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ARTERIAL PRESSURE.[1]

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My first duty is to express my great pleasure in having this opportunity of bringing before the Society some of the important facts relating to arterial pressure as an introduction to what, it is hoped, will be an interesting discussion. My original intention, when speaking to the Senior Secretary on the subject, was simply to bring a paper before you dealing with certain aspects of the subject which have been, during the last few years more especially, occupying my attention; but it will certainly be more interesting to us all to have an open discussion of the subject at large.

Definition of Terms.—Before entering upon the consideration of the different divisions into which the subject naturally falls, it is obvious that we must be at one in regard to the terminology to be employed. The *maximal*, or *systolic, pressure* is estimated by the amount of force which will entirely arrest the flow of blood in the vessel undergoing investigation. In regard to this point there is probably no difference of opinion whatsoever, except with reference to the relative importance of the resistance offered by the wall of the vessel—a matter which will engage our attention shortly. The *minimal*, or *diastolic, pressure* is estimated by the point at which the greatest lateral oscillation of the arterial walls is found. This is ascertained by the level at which the greatest systolic and diastolic movements of the column of mercury are observed. This part of the subject has given rise to more difficulty than any other in the study of arterial pressure, and has led to wider discrepancies of opinion. Marey, for example, regarded the greatest oscillations as being an expression of the mean pressure. In this belief he was followed by Hill and Barnard, who expressed a similar opinion, but since the investigations of Howell and Brush we know that the greatest oscillations are found to indicate the diastolic pressure, their careful investigation having absolutely proved this point. The *mean pressure* is taken to be a point half-way between the maximal and minimal, while the *pulse pressure* is a term employed to

[1] The introduction to a discussion at the Medico-Chirurgical Society, 30th November 1910.

denote the amount of difference between the maximal and minimal pressures. The use of this latter term—*pulse pressure*—cannot be commended, as it savours of ambiguity. The term, however, has become implanted in modern literature, and when that is the case it is difficult to modify use and wont.

The arterial pressure, as measured by our clinical methods, is the *lateral* pressure, or the pressure which is exerted on the walls of the vessels at a right angle, approximately, to the line in which the onward current of blood flows. The *terminal* pressure may be regarded as this lateral pressure, in addition to the velocity of the blood. The general conception is that the lateral pressure in an artery is about equal to the terminal pressure in the branch of the vessel immediately beyond the point at which the lateral pressure is estimated.

In the discussion of various problems connected with arterial pressure it is useful to take into consideration the *co-efficient* of pressure, as it provides us with a convenient method of estimating the validity of the circulation. The co-efficient of pressure consists in the pulse pressure, divided by the systolic pressure. Dealing with anyone in ordinary health, we may say (expressing the pressure in millimetres of mercury) the systolic pressure is 120, the diastolic pressure is 90, and the pulse pressure is 30. This gives us:—

$$\frac{\text{P.P.}}{\text{S.P.}} = \frac{30}{120} = \frac{1}{4}$$

which is the co-efficient of pressure. We may further assume that the pulse pressure, multiplied by the pulse-rate, is equivalent to the *velocity;* while the systolic pressure, multiplied by the pulse-rate, expresses the *work done.* From this it is easy to calculate the efficiency of the heart. For anyone in perfect health, in formulæ similar to those of Tigerstedt and Hirschfelder, we may state the condition of matters in the following way:—

$$\frac{\text{P.P.} \times \text{P.R.}}{\text{S.P.} \times \text{P.R.}} = \frac{\text{Velocity}}{\text{Work}} = \frac{30 \times 70}{120 \times 70} = \frac{2100}{8400} = \frac{1}{4}$$

which gives the efficiency of the heart in health.

Let me, as a contrast to this, state what may occur in an instance of heart block:—

$$\text{S.P.} = 270\,;\ \text{D.P.} = 90\,;\ \text{P.P.} = 180\,;\ \text{P.R.} = 30 \quad \frac{180 \times 30}{270 \times 30} = \frac{5400}{8100} = \frac{2}{3}$$

In a case of aortic disease the departure from normal may be even greater:—

$$\text{S.P.} = 160\,;\ \text{D.P.} = 80\,;\ \text{P.P.} = 80\,;\ \text{P.R.} = 90 \quad \frac{80 \times 90}{160 \times 90} = \frac{7200}{14400} = \frac{1}{2}$$

Influences at Work.—The factors which maintain the arterial pressure must be briefly referred to. The first and greatest influence is the *energy of the heart.* Although many other causes are in operation in the maintenance of the arterial pressure, this is the most important. The heart still remains, as Harvey said, "the Sun of the Microcosm." If, for any reason, there should be an increased output of energy in the heart, the arterial pressure is augmented; and if, on the other hand, there should be diminished cardiac energy, the arterial pressure will necessarily fall, other conditions remaining constant.

The condition of the *arterial walls* plays a prominent part in the maintenance of the pressure. When the arterial walls are in conditions of health, so as to permit of the uninterrupted passage of the wave of increased pressure produced by cardiac activity, and when the walls retain their power of *elasticity* (meaning the property by means of which, after any change of form, they return to their previous condition) the state of matters is most favourable for the circulation. With every pulsation of the heart there is a corresponding increase of pressure within the arteries, and some of this pressure distends the arteries, giving rise to potential energy which, during cardiac diastole, becomes kinetic. There has been a considerable amount of misconception as regards the condition of the arterial walls. In the first place, the term elasticity has been used with very different conceptions, but, taking it in the sense employed by Kelvin and Tait, we may hold that it is the property by means of which a body that has been distorted by any force returns to its original bulk and shape on the removal of that force. In the second place, the *resistance* of the arteries to external pressure has been the subject of considerable debate within recent years. According to many authorities, as may be found expressed by Janeway, the resistance of the arterial walls to external pressure may be regarded as a negligible quantity; while, according to others (and here Russell may be regarded as the protagonist) the external resistance of the arteries is the most important factor in arterial pressure, as gauged by clinical methods. Here, as in many other matters, the truth lies somewhere between the two extremes, but it is certainly nearer the statement of Janeway

than of Russell. The question can easily be solved by testing the resistance of the arteries by the methods employed by Tait in the investigation of distensible tubes. A piece of the brachial artery (which is specially selected seeing that it is the artery in which we estimate arterial pressure most commonly) is tied at its two ends to two pieces of glass tubing, and fixed, by means of these, inside a glass tube, with rubber stoppers at each end, through which the two pieces of glass tubing are inserted. By means of two other glass tubes a stream of water is caused to flow from a reservoir, while another stream of water is led through the artery. Each system, internal and external, is in communication with a mercurial manometer, and when water is allowed to flow through the two systems, the amount of resistance required to compress the artery can be accurately determined. In my own observations the amount of arterial resistance ascertained in this way has never exceeded 15 mm. Hg, but in the series of 49 cases carefully studied by Herringham and Womack they found two instances in which the pressure was above 30; one of these gave 33, and the other 34 mm. Hg. With these two exceptions, in no case was the obliterating pressure above 24 mm. Hg. It may be held that experiments upon arteries after death do not help us as regards sphygmomanometric investigations during life, but it is to be remembered that the tonus of the vessels does not cease at the moment of death; on the contrary, it remains for a variable period.

The *resistance in the arterioles* is, next to the cardiac energy, the most important factor in the production of arterial pressure; in fact, the resistance and the pressure are reciprocal. The resistance in the arteries is produced by tonus, which is the result of the antagonistic action of vasoconstrictor and vasodilator nerves. There is a great difference between the action of these two sets of nerves; the former are constantly in action, the latter are only brought into action when they are required. These agencies are always in a condition of variation, so that the general and local vasomotor tone may be regarded as perpetually undergoing alterations.

The *character of the blood* undoubtedly plays a prominent part in the arterial pressure. The *density* appears to me to be in a direct ratio to the pressure. The few observations which have been made by myself personally have been with the view of illustrating the condition of matters in splenomegalic polycythæmia. These observations have led me to the belief that the

specific gravity and the pressure bear a direct ratio to each other. This appears to be the general conclusion of most observers. The *viscosity* of the blood might be thought to stand in the same relation as the density, and here again the few individual observations made by myself would lead me to that belief. A much larger series of investigations, carried out by Welsh at my suggestion, and shortly to be published, throws considerable doubt upon this matter, and it is one which will require careful investigation in the future. The presence of *chemical impurities* in the blood increases, for the most part, the pressure, but certain impurities bring about the opposite result. Whether these effects are the result of direct toxic blood on the arterioles or on their nerves is at present uncertain. The *quantity* of the blood in the circulatory organs must have some influence, and it might naturally be expected that increased volume would result in higher pressure. The vasomotor nervous system, however, has such ample powers of compensation that the quantity of the blood in itself has not, probably, such influence.

The local condition of arterial pressure in any part of the body is not merely modified by vasomotor activity, but also by mechanical interference. In other words, alterations in the *access of blood* bring about modifications in local pressure. It has long been known that any obstruction increases the proximal and lessens the distal pressure. The external pressure of an aneurysm or a neoplasm, or the blocking of the lumen by embolism or thrombosis, reduces or terminates the pressure, and this often without any alteration in the condition of the wall beyond the obstruction. A very interesting instance of this was under my care in the Royal Infirmary just about a couple of years ago. A man of middle age was admitted with aortic disease. The left arm furnished a beautiful example of Corrigan's pulsation, while there was nothing of the kind to be found in the right. When the pressure was tested in the two arms it was found that in the right arm the systolic pressure was 74 and the diastolic 55 mm. Hg; while in the left arm the systolic pressure was 130 and the diastolic 90 mm. Hg. The patient died somewhat suddenly, as the result of his cardiac disease, and after death a thrombotic obstruction was found at the origin of the right subclavian artery. The brachial arteries were carefully tested after death by Dr. Rainy and myself, and we discovered that the resistance in each artery was practically the same, the figures being 11 mm. Hg in the right brachial and 10 mm. Hg in the left.

Methods of Investigation.—Amongst the methods of estimating the arterial pressure from the *physiological point of view* the earliest observations were made by Hales, who ascertained the height to which the column of blood could rise in a glass tube, connected with one of the larger arteries in the horse. At a later date the use of the mercury manometer was introduced by Poisseuille, and the method was rendered more available by Ludwig. Still more recently the introduction of the elastic manometer, such as the membrane of Chauveau and Marey and the metal spring of Fick, may be particularly mentioned. It is not necessary on the present occasion to dwell on these physiological instruments.

Attempts at the *clinical estimation* of arterial pressure have a respectable antiquity, and it is possible from the writings of Aristotle, Herophilus, and Galen to bring forward much of importance as regards this aspect of the study of the pulse. In the works of Galen the investigation of the pulse is carried out with what, in this age, appears to be excessive refinement. There cannot be a doubt of the extreme importance of educating the sense of touch in the estimation of arterial pressure, and in my own clinical teaching, after impressing upon those who give me the privilege of teaching them that their first duty lies in trying to ascertain, with as much accuracy as possible, the state of the arterial walls, the next place is given to the resistance within the vessels. Although, to my way of thinking, it is quite as scientific to attempt the estimation of the temperature in a case of fever by means of the unaided thermic sense of the physician as to try to gauge the arterial pressure by means of tactile sensibility, yet it must be admitted that we are sometimes inclined to trust too much to our modern instruments. It is therefore necessary, before teaching the use of such instruments, to educate our clinical clerks most carefully in the methods of estimating arterial pressure by means of the sense of touch.

The first attempts at the *clinical estimation* of arterial pressure by *modern instruments* were made by Vierordt, who, using one of his own sphygmographs, attempted, by attaching different weights to it, to ascertain the amount of pressure necessary to overcome the arterial pressure. The first observer, however, who really brought the clinical estimation of arterial pressure within practical possibilities was von Basch, who was followed, at a somewhat later date, by Potain. Marey, Waldenburg, Hoorweg, Hürthle, Bloch, Mosso, and Oliver, during the last quarter of the

previous century, applied themselves to the problems arising from the study of arterial pressure in man, and also paved the way for future advances; but it is not too much to say that von Basch and Potain deserve the credit of being pioneers in our clinical methods. The present period is entirely due to the introduction by Riva Rocci of the method of circular compression of a limb, so as to obliterate one of the larger arteries and estimate the effects by examination of itself or of one of its branches further from the heart. It may be said at once, without fear of contradiction, that, with the exception of those sphygmomanometers which apply this method, there is none which need be seriously considered. The others are of historic, but not of scientific, interest. Another point is that the only reliable instruments are those which are provided with a column of mercury. All the others, whether employing an aneroid, a column of air, or a tube of spirit, are not to be trusted, unless they have been standardised immediately before use by comparison with a mercurial manometer. Judged by these standards, the instruments of Marey, Hürthle, and Mosso, which embrace the extremities, after the manner of the plethysmograph, and that of Gärtner, which estimates the pressure after the removal of the compression of a finger by the return of colour to the skin, as well as the instruments of Hill and Barnard, and of Oliver, cannot be recommended. On the other hand, the apparatus of Riva Rocci, Stanton, Cooke, Janeway, Martin, French, and Mummery are at once perfectly simple and absolutely reliable.

In the last two or three years *graphic records* of the arterial pressure have been obtained. The sphygmomanometer of Erlanger, while based upon the same principles as that of Riva Rocci, was the earliest attempt to furnish graphic records of arterial pressure in clinical investigation. A tube connected with the column of air, leading from the cuff to the mercury of the manometer, communicates with a hollow rubber ball, which responds, by distending and contracting, to every oscillation of the air and the mercury. This ball is surrounded by a glass bulb, leading by a tube to a tambour connected with a lever, which records the oscillations upon a smoked cylinder. These tracings furnish a complete record of the oscillations of the column of air, but do not furnish any record of the height of the column of mercury by which the pressure may be estimated. It is, accordingly, necessary to watch the level at which the mercury stands while noting the oscillations. The return of the arterial pulse at the wrist coincides with the com-

mencement of the large oscillations, and the point at which the large oscillations suddenly begin to diminish gives an indication of the diastolic pressure.

The sphygmomanometer recently introduced by me also takes advantage of the principle of circular compression in order to estimate the maximum or systolic arterial pressure, while it at the same time gives the minimum or diastolic pressure by the oscillations of the mercury. It has a mercurial manometer, the lumen of which is exactly that of the ordinary physiological kymograph. The air contained in the armlet can be increased, and the pressure on the limb therefore elevated, by means of a large syringe, and the pressure may be raised quickly or slowly according to requirements. By means of a valve the pressure may also be lowered quickly or slowly. A float rests upon the mercury, surrounded, as is usual in the physiological laboratory, by alcohol, and an upright rod of aluminium leads to a horizontal arm which writes on the revolving cylinder. In order to have the absolute zero, a fixed arm traces the abscissa upon the cylinder, which is driven by a clockwork placed horizontally, as in the instrument of Erlanger. The pulsations of the artery below the point of compression are recorded by means of a transmission sphygmograph. This consists of a tambour placed in contact with the brachial or radial artery, as may be most convenient, by a pelotte resting upon the vessel. This tambour is brought into communication by rubber tubing with another tambour, the movements of which are recorded on the cylinder simultaneously with the movements of the kymograph. The best tracing is obtained when the tambour in contact with the artery is larger than that connected with the recording lever, by means of which the movements are amplified. As usually employed, the pressure is raised with one steady, forcible expulsion of the air contained in the syringe until a high level is reached—150 or 160 mm. Hg being sufficient under ordinary circumstances. The valve already set at the slow escape allows the pressure to fall gradually. If sufficient pressure has been employed, the tracing from the artery, after a few oscillations due to the inertia of the mercury, shows an entire absence of all movements.

In the interpretation of the tracings there is one point which may always be depended upon with a reasonable degree of certainty; the point at which the pulsation returns in the vessel below the seat of compression is approximately the systolic pressure. This has been admitted ever since the observations of Vierordt and von Basch. The middle of the oscillation at this point is therefore

chosen as the index of systolic pressure. It is perfectly true that it is not the end pressure. The top of the first wave which appears gives the maximum systolic pressure indeed, but it is the lateral and not the end pressure which is recorded, and therefore the first pulsation by the method of circular compression, as was shown by Masing, does not give the absolute maximum. The determination of the minimum or diastolic pressure is not such an easy problem. Marey suggested that the point at which the largest swing of the instrument occurred was an index of the mean pressure, and this was adopted and amplified by Roy and Adami. It has been proved experimentally, however, by Howell and Brush that this does not indicate the mean, but really records the diastolic, pressure. Masing, who obtained some tracings from the artery below the seat of compression, believes that the greatest movement of the sphygmographic tracing marks the diastolic pressure. This must, however, be an error, as the greatest amplitude of oscillation of the sphygmographic curve is very commonly found after the pressure in the armlet has been allowed to fall nearly, if not quite, to zero, and the results obtained by this method of estimation are unmistakably erroneous. My own method of obtaining the diastolic pressure is to ascertain where the greatest amplitude of oscillation occurs in the kymographic curve, and to take the middle point of this as the expression of the diastolic pressure.[1]

A very beautiful modification of my sphygmomanometer has been introduced by Singer, who has reduced the size of the apparatus, and has applied to it an ink pen, so that records can be obtained without the troublesome necessity of smoking the paper on the cylinder.

During this same period, when our attention has been applied to the graphic investigation of arterial pressure, *auscultatory determination* has also been studied. Korotkow, Ettinger, Bozowski, Krylow, Ehret, Fischer, and Gittings have, during the last five years, paid much attention to this subject, which is, both pathologically and clinically, of very great interest. These determinations are carried out with an ordinary sphygmomanometer of the Riva Rocci type, applied, in the usual manner, to the arm, and the brachial artery is carefully auscultated at a point about half an inch below the armlet. The arm is compressed in the ordinary

[1] The method of palpation of the brachial artery, advocated by Ehret, should have been mentioned. He finds that the brachial artery just below the cuff gives a peculiar hammering vibration at the time of the greatest oscillations, and this characteristic change is the criterion of diastolic pressure.

way, and when the column of mercury is allowed to fall, on releasing the valve, a distinct sound, with the character of a thud, is heard. This denotes the maximum, or systolic, pressure. Variations in this sound follow, and at last, with still further lowering of the column of mercury, the sound altogether disappears. This is generally understood to correspond to the minimum, or diastolic, pressure. This method has proved of much interest in my own wards, as we have found in it a very definite way of estimating the acuteness of sensibility of the observer. When the same observer listens to the sounds, and, at the same time, applies his fingers to the arterial pulse, the audible and palpable phenomena occur at exactly the same moment of time. If, on the other hand, one observer listens to the brachial artery whilst another feels the arterial pulse, a discrepancy is perfectly certain to take place. It seems to me, from my own observations in a large number of cases, in hospital as well as private practice, that the auscultatory determination cannot replace the previous tactile determination. It nevertheless furnishes a useful additional method to our modes of investigation.

Possible Fallacies.—It may very pertinently be asked, Are there no fallacies in our modern methods? It seems to me that our new methods of investigation provide few, if any, sources of error. With a good and simple instrument, the results which we obtain contain few possible sources of error in respect of determination, leaving at present out of account the question of interpretation. Riva Rocci and Henson have proved the accuracy of these methods on the dead body.

With all these instruments the influence of the personal equation is reduced to a minimum, and the question involves merely the variable acuteness of sensibility of the observer. This has been already referred to in my remarks on auscultatory determination. Russell, as has already been mentioned, believes that the wall of the artery under compression has a great influence on our results, and that degenerative conditions of the wall, in addition to increased contraction of its muscular tunic, account for some of our highest readings. It must be remembered, however, that the middle coat of a large, is relatively much thinner than that of a small, artery, and my own opinion is that Russell mistakes the increased pressure produced by contraction of the arterioles for changes in the walls of the large arteries which we are in the habit of investigating. Henson, from a long series of observations, showed that the resistance of the vessel walls under ordinary cir-

cumstances was, on an average, equal to 3 mm. Hg, and that, even when they were much sclerosed, it was never higher than 20 mm. Hg. The observations of Herringham and Womack showed that in only 2 out of 49 cases the resistance was above 24. We may therefore, until the contrary has been proved, assume that any changes in the vessels will not account, at most, for more than 20 or 30 mm. Hg.

It is clear that the limitations by which we are surrounded in attempting to estimate the absolute value of arterial pressure consist, to a great extent, in the difficulties by which we are beset in analysing the different factors maintaining the pressure. The relative amounts of cardiac energy and of vascular resistance are still, as was remarked by me in the Toronto discussion, a sealed book. A relatively low general pressure may be one which is nevertheless too high for the energy of the heart, while on the other hand a pressure which appears to be abnormally high may be one which is quite low in relation to the strength of the heart. The fact that spasm of the arterioles and smaller arteries has an important influence in modifying the pressure must always be borne in mind.

It must be added that when there is any very high degree of adiposity or any extreme amount of œdema, readings with the sphygmomanometer are rendered less easy and more doubtful. It is further to be noted that if there should be any liability to tremor, accurate observation is rendered less easy.

The pressure should always be estimated while the patient is in a position as nearly as possible horizontal, and, at the same time, resting comfortably. The limb which is employed for investigation ought to be absolutely at rest.

Arterial Pressure in Health.—The *normal limits* of arterial pressure require a brief notice. From numerous observations with our modern instruments by a large number of reliable observers it may be assumed with certainty that the normal systolic pressure in the young male adult does not exceed the limits of from 90 to 130 mm. Hg, while the diastolic is from 70 to 100. It is less in healthy women than in men of the same age, and still less in children. In healthy individuals variations in the arterial pressure take place without the presence of abnormal conditions, and must be accounted as normal. Daily fluctuations, probably dependent upon the condition of the whole system, are always found. The pressure usually reaches its highest level in the forenoon, after which it falls, frequently,

however, manifesting another but smaller rise in the afternoon. Recognising that such changes exist, we will be wise if we always take our readings in any individual at the same hour.

When the pressure is studied by means of continuous tracings smaller variations, both in the level of the pressure and the amplitude of the oscillations, may be observed. They are clearly analogous to the Traube-Hering curves seen in physiological tracings taken with the kymograph, and they undoubtedly depend upon changes in the influence of the vasomotor nerves.

The *external temperature* must exert some influence, but we still remain in want of sufficient observations upon the subject. So far, the general result of the investigations which have been made (by Müller, for instance) shows us that when an individual is exposed to any considerable alteration of the external temperature the arterial pressure is apt to rise.

The *atmospheric pressure* exerts considerable influence over arterial pressure. Most of our knowledge upon this subject is due to Crile, who has shown that the external atmospheric pressure and the internal arterial pressure stand to each other in a direct ratio.

Posture exerts a certain amount of influence. There is not much difference in arterial pressure between the recumbent and the sitting postures, but there is a much greater difference between sitting and standing. It is the diastolic pressure which undergoes the greatest changes, so that in this way the pulse pressure is diminished in the upright position.

The *influence of food* is not constant. After a meal the pressure sometimes rises and sometimes falls. Diminution of pressure is easily accounted for by the diversion of blood into the abdominal viscera, but this may be balanced by reflex stimulation of the vasomotor system. After the contents of the alimentary tract have begun to enter the circulation there is certainly a tendency to elevation of the blood-pressure.

The question of food at once brings up the analogous subject of the effect of certain articles much employed in daily life. It is almost certain that the usual influence of *alcohol* (after a slight initial rise produced by increased frequency of the heart) is to cause diminished pressure. *Tea, coffee, and cocoa* have not yet received the attention which they deserve, and a series of observations upon these substances would be most helpful. In regard to *tobacco*, we well know that its effects upon the vaso-constrictor apparatus bring about a very great increase of arterial

pressure, and this view entirely accords with a great series of observations made in France, showing that arterial sclerosis is frequently brought about by excessive vascular contraction, resulting from the abuse of tobacco.

Muscular exercise produces remarkable effects. Gentle exertion has little, if any, influence in raising arterial pressure, even when it is continued for a considerable length of time. Violent muscular exercise is followed first by a considerable rise of pressure, and this is succeeded by a striking fall at a later stage. The observations of Gordon, carried out when he was my house physician, are sufficient to prove these facts.

Mental exertion has similar effects, but these vary with the intensity of psychical processes. This is a matter which has been tested very frequently in my clinique, both on myself, my staff, and my clerks, with results which are extremely uniform.

The arterial pressure certainly falls during *sleep;* it is lowest in the early hours of normal sleep, rising gradually from that period until the hour of waking. Lewis Bruce, as well as Brush and Fayerweather, have brought before us most interesting observations upon this subject.

The *age of the individual* has considerable influence. Until middle life there is no great departure from the normal limits of arterial pressure, but after that period there is undoubtedly a much greater tendency to a higher level. Clifford Allbutt has devoted his attention to this subject, and to myself personally his teaching has been of the greatest benefit. Russell has criticised his opinions very freely, but to my way of thinking he has failed to shake the foundations upon which Clifford Allbutt's views are based. The real facts can only be ascertained from a very large series of observations. There are undoubtedly many individuals who retain comparatively low pressure during long lives, while in others a high pressure begins almost in youth. As a general rule, however, increase of age brings with it elevation of pressure.

Arterial Pressure in Disease.—In attempting to give a summary of the probable modifications of arterial pressure in pathological conditions, it seems to me that it will be advisable, in the first place, to deal with alterations which mainly *affect the circulation.* It is impossible to do this, however, without bringing into the field of discussion a large number of external agencies connected with every part of the body, seeing that the circulation is not merely subject to purely mechanical alterations, but also to chemical

changes, and perhaps even more subtle influences arising out of metabolic processes, while, at the same time, the heart and the blood-vessels show unceasing response to nervous influences, and the circulatory variations in turn react upon the entire organism. We must assume that the cardiac energy and the arterial pressure are reciprocal, while arteriole contraction and arterial pressure stand likewise in a direct ratio to each other. These general principles are necessarily subject to various modifications from changes occurring in any of the factors concerned in the physiological or pathological processes present in any given circumstances. There is a general correspondence, universally recognised, between the level of pressure and the cardiac conditions. When the arterial pressure is high the cardiac impulse is forcible, the second aortic sound is loud, and even the first sound produced by the left ventricle may gain in its intensity. When a case manifesting such conditions is watched for any length of time some increase in the size of the heart is made out by the ordinary clinical methods of palpation and percussion, as well as by the screen and the skiagram. In such cases, also, tracings obtained with the capillary electrometer, or with the string galvanometer, show us, as has often been observed by myself, that the electromotive changes are excessive; but, in addition to such results, there are some even more characteristic effects arising from the relations of the arterial pressure and the cardiac energy. No one could have been more sceptical than myself in regard to the possibility of aortic diastolic murmurs without valvular disease, it having always appeared to me that the origin of the aorta was so resistant as to preclude the possibility of stretching from stress leading to a strain. Some years ago, however, the stern logic of fact caused me to recognise that my views had been based upon insufficient experience, and in a paper in the *Edinburgh Medical Journal* some cases of diastolic aortic murmur without valvular lesions were published. In these cases the diastolic murmur appeared, disappeared, and reappeared, according to the condition of arterial pressure. These cases, however, were surpassed in interest by a recent experience in my ward. A man of middle age was sent in on account of aortic disease, and on admission it was found that a very musical diastolic murmur could be heard by anyone standing at the foot of the bed, *i.e.* about four feet from the heart. This murmur, as usual, was louder on standing than on sitting, and yet more so than on lying down, while after walking round the ward it became still further exaggerated. His arterial

pressure was found to be 95 diastolic and 145 systolic on admission. After a few days the murmur became so much diminished in intensity as to lose all of its musical character, and it could only be heard on auscultation with the stethoscope. After a few days more rest the murmur absolutely disappeared. It occurred to me that it might be of interest to find out the relation between the arterial pressure and the murmur. After quiet rest in bed the maximum arterial pressure was found to be 115, and with this pressure no murmur was audible; with gentle exertion the arterial pressure rose to 130, a soft, aortic, diastolic murmur developed. After some strenuous exertion, such as walking quickly three times round the ward, the maximum arterial pressure rose to 145, and the murmur became so loud and musical that the patient himself could hear it. It was obvious that in this case there was not merely stretching of the fibrous and elastic tissues forming the aorta, but also of the muscular fibres surrounding the aortic orifice.

Entirely analogous results have been obtained on investigating the pulmonary artery, and the pulmonary diastolic murmur of high pressure described by Steell, Duckworth, Barr, and myself is undoubtedly produced in the same way. Keith has shown us that a loss of tone in the muscle fibres surrounding the mouth of the vessel allows stretching of the fibrous tissue.

Low arterial pressure is, in general, associated with feeble cardiac impulses and short, high, weak sounds. The conditions in which this group of phenomena is found are frequently observed in surgical practice, and the work of Cushing and Crile demand our most careful attention.

Let me now direct your attention as briefly as possible to some conditions of arterial pressure in general and special affections. Amongst the *general diseases* let us take, as an example of the *infective* type, typhoid fever. Upon this subject the results of all observers are wonderfully uniform. In the overwhelming proportion of cases of typhoid fever the arterial pressure is reduced. The pressure begins to fall during the first week, and continues to fall during the second, third, and fourth week. During the fifth week it begins to rise, and gradually regains the normal. The most important statistics upon this subject are contained in the observations of Crile. From the modern arrangements in our Royal Infirmary few opportunities have been afforded me, during the last few years, of watching cases of typhoid fever, but the few observations made in my wards entirely accord with Crile's results.

Amongst *toxic* conditions let me cite the affections due to lead. In all the cases which have been under my observation during the last few years the pressure has been elevated, and the various symptoms stand in a direct ratio to the rise of arterial pressure. This is the case in regard to the digestive, muscular, and nervous symptoms.

Lastly, amongst such general diseases there can be little doubt that *metabolic* disturbances, in which the arterioles are subject to the baneful effects of imperfect tissue changes, are followed by high arterial pressure. A very large number of conditions, which we sum up under the heading of chronic gout, are associated with high pressure, and pave the way, by means of it, for the structural alterations in arterioles and arteries affecting the entire organism. Such conditions are of everyday experience.

Turning now to *special diseases*, let me, in the first place, refer to affections of the *vessels*. There cannot, in this age, be any doubt that, without any structural alteration in the circulatory organs, including heart and vessels, there are conditions in which periods of pressure changes, both low and high, occur through the influence of the vasomotor system. To such conditions Pal gives the name of *vascular crises*. They are attended by alterations in heart-beat and pulse-wave, and accompanied by modifications in the amount of every secretion, as well as in the appearance of the skin, and they give rise to very considerable disturbance of the nervous system, particularly in its subjective aspect. Such crises are frequently produced by causes of purely nervous character, but they are also produced by various poisons circulating in the blood. They are apt to lead, in time, to structural changes in the arterioles and arteries, particularly in the myocardium and in the kidney. Such conditions frequently manifest all the features of vicious circles, closely studied lately by Hurry, as the alterations in pressure are very apt to lead to nervous instability in varied forms. While crises of high arterial pressure are more common than those manifesting low pressure, the latter condition is also sometimes found as a critical phenomenon, and here again the tendency to a vicious circle is apt to manifest itself, as low pressure frequently brings about nervous depression. The arterioles and small arteries, therefore, may be regarded as subject to many influences leading to alterations in pressure, which bring in their wake undoubted pathological conditions.

When we approach the subject of *arterial sclerosis* we find a general consensus of opinion that endogenous poisons, no less

than those which are introduced from outside, produce definite arterial degeneration. One important point is concerned with the question whether high pressure leads to arterial sclerosis. There can be no doubt that the question must be answered in the affirmative, and amongst many workers upon this subject it is a duty to cite Clifford Allbutt, Savill, and Russell. The former of these observers has indeed classified out a series of causes under the categories of toxic, hyperpietic, and involutional, but he frankly admits that there is no sharp dividing line between these classes. That this is far from being a solution of the whole problem must be obvious, when we consider the fact that there is frequently arterial sclerosis without high pressure. In 506 cases Groedel found 35 per cent. in which there was no elevation of pressure, while Rudolf, in collaboration with Ellis and Robertson, found that the pressure was only above normal in 50 per cent. of well-marked cases of arterial thickening. Such facts have been under my own observation for many years, and one of the most striking corroborations of this statement is to be found in the observations of Elliot Dickson upon the arteries and pressure in miners, where, with extremely common arterial changes, there is seldom any modification in arterial pressure.

The relation of *angina pectoris* to arterial pressure forms a somewhat complex subject. While there is a variety of angina pectoris of occasional occurrence which seems to be entirely due to spasm of the arterioles—the angina pectoris vasomotoria of Landois and Nothnagel—and while in a great many cases of organic angina pectoris the attacks are preceded and attended by rise of arterial pressure, due to spasm of the arterioles, there are numerous instances in which the arterial pressure is below normal. This is a fact to which attention has been more particularly called by Morison. It is, nevertheless, in such instances particularly that the pressure, although below normal, may be relatively too high for a weak and failing heart, and the caution already expressed should, in this place, be once more recalled. It is a well-known fact, to which Mackenzie has more particularly invited our attention, that in cases of arterial sclerosis there is a greater tendency to spasm of the arterioles than in ordinary conditions. Every observer who looks into the matter with an open mind must be struck by the enormous variation in the appearance of the arteries when a spasm of the arterioles is present, as compared with their condition in the absence of a spasm. A few years ago Pal showed that vessels which were almost straight with a

pressure of 95 became highly tortuous when the pressure rose to 200 mm. Hg.

Turning to the heart, we find in *acute pericarditis* an affection with a great tendency to bring about a lowering of arterial pressure, through its weakening effect upon the myocardium, and it is one of the affections in which a favourable or an unfavourable prognosis may be reached, according as there is little or considerable lowering of the pressure. All the recent cases of pericarditis which have crossed my path have made excellent recoveries, and on looking over their records, the striking feature is that in no case was the arterial pressure below 125 mm. Hg. When the pericardium is distended with fluid, as the result of an acute attack, the arterial pressure falls to a level which is alarming, and which is one of the surest indications of the necessity for active intervention. In the case of *acute endocarditis* there is likewise a liability to a fall of arterial pressure, and here, again, the prognostic significance of the arterial pressure cannot be overestimated. Amongst the various *valvular lesions* there is an immense series of different pressure results. In most of them, until fatal cardiac failure steps in, there is but little interference with arterial pressure, so wonderful are the powers of compensation possessed by the circulatory system. In aortic disease there is a most striking instance of remarkable divergence between the systolic and the diastolic pressure. This has been already referred to in the introductory remarks upon the co-efficient of pressure and the efficiency of the heart. It is very common in aortic disease to find a systolic pressure of 170 or 180, while the diastolic pressure may only be from 70 to 80. Even greater variations of pulse pressure have been recorded. Another interesting point in aortic disease, with insufficiency, is that the difference between the arterial pressure in the arms and legs is much greater than in health. To this point attention has been particularly directed by Hill and Hare. It has been suggested by the former that the condition is a reflex one, intended by Nature to prevent cerebral anæmia. Other valvular diseases produce comparatively little alteration upon the arterial pressure, except in so far as they bring about alterations in the efficiency of the myocardium. Diseases of the *myocardium* yield widely different readings; in fact, the results entirely depend upon whether the myocardial troubles are due to primary alterations in the heart muscle or are secondary to disease elsewhere. In such affections as myocarditis, following upon diphtheria or other

acute disease, the arterial pressure, in general, falls. The same occurrence takes place in the fatty degeneration attendant upon any grave disorder of the blood. On the other hand, in fatty infiltration the arterial pressure is frequently high, simply because in such conditions impure blood in the arterioles causes increased resistance. Needless to say, when the myocardium is undergoing hypertrophy, to compensate for increased resistance, the pressure is high. One of the most interesting of all myocardial conditions is to be found in heart block, in which, apparently from the long intervals between the ventricular systoles, there is in most instances a very high systolic, and a very low diastolic, pressure. Attention was called to this fact by myself about five years ago.

In affections of the *respiratory system* there are great variations of arterial pressure. For the most part, in asthmatic conditions there is a tendency to high pressure, while in pulmonary tuberculosis, as a general rule, the pressure is low. In pneumonia there are very different levels of pressure in individual cases. In the remarks on prognosis some useful indications will be brought forward in connection with this subject.

The arterial pressure in *glandular diseases* constitutes one of the most interesting subjects in modern medicine. In myxœdema, as is universally recognised, the pressure tends to be considerably above the normal; in exophthalmic goitre, although in general the pressure is below the normal, this fact is not of universal application. As the vasomotor control of the arterioles appears to be in a more unstable condition than in most diseases, it can be no wonder that the pressure is very variable. Addisonism furnishes one of the most fascinating problems in its relation to arterial pressure. In the fully-developed condition the arterial pressure is always low, because in it the medullary portion of the suprarenal body is destroyed, and the production of pressor substance is accordingly annulled. There may be a deep discoloration of the skin without alterations in arterial pressure, and in these cases we may assume that the cortical portion alone is diseased; on the other hand there may be profound depression, without discoloration, but with great reduction of the arterial pressure, and in such cases it is certain that the medullary portion of the gland is that which is affected. To these conditions attention has, within the last two years, been particularly directed by Rolleston and myself. Acromegaly sometimes gives high and sometimes low pressure. Here again the different effects may be correlated with the particular portions of the pituitary body which are implicated.

Amongst affections of the *kidney*, in acute nephritis, the pressure is in general elevated, but this is not so universal as is commonly supposed. In many such cases the pressure is really below normal. It has often seemed to me that the state of the pressure may vary according as the process affects the glomeruli or the tubules. This, however, is a subject upon which observations are greatly wanted. In most instances of chronic nephritis the arterial pressure is high, and it is particularly high in most cases of uræmic poisoning. The highest pressure which we meet with is found in chronic interstitial nephritis, with arterial degeneration and cardiac hypertrophy, especially (as was shown by Hasenfeld and Hirsch) when the splanchnic arterioles are implicated. In waxy degeneration of the kidney the arterial pressure is always low, unless the process is accompanied by a high degree of interstitial nephritis.

The relations which exist between alterations of arterial pressure and disturbances of the *nervous system* are of the most interesting description, seeing that a large number of nervous affections are produced by changes in arterial pressure, while, on the other hand, nervous disorders frequently bring about very definite modifications in the arterial pressure. In a considerable number of cases of adolescent insanity, and more particularly in precocious dementia, the arterial pressure is extremely low. The recent work of Rae Gibson has thrown a good deal of light upon the connection between the two conditions, and the facts which he has brought forward have been fully borne out by a few cases under my own care. In cases of melancholia there is, as a general rule, a pressure rather above the normal, while in mania, on the other hand, the pressure tends to be subnormal. In most of the painful affections, such as neuralgia and the crises of tabes dorsalis, the pressure is almost invariably above the normal during the attacks of pain. Pal believes that in such cases the cause of the pain is a vascular crisis. Possibly the most remarkable series of phenomena appearing on the borderland of circulatory and cerebral changes is to be found in the facts connected with cerebral hæmorrhage. It cannot for a moment be doubted that the essential factors in bringing about cerebral hæmorrhage are structural changes in the arteries and increased pressure within them. These points are, however, eclipsed in importance by the clinical observations that after a hæmorrhage has occurred the arterial pressure goes on steadily rising, and the result of the beautiful series of investigations by Harvey Cushing is to show that the

object of this high arterial pressure is to maintain the circulation in the vital centres at the base of the brain.

Arterial Pressure in Prognosis.—From the point of view of prognosis the study of the arterial pressure is of the greatest practical importance. In *acute disease* the relation between the rate of the pulse and the height of the pressure is of the highest value. In an address given by me some years ago the rule was enunciated that in cases of *pneumonia,* when the pulse-rate per minute does not exceed the height of the pressure in mm. Hg, the outlook is hopeful, but that when the converse occurs the prognosis is grave. Every observation which has been made since the date of that address has more firmly convinced me of its value. It has recently been emphasised in a most interesting lecture by Hobart Amory Hare. Within recent years pneumonia is the acute disease which has been most frequently under my observation, but it seems to me quite clear that some such formula might well be applied to other acute affections. In almost all of them, as has been stated, there is a tendency to a fall of the pulse-rate. In all of the acute diseases, if there is too great a tendency to fall of pressure, there must, in the mind of the observer, be considerable anxiety. Acute *nephritis* furnishes an interesting example of a disease in which the condition of pressure is of use from the point of view of prognosis. It has already been remarked that in many cases of this affection there is no increase, and that a decrease may even be present. If there is a considerable increase the prognosis is less favourable than when there is no rise at all.

Amongst *chronic diseases* most instances of anæmia result in diminished pressure, but this is by no means to be regarded as a general rule, as chlorosis has frequently increase of pressure. With the exception of such cases, it may be said that the lower the arterial pressure the worse the outlook for the patient. The same is true in *cachectic* conditions.

In chronic *nephritis* the arterial pressure is, in general, above the normal, but the degree varies according to the variety of the affection. In chronic *parenchymatous* nephritis, resulting in the *large white fatty kidney,* the arterial pressure is usually elevated, but by no means to the same degree as in interstitial nephritis. An interesting point, bearing upon the prospects of the patient, is that a rise of pressure above what has been found to be the normal for the case is very commonly the herald of uræmia. In chronic parenchymatous nephritis of the type ending in the *small white granular kidney* the pressure is considerably higher, but here,

again, a sudden rise is apt to portend the onset of uræmia. It is in chronic *interstitial* nephritis—the type which terminates in the *small red granular kidney*—that the arterial pressure reaches such enormously high readings, and it is in such cases that the prognosis is so largely dependent upon the influence of remedial measures upon the pressure. In severe cases, with cardiac, respiratory, and nervous symptoms, as well as extremely high arterial pressure, we are sometimes absolutely unable to influence the course of the disease to any appreciable extent.

Treatment of Changes in Pressure.—The last subject to which attention must be directed deals with the treatment of the conditions in which the arterial pressure is abnormal. In order to preserve the arterial pressure as nearly as possible at its normal level every method by which the general health can be best maintained must be adopted. Attention to the skin, both by baths and clothing; regulation of the diet, both as regards fluids and solids; care of every eliminating channel; and sufficient muscular exertion, together with adequate rest—all such matters must be taken into the most careful consideration. It must be obvious that there is no absolute rule to be employed in cases of departure from the normal; each instance must be judged and treated in accordance with its particular nature. When the arterial pressure is too low, as in acute diseases, it may be necessary to use digitalis, or strophanthus, or strychnine, or suprarenal substance, or its active principle, adrenalin. When, on the other hand, there is a great tendency to high pressure, the nitrites, the iodides, the hot pack, the vapour bath, and even venesection may require to be adopted, along with drugs which increase the action of every channel of elimination.

Such are the points connected with arterial pressure which seem to me to be those from which a most interesting discussion may arise, and upon which the Society will certainly wish the opinions of the members.

DISCUSSION.

Dr. Wm. Russell said in the ten minutes allotted to him he would attempt as clearly as possible to indicate what he regarded as the central phenomena in the important question before the Society. He was sure Dr. Gibson would understand that it was from no disrespect to him that he avoided much of what had been said. He would begin at the extreme end of the phenomena and mention a case which was at present in his ward in the Royal Infirmary. The patient came in

with a pressure of 295 mm. of mercury. With rest in bed and treatment it fell to 165, showing a fall of 130 mm. of mercury. This pressure swung back again to 180, and kept fairly steadily between 180-210, but while still in bed, this patient would develop a brachial pressure of 70 mm. greater than this, associated with symptoms which it was impossible to enter into here. This rise was invariably associated with a definitely palpable contraction of the brachial artery, diminution of its size, and hardening of its wall. There was no question as to what took place in the brachial artery. This was important, as the common statement, which all of them had been long familiar with, was that when pressure rose the periphery was constricted, but unfortunately the periphery had been regarded as capillaries and arterioles.

Nine years ago, before this Society, he had described a hypertonic contraction of the arteries. At that time he had not investigated farther back in the circulation than the radial artery. When, however, the modifications of the Riva Rocci instrument came into use, he found it was necessary to go farther back—from the radial to the brachial artery. He had found that the brachial artery was one which was often readily palpable, and the condition of its walls readily discovered. On post-mortem examination he found that the brachial artery might have a muscular coat which had undergone great hypertrophy, and that the wall had therefore become thickened. He had in his possession brachial arteries like pieces of caoutchouc, and yet the contention was that these thickened brachial arteries had nothing whatever to do with the compressibility of the artery. It was difficult to realise how it was possible to contend that this was so. After death an artery like that alters enormously; it becomes like a piece of thick cloth; it is no longer the elastic tube with its tonic and hypertonic wall. It seemed to him that that was the explanation of the fact that such eminent clinicians as Sir Clifford Allbutt and Sir Lauder Brunton talked about blood-pressure and heart-power while absolutely ignoring this condition of the vessel wall.

To-day he had read Janeway's latest contribution to this subject; it had just been published, and he went the length of maintaining that the calcified wall had no influence whatever on the compressibility of the artery. He would leave it to them to consider whether this was a reasonable position and one which clinicians could really accept.

Going on to explain another change that the thickened brachial artery undergoes, Dr. Russell referred again to the patient with a blood-pressure of 200. When the brachial underwent contraction the pressure was necessarily thrown back upon the aorta. Now there had been a difficulty here, and it was caused by the fact that there were certain cases with thick arteries which did not give a high reading. He had recently had a man in his ward with calcareous radial arteries and sclerosed brachials, in whom the brachial artery was not only

thickened but dilated and atonic; it had a thick wall, but it also was a big vessel (shown on blackboard), and whenever one got a large tube like that it was easier to constrict than a small tube like that (demonstrated). This man, a moulder by occupation, had a big heart, but he died from heart failure; his lungs had become œdematous, he was rapidly pouring out fluid into both pleuræ, and yet when dying he gave a pressure of 140 mm. of mercury. That is not a high pressure, but that man's heart was not working—if you take 120 as the average power of the heart—a quarter of the average. The forearm gave a reading of 30 mm. higher than the upper arm owing to the calcareous condition of the wall. At the post-mortem examination the muscle of the left ventricle was soft and fragmented, and that of the right largely replaced by fat, yet we were asked to believe that the arterial wall was negligible.

With their permission he would now go to the other extreme, away from the state of affairs in sclerosed vessels. This to him was the more interesting part of the subject, namely, the cases where there was a relatively low pressure. He would illustrate this by a patient who was convalescing slowly from an acute pericarditis with a distinctly feeble heart. In this patient, as long as the pressure kept to 120 or 125 mm., everything went on pleasantly and there was no discomfort, but if from constipation or unwise feeding or from mental disturbance the vessels become hypertonically contracted (it is a fact that they become hypertonically contracted, for the contraction can be seen and felt taking place in the brachial artery), the pressure rose to 140-145 when lying in bed. Constriction of the periphery has occurred, but that includes the brachial artery. When the brachial becomes constricted, it becomes thickened as the result of the hypertonic contraction, but the rise of pressure takes place behind this in the arch of the aorta. He could not, and no one could, say what the aortic pressure was. This rise in aortic pressure embarrassed the feeble heart.

The essential clinical fact was the constriction of the arteries up as high as the brachial, and it is that constriction which is being read; there were no means of estimating how much pressure that constriction had thrown back on the aorta, and it might be quite easily overestimated, because, as Dr. Gibson pointed out, the heart very readily strikes a balance and recovers itself.

Another case of the same kind was that of an old gentleman, aged 80, suffering from angina minor. He had a pressure of 140-145 when an attack of angina minor was present; the attacks were associated with a palpable and definite tightening up of the radials and the brachials, and whenever the pressure was reduced to 120 there was no further heart trouble, and the radial and brachial arteries became soft. It was necessary to determine what would prevent the constriction, and in this particular case only very small quantities of iodide of potassium

were required to do so. The action of iodide Dr. Edwin Matthew would no doubt be able to say more about, but one plain fact was that in this old gentleman's case the pressure was 120 in the soft tonic vessel, and that when the brachial became hypertonic it went up to 140. The pressure was thrown back on the aorta, and the feeble heart was embarrassed. A rise in brachial reading in such a case meant a rise in aortic pressure. No one questioned that, but he would say this, that we were estimating not the pressure in the aorta but the compressibility of the brachial. The difference in the readings was the result of hypertonicity. The symptoms were those of heart embarrassment from constriction of a much wider periphery than had hitherto been recognised; the constriction threw the pressure back on the aorta, and therefore a greater strain was put on the left ventricle.

Dr. Rainy directed attention to certain physical aspects of the subject which were apt to be overlooked in purely clinical discussions.

He pointed out that in those methods of determining blood-pressure which depend on the use of an armlet, one had to assume that the tissues between the armlet and the vessels are so plastic that they may be practically considered to transmit the pressure in the same way as a sac of fluid would do. This assumption that the rigidity of the tissues is negligible is probably justified in ordinary cases, but, as Janeway and others have shown, there are certain conditions in which it is untrue. If, for instance, œdema is present, the method of armlet compression is quite unreliable. A not unreasonable suggestion for this is that the tissues, in consequence of the tension produced by the œdema, become more or less rigid, just as a pneumatic tyre gets rigid when fully inflated, and thus interfere with the transmission of the applied pressure.

The question of the allowance which may have to be made for resistance due to rigidity of the vessel wall appears at first sight to be a more difficult one, but, fortunately, blood-vessels remain alive for many hours after somatic death, and further, they can be maintained in life after they have been removed from the body. There is thus no difficulty in ascertaining the influence of this factor by experimental methods. Recent work by Stevenson has shown that transfusion of a vessel with a fluid which acts as a vasoconstrictor can induce a condition of hypertonicity, and by a simple piece of apparatus the difference between the external and internal pressures when the vessel is just flattened by the external pressure can be ascertained. The difference between these two obviously gives the exact amount of resistance to compression which such an artery offers. Stevenson's results show that there is a wide degree of variation within physiological limits. Roughly, in ordinary cases it will be found to be less than 10 per cent., and even where there is marked hypertonus it rarely exceeds 30 per cent., although in a few extreme instances it proved

much greater. One must therefore acknowledge that the vessel wall does affect the readings, but not to an extent which can materially vitiate the observations in the immense majority of cases.

He further pointed out that the blood-pressure in all the larger arteries is very nearly equal to that in the aorta, and that when one remembers that the resistance to the flow of ordinary fluids through tubes varies inversely as the fourth power of the diameter, it becomes obvious that practically the whole fall of pressure takes place in the arterioles and capillaries.

He also directed attention to the fact (which appears to be too much disregarded) that it is merely an assumption, and in many cases an unwarrantable assumption, to say that disappearance of the pulse coincides with closure of the vessel, for, as a matter of fact, the pulse disappears before the vessel is obliterated, as can be shown alike by experiment and by the application of mathematical formulæ.

In conclusion he referred to some of the earlier mathematical literature on the subject, and especially to two most valuable articles by Dr. Thomas Young, one of which was his graduation thesis, the other the Croonian Lectures which he delivered at a later period of his life, whilst in Germany the work of the brothers Weber, both on "Wave Motion" and on "The Application of the Study of Wave Motion to the Pulse," remains a standard authority to the present day.

Dr. Cargill Knott said it was difficult for the mere physicist to speak, but some remarks Dr. Rainy let fall require a little supplementing—he was not quite sure that he understood them. There is no doubt that the strain is very much greater in the large tubes. Then there was the question of the way in which the pulse falls off along these tubes. He always thought—he might be wrong—that the fading away of the pulse towards the periphery was due really to viscosity acting in the fine tubes. He had made an experiment, which Dr. Russell had expounded to him some years ago, namely, when the arteries in the arm are compressed the compression closes the pulse at the wrist. That seemed to show—though Dr. Rainy might be right—that a resistance had been produced there which was comparable with the resistance in the smaller arteries where the pulse is not at all conspicuous. It was a difficult question how that would affect the whole circulation; the whole circulation is affected by all those various forms of tubes, and the fact that by this constriction you destroy the pulse brings the arteries of the arm into somewhat the same category as regards the smaller arteries. In that case it might be that both points of view are sound enough, but he would be sorry to be dogmatic on the question. It is much too delicate a thing for a pure physicist to say much about; the physicist pretends to know nothing beyond pure physics. He might be speaking rashly, but that was what struck him

when hearing Dr. Rainy, and he thought it possible that both Dr. Rainy and Dr. Russell were right.

Dr. Elliot Dickson said he was not going to give any mathematical calculation, but as a country physician who had done some work with regard to this subject he should like to say something about arteriosclerosis in apparently healthy men. He and his brother, Dr. Arnott Dickson, had taken a series of 500 miners who were apparently healthy —who did not complain of any symptoms referable to their circulatory apparatus. They took the blood-pressure by the Riva Rocci instrument in these 500 men of all ages and found that in no less than 456 the arteries were thickened. Out of 116 men under 20 years of age 100 had easily palpated arteries. Of the 500 only 2 had atheroma, and they were old men. Only 44 out of 500 had radial arteries which could not be palpated. The blood-pressure was 140 mm. or over in 6 per cent. of the cases, and in each of these the urine was tested for albumen, with a negative result. In 469, or 94 per cent., the blood-pressure ranged within normal limits; all had thickened arteries; in some of the cases this was very obvious, especially in men with thin arms, where the arteries could be tested all the way up to the axilla. Over and over again the brachial was traced right up to the axilla; it could be picked up between the fingers and thumb and rolled about; it felt like a lead pencil. Tracings had been taken of brachial arteries, by Dr. Gibson's kindness, such as had been mentioned, showing readings of 120-118, and one of 95 mm. of systolic blood-pressure, so that whatever increased blood-pressure is due to, there can be no doubt that thickening of the brachial arteries is at least not the only factor. No doubt, with thickened arteries, there is a tendency to increased blood-pressure, but that was more evident when taken in connection with increase in age of the patient. In arteries of the same degree of thickening, so far as could be detected by the finger—and after considerable practice one could tell pretty well—there were great variations (from 85-165 mm., the former being obtained on three different occasions in a youth of 18). The great point about this series of cases is that the men were healthy and had nothing wrong with their circulation.

There were many cases of hypertonic contraction in men who came into the surgery to have teeth extracted, &c.; after such slight operations, as was to be expected, the reading was generally about 20 mm. higher than was found on testing the men on a second occasion.

So far as the cause of the thickening of the arteries in this series of cases goes, it is rather difficult to explain. All the cases have thickened arteries with low or normal blood-pressure. They correspond with what Clifford Allbutt calls toxic arteriosclerosis. The toxin in this series of cases has to do with conditions of work underground. He had known and tested cases where the arteries were perfectly

normal and where thickening developed within a year after working underground. The nature of the toxin is somewhat difficult to ascertain, but probably it has something to do with the ventilation underground. There is an enormously high percentage of carbonic acid gas in coal-mines; ·7 per cent. represents good ventilation from a coal-miner's point of view; ·04 above ground. Whether that was a factor in producing thickening of the arteries or not it was impossible to say meanwhile.

Dr. Oliphant Nicholson spoke of blood-pressure in regard to pregnancy.

Dr. Shennan said he rather hesitated to enter into a discussion which had been, so far, completely clinical, but one or two things mentioned had suggested several points to him. Firstly, as to the terminology. The previous speakers had used the terms *atheroma, arteriosclerosis, arterial sclerosis, calcification of the arteries;* probably the best term was *arterial sclerosis,* because it included all the others. It might be taken that high blood-pressure was the prevalent end of these conditions, the usual conditions found post-mortem being arteriosclerosis—general thickening of the arterial wall—often associated with atheroma, meaning, by the latter term, localised thickening of the wall, with constant degeneration of the thickened portion. One point in connection with calcification of arteries was that, in thickened arteries which were also calcareous, there was a constant feature, namely, that in the portions of the arteries corresponding to the flexures, *e.g.* the joints of the extremities, the calcareous deposit was not seen. That could be verified by X-ray photographs of the separated arterial tree. One could also tell where the flexures had been, such as the knee-joint, the elbow-joint, the groin, by the fact that the vessel was still quite pliable, the parts between the joints being resistant and calcareous. This should be taken into account in considering the resistance offered by the wall of the vessel to recording instruments.

With regard to the quantity of blood. He had recently been watching post-mortems in Münich. What had struck him as amongst the most common pathological changes found there were hypertrophy of the heart (Münich beer heart) and arteriosclerotic atrophy of the kidney. There was a general idea that it was the *quantity* of fluid (beer) which was the cause of the hypertrophy; if in place of the quantity the *toxic* effects of the beer were specially to be taken into account, it would be a terrible indictment of Münich beer.

With regard to the viscosity of the blood, he had noticed in one or two cases of polycythæmia that the heart had not been hypertrophied at all; in one case the coronary arteries had been extremely atheromatous, in fact a fine thread could scarcely be passed through the vessel. The hypertrophy was an interesting point, because Zétulle had stated that great hypertrophy might occur although the left

coronary was practically impervious. That there was some arterial communication between the coronary arteries was generally allowed.

Dr. Gibson had referred to interstitial nephritis and chronic parenchymatous nephritis; were they to assume that in all forms of chronic nephritis, and many people included with these the sub-acute forms, high blood-pressure was always present? Does chronic nephritis include the small red granular kidneys, and the white kidneys with coarse granulations? He himself recognised a small red kidney due to chronic nephritis, and another small red kidney due to primary arterial disease. In dealing with post-mortem examinations he was inclined to think that in renal disease, the result of arteriosclerosis, hypertrophy of the heart was distinctly greater on an average than in other forms of chronic kidney disease.

With regard to amyloid disease and chronic nephritis, amyloid disease, it had been said, was always associated with low blood-pressure, chronic nephritis with high blood-pressure; but the curious point was that one hardly ever met with pure amyloid disease of the kidney; it was always associated with chronic interstitial nephritis or chronic parenchymatous nephritis. It was a rather nice point to reconcile these statements.

Dr. Edwin Matthew referred to the action of certain drugs on blood-pressure.

Dr. Church remarked on the great usefulness of the blood-pressure recording instrument, and advocated that it should be found in every general practitioner's consulting-room. It was important in giving medicines where the blood-pressure was high not to give a remedy which would increase it, and where low a remedy that would lessen it. He had found to give something which would increase the blood-pressure where a patient was suffering from sleeplessness due to high pressure was harmful, whereas something which reduced the pressure generally induced sleep.

The time at which the blood-pressure was taken was also an important point; the pressure varied greatly in accordance with the time. For instance, the pressure after breakfast would be found to be 15 or 20 mm. more than that before breakfast; after a smoke, 20 or 30 mm. more than before the smoke.

With regard to iodide of potassium, it was no doubt very helpful, but its use might be greatly increased by the help of a hot bath. He had seen a hot bath bring down the blood-pressure 25 or 30 mm. It was wonderful what a hot bath could do in lowering pressure and adding to the comfort of the patient.

As regards children, it might be that the blood-pressure affected the physical power of the child and would be a guide of what the child was able for both physically and mentally. He had tested children

and found that the physical, and particularly the mental, capacity was in keeping with the amount of arterial pressure.

Dr. G. A. Gibson thanked the President for his graceful remarks, as well as those gentlemen who had taken part in the discussion and those who had assisted the debate by their presence. He hoped that the success of the discussion would be gratifying to the Council of the Society.

With regard to the remarks of the President, he did not think he had ever in his life asserted that the condition of the arterial wall was negligible, although he had not gone as far in the other direction as Dr. Russell. In the work of Herringham, to which he had referred, published in *The Transactions of the Royal Society of Medicine*, there were recorded resistances of 32 and 34 mm. Hg in the brachial walls, but what is such a figure in a pressure of over 300? That was his great difficulty.

Turning to Dr. Russell's contention, he "nailed his colours to the mast," since the high pressure is mostly due to the resistance produced by the contraction of the arterioles, possibly also of the capillaries, although that is open to doubt.

It was a pleasure to have listened to the observations of Dr. Rainy, who had explained a good many points very clearly indeed, especially in regard to the wall of the vessel; his statement, that the arteries removed from the body are still to be regarded at the time of the investigations as living, largely discounts the objections of Dr. Russell, that the condition of tonus has been lost.

Dr. Cargill Knott tried to smooth over the breach between Dr. Russell and Dr. Rainy, and seemed to think there might be a *via media* between the two views; as regards some of his statements, he thought Dr. Cargill Knott had argued from rather narrow premises to sweeping general conclusions. Such was the impression left on his mind, but he might have misunderstood the meaning of what was said.

Dr. Elliot Dickson's observations were well known to him, and he had been consulted by him some years ago on the subject. The view that the thickening of the arteries in his series of cases was entirely due to carbonic-acid gas was, he thought, the correct one; it was surprising how much carbonic-acid gas there was in the Fife mines.

He was interested in hearing Dr. Oliphant Nicholson's remarks in explanation of a subject in which he himself felt a great interest, but of which he had little practical knowledge; Dr. Nicholson's work on the effect of thyroid in post-partum conditions led him to expect this interesting communication from him.

With regard to Dr. Shennan's observations on arteriosclerosis and amyloid disease, he entirely agreed with Dr. Russell that arteriosclerosis is entirely different from atheroma, in fact the latter is more like endarteritis deformans in its general conformation. The President had

somewhat cleared up the point as regards the condition of the kidney. Under the term chronic interstitial nephritis there are several varieties, but most of these may be regarded as being part of a general disease—a widespread process. One observation he would like to make. The President would remember well that about the time when they were undergraduates they used to see a great many cases of waxy disease in this city; now they saw comparatively few. It used to be more common in Edinburgh than in the South. (The President here said that was not so, the lessening of the disease was due to antiseptics. Dr. Gibson had gone to Birmingham from Edinburgh and had found it less common; the President had gone to Newcastle and found it quite as common; it was purely antiseptic. He agreed with Dr. Gibson in the fact that waxy disease was less common now, but not with his explanation of the fact. Dr. Gibson rejoined that in the Seventies Birmingham could not show any waxy disease, and this could not at that date be due to antiseptic surgery.)

To Dr. Matthew's observations on therapeutics he had listened with the greatest interest. He could not help thinking, when Dr. Matthew was telling of his success with the iodides, with which most of them were in harmony, that in severe spasm he had found iodide of ethyl act at once, just as nitrites do. No doubt it was necessary to get free iodine into the blood.

With Dr. Church he agreed as regards the value of the sphygmomanometer in general practice, and hoped it would not be long before the instrument, in one or other of its simpler but reliable forms, was in the hands of every practitioner.

In conclusion he desired to express his grateful appreciation of the generous way in which his introductory remarks had been received by the Society.