileus in only 2 of these 34; but in the same paper stated, "It must be assumed, however, that some of our cases of diffuse peritonitis had a paralytic ileus, and therefore the above statistics may be misleading" (18 of the 34 deaths were stated to be due to diffuse peritonitis). McNeill Love³ has recently reviewed a series of 1,500 cases at the London Hospital (1919-22), and in a set of 421 of these, in all of which the operation was performed through the gridiron incision and drainage established through this, he records 102 complications, and of these 17 were instances of ileus or intestinal obstruction (10 fatal) and 14 of general peritonitis.

Furthermore, we must not lose sight of the occasional and very unwelcome appearance of acute obstruction as a remote sequel of appendicitis; this may occur months or years afterwards, and is particularly prone to follow operations for acute appendicitis that have required drainage. It is noteworthy because the patient and even his doctor may be disposed to minimize the gravity of the trouble during its early stages, knowing that the most common cause of acute abdominal disease—the appendix—has been previously removed; yet many of these late attacks, two, three, or more years after the appendix operation, are due to strangulation of the bowel and call therefore for immediate recognition, whilst those developing some weeks or months afterwards are more commonly due to adhesions. McGlannan⁴ has recently recorded 276 cases of acute intestinal obstruction in 25 of which there had been a previous operation for acute appendicitis, and in 23 of these drainage had been employed.

It is, however, with intestinal obstruction as an immediate complication of appendicitis that I am concerned here, and I will now describe the pathological conditions, as seen at operation, responsible for obstruction. At least three distinct types can be recognized.

1. Adhesion by Lymph Exudate of Lower Ileum to Pelvic Wall or, less frequently, to Neighbouring Loops of Small Intestine.

This I believe to be the commonest cause of intestinal obstruction occurring before or soon after operation for acute appendicitis. This condition is most commonly set up by perforation or gangrene of the pelvic appendix; in