The sphygmometer : its value in practical medicine / by William Russell.

THE

SPHYGMOMETER:

ITS VALUE IN CLINICAL MEDICINE.

WILLIAM RUSSELL.

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ITS VALUE IN PRACTICAL MEDICINE

BY

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TO

THE PRESIDENT AND COUNCIL OF THE
ROYAL COLLEGE OF PHYSICIANS,
EDINBURGH

WHO HONOURED ME BY ASKING ME TO DELIVER THE

GEORGE ALEXANDER GIBSON
MEMORIAL LECTURES,
1920

PREFACE

THE chapters of this book fall into two groups. One group consists of an extension and elaboration of the "George Alexander Gibson Memorial Lectures," which the President and Council of the Royal College of Physicians did me the honour of asking me to undertake, and which were delivered on the 10th and 12th of May, 1920. The second group comprises lectures delivered to graduates or to students in Edinburgh. The chapters which form this group are reproduced as they were originally published in the medical journals. The rewriting of these, so as to avoid repetition, has been found to be impracticable; while, on the other hand, there are advantages in reproducing them in their original form, as they dealt with the subject in the way which was most appropriate at the time, and, in view of the still unsettled state of opinion, is still appropriate.

In dealing with the subject one cannot but deplore the fact that it has not yet been generally accepted and taught to students that sclerosed arteries are not necessarily or even usually atheromatous, and that the sclerosed artery seldom if ever becomes a rigid tube, but retains its active responsiveness to blood and other conditions. Sound pathology is as necessary as accurate physiology for the doing of satisfactory work in practical medicine, and there lies a very special responsibility on the teachers of these two important subjects.

Preface

The publication of this work is the outcome of my having undertaken to give the Gibson Memorial Lectures. For the purpose of the lectures I gleaned material almost entirely from my private case-books; while the lectures to graduates or to students were delivered either in the University or in the Royal Infirmary, and dealt with cases in my wards at the time they were delivered. The combination of private and of hospital observation has advantages which it is hoped will be evident to those interested in the subject.

The estimation of heart power is so important, and is so closely incorporated with all pulse questions, that a chapter has been added on this subject, which is largely made up of a lecture delivered during war-time, as a help and a guide in determining what was then a very vital question for many of our young men called to serve their country.

W. RUSSELL.

EDINBURGH,
December, 1920.

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INTRODUCTION

THE main object the writer has in view in presenting this book to his professional brethren is to illustrate the value of sphygmometer observations in the daily work of the physician and the family practitioner. As a preliminary to that object it is necessary to deal shortly with the long-established practice of feeling the radial pulse. The pulse-wave in the radial artery was accepted as indicating the rapidity, the regularity, and the strength of ventricular contraction. Words were in use, such as *compressibility*, *pressure*, *tension*, to define the amount of force required to stop the passage of the pulse-wave. This to persons of critical mind involved the consideration of the condition of the artery wall, for to them it was clear that when it was thickened, *compressibility* was altered and *more pressure* was required to arrest the passage of the pulse-wave. The word *tension* came into use, and led to complete confusion, as the word was used sometimes to define the pressure of the blood on the artery wall, at other times the condition of the wall itself, without defining what was meant or indicating what difference, if any, there was between it and the other words in use.

In 1901, before the sphygmometer was used in practical medicine, the present writer published in the *Lancet* and

in the *Transactions of the Medico-Chirurgical Society of Edinburgh* a paper on " Arterial Hypertonus and Arterio-Sclerosis," in which were given the results of years of observation on these conditions. Part of that communication is utilized now, as the observations then recorded enabled him to interpret aright sphygmometer findings, and ultimately to realize the value of sphygmometer observations in many abnormal states.

In that communication it was shown that arterio-sclerosis in the radial artery and in other arteries consisted of a thickening of the artery due to thickening of the muscular or middle coat, and had no resemblance whatever to atheroma. Atheroma occurs in patches, and both it and calcareous infiltration are comparatively rarely found in the radial artery even of old people. Arterio-sclerosis, on the other hand, is a uniform thickening in their whole length of the middle coat of arteries, and is very common. The difference between the two conditions is shown in Figs. 1, 2, and 3 as it is seen in the radial artery.

The thickened state of the wall referred to was associated with the clinical terms *high tension* and *high pressure* applied to the pulse. There was no attempt clearly to separate the pressure of the pulse-wave on the wall and the condition of the wall itself as a living muscular tube.

It was further shown that the idea that the thickened artery wall made the artery a rigid tube had to be abandoned; that, on the contrary, the thickened artery retained its power of contracting and relaxing, and when contracted its wall was further thickened and its lumen proportionately diminished, as shown in Fig. 3. The term *hypertonus* was applied to this contraction, as it

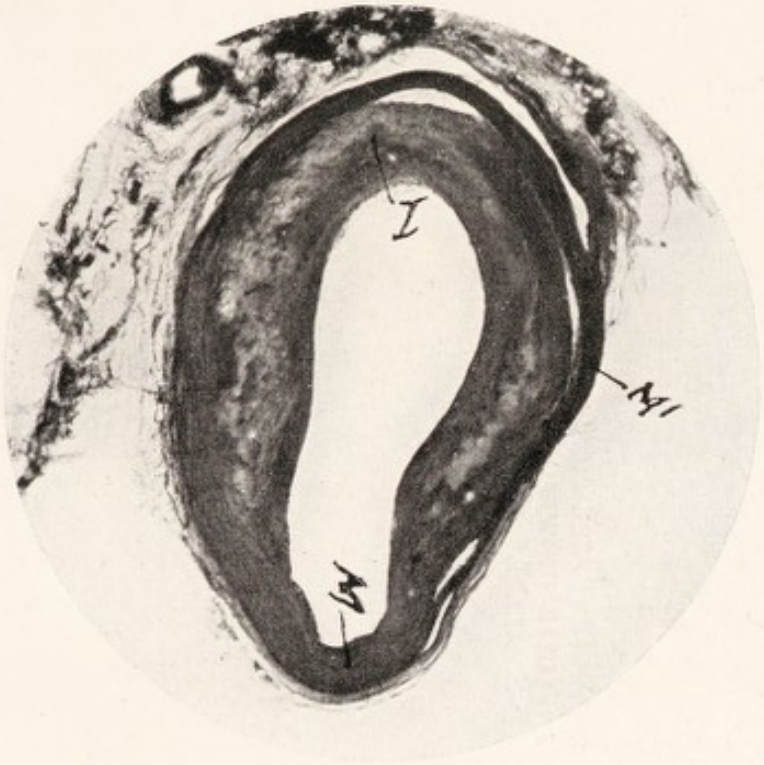

FIG. 1.—ATHEROMA OF RADIAL ARTERY.

I, Thickening and degeneration of intima ;
M^1, atrophy of media corresponding to atheromatous part ;
M, thickened media where there is no atheroma.

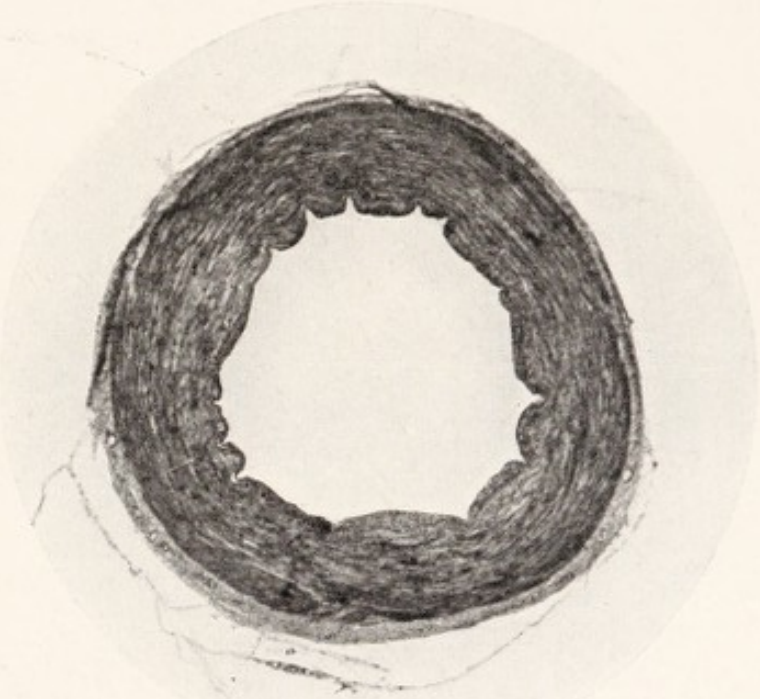

FIG. 2.—ARTERIO-SCLEROSIS OF RADIAL ARTERY, SHOWING THICKENING OF MIDDLE — *i.e.*, MUSCULAR—COAT.

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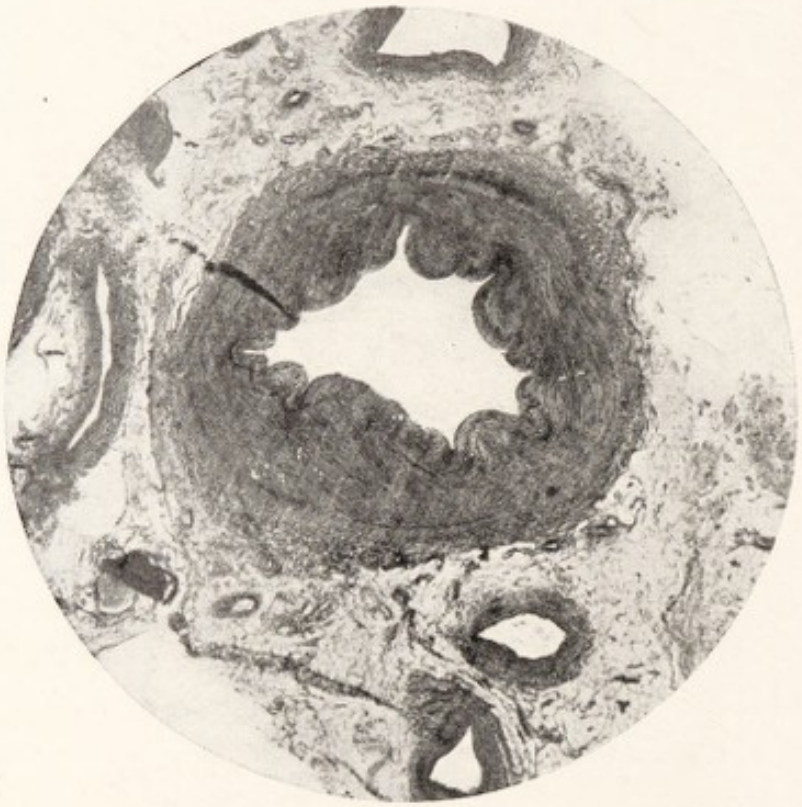

FIG. 3.—ARTERIO-SCLEROSIS AND HYPERTONUS OF RADIAL SHOWING THICKENING OF WALL AND SMALL LUMEN.

was an exaggeration of normal *tonus;* and thickened arteries seemed to be particularly liable to become hypertonic.

The use of another term—namely, *elasticity*—as applied to arteries, obscured the fact that, quite apart from the idea of stretching and recoil, the muscle of the wall was not only a living, but an active mechanism, determining the lumen of arteries, and acting under many different influences.

It is important to understand that *hypertonus* occurs both as a prolonged and as an oft-recurring increase of the normal degree of contraction which vessels have in virtue of their muscular coat, and which is physiologically called *tonus*. Hypertonus carries with it an important influence on the circulation, apart from any alteration in blood-pressure—namely, a diminution in the blood-containing capacity of the vessels from a lessening in their lumen. The diminution in lumen necessarily occurs when an active contraction of the vessel wall is present. In diseased conditions it has very important results, and determines symptoms of grave significance.

Its recognition clinically can usually be made by means of the finger.

Evidence in support of these contentions was submitted in the paper referred to, and with greater fullness in my book on "Arterial Sclerosis, Hypertonus, and Blood-Pressure," which has been out of print during the period of the Great War, but which, it is expected, will be reissued shortly. Here, however, it may be stated that such confirmation as was then available was obtained by means of sphygmographic tracings and arteriometer observations.

In considering the cause of hypertonus, I at that time

regarded it as due to errors in primary or in secondary digestion; but as the question of causation will be dealt with more fully later, further reference to it is not necessary at this stage.

Having dealt with the radial artery in the manner indicated, the next step was to examine the brachial artery in the same way. It was found that the brachial artery became thickened and hardened in the same way as the radial, and that the thickening and hardening were, of course, easily appreciated by the finger. It was also easy to recognize by the finger that it tightened up, became smaller and harder—that the tightening up relaxed. *Hypertonus* occurred in it as in the radial, and could sometimes be more easily noted by the finger than in the radial.

The following figures (4 and 5) illustrate the thickening of the wall, due to thickening of the middle coat, when compared with a normal brachial artery. The figures further establish my contention that arterio-sclerosis is totally different from atheroma, and that vessels the size of the brachial participate in it.

Fig. 6 is a section of the brachial artery, showing the ordinary lax condition of a normal brachial after death; the other (Fig. 7) shows the appearance produced by contraction of the muscular coat—by, in fact, hypertonic spasm. These two illustrations are from the same brachial artery. They are given here, not only to illustrate the important fact that the living artery can contract so much that its lumen is almost obliterated, but to press the truth that the sclerosed artery retains this power of contraction, and must not be thought of as a rigid tube which has lost its contractility. It is only when this is fully realized that the sphygmometer becomes an instru-

FIG. 4.—ARTERIO-SCLEROSIS OF BRACHIAL ARTERY SHOWING THICKENING OF MEDIA.

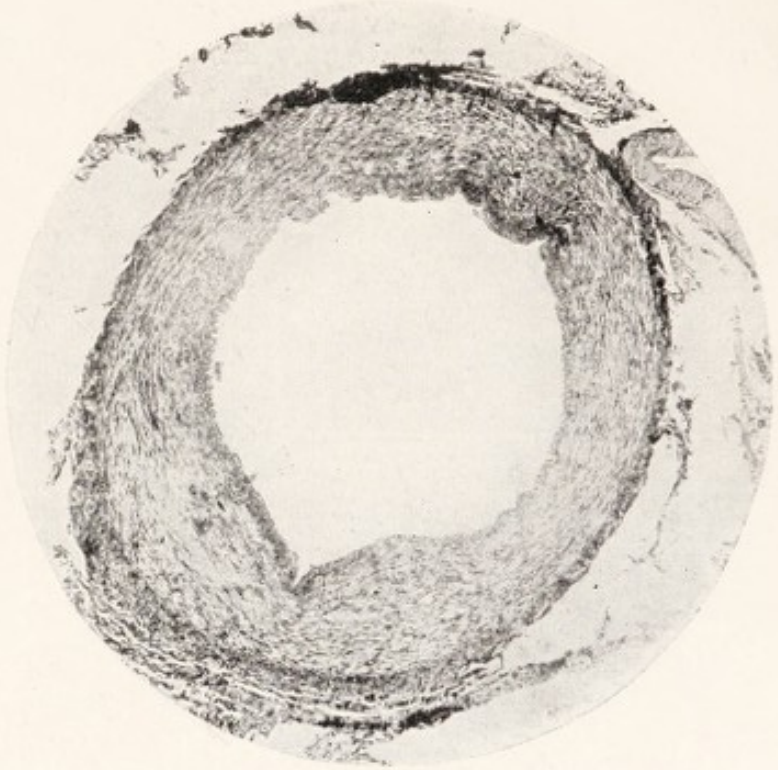

FIG. 5.—ANOTHER BRACHIAL ARTERY, SHOWING THICKENING OF MIDDLE COAT

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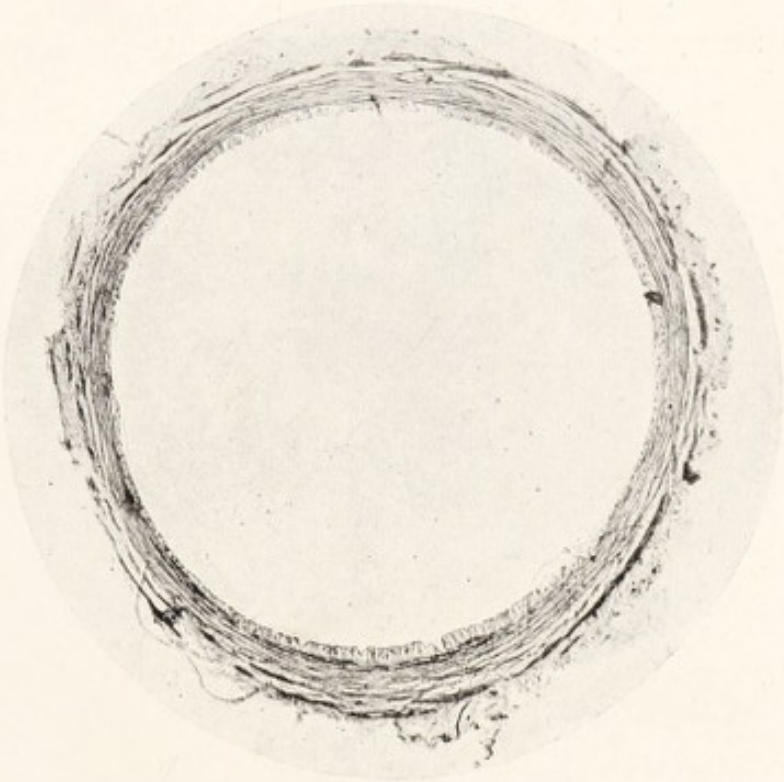

Fig. 6.—Normal Relaxed Brachial Artery.

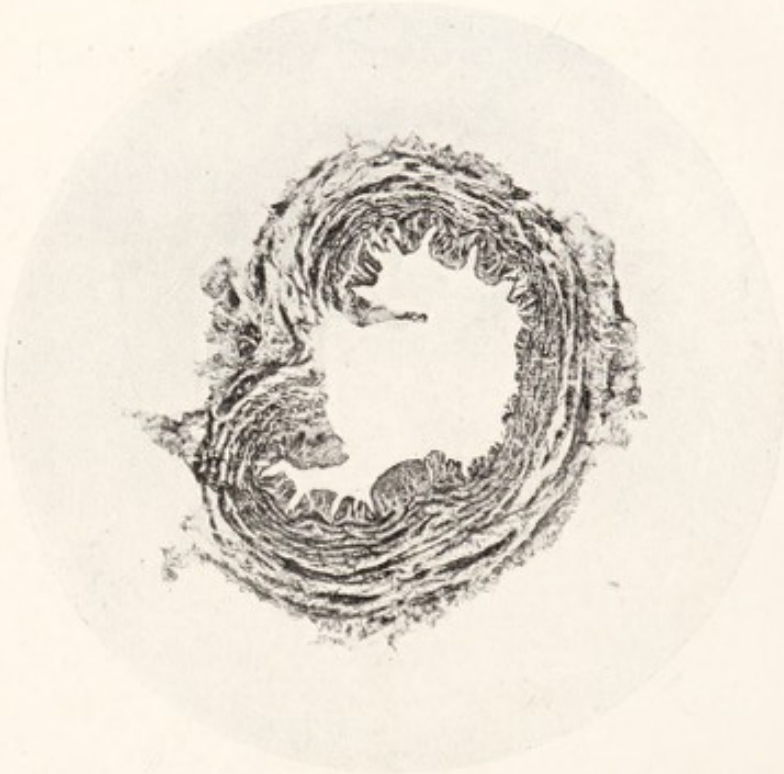

Fig. 7.—Hypertonic Contraction of the same Artery as Fig. 6.

ment of great clinical value to the practitioner seeking knowledge of his patient's condition by every available channel. It is perhaps the most important instrument put into the hands of the practitioner since the introduction of the stethoscope.

CHAPTER II

THE SPHYGMOMETER AND ITS PROBLEM

THE sphygmometer was introduced into practical medicine as an instrument that would accurately measure "blood-pressure." By blood-pressure was meant the pressure of the blood upon the arterial wall, such as would be recorded if a cannula were introduced into the radial artery and attached to a manometer. "Blood-pressure," of course, represented the power of the left ventricle. This proposition might have been accepted without cavil as correct for a normal artery with a normal measure of tonus. The astounding fact was that the proposition was accepted as applicable to all conditions, irrespective of pathological changes, in the vessel wall itself. Eminent physicians held that the sphygmometer gave the true brachial blood-pressure, and allowed only 5 mm. Hg pressure for the arterial wall, no matter what its condition. Owing to this extraordinary position, sphygmometer observations are still constantly referred to as "blood-pressure." The writer's contention has always been that thickening of the artery wall from sclerosis, from hypertonus, or from hypertonus in a sclerosed artery, was the important factor in sphygmometer readings.

The following paper is reproduced here, as it deals with the vital question of the estimation of blood-pressure:

The Clinical Estimation of Blood-Pressure : The Finger and the Hæmomanometer.*

To the clinician the estimation of blood-pressure means the estimation of the driving power of the left ventricle. It is mainly as a means of judging of heart power that the pulse is felt. No doubt peripheral resistance from various causes will immediately lead to a rise of blood-pressure in the aorta, but the increased pressure can only be maintained there by increased effort on the part of the ventricle. The result is that blood-pressure represents heart power, no matter what the factor may be which precedes the calling out of increased action. It is this simple fact that makes the estimation of blood-pressure a question of prime importance to the clinician.

The clinical estimation of blood-pressure has in the past been made through the medium of the finger placed on the radial artery. It was known that the radial artery varied in the thickness of its wall, and it was this factor that led to the use of such terms as " tension " and " compressibility." Whatever confusion existed as to the exact meaning of the terms in use, the fundamental aim was to estimate the blood-pressure. In a tube the wall of which varied so greatly in thickness as the radial artery, the estimation could not have been made were it not for the pulsatile movement of the blood and the effect of that movement on the arterial wall. Whatever the difficulties might be, the aim of the clinician was always the same, and the skill which could be acquired is nowhere better

* A paper read in the Section of Medicine at the Annual Meeting of the British Medical Association, Sheffield, 1908 (*British Medical Journal*, October 10, 1908).

portrayed than in the little book on "The Pulse" by the late Sir William Broadbent.

Recently hæmomanometers and sphygmometers have become widely used, and are held by some clinicians to be a means of determining blood-pressure with an accuracy hitherto not attainable. The contention is that the amount of pressure, as measured by a manometer, which is required to obliterate an artery is practically the blood-pressure inside that artery. It is maintained that, no matter how thick the arterial wall be, it is practically negligible, or at most requires only some 5 mm. Hg to overcome its resistance. Hæmomanometer readings run, say, from 80 to 100 to 200 or 300 mm. Hg, so that after deducting 5 or so the remainder represents the pressure exerted by the blood on the vessel wall.

Now, we were all taught that the "compressibility of the pulse" depended not only on the pressure of the blood inside the artery, but also on the thickness of the arterial wall. The modern cult assures us this was wrong; that all we really felt was blood-pressure; that when we thought there was low blood-pressure inside a thick-walled radial artery it was an illusion, for this new mechanical device gives a high reading, and the vessel wall being negligible, there is no other possible interpretation of the phenomenon. All who think otherwise are relegated to a region of ignorance inhabited by those who would dispute the claims of the clinical thermometer as compared with the thermic sensibility of the hand.

If it is thought by anyone that this is exaggeration, I commend to his perusal the report of the discussion on arterio-sclerosis at the Montreal meeting of the British Medical Association.

This extraordinary contention is based upon experi-

ments made on arteries taken from the dead body, and said to have been " sclerosed." I need only say that the term meant that they were, or were believed to be, thickened. The conclusions drawn from these experiments have been accepted by some clinicians apparently without question, while other clinicians have not accepted them. In two recent reviews which appeared in the *British Medical Journal* the reviewer, who was presumably a man of outstanding authority, definitely stated that. The reviewer of one of these in the *Lancet*, commenting upon the contention that the vessel wall was a factor, said my conclusions " would be anticipated."

I am fully aware that the common sense of many in the profession does not see that the contention I have indicated can be correct, and I hope to satisfy at least that section of my brethren that their scepticism has been well founded.

Radial Arteries.—I begin my proof by showing you sections of radial arteries, all of which had been recognized by myself during life as thickened. You will note how greatly they vary in size, in thickness of wall, and in the relation the lumen bears to the thickness of the wall. This last point is important, for a tube with a bore of $\frac{1}{4}$ inch and a given thickness of wall requires more power to compress it than a tube of $\frac{1}{2}$-inch bore with the same thickness of wall. The arteries from which these sections were taken were uniformly thickened, not atheromatous, and it is to this change that I have proposed that the term " arterio-sclerosis " ought to be confined.

Brachial Arteries.—I next draw your attention to sections of three brachial arteries, but before doing so I show you the pressure-chart in the first two cases. In the first case the pressure was about 100 mm. Hg, in the second it ranged between 265 and 220 mm. Hg.

The *first* slide is from the perfectly normal radial artery of a girl of eighteen who died of pneumonia. It is to be noted that the internal surfaces are in contact, as the surfaces of a thin rubber band would be if similarly placed. The wall is thicker than it was at the time of the removal of the artery from the body as the result of the action of the formalin in which it was fixed.

The *second* is from a patient who was under my care and observation in the Royal Infirmary, Edinburgh. She had granular kidneys and very thick, hard arteries. The hæmomanometer readings varied between 265 and 220; below the latter figure we never found them. After death the brachial artery was procured and put in 5 per cent. formalin solution. This vessel I tested by pressing upon its wall directly with Oliver's hæmodynamometer, and found the pressure required to bring its surfaces together was equal to 120 mm. Hg. With the encircling bag the pressure required would have shown a manometer reading of from 160 to 200 mm. Hg. This artery felt like caoutchouc.

The *third* is from a brachial artery of great importance for our present purpose. It was taken from a man I had watched for over a year, and who had a pressure of 200 to 220 mm. Hg. I got this artery about two hours after death, and as soon as possible I attached it to a schema through which I could let water flow and know the pressure exercised by the water. I then placed the artery along an assistant's finger, covered it with some folds of gutta-percha tissue, and surrounded the finger with the smaller bag or wristlet of Oliver's original form of modified Riva-Rocci. The bag was connected with a manometer, and it was found that with a stream of water trickling through the vessels it required a pressure of about 100 mm. Hg

to stop the flow through it. The artery was then put into 5 per cent. formalin solution, and after it was fixed in this way I again passed water through it and found that it required a pressure of 160 mm. Hg to stop the flow. This is the artery which I hold in my hand; it feels like a piece of india-rubber, but not very much harder than it felt during life. If this artery had been tested some longer time after death it would have lost all that tonicity of wall which the living muscular coat, as we all know, gives to it. I used this artery so soon after death that it had not lost this, as was shown by the fact that it contracted and thickened by the mere handling of it. Placing it in formalin solution fixed it, and made it retain at least the consistence it had at the time it was tested. The formalin, I believe, raises the consistence above the ante-mortem consistence; but the formalin-hardened artery more nearly represents the consistence of the living vessel than an artery which has undergone post-mortem relaxation and softening.

Conclusions.—I contend that these observations bring us back to the common-sense view that a thick-walled vessel requires much greater pressure to stop the pulse-wave travelling along it than a vessel of normal thickness requires. In Case 2 with a hæmomanometer pressure varying from 220 to 265 mm. Hg, the vessel wall represented somewhere about 150 mm. Hg or more. In Case 3, with a pressure varying from 200 to 220, the vessel wall represented a pressure of 100 to 140 or more. I submit that the doctrine that the thickened vessel wall is negligible, or that the part it plays never exceeds 20 to 30 mm. Hg, is a very grave error when the hæmomanometer is used to help clinical opinion or diagnostic judgment.

I ask your attention to the other extreme of clinical

phenomena. The next section of the brachial artery (Case 4) is from a woman who died of malignant disease of the liver and omentum. For days before death the radial pulse was a mere flicker. I would have not estimated the blood-pressure at more than 20 mm. Hg, that figure quite corresponding with the extreme prostration and debility, which was, of course, shared in by the heart. This patient's heart was not doing more than one-fifth or one-sixth of the work of a normal heart, and to contend otherwise would be, I take it, an expression of clinical incapacity. Yet when the hæmomanometer was applied over the brachial artery it required a pressure of 95 mm. Hg to arrest the flickering pulse in the radial artery. At the post-mortem examination the brachial artery was found relaxed, with its internal surfaces in contact. I passed a probe along the interior of the vessel, and placed it in formalin solution, with the object of allowing the vessel to firm up round the probe. The vessel so hardened, when tested as the others were tested, was found to require a pressure of 60 to 70 mm. Hg to stop the flow of water through it.

What happens in cases of this kind—and I could give you many such—is this: As the circulation becomes feeble the brachial and other arteries contract to accommodate themselves to the smaller volume of blood passing along them; this means a thickening of the wall and a diminution in the lumen, with the result that the hæmomanometer reading is relatively high, while the true blood-pressure is very low.

Meanwhile I contend that the finger is still the main means of estimating blood-pressure in clinical work; that in using the hæmomanometer the vessel wall becomes a disturbing factor whenever we are dealing with patho-

logical conditions; and that in the high pressures obtained from thickened arteries the arterial wall is the predominant partner.

Diastolic Pressure.

So-called "diastolic pressure" has received much attention from the time when Dr. George Oliver introduced the hæmodynamometer. That instrument was used to press upon the radial artery, and had a clock-face marked in sections representing mm. of Hg, and armed with a pointer which swung with every pulse-beat. As pressure on the artery was increased the swing of the pointer increased up to a certain point, and then decreased. The point of maximum swing was thought to be true blood-pressure. Later the stethoscope was used to listen to the sound developed in the brachial artery as the vessel was being compressed by the armlet applied to the upper arm. Observations made in this way are interesting, but they, no more than the "systolic pressure," which is the amount of pressure required to obliterate the pulse-wave, can be accepted as recording blood-pressure. It is evident that the factor of resistance in the vessel wall occupies the same place in both sets of observations, and in neither is it a record of blood-pressure.

CHAPTER III

SCLEROSED ARTERIES WITH HIGH SPHYGMOMETER RECORDS

DISCUSSIONS still take place on the question of the reduction of " blood-pressure," meaning thereby the reduction of sphygmometer readings. It seems to me that this is the unfortunate result of the continuance of the use of the word which accompanied the introduction of the instrument into practical medicine. The contention that the vessel wall was a negligible factor in what the sphygmometer recorded has been largely abandoned, no doubt with reluctance by many; but observations and investigations made on both sides of the Atlantic have confirmed my contention that a sclerosed artery wall was an important factor in determining sphygmometer reading and that hypertonus, even in a normal artery, was also a factor. The fact that sphygmometer reading does not record " blood-pressure," and therefore is no index to heart power, has tended to discredit the use of the instrument. This is unfortunate, although more or less inevitable. It will be shown that the instrument is of great value where the information it provides is correctly interpreted and correlated to information obtained through other channels of observation.

As illustrating and supporting this contention it is proposed in the first instance to deal with cases giving high sphygmometer readings. High-pressure cases vary,

of course, in the height of the readings they supply. In the cases dealt with in the following sections the arteries were anatomically, and therefore permanently, thickened and hardened—that is, sclerosed, in the sense in which the word is here used.

Cases of high pressure in which albumin was present in the urine will be dealt with in the first place.

A.—High Pressure with Albumin in Urine.

Case 1.—Mr. G., aged sixty-three, was a typical case of uræmic dyspnœa with cyanosis. The dyspnœa is frequently referred to as "cardiac asthma," a term which misleads in more than one direction. He had had many similar attacks. There was albumin in the urine, and the brachial pressure was 280 mm. Hg. Attacks of this kind are frequently relieved by the administration of erythrol tetranitrate. One grain may be given, its effect watched by means of the sphygmometer, and the dose repeated two or three times, but not more frequently than hourly. If relief is obtained iodine may be given, or spirit of nitrous ether. Attention has to be paid to clearing out the alimentary tract by means of blue pill or calomel, and later by means of cascara sagrada, phenolphthalein, and taraxacum. Salines, in my experience, are not satisfactory. In urgent cases bleeding may be adopted. The diet ought to be restricted to milk, fish, and vegetable foods. With a fall in sphygmometer reading relief is procured, but no attempt ought to be made to reduce the readings to normal. When relief of dyspnœa and cyanosis is procured no further heroic measures ought to be employed, and any further lowering of pressure must be obtained by means of the other

measures indicated. These measures may be successful in still further reducing the readings; and, even if they are not, they will delay or prevent recurrence of the dyspnœic paroxysms. The explanation of the paroxysm is that a wave of hypertonic contraction has affected the arteries, and when that is stopped relief is obtained. The anatomical thickening is permanent, and gives a continuously high reading up to 200 mm. or more. The hypertonic wave can to a certain extent be kept in check. It is determined by blood conditions which can only be modified in view of the sclerosed vessels and the accompanying inefficiency of the kidneys. The life of such patients can only be prolonged and made more comfortable. Cure in its full meaning cannot be effected, but the immediate danger, consequent upon the continuation of the paroxysm, can be averted; while much can sometimes be done to lessen the tendency to, or to diminish the severity of, the paroxysms.

Case 2.—Mr. G., aged fifty, seen in consultation, was suffering from severe headache over the occiput and vertex. He had had many severe attacks of dyspnœa, "uræmic asthma." The arteries were thickened, the heart enlarged, there was a small amount of albumin in the urine, and the brachial pressure was 220 mm. Hg. The general principles of treatment already referred to were applicable in this case. The immediate prognosis was not so bad as in the former case.

Case 3.—Mr. C., aged fifty-nine, from the West of Scotland, consulted me in February, 1912. He complained of pain in the left hip, from which he had suffered for eighteen months, and had been off work for six months. The hip condition was evidently rheumatoid arthritis. The vessels were thickened, the heart enlarged, and the

brachial pressure was 250 mm. Hg. The urine was pale, and contained much albumin. In a patient showing these changes there was nothing for me to do but to write to his ordinary medical attendant drawing his attention to the serious condition present.

CASE 4.—Mr. N., aged fifty-seven, a gamekeeper in the North of Scotland, was sent to me in March, 1910. He had a profuse nose-bleeding early in January, which had been repeated frequently. This had made him anxious and nervous, although he had been able to attend to his duties. His appetite was good, and the bowels moved freely daily. He had no other symptom of unhealth. The *pulse* was 124, the artery thick, and the brachial pressure 200 mm. Hg. *Heart:* The apex was out and down, 2 inches beyond the mammary line; there was a somewhat diffuse impulse over the precordia; the first sound at the apex was impure, and there was an aortic systolic murmur. *Abdomen* was normal, save that the area of stomach tympanicity was large. *Urine* was pale in colour, specific gravity 1012, faint cloud of albumin. *Eyes:* No albuminuric retinitis. *History:* He had been a large flesh-eater all his life. He did not drink beer, and he often did not taste whisky for weeks or months.

Remarks.—Here was a patient who had for three months recurring epistaxis; a pulse beating over 100 per minute; a high brachial pressure, notwithstanding repeated hæmorrhage; a dilated and rapidly acting heart; and albumin in the urine. This was clearly a patient who ought to be in bed, carefully watched, and treated by his medical attendant, or sent into a hospital or a nursing home. The *treatment* indicated in a case of this kind is, firstly, rest in bed, and a dietary without red flesh. Rest alone may lessen the pulse-rate, improve

the heart condition, and bring the brachial pressure down somewhat. Further treatment might be the administration of iodide of iron, with a view to obtaining the tonic action of iron with the special action of iodine. The heart dilatation might even require a course of strophanthus. Later the patient should have strongly impressed upon him the necessity of avoiding red flesh, and of making his diet as spare in quantity as possible. In this patient a laxative did not appear to be required. The immediate prognosis is fairly good in such cases if the necessary conditions are attainable; but the thickened arteries remain and the albumin in the urine persists. The later outlook is bad.

Case 5.—Mr. B., aged fifty-nine, a commercial or business traveller residing in London, consulted me in November, 1910. He complained of feeling very nervous, and as if his legs would break down under him. He was sleeping badly. He attributed his symptoms to overwork, owing to the illness of his principal throwing more work and responsibility on him. There was a history of retinal hæmorrhage four years ago. He was a bachelor, and had syphilis as a young man. He had always been a free feeder and a free drinker, although not taking liquor to excess in the popular sense. The pulse was 76; the brachial pressure 210 mm. Hg; the right radial was not so hard and thick as the left; the right brachial was hard, resistant, and tortuous. The wave in the arteries was small and weak. The heart's apex was 1 inch beyond the mammary line; the first sound was faint, and the aortic second sound accentuated. The urine was pale and showed a faint trace of albumin. There were hæmorrhages in the fundus of each eye. I advised rest, and gave potassium iodide (5 grains) and tincture of squill (10 minims), to be

taken three times a day; and as a laxative 2½ grains of colocynth and hyoscyamus pill.

I saw him early in January, 1911. He was feeling better, and had no distressing symptoms, but he had not been able to rest. Pulse and brachial pressure were much the same as they had been in November. The urine had a specific gravity of 1020, and there was no albumin. Ten grains of bicarbonate of soda were added to each dose of the iodide and squill mixture.

He was seen again a fortnight later, when the pulse and pressure were much the same as before, but he was feeling done up, and I strongly represented to him the necessity of stopping work.

Remarks.—This is not an uncommon class of case to see in the consulting-room, and, in spite of much talk about the early diagnosis of disease, the fact is that cases of this kind have had no or little medical attendance before they are seen by the physician, or by the ophthalmologist, who has been consulted regarding impaired eyesight. The first essential of treatment is physical repose in a nursing home, or hospital, or at home, but men of this stamp, under the pressure of circumstances, keep at work until a complete breakdown or disaster of another kind overtakes them.

Case 6.—Mr. C., aged sixty-seven, had a men's tailoring business and was in easy circumstances. He was seen in consultation in May, 1916. He had felt quite well until two months before, when he began to be breathless; he had not improved, but was losing ground. He was pale and languid-looking. The radial arteries were thick and tortuous, with a poor pulse-wave; the brachial pressure was 190 mm. Hg. The heart apex was 1½ inches beyond the mammary line, and its action was feeble.

The urine contained albumin and phosphates. In spite of all efforts to improve the myocardium, he became progressively feebler and died in a few months.

Remarks.—This is another illustration of a man who had enjoyed good health, in so far that he had no symptoms leading him to seek advice until he suddenly began to get breathless when performing his ordinary duties, and yet there was advanced arterial thickening with kidney disease, and a myocardium which suddenly began to give way and steadily got worse notwithstanding all the measures taken by his doctor to improve the condition. This is only another example of the fairly common experience that arterial, kidney, and heart changes may be fully established before the individual has had symptoms which suggest to him the need of consulting a physician or of seeing his family medical attendant.

The foregoing cases illustrate the well-recognized class of atrophic or granular or cirrhotic kidney characterized by sclerosed arteries, enlarged heart, albumin in the urine, and a high brachial pressure. The treatment can only be palliative. The prognosis is worse than in the next series, and yet it is surprising how much can be done for some of these cases when conditions of life can be made fully satisfactory. The following case illustrates this fact:

Case 7.—Miss C., aged fifty-two, was sent to me in May, 1909. She was a member of a gouty county family. The history was that in February, 1908, she had an attack in which she had difficulty in speaking, and used wrong words. She had been kept in bed for a month at that time. In 1900 there had been a loss of memory for ten days, but speech was not then affected. Memory remained impaired for a year. When seen in May, 1909, she still had difficulty with some words. The radial artery was

hard and the brachial pressure was 220 mm. Hg. The apex of the heart was down and out. The urine contained considerable albumin, although there was none when her medical attendant had examined it. The general line of treatment as suitable to such cases, and already stated, was followed. She was seen in September, 1909, when the brachial pressure had come down to 180 mm. Hg and the urine showed no albumin. She was seen again in July, 1910, by which time she had greatly improved in general health and was feeling well. The pressure had fallen to 165 to 170 mm. Hg. In 1920 she was alive.

Remarks.—This case looked a very bad one, and yet under treatment directed to diet, to daily intestinal evacuation, and the administration of iodides, she made a very satisfactory improvement. The arteries were thickened, and no heroic measures were attempted to reduce the sphygmometer readings to normal. The fall of 50 mm. Hg represented the relaxation of the hypertonus, and left the thickened artery with its pressure of 160 to 170 mm. Hg. So long as the hypertonic wave can be prevented these cases do quite well. The special danger in this patient was the evident tendency to brain angiospasm as part of a general hypertonic wave, and that prolonged spasm might directly or indirectly lead to permanent destruction of brain areas.

B.—High Pressures without Albumin.

The next series of cases illustrate the existence of sclerosed arteries with high sphygmometer readings, but no albumin in the urine.

Case 8.—Mr. P., aged fifty-one, was suffering from mental disturbance characterized by temporary loss of

memory, then giddiness, and a "swimming sensation" on movement, and then insomnia and great restlessness—all pointing to serious disturbances in brain circulation. The radial artery was thick and hard, and the brachial pressure varied between 220 and 250 mm. Hg. The heart was 1½ inches beyond the mammary line. The weight was 16 stone. There was no albumin in the urine. The history was that he had been, to his doctor's knowledge, a huge eater for twenty years, and had been warned many times about his habits in that direction. His diet had been curtailed and he had been kept at rest. He had been treated with nitrites, and iodides had been given up to 40 grains a day, but in spite of all these measures the pressure was never below 210 to 220 mm. Hg.

Remarks.—In this patient the arterial sclerosis was the result of the big feeding, extending probably over all his life. The condition of the arteries could not be altered, but the treatment had almost certainly prevented or lessened the risk of a great hypertonic wave involving the cerebral circulation so pronouncedly that disaster would almost certainly have ensued. I could do nothing but endorse the medical attendant's opinion of the gravity of the condition, and enforce the necessity of adhering rigidly to the advice and treatment already given.

Case 9.—Mr. M. W., aged fifty-two, was brought to see me by his medical attendant in April, 1908. The history was that three years ago he had disturbances of sensation in the right arm and leg. At that time he went for treatment to Harrogate and to Aix. Two months before I saw him he had again numbness in the right side extending over the whole half of the body. Before that the sight of the left eye had become affected. The arteries were thick and the pulse-wave poor. The

brachial pressure was 230 mm. Hg. The urine contained neither albumin nor sugar. There were recent retinal hæmorrhages.

Remarks.—This patient's history was that he was a colossal feeder and a big beer-drinker. His weight varied between 15 and 17 stone. His doctor, who was a keen observer, and much interested in the sphygmometer, was supported by my strongly expressed opinion that the condition required somewhat drastic changes in his mode of life, particularly with regard to feeding and beer-drinking. The treatment was carried out with considerable vigour, with benefit, although ultimately it had to be considerably relaxed. The doctor found that the medicine which suited best was spirit of nitrous ether. He lived for two or three years, and was able to get about freely and to act as his own chauffeur.

Case 10.—Mr. H., aged eighty-two, was seen in consultation in June, 1914. The history was that after dinner some days previously he was seized with difficulty in speaking. When I saw him the difficulty was much less, but still present in some degree, and mentality was definitely impaired in the direction of slowness. The arteries were thick and irregular, and the brachial pressure was 190 mm. Hg. There was no albumin in the urine. I saw him several times in December of the same year. Speech was completely restored and mentality much improved. He died five years later from the results of enlarged prostate.

Remarks.—This is a typical case of brain angiospasm in a man advanced in years, with sclerosed arteries and no albumin in the urine, who had led a strenuous life and was neither a big feeder nor a wine-drinker, save in very moderate quantity. The association of arterio-sclerosis

with enlarged prostate, I am informed by my friend Dr. John MacDougall, formerly of Cannes and Carlisle, is usual; that is to say, that enlarged prostate occurs only or mainly in persons with marked arterio-sclerosis. It was this complication which proved fatal, and had it not been for this the patient might have lived for years by continuing to follow the simple and frugal method of living which he had no difficulty in adopting under medical advice.

CASE 11.—Mr. F., aged fifty-five, was seen in July, 1912, when his symptoms indicated attacks of angina pectoris, coming on when walking, and sufficiently severe to make him stand still until the pain passed off. The arteries were hard and the brachial pressure 180 mm. Hg. The heart apex was 1½ inches beyond its normal limit. There was no albumin in the urine. The bowels moved easily daily, and had always done so. In October he had an attack of angina in my room, characterized by severe precordial pain, flushing of the face, the pulse irregular and rapid, beating 100 per minute, and the brachial pressure 220 mm. Hg. In January, 1913, he had two epileptic fits, in one of which the tongue was bitten. After this he was kept in bed for some time, his diet was strictly regulated, all alcoholic liquors and tobacco were stopped, and iodide of potassium was continued in larger doses. He took a long holiday, and was able to resume work. He kept fairly well, but had to avoid all physical effort, although he continued to walk a little very slowly. He reported himself from time to time, and was able to continue his office work right through the period of the war, but required great watchfulness. Early in 1919 he took a sharp but short attack of influenza, during which he nearly died, but began to convalesce, when he

was suddenly seized by an attack of angina pectoris and died in a few minutes.

Case 12.—Mr. H., aged fifty-eight, was seen in August, 1916, when he was suffering from breathlessness on slight exertion and giddiness. The tongue was dry and furred; the bowels constipated, sometimes not moving for two or three days. The brachial pressure was 210 mm. Hg. The apex of the heart was 2½ inches beyond its normal position; the aortic second sound was much accentuated. There was no albumin in the urine. The history was that the patient had led a very strenuous and successful business life, entailing much expenditure of nervous energy, and that he had always been a big red-flesh eater. The treatment was rest, daily attention to the bowels, the reduction of red flesh to a minimum, and iodide of potassium and bicarbonate of soda to be taken thrice daily. He was seen again in the end of May, 1917, when he was suffering from giddiness, headache, and discomfort and pain over the precordia. He could only lie on the right side, and night urination was frequent and very troublesome. On June 5 he went into a nursing home. There he was kept in bed, the bowels regulated by means of a daily laxative, and a radiant-heat bath given every second day for some time. He improved greatly in every respect, and with the improvement the pressure fell to 180 mm. Hg, and under ½-grain doses of erythrol fell to 150 mm. without causing discomfort, but this remedy was not continued. He left the home very much improved, all his previous symptoms being relieved or removed. He returned to the home for a short time on one or two occasions, and was always better for it, but it was difficult for him to give up his business and feeding habits. After a long interval I saw him in the

spring of 1920, after he had come through a time of much mental stress. He was in a highly nervous state, with furred tongue and the bowels kept acting by means of a saline. The pressure swung between 200 and 220 mm. He had been several times at one of the English spas with little benefit, and he had resumed his former habits as regards food. He again went into a nursing home, and had his diet reduced in quantity and containing little red flesh. He was kept in bed, and the bowels were regulated by means of phenolphthalein, which acted admirably in doses of 2 grains or even less. He was given iodide of potassium and had a radiant-heat bath every second or third day. During the fortnight in the home he improved greatly; the nervousness was quieted, he slept well, and was feeling much better. The brachial pressure moved between 190 and 220 mm. He then spent a month at a hydropathic, which was of no benefit, but rather the reverse. Red meat was excluded from the diet, and the bowels were regulated. The pressure under this treatment was reduced to 180 mm., and unpleasant symptoms again disappeared.

Remarks.—This patient illustrates the result of a highly strenuous life in a *very live man*, eating largely of red flesh and suffering from constipation. There was no question of alcoholism or of specific disease. The arterial sclerosis was clearly the result of the factors specified, and of these the big red-flesh feeding was the most important. How much can yet be accomplished in this particular patient it is difficult to say, but there is ground for hope that if he can be definitely weaned from business and from big flesh feeding his life may be prolonged for years.

C.—Early Arterio-Sclerosis.

A good deal of talk is heard in the profession from time to time of the early recognition of diseased processes and the means by which this knowledge is to be reached. The axiom that "a man is as old as his arteries" is one of the axiomatic *half*-truths which unfortunately obstruct the light which is the *whole* truth. It is also axiomatic that a "man may be older than his arteries." Arterial sclerosis is common in the later decades, but it is no necessary part of advancing years. Until that fact is recognized the beginning of change is not likely to be recognized. The examples of thickened arteries without albumin in the urine given in the preceding section occurred mainly in men between fifty and sixty years of age; but men are met with twenty or thirty years older with arteries which are perfectly soft, and who have a brachial pressure only 10 or 20 mm. above 100. I know a man over eighty years who has led a very strenuous life, and yet whose vessels are as soft as they ever were, and he has come through several very severe illnesses. Such instances are merely illustrations of many. The thickening of vessels in otherwise healthy men is a very slow process, and is long in developing. And it is only when we understand the influences which begin the arterial change that true prevention becomes possible, and in this case wider understanding on the side of the profession has to be supplemented by more intelligent knowledge on the part of the public. This matter will be dealt with more fully later.

The three following cases are given to illustrate the earlier stages of arterial sclerosis:

Case 13.—Dr. O., aged thirty-nine, consulted me about

symptoms indicating arterial spasm in one leg. He had been unfortunate enough to get a finger poisoned by a syphilitic patient three years before I saw him. He had been treated in accordance with the best knowledge of the time. The arteries were slightly thickened. The brachial pressure was 150 mm. Hg, while the pressure with the bag on the forearm was only 130 mm. The apex of the heart was 1 inch beyond its normal position. As regards dietetic habits, he was a big protein feeder, taking meat three times a day. He improved under iodide of potassium, and lost the symptoms of angiospasm in the leg. The brachial pressure only came down to 140 mm., and the forearm to 125 mm. He went abroad soon after that time.

The case illustrates the combination of syphilitic infection and big flesh-eating in the earlier period of life.

Case 14.—Mr. E., aged fifty-eight, consulted me in February, 1911. He owned and farmed a small property. He was unmarried. He complained of "not being able for much," and could not get "stronger," although he had a good appetite and had taken tonics of various kinds. He felt languid and heavy. The tongue was coated posteriorly, and the bowels were always constipated. The brachial pressure was 155 mm. Hg; the heart was normal in size. The urine contained neither sugar nor albumin, but gave a wide and deep purple ring with nitric acid. He was a free feeder and fond of flesh food. He usually took whisky at least once a day, at night. I very strongly impressed upon him the necessity of giving up spirits entirely, and of greatly reducing the quantity of food. The regulation of the bowels was also attended to. Under this treatment he rapidly improved, and the brachial pressure fell to 120 mm. I again dwelt on the necessity

of continuing the line of treatment which had been so beneficial.

1914. He was not seen again until 1914, when he returned with the same type of symptom, and on inquiry it was ascertained that his former habits as to food had been resumed. The pressure was 150 mm. He again improved, and during 1915 and 1916 continued reasonably well, with a pressure of 130 mm.

1917. In 1917 there had been another relapse, and the pressure had risen to 150 mm.

1918. During the few days in 1918 when I had the opportunity of seeing him the pressure had risen, and kept between 165 and 175. I again spoke very seriously to him, but he wanted to go for a change to the seaside, and passed out of my care. The next I heard of him, early in 1919, was that he had a cerebral hæmorrhage which was promptly fatal.

Remarks.—So far as I could ascertain, the amount of whisky taken was moderate; there was never any suspicion of excess in the ordinary sense. He was, however, a big flesh feeder, took little exercise, and was habitually constipated. That his feeding habits as life advanced led to increasing hypertonus of his vessels is undoubted; that this was capable of being controlled, when first I saw him, was equally certain; but I had no continuous control or influence over him, as I only saw him when it pleased him to come to see me when other business brought him to the city.

Case 15.—A lady, aged about fifty, had been under sanatorium treatment and feeding for two years, and had completely overcome a small tuberculous invasion of one apex. She had gained much in weight, and her brachial pressure was 160 mm. Hg, due presumably to the

thoroughness with which feeding regulations had been carried out. I saw her within a fortnight again, after she had come through a week of great anxiety and severe mental strain, which had led to insomnia. I again took the pressure, and found it 220 mm. Hg. A week later, when the anxiety had passed and the strain subsided, it had fallen to 180 mm., and later fell still further.

Remarks.—The points of special interest here were, *first,* the effect of a liberal dietary in raising sphygmometer reading; *second,* the very striking effect of a week of emotional perturbation with insomnia; and *third,* the fall of brachial pressure when the emotional strain was relieved.

CHAPTER IV

THE CLINICAL VALUE OF HÆMOMANOMETER OBSERVATIONS*

GENTLEMEN,—I would remind you that arteries have two sets of movements. The first of these is *pulsatile movement.* This movement is the result of the volume of blood shot into the aorta by the left ventricle and of the elasticity of the arterial wall. The second is, however, the more important movement—it includes both *contraction and relaxation.* This movement the arteries have in virtue of their muscular coat. By means of that coat they can contract so much as greatly to diminish their lumen—almost, if not quite, to close it. That being so, the first question we ask is, How is this movement determined? And the first answer to that is, Through the vaso-motor nervous mechanism. This is shown by the effect that emotion, for instance, has upon the circulation. But it is also indicated by the contraction of the arteries in one area when the vessels in another area are dilated. When, for instance, the vessels in the splanchnic area dilate and contain much blood, the systemic arteries contract, and this, I presume, is determined through the medium of the vaso-motor nervous mechanism. It is probable, I think, that the arteries always contract when the volume of blood in them diminishes, and that such contraction is brought about through the nervous mechanism. It is,

* A post-graduate lecture (*Lancet,* February 13, 1909).

I think, specially these regulative adaptations of the circulation that are presided over by the vaso-motor nervous mechanism. You all know the predominant place given to this mechanism; we have, indeed, been taught that the movements of arteries are altogether determined by it. You will look in vain for much definite reference to anything else.

I want, however, to draw your attention to another factor which I hold has been practically overlooked, and that is that *the arteries contract and relax in response to the composition of the blood*—in response to the direct influence on the vessel wall of various substances present in the blood. The pharmacological and physiological side of this is that various substances which we use in medicine—digitalis, ergot, adrenalin, and so forth—are known to have this direct action on the vessel wall when introduced into the circulation. That being so, I do not require to argue very strongly that other substances can have the same effect upon the arterial wall. If we find that under certain clinical conditions there is the same contraction of arteries as follows the giving of digitalis to a patient, we are entitled to assume that other substances than those the action of which has been determined experimentally have this particular effect. I cannot elaborate this question now, but merely state that there are certain conditions which are well known to us as physicians in which, I contend, this contraction of arteries is to be observed. Of these there is, in the first place, the retention of waste material in the circulation, substances which, instead of being excreted, are retained. These, I hold, irritate the vessel wall directly and lead to its contraction. Probably even an excess of nutritive material taken up from the intestine has this effect.

Another condition which assuredly leads to this state of the arteries is the absorption of putrefactive products from the intestinal tract. These substances, whatever their chemistry may be, and which are present in the blood, have the direct effect of stimulating the muscular tissue of the arterial wall to contract. There are special toxins which I only require to mention to convince you that substances present in the blood can have the effect indicated. Take syphilis and lead-poisoning; the changes which ultimately take place in the vessels are the result of the prolonged action of a toxin in the blood. Kidney disease, with its slow uræmia, produces the same anatomical alterations. In all there is the muscular stimulation to begin with, which goes on to medial and intimal changes. Tobacco and alcohol in many persons have the temporary effect of causing arterial contraction, and alcohol may certainly lead to permanent anatomical changes.

The next important fact is that not only normal vessels contract and relax, but that thickened vessels retain the power of contracting under the stimulus of substances present in the blood. In sclerosis the muscular coat is hypertrophied, and the arteries are not the seat of a fibroid degeneration, as has been so widely accepted from Gull and Sutton's teaching. The hypertrophy does not abolish muscle action in the arteries any more than it does in the heart. The sclerosed artery (using the term in the restricted sense in which I have advocated) can therefore tighten up and it can relax.

The Estimation of Arterial Contraction.

The next question we ask is, How is this constriction of arteries to be judged of, and how is it to be estimated? In the examination of the pulse there are two important factors: the one is the pressure of the blood inside the vessel, the other is the thickness of the vessel wall. Now, the pressure of the blood inside the vessel, *plus* the compressibility of the vessel wall itself, is determined by the sphygmometer. The bag surrounds the upper arm, and is distended with air until the pulse-wave is arrested in the brachial artery—until there is no longer any pulse felt at the wrist.

Now, I do not propose, gentlemen, because it would take many hours probably, to give you all the proofs that I could give you that that statement is correct. I am afraid I must ask you to accept it from me that this instrument determines these two things—namely, the pressure of the blood and the resistance of the vessel wall, and in spite of all that has been written and all that has been said in recent years to the contrary I maintain this. I shall only remind you that we were all taught that the compressibility of the pulse depended in part, and often very largely, upon the compressibility of the vessel wall. That this should ever have been seriously questioned will soon be forgotten. The results obtained by an instrument like this must be greatly influenced by the changes in the arterial wall such as I have described.

It follows that when the vessel walls are thickened you can only judge of the internal pressure by the fingers. There is no other way of separating the blood-pressure from the thickness of the arterial wall. The limit of blood-pressure is known, so that whenever there is a read-

ing with the hæmomanometer above that limit it means that the vessel wall is thickened. Now, this thickening of the vessel wall may be due to two factors. It may, in the first place, be due to the anatomical changes which I have shown to you. It may be due, on the other hand, to a contraction—a mere muscle contraction or spasm of the muscular coat. I need not elaborate this point to you. You realize that if the muscle coat of an artery contracts it thickens the wall and diminishes the lumen. You also know that it requires greater pressure to obliterate an artery the lumen of which has a diameter of $\frac{1}{8}$ inch than of $\frac{1}{4}$ inch, even if the wall be of equal thickness in each. This fact is of great importance, because the normal artery can tighten up in the way I have indicated to you so as almost to obliterate its lumen and to allow little blood to pass through it But in many instances you have the two factors together —in many cases you have a vessel with permanent anatomical thickening *plus* the contraction or spasm of its media. No means in our hands can alter the permanently thickened arterial wall, but the element of spasm is quite within control. The importance of recognizing that the sclerosed artery is not atheromatous, and is not the seat of such fibrosis as the clinician has often thought to be the case, but of such changes as I have shown, is this, that the changes I have described permit of, and explain the hypertonic contraction of, thickened arteries.

The necessary outcome from the foregoing is that there is often a reading of the hæmomanometer which is quite within the limits of normal blood-pressure, and yet does not indicate normal blood-pressure, but indicates a blood-pressure much below normal inside a contracted artery. In other cases a reading is much above normal,

and yet the high reading is associated with a low internal pressure and a greatly thickened arterial wall.

Now, while all this is true, the sphygmometer is an instrument of really very great assistance when it is combined with the use of the finger, and combined with such a use of the finger as we have all acquired. You have to put out of your minds altogether the extraordinary contention that the finger is no longer of any use for feeling the pulse. This instrument is of great use when combined with reliance upon our fingers, for of course we can estimate the pressure of a flowing fluid inside a tube when a pulsatile wave is transmitted through the fluid, no matter what thickness the wall of the tube be. The error to which I have just referred has arisen partly in this way. The blood-pressurists always begin at the physiological starting-point—that is to say, no matter what power the heart is showing, the blood-pressure is assumed to be 100 to 120. This conception, when reduced to plain terms, means that a heart doing a half or a third or a fourth of its normal work—and you know that many a heart does only a half, a third, or a fourth of its normal work—yet maintains a normal blood-pressure. You appreciate how absurd such a proposition is to the clinical pathologist. I may give you a recent case to illustrate this. The case was that of a woman dying from malignant disease of the abdomen. She was constantly vomiting; for days she was unable to retain anything swallowed. The pulse was a mere flicker, and the heart was steadily and slowly giving out, yet her brachial pressure was 95 mm. Hg, and the normal is from 100 to 120. According to the people who hold that these readings give blood-pressure, this exhausted and feeble heart was maintaining a blood-pressure some 10 mm. Hg below normal. The clinical

pathological picture I form of such a condition is that the amount of blood in the arterial system has diminished—it has accumulated on the venous side—a very small amount being thrown into the aorta with each systole. As the quantity of blood in the arterial system diminishes the arteries contract—the brachial artery contracts—accommodating themselves to the diminished volume of blood. The thickening of the artery wall by such contraction is the factor which requires much the larger portion of the pressure of 95 mm. Hg necessary for the arrest of the pulse at the wrist. The foregoing is a mere outline to you of the propositions which I hold in connection with the investigation of the circulation by means of the hæmomanometer.

There are many types of cases in which the phenomena which I have indicated may be noted, and in which observations made by the finger *plus* hæmomanometer readings become of important value.

Neurasthenic Type.

The first type that I may mention—I can only indicate to you very briefly certain lines along which you can make observations for yourselves—is the neurasthenic type. I can best illustrate this by the case of a patient sent by a medical friend for advice and treatment.

The patient was a woman, forty years of age, a neurasthenic. She was somewhat sallow, poorly nourished, and complained of languor and feebleness, of weakness and difficulty in performing even her domestic duties. She had been treated by tonics of various kinds. Her diet was of the ordinary mixed description, and the intestinal evacuations were scanty and irregular. The pulse

was small and feeble, with a small tightened-up artery. The pressure was 115 mm. Hg. I regulated the diet and gave iodide of iron internally. Within a week she saw me again. She said she was quite well. The symptoms which had oppressed her were gone. She declared she had not been so well for years. The radial artery was a much bigger vessel, and there was a fairly large wave in it. She had put on some 5 pounds of weight within the week. The brachial pressure had fallen to 105 mm. Hg. The hypertonus had been overcome, and with that the pressure had fallen.

This case indicates to you the value of this instrument along with your finger. That little tightened-up radial artery was plain to me. Her pressure was normal, yet by still further lowering it she was relieved of all her symptoms. In a case such as this the condition is an autotoxic one, the toxin in the blood irritating the vessels and causing their contraction. It is a condition you often find associated with neurasthenia—people who have nervous systems that are very susceptible to the toxin present in the blood. Those people have nervous systems asthenic to certain deleterious influences. Many are particularly susceptible to toxins coming from the digestive tract. By preventing the toxic absorption or favouring the toxic elimination you often enable the nervous system to battle successfully against its own inherent weakness.

Asthmatic Type.

Another type is the asthmatic type. In many asthmatics you will find the arteries tightened up during the asthmatic seizure and relaxing with the cessation of the seizure. These changes in the radial artery can be easily

recognized by the finger, while the hæmomanometer shows that with the relaxation the pressure falls 30 to 40 mm. Hg. I regard this as a very important observation, for it suggests that in some asthmatics the paroxysms may arise, not from spasm of the bronchi, but from spasm of the pulmonary vessels corresponding to the spasm in the systemic vessels.

The Cardiac Type.

You have the same phenomena taking place in the arteries in patients with heart disease. In cases of heart weakness or of valvular lesion, if the systemic arteries tighten up you do not require me to argue that this throws a great strain upon the heart and leads to very serious embarrassment. As a matter of fact, one frequently finds in persons with old-standing heart lesions that when they begin to have heart symptoms they are due to this arterial constriction, and that you relieve them at once with a vaso-dilator, and you can prevent recurrence by eliminating from the blood the substances which irritate the vessel wall. The myocardial cases—people with fatty heart—are the same. You cannot more assuredly kill people with fatty heart than by suddenly tightening up the arterial system.

In angina pectoris the great majority of cases are cases in which during the paroxysm the whole arterial system gets tightened up. One of the last cases I saw had a pressure of 140 mm. Hg. I did not see the patient in an attack, but from the history given to me by her medical adviser I was satisfied that it was a case of angina pectoris. The medical attendant was fortunate enough to see the patient in an attack, and found that the pressure was 190. He promptly administered a vaso-dilator, with the

result that the pressure fell to 140 and she was relieved. The frequency of the attacks was rapidly lessened by appropriate treatment. Cases of this kind one could multiply, but it is unnecessary. All that I want to indicate is that you have these vascular phenomena in angina pectoris. I do not say that there is no attack of angina pectoris without this contraction of systemic vessels. It is almost certain, I think, that such vessel contraction may be confined to the coronary arteries, but such a local spasm would equally account for the anginous paroxysm.

Digestive Type.

This is one of the commonest types. The vessel constriction may be due to constipation or to some particular dietetic substance. The effects vary greatly in different individuals. In some the effects are marked, and the sustained or repeated tightening causes permanent structural change. On this digestive side let me indicate how undesirable it is to get faddy about these things. I know a gentleman who has cured his hypertonus by giving up drinking tea. It does not follow that either you or I require to do likewise, for it has not that effect upon me. There are people who, because they are poisoned by some special food or fluid, think that their poison must be poison to everyone. I say this because I do not want any one of you to suppose that I am a faddist. I am only a faddist to this extent—I am sure that there are many people who cannot do what you or I can do, and that if they want relief from their symptoms they must abandon certain things, whether it be tobacco, alcohol, or free protein feeding. There is no doubt there are marked idiosyncrasies in the matter of diet, and these

assert themselves the more as life advances. We meet it amongst navvies as well as amongst the higher classes. I am not sure that I did not see a case of this kind lately. A young man aged twenty-five years was brought to see me. His medical attendant was dissatisfied with him. His arteries appeared to be thickened, and nothing could be found to account for his condition. His pressure was 220 mm. Hg. I had not seen anything like this except in granular kidney. There was, and had been, no albumin; there was nothing in the eyes to show that the kidneys were affected, but his pressure was 220 and his vessels were somewhat thickened. I took a gloomy view of the case, but advised that he ought to be taken home, kept in bed, and nourished almost entirely on milk. I saw him four weeks later, and the pressure was down to 135, and since then it has fallen to normal. I have left it as a problem for his medical man to find out whether his patient has not an idiosyncrasy to all red-flesh foods. I can say nothing more definite upon this unusual case; but I am prepared for the most extraordinary idiosyncrasies. The practical point is that if you can influence the pressure by rest and diet you may depend upon it that the future of your patient is in your hands. The future of that patient lies with his medical attendant. Had the condition been wrongly interpreted it would assuredly have gone on to sclerosed vessels and granular kidney.

Renal Type.

Next there is the renal type. I need not say much to you about this type. I have shown you the changes in the arteries and in the kidneys. In this type the hæmomanometer reading often varies from 240 to 290 mm. Hg. The

difference is not a variation in blood-pressure, but indicates a variation in the arterial wall. When the thick vessels contract the hæmomanometer reading rises, and as they relax it falls. This record by the hæmomanometer of the change in the vessel wall is most important if you accept my contention that the vessels tighten up under the direct influences of substances present in the blood, a rise in pressure in such a case warning you that your patient is in dangerous proximity to an attack of uræmia.

Cerebral Type.

Lastly, there is the cerebral type. I do not know that there is any type more important than this type. It is most commonly seen in old or elderly people—people who no doubt very often have atheroma of the cerebral vessels. The cerebral type gives you many varieties of phenomena—excitement and delirium to apathy and semicoma; temporary to permanent paralysis. The hæmomanometer and the finger combined can help you greatly in these cases. In many of them the changes affect the systemic vessels as well as the cerebral vessels. In the brain, when these thickened or atheromatous vessels tighten up, their lumen is diminished and there is a diminution of the blood-supply. If you use the finger and the hæmomanometer you will often be able to regulate these cases, if you recognize that you have to think of vessel thickening as shown by the hæmomanometer, and of heart power as judged of by the finger on the artery. In many old people your finger tells you that there is a feebly-acting heart in spite of a high hæmomanometer reading, and that if you relax the arteries with the one hand you must encourage and help the heart with the other. You require a fairly strong heart to send the blood

on after the vessels are relaxed. You will often prevent cerebral softening by properly appreciating these relations, and often rapidly cure your hemiplegias. I have no hesitation in claiming that I have done that for people over and over again, and we can all do it.

The subject I have brought before you is one full of interest. I have merely given you an outline sketch of it, but one, I hope, which will arouse your interest sufficiently to lead you to look into it further and satisfy yourselves that these things are true; that this action of the blood upon the vessel wall is a truth which we have ignored—why, it is not my business to inquire; and that the muscle movement of the vessel wall has also been practically ignored. In these two observations lies the explanation of the production of arterio-sclerosis, confining that term to the medial and intimal changes which are the common anatomical changes in the thickened radial and other arteries so well known to the clinician. In conclusion, I have indicated to you that the hæmomanometer is of clinical value as giving you a record of the contraction and relaxation of the arterial wall rather than of the blood-pressure. This interpretation will, I believe, soon come to be recognized as much more practically and clinically useful than the other.

As an illustration of the cardiac type the following case is introduced here. It formed part of a clinical lecture on " Dyspnœa in Cardiac Disease."

Case 15A.—*Double Aortic Lesion with Great Dyspnœa due to Systemic Angiospasm.*—This patient, aged forty-one, a labourer at Rosyth, was admitted on November 25, 1914. He was admitted with marked dyspnœa, the respirations were about 34 per minute, and there was great distress referred to the precordia. He could not

lie down, and rested his arms and his head on the bed-table in front of him. The history he gave was that eight weeks before admission he got a bad cold with a bad attack of breathlessness, but no pain. He had several other attacks of breathlessness, the worst attack having occurred just before he was recommended for admission to the Royal Infirmary. We further learned that he had had rheumatic fever at the age of twenty and enteric fever at thirty. On examination the heart's apex was found to be in the sixth intercostal space, 1½ inches external to the mammary line; it was forcible in character, but regular. There was a marked systolic and diastolic aortic murmur, both murmurs being more or less audible over the whole precordia. In addition to constant distress and dyspnœa which prevented the patient lying down, he suffered from frequent exacerbations of these; the exacerbations not only came in waves, but left him with a feeling of great weakness and exhaustion. The pulse varied in frequency from 76 to 98 during his first days in hospital. The Resident found that he could control the more severe paroxysms by means of erythrol tetranitrate or whisky. This went on for some days, but notwithstanding the fact that the patient was kept as quiet as possible there was no appearance of any definite improvement in his condition. While his radial pulse was typical of aortic regurgitation, the artery gave me the impression of being hypertonic, and his brachial pressure stood at 170 mm. Hg. Judging from former experience in similar cases, I ordered iodide of potassium to be given in 10-grain doses three times a day. The effect of this drug was very striking; each dose improved his condition; within forty-eight hours the distress had disappeared, the patient was able to lie down, and the brachial pressure

had fallen to 137 mm. Hg. From that time improvement continued; there have been no relapses, and there have been no paroxysms of dyspnœa or of pain. The pulse has fallen to about 80 and the respirations to 26, the heart is steady and working satisfactorily, the patient sleeps well, and he is steadily regaining strength.

Remarks.—Such is a brief summary of this interesting case. In this patient the evidence of a double aortic lesion of considerable magnitude was unequivocal. The chill, of which he told us, had evidently been the determining cause of the symptoms of which he complained—namely, precordial pain and breathlessness. Up to that time there had been what might be called a working equilibrium in his circulation, the equilibrium being, however, suddenly upset by the entrance of this new factor. I ask you to note that the influence of the chill did not operate directly on the myocardium. The heart muscle showed considerable power, and there was no suggestion of failure or of undue dilatation. Yet the heart was disturbed and was evidently struggling. None of the causes which have been dealt with in the previous cases as determining dyspnœa were present here. The myocardium, although struggling against an obstacle, had not given way. When we pass to the determination of the nature of this obstacle the explanation was suggested by the state of the radial arteries—they gave the impression of being tightened up. At this point let me again explain to you what I have already explained oftener than once, that when the systemic arteries, in virtue of the property of their muscular coat, become constricted or hypertonic, no matter what the cause be, the blood-pressure is lowered in the arteries and raised in the aorta. This raising of the blood-pressure in the aorta requires

increased effort on the part of the myocardium of the left ventricle to open the aortic cusps, and this effort explains how systemic angiospasm or arterial hypertonus may be the determining cause of dyspnœa in valvular lesions. It need not be argued, for it is a matter of common clinical experience, that when strain is put upon the heart dyspnœa results, and that the duration and degree of dyspnœa are in proportion to the duration and the degree of strain. The opinion that arterial hypertonus was the factor operating in this case was confirmed by the effect of the treatment. The treatment was directed to counteracting the sustained arterial hypertonus, and the drugs which experience has shown us are most effective for this purpose are iodide of potassium and iodide of sodium. The result added further confirmation to our previous experience: the relief it gave was marked, and the effect was quickly produced. The change in the character of the pulse was readily appreciated by the finger, while the fall in sphygmometer reading showed that the vessel wall had responded to the influence of the iodide administered. The effect of the drug was to relax the hypertonus of the arterial system, and thereby to increase the capacity of the arterial channels. By doing this the undue pressure in the aorta was taken off and the effort required from the left ventricle to open the aortic cusps was lowered. The ventricle had now a weight to carry that was so reduced that it was carried without conscious effort. All this relief was effected very rapidly, and the heart showed, as we had anticipated, that its myocardium was quite equal to coping with the valvular defects so long as this extra factor of arterial hypertonus, with its heightened aortic pressure, was kept out of the scale against it.

CHAPTER V

INTERMITTENT CLOSING OF CEREBRAL ARTERIES: ITS RELATION TO TEMPORARY AND PERMANENT PARALYSIS*

GENTLEMEN,—I have been seeking a new word to express the kind of vessel action that I propose to speak to you about this afternoon, but I have failed in the search. Many of you must know the term "intermittent claudication," which has had a place in medical literature for about eighty years. It was introduced from veterinary medicine, where it was first used to describe recurring attacks of lameness or limping, with pain in the affected limbs, which occurred in a mare, and were due to obstruction to the arterial blood-supply when the muscles were put in vigorous action. In human medicine the term has always carried with it the conception of loss of power as a result of the closing of arterial channels. A record of mere limping in a horse would be of no more interest to the veterinarian than to us; it was the *cause* of the limping that was of interest; it was this that led to the introduction of the term into human medicine. It is this association of effect and cause that makes the term of interest to us. I have tried to manufacture a new word from "claudĕre," to close, like "claudication," which is derived from "claudēre," but have not succeeded, and so I use the simple word "closing."

* A post-graduate lecture (reprinted from the *British Medica Journal*, October 16, 1909).

I ask your attention to a corresponding loss of brain power due to intermittent closing of cerebral arteries, as it is seen in temporary paralysis, and as it takes a part in the production of permanent paralysis.

As illustrating the subject I give you briefly, in mere outline, the salient points in the case of a patient who was in my male ward in the Royal Infirmary from March 5 to 31.

Mr. M., aged fifty, a farmer, was admitted for observation. The history was that during the months of May, June, and July, 1908, he had three attacks, characterized by tingling in the right arm and hand, a stiff and numb feeling in the right side of the face, and difficulty in articulating. In August he had an attack of complete loss of power on the right side, and stumbling speech. During this attack the symptoms passed off and again returned. Within ten days he had three similar attacks, but not so severe as the first. In November and December he had slight attacks. In the end of December he noticed that when reading he could not see the beginning and the end of a long word. This eye condition led him to consult Dr. Wilson Black of Inverness, who kindly sent me a report on the condition of the eyes, and I show you the perimeter chart of the field of vision at that time. There was a right homonymous hemianopia, but not of the entire right half of the field, only of its outer half.

On admission, the patient weighed 13 stone, looked robust and well nourished. The pulse was 84, regular, and of moderate force; brachial pressure was 210; the vessel wall was thick, but not tortuous. The heart dullness extended to the nipple line. The amount of urine passed in twenty-four hours measured from 50 to 60 ounces; the specific gravity was 1020, and it contained a trace of

albumin, but no casts were found. The blood was rich in quality. There was no trace of paresis anywhere, no alteration or loss of the ordinary or of any of the special senses. The reflexes were all normal. Speech was clear. The field of vision in both eyes was normal. Dr. Arthur Sinclair, who examined the eyes, found in the left fundus an area of slight local œdema.

In this patient the symptoms may be summarized thus: Extending over a period of three months there were recurring *premonitory* symptoms; in one month there were four attacks of hemiplegia or of hemiparesis, during which time he was medically treated; slight recurrences of premonitory symptoms; and, finally, a temporary and partial right hemianopia. From all these he had recovered by the time he was admitted to hospital, and during his sojourn there there was no return of the symptoms. He had slight digestive disturbance when admitted, which was readily rectified. The arterial pressure varied between 188 and 234. An exclusively milk diet for some time, the administration of iodide of potassium in 10-grain doses thrice daily, and free action of the bowels, made no permanent impression on the hæmomanometer readings. On leaving hospital the diet was regulated, and when I last heard of him there had been no return of the special symptoms.

Causes of Sudden Hemiplegia.

Before discussing this case, let us look at the causes of sudden hemiplegia or hemiparesis. They are hæmorrhage, embolism, thrombosis, and, I add, temporary closing of cerebral arteries. The effects of these are, in the case of hæmorrhage, laceration of brain tissue; in the cases of embolism and thrombosis, necrosis and soften-

ing of brain tissue. In both laceration and softening there is no restoration of destroyed brain tissue. In temporary closing of arteries there is impairment of function in the part of the brain affected—a halting of brain function from closing of the channels which convey the blood, a condition comparable to intermittent claudication.

Differential Diagnosis.

Embolism.

Turning to the question of differential diagnosis, a diagnosis of embolism requires at least the coexistence of mitral or aortic disease, or disease of the aorta itself, in addition to sudden paralysis. Putting this aside, there are the other two causes, hæmorrhage and softening; the latter as resulting from thrombosis, and much more common than embolism.

Hæmorrhage and Thrombosis.

With regard to thrombosis, thrombus, once formed, does not break up and disappear in some mysterious way; it is a difficult thing to get rid of, and we cannot appeal to recurring formation and disappearance of thrombus to explain our clinical phenomena. That is not pathology. With regard to hæmorrhage, there seems to me to have been a marked tendency in later years to regard all cases of sudden hemiplegia as caused by cerebral hæmorrhage. However this teaching may have arisen, I do not think it is correct. Tooth agrees with Gowers in thinking that thrombosis is the commonest cause of hemiplegia, and that is my experience. The outstanding symptom which differentiates the two conditions is loss of consciousness. Trousseau, following Récamier, pointed this out long ago.

He said that "complete loss of motor power, without accompanying coma, belongs more especially to softening"; and he repeated it thus: "Whenever hemiplegia complete and absolute occurs suddenly, without loss of consciousness, softening of the brain may be diagnosed." Tooth accepts Trousseau's position as the "broad rule" to which there are many exceptions. Liddell says that two-thirds or so of all cases of hemiplegia due to hæmorrhage show loss of consciousness. If these estimates are even approximately correct, about 1 in 5 cases of hemiplegia without loss of consciousness is due to hæmorrhage. This symptom is therefore of predominant importance in the differential diagnosis of these two conditions.

Temporary and Intermittent closing of Cerebral Arteries.

In this condition the closing of the blood-channels is initiated by the channels themselves, and is effected by means of their muscular coat. It is a local closing, and may be partial or total—that is to say, it may only lessen the amount of blood passing, or it may completely arrest the flow of blood to the part. As a result, there is either impairment or complete suspension of function until the vessel opens and permits the blood to resume its course.

That such local closing of arterial channels is possible is seen in the local syncope of "dead fingers," in the "local syncope" of Raynaud's disease, in some cases of migraine, in the vessels of the retina in quinine blindness, and in temporary partial blindness seen to be due to closing of a branch of the retinal artery. A corresponding explanation is required for the phenomena which were exhibited in

Mr. M.'s case. His case is only one of many I could give you. They are known to all physicians and practitioners of experience, and, were it necessary, I could gather striking examples of such recurring phenomena from medical literature. The complete recovery which occurred in Mr. M.'s case could not have occurred had there been either hæmorrhage or softening in the motor regions affected, or in the part of the optic radiation which determined the hemianopia.

A Working Hypothesis.

The explanation, moreover, provides a working hypothesis of great practical value, and this is the reason of my bringing the subject before you to-day. Take the phenomena in Mr. M.'s case. Extending over three months there were symptoms commonly regarded as premonitory, or as the threatenings of cerebral hæmorrhage. But what is meant by *threatening* of cerebral hæmorrhage? It is either hæmorrhage or not; there is no half-way stage in hæmorrhage, although the quantity varies greatly. But these threatenings also precede thrombosis and softening; they also precede the paralysis due merely to temporary arterial closing, as in Mr. M. The "paralytic stroke," due to any of three different pathological processes, has the same premonitory threatenings, and it is of no small practical importance that there should be a clear picture of the clinical pathology of this stage. These threatenings can only, I take it, be due to irregular vessel action, and so to irregular blood-flow through the part. There must be vessel constriction, local in site, varying in degree and in extent, coming and going, intermittent.

Effects of Intermittent Closing.

The effects of intermittent closing may be placed in four groups:

1. It leads to impairment or suspension of the function of the part affected, as manifested by sensory disturbances and motor faltering or paralysis.

2. It favours thrombosis in certain states of the vessel wall, or of the blood itself, or of both combined.

3. If the heart be weak the blood-flow is more easily arrested, thrombosis is favoured, and the risk of softening is greater.

4. If the heart be strong and the vessels not sound, hæmorrhage is liable to occur. The hæmorrhage, however, does not take place in the closed portion of vessel; it must take place on the proximal side, where the pressure is raised because of the distal closing.

The significance of the premonitory warnings and threatenings is thus seen, and the aim of practical therapeutics is made clearer.

Etiology and Treatment.

We have thus a picture of intermittent closing of arteries occurring in different parts of the brain, occurring in persons of very different types, and occurring, as in our case, at the early age of fifty years. If you ask about the etiology of this irregular vessel action, we are at once brought face to face with fundamental problems. There are questions of constitution such as are embraced in the words " gouty " and " rheumatic "; there are questions in metabolism, such as the accumulation of the waste products of cell activity, the fatigue toxins, and the inefficient

removal of such. There are questions of food and drink; there is the condition of the bowels, and the absorption of toxins from the intestinal tract. There are questions of skin and kidney action. There are questions of mental work and of mental repose; there is emotion and passion. In no department of our work is it so necessary to form a sound all-round estimate of the patient; habits, digestion, bowel action, kidney efficiency, the cardio-vascular system, have all to be reviewed and estimated aright if we are to be guided aright. What is to be aimed at is the lowering or removal of the abnormal vessel irritability. In the robust plethoric subject the threatenings of which we have spoken have long been treated by active purgation, diminished food-supply, physical and perhaps mental rest. All these measures are good, but the plethoric type does not supply the greater part of the cases we are now considering. In many cases active purgation and all extreme measures are to be avoided as positively hurtful. Rest, physical and mental, gentle bowel action, spare diet with no red flesh, are the general lines of treatment. The irritable tonicity of the vessel wall is in many cases wonderfully controlled by spiritus ætheris nitrosi. When symptoms appear to be more urgent, I have much confidence in erythrol tetranitrate in $\frac{1}{4}$ to $\frac{1}{2}$ grain doses, repeated every three, four, or six hours, as may be required. The action is temporary, but it allows time for the bowels to act fully and for the elimination of the excess of waste products in the tissues. Thereafter restricted diet and daily bowel action may prevent the recurrence of symptoms. In other cases the vessel irritability is markedly allayed by the administration of iodide of potassium. I would very strongly warn you against violent purgation with calomel and similar drugs

in persons with a feeble circulation, for the lowering of such a circulation is the surest way to ensure softening and even subsequent hæmorrhage. The feeble circulation has to be recognized behind thickened and incompressible vessels; see to it that you are not deluded into thinking that there is a strong heart because there is a high hæmomanometer reading. A tonic, as nux vomica, may be a valuable addition to spirit of nitrous ether or to iodide of potassium in cases of this kind.

By a true estimate of the patient as a whole, by the wise use of the simple methods indicated, the irritability of the arterial wall disappears and can be prevented returning to any alarming extent. In this way we can by foresight prevent such truly pathetic cases as must be known to most of you.

CHAPTER VI

RECURRING ARTERIAL HYPERTONUS IN GRANULAR KIDNEY AND IN MIGRAINE*

GENTLEMEN,—There are two patients in the adjoining ward whom you will have the opportunity of seeing and examining for yourselves after I have given you an account of the clinical phenomena which have been noted in them during the time they have been in the ward.

Granular Kidney.

CASE 16.—The patient is fifty-two years of age. She has had a checkered life, has been in this infirmary and in the workhouse hospital on more than one occasion for various ailments, and she frankly acknowledges to having been "fond of a good big dram." The details of such a life do not require to be dwelt upon; the results of it at fifty-two years of age are illustrative of a large group of cases, not all, however, presenting a personal and social history of hard work, poverty, ill-treatment, and bad habits. The patient was admitted on June 10, 1910. She stated that for some weeks she had been breathless, especially on going upstairs, while latterly it was continuous; she suffered from attacks of dizziness; her legs at times became weak and shaky, and she had difficulty with her speech at times—she stammered and could not

* A post-graduate lecture (*Lancet*, December 3, 1910).

get the words out. She sought advice at the Cowgate Dispensary, was recommended to the infirmary, and was admitted under my care.

On admission the pulse was 80, the respirations were 24, the temperature was 97° F., and the brachial pressure was 295 mm. Hg. She was kept in bed, put on milk diet, and was given a dose of Henry's solution at night. The radial artery was thickened, but did not give the impression of anything like the pressure given by the brachial. The heart dullness at the apex was in the nipple line; the sounds were clear and slapping, and there was no murmur. The lungs were emphysematous, but otherwise normal. The urine had a specific gravity of 1017, and showed a faint trace of albumin. The blood-count and the amount of hæmoglobin were normal. The bowels moved once or twice daily. By the 14th the pressure had fallen to 185 mm. Hg. She was then given 5 grains of potassium iodide three times a day. On the 18th the pressure had fallen to 165 mm., and she was allowed light diet, which includes chicken and fish. From that date to July 2 the pressure varied from 165 to 210 mm., the level being somewhat higher after the increase in her diet than before that. On the forenoon of July 4 she was not so well, and when I saw her, her face was flushed, she was propped up in bed with pillows, she looked dazed, and when spoken to could not articulate; the pulse was rather over 100, the respirations were 24 and laboured, and the brachial pressure had risen to 268 mm. She was given erythrol at once, and at night had a dose of mannitol. She rapidly regained her ordinary condition, had a good night, and next morning the pressure had fallen to 190 mm., and she felt in her usual health. On August 12, with her pressure only at 202 mm., she complained of headache, a tingling sensation in the right

limbs, right side of the trunk, and in the corresponding side of the tongue. These sensations continued, and she was given erythrol. On the 14th the pressure was 186 mm. On the 30th the patient was noted to be particularly well in the morning, but at about 6 p.m. she became restless; at 7.30 she complained of violent pain in the head, was breathless, and had difficulty in articulation; at 8.15 the pressure was 258 mm., and she was given erythrol; and at 9.30 the pressure had fallen to 207 mm., and as she was drowsy she was not disturbed again, and she soon fell asleep. The following morning the pressure had fallen to 175 mm., and since then she has been in her usual condition, the pressure keeping about 200 mm.

The course of the pressure is seen in the sphygmometer chart, Fig. 8.

This patient is suffering from the effects of granular kidney and extensive arterial change, and I bring her case before you to illustrate the clinical lessons such a case supplies, and to impress upon you some points which it is of the first importance for our patients that we should understand the true significance of and be able to treat intelligently, and then successfully up to a certain point.

The patient was kept in hospital until December, when she was sent to the workhouse hospital. She was able to be out of bed and assist in the ward work. The pressure was steady at about 180 mm.

You will note that this woman on admission had a vessel pressure of 295 mm. Hg, one of the highest pressures I have had in this ward, and that rest in bed, the administration of a saline purge, milk diet, and later 5 grains of potassium iodide thrice daily, brought the pressure down 100 to 130 mm. During the period she has been in hospital she has sometimes shown symptoms which

are classed as uræmic, such as restlessness, incoherence, dyspnœa; at other times symptoms that are regarded as premonitory of cerebral hæmorrhage or cerebral softening

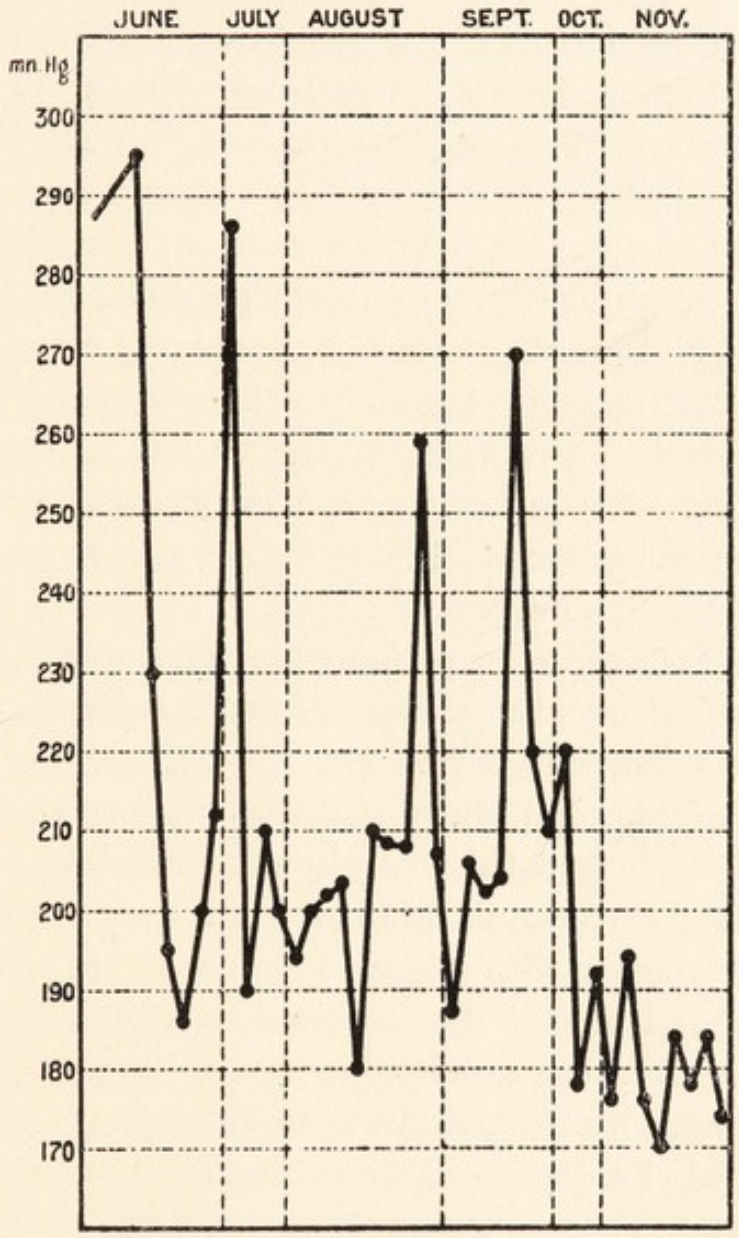

FIG. 8.

from local anæmia or thrombosis, such as tingling in one half of the body, confusion of thought, difficulty in articulation, even loss of articulate speech. She has had several minor manifestations in one or other of those

directions; but she has had two or three graver attacks, in the first of which I was fortunate enough to see her and at once to recognize its nature. In all these attacks I ask your attention to the fact that the brachial pressure showed a great rise—a rise which would doubtless have risen still higher had it not been arrested by therapeutic interposition. With such interposition, however, the pressure fell, as on July 4, from 268 to 190, and on August 30 from 258 to 175 mm. With the fall of pressure all the urgently alarming symptoms disappeared; in fact, the arrest of its upward movement, and a fall even of 15 or 20 mm., brought visible relief to the symptoms, and this was effected in a remarkably short space of time. The association of a marked rise in hæmomanometer reading with such symptoms as I have indicated is in my experience always to be noted.

Neither the ordinary pressure nor the rise in pressure in these cases can always be estimated correctly by the finger on the radial artery. None of you would guess from this patient's radial artery that the pressure would be as high as it is; indeed, those of you who study blood-pressure by the finger will think that it is even lower than normal. I entirely agree with those of you who may think so. The finger, I hold, is the only means by which you can estimate true blood-pressure. True blood-pressure to the clinician means the power of the left ventricle, and nothing else, as that can be estimated by the finger on an artery. The hæmomanometer reading, on the other hand, is only in part the result of blood-pressure. The patient we are considering has a variation in her hæmomanometer reading from 295 to 165—a margin of 130 mm. —and certain eminent persons would ask us to believe that this is the margin of power in this woman's left ventricle,

a margin which no healthy man in this room possesses. But I do not propose to argue this point: I have done that in other places. I merely state that this great margin of pressure, that these great variations in pressure, indicate to us the condition of the arterial wall—the degree of hypertonic contraction or of relaxation of it. The armlet being applied to the upper arm gives us the condition of the brachial artery, which every now and again differs greatly from the bit of radial artery to be felt at the wrist. This observation as to the state of the brachial artery, added to your examination of the radial artery and pulse, you will find a great help in estimating correctly the state of the circulation in your patients.

Let me repeat that in this patient we had, and will have again, symptoms of uræmia, and symptoms which presage cerebral softening or hæmorrhage; that when these threatenings are present the hæmomanometer reading is found to have risen many millimetres. My contention is that the raised reading is due to hypertonic contraction of the artery from which the reading is taken. The symptoms are associated definitely with this phenomenon, for the moment you arrest the constrictive action the symptoms begin to disappear, and altogether disappear with the cessation of the hypertonus and the consequent fall in the hæmomanometer reading.

To you as graduates in medicine I need not labour to prove the great value, therefore, of this instrument if you use it aright and interpret its readings correctly. I shall only ask you to realize what the phenomena are which are commonly recognized as presaging paralysis, hemiplegia being the commonest, associated or not with aphasia. Very many of these cases are preceded for days or for longer periods by recurring sensations of numbness

or tingling, by temporary slight loss of power in a hand, an arm, a leg, difficulty in articulation, temporary loss of speech, feeling of mental confusion, temporary loss of memory, and so on. All these you will find associated with arterial hypertonus and a high hæmomanometer reading. The nervous phenomena can only be explained by the participation of the vessels in varying parts of the brain in a constriction in which the whole systemic circulation is affected. These local angiospasms in the brain lead to anæmia of the areas involved, the clinical phenomena being referable to the areas so affected. The angiospasm in brain areas favours thrombosis, and its consequent deplorable brain softening, or leads to hæmorrhage on the heart side of the region of spasm.

Now, I unhesitatingly assert that if these phenomena are duly observed, correctly correlated and interpreted, you have it in your power in many instances to save your patients from disaster, preventing their being deprived of the power of locomotion, of speech, of mental power, perhaps for years longer than would otherwise be the case.

The patient who is the text of these remarks is but one of many similar cases I could describe to you. I am glad that a case of the kind happens to be in the ward at present, so that you may see the case and note the difference between the radial pulse and the high pressure required to obliterate the brachial artery, and realize that this simple instrument, the hæmomanometer, is really a great help in your everyday medical work if you use it as a guide to the state of the vessel wall more than as a guide to blood-pressure, although in other classes of cases it can even help you in judging of that.

Migraine.

CASE 17.—The second case to which I ask your attention is also of interest, although not of such urgent importance as the preceding one. The patient, a woman aged forty-nine years, was sent to me in May, 1909, by Dr. W. C. McEwan of Prestonpans, as she was suffering from migraine, having an attack almost every week, the attack lasting three or four days. At that time the radial artery was small and wiry, and the brachial pressure 150 mm. Hg. The bowels were constipated, the tongue coated, the breath offensive, and there was a trace of albumin in the urine, which also showed much indican. I advised that she should have a laxative pill nightly of cascara, belladonna, and nux vomica, and iodide of potassium thrice daily. Under this treatment she improved, but as the attacks returned she was again sent to see me, and was admitted into the adjoining ward on August 12, 1910. On admission the pressure was 205 mm. She was treated with enemata to unload the bowel, and got a colocynth and hyoscyamus pill to keep the bowels moving. Under this treatment and rest in bed the pressure fell and kept between 164 and 172 mm. On August 21 the pressure was 164 mm. On the 23rd, in the evening, she complained of headache, vomited twice, and she knew that one of her attacks had begun. She vomited several times during the night. On the 24th her condition was unchanged; she vomited twice, and the pressure was 192 mm. On the 25th her condition still remained the same, and the pressure was 194 mm., when she was given ¼ grain of erythrol, the hour being 12.25 p.m.; by 12.55 the

pressure had fallen to 142 mm.; by 1.50 it had risen to 168 mm., and by 7.45 to 179 mm. On this date (the 25th) she vomited six times between 1.30 p.m. and 9 o'clock, and as her symptoms were not at all relieved by the fall of pressure produced by the erythrol, the drug was not repeated. On the 26th the headache was much less and the vomiting had stopped. The attack had lasted part of three days. On the 27th there was neither headache nor sickness, and the pressure had fallen to 138. On September 1 the pressure was reported as rising steadily, and had reached 172. The patient thought she was to have another attack, as she had a feeling of fullness in the right arm, a symptom she had noticed previously as indicating the approach of an attack. On the 2nd, in the morning, headache began, and at 10.30 a.m., when I saw her, the pressure was 203. I ordered 10 grains of phenacetin every hour for three times. At 1 p.m., the pressure being still 203 mm., she was given the first dose; at 1.45 the pressure was 187 mm. and the headache was slightly relieved; at 2.30 she was given the second dose, and at 2.45 the pressure was 184 mm. and the headache continued relieved; at 3.30 she was given the third dose, and at 4 o'clock the headache was distinctly better and the pressure was 178 mm.; at 7 p.m. the headache was much better and the pressure was 178 mm. There was no vomiting, and the patient had a good night, sleeping soundly. The following morning, September 3, she expressed herself as feeling quite well, and the pressure had fallen to 158 mm. She has remained well down to the present.

The course of the hæmomanometer readings may be tabulated thus:

	May, 1909	..	150 mm. Hg.		
	August 12, 1910	..	205	,,	
	,, 13-20, ,,	..	198-165	,,	
	,, 21, ,,	..	164	,,	
	,, 24, ,,	..	192	,,	
Attack	,, 25, ,,	..	194	,, ..	12.25 p.m., erythrol.
			142	,, ..	12.55 ,,
			168	,, ..	1.50 ,,
			179	,, ..	7.45 ,,
	,, 27, ,,	..	138	,,	
	Sept. 1, ,,	..	172	,,	
Attack	,, 2, ,,	..	203	,, ..	10.30 a.m.
			203	,, ..	1.0 p.m., phenacetin.
			187	,, ..	1.45 ,,
			184	,, ..	2.30 ,, phenacetin.
			184	,, ..	2.45 ,,
				,, ..	3.30 ,, phenacetin.
			178	,, ..	4.0 ,,
	,, 3, ,,	..	158	,, ..	morning.

August 24 and September 1 are the dates of two attacks, the first treated with erythrol and the second with phenacetin.

This case raises various questions which cannot at present be discussed. Some of these questions are: Are the attacks primarily of nervous origin, and, if so, are they neurasthenic or epileptoid? Are they nervous manifestations of early cirrhotic kidney, or are they the result of a primary auto-intoxication due to some error in metabolism, or to the retention of some product not being regularly excreted? We are engaged in the investigation of the urine with a view to the possible determination of this last question, but meanwhile we have not got far enough to entitle me to speak about it. I bring the case before you as showing the interest that is attached to hæmomanometer observations, and that the instrument is well deserving of a place in ordinary medical work. In this patient I have shown that her attacks are associated with a marked rise in hæmomanometer readings, but it would appear that the mere reduction of pressure, as in the attack beginning on August 23, did

not modify the course of the attack in any way. It may be that had the erythrol been given sooner and been persevered with the result might have been different. However that may be, the difference between that attack and the attack on September 2, which was at once treated with phenacetin, is very striking; in it the symptoms were promptly arrested and aborted, and along with that there was in three hours a fall in pressure of 25 mm. By the following morning a fall of 45 mm. had occurred, accompanied by the relief of all the symptoms. In this attack headache was arrested and vomiting was prevented by the administration of phenacetin, the drug above all others that arrests migraine.

Since the above was written this patient has left hospital. She left on October 15, having passed nearly a month without an attack, although in that time she had two threatenings of attacks, which have been designated in the subjoined table "abortive attacks." These abortive attacks were characterized by a feeling of drowsiness and by urine changes as indicated below. The examinations of the urine which were being carried out, as mentioned above, have been completed. They are given on p. 67, and form an interesting contribution to the question of chloride excretion in migraine; they show a relationship between the migrainous attack, the occurrence of polyuria, and the discharge of a large quantity of chloride followed by a big drop in chloride excretion.

I have to express my indebtedness to my residents, Dr. R. W. Lang Todd, Dr. T. Welsh, and Dr. A. L. Taylor, for their watchful interest in these cases.

AMOUNT OF URINE AND CHLORIDES EXCRETED IN RELATION TO ATTACKS OF MIGRAINE IN CASE 17.

	Date.	Oz.		
	Aug. 29	30		
	,, 30	34		
	,, 31	87		
Attack	Sept. 1	72	10·2 grammes total chlorides.	
	,, 2	87	16·2 ,,	,,
	,, 3	48	None.	
	,, 4	42	0·49 gramme total chlorides.	
	,, 5	50		
	,, 6	108		
	,, 7	—		
Attack	,, 8	105	23·46 grammes chlorides.	
	,, 9	34	0·286 ,,	,, (vomit 44 oz.).
	,, 10	17	0·376 ,,	,, (,, 11 ,,).
	,, 11	38	1·60 ,,	,,
	,, 12	28	None.	
	,, 13	69	0·34 grammes chlorides.	
	,, 14	64	1·07 ,,	,,
	,, 15	100	7·76 ,,	,,
Attack	,, 16	40	3·88 ,,	,, (vomit 8 oz.).
	,, 17	37	9·4 ,,	,, (,, 74½ ,,).*
	,, 18	34	1·07 ,,	,,
	,, 19	20	0·23 ,,	,,
	,, 20	29	1·8 ,,	,,
	,, 21	53	1·5 ,,	,,
Abortive attack	,, 22	124	11·2 ,,	,, (no vomiting).
	,, 23	52	4·3 ,,	,,
	,, 24	56	3·6 ,,	,,
Abortive attack	,, 25	120	13·9 ,,	,, (no vomiting).
	,, 26	78	8·2 ,,	,,
	,, 27	78	6·0 ,,	,,

DISCUSSION ON BLOOD-PRESSURE AT THE EDINBURGH MEDICO-CHIRURGICAL SOCIETY.†

Dr. William Russell said that in the time at his disposal he could not debate the question before the society. He could only try to define what he considered to be the

* Containing 6·5 grammes chloride.

† Reprinted from the *Lancet*, December 10, 1910.

essential phenomena in the subject under consideration. He would begin at the extreme end and take as an example of it a patient admitted to hospital with a brachial pressure of 295 mm. Hg. Under rest and treatment it fell to 165. It rose and kept about 190 to 210, but it would suddenly rise 70 to 80 mm. or more, with symptoms which there was no time to describe. This patient had thickened vessels, and along with the rise in pressure the vessels, including the brachials, underwent hypertonic contraction.* Nine years ago he had made a communication to the society on arterial hypertonus, in which he pointed out that hypertonus occurred in normal and in sclerosed radial arteries; with the advent of the modifications of the Riva-Rocci instrument he had carried his observations to the brachial artery, and found that it became greatly thickened, and that it could in favourable cases be both felt and seen to diminish greatly in size and increase greatly in the thickness of its wall during such hypertonic contraction. When this contraction took place the pressure in the aorta was raised—how much there was no means of determining, but there was great power of restoring the balance and relieving the heart. The essential phenomenon was the arterial hypertonic contraction, and that it embraced such vessels as the brachials. His contention was that the hypertonically contracted and sclerosed arterial wall was a large factor in the production of the brachial reading. A source of fallacy occurred from the fact that in some cases a brachial artery, although thickened, was dilated and atonic, and such a vessel gave a relatively lower reading. A man had died in his ward last week who came in with a failing big heart—failing on both sides; the lungs were congested,

* *Lancet*, December 3, 1910, p. 1602.

and he began pouring out fluid into both pleuræ; the heart sounds could not be heard; the pulse was a mere flicker at the wrist; the radial arteries were calcareous; the brachials were large, sclerosed, and tortuous; and the brachial pressure during this state of affairs was 140 mm. Hg. In one set of observations he found the pressure in the forearm was 30 mm. higher than that in the upper arm. At the post-mortem examination the myocardium of the left ventricle was soft and fragmented, and that of the right considerably replaced by fat. Yet eminent clinicians write on blood-pressure and heart power, estimated by the hæmomanometer, without a reference to the arterial wall. To him that position was beyond comprehension, and he left it to them as to whether they were prepared to accept it. He would now ask their attention to the other extreme, and could illustrate this by reference to two patients: the one convalescing slowly after an acute pericarditis was quite comfortable with a brachial pressure of 120 mm. When, from constipation, an error in diet, or emotion, the pressure rose 15 or 20 mm. he got cerebral breathing and the pulse intermitted. When this rise took place the radial and the brachial arteries could be felt to have become smaller from hypertonic contraction—that was not an opinion, that was a clinical fact—the contraction threw an increased pressure into the aorta, and the feeble myocardium was embarrassed. The symptoms could be promptly relieved by giving a vaso-dilator. The second case was a medical friend, aged eighty years, who was having recurring attacks of angina pectoris minor. During these attacks the radials became small and the brachials also became contracted, with a very poor wave in them; the pressure showed 140 to 150 mm. Prompt relief could be obtained

by a vaso-dilator, and the pressure fell to 120 to 125. He found that they entirely overcame this vessel sensitiveness, and removed the symptoms by the administration of a very small quantity of iodide of potassium. In both these cases the essential and fundamental clinical phenomenon was the arterial hypertonus, just as it was when the vessels were sclerosed. The constriction might or might not embarrass the heart. No one knew what the pressure was in the aorta; and with the armlet of the hæmomanometer they measured the changes in the arterial wall as well as the pressure inside it.

CHAPTER VII

THE ESTIMATION OF HEART POWER: THE TERMS BLOOD-PRESSURE, HYPERTENSION, HYPERPIESIS, HYPERTONUS *

GENTLEMEN,—A desire expressed by some of you that I should give a clinic on the present position of "blood-pressure" was communicated to me, and I of course at once expressed my willingness to speak to you on the subject.

At the outset permit me to say that in the consideration of many clinical problems the accuracy of our first step often determines the accuracy of our further steps, and of the conclusion we ultimately arrive at. As illustrating this proposition, I would impress upon you that it is necessary to realize that, when we as clinicians feel the radial pulse, the initial and primary object in doing so is to obtain information as to the vigour or strength of the heart. The second object is to gain some knowledge of the condition of the channels through which the blood is propelled. As directly bearing upon our subject, I recall to you various terms used in relation to it.

Blood-Pressure.

This term means the pressure exercised by the blood inside the vessel, and the driving power of the heart is judged of by the estimate we form of this pressure of the

* A post-graduate lecture (reprinted from the *British Medical Journal*, March 23, 1912).

blood upon the arterial wall. In much recent writing the term unfortunately is used to indicate, or as a mere synonym for, hæmomanometer readings. Whatever the blood-pressure be inside the artery, it represents the power with which the blood is driven out of the left ventricle. This is the plain and fundamental fact, and this is the reason why the examination of the pulse occupies such an honoured place in our clinical methods. Other words are, however, in use in the study of this same problem of heart power.

Hypertension.

The term " hypertension " is at present in common use, and has, I think, been popularized by the French school. In past years high and low tension pulses were spoken of. The word " tension," applied in this way, was, however, long regarded by critical clinicians as unsatisfactory The question always was whether it merely meant the amount of pressure exercised by the blood on the arterial wall or this combined with changes in the wall itself. So far as I am able to see, the use of the term " hypertension " in no way answers that question. Some clinicians steadily abstain from using the word " tension," in any of its possible meanings, as applied to the pulse, and with this attitude I am in complete accord. Without clear thinking and the use of suitable words the confusion which surrounds this subject will continue.

Hyperpiesis.

The term " hyperpiesis " has been introduced by our veteran clinician, Sir T. Clifford Allbutt. Etymologically it means excessive pressure, and throws no light on the problem of how much is blood-pressure and how much is

arterial wall. If it carries with it the idea of fullness, the question at once arises of the relation of fullness to pressure. In measuring arterial pressure with the hæmomanometer, the question remains, Are we measuring the pressure-wave only, or are we not also measuring vessel wall ?

Hypertonus.

The only other word I need refer to is " hypertonus." I am commonly regarded as having definitely applied it to an arterial condition. It means a contraction of the muscular coat of arteries over and above the normal or average tonus. For many years, before the hæmomanometer came into use, I had watched the occurrence of this over-tightening up of the arteries and the relaxation of the over-tightening. It is a clinical phenomenon which is as certainly present and is as easy of recognition as any phenomenon appreciable by the senses.

Arterio-Sclerosis.

I must next ask your attention to the term " arterio-sclerosis." It was introduced almost as another word for " atheroma." The thickened radial arteries which are so commonly met with clinically are, however, only rarely atheromatous; the thickening is due to hypertrophy of the middle—that is, the muscular—coat. This thickening may occur with hardly a trace of atheroma, and most of the arteries with a muscular coat may be so thickened. It is a very common condition.

The Hæmomanometer.

The modified Riva-Rocci hæmomanometers all have an armlet, which is usually applied to the upper arm. The bag encircling the arm has air pumped into it until the

pressure has stopped the pulse-wave, as indicated by the disappearance of the radial pulse at the wrist. The pressure in the bag is shown by a **U**-shaped mercurial manometer, or by a spirit manometer as introduced by Dr. George Oliver, or by a neat little mercurial manometer recently introduced by that ingenious physician. It does not really signify what manometer is used, for they are, of course, all carefully standardized, and either of the two latter is easily carried about. The armlet is practically the same in all.

The Claim.

The armlet, when applied to the upper arm, obliterates by pressure the lumen of the brachial artery. This artery, when perfectly normal, is so thin-walled that when empty it collapses by its own weight. In this condition of artery the pressure required to close the vessel corresponds with the blood-pressure inside the vessel. This physiological fact was taken up by some distinguished physicians, who maintained that, no matter what pathological changes might be present in the artery compressed, this instrument recorded blood-pressure—that is, recorded the same pressure as would be recorded were a cannula inserted into the artery and attached to a manometer.

The Author's Position.

My contention has been, and is, that when the artery wall is thickened the thickening increases the resistance to compression, the resistance then becoming *blood-pressure plus vessel wall.*

The difference between the thickness of the normal brachial artery, which, as I have said, collapses by its own weight when empty, and an artery thickened by sclerosis

—that is, by muscular hypertrophy—is great. In addition, however, to the muscular thickening, the thickened muscular coat retains the power to contract and to relax. When it contracts the wall becomes still thicker, and the lumen becomes still smaller. Now, I submit to your common judgment that, when you put the armlet over such a thickened vessel, you have not only to overcome the resistance of the blood-pressure, but of the hypertonic vessel wall in addition. The phenomena have passed from the physiological to the pathological, and therefore to the clinical.

As it is true that the thick-walled vessel requires greater force to obliterate its lumen than the thin-walled vessel requires, it follows that when the thick muscular coat contracts beyond its normal *tonus* the vessel wall is further thickened, and the lumen is further diminished, so that still greater pressure is required to obliterate the lumen and to stop the pulse-wave. The *hyper*-tonically contracted sclerosed artery is more resistant to pressure than the tonically contracted one.

Three Factors in Hæmomanometer Observations.

It thus follows that in taking hæmomanometer observations three factors of clinical importance have to be considered—namely, (1) blood-pressure; (2) structural changes in the wall of the artery; (3) hypertonus of the muscular coat. Upon the correctness of your estimate of these separate factors will depend the correctness of your conclusions, and will determine the real value of this instrument in your clinical work. If you take its readings as only giving the first factor—namely, blood-pressure—you will make the most appalling and disastrous mistakes, and I have known such to be made.

Recent Experimental Work.

I am aware that this subject can appear so clear to you, and my reasoning be so convincing, that further evidence may be regarded by you as unnecessary. But the fact remains that my contentions have not yet met with universal acceptance, and to help the conversion of our doubting friends I may tell you of some recent experimental work on the subject.

In the first place I shall refer to Dr. J. C. Janeway, of New York, who, I think, wrote the first book on blood-pressure after the hæmomanometer came into common use. He was a pure blood-pressurist, and held that there was no evidence that the condition of the arterial wall affected hæmomanometer readings—that the readings always meant blood-pressure, and nothing else.

He has recently taken up the question experimentally, in conjunction with Dr. E. A. Park, using arteries taken from freshly-killed cattle for the purpose. It is interesting to find from the record of these experiments* that jointly they are satisfied that *hypertonus in normal arteries* may represent a pressure of 51 to 68·5 mm. Hg. This is a large rise from the previous zero, and it is acknowledged that the effect of hypertonus had been overlooked—not, of course, by me, either clinically or pathologically. They say: "We must acknowledge that Russell has called attention to a hitherto *insufficiently recognized* influence of arterial contraction on our clinical readings." They would have been more correct had they said *totally unrecognized*. In the sentence immediately preceding this halting acknowledgment the statement is made that "it

* Janeway and Park, *Arch. of Int. Med.*, November, 1910, vol. vi., p. 586.

seems to us reasonably proved that Russell's contention, that hypertonic contraction of the arteries, and not high blood-pressure, is the cause of the high readings obtained with clinical instruments, has no basis in fact." I am entitled to claim that when my work is criticized it should be presented accurately, and the sentence just quoted misrepresents my contentions, and, indeed, has " no basis in fact." My critics have not acquainted themselves with my work as they ought to have done, seeing my book was in their hands. They overlook the clinical and pathological fact upon which I based my contentions—namely, that the structural change present in the majority of the thickened arteries, recognized as such during life, was hypertrophy of the tunica media. I suggested that the term " arterio-sclerosis " should be confined to this condition, as it was the commonest form of thickening in radial arteries, and had no resemblance whatever to atheroma. It is clear that my critics have entirely failed to realize the significance of that contribution to clinical and pathological knowledge. They are entitled, perhaps, to confuse atheroma with other arterial thickenings, but they are not entitled to represent me as doing so. Having failed to note the significance of my pathological findings, they also failed to grasp the contention that arteries with such a thick muscular wall presented much greater resistance to compression by the armlet of the hæmomanometer than normal arteries present. The further point they have also, as a consequence, missed—namely, that the hypertrophied tunica media retained its power of passing from tonus to hypertonus, and that hypertonus in such an artery leads to further thickening of the wall and to diminution in the size of the lumen, and consequently to still greater resistance to compression by the armlet. My

critics have acknowledged that the hypertonus of a normal artery may give an increase of pressure of from 50 to 68 mm., but they will also find that when hypertonus is present in an artery with a hypertrophied muscular wall the resistance is greatly added to. I may mention that arteries with such walls as I describe will not be found in cattle; they must be sought for elsewhere.

My critics, however, have experimented, not only with normal, but with atheromatous and calcareous vessels, and speak of these changes as arterio-sclerosis. The use of the term "arterio-sclerosis" for all arterial thickenings and degenerations is deplorably inaccurate, and the matter under discussion will not be satisfactorily settled until totally different pathological processes cease to be called by the same name. Clinicians must accept the fact that the thickened arteries with which they so frequently meet are not usually atheromatous, but are thickened as I have indicated and act as I have indicated. The atheromatous vessels used for the experiments were suspended in a glass cylinder, and the compressing medium was water introduced into the cylinder. It is shown that the water picks out the unaffected portions of such arteries, and compresses the artery at these points, and on this observation it is contended that *arterio-sclerosis* (which in the vessels used clearly means atheroma) has no effect in making the vessel more resisting to pressure. The observation so far as atheromatous arteries are concerned is correct, and is in harmony with our pathological knowledge of the scattered and patchy character of the lesions in atheroma, but a grave mistake is made in not allowing for the difference between the experimental method adopted and the clinical method. The observers might

have seen that, while a water-jacket would necessarily pick out and compress the smallest normal areas in the course of an atheromatous vessel, the armlet assuredly bridges over such areas unless they are extensive. No doubt the pressure exercised by the compressed muscle of the arm is spoken of as "fluid pressure," but to be led into experimental or clinical error by the use of this term, in a matter of this kind, is not reasonable. Muscle is not fluid in the sense in which water is fluid. The same method has been used by other experimenters, but Drs. Janeway and Park are the first to find that normal arteries, when hypertonically contracted, may add over 60 mm. to the pressure required to obliterate them. From this observation the authors of it must see that the conclusion is unavoidable that the muscular thickening, to which I hold the term "arteriosclerosis" ought to be confined, must add to the resistance to compression, and that when hypertonus is also added there is a great additional resistance offered to the air-bag.

I may state, what I have stated elsewhere, that in this condition the thickening is uniform and not patchy. It is, perhaps, also necessary to state that the hypertrophied muscle of an artery wall may lose its tonus, as the hypertrophied wall of a left ventricle may, and when it does so it relaxes, and resistance is then, of course, lowered. Thickness and tone of wall and size of lumen must always be considered together, if accurate understanding is to be reached.

In the *British Medical Journal* in 1908 I published observations made on a thickened brachial artery, obtained shortly after death, tested by means of an air-bag, and found that it indicated a pressure of something

over 100 mm. Hg, and I do not think that this is the limit of resistance of the arterial wall.*

In July, 1911, Dr. Leonard Findlay published an interesting series of observations on brachial pressure, as compared with digital pressure, at different ages.† The digital pressure was measured by Gaertner's tonometer. In early life there is little difference between them—that is to say, the blood-pressure is much the same in the finger as in the brachial artery. As life advances, however, there appears a marked difference between the two, until between forty and fifty years of age there may be a difference of 88 mm. Hg. In cases of Bright's disease this difference between brachial and digital artery pressure may reach 100 mm. Hg. He thinks the explanation is to be found in the arterial hypertonic contraction. He also experimented on cats with adrenalin, and found that the *difference* between the carotid and the paw pressure rose from 50 to 140 after adrenalin injection. His explanation is that "the constricted arteries cut off the pressure wave," that the pressure wave is different in rigid and in elastic tubes, and that when "arteries are more or less sclerosed, or in acute Bright's disease where the arteries are tonically contracted, the elasticity of the vessel is diminished and more nearly approaches the character of a rigid tube."

Experimental work is thus confirming my clinical and pathological contentions.

* W. Russell, *British Medical Journal*, 1908, vol. ii., p. 1076: "Arterial Hypertonus, Sclerosis, and Blood-Pressure" (Green and Sons).

† Leonard Findlay, *Quarterly Journ. of Med.*, vol. iv., No. 16, July, 1911.

Illustrative Cases.

I next ask your attention to three cases which illustrate how valuable and interesting hæmomanometer observations are when properly interpreted.

Case 18—*Cerebral Symptoms due to Hypertonic Contraction.*—This patient was under my care in Ward 27 in September, 1910. The phenomena she then presented consisted of cerebral symptoms whenever the arterial pressure rose, as it could do, from 200 to 260 to 290 mm. I showed her to the post-graduates, and her case is recorded in the *Lancet* of December 3, 1910. I had lost sight of her, so that her being here now has been quite fortuitous so far as we are concerned. She has kept fairly well, but is threatened with a return of her symptoms, and whenever these threaten there is an associated rising of her brachial pressure. At present her pressure keeps about 200 mm., but as soon as it begins to rise she develops cerebral symptoms, which are promptly removed by the administration of a vaso-dilator. If left alone the pressure can rise from 200 to about 300 mm., and we are asked to believe that this is the result of a variation in this woman's heart power. As a matter of fact, what happens is that her brachial artery becomes markedly hypertonically contracted, and it is this which gives the high reading; when the artery is relaxed by erythrol the reading rapidly falls to 200 again. It is this recurrence of hypertonic contraction which is this patient's danger. It is not any variation that the hypertonus may lead to in stimulating the left ventricle to increased effort that is the essence of the clinical phenomena. It is not a heart instability, it is a vessel instability; and if we miss that we miss the essential factor in the whole series of phenomena which

the case can present; we fail to understand the case, and what we fail to interpret aright we cannot treat aright.

CASE 19—*Arterial Thickening due to Syphilis.*—I next show you a man, aged forty, who was admitted on account of severe and constant headache, and who had syphilis three years ago. His radial and brachial arteries were somewhat thickened, and his brachial pressure varied between 140 and 150 mm. The pulse-wave was small and feeble; there was no heart enlargement discoverable by percussion; the heart impulse was feeble and the sounds were very faint, the first sound at the apex and over the right ventricle being barely recognizable. He was anæmic, with a red-blood count of 2,560,000 and 46 per cent. of hæmoglobin. He was put on large doses of potassium iodide. The headaches soon yielded to this treatment; at the same time the changes in his arteries and in the vigour of his circulation were of particular interest, for one does not often have the opportunity of following the changes as we had in this patient. When he had been taking the iodide for some time the arteries became quite perceptibly softer and larger, and along with this the pulse-wave became larger and fuller, and the first sound of the heart came out clearly and definitely. These phenomena were as definite to my house-physicians as to me. With these marked changes in the circulatory phenomena there was, however, no change in the hæmomanometer reading.

I have seen this kind of thing before, and the interesting clinical facts which are to be noted in such cases are—(1) That the hypertonic thickening of the vessel has yielded; (2) that the power of the left ventricle has been restored, and the pulse has become larger and stronger; and (3) that there has been no change in the hæmo-

manometer reading. No one who still claims to know anything about the pulse as measured by the finger would for a moment doubt that there had been a marked increase in heart power in this patient. Such an increase was confirmed by the change in the character of the first sound and of the impulse. Yet there was no change in hæmomanometer reading; and there ought to have been, did this instrument record blood-pressure and blood-pressure only.

From my standpoint, the explanation is that what was gained in heart power was balanced by the relaxation of the hypertonus. That is to say, if the blood-pressure rose 60 mm., it was balanced by a loss in the resistance of the vessel wall of 60 mm.

Again you see how entirely misleading this instrument will be if you take it as the measure of heart power in your clinical work, and yet how interesting and valuable it is when you learn to interpret its records aright. I shall not detain you by showing you other cases; but I should like to tell you about a medical friend whom I saw a few days ago, as he illustrates another aspect of the subject we have been considering.

Case 20—*Angina Pectoris Minor with Hypertonic Spasm.*—A few months ago this friend consulted me about a recurring pain in the left chest over the precordia and posteriorly. It was difficult to be quite satisfied as to the meaning of this pain, for the heart was perfectly sound and the brachial pressure was only 125 mm.; the vessels were soft and the pulse regular and of good volume. He, moreover, was a strong cyclist, and his cycling did not make things worse, nor did it bring on the pain. With treatment he got relief for three months, but the pains had returned; they were more severe and very perturbing, as they came on at unexpected moments, and

when they supervened he had to stop what he was doing. There was a suggestion of the pain going down the left arm. He was anxious that I should not think his symptoms merely fanciful. The pulse was 80 and the pressure 125 mm.; the heart sounds were clear and closed. After the examination we sat talking for some time, when he told me that an attack was coming on. His colour did not change, but he leant forward, resting his elbows on his knees, and was evidently considerably distressed. I again felt his pulse and found it distinctly smaller, and instead of beating 80 it was only beating 60. I asked him to take his coat off, and on taking his pressure found it had risen from 125 to 145 mm.

This observation, to my mind, at once settled the question of diagnosis. This was a true angina pectoris minor, a condition I described years ago. My interpretation of the phenomena is that the state of the vessels, as revealed to the finger and shown by the rise in the hæmomanometer pressure from 125 to 145 mm., indicated that a hypertonic spasm had taken place; that this increased the pressure in the aorta, and thereby perturbed the heart, which, instead of being accelerated, was slowed down from 80 to 60 beats per minute. To speak of this as a neurosis almost always suggests that the symptoms are either fanciful or that the condition is mainly determined by a mental and central influence; and no men more warmly resent this view when applied to themselves than medical men do. Personally I interpret the phenomena as being determined by peripheral conditions, and not by central ones. To me this is a vessel irritability, and its successful treatment lies in the removal of the cause keeping up that irritability. In this particular case the cause was probably tobacco.

Again I point out to you that the variation of hæmomanometer reading was due to change in the vessel wall (the brachial artery); a mere increase of heart power represented by 20 mm. would not give rise to such symptoms. My patient could, I doubt not, stimulate his heart by cycling above that and without discomfort, but the moment a general angiospasm occurs the symptoms appear. It is always a question whether or no the coronary arteries participate in the spasm, but the practical fact is that if you counteract the general angiospasm the symptoms disappear. Your patient is cured when you have removed the tendency to this hypertonic spasm. The foundation fact is the vessel spasm, for the moment you relieve it the symptoms disappear; the spasm affects the brachial as well as the smaller arteries, and, I repeat, the hæmomanometer gives you the record of it, when the vessels are normal as well as when they are sclerosed.

There are always the three factors to keep in mind—namely, blood-pressure, structural change in the vessel, and hypertonus. There is no escape from the exercise of discretion and judgment when using this instrument, and those are the points; the significance and value of each have to be determined in every individual case. The instrument has opened no *royal road* to the determination of the all-important question of heart power, as we are called upon to judge of it clinically.

CHAPTER VIII

MOTOR AND SPEECH PARALYSIS DUE TO CEREBRAL ANGIOSPASM*

Introductory.

Definition.

GENTLEMEN,—Angiospasm is a spasmodic constriction of vascular channels, no doubt mainly on the arterial side, which implies that the wall of the bloodvessels is an active muscle structure. It also implies that such constriction can be limited in area, can be local as distinguished from general, and that we are entitled to think of spasm in cerebral vessels as sometimes focal and sometimes general, but not necessarily and always general.

Effects.

The effects of angiospasm depend upon the organ which is the seat of the condition. To-day I do not dwell upon angiospasm in the stomach or intestine, in the lungs, or in the heart's coronaries, but confine myself to the phenomena which we contend follow upon this condition in the brain.

I ask you to picture to yourselves what occurs during angiospasm. All spasm varies in degree or intensity, so you have to picture variable degrees of spasm in the muscular coat of arteries. All spasm here, as elsewhere, leads to diminution in the sizes of the lumen of the affected

* A post-graduate lecture (*Lancet*, November 16, 1912).

vessels, as the muscle fibres are arranged round the vessels with their long axes in the transverse way of the lumen. Such spasm, therefore, necessarily leads to a constriction of the lumen, and may be sufficiently pronounced practically to obliterate the lumen, so that neither blood-corpuscles nor serum flow along it; at other times serum may pass and no corpuscles, or a single file of corpuscles may be driven slowly along. The degree of spasm determines, therefore, the measure of the anæmia of the affected area, and this, again, determines the intensity of the clinical phenomena, whether it be complete or partial loss of motor power or of sensibility; while the portion of brain affected determines the character of the phenomena, whether motor, sensory, or mental.

As spasm tends to be temporary and evanescent, so the phenomena the result of spasm tend to be evanescent; but if the spasm be continued and pronounced, softening results, and when softening ensues loss or impairment of function is permanent.

You realize, of course, that the symptoms are not due to vessel spasm *ipso facto*, but to the arrest of function from curtailment of blood-supply to the part. It is the anæmia which is the cause of the phenomena, and anæmia may be the result of thrombosis or embolism; spasm is only an additional cause, although we contend that it is a much more common cause than is generally recognized. I do not propose to labour the propositions that vessel spasm can and does occur, and that it may be confined to a limited area. We all know it in "dead fingers"—a solitary digit sometimes being pale and bloodless—in Raynaud's disease, and in the constriction of retinal vessels producing blindness.

Causes.

The causes of such spasm are, for instance, local cold; local heat, as in the stopping of uterine hæmorrhage by hot douching; nerve influences, as shown by the effect of emotion; and in the various angioneuroses. But there is another equally important group of causes—namely, where vessel constriction is brought about by direct action on the vessel wall, as is the case with adrenalin, digitalis, etc. In addition, there seems to be no doubt clinically that the waste substances retained in the blood act thus upon vessel wall. That waste substances produced as the result of muscle activity accumulate and can affect muscle action has been shown by their paralyzing action on muscle, while washing out the bloodvessels enables the muscle to remanifest its function of contractility. As regards angiospasm in the brain, I think it is probably caused by waste products irritating or stimulating the vessel wall directly, but later I shall again refer to this point.

Personal.

I ask you to permit me to make a little personal explanation here. Twelve years ago I drew attention to the cerebral symptoms associated with hypertonic contraction of systemic arteries, and suggested a corresponding state of cerebral vessels to explain the symptoms. I supported the suggestion by the clinical observation that vessel relaxation induced by a vaso-dilator led to relief of the symptoms. I was not then, and am not yet, conscious of having borrowed this explanation from any source whatsoever. I have written a good deal on the subject since then, and I have also found some references to the phenomena in question, accompanied by the sug-

gestion that they were due to local vessel spasm or to temporary local anæmia. I claim, however, that my contributions to this subject have definitely drawn attention to it, and I hope established it as an interesting condition, the understanding of which is highly useful to the practical physician. I shall refer to the historical aspects of the subject under the various classes of phenomena with which I propose now to deal.

Varieties of Phenomena.

Those cases which show motor symptoms may be regarded as exhibiting partial or complete abeyance of function in brain areas rather than irritation of areas. This distinction is of significance in so far as it may be taken as an evidence of impaired local nutrition—that is, of anæmia rather than of irritation. If we adhere to the postulate that the symptoms are due to anæmia of the affected areas—and I ask you to do so for the present, at least, as otherwise I fear my argument cannot be followed —we shall understand that two factors will require to be kept in mind when dealing with individual cases—namely, (1) the probable condition of the blood-channels, and (2) the driving power of the left ventricle. Although my main thesis in this lecture is the anæmia due to vessel spasm, I must indicate to you that anæmia may be determined by atheroma or obliterative endarteritis associated with a feeble heart. I can illustrate this class of case as follows:

Feeble Heart Cases.

I had recently a patient in my male ward who had recurring attacks of inability to utter an articulate word, associated at times with slight paresis of his right limbs.

The speech phenomena were present during the night, so that when he awoke he was unable to ask the night nurse for anything he wanted. As this man's general condition and heart power improved this disappeared. The explanation I offered of the speech phenomenon was that the brain vessels were almost certainly atheromatous, that the feeble circulation was at its feeblest during the night, and that the aphasia and paresis were due to local anæmia from the operation of these two conditions. That the explanation was reasonable was borne out by the cessation of the phenomena when the circulation became stronger and able to keep up a sufficient flow through his narrowed vessels—narrowed structurally, not by spasm.

In case the apparent novelty of my explanation should lead you to doubt its soundness, I may tell you that Brissaud in 1893 recorded the case of a woman suffering from heart failure who exhibited recurrent aphasia with right hemiplegia. At each return of cardiac asystole there was a corresponding return of the paralysis of the right limbs with transitory aphasia. In this case, I venture to think, there must have been atheroma of cerebral vessels as well as the recurring heart failure.

This case, you will grant, strengthens my position, and you will realize how important it is that cases of the kind should not be misunderstood. If such a case is regarded as one of " high blood-pressure " with threatening hæmorrhage, and relaxing and depressant treatment is adopted, you realize the evil consequences which must follow on such a diagnostic error. It is stimulant, and not depressant, measures which are required.

Migraine and Temporary Paralysis.

I shall also not dwell on the association of migraine and temporary paralysis, as the association is now widely known. For historical purposes I want to state, however, that Ball, a French physician, described the case of a patient who suffered habitually from migraine, and who had twelve attacks of aphasia in nine months, accompanied by slight paresis and convulsive movements in the right hand. He supposed these cerebral phenomena were due to a temporary anæmia. I do not know whether he is to be credited with attributing the anæmia to angiospasm. These brain symptoms occur in migraine without arterial disease, while the hemianopia, which is not uncommon in migraine, is associated with angiospasm of the blind area in the retina, the blindness passing off when the vessels relax. That we should extend a corresponding explanation to the brain symptoms is inevitable, and surely highly reasonable.

Motor Paralysis and Aphasia.

This is the commonest type, and forms the largest group of cases. It consists of persons usually at or beyond middle life, frequently giving unmistakable evidence of arterial thickening or of atheroma, and not as a rule showing heart feebleness. The group illustrates angiospasm in cerebral vessels affected by either atheroma or obliterative endarteritis. In passing, I would remind you that atheroma is common in cerebral vessels when its presence elsewhere is clinically negligible.

Transitory Hemiparesis.

I cannot do better than begin the consideration of this big group by reading a letter I received some six weeks ago from a medical practitioner in Southsea about a valued old friend of mine. He says: "I am writing you about Mrs. ——. A fortnight ago she suddenly lost power in her left arm and hand and lower extremity. This loss of power was transitory, and two days later there was no sign of it. Her blood-pressure was 200. I kept her in bed for a week, gave small doses of calomel at night, advised light diet, and also gave her a grain of sodium nitrite and 10 grains of potassium nitrate three times a day. This brought her pressure down to 175." This letter was dated July 25. I saw the lady on August 17, and again on September 4. She was well, and had had no return of the symptoms mentioned. In this patient you will note that there was a high arterial pressure. This illustrates what you will often find—namely, that during an attack of paresis there is a marked heightening of the ordinary arterial pressure shown by the individual, the result of the hypertonic contraction of the systemic arteries. The heightened pressure was readily lowered in this patient by the excellent measures adopted by her Southsea physician. It was the frequent association of such paretic seizures with hypertonic contraction of the systemic vessels, and the disappearance of the symptoms when the vessels were relaxed by a vaso-dilator, that led me to suggest that a like constriction of vessels in a brain area was the cause of the paresis in such cases.

Another illustration of this evanescent hemiparesis is afforded by a man, aged forty-seven, who died in the Royal Infirmary five days ago. He was the subject of

advanced kidney disease, but he was also aphasic, the aphasia presenting some very interesting features. The history we obtained was that three or four years ago he had " a shock," in which the right side of his body was paralyzed, but from which he recovered. We were also informed that he had had similar attacks repeatedly, all of them apparently of short duration. He was admitted to my ward on May 25, the aphasia having come on three days before that and lasting to the date of his death on August 31. There was no other paralysis. He was treated as a kidney case, and it was of his kidney condition he died. In the brain there was an area of softening, about the size of a pea, far forward in the white pathway of motor speech, and no other lesion. Now, this man had had several attacks of hemiplegia, from all of which he recovered, and none of which left any visible trace in his brain. The aphasia, which was permanent, was due to the permanent break in the communicating white fibres of his speech centres.

This is not the first brain I have seen from a patient who had had a temporary hemiplegia of which no trace was left in the brain, and I bring this last case before you as a further support to my contention that these temporary paralyses have an evanescent cause, and that the only reasonable explanation is the one I have offered you.

I would add to this that the site of angiospasm is very frequently in the great motor pathway, in or above the internal capsule. And if the question is asked why this position should be a common one, I can only answer that in my experience softening of grey cortex is not common; that the motor pathway, especially in or near the internal capsule, is the common seat of hæmorrhage and of softening. It would seem as if it were not the centre, but the

busy thoroughfare, which is so apt to be damaged by either of these, and which I think is the seat of angiospasm also.

Historical.

I shall now endeavour to strengthen my contentions by pointing out to you that the explanation I have offered has been proposed before, although it has never attracted sufficient attention to make it of practical value to the practitioner in his work. The only British author who refers to the subject is Bastian; he quotes a case published by Daly in 1887 in which "recurring attacks of transient aphasia and right hemiplegia," lasting for from three minutes to three hours, occurred in an elderly gentleman. Daly supposed that the attacks were due to spasms of vessels brought about under the influence of gouty and uræmic poisons in the blood. Bastian himself, in view of this, suggests that "poisons circulating in the blood may lead to contractions of the small arterioles, thus temporarily cutting off more or less completely the blood-supply of cortical centres."

In 1886 Dr George Peabody delivered an address to the Practitioners' Society, New York, entitled "A Contribution to the Symptoms and Pathology of Endarteritis Obliterans." He brought forward three cases, "not as something new," but as bearing upon the pathology of a condition about which little work had been done. One of these cases was a man, aged fifty-six, who had an attack of transient right-sided hemiplegia. Then in the course of ten days he had four or five attacks of aphasia with incomplete hemiplegia. Finally, he died in a complete attack, in which he had complete right-sided hemiplegia, with loss of consciousness. At the post-mortem

examination there was found extensive endarteritis of the vessels of the brain, but no local lesion, œdematous areas, or focus of hæmorrhage. The arteries were diseased, but not sufficiently encroached upon to account for death. Commenting upon this, Dr. Peabody states that death may result from this lesion—*i.e.*, endarteritis obliterans—with striking clinical evidence of destruction of motor areas in the brain, which necropsy reveals to be intact. He adds that "the only explanation which suggests itself is that, in addition to the partial obliteration of the area affected, there must have been a spasmodic contraction of the vessel or vessels which was sufficient to cause complete local arrest of the circulation. The duration of the spasmodic contraction varies. In some attacks it lasts only a few minutes; then all the symptoms pass away as rapidly as they came. In others the duration is prolonged to such an extent that life comes to an end before it ceases, and yet the duration may not be sufficiently prolonged to cause softening of the brain area affected." Peabody may have got the idea of brain arterial spasm from a paper by E. G. Loring in this same year (1886) on spasm of arteries of the retina causing temporary and permanent blindness.

Again, in 1891 Dr. Peabody, in a communication on the relations between arterial disease and visceral changes, describes the same case and offers the same explanation. This latter communication was made at a meeting of the Association of American Physicians. He again made no claim to the discovery, and in the discussion which followed this part of the paper seems not to have been touched by the members present, the gathering including Sir W. T. Gairdner and other British physicians.

Sir William Osler, before the Canadian Medical Associa-

tion in 1911, seems to have quoted Dr. Peabody's 1891 communication as the first statement of spasm of the arteries as a cause of paralysis, transient or otherwise, and appears not to have referred to the 1886 communication. How little this communication of Dr. Peabody influenced medical teaching or thinking is shown by the absence of any reference to this condition in Sir William Osler's excellent and popular book on the " Principles and Practice of Medicine " down to the 1909 edition at least, so far as I have been able to discover.

Now, gentlemen, I have supported my contentions by showing you that others have offered the same explanation of the phenomena in question as I have offered, and that, whatever credit may be due to me for having repeatedly written on this subject, and so drawn the attention of my brethren to it, I, no more but rather less than Dr. Peabody, can claim to be the " discoverer." Sir William Osler, who made this subject the theme of a holiday lecture in Canada last year, claimed for Dr. Peabody what Peabody himself disclaimed.

CHAPTER IX

PHENOMENA ATTRIBUTABLE TO CEREBRAL ANGIOSPASM*

I PROPOSE to speak to you to-day on some points bearing upon the blood circulation in the brain, and of phenomena due to disturbances of that circulation. Owing to the position of the brain within the cranial cavity, local disturbances of the circulation are difficult of proof; they can only be assumed from the clinical phenomena which may be present, and from our knowledge of the dependence of local function upon an efficient local blood-supply.

The special condition I propose to speak about is spasm constriction of the arterial channels in localized areas of the brain; the term "cerebral angiospasm" is given to the condition. Although the cerebral vessels cannot be seen, the vessels of the retina can be examined by means of the ophthalmoscope, and it has been clearly established that angiospasm occurs in these vessels. Various observers have noted that a temporary loss of vision of one half of the retinæ may be associated with marked diminution in the size of the retinal vessels corresponding to the blind field, and that vision returns with the relaxation of the constricted vessels and the re-establishment of the normal blood-supply to the part. From these observations alone it is fair to postulate that corresponding constriction can take place in the arteries in localized areas of the brain.

* A clinical lecture (reprinted from the *British Medical Journal*, May 16, 1914).

Migraine.

This can be illustrated from a common and well-known disorder—namely, migraine.

It is a well-established clinical fact that in migraine such disturbance of vision as has just been referred to is by no means uncommon, and that it can be shown to be associated with the change in the retinal vessels referred to. In migraine, however, there are other temporary phenomena which must have their origin in the brain; of these, perhaps one of the commonest is partial or complete loss of power of one side of the face, of one limb, or of both limbs on one side. Other temporary phenomena are characterized by disordered sensation or temporary loss of sensibility, the distribution of such phenomena being usually, if not invariably, one-sided, and affecting either side. These phenomena, and the explanation which I have given you as to their causation, are now pretty generally recognized and accepted.

Loss of Memory.

There is, however, another set of phenomena which I venture to think may be explained on the same hypothesis —namely, the loss of memory of recent actions which ordinarily imply not only active volition, but conscious volition; this set of phenomena is illustrated by what frequently occurs in old people. Old people tend from time to time to get restless and sleepless, and noisy or active during the night. In an attack of this kind old people will get out of bed, disturb the other inmates of the same room by interference with the bedclothes and by other acts; they will go to the fire and interfere with it; light matches without any apparent object in view; and

the following morning have absolutely no recollection of what had happened during the night; they may even state that they had a good night and slept well. Periods of mental excitement and incoherence come on in the same way, and may be followed by mental coherence during the day; and here also there is no recollection of the restlessnes and incoherence of the night. Cases of this kind may show, during the periods referred to, a definite constriction of systemic arteries; and when this occurs it is found that the most suitable remedy for removing the symptoms is the administration of a vaso-dilator. So impressed have I been with this experience that, whenever such cerebral symptoms occur as I have referred to, and if the condition of the arterial pulse even suggests arterial constriction, the routine practice is to administer a vaso-dilator; and it is quite remarkable the number of patients who respond to this treatment and whose attacks may be thereby arrested. The two drugs which I use mainly are phenacetin and erythrol tetranitrate. From the character of the phenomena and the effect of treatment, it seems to me reasonable to explain such cases on the lines I have indicated.

I beg you to note, before passing from this point, the importance of the proposition that a person who is awake and able to move about, and is able to do acts which ordinarily imply conscious volition, may have recurring periods during which he is absolutely unconscious of his acts and doings.

The case of Walter S., which follows, is, I believe, a striking example of this general proposition. In this particular case there were, apparently, definite epileptic phenomena; yet to designate the phenomena to which I am specially referring as epileptoid does not help much;

whilst a similar forgetfulness in the nocturnal disturbances of old people cannot be regarded as epileptoid. The presumption is that the explanation of the phenomena in old people is applicable also to the phenomena in this case.

Case 21.—W. S., aged forty-five, shale-miner, was admitted on October 30, 1913, complaining of fits of unconsciousness. This patient had been previously admitted to the hospital on February 14, 1913, and was discharged on March 14. At that date he was admitted because of sudden fits of unconsciousness. He stated then that he suddenly became unconscious without any previous warning, and that these attacks had been present for over two months; no twitching or muscular movement of any kind took place during the attacks, and he was not pale or cyanosed during them. They took place at intervals of about a week. While in hospital he had two fits which lasted about one minute, from which he slowly recovered, and was quite conscious in about five minutes. A positive Wassermann reaction was in accordance with the specific history given by the patient.

When admitted on October 30, the patient stated that since January, 1913, he had been subject to attacks of mental abstraction, during which he was unconscious of his actions. The attacks lasted for from two to five minutes, but he neither knows he is having an attack nor has he any warning that an attack is coming on. After an attack he is told of his having done things which he has no recollection of having done; the things he does are often "foolish"—as, for instance, he would empty a full pipe and fill the pipe with brown paper without having any recollection of doing so; in one attack he caught one of his children, and threw the child out of the house.

When I questioned him about what he had done to his child he was evidently much perturbed emotionally by the matter being recalled to him, and assured me that he had no recollection whatever of having done such an act. He has one or two attacks weekly, and they are not preceded by any aura.

Circulatory System.—The left border of the heart at the apex was slightly outside the nipple line. There was a systolic murmur at the base of the heart. The pulse was regular in rate and in rhythm. There was a marked difference between the two radial pulses, the right being much more forcible than the left. The vessel pressure was 126 mm. An X-ray examination showed an aneurismal dilatation of the first part of the aorta.

Respiratory System.—The respiratory system showed nothing abnormal.

Nervous System.—The organic and skin reflexes were normal. The plantar reflexes showed normal flexion. The tendon reflexes in the arms were brisk. The knee-jerk was somewhat exaggerated on the right side and somewhat diminished on the left. Both pupils reacted normally to light and to accommodation; there was slight horizontal nystagmus. The other systems presented no abnormal phenomena.

On November 3, about 11 p.m., the patient was unconscious and presented a series of clonic contractions of the muscles of the extremities which lasted about two minutes. The patient frothed at the mouth—mucus being blood-stained—but he did not bite his tongue; he slept afterwards.

On November 7, at 10 p.m., the patient was noticed to be looking queer, and on examination it was found that he was lying on his back, with his eyes wide open and turned

upwards; on being spoken to several times he made no response; in a few minutes, however, he recovered and went to sleep. Several attacks occurred during the following days, and on November 12 the patient left hospital against the advice of the house-physician.

Mental Obscuration with Attacks of Greater Obscuration and Inability to Work.

The next type of cerebral phenomenon to which I would draw your attention is characterized by a complaint of sleepiness, of entire want of energy, and of marked forgetfulness. Such symptoms are the result of grave disturbance of the circulation in certain brain areas. Such phenomena may, indeed, indicate more than mere anæmia of brain areas, for it is found that when there has been marked impairment of cerebral function, such as is implied in persistent forgetfulness, it commonly indicates destruction by softening of the area implicated. The softening is the result of cutting off of the blood-supply to the area affected, and I believe this often begins in local vessel spasm and is succeeded by local thrombosis. When such phenomena are temporary, the fact implies temporary diminution in the supply of nutriment to the area involved. The evanescent character of the phenomena, associated as it frequently is with tendency to recurrence, must be regarded as due to variations in the supply of blood to the affected area; while the permanent loss of function implies permanent arrest of blood-supply, and, as an inevitable consequence, necrosis and softening of the area affected. Even when such softening has occurred, there may be added to the permanent symptoms accessory phenomena which appear and disappear from time to

time. Such accessory phenomena may be a feeling of being dazed and of entire inability to attend to business of any kind. These accessory phenomena may, as has been said, pass off in the course of some days, and may only reappear at intervals of weeks or months. They are also, in my opinion, to be explained by the occurrence of impaired blood-supply to regions which may adjoin the softened area, although they may, of course, have a totally separate site. It is on this hypothesis that satisfactory treatment of such cases is carried out. The aim of treatment is to reduce the irritability of the vessel wall which leads to such local spasm. In some cases, no doubt, such temporary diminution of blood-supply as is here pictured may be the result of periods of special heart enfeeblement associated with marked atheroma of cerebral vessels; but in my experience this only accounts for the phenomena in a limited number of cases, the phenomena being more commonly met with in cases in which there is no question whatever of such heart enfeeblement as would lead to insufficient supply of blood. The following case of James B. illustrates these contentions:

Case 22.—J. B., aged sixty-two, an umbrella-maker, was sent by Dr. Calder. He was accompanied by his wife. He complained of being continually sleepy, having no energy, and being very forgetful. This condition had lasted nearly a year, and had been preceded by severe epistaxis. The bowels moved daily. He had been a hearty eater, but had never taken alcohol.

His wife informed us that during the past year he had had three attacks of being dazed and unfit for work; the two first had lasted for ten days, while the one he was at present suffering from began a fortnight previously. She

stated that his mind was never quite clear between the attacks. On questioning him he said that he used to read a good deal, but that he had given up reading for the past year. The arterial pressure was 220 mm. Hg, and there was a small amount of albumin in the urine.

My interpretation of the phenomena in this case is as follows: About a year ago this patient had an attack in which he was dazed and unfit for work; the attack lasted for ten days, and was preceded by severe epistaxis. The arterial pressure when he came to hospital was 220 mm., and there was a trace of albumin in the urine. What happened at the time of his attack was that his systemic arteries were experiencing a wave of hypertonic constriction; that a corresponding condition was present in at least part of his brain, producing symptoms referable to mental areas, as indicated by his being dazed and stupid; and that this had lasted long enough to allow of softening to occur in one of those areas. He made a partial recovery as the brain vessel spasm subsided, but the recovery was only partial, as the area of softening remained. He was able to attend in some degree to his business, for he had no motor paralysis, but his memory was impaired and he had lost his former energy. Since that first attack he had had two other attacks in which he became dazed and was quite unable to attempt to go to his place of business. The attack from which he was suffering when we saw him had lasted longer than the two first. These attacks mean, in my judgment, a recurrence of the constriction of brain vessels, and each attack, if it leads to a fresh area of softening, will leave him on a lower mental level than before.

This, to my mind, is not only the picture of what has been happening in this particular brain, but it represents

common occurrences and experiences, and the great advantage of having a correct moving and living picture is that in many cases of the kind the brain angiospasm can be stopped and your patient saved from cerebral softening.

Temporary Losses of Ability to Perform Ordinary Work.

Another set of phenomena is illustrated in the case of a patient, J. W., to whom fuller reference is made later. This patient was thirty-one years of age, and was a joiner by trade. His mother stated that for nine years he had from time to time suffered from "stupid" attacks, characterized by inability to perform the most ordinary procedures necessary for doing his work as a joiner; he would, for instance, be incapable of taking the simplest measurements necessary to the carrying out of simple pieces of work. A similar mental inability to deal with other matters showed itself. The attacks lasted for two or three days, and gradually wore off, and as they wore off he began again to understand things and to be able to do his work. He also showed that, while he recognized his mother as "mother," he could not understand how it was she was his mother. Notwithstanding this strange obscuration of his mental processes, he was able to go about as usual, and would even go to his workshop with a view of working; but even when put to the simplest of operations he could not carry them out. These attacks have come and gone, some of them having been short attacks, others of them having lasted longer. In this case, again, it seems to me difficult to explain the phenomena otherwise than by attributing them to local impairment of nutrition in the areas affected, and that local vessel spasm alone enables us to form a tangible view of the phenomena.

Merely to give the phenomena the designation of being epileptoid, again, seems to me to lead us nowhere. The following is the more detailed account of this case:

Case 23.—J. W., aged thirty-one, was sent to me on February 11, 1914, by Dr. Bowie with a note saying that the patient had an attack about a year and a half ago similar to that from which he was at present recovering.

We found it impossible to get a satisfactory history from the patient himself. His mother, who accompanied him, gave us the following history—namely, that about nine years ago he began to have attacks in which he was stupid in ordinary work; he could not even take ordinary measurements. The attacks lasted for two or three days and gradually wore off, and as they wore off he again began to understand things and to be able to do his work. His father died in July, 1912, and in the following October he took an unusually bad turn and did not go to work until January. His mother informed us that during that time he walked about and saw her, but would say to her that he did not understand she was his mother; he at that time could not be made to understand anything in connection with any papers that required to be signed bearing upon his father's affairs. He gradually got over that attack and was pretty well during all 1913, but on the Monday before he was sent to see me he went to the joiners' shop in which he worked, but was quite unable to do the simplest operations necessary for his work.

Epileptoid Attacks simulating Fainting Attacks.

The following is an example of epileptoid attacks resembling fainting:

Case 24.—L. S., a young woman, was sent to me by Dr. Deuchars with a history of continually having faint-

ing attacks, the nature of which was not apparent, and she was sent for my opinion. Her employer was much interested in her, and was anxious to know if he could do anything for the girl.

The story we get from the patient of her attacks is that in the forenoon she feels sick, loses control of her muscles, and sometimes falls. These attacks come on quite suddenly with a feeling of extreme nausea and pain in the head in the right temple region. She never vomits. She has on two occasions suffered from concussion as the result of knocking her head upon the floor. The lesser attacks last for about a quarter of an hour, as she has been told. She is so upset by the attacks that she has to rest the whole day thereafter. The attacks are more apt to come on when menstruation is due, but they are not confined to that time. The girl's colour was good, and there was no appearance of anæmia. The heart sounds were closed and distinct. The pressure was 144 mm. The bowels were kept regular by means of a laxative, and there was no albumin in the urine.

You have had lectures on the grave and the minor forms of epilepsy, and I only indicate to you at present that epileptoid phenomena sometimes respond to vaso-dilators, and that there is a vessel theory of epilepsy.

Vertigo due to Brain Angiospasm.

The following two cases illustrate this relationship:

Case 25.—J. W., aged forty-nine, a labourer to an electrical company, recommended by Dr. Hamilton. Admitted January 23, 1914, complaining of giddiness of ten weeks' duration. The patient drinks a good deal of beer, and smokes 4 ounces of tobacco weekly. He has a

comfortable home, and has a family of eight children alive and well.

History.—Ten weeks ago he began to suffer from giddiness, which came on after stooping and was very severe. In one day he had three attacks of a sensation of coldness and numbness on the left side of the body, including the face, the trunk, and the limbs. He at the same time experienced a feeling of nausea and a salt taste in the mouth. On one occasion he fell. His bowels, which as a rule act well every day without medicine, had been acting sluggishly prior to the onset of the attacks referred to; there was no vomiting, but he said his sight was bad and he had a slight headache; the giddiness was worst four days after the attack of numbness.

After two weeks' illness he was admitted to the Hawick Hospital, where he stayed for five weeks. Since then he has been two weeks at home, when at the request of Dr. Hamilton he was admitted to the Royal Infirmary. His sight had improved a little, but he was still subject to threatenings of attacks similar to those described. He said, however, that he was able to avert them by lying down, when after ten minutes he felt perfectly well again. He had these attacks as a rule once or twice a day, but sometimes missed a day. He stated that the attacks began with a gripping sensation round the left upper arm.

Condition on Admission.

Nervous System.—Intellectual and mental functions satisfactory. He has slight headache in the frontal region, and also pain shooting along the vertex. He still suffers from giddiness, which is so marked sometimes that turning in bed to use the spittoon may make him feel as if he would fall out of bed. Voluntary movement is in no

way affected. Sensibility to touch, pain, and temperature are normal. Reflexes are normal; there is no incoordination and no Rombergism, although the patient says that when first tested for this by his own doctor he was very unsteady.

Special Senses.—The pupils are normal, and react to light and accommodation. Vision is good; the patient can read, and the discs, as reported on by Dr. Sim, show a little obscuration of the margins within physiological limits, and the vessels did not appear to be in any way abnormal. The patient was a little deaf on the right side. Dr. Logan Turner, who kindly examined his ear condition, reported that the patient had evidence of old-standing middle-ear disease; that he had a large perforation and some moisture on the right side, and this ear had discharged for many years. Dr. Turner further stated that the tests went to show that the deafness was entirely of the middle-ear type, and that the inner ear was not affected.

Circulatory System.—The pulse varied from 68 to 80 or more, and was regular in force and type. The arterial pressure was 140 mm. The apex of the heart was in the sixth interspace, 1 inch outside the nipple line; the sounds were pure.

Respiratory System.—There is a history of winter cough for some years. The lungs were normal.

The abdominal organs were normal. The urine was of acid reaction, specific gravity 1025, and showed no abnormal constituents.

He was treated with 10 grains of potassium iodide three times a day. On February 6 the dose was increased to 20 grains three times a day. On February 9 he had greatly improved, the headache and the giddiness having

completely disappeared, and he was allowed to get out of bed. He was discharged on February 14, having showed no recurrence of his symptoms. On February 10 his arterial pressure was 110 mm., and his pulse was between 72 and 80.

The preceding facts suggested that the phenomena might be the result of brain angiospasm. The fact that the vertigo had been preceded by such marked disturbance of sensation on one side of the body from the face downwards, and that this abnormal sensation had appeared and disappeared several times in the course of one day, suggested this view. The action of potassium iodide in counteracting the tendency to vessel spasm seems to me from clinical experience to be definitely established. In this case the effect of the drug on the patient was eminently satisfactory, and my contention with regard to its action is supported by the fact that with the disappearance of the cerebral phenomena the pressure was found to have fallen from 140 mm. to 110 mm.

Case 26.—Mr. W., aged sixty-seven, a solicitor, was sent to me on February 12, 1914. He complained of attacks of headache, referred to the back of the head, and of giddiness, which might or might not be accompanied with vomiting. These attacks came on if he applied his mind very closely to his work, if his work worried him, or if he became excited in discussion or controversy. The giddiness might be so pronounced as to compel him to stop work and to go home in a cab. Rest led to disappearance of these symptoms.

The pulse was 60, and the arterial pressure was 110 mm. On lying down the pulse was definitely fuller than when sitting. The vessels were soft. The heart at the apex was in the mammary line; the sounds were fairly good

and closed. The abdomen presented no abnormality; to regulate the bowels apenta was taken regularly. The urine presented no abnormal characters.

In this patient the headache and giddiness were primarily cerebral, and were to be regarded as the result of disordered cerebral circulation, due to too close mental application, or to emotional influences. There could be no doubt that there was an imperative call for complete mental rest away from the patient's ordinary environment. Here, again, on the hypothesis of local cerebral angiospasm being the cause of the phenomena, the danger was not that in one of these attacks cerebral hæmorrhage would occur, but that the angiospasm might be so pronounced and of sufficient duration to lead to cerebral softening in the more purely mental areas of the brain.

CHAPTER X

HYPERTONUS IN NORMAL ARTERIES. A HIGHLY SENSITIVE AND RESPONSIVE LIVING MECHANISM

In this chapter hypertonic contraction, as it occurs in normal arteries, is to be considered. This part of our subject is of great interest and of much importance. Its importance and interest follow upon recognition of the fact that the arteries with muscular coats, while possessing a normal tonus, are liable to become hypertonic. This means that the artery is unduly contracted, somewhat smaller and thicker to the sense of touch in the finger applied to it; that this necessarily implies a diminution of the lumen of the vessel and a fall in the blood-pressure inside it. It will be seen later that this hypertonic contraction can be caused by a number of agencies, and these will be separately examined. What is at present sought to lay emphasis upon is the frequent occurrence of this phenomenon: that it is always associated with a relative rise in sphygmometer reading, and a fall when the condition determining it is removed. It is sought here to show that this arterial movement is one of the most, if not the most, sensitive and responsive *living mechanisms* in the body, and that it provides a special channel by which information can be obtained of what exactly is happening in our patient when he seeks our expert assistance for his relief.

The following cases illustrate various aspects of this phenomenon; but it is necessary to state that sphygmometer observations were made in them, sometimes because a radial artery felt hypertonic, or from mere curiosity to know what the brachial pressure was. It was not until such records had accumulated that the far-reaching significance of the observations became strikingly apparent.

A.—Varied Phenomena and Hypertonus.

CASE 27—*Migraine, Palpitation, Constipation.*—Mrs. G., aged forty-seven, was seen on September 5, 1917. The complaints were palpitation on slight exertion; a bad attack of migraine weekly, so that she had hardly recovered from the effects of one attack before another came on; and constipation, the bowels only acting when she took medicine. She had for some time been taking Epsom salts every morning without any improvement in the other symptoms. The pulse was small and contracted; the brachial pressure was 125 mm. Hg. The heart was normal in size, and there were no murmurs. The urine contained neither sugar nor albumin. I prescribed a laxative to be taken every night instead of the Epsom salts; 10 grains of phenacetin as soon as the migraine threatened, and to lie down for half an hour; if not relieved to take a second dose of 10 grains, and to lie down for another half-hour or longer.

She came to see me again on September 21, when she assured me she had not felt so well for years; she had had no attack of migraine, and half a pill at night had been sufficient for the bowels. The radial artery was softer and larger, and the pulse-wave larger. The pressure was

105 mm. Hg. The patient was relieved of her miseries, and the brachial pressure had fallen 20 mm. Hg.

She was seen in April of the following year, when the improvement had continued. To suggest that this fall of 20 mm. represents a fall of 20 mm. of blood-pressure inside the brachial artery is to my mind absurd. This is a record of hypertonic contraction of the brachial, and of its relaxation when suitable measures were taken for the relief of the condition.

CASE 28—*Gastric Case.*—Mr. C., aged thirty-four, was suffering from hypochlorhydria and air-swallowing, and no constipation. This case has been published elsewhere in full. Before treatment the brachial pressure was 145 mm. Hg; after treatment, which was curative, the brachial pressure had fallen to 115 mm., a fall of 30 mm.

CASE 29—*Constipation.*—Mr. S., aged forty, consulted me in 1920. He complained of flatulence, of feeling heavy and depressed, and of constipation. The tongue was clean and moist. The heart was normal; the urine precipitated phosphates, and gave a deep ruby ring with nitric acid. The pressure was 125 mm. Hg; the pulse-wave was poor, the vessel hypertonic. He was given glycerophosphates and formates, and as a laxative phenolphthalein. He was seen a month later and was much better, the bowels acting satisfactorily. The ruby ring in the urine was much less. The pressure had fallen to 115 mm. Hg.

CASE 30—*Functional Angina Pectoris; Tobacco and Constipation.*—Mr. R., aged fifty-two, consulted me in July, 1917. He complained of attacks of precordial pain brought on by slight exertion, and from which he had suffered for three years. His radial artery was thick. The tongue was coated, and he suffered from constipation.

The heart was normal in size and in its sounds. The brachial pressure was 140 mm. Hg. He was not a big feeder, but he did business with farmers, which led to drinking a good deal of whisky. He also smoked a great deal. He was strongly advised to lessen or stop the consumpt of tobacco, and to diminish the quantity of whisky he drank. I thought that his vessels were more hypertonic than sclerosed, and that probably the condition was due rather to tobacco and constipation than to whisky. I gave him a mild laxative pill and a mixture containing iodide of potassium, bicarbonate of soda, and tincture of belladonna. He was to return to see me if he did not improve, but he has not yet reported himself.

CASE 31—*Highly Nervous Patient; Rheumatic Nodules; Constipation.*—Mrs. G., a middle-aged lady, consulted me in August, 1917. She complained of extreme nervousness, and was subject to recurring periods of great nervousness and restlessness. There were nodules on the fingers. The tongue had a thick white fur, and she was constipated. The brachial pressure was 170 mm. Hg, and the apex of the heart was considerably beyond its normal position. There was no albumin or sugar in the urine. She went into a nursing home, and under rest and treatment rapidly improved; the nervousness and restlessness entirely subsided, and the pressure fell to 120 mm., a fall of 50 mm.

CASE 32—*Feeling of Beating and Fullness in Head; unable to do Mental Work; Easily Tired.*—Mr. E., a student, aged twenty, consulted me on account of a feeling of beating and fullness in neck and back of head, and that it rendered him unable to study when these feelings came on. He was also easily tired. The tongue was coated posteriorly, the bowels moved freely, the

appetite was good, and he slept well. The brachial pressure was 135 mm. Hg. The heart was normal. The urine contained no albumin, but gave a marked colour ring with nitric acid, and precipitated a considerable amount of phosphates on heating. I prescribed a mild laxative pill to be taken nightly, and a mixture containing small doses of sodium salicylate, tincture of nux vomica, compound tincture of rhubarb, and compound infusion of gentian. He rapidly got well; the tongue cleaned, the phosphates disappeared from the urine, and nitric acid gave only a faint ring. The pressure had fallen to 120 mm.

CASE 33—*Cramp in Leg; Heart Thumping and Rapid.*—Miss E., aged fifty-three, was brought to me by her doctor in July, 1910. She had suffered from severe "cramp" in one leg, which her doctor rightly interpreted as due to arterial spasm, as during the attacks he could not feel the pulse in the posterior tibial artery. I saw her after a course of iodide of potassium and sodium. The urine contained no albumin. The heart's action was thumping and rapid. The brachial pressure was 160 mm. Hg. I advised that the iodides should be continued, that the diet ought to be spare, and that the daily easy action of the bowels should be attended to.

I heard of her improvement, but did not see her again until 1913, when the heart was acting quietly and steadily and the brachial pressure had fallen to 120 mm. At the time of writing this (1920) the lady keeps well, and is fully able for her domestic and social duties.

CASE 34—*Heart-Strain, with Attacks of Angina Pectoris Minor on Exertion.*—Dr. D., aged forty-nine, was suffering from recurring attacks of the minor variety of angina pectoris when he made any physical effort. This neces-

sarily became an impediment to his usual activities. The heart had been pretty severely tried, and was showing signs of resenting it. The pulse was 80 and the brachial pressure 125 mm. Hg. On one occasion he had an attack in my room, during which the pulse fell to 60 and the pressure rose to 145 mm. Under rest, a mild laxative, and a mixture containing iodide, nux vomica, and rhubarb, he got rid of his symptoms, and has continued at work during the last nine years.

CASE 35—*Dull, Heavy, and Wanting in Energy; Albumin in Urine for Twenty Years.*—Mr. R., aged forty-two, consulted me complaining of feeling dull, heavy, and wanting in energy. The appetite was poor and sleep indifferent. He was not married, and was an abstainer from alcohol and tobacco. He used a compound aloin pill for the bowels, which were usually constipated. He knew that for twenty years there had been albumin in the urine. The radial artery was a little thick; the heart was sound and showed no enlargement; the brachial pressure was 140 mm. Hg. The urine had a specific gravity of 1020 and contained albumin. I treated him with iodide of potassium and tincture of squill. He rapidly improved, and lost his sense of mental and physical languor. With the improvement in his symptoms the pressure fell to 125 mm. The albumin in this patient was evidently not the result of a chronic nephritis, and was to be regarded as belonging to the orthostatic group. I saw some months ago a corresponding case, a man who had led a strenuous life in Egypt and had been declined for life assurance twenty years ago because of albumin in the urine. During those twenty years he had not only led an arduous life, but had come through more than one very severe illness. The albumin had persisted, and was

present when I saw him; but there was no arterial sclerosis, although both his heart and his nervous system required rest and building up.

CASE 36—*Unsteady when Walking.*—A clergyman, aged seventy-two, consulted me in the end of April, 1915. He complained of feeling unsteady when walking, and that the objects about him appeared to be unsteady. The bowels tended to be constipated. The urine contained neither sugar nor albumin, but gave a marked red-purple ring with nitric acid. The brachial pressure was 175 mm. Hg. A pill containing cascara and taraxacum was prescribed to be taken nightly, and a mixture containing 5 grains of iodide of potassium to each dose to be taken thrice daily with meals. He was seen again twelve days later. The bowels had moved easily with the pills; he had quite lost the feeling of unsteadiness, and the eye phenomena had gone. The pressure had fallen 35 to 40 mm.—to 135 to 140.

A year later he reported himself. There had been no return of the symptoms, and the brachial pressure had evidently kept down, for it was 135 mm.

He continued in active work and was keeping well, although by 1920 he had reached the age of seventy-seven, and was finding that he had not the physical endurance of earlier life. This patient was an abstainer from both alcohol and tobacco.

CASE 37—*A Keen Brain-Worker, breaking down from Time to Time.*—This patient was a lawyer, aged sixty-two. He was a very hard and keen worker in his profession, taking a hand in many things. From time to time he broke down and had to stop his routine duties. He consulted me in April, 1919, when he was on the verge of another break. The bowels moved daily by means of a

small dose of saline. The heart and urine were normal. The brachial pressure was 170 mm. Hg. Iodide of potassium in 5-grain doses was prescribed to be taken three times a day with meals. He was seen again in sixteen days, when he reported himself as feeling much better and surprised at the speedy relief he had obtained as compared with previous experiences. The brachial pressure was 135 mm., a fall of 35 mm., under the influence of iodide alone. There was no question of alcohol or tobacco in this patient.

The foregoing cases have been selected, out of many others, with a view to illustrate this side of our subject, and to indicate the wide field of interest it covers. Mere multiplication of examples would not be any more illuminating.

B.—Tobacco-Smoking and Hypertonus.

Tobacco-smoking in excess for the individual gives rise to a variety of symptoms, more or less alarming as the person affected is or is not able to interpret his symptom aright. It is not proposed to refer in detail to individual cases, some of which occurred in professional brethren whose habits in all other respects were known to be above suspicion of excess. Many of them could be regarded as pure cases of tobacco overuse. The symptoms noted have varied somewhat, but the predominant type has been precordial pain coming on as a result of physical effort hitherto performed with ease. The severity of the symptom varies, but it may be sufficiently severe to give rise to considerable anxiety, and may come on at night and prevent sleep. Another symptom is breathlessness on exertion, which may be accompanied by precordial

pain, or even heart intermissions. In still rarer instances there is giddiness, not of a pronounced degree, but sufficient to cause considerable discomfort. When cases of this kind are examined, the radial is found hypertonic, thickened in some measure, but not sufficiently to indicate a markedly sclerosed vessel, while the duration of the symptoms and the previous history suggest mere hypertonic contraction. The heart is not enlarged, but the first sound has the jerking character which suggests tobacco. The urine is normal. There may or may not be a tendency to constipation. The brachial pressure in these cases is not above 160 mm. Hg, and may be considerably lower. The mere figure of the sphygmometer reading must not be acted upon; a reading of 140 mm. may not be an absolutely high figure, but it will be found that, under treatment appropriate to the condition, such a reading may fall to 120 mm., while the symptoms have disappeared. When treatment is successful a fall of from 15 to 20 mm. occurs, and the radial artery loses all or most of its thickening. I have seen a case in which the pressure was only 130 mm.—not a high pressure for a man about sixty years of age—but when symptoms were corrected the pressure was only 115 mm., and the little hypertonic thickening of the radial disappeared.

Treatment in all tobacco cases, more especially the pronounced ones, ought to be to stop the use of tobacco for a time. The symptoms are also materially alleviated by the administration of the iodides and belladonna.

C.—Epileptoid Cases.

Under this subheading five cases are recorded, or referred to, which were characterized by epileptic or epileptoid seizures; in only two, however, were sphygmometer

observations made. The other three cases were seen before the sphygmometer was in use, but there is no doubt that in them also the instrument would have provided the same kind of information, had it been in use, which it provided in the first and second cases. These cases are not common, for the five mentioned here are perhaps all I have seen during the last twenty years.

CASE 38.—Mrs. H., aged sixty-five, was sent to me in March, 1911. The history was that during the previous year she had four fits, evidently epileptoid in character. After the last fit she had remained unconscious for an hour. The face was florid and showed injected venules; the tongue was coated; the teeth were very bad; the bowels were regular; the urine was small in amount, and contained neither sugar nor albumin. The heart's apex was slightly beyond its normal position. The brachial pressure was 150 mm. Hg. In view of the general condition, 5 grains of salicylate of sodium and 5 grains of potassium iodide were prescribed to be taken twice a day. The result was that very free diuresis was set up, and the pressure fell to 125 mm., and she felt much better than she had felt for a long time. The remains of teeth were removed and an efficient denture provided.

I heard of her regularly until the middle of the war period, and she had remained quite well.

CASE 39.—E., a clergyman, aged forty-four, consulted me in the end of July, 1911. The history was that in the end of April he had " fainted," and was unconscious for three hours, and then slept. There was jerking of legs, arms, and face. During the four years preceding that attack he had had three or four short " faints " in which he fell, and might bump his head, but he got up almost immediately. Since the April attack he had

several minor ones in which he fell. There had been no involuntary passing of urine or fæces in these attacks. The radial artery was large and thick. The heart was normal. The tongue showed a dryish white fur. He wore artificial dentures, and the gums were sound. The bowels tended to be constipated. The urine contained neither sugar nor albumin. The brachial pressure was 145 mm. Hg.

He was seen again on October 4, after a long holiday. He looked much better, and he had had no more "faints." The tongue was moist and red. The brachial pressure was 125 mm.

He continued his holiday until October 20, when I again saw him. He was feeling much better, and was anxious to return to work. The brachial pressure was 115 mm., a fall of 30 mm. since I first saw him three months before.

The other three patients were seen before the sphygmometer was used in medicine. In each of the three there was a distinct history of one or more epileptic fits of recent occurrence. One was a lady over sixty, another a male about fifty, and the third a young married lady who had her first baby. In all three, in spite of a very gloomy outlook on the part of their medical attendants, I believed that they were due in the first case to extraordinary inattention to intestinal evacuation, in the second case to a like cause in a man leading too sedentary a life, and in the third to a gastric upset caused by some unsuitable food. They all three not only recovered, but they are still alive, and have had no return of the condition, except once in the case of the older lady, from recurrence of inattention to the bowels.

CHAPTER XI

INTERPRETATIONS—A NEW REVELATION —ETIOLOGY AND CONCLUSIONS—TREATMENT

Interpretations.

FROM the facts recorded in the preceding chapters it is submitted that the sphygmometer ought to be given in clinical and practical medicine a very high place. Its practical value lies not in providing an assured record of heart power in terms of "blood-pressure"; that view has to be definitely abandoned. The varying record it supplies in the same patient is for practical purposes a record of the condition and state of the arterial wall. Of course there is blood inside the artery, and so long as a pulsatile wave passes along its channel there is a "blood-pressure" inside; but that may be approaching zero when a manometer pressure of over 100 mm. Hg is required to flatten the artery by means of the armlet, so as to arrest the passage of the pulse-wave. The millimetres of internal pressure are trifling in such cases. There are other cases in which there are permanently thickened arteries with a permanent high pressure due to the state of the artery wall, plus a well-sustained blood-pressure; but these thickened vessels often show an extraordinary range of contractility, readily tightening up, becoming in their hypertonic spasm like spastic muscle. Such vessels provide a range of sphygmometer reading from 175 to 295 mm., a range of 120 mm.

or even more. This range is due entirely to vessel wall and to altered lumen consequent upon the angiospasm. When a tube like the brachial artery contracts its lumen the pressure of the flowing fluid inside it falls, while the pressure required to stop the wave in the contracted artery rises. Increase of internal pressure is proximal to the area of contraction. Experimental physiology measures blood-pressure by a cannula in the aorta, and speaks and writes of it in that sense; the clinician has to judge of it primarily by his sense of touch in the arteries of the limbs, while he also obtains valuable information by examining the heart. It is this contracting and relaxing, this play of tonus, in thickened vessels which the sphygmometer records, and which is of incalculable clinical value. The teaching of the thickened artery as a rigid tube has to be abandoned, and all the rigid ideas associated with it.

A New Revelation.

The sphygmometer has, however, done more than this or practical medicine, and for the physiology of medicine (and true medicine is based on true physiology; in neither do half-truths work out satisfactorily). In the chapter on hypertonus in normal arteries records are given of cases taken from my private case-books, containing observations spread over the years since the sphygmometer became a clinical instrument. The sphygmometer observations were merely part of a general medical examination, while no doubt often prompted by my finger's appreciation of hypertonic radial arteries and a legitimate curiosity to test my finger's dependability. When specimen cases are grouped together it is evident that this new clinical instrument makes an important new revela-

tion to us. It seems to establish beyond reasonable question that the *play of tonus* in the arterial wall represents a highly sensitive and responsive living mechanism, and reveals a delicacy of circulatory responses and adjustments we had not visualized, and so failed to apprehend that observations made by the sphygmometer have been often unintelligible, and thrown aside as such. A doctrine of averages arose here, as in so many other departments of medicine. In middle life a sphygmometer reading of 140 mm. or more was looked upon as average, and therefore as having no connection with or relation to the symptoms, of whatever kind they might be, of which a patient complained. The instrument has revealed a new and valuable source of information, which has nothing to do with questions of averages of ages and pressures. Tightened-up arteries with a pressure of 140 to 160 mm. always carry a lesson. Reliance on averages, and making averages a serious factor in diagnosis, is as futile in this department as in the age-incidence of cancer of the stomach or of pulmonary tuberculosis when we are face to face with individuals as patients. The sphygmometer must not, however, be given an undue place in practical medicine. There is always a desire for methods which give absolute guidance to diagnosis and prognosis, but the number of these is limited. Accurate diagnosis and prognosis will always depend upon the judgment which is directed to the interpretation of available facts. The sphygmometer has made an important and far-reaching revelation to us; the use we make of it in individual instances will depend upon the completeness of our grasp of a great practical physiological fact, and on the judgment with which we can utilize it when physiology is overtaxed and overburdened and seeks medical assist-

ance. So much is said to explain how no rules, beyond the indications given in the preceding chapters, ought to be laid down for the interpretation of sphygmometer readings. Rigid rules applicable to every case cannot be formulated. Rising brachial pressure is no necessary part of advancing years, and if that fact is fully realized, certain inferences inevitably follow. There is no escape from these inferences and their progressive results, save by acting on the recognition of their inevitableness.

Etiology and Conclusions.

The question of the etiology of arterio-sclerosis, in some of its aspects, has been practically answered in some of the earlier chapters, but it requires somewhat more detailed examination. In this connection we are in a position to recognize the beginnings, and to trace the progress of the changes up to the fully developed products. The beginnings are in the hands of every practitioner who can remould or apply his understanding of pulse and arterial wall, and give up thinking of sphygmometer readings as "blood-pressure." Schemes for the study of the beginnings of disease are at present being boomed, and here is a wide field for observation, which only requires a sphygmometer and judgment to use it aright; and a field which more or less includes the whole community.

The fully developed picture of arterio-sclerosis is the result of two sets of antecedents—(1) gastro-intestinal errors and (2) syphilis. The latter is sometimes associated with the former, and then it is difficult to say which is the predominant factor.

Gastro-intestinal Errors.—These embrace a variety of conditions, the consideration of which implies an approxi-

mately correct knowledge of foods, of the processes of digestion, absorption, assimilation, and excretion. To all this has to be added the factors which form the individual equation. There is no escape from this problem. Apart from mere idiosyncrasy to certain foods, there is a very wide margin of individual capacity with regard to the amount and kind of food the stomach can deal with satisfactorily. It has been shown that disordered stomach function may lead to arterial hypertonus, which ceases when the proper remedy is applied to the gastric disturbance. A like hypertonus may be removed by a suitable laxative, mere clearing out with saline not necessarily being effective. But there are, on the other hand, the physically vigorous men, leading a strenuous life in business, big feeders and fond of red flesh, who do not know they have a digestive system apart from the conscious satisfaction of eating a big meat meal, and the need for an occasional purge to counteract a "bilious attack." Such is the history of men with advanced arterio-sclerosis who are permanently breaking down between fifty and sixty years of age. In the same type, but on a lower platform physiologically at the same time of life, are men who seek advice and under direction modify their mode of living, become very moderate in eating and drinking, and attend more carefully to regular action of the bowels if that is required. These two divisions of the same type are of special interest because of the questions they raise as to the steps between the big intake of food and the blood condition which produces a steady arterial hypertonus with a steadily increasing myocardial hypertrophy to compensate for the arterial constriction. There is no defect or deficiency in digestive ability. Such people may never have known

what indigestion means. The chemical stages have all the appearance of being performed perfectly, and nutrition is well maintained. Herter's view was that the excess of digested material was not absorbed, and, not being absorbed, readily fell a prey to the putrefactive organisms normally present in the intestine, and that the toxins thus produced were absorbed and acted injuriously. The same doctrine of putrefactive organisms acting on retained excrement in constipation is used to explain many symptoms. There is reason in this, but the doctrine, like the word *neurasthenia* or *neurosis*, is a cloak to much ignorance and to not a little charlatanry mixed with fanaticism. While *auto-intoxication*, from the bacterial standpoint, has its own place, it tends to be placed out of perspective. In itself constipation, which is a colon retention, and may be due to atony or spasm, has a reflex action through the sympathetic or para-sympathetic nervous system upon the vascular system, especially of the brain. The clinical proof of that is the sudden relief to brain heaviness and dullness immediately the lower bowel is emptied in some people who suffer now and again from constipation. A further aspect of this bacterial auto-intoxication doctrine is the fact that the liver has the power of dealing with so many injurious products that one's appreciation of its great scope and its great margin of power increases rather than lessens. To me a still more important part may be played by the steady absorption of more digested material than is required for nutrition; for it falls to the liver to dispose of the excess. This means a continuous strain, and the using up of substances required for the efficient removal of the normal waste of the body. It is, I think, probable that there is a steady acidosis under these

circumstances. Dr. Sellards, of Harvard Medical School, has shown that this is definitely present in the phase of arterio-sclerosis with angiospasm and dyspnœa—that is, of "uræmic or cardiac asthma." This may quite possibly be the process slowly but surely acting and ending in the arterial thickening and irritability which characterize the fully-developed malady. Mere abstinence from red flesh may have the effect of materially reducing a persistent hypertonus, while the relief of constipation fails to act as effectively. Valuable indirect support to the influence of a rich protein dietary is provided in a paper by Dr. C. R. Box, published in the *British Medical Journal* of March 13, 1920, in which he records the successful treatment of tubular nephritis with dropsy by a protein dietary after Epstein's method. Copious diuresis was established, with complete disappearance of dropsy. During the treatment the pulse pressure steadily rose and reached 220 mm. Hg. That the steady use of alcohol or of malt liquors operates along the same chemical lines will be more readily accepted. Yet with regard to both food and drink it must be restated that some persons can take quantities without discomfort which would make other persons ill. One test at least can be applied by the use of the sphygmometer, but I think alcoholic liver cirrhosis may occur without arterio-sclerosis.

Nervous System.—The influence of the nervous system does not require much space to deal with it. That the vessels are acted upon by emotion through the medium of the nervous mechanism is beyond question. It may be that the same mechanism operates in the people who live intensely, and that it is more responsive in the big protein feeders than in spare feeders; but I do not

think that mental stress by itself can produce arterio-sclerosis.

There is, however, another aspect of this question—namely, this: granted the absorption of bacterial toxins from the intestine to be an important factor, do they act through the nervous mechanism, or directly on the arterial wall through the agency of blood composition? Personally I hold to the latter view, and have dealt with it fully in my former book. That constipation may affect the vessels reflexly has been already referred to; and there is, of course, no doubt as to the active reflex between the splanchnic system and the systemic vessels. There seems to me to be little doubt that arterio-sclerosis is the result of long-continued blood conditions, due to errors in diet, digestion, intestinal excretion, or to the absorption of an excess of digested protein food impairing the efficient removal of the normal waste material of cell-protoplasm activity.

Treatment.

Details of treatment in many of the cases recorded in previous pages have been given, so that any elaborate disquisition seems to be unnecessary. If the broad lines of the causes of hypertonus in arteries, either sclerosed or normal, have been followed, it is evident that relief is to be obtained by removing the cause, and prevention by avoiding the cause.

In bad cases the main remedies are physical rest, low diet, daily bowel evacuation, radiant-heat baths, erythrol tetranitrate to overcome special symptoms due to hypertonic spasms, spirit of nitrous ether, the iodides and alkalies combined with squill if the heart requires stimulation. In extreme cases blood-letting is useful. In milder

cases, or when normal vessels are hypertonic, my favourite laxatives are phenolphthalein, cascara sagrada, and taraxacum; the first may be given in tablet, the other two together in pill or in liquid form as liquid extracts combined with liquorice or glycerine, or the three may be given together in pill. Belladonna and nux vomica may be added to either form. Purgation is to be avoided save in the urgent cases.

A few words of warning ought perhaps to be added with regard to old people with sclerosed arteries. In many such cases there is no call for interference. Medical interposition ought to be confined to advising as to diet and bowel evacuation. If symptoms emerge pointing to brain angiospasm, small doses, which may be increased if necessary, of erythrol may often be given with great and speedy benefit. In some cases spirit of nitrous ether is sufficient. Stricter attention to diet is indicated, and a daily laxative is often necessary. Phenolphthalein is the laxative most commonly useful in old people.

CHAPTER XII

THE ESTIMATION OF HEART POWER AND THE RELATION OF THE RIGHT SIDE OF THE HEART TO OVERSTRAIN*

THE estimation of heart power is so closely associated with the questions dealt with in the preceding chapters that it is appropriate to deal with it shortly here. In view of what has been said in a previous chapter, it follows that the time-honoured practice of feeling the pulse-wave is the first step towards forming an estimate of heart power. The vigour and force of the wave inside the artery, no matter what the condition of the arterial wall may be, is readily estimated by the educated finger when mixed ideas of " tension " are put aside. A feeble, small wave has a feeble left ventricle behind it as a rule, if the artery felt is of average size. From examination of the pulse the next step is the examination of the heart. Its size and its sounds enable us to judge of its power. And here average size and average loudness and character of sounds become the standard by which other hearts are to be judged. The importance of noting the phenomena referred to in the following lecture in cases of heart strain cannot be overstated, and cases of overstrain are so common that the lecture is reproduced here in its entirety.

Gentlemen,—I ask your attention to-day to the consideration of the clinical phenomena which, when correctly interpreted, enable us to arrive at certain conclusions as

* Reprinted from the *British Medical Journal*, June 24, 1916.

to the condition of the right side of the heart. At the outset we must recall the anatomical relations of the right heart. A frontal view of the heart is made up of right auricle and ventricle, and, at its left limit, of a strip of left ventricle about ½ inch in breadth. The origin of the pulmonary artery, from that part of the right ventricle called the *conus arteriosus*, is in the second left intercostal space, while its position, when not thus situated, can often be precisely determined by palpation and auscultation, as these define the point at which the closure of the pulmonary cusps is best felt and heard. The right ventricle lies under the third, fourth, and fifth spaces, extending in the fifth space to within ½ inch to 1 inch of the apex of the left ventricle. The right border of the heart forms an arc the convexity of which is to the right, and which lies in the third, fourth, and fifth intercostal spaces, extending in the fourth to 1 inch or 1½ inches from the right sternal border.

Methods of Examination.

Percussion.

The position of the left and right borders of the heart can be determined by percussion in the different intercostal spaces. The right border is the more difficult of the two to determine, but I have explained to you on previous occasions how it can be done, and I have warned you that a right border represented as a line along the sternum may be the border of the right lung, but cannot be the right border of the heart. The way you must look at this matter is that the right border even of the normal heart is an inch or more beyond the sternum, and that you have to learn to find it. It is often considerably to the right of its normal limit.

Inspection.

Pulsation in the second left space is usually not due to the pulmonary artery, but to the conus arteriosus, and results from the fact that this part of the right ventricle dilates early, and that as it dilates it occupies the space carrying the origin of the pulmonary artery outwards and upwards, so that it may lie under the second rib, and be an inch or more to the left of the sternal border. Pulsation in the third, fourth, and fifth spaces, in the latter of these to within an inch of the apex, is always caused by the right ventricle, and all ideas based upon the phrase "diffuse apex-beat" are erroneous. Such pulsation is a diffuse cardiac impulse, not a diffuse apex-beat.

Auscultation.

In auscultating the heart the site of the apex of the left ventricle is auscultated, first, to determine the intensity of the first sound as produced by systole of the left ventricle; and, secondly, as the first step in determining whether a mitral lesion is or is not present. Having begun with the apex, and passing from it towards the sternum in the fifth, fourth, and third spaces, the loudness, faintness, or absence of the first sound is noted as an indication of the strength of the right ventricle. The old idea that the first sound as heard over the right ventricle was attributable to the left, as the thin-walled right ventricle was too weak to produce sound, has been more or less abandoned. The right ventricle is now credited with at least some part in the production of the first sound as heard over it. I, however, ask you to go further than that. I would advise you to look at this matter from a different standpoint—namely, that the first sound originating in the left

ventricle and murmurs produced at the mitral orifice, as a rule and in the majority of cases, are not passed through the interventricular septum to the anterior wall of the right ventricle. The first sound as heard over the right ventricle is therefore *not* produced by the contraction of the left ventricle. As regards murmurs produced at the mitral orifice, I again impress upon you that, as a rule, these murmurs are not heard to the right of where the ventricular septum forms part of the anterior aspect of the heart. We have frequently in the wards cases—and there are two or more such cases there now—in which loud systolic mitral murmurs are not heard over the body of the right ventricle, while they are well heard outwards and upwards to the left of the apex. Such cases must be accepted as the standard by which we are to judge of the true area of limitation as well as the direction of propagation of mitral murmurs. You must abandon any erroneous views you may have learnt that mitral murmurs are commonly conducted over the whole precordia—conducted even to the base. The rule is that when a systolic mitral murmur is audible at the apex, and a murmur at the same time is heard to the right of the ventricular septum, this latter is a separate and superadded murmur, and is tricuspid in origin. In cases of marked incompetence the tricuspid murmur is often heard so high on the sternum that it may be thought to be aortic in origin. When the murmur thus raises the question of its seat of production, it will be found that on making the patient sit up in bed the murmur will disappear from the manubrium sterni if it is tricuspid in origin, and will reappear when the patient lies down. An aortic systolic murmur does not disappear and reappear with these changes in posture. The presence of a tricuspid murmur

can thus, in the majority of cases, be readily determined. In judging of the right heart, the power to determine with accuracy the presence or absence of this murmur is essential, as it gives important evidence as to the condition of the right heart.

Let me impress upon you the points I have endeavoured to expound to you. They are—(1) That pulsation in the second, third, fourth, and fifth spaces, ½ inch or more within a vertical line drawn through the apex, is caused by the right ventricle; and (2) that you have to differentiate tricuspid from mitral murmur on the lines I have formulated for you.

Abnormal Phenomena.

Visible Pulsation of Right Ventricle.

From these elementary points, which it has been necessary to dwell upon owing to the prevalence of misconceptions, we pass to the consideration of their bearing on the investigation of the condition of the heart. In the dorsal decubitus and in normal conditions the pulsation of the right ventricle is not visible save in persons with exceptionally small and thin chests. Visible pulsation means either that the right ventricle is dilated or that the left heart is dilated and has lifted the right ventricle into closer contact with the chest wall. With regard to the first of these explanations, I would warn you against a too facile belief in dilatation of the right ventricle; as a matter of fact, although various interesting changes occur in the right ventricle, dilatation such as is common in the left ventricle does not occur, for the simple reason that a very strong muscular band passes from wall to wall preventing it. The conus arteriosus readily dilates, and when it does so it carries the origin of the pulmonary artery

outwards and upwards. With regard to the second explanation, it has this important practical significance, that visible pulsation of the right ventricle may be proof of dilatation of the left heart, and herein, perhaps, lies its chief value as a clinical sign.

Accentuation of the Pulmonary Second Sound.

Returning to the auscultation of the right heart, you only require to be reminded that the second sound produced by the closure of the pulmonary cusps may be accentuated in its own area. The accentuation means heightened pressure in the pulmonary artery, and, when primary lung changes are excluded, it means increased fullness, with the consequent degree of increased difficulty, in the pulmonary circuit. Accentuated pulmonary second sound thus indicates the measure of impairment of the left ventricle which has determined the increased difficulty in the pulmonary circuit. Whenever the left ventricle is overstrained some degree of dilatation occurs. This necessarily means an increase of residual blood in the ventricle, a corresponding measure of greater distension of the left auricle, and the increased engorgement of the pulmonary vessels which leads to the accentuated pulmonary second sound. You have here, then, a second means provided by the right heart of helping your judgment as to the state of the left heart.

Systolic Murmur in Pulmonary Artery.

The presence of a systolic murmur in the second left intercostal space close to the sternum and strictly limited to this area is frequently noted. The conditions in which the murmur is present shall be presently dealt with; meanwhile I ask you to note that the murmur is heard

over the first part of the pulmonary artery; that this is the position of this part of the artery is confirmed not only by observations made after death, but also by the fact that, either by palpation or auscultation, or by both, the vibrations caused by the closure of the pulmonary cusps or the localized intensity of the sound enables us to locate with absolute precision the exact point where the artery joins the conus arteriosus. An interesting and important characteristic of the murmur is that during inspiration, especially during a somewhat deep inspiration, the murmur disappears, to reappear during expiration.

As to the conditions in which this murmur is present, chlorosis may be taken as an example. In chlorosis three murmurs may be present over the precordia—namely, a mitral systolic, a tricuspid systolic, and a pulmonary systolic. The mode of differentiating tricuspid from mitral and from aortic systolic murmur has been already dealt with. That these murmurs are not due primarily to the impoverishment of the corpuscles in hæmoglobin is proved by the fact that the murmurs are pronounced in a case of chlorosis if the patient, usually a young woman, has been struggling to carry on her ordinary working duties in spite of an increasing sense of physical weakness. When such a patient comes into hospital and is kept in bed, two of the three murmurs commonly disappear in a few days, long before any material improvement has been effected in the blood. The mitral and tricuspid murmurs are due to dilatation and to such myocardial weakness as allows of leakage at both these orifices during systole. In other conditions of debility these murmurs may also be present. Apart, however, from the presence of mitral and tricuspid murmur, the systolic murmur in the pulmonary artery is frequently present as the only murmur heard

over the precordia, and it is this murmur I specially want to explain the significance of, and to show you that its presence enables us to judge of the condition of the left heart. The explanation I offer to you of the mode of production of this murmur is as follows: We have already seen that the right ventricle is lifted by dilatation of the left heart into close contact with the chest wall, so that its pulsations may be visible in the third, fourth, and fifth left spaces; systole of the right ventricle, especially systole of that portion of it called the conus arteriosus, carries the origin of the pulmonary artery obliquely downwards and to the right; if you endeavour to simulate this action, you will find that the anterior and posterior surfaces of the artery at its origin are approximated, and that a relative narrowing is thereby produced. This narrowing is, to my mind, undoubtedly the mechanism by which the murmur is produced. It explains the disappearance of the murmur during inspiration, for with inspiration the heart has a freer forward movement and the kink is prevented. This explanation gives an important value, and I believe the true value, to this murmur. Its presence becomes a proof of a dilated left heart with, usually, an enfeebled myocardium. It is present in anæmia, in febrile conditions, in debility, and as a result of heart strain. It belongs to the class of murmurs which are called *functional* to differentiate them from murmurs due to endocarditis and called *organic*. The murmur is present in cases of organic mitral lesion when the origin of the pulmonary artery has not been carried to the left and upwards by the dilatation of the conus arteriosus.

Pulmonary Diastolic Murmur.

Pulmonary diastolic murmur not due to endocarditis at this orifice is, in my experience, very rare. I have, however, recently seen a case of the kind. The patient was a young Royal Field Artillery officer who had returned to light work after recovering from an extremely feeble heart following upon a febrile attack in Flanders. The light work had proved too much for him, and he showed a faint diastolic murmur over the pulmonary orifice, a faint systolic tricuspid murmur, a right auricle considerably to the right of its normal position, and a marked ventricular systolic wave in the neck veins. This was a case of pure strain in a heart that had materially improved in myocardial strength, but not sufficiently to withstand the strain of the work laid upon it. The murmurs disappeared under physical rest and medicinal treatment.

Explanations.

The explanations already given of the phenomena with which we have dealt may be supplemented with advantage. In the first place, feebleness or absence of the first sound over the right ventricle means weakness of the myocardium of that chamber, and in stout people it commonly means fatty infiltration. Cases of this kind die suddenly from some sudden physical effort, death resulting from right heart failure, not left failure. As we have seen, accentuation of the pulmonary second sound, systolic pulmonary murmur, systolic tricuspid murmur, diastolic pulmonary murmur, are the auscultatory phenomena, having origin on the right side of the heart, which may be present. They all indicate strain, varying in degree, of course—strain in the sense that the

myocardium is being called upon, or has been called upon, to do more than it can do comfortably or satisfactorily. Such strain leads to dilatation, sometimes of the left heart, at other times of the right, sometimes of both. Some of the phenomena produced on the right side of the heart become the index and guide to strain of the left heart; these are especially accentuation of the pulmonary second sound, pulmonary systolic murmur, and pulmonary diastolic murmur. The presence of tricuspid murmur shows, of course, the right ventricle is dilated, or atonic and enfeebled, either from initial strain or from the strain due to heightening of the pulmonary blood-pressure the consequence of initial strain of the left ventricle. The auscultatory phenomena result from relaxation and dilatation, and it is that fact which gives them their clinical significance and importance. Visible pulsation of the right ventricle has the significance already dwelt upon, and it is sometimes more valuable as an index of left-sided than of right-sided dilatation.

Venous Pulsation.

I now ask your attention to a further source of information—to another set of phenomena from which important deductions can be made, and which may lead very promptly to accurate diagnosis. I mean pulsation in the veins of the neck. There are two methods of making observations on these: the first is by means of the unaided eye. Fullness and pulsations, the rhythm of pulsations as timed by cardiac systole, whether they are entirely synchronous with systole, or what their time relation to systole may be, can be seen and noted, and let me impress upon you that this field of observation ought to be diligently explored by you. The second method of investigation is

by means of that beautiful clinical instrument invented by Sir James Mackenzie, and known as Mackenzie's polygraph. The latter method gives details which the eye cannot provide; at the same time I repeat that the unaided eye carries us a long way.

Pulsations in the veins of the neck are, of course, determined by the right side of the heart. This simple proposition is fundamental, and when it is fully grasped the second step is inevitable—namely, that such pulsations can only be caused by contraction of the ventricle or of the auricle. Pulsations are therefore systolic in time when ventricular in origin; all other pulsations are diastolic in time, and have their origin in the auricle. In the con-

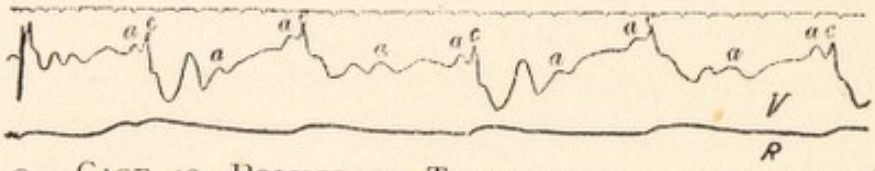

FIG. 9.—CASE 40. POLYGRAPH TRACING, 2 TO 1 HEART-BLOCK.

sideration of this aspect of our subject I confine myself to cases which are at present under observation, and I shall show you on the screen tracings made by means of the electro-cardiograph and by means of Mackenzie's polygraph.

CASE 40.—R., a woman aged sixty-five, had a pulse-rate of 40 when I first saw her. The pulse-rate corresponded with the number of ventricular systoles. On inspection of the veins in the neck there was visible a wave synchronous with ventricular systole, and between each of these another wave, which was presumably auricular systolic in time, and it was so regular that there was little doubt that the case presented the phenomena of a 2 to 1 heart-block. I show you the polygraph tracing (Fig. 9),

in which each wave marked *a* is produced by the auricle which is contracting quite rhythmically; the wave *c* is the

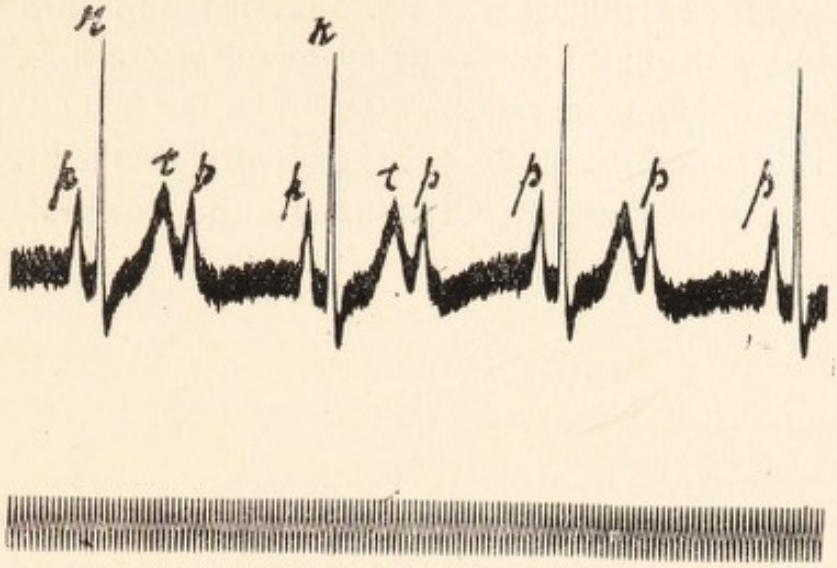

FIG. 10.—CASE 40. ELECTRO-CARDIOGRAM, 2 TO 1 HEART-BLOCK.

communicated wave produced in the carotid artery. A few days later the electro-cardiogram (Fig. 10) was taken, and you note that each wave *r* which is near the commence-

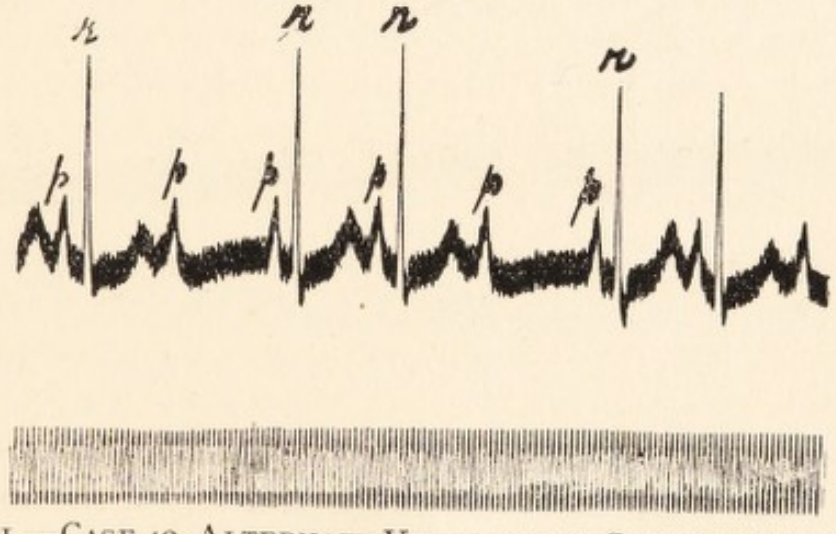

FIG. 11.—CASE 40. ALTERNATE VENTRICULAR SYSTOLES PRECEDED BY TWO AURICULAR SYSTOLES.

ment of the ventricular movement is preceded by two auricular contractions marked *p*. The diagnosis of a

2 to 1 heart-block was thus confirmed. This patient's heart was improving, and even at the same séance as gave us Fig. 10, Fig. 11 was also obtained, and shows that only every alternate auricular systole *p* was *not* followed by the ventricular wave *r*.

CASE 41.—W., a man aged sixty, with marked cardiac irregularity, showed in the neck to the unaided eye a

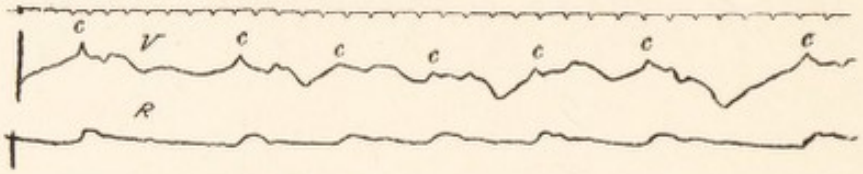

FIG. 12.—CASE 41. POLYGRAPH TRACING.

confused succession of undulatory movements, in addition to the respiratory distensions and emptyings. The polygraph tracing from the neck and the radial artery (Fig. 12) gives the position of the communicated carotid pulse marked *c*, and you note there is an absence of the auricular wave present in the preceding tracings. In fact, there is

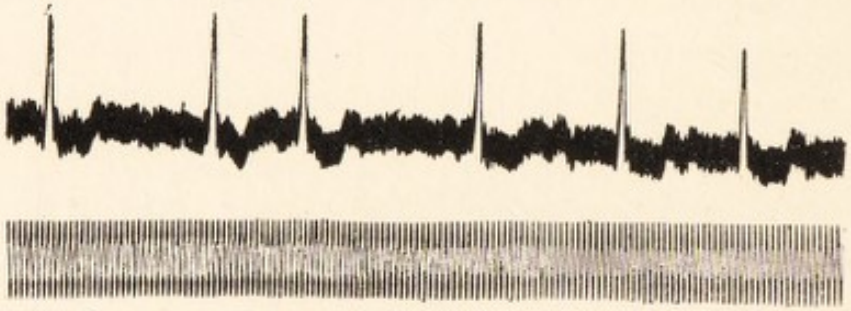

FIG. 13.—CASE 41. ELECTRO-CARDIOGRAM; NO AURICLE WAVE.

no evidence here of rhythmic auricular systoles; there is practically what used to be called paralysis of the auricle, now known as auricular fibrillation. The electro-cardiogram (Fig. 13) bears out the diagnosis of extreme irregularity, the absence of a true auricular wave, and the opinion that the patient had a fibrillating auricle.

Case 42.—S., a man aged fifty-four, with a systolic mitral murmur and a large heart, showed a ventricular systolic venous wave in the neck. The tracing (Fig. 14) shows the position of waves *a* and *c* as taken by the polygraph.

The timing of visible venous pulsations is sometimes difficult, but in many cases ventricular systolic and auricular systolic waves can be separated, and their

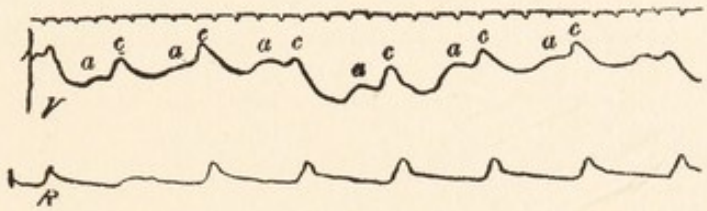

Fig. 14.—Case 42. Polygraph Tracing, in which the Venous Pulse seemed to be Systolic.

presence must be a measure of the fullness of the right side, especially of the right auricle.

I do not follow our subject further to-day, but you will understand that the study of the means by which the right side of the heart can be investigated, and the method of interpretation of the phenomena which I have presented to you, are able to guide you to a true estimate of many a heart that will come under the consideration of some of you, probably in the near future. The matters I have dealt with lie at the foundation of a true understanding of overtaxed hearts.

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