The Goulstonian Lectures on the physics of the circulation : delivered before the Royal College of Physicians at the examination Hall Victoria Embankment, on Feb. 27th, 1894 / by Paul M. Chapman.


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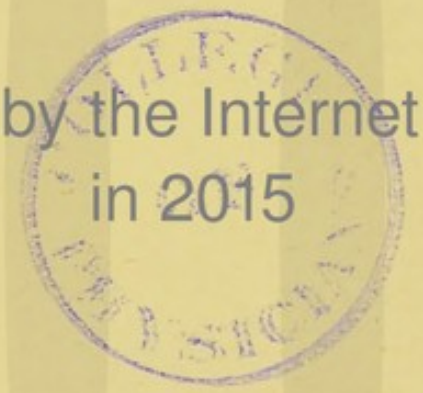
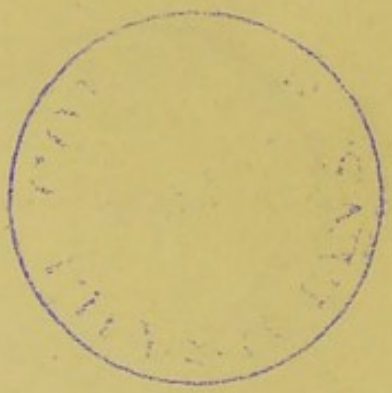




THE LANCET, MARCH 3, 1894.

The Goulstonian Lectures

ON

THE PHYSICS OF THE CIRCULATION.

Delivered before the Royal College of Physicians at the Examination Hall, Victoria Embankment, on Feb. 27th, 1894,

BY PAUL M. CHAPMAN, M.D., F.R.C.P. LOND.,
PHYSICIAN TO THE HEREFORD GENERAL INFIRMARY.

LECTURE I.

MR. PRESIDENT AND FELLOWS,—It has been my object, ever since my observation was first directed by Dr. Burdon Sanderson to the consideration of the graphic representation of the heart's action, to endeavour to obtain a more accurate clinical knowledge of the state of the whole circulation than is obtained by the ordinary methods of the physician. For this purpose I have studied the literature of the subject and the results published by physiological laboratories in England and on the Continent, at the same time endeavouring to carry on clinical observations of my own by methods which I shall describe in the course of these lectures. It would be impossible to treat the whole subject of the "Physics of the Circulation" in three short lectures, and some explanation is due from me as to the selection of my title, for which I must ask your indulgence. The aim of these lectures is to commend as strongly as possible a more close and accurate observation of the state of the circulation in the sick than is now employed in clinical medicine. The cardiograph has practically never been used by physicians, while the sphygmograph has fallen into disuse. The indistinct and largely imaginative use of the senses of hearing and touch has led to the disuse, and in England to the distrust, of accurate instrumental measurement in medicine in favour of the employment of what we like to call our "own unaided observation." I believe that partly reluctance to take trouble and partly the sense that our own individuality, rather than great accuracy in diagnosis, is the main factor in creating what is known as a successful practice, have more to do with this feeling than we are ever likely to acknowledge to ourselves. Be this as it may, it is the conviction of others besides myself that the employment of the cardiograph alone may give us definite proof of conditions otherwise not recognisable—still more, that the combined use of the cardiograph with the sphygmograph may be the only means in certain cases of arriving at a definite diagnosis. It is especially for the latter method of observation that these lectures are intended to be some preparation, and this can only be done by entering somewhat largely into the subject of the "physics of the circulation," which will be dealt with in a kind of summary of our present knowledge. The graphic representation of the arterial pulse and its relation to the cardiographic tracing can then, and then only, be properly discussed. It will be found, in the course of these lectures, more convenient to consider the cardiographic tracing first by itself separately

in connexion with the intra-cardial variations of pressure, and again secondarily in connexion with the pulse tracing. In discussing the mechanism of the circulation it is of the first importance to accustom the mind to regard all changes in blood flow through any particular bloodvessel or cavity as conditioned by variations of pressure. To say that during ventricular contraction the atrio-ventricular valves are closed, and that consequently the blood is forced out of the heart into the arterial system, may be correct in fact, but it is a mode of expression not so helpful to the understanding as to say that, owing to certain detailed circumstances, the intra-ventricular pressure is raised so far as to preponderate over the pressure in the aorta. I shall therefore, whenever possible, speak of blood flow in any individual as being immediately conditioned by variations of blood pressure, though, in comparing different individuals, it must never be forgotten that a stream meeting with little obstruction may, with less driving power behind it, attain the same velocity as a stream driven with much greater power if the obstruction is correspondingly great.

Before proceeding to the consideration of clinically observable conditions it will be necessary to review shortly the state of our physiological knowledge at the present date—(1) as to the state of the heart and its contents during a revolution, and the mode of production of the heart sounds, (2) the effect on the circulation of contributory influences such as respiration, at which part of these lectures some words on the venous and capillary circulation will be convenient, and lastly (3) as regards the sequence of events within the arteries, the sphygmographic tracing, simultaneous tracings, and the use of the combined method. A full historical account of our gradual advance in knowledge would not here be possible. It will not even always be possible to pause in order to give my acknowledgments to those who have furnished me with facts. I can, however, at the outset refer to Marey's initiative work in France ; to the works in German of v. Frey and Krehl, and of Hoorweg of Utrecht, and especially to the valuable articles of Hürthle of Breslau[1] and to the exhaustive summary by Tigerstedt ;[2] and in England to the physiological work of Burdon Sanderson, Waller, Roy and Adami, and others, and to the suggestive clinical observations of Broadbent, Ewart, and Douglas Powell. The latest contribution to the study of the mode of production of the first heart sound is furnished by Kasem-Bek[3] by experimental inquiry conducted in the laboratory of Joh. Dogiel of Kasan, who had in 1863, in conjunction with C. Ludwig, made experiments of the following nature. They took a dog, opened its chest, and one after the other tied the upper and lower venæ cavæ, the pulmonary veins, and lastly the aorta. The heart was then excised and immersed in a retort filled with defibrinated blood, and the narrowed end of the retort was closed with an elastic membrane and connected with the elastic tube of a König's stethoscope ; under such circumstances the heart, when contracting normally, gave rise to a sound. Dogiel and Ludwig therefore argued that the noise made by the contracting muscle plays the most essential part in the production of the first heart sound, the system of many branched bundles of muscle superposed in layers giving a tone or noise by their sudden contraction. In 1869 Guttmann and Rosenthal, repeating these experiments, considered the sound obtained to be hollow and toneless in comparison with the normal sound, and thought that for its production the contraction of the papillary muscles would

be sufficient, by whose means the valves were put into vibration but not closed. Guttmann concluded that the first sound was a mixed one and was chiefly caused by the tension on the valves, but secondarily through muscle contraction. Von Bayer, in 1870, by connecting a glass tube with the ventricle through the aortic opening and suddenly letting a column of water into the heart, obtained a sound shorter, duller, and higher-pitched than the first heart sound. Since this sound strikingly differed from the normal, he concluded that the first heart sound was a muscle sound. Ostroumoff, in 1873, criticised Dogiel and Ludwig's experiments, saying that in them the heart was not wholly emptied of blood. When this was done he obtained no sound at all which could be heard through the chest wall. In a further series of experiments he excited the heart by electricity and could not thus satisfy himself of any dependence of the heart sounds on muscular contraction. The opponents of the muscle theory make chiefly two objections to the supporters of it—first, that the heart has never been so completely emptied of blood as to avoid the possibility of sudden valvular tension ; and secondly, that the muscular bundles of the ventricle are not in a condition to produce a tone, since the heart contraction is not tetanic, while Helmholtz has proved that only tetanic muscular contraction produces a tone. With regard to the last objection, the bundles of muscular substance in the ventricle cross and recross each other, and are so overlaid one upon another that by a sudden contraction it is quite possible that they could give rise to a sound and not merely to a rustle. Kasem-Bek's experiments seem to be fairly conclusive with regard to the first objection. They were of two kinds. First, a dog's heart was rapidly removed and, with the apex uppermost, was slightly compressed in the hand. It was then put into the retort apparatus described by Dogiel and Ludwig. Since, according to Guttmann, the valves might be made tense even in a heart emptied of blood, Kasem-Bek excised a portion of the auricular wall and thrust short gutta-percha tubes shaped like ear specula into the auriculo-ventricular openings, with the funnel-shaped expansion above and the narrow ends within the ventricles ;

[1] Pflüger's Archiv für Physiologie.
[2] Physiologie des Kreislaufes.
[3] Pflüger's Archiv, Band xlvii., 1890.

these were either fixed in position by pins or by closing the auricles over their expanded ends. After every experiment he ascertained that no blood was in the ventricle. When the heart was now auscultated he heard, with strong and regular contractions, a loud and well-continued tone, which differed little from that heard over the apex before the experiment, except that the sound was louder in the excised heart. In his second series of experiments, with the heart *in situ*, he opened the pericardium and passed one index-finger round the aorta and pulmonary arteries, the thumb being in front at the auriculo-ventricular limit. When the thumb and finger were compressed so as to prevent blood entering the heart there were heard on auscultation through the uninjured chest wall on the left side a first and second sound once ; then the second sound disappeared, while the first sound persisted, though less loud than before. Krehl [4] comes to the same conclusion as Kasem-Bek. The consideration of the first sound as a muscle sound persisting in a measure throughout the systole originated with Turner in 1829, and the same view was held by C. J. B Williams and the committee of the British Association in 1836. It is,

I think, after the experiments I have related, as far as possible proved that the first sound is a muscle tone, to which, indeed, other sounds are superadded, especially those arising from the vibrations of the mitral and tricuspid valves, also from the vibrations of the semilunar valves, and from the vibrations of the blood column immediately above them. With regard to the second sound, it will suffice to say that it disappears when the valves are destroyed, and that it consists of two sounds united, due respectively to the closure of the semilunar valves and to the vibrations of the blood column above them. We have, of course, two first and two second heart sounds, for sounds are conveyed by both sides of the heart. These, under normal circumstances, are always heard synchronously. A reduplication of sounds, when heard, is most frequently in connexion with the second sound at the base of the heart; and it is to be remembered, as has been shown by Roy and others, that the distensibility of the pulmonary arteries is greater than that of the aorta, and that the blood pressure therein is both lower and subjected to more frequent variations owing to the disturbances due to respiration. The first sound being a complicated sound chiefly due to contraction of muscles, the fibres of which are common to both ventricles, a true reduplication of it is never heard. An interesting case of apparent reduplication of both sounds will be discussed when the graphic representation of the heart's movements is under consideration. The first sound of the right heart is best heard near the right edge of the sternum, in a limit between the fourth intercostal space and the fifth rib, and the first sound of the left heart in the fifth left interspace or in the fourth intercostal space directly above. The second sound of the right heart is best heard near the edge of the sternum, in the second left intercostal space, and the second sound of the left heart at the right edge of the sternum, at the limit of the first rib and second intercostal space. Vierordt,[5] by placing between the ear and the chest wall a column of thin indiarubber discs and estimating how many of these discs would have to be interposed before the sound vanishes, has judged the relative intensity of the heart sounds. It appears that the first sound is relatively louder on the left side, especially in childhood, and that the second sound, on the contrary, is considerably louder over the pulmonary artery, diminishing up to the age of forty. After that age the aortic second sound becomes slightly louder than the pulmonary second sound. We must assent, I think, to the summing up of Tigerstedt: "So much is certain, that our theory of the normal heart sounds, and, in dependence therewith, our explanation of heart murmurs, is still in many essential points confused, and not to be depended on for the more delicate diagnosis of organic heart lesions."

As to the production of the apex beat we have to consider the changes of position and of form of the heart during systole. An interesting experiment was performed by Berry Haycraft,[6] who noticed that if a needle is inserted through the chest wall into the base of the heart the end projecting from the thorax moves upwards at each systole; if the needle is passed through the thorax nearer to the apex the excursion of the needle is less. If it is inserted into the apex, very little movement is communicated to the needle. As may be seen by direct inspection of the exposed heart during systole, the base approaches the apex, and the latter is

[4] Ueber den Herzmuskelton, 1889.
[5] Die Messung der Intensität der Herztöne. Tübingen, 1885.
[6] Journal of Physiology, 1891.

the least movable point of the heart. According to Tigerstedt

the approximation of the base to the apex can, at least in part, be attributed to the recoil set up as the blood streams through the arterial orifice. However, it appears that in the bloodless frog's heart beating *in situ* the base approaches the apex, although not so markedly as when occupied in driving out blood; it, therefore, to some extent probably depends on the peculiar arrangement of the muscular fibres of the heart. But for mammals Guttmann,[7] Jahn,[8] and others say that when the veins are ligatured the base does not approach the apex. Further, it is possible that the extensibility of the large arteries can, by an increase in length when the blood is driven into them, act in favour of an approximation of the base to the apex;[9] whether distension of the aorta, or recoil from the rush of blood, can play any part in the production of the apex-beat is, however, to me doubtful. As will be shown later, no blood can pass into the aorta until the intraventricular pressure is raised sufficiently to overcome the pressure in the aorta; for this a certain time is necessary. The apex-beat appears to be caused by the peculiar spiral arrangement of the muscular fibres whereby the point of the heart rotates during systole from left to right and is tilted up. That the contraction itself is very sudden will appear from the fact that the rapidity of propagation of the wave of contraction throughout the heart has been estimated by Waller to be at about five metres per second; we may therefore assume that the apex-beat occurs at the commencement of systole, or at least at such a time as to exclude the supposition that it is caused by elongation of the aorta owing to its reception of the contents of the heart. In this connexion it may be convenient to mention that according to Roy and Adami[10] the papillary muscles contract later and cease to contract earlier than the ventricular wall in a ratio of 5 to 8. Fenwick and Overend[11] find that the papillary muscles contract one-twentieth of a second later than the heart's apex. The idea is commonly held that the pulmonary and aortic valves are closed by the regurgitation of blood against them owing to the relaxation of the ventricles and the consequent alteration in pressure on the two sides of the valves. This is not exactly the case. During systole the blood is forced through the orifices, which at that time are mere chinks owing to the pads of muscle which take their origin from all sides of the ostium. Vortices are thus created in the space between the arterial root and the edges of the valves. These vortices tend to press the edges of the valves together, and the valves consequently close at the moment that the blood actually ceases to stream through the narrow crevice. By this means all regurgitation is avoided, the valves being already closed before the recoil of the blood column against them. Were it not for this, some little regurgitation would be constantly occurring, and, indeed, if the necessary muscle pads which support the semilunar valves are insufficiently developed, an aortic insufficiency may appear in spite of perfectly normal valves.[12] The muscular arrangement at the venous orifices has also its uses in favouring blood flow. When fluid flows out of an opening in the bottom of a vessel the lateral pressure forces the fluid through the opening in a funnel shaped stream, so that the mean section of the stream is smaller than that of the opening. During relaxation of the auricles the venous orifices are slightly enlarged, so that the largest possible amount of blood may flow through the orifices in a given time. In discussing the graphic records of the variations of pressure within the heart and bloodvessels reference has particularly to be made to the recent work of Hürthle and v. Frey, and to that of Roy and Adami in England.

Simultaneous measurements of the amount of the variations in pressure can only be done in the physiological laboratory; I shall, therefore, merely give their results where useful for the purpose of a better understanding of the cardiographic and sphygmographic tracing, and I will not attempt to describe the different forms of manometers employed, though, according to the various kinds of instruments used, rather different tracings are obtained of the intra-ventricular pressure. V. Frey and Krehl say that the tracings obtained by other instruments than their own are owing to the use of an open sound, which, being inserted too far into the ventricle, is liable to be blocked by the contraction of the papillary muscles. With

[7] Archiv für Pathologische Anatomie, 1871, p. 540.
[8] Deutsches Archiv für Klinische Medicin, 1875.
[9] Giraud Teulon, Gazette Médicale de Paris, 1856.
[10] The Practitioner, 1890.
[11] Brit. Med. Jour., 1891, p. 1117.
[12] Hesse, Archiv für Anatomie und Physiologie, 1880, p. 338; and Krehl, Abhandlungen der Sächischen Gesellschaft der Wissenschaften, 1891. p. 348.

regard to this criticism it is remarked by Tigerstedt that it does not apply to the results of Chauveau and Marey, since those investigators used a closed bulb filled with air, which was not entirely driven out when the bulb lay in the ventricle during systole. Owing to the disturbance produced in the heart by the insertion into it of sounds through the jugular vein or carotid artery, it is difficult to estimate by this means the maximum pressure within the cavities. In the right auricle of the dog this is estimated at about 20 mm. of mercury.[13] Jager's estimate from three experiments is as follows:

	I.	II.	III.	
Left ventricle ...	234	174	176	mm. of mercury.
Aorta	212	162	158	
Right ventricle...	28	44	72	

The pressure in the right ventricle as compared with that in the left, according to different writers, is as 1 to 3, 1 to 4, or 1 to 5. The intra-ventricular pressure curves of Chauveau and Marey, and also those of Fredericq and Hürthle, show a slight upheaval just before the main rise of pressure, attributable to the alteration in the ventricular pressure due to the contraction of the auricle. The degree of its separation from the main rise is dependent on the rapidity with which that rise ensues, and it may thus be merged in the upstroke or altogether lost. The same slight upheaval may be noticed in some tracings of the pressure within the aorta and carotids. Hürthle has obtained it in dogs with very low aortic pressure, showing that the influence of a sudden rise of pressure within the ventricle caused by the auricular contraction may be felt within the aorta. It is known that the auriculo-ventricular valves close at the commencement of ventricular systole; the time of their opening is inferentially assumed to occur at the commencement of diastole. In considering the arterial pulse wave it is important to know at what point of the heart tracing the semilunar valves open and blood enters the aorta, and also at what point the valves close. The arterial pulse can, if this be known, be divided into a systolic and a diastolic portion.

With regard to the opening of the semilunar valves after ventricular contraction commences, a certain time must elapse before the pressure within the ventricle rises to such a point as to overcome the pressure within the aorta. Until this point is reached the semilunar valves must necessarily be assumed to be closed. According to Chauveau and Marey, in the horse the rise of pressure in the aorta begins about

0·1″ after the commencement of ventricular contraction,[14] and the pressure curve of the ventricle does not show a disturbance at this juncture. The time difference between the commencement of rise of pressure in the ventricle and the expansion of the aorta, according to Hürthle,[15] is in the dog only from 0·02″ to 0·04″, the interval being less the lower the blood pressure in the aorta. We must take into consideration that the pressure remaining within the ventricle at the end of diastole may influence the length of time which elapses between the commencement of contraction in the ventricle and the rise of pressure in the aorta ; the state of the auriculo-ventricular valves, therefore, and the blood pressure within the auricle and pulmonary veins may also affect the time of delay before the aortic rise. The time of delay is, however, little noticeable in comparison with large variations in pressure. By comparative simultaneous tracings of the heart's apex and the carotid pulse in man, and allowing for the delay in transmission along the artery from the semilunar valves to the point of the carotid artery whence the tracing is taken, we find that the semilunar valves open at about the summit of the first rise in the cardiac tracing, and that while the blood is entering the aorta the direction of the tracing is changed, continuing more or less parallel with the base line—i.e., the horizontal. The delay in transmission from the semilunar valves to the carotid is thus accounted for inferentially. At every beat of the heart blood enters the aorta, and a wave is set up in the arteries, this being of course felt earlier in the carotids than in the radial artery, the time difference between these two points being about 0·0786″. The length between the two points is about 60 cm. The length of vessel from the semilunar valves to the carotid is about one-third that from the carotid to the radial—i.e., about 20 cm.—and the time occupied in traversing it is inferentially one-third of 0·0786″, or 0·0262″, and from the semilunar valves to the radial 0·0786″ + 0·0262″ = 0·1048″.

[13] Goltz and Gaule, Archiv für die Gesammte Physiologie, 1878, p. 106; Magini, Archivio Italiano de Biologie, 1887, p. 127.
[14] Mémoires de l'Académie de Médecine. 1863, pp. 305-6.
[15] Archiv für die Gesammte Physiologie, No. 49, p. 56 et seq.

If one takes a synchronous tracing of the apex-beat and carotid pulse it is found that the apex curve begins to rise considerably earlier than the carotid pulse—i.e., much earlier than 0·0262″, and more like 0·1182″. Subtracting 0·0262″ for the time of transmission between the semilunar valves and carotid, a time of 0·092″ remains, which must be purely cardiac. This is the time during which the intra-ventricular pressure is rising to overcome the aortic pressure ; in other words, it is the time from the commencement of ventricular contraction to the opening of the semilunar valves. This time is called by German writers the *Anspannungszeit*, or time of extension or stretching, or *Verschlusszeit* or time of confinement, and by Keyt it is termed more conveniently and clearly the "præsphygmic" interval, since in observations on the heart and pulse it is frequently necessary to consider or to eliminate the præsphygmic interval. Tigerstedt gives the estimations for the interval of various authorities :—

Præsphygmic interval in seconds.	Authorities.
0·8	Marey.
0·073	Rive.
0·096–0·087	Edgren.
0·07	Grunmach.
0·054	Keyt.

The interval varies inversely as the pulse frequency, being according to Keyt about one-tenth of the systolic period. The semilunar valves must close when the aortic pressure exceeds that in the left ventricle. Whether the second sound is coincident with their closure is not made out; it may occur just subsequently to their closure by the vortices set up behind the valves, the second sound being coincident with their tension, though not with their closure. The time of closure is to be found by comparative estimation of the pressures in the ventricle and aorta. By this means v. Frey has estimated the closure to take place in the upper half of the drop in the apex tracing. Hürthle [16] places it shortly after the commencement of relaxation of the ventricle. These estimations are probably correct, though Chauveau and Marey place it much lower down the limb. The outflow of blood from the ventricle lasts, then, from the opening of the semilunar valves to this point. This period is termed by German writers the *Austreibungszeit*, or time of expulsion. Hürthle remarks that the duration of this period only varies very slightly if the pressure within the aorta is increased (0·178″ to 0·195″, dog). According to Roy [17] the fibres of the heart wall shorten less during contraction when the arterial blood pressure is high. This is not to say that their persistence in contraction or, in other words, the duration of systole is shortened. The effect, however, of the diminished shortening of the muscular fibres of which Roy speaks is to increase the quantity of residual blood which is left in the ventricle at the end of each systole. Ordinarily during systole "the lower part of the ventricular cavity closes completely, the musculi papillares coming into contact with one another; the upper part of the cavity, however, lying between the valves and the papillary muscles, does not become emptied." [18] This residual blood being increased in quantity, compensation has to occur by increased dilatation. Up to an undefined point this dilatation may be physiological, but it is easy to see that it may become pathological. It may indeed in excess lead to deficient closure of the auriculo-ventricular valves, so that we can in this manner obtain a mitral incompetency from simple increase in the aortic blood pressure. Since cardiographic tracings are readily obtainable it is important to consider the apex-beat somewhat closely and to see how far the curve represents a curve of intra-ventricular pressure and in what way it varies with an increase of frequency.

Contrary to the generally accepted view the apex-beat is stated by Mariannini and Namias [19] in 86 per cent. of women and in 62 per cent. of men to be situated in the fourth intercostal space. I think this cannot be so with English people, with whom the apex-beat is certainly more frequently felt in the fifth intercostal space. The apex tends to fall away from the chest wall when the patient is lying flat on his back. When in this position, if he rolls over on to the right side the apex-beat may disappear under the sternum. If he rolls to the left the apex comes most closely into apposition with the chest wall. On this

[16] Archiv für Physiologie, No. 49, p. 45.
[17] The Practitioner, 1888, p. 347.
[18] Brit. Med. Jour., Dec. 15th, 1888.
[19] Archivio Italiano de Biologie, 1883, p. 143.

account, and because when so lying the respiratory movements of that side of the thorax are limited, the last is the most convenient position for taking cardiographic tracings, the left arm being flexed and the palm of the hand lying

under the left cheek. The apex-beat is caused by the sudden hardening of the heart in contraction against its contents, and by the apex being thrown forwards and upwards as the heart rotates on its own axis to the right, while the apex comes into a perpendicular with the basal openings. For the reasons which I have already given we can exclude the participation of extension and recoil of the aorta. The base of the heart may indeed approach the apex in systole, partly owing to these causes, but the apex is not thrust against the parietes of the chest by this means. A distinct apex-beat occurs in an excised heart emptied of blood, though the tracing, it is true, obtained from a bloodless heart is not the same as when blood is circulating through it. The tracing of the apex-beat is, by comparing it with tracings taken from the wall of the exposed heart, shown to be a tracing of ventricular muscular contraction. How far this is modified by the blood pressure within the ventricles becomes a matter of exceeding interest, the question whether the heart tracing is any guide to a knowledge of intra-ventricular states being all-important if it is to assist us in our physical examination of the sick. The general correspondence of synchronously obtained curves of ventricular contraction and of intra-ventricular blood pressure is so close as to be in favour of the view that a ventricular apex curve, or "cardiogram," is indeed, to some extent, a curve of intra-ventricular pressure.

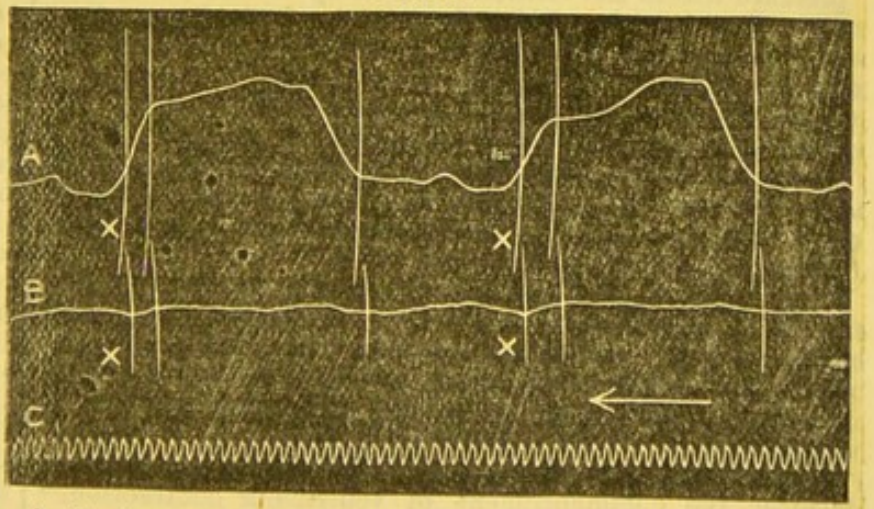
From cardiograms obtained from the wall of the exposed heart Roy and Adami give a scheme of three forms of tracings obtained by exerting different grades of pressure on the ventricle. It will be seen that when there is any approach to a distinct angle of drop consequent on the relaxation of the ventricle the duration of the whole period of contraction is perfectly measurable, the point of rise corresponding in each case. The curve indicated as No. 1 would be discarded as fallacious by any experienced cardiographist. With his cardiograph he would exercise more pressure on the interspace until he obtained a sharper angle of drop, at the least as in II. It is a matter for regret that, because such a tracing as No. 1 can be obtained by using light pressure, Roy and Adami should draw the conclusion that it is difficult, and in most cases impossible, to measure from the cardiogram the different phases of the heart's revolution. Such tracings as that in No. 1 either need not be obtained or may be rejected. Moreover, the assertion that the duration of systole of the ventricle can as a rule be exactly measured is greatly strengthened by numerous and conclusive experiments, which show that in the same individual the duration in time of ventricular contraction for any increase in pulse frequency undergoes a steady and uniform diminution, and that there is no difficulty or source of error in measuring these changes by means of acceptable cardiographic tracings.[20] That the cardiographic tracing varies as it is obtained from different parts of the exposed heart wall (v. Frey and others) need not concern us, as we have to deal with the apex-beat only. Extreme physiological accuracy we cannot hope to attain, but by comparison of contraction curves and of pressure curves it is obvious that we are in possession of means of observation of a most valuable kind. The curve of muscular contraction is modified by the contraction of the muscle on its contents, as is seen by comparison of the full and empty heart during contraction. Hürthle[21] thinks that the duration of the cardiogram sufficiently corresponds to the pressure curve of the heart, while v. Frey also thinks that the cardiogram and

contraction curve taken from the exposed heart are of about the same duration, yet he does not think that much information can be obtained from the cardiogram as to the course of pressure in the ventricle.[22] From the cardiogram nothing can be learned as to the time of closure or of opening of the auriculo-ventricular valves. The time of opening or of closing of the semilunar valves has been the subject of continual discussions. According to Edgren and v. Martius, if we take a simultaneous tracing from the heart and carotid and allow 0·0262″ for transit of wave from the heart to the point whence the carotid trace is taken, we find that the commencement of the rise of pressure in the aorta corresponds to the top of the first rise in the heart tracing, and that con-

[20] See table of duration of systole for different pulse frequencies.
[21] Loc. cit., No. 49, p. 93.
[22] Ibid., p. 118.

sequently it is at this point that the semilunar valves are opened. Hilger[23] places their opening 0·02″ to 0·01″ before the point is reached. The moment of closure of the semilunar valves is estimated by a comparison of the pressure curves from the aorta and left ventricle. The point at which the aortic pressure is suddenly in excess is somewhere in the line of drop. By marking off the first and second sounds as they are heard, and continuing to do so rhythmically in order to avoid as far as possible error due to the personal equation of the observer (from 15″ to 20″), attempts have been made to obtain the exact point of closure. The method is so full of error that the results are not to be trusted, but the moment of closure may perhaps safely be assumed to be in the line of drop—higher according to Marey, lower according to Edgren. Whether it occurs in the first or second half of the descending line is, according to Tigerstedt, not to be decided owing to errors in the method of marking. I am glad to be able to show a tracing which I have myself obtained from a patient, which is of much interest in this relation. A sharp "knick" was felt by the finger when pressed into the second intercostal space near the sternum, clearly corresponding to the closure of the semilunar (pulmonary) valves. Together with an apex cardiogram a simultaneous tracing was obtained from this point by means of a spring and air transmission. It will be seen that the "knick" in question corresponds in time with the lower part of the descending limb of the cardiogram.

FIG. 1.



A, Cardiogram. B, Second intercostal space. C, Harmonium reed. The arrow indicates the direction in which the tracing has been made.

About halfway down the descending limb of the cardiogram a slight bulge is often found. On Edgren's tracing of a normal curve he gets this lower down the limb, so that it appears almost to be an upheaval at the end of the drop. Neither of these points, however, appears to be synchronous with the closure of the semilunar valves, and the appearance is probably caused by the rush of blood from the auricle into the ventricle, due to the relative change of pressure between the two cavities owing to ventricular relaxation.[24] Previous to the great main rise in the cardiogram there is an upheaval corresponding to the contraction of the auricle, which immediately precedes, and is even in continuity with, ventricular systole. In my experience this may lead to error in measurement of the duration of ventricular systole. With light pressure of the cardiograph it may often merge into the ascending limb, and being measured as systolic an excessive length of ventricular systole may be simulated. For all purposes of measurement it is important that the cardiogram should be very sharp and clear, and the main systolic rise should take place from a distinct angle, showing that it is due to a marked and sudden change. The upheaval due to auricular contraction is by no means always to be obtained. It is necessary to have a favourable case, a sensitive instrument, and the luck which is born of experience.

In the cardiographic tracing on the next page we have, therefore, the following points to consider :—

As to measurement of the duration of ventricular systole, if we assume the valves to close somewhere in the line of the descending limb it should, to be exact, be measured from B to this point. Since this point is not visible on the tracing, and since we know that the angle (at D) where the sudden drop begins certainly corresponds to the cessation of active contraction of the ventricle, it seems to be more prudent to

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[23] Zeitschrift für Klinische Medicin, 1891, p. 163.
[24] Fredericq, Centralblatt für Physiologie, 1892, p. 257.

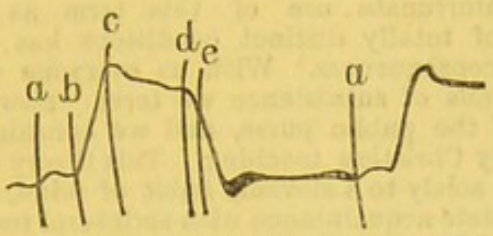
measure to the angle of the drop when comparing relative "durations of systole" one with another. Baxt and v. Frey measure to the bottom of the drop. Volkmann and Donders in their measurements of systole refer to the interval between the two heart sounds. The measurement from the beginning of the main systolic rise to the angle of the drop has, however, the great authority of Hürthle and of Burdon Sanderson, and whenever speaking of the "duration of systole" I mean, and think that we should for practical purposes be understood to mean, the time measurement between these points.

I have carefully said that the angle of drop corresponds to the cessation of active contraction of the ventricle, as it appears that although the actual expulsion of blood ceases at this point, yet the muscle does not suddenly relax, a certain short time (*Entspannungszeit*) intervening. Any calculations which endeavour to include this period in the systole, or which are made by estimating the time interval between the first and second sounds, will be somewhat in excess of those which I have published in previous papers.

In conclusion (I quote from Tigerstedt) "the apex curve, as was first remarked by Chauveau and Marey, represents a combined pressure and volume curve of the ventricle. It is a pressure curve, for the knob placed against the chest wall exerts a pressure against which the heart by systole performs work. At the same time it is a curve of volume, in so far as it is also influenced by the changes in the volume of the heart. By the filling of the heart during diastole the curve gradually rises from the abscissa ; by the sudden emptying of the

ventricle when the semilunar valves are opened the pressure of the heart against the chest wall will be somewhat less, and the lever can now no more follow the progressive increase of ventricular pressure, but describes a plateau." Volkmann

FIG. 2.



a, *b*, auricular systole; *b*, *c*, *Verschlusszeit*, or time of confinement; *c*, *d*, *Austreibungszeit*, or period of expulsion; *d*, points of cessation. Here a drop occurs, at the bottom of which is usually a notch due to the moment of inertia of the lever. Between *e*, *a*, a slight rise takes place as the ventricle slowly fills and the blood pressure increases, till at *a* it is again suddenly raised by contraction of the auricle.

for a pulse frequency of 84 gives the interval between the first and second sounds as 0·375″, and the diastole as 0·380″. Donders measures the same interval—from 74 to 96 per minute—as varying from 0 327″ to 0 301″. For a difference of from 63 to 124 per minute in the same case the duration of systole sank from 0·382″ to 0 199″. Landois gives the duration of systole between 53 and 113 from 0·346″ to 0·190″, and Edgren gives the interval between the heart sounds for a pulse-rate of 70 as 0·379″. Thurston for pulse variation from 47 to 128 gives the duration of systole between 0 347″ and 0·256″. Generally speaking, and this is also true of variations arising from artificial stimulation of the inhibitory or of the acceleratory nerves, the difference between a slow and rapid pulse frequency is more at the expense of diastole than of systole. It will be realised that among all these somewhat conflicting statements it is very important to determine some standard of duration of systole for different pulse frequencies. Only by so doing, or by attempting so to do, shall we be able to institute useful comparisons of the cases we meet with. So long ago as 1882, in a report to the Scientific Grants Committee of the British Medical Association (Aug. 19th) I endeavoured to give the average duration of systole for every increase of five beats in pulse frequency per minute from 46 to 130, the measurements being taken from the initial systolic rise to the sharp angle of drop, which, as I have said, is taken by Burdon Sanderson and by Hürthle as the termination of outflow of blood from the ventricle. The range was from 0 36″ to 0 21.″ There is no absolutely fixed rate of diminution. For every five beats the diminution is about 0 0085 ″ My table, which has been republished in "Landois and Stirling's Text-book of Human Physiology," p 97, has, however, been of the greatest use to me for purposes of comparison, and for establishing abnormalities, and its general correctness becomes more evident to me as my experience becomes more extended. I therefore give the table again, together with another table which is artificial and diagrammatic, but which will immensely facilitate the calculations which have to be made in measuring cardiograms and estimating pulse frequency per minute.

Experimental Table.

Frequency per minute.	Duration of systole in seconds

130	.....	0·2100 (exertion).
116 to 120	.....	0·2350 (mixed cases).
111 to 115	.....	0·2475 (mixed cases).
106 to 110	.....	0·2675 (exertion).
101 to 105	.....	0·2543 (Turkish bath).
96 to 100	.....	0·2540 (Turkish bath).
96 to 100	.....	0·2730 (normal).
91 to 95	.....	0·2690 (mixed cases).
86 to 90	.....	0·2800 (Turkish bath).
86 to 90	.....	0·3200 (exertion and normal).
81 to 85	.....	0·3030 (Turkish bath).
81 to 85	.....	0·3250 (exertion and normal).
76 to 80	.....	0·3032 (Turkish bath).
76 to 80	.....	0·3300 (exertion and normal).
71 to 75	.....	0·3033 (Turkish bath).
66 to 70	.....	0·3200 (mixed cases).
61 to 65	.....	0·3200 (mixed cases).
56 to 60	.....	0·3460 (normal).
56 to 60	.....	0·3200 (Turkish bath).
51 to 55	.....	0·3425 (normal).
46 to 50	.....	0·3600 (normal).

Diagrammatic Table for facilitating Calculations.

Time of total revolution in seconds.	Time of systole in seconds.	Time of diastole in seconds.	Pulse-rate per minute.
1·333	0·3600	0·9733	45
1·200	0·3515	0·8485	50
1·0909	0·3430	0·7479	55
1·000	0·3345	0·6655	60
0·9230	0·3260	0·5970	65
0·8570	0·3175	0·5395	70
0·8000	0·3090	0·4910	75
0·7500	0·3005	0·4495	80
0·7060	0·2920	0·4140	85
0·666	0·2835	0·3831	90
0·6316	0·2750	0·3566	95
0·6000	0·2665	0·3335	100
0·5701	0·2580	0·3121	105
0·5454	0·2495	0·2959	110
0·5217	0·2410	0·2807	115
0·5000	0·2325	0·2675	120
0·4800	0·2240	0·2560	125
0·4615	0·2155	0·2460	130
0·444	0·2070	0·2374	135
0·4286	0·1985	0·2301	140
0·4138	0·1900	0·2238	145
0·4000	0·1815	0·2185	150
0·3871	0·1730	0·2140	155
0·3750	0·1645	0·2105	160
0·3636	0·1560	0·2076	165
0·3530	0·1475	0·2055	170
0·3428	0·1390	0·2038	175
0·3333	0·1305	0·2028	180
0·3246	0·1220	0·2026	185
0·3158	0·1135	0·2023	190
0·3077	0·1050	0·2027	195
0·3000	0·0965	0·2023	200

The time of rest and of work in the day, and the considerations thereto belonging, and the influence on fatigue of the heart muscle by merely slowing the pulse by means of drugs, or by lengthening the systole or diastole relatively to each other, become extremely interesting from this point of view.

The Goulstonian Lectures

ON

THE PHYSICS OF THE CIRCULATION.

Delivered before the Royal College of Physicians at the Examination Hall, Victoria Embankment, on March 1st, 1894,

BY PAUL M. CHAPMAN, M.D., F.R.C.P. LOND.,
PHYSICIAN TO THE HEREFORD GENERAL INFIRMARY.

LECTURE II.

MR. PRESIDENT AND FELLOWS,—With regard to the behaviour of the heart during diastole, until the last fifteen years it was generally assumed that the ventricle relaxed, but did not actively dilate, and exerted no suction power during diastole. Goltz and Gaule, in 1878, found that there was a negative diastolic pressure in the dog's ventricle when acting well and strongly. When the heart's movements were weak, and the heart no longer completely emptied itself during systole, the suction power diminished. De Jager[3] found in the right ventricle a negative diastolic pressure of from 5 mm. to 38 mm. Hg., and in the right auricle a negative pressure of from 2 mm. to 6 mm. Hg. In all these experiments the heart was laid bare to avoid suction due to respiratory movement. It is difficult to understand how the elasticity of the muscular fibres can suffice to bring this about. Stefani,[4] for the elucidation of this point, performed an ingenious experiment. He filled the pericardium with water, and estimated the pressure which was necessary to hinder the diastolic expansion of the ventricle. He found that this must be much greater when the vagi were intact than when they were divided. The vagi, therefore, appear to influence the active expansion of the ventricle. Whether, as Stefani appears to assume, this is due to a direct influence of the vagi on the heart muscle, or whether the section of the vagi lowers the blood pressure only, is not quite satisfactorily proved.

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[2] THE LANCET, Oct. 28th, 1893.
[3] Archiv für die Gesammte Physiologie, 1883.
[4] "Intorno al Modo con cui il Vago agisce sul Cuore." Memoria letta all' Accademia di Ferrara nella, 1 Dicembre, 1882; Cardio volume, press-pericardica, 1891.

It is theoretically possible that if the contraction of the circular fibres of the ventricle should persist longer than that of the longitudinal fibres a negative pressure might be produced. This has been pointed out by v. Frey.[5] Rolleston[6] has remarked that the narrowing of the venous orifices through the contraction of the circular fibres can give rise to a suction in the ventricle if this occurs later or persists longer than the contraction of the remainder of the ventricular wall. Gaule and Mink[7] refer to the arterial openings. Here the muscular fibres take their origin from the fibrous ring in which the aorta is sunk, and surround the ventricle in spiral fashion; so that at the moment when the ring is widened by the filling of the aortic sinus after the occurrence of the

closure of the semilunar valves an unfolding of the point of origin of the muscular fibres occurs, and thereby an unwinding of the spiral. Lastly, the flow of blood into the coronary arteries tends to straighten their curve, and so leads to an enlargement of the ventricular cavity (Brücke, Rolleston). The amount of blood expelled from the heart, and the method by which the estimation is arrived at, are familiar to everybody from the text-books of physiology. If we assume the quantity expelled to be four ounces, and we know the velocity attained by the blood to be about 0·5 metres per second, and the pressure in the aorta to be about 150 mm. Hg, we can, by multiplying these two together, estimate the amount of work performed by the heart. If there was no obstacle arising from the high pressure in the aorta the amount of force necessary for the expelled blood to attain a velocity of 0·5 metre would be very small—viz, from 0·64 to 1·28 gramme metres. To overcome, however, the high aortic pressure a force of from 102·0 to 204 gramme metres is needed. The total work of the heart is therefore a union of these two—viz, from 102·64 to 205·28 gramme metres. By far the largest part of the work of the heart is therefore expended in overcoming the obstacles in the vascular system. We do not sufficiently realise ourselves what a very small part of the heart's work is available for imparting to the blood its requisite velocity. It is but about 1-200th part of its whole work. The importance of treasuring this small margin of force, and the readiness with which the blood flow can be diminished by any increase in the vascular pressure, should be continually borne in mind. To sustain the equality of the circulation the right ventricle should expel at each systole the same quantity of blood as the left ventricle. Assuming the pressure in the pulmonary artery to be two-fifths of that in the aorta, the work expended by the right ventricle would be from 40·8 to 81·6 gramme metres. According to physiological experiments, while the *force* of contraction varies, the duration of ventricular systole is independent of arterial or venous blood pressure. My own table seems to show considerable discrepancies between lengths of duration of systole under exertion (when the blood pressure is high) and under the influence of the Turkish bath (when the pressure is low and the cardiogram and pulse tend to dicrotism), but the shortened systole may be due to nervous influences. In an experiment made by me some years ago both the subclavians and crurals and the abdominal aorta were simultaneously compressed without altering in any way the duration of systole. I have met with very short systole in pernicious anæmia and in fatty heart. In these, as in all other cases, it is probable that marked alterations in the duration of ventricular systole must be taken as evidence of want of nutrition of the heart wall or of the presence in the blood of toxic substances. A rise or fall of pressure in the aorta will, on the other hand, affect the *frequency* of the pulse, which sinks by rise of pressure and rises by fall of pressure through the influence of stimulation of the vagus centre or of the inhibitory nerves respectively.

In natural breathing, when the air vesicles are dilated by inspiration, the pressure on all sides of the lung capillaries is less, and the capillaries are not only lengthened but widened out in all directions; their capacity is, therefore, made greater and the blood pressure within them sinks, involving negative pressure in the large veins which open into the heart and facilitating onflow along the pulmonary arteries. Blood, therefore, streams into the lung. If, at the termina-

tion of expiration, the quantity of blood in the lungs is from one-fifteenth to one-eighteenth of the total quantity of blood in the body, at the termination of inspiration it will be from one-twelfth to one-thirteenth. It is obvious that, if the

[5] Die Untersuchung des Pulses, p. 94.
[6] Journal of Physiology, 1887, pp. 25–254.
[7] Centralblatt für Physiologie, 1890.

lungs are distended by artificial inflation through the trachea, the intra-vesicular pressure will be positive from the first and will be very great when the distension is complete. The effect of this will be to hinder the onflow from the pulmonary arteries and to hasten the onflow along the veins, the total amount of blood in the lung in full artificial inflation being only about one-sixteenth of the total mass of blood. Now, in natural inspiration the pleural negative pressure is increased. This negative pressure acts in a contrary direction to and hinders the heart's systole, giving the right ventricle some slight trouble to empty itself. Diastole, on the other hand, is facilitated. In ordinary unforced inspiration these influences are, however, very slight, the main fact being that, in inspiration, the suction power brings such an increased flow of blood to the heart that the right ventricle is distended with blood previous to contraction, and therefore more completely fills the pulmonary artery by its systole. In expiration, on the other hand, the heart's systole is favoured and diastole is hindered. The flow of blood into the left heart is less, and there being less room in the lung capillaries the blood pressure in the pulmonary artery rises. An interesting observation was made by Lichtheim, who found that after ligaturing completely one pulmonary artery the same amount of blood continued to reach the left heart and the aortic pressure was not diminished. Even after a large part of the remaining pulmonary artery was rendered impervious by the creation of artificial emboli, not until about half the lung was rendered bloodless did the aortic pressure sink at all.[8] The pulmonary artery appears to be subject to very wide variations of distension. The actual work done, however, by the left ventricle is subject to much greater variations than that done by the right ventricle. Waller[9] by exciting the spinal cord obtained a very strong contraction of the peripheral vessels of the systemic circulation, whereby the aortic pressure was greatly increased and the left ventricle could not empty itself. By this means the pressure in the left auricle was raised from 25 mm. to 30 mm. Hg., and its walls ceased to contract. During all this time the right auricle and ventricle continued to contract normally. The distensible capacity of the pulmonary artery relieves it from any great variation in pressure. Tigerstedt remarks that this capacity and distensibility play the same *rôle* in the smaller circulation as the liver does in the larger. The blood collects in either, to the relief of the cardiac circulation, which would otherwise be constantly embarrassed.

We are now prepared to consider the effect of the variations of the pulmonary circulation in respiration on the pressure in the systemic arteries. In natural inspiration the first effect of the negative pressure in the intra-thoracic capillaries and veins is to diminish the flow out of the veins into the left auricle; the aortic pressure consequently falls. There is a second effect a moment later, for, the flow of blood into the distended pulmonary veins and capillaries being facilitated, the lung is gorged, blood pressure rises, the left auricle becomes distended by an increased flow of blood, and the aortic pressure rises. In natural expiration the capacity of the lung capillaries is diminished and pressure is exerted

on the veins; the first effect is a rise of blood pressure in the veins, more blood enters the left auricle, and the aortic pressure consequently rises. The second effect occurs a moment later, less blood enters the contracted capillaries, the blood pressure in the veins falls, less blood enters the left auricle, and the aortic pressure falls. It follows that in prolonged laboured inspiration the aortic pressure rises, while it falls in prolonged expiration. I may here mention, though it is not a question of mechanics, the well-known fact, dependent on nervous influences, that the pulse frequency is increased in inspiration and diminished during expiration. The blood supplied to the heart and the corresponding extent of excursion in the heart's movements also vary in the same way. The respiratory movements of course do not affect the intra-thoracic pressure only, but also the abdominal. The abdominal pressure rises, while the intra-thoracic pressure falls; the effect of this is to hasten the blood onwards towards the heart and to diminish the return of blood from the legs to the abdomen. This may contribute to the rise in inspiration of the aortic pressure. In slow, deep respiration the maximum aortic pressure is reached at the commencement of expiration, the minimum at the beginning of inspiration. It is clear that to maintain an equable circulation the heart must be supplied with the same

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[8] Die Störungen des Lungenkreislaufs. Berlin, 1876.
[9] Archiv für Anatomie und Physiologie, 1888.

quantity of blood as is driven from it at each systole. It is customary to imagine that the difficulty of return to the thorax of a column of blood extending from the foot to the heart is very great, and that it is only overcome by the suction power of inspiration, and of cardiac diastole, combined with the effect of lateral pressure of the acting muscles and of the pulsating arteries against the blood column in the veins which lie in contact with them. That the difficulty is not so great as is generally imagined will be apparent from the consideration that the mechanical obstruction to the return of fluid in the ascending limb of an arc is neutralised by the increased facility offered for the flow of blood in the descending limb. As Tigerstedt points out, when the thorax is opened so as to exclude any inspiratory suction power the blood returns to the heart without the least difficulty; the return is, indeed, favoured by the greater sectional area existing in the larger veins compared with that in the arteries.

Before entering upon the study of the pulse wave in the arteries it is well to consider what obstacle the capillaries offer to the wave of impulse sent through the blood. Is the wave lost before it reaches the capillaries? If not, what is the nature of the obstacle which they offer to the impacting wave? Part of this question will be considered when we discuss the movements of waves in elastic tubes, but some facts relating to the capillaries should be recapitulated.

Owing to our visual memory of the capillary network as seen through the microscope, we are, I think, inclined to imagine the length of the capillaries—that is to say, the distance between the finest arterioles and the extreme venous radicles—to be much longer than is the case. The distance has been estimated at about 0·2 mm. Through this short distance the red corpuscles move with a velocity of a millimetre in one second or in half a second. The red corpuscles, because of their large size and specific gravity, take the axis of the stream owing to purely mechanical reasons. Owing to mechanical reasons also, the corpuscles being

flat and the pressure behind having a tendency to be equalised over their surface, they travel flat to the stream, with the shortest diameter parallel to the direction of blood flow. The white corpuscles, being rounder and smaller and lighter, mechanically take the wall, and, getting into the peripheral part of the stream, since the rapidity of flow is greatest on that side of them which is nearest to the axis of the stream, they are rolled along the wall of the vessel. Now, since the red corpuscles take the axis of the stream, and the mean velocity is half that of the axial stream, the mean velocity of blood flow will be about half that of the red corpuscles. We know that the velocity of blood flow in the aorta is from 0 3 to 0 5 up to 1·0 metre per second, while in the same space of time the blood in the capillaries traverses only about as many millimetres. This is owing partly to the much larger section of the whole capillary system, compared with that of the aorta, and partly to the loss of velocity through friction, which, indeed, is not a "loss" in the ordinary sense, since it becomes disponible as heat.

It is now known that the capillary walls are contractile, and that the diameter of the capillaries varies independently of the arterial blood pressure. When they thus vary it is probably in response to the chemical constitution of the blood, which is altered in correspondence with the varying conditions of tissue change in the part. Irritation of the nerves which supply the capillaries has not yet been directly shown to influence their diameter, though variations in their diameter may assist the arterioles in regulating the blood supply to an organ. The capacity of the arterioles and capillaries considered as a whole is so great that, were they all filled with blood to the fullest capacity, the quantity of blood in the body would not be sufficient to maintain the normal aortic pressure. The pressure and capacity are constantly varying in different parts of the arterial and capillary circulation, accordingly as each organ is called into greater or subsides into less activity. By this arrangement the aortic pressure remains the same and contributes to the increased flow of blood to the part excited to activity. These facts explain in some measure the beneficial action of some of the drugs called "derivatives," such as ipecacuanha, which, by dilating the vessels generally, equalise the circulation and relieve some part which is receiving too great a supply of blood. The actual blood pressure within the capillaries has been estimated by observing what superficial pressure is necessary to deprive some part of a mucous membrane lying against a hard basis of its colour. Such a situation is found in the gum of the rabbit, where v. Kries found that the capillary pressure was about 33 mm. Hg, or about one-third to two-sevenths of the aortic pressure.

Owing to the independence in contractility shown by the capillaries and to the large size of the individual red blood corpuscles compared with their lumen, together with the fact that we are not here dealing with a mere channel of conveyance, but with a part actively engaged in tissue changes, to which the variations in the conditions of the arteries are merely subservient and contributory, and also considering the fact that pulsation is not felt in the veins on the other side of the capillary network, we may be sure that if an impulse wave arising in the aorta is of such a length as to reach this point it will meet with such an obstacle as to be possibly reflected back again along the channel by which it came. Still carefully distinguishing between velocity of

blood-flow and velocity of transmission of pulse-wave, which latter will be considered later, we find, by the use of Ludwig's "Stromuhr," the mean velocity of the blood in the arteries in one second of time to be about 0·3 to 0·5 metre per second. The experiments made by Hering and Vierordt to estimate the time occupied by the blood in traversing a complete circuit are well known. A solution of ferrocyanide is injected into a jugular vein of one side and is detected after the sought-for lapse of time in the other jugular or in a crural vein. Hering has estimated the shortest time in which any portion of the blood completes a circuit to be 26·2″. This must not be taken as the average time of completing a circuit. As already said, the maximal velocity is attained only in the axis of a tube, and the minimal near the parietes, the mean velocity of blood flow being but the half of the maximal, or axial, velocity—that is to say, fifty or sixty seconds per circuit.

Vierordt[10] estimates that the mean duration of a circuit varies with different mammals and is shorter in small than in large mammals. He finds that the number of heart beats which is necessary in every animal to drive the blood through the whole circuit is independent of its size and is equal in all animals, being about 28 to 29 heart beats. Tigerstedt, however, finds that in the rabbit the number of heart beats necessary is 120, and nearly all his measurements differ so widely from those of Vierordt that the subject must be considered as being still under discussion.

The blood pressure alone gives no indication of the velocity of the blood, which may be slow or quick with a high pressure according as the obstacle is greater or less. This is excellently and usefully shown by Dr. Waller's table:[11]—

Heart Force.	Arterioles' Resistance.	Blood Pressure.	Blood Flow.
1. Constant	Increased	+	−
2. Constant	Diminished	−	+
3. Increased	Constant	+	+
4. Diminished	Constant	−	−
5. Increased	Diminished	+	+
6. Diminished	Increased	−	−
7. Increased	Increased	+	−
8. Diminished	Diminished	−	+

Inside rigid tubes of varying diameter, into one end of which fluid is injected, the velocity of the fluid is equally great in every section of the tube, but the side pressure grows less the further it is taken from the point of injection. The tension of the fluid in any part of a horizontal rigid tube is the side pressure, which can be measured by leading off perpendicular tubes from various points of its length. The larger the bore of the main tube the less is the obstruction, and therefore the lower the height of the fluid in the perpendiculars. The height of the fluid in the perpendiculars declines in relation to the distance from the initial pressure, owing to the increased friction of each added length of tube. The height of the fluid in the perpendicular tubes is a measurement of the side pressure in the main tube at that point and, of course, marks the excess of the inner pressure over the atmospheric pressure. If the sectional diameter of the tube varies, fluid still flows out at the open end in a constant stream, therefore in every unit of time the same quantity of fluid must flow onward in every section of the tube; the velocity in the different parts of the tube will, in consequence, be inversely as their sectional area. In this case, besides the loss of side pressure due to friction of the kind above mentioned, a further loss occurs from the friction arising at the junction of a wider with a narrower portion of the tube; if, on the other hand, a wider tube follows a narrower one

the pressure may either remain unchanged from what it

[10] Die Erscheinungen und Gesetze der Stromgeschwindigkeiten des Blutes, p. 56. Frankfort on-Maine, 1888.

[11] Handbook of Physiology, p. 60

was in the narrower portion or actually rise. If branched rigid tubes lead off from a main trunk there is a greater facility of flow through them than through a single tube. If their united section is larger than that of the main stem the quantity of fluid passing in each unit of time through the thus created larger section remains the same, but the velocity of flow is less, being inversely proportional to the larger sectional area; the relation between fluid and surface is, however, increased, owing to the smaller diameter of each of the main individual branches into which the stem divides, and therefore the obstacle interposed by friction is also greater. The increased friction is hence opposed to the increased facility of flow which would otherwise be the consequence of the larger sectional area. When the divided branches are gathered up again into one stem there are less friction, a smaller sectional area, and a greater velocity. If a simple continous stream flows through a system of elastic tubes the same considerations hold good and apply to the flow in the capillaries and veins. When fluid is driven into elastic tubes by intermittent rhythmical action, as it is into the arteries, special considerations arise. The fluid driven into the tube has not to displace the whole mass of fluid beyond it at one time. The blood driven into the aorta from the heart meets, it is true, with the inertia of the mass of fluid against which it impinges, with the friction in the many arterial branches, and with the obstruction of the capillaries, but the root of the aorta is distended by the increase of blood volume. This distension exerts a pressure on the enclosed fluid, which continues when the heart has ceased to contract, and consequently the blood continues to stream onward until the root of the aorta has returned to its former tension. The force of the heart is, in fact, transmuted into terms of elastic tension, which acts during the period of the heart's rest; the expenditure of work is, therefore, much less than if the whole mass of blood had to be moved forward *en masse*, as would be the case if the tubes were rigid.

As has been shown by Roy,[12] the arterial walls become more rigid as the pressure is raised above the general mean pressure of the artery of the animal from which it is taken. The rigidity begins in excess of a point up to which the distensibility of the artery increases with each increment of pressure, the maximum cubic extensibility of an artery lying about its ordinary mean interior pressure In a distended artery, in which the blood pressure is already in excess, the advent of an additional volume of blood causes the interior pressure to rise greatly in excess of the rise which would occur in the same artery if the blood pressure were lower and the elasticity greater. With higher arterial pressure, when the arterial walls are distended to the point of rigidity, the less can the artery be widened by the same addition of blood. I have already mentioned his remarks on the occurrence of dilatation of the heart from such high aortic pressures owing to incomplete emptying of the ventricle.

That many difficulties lie in the way of measuring, with complete accuracy, the blood pressure in the sick is not a reason why any approximate estimation of the pressure should be completely neglected, as is now almost universally done. A useful sphygmomanometer can be made for a few shillings, and its application would in most cases be accurate enough

if we could always feel, at the wrist or elsewhere, an easily accessible artery lying on a flat and rigid bed. It is better to employ a slight extension of the wrist, as, owing to the increased tension put upon the fascia, the amount of yielding beneath will then be reduced to a minimum. I have been using a sphygmomanometer made by myself on a simple plan suggested to me by Dr. Waller. A U-shaped piece of glass tubing, one limb of which is bent at a right angle, is connected by means of a perforated indiarubber cork with an ordinary indiarubber finger-stall. This is fixed on an upright frame scaled in millimetres. When the finger-stall is applied over an artery, pressure is exerted until the distal pulse beyond it is no longer perceptible. The ulnar artery should always be compressed in order to avoid the recurrent pulsation through the palmar arch, to which attention was first called by Dr. Waller, and which has since been described by Dr. Dickinson and by Dr. Douglas Powell. Since I have used the sphygmomanometer I have met with the phenomenon of recurrent pulse so frequently that I have ceased to consider it as being of much clinical significance, though Dr. Douglas Powell has described it specially in connexion with the pulse of high tension.

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[12] Journal of Physiology, vol. iii.

By direct measurement in mammals we know that the mean blood pressure is remarkably independent of the kind or size of the animal. By analogy we may assume that the aortic pressure in man varies between 100 and 200 mm. Hg, the mean pressure being about 150 mm. Hg.

In using the sphygmomanometer which I have just described we must allow for certain errors. According to von Basch,[13] in order to occlude an artery through the overlying skin, it is necessary to apply a pressure of from 6 to 8 mm. Hg higher than when the artery is exposed. Again, a certain amount of pressure is necessary to occlude even an empty exposed artery. V. Basch estimates this to be about 1 mm. Hg for a normal and about 5 mm. Hg for a sclerosed artery. We have therefore, to allow for :—

	Millimetres of Mercury.	
	Minimum.	Maximum.
1. Overlying skin	6	8
2. Unfavourable position of artery	20	60
3. Compressibility of arterial wall	1	5
4. Difficulty in estimating disappearance of pulse	5	5
	32	78

Tigerstedt concludes that the sphygmomanometer is suitable for estimations of the blood pressure on one and the self-same person within certain limits of time and thinks it has proved to be of great use in clinical medicine. I certainly think that when employed with due care it gives a much more accurate knowledge of the blood pressure in the radial than can be gained by the sense of touch, and I have used it as an indication for treatment with the best results. It would be interesting to ascertain how many physicians ever use one in their practice.

In clinically considering a low blood pressure we have to remember that the arterial blood pressure sinks—(1) when the quantity of blood expelled from the heart in a unit of time is less ; (2) when the energy of the heart declines ; and (3) when the vascular obstacles are less ; and that it rises when either of these is in excess.

If the venous supply of blood to the heart is sufficiently

voluminous and rapid, and the heart is acting with energy, a rise of arterial pressure may ensue from increased frequency of heart beats. The reverse is also true. It is a suggestive fact that the blood pressure in the aorta is not raised so high when the abdominal aorta is compressed as it is when the splanchnic nerves are strongly stimulated. In the first case a quantity of blood remains in the arterial system on the peripheral side of the ligature ; in the latter this blood is by the contraction of the arteries driven out into the venous system, and consequently a larger mass of blood returns to the heart in order to be driven into the aorta. It is only after the aortic pressure becomes very high that the heart begins to expel a smaller quantity of blood at each systole.

Both contracting auricles discharge their contents into the ventricle at about the same pressure (20 mm. Hg) ; we may assume, then, that this will represent the pressure in the ventricle just before its contraction. The pressure then rises in the ventricle until the semilunar valves are open, and continues to rise, after they are open, to a maximum, prior to the relaxation of the heart muscle. The higher the aortic pressure the greater is the work thrown upon the heart in raising the intra-ventricular pressure. It must be repeated that this extra work is not shown by an increase in the duration of the ventricular systole ; none of the many measurements I have made lend any support to such a view. It is said that the slope of the initial rise in the cardiac tracing is lower, but this may not always be the case, for the heart's increased energy is shown by the vigour of its contraction, which succeeds in doing a larger amount of work in the same period of time. Any diminution of work done is therefore to be expressed, not in terms of the time employed in ventricular contraction, but in terms of the reduced amount of blood expelled into the aorta, which, otherwise expressed, means that a larger amount of residual blood remains in the ventricle. If the supply of blood to the heart is kept up, the volume of expelled blood is, between from 58 to 147 mm. Hg., independent of the aortic pressure. Indeed Roy and Adami,[14] as also Johannsson and Tigerstedt, have shown that within certain limits the heart may expel a greater quantity of blood against an increased aortic pressure than against a lower pressure. The rule is, as

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[13] Berliner Klinische Wochenschrift, 1887.

[14] Brit. Med. Jour., 1888, p. 1321 et seq.; and Philosophical Transactions, 1892.

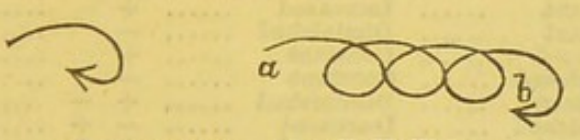
I have said, that the quantity of blood expelled at each systole is less when the aortic pressure becomes very high. This conclusion is borne out in another way. Hürthle[15] found that the variations in aortic pressure during a whole heart period were as a rule less with higher than with low pressures. Now we know that the extensibility of the aorta grows less with high pressures—that is to say, it becomes more like a rigid tube—and that consequently additions of blood from the heart must cause a much greater increase of pressure when the pressure is already high than when it is low. Since, however, it is shown by Hürthle that the aortic pressure varies within small limits only, it is to be concluded that when the aortic pressure is very high less blood is expelled into it at each systole than is the case with a low aortic pressure.

Alterations in the arterial pressure by variations in the quantity of the whole mass of blood through blood-letting or through transfusion are very transient. After blood-letting, fluid is quickly absorbed from the interstitial tissues, while the secretions from the kidneys, salivary glands, &c.,

are diminished. Transfusion, on the other hand, if excessive, may lead to rapid heart failure; if moderate, blood will collect in the distended liver and abdominal veins, while the urinary secretion is increased. Alterations in arterial blood pressure are, in short, much more dependent on the dilatation or contraction of the arterioles, arising from vaso-motor or other influences, than from these causes.

Before entering upon the study of the graphic representation of the pulse wave it is of advantage to consider shortly the nature of the wave movements of incompressible fluids when these are set up (1) with free surface, and (2) in elastic tubes. The general fact that the particles of the fluid set in motion describe an elliptical path, but are not themselves propagated onward with the travelling wave, is sufficiently familiar to need no further mention. The terms "positive wave" and "negative wave" require, however, some notice. Separating clearly the idea of an onward moving mass of fluid from the onward movements of a wave set up in it, we find that the particles of fluid participating in the wave describe, successively, elliptical paths, each of which may be divided into two kinds—viz., (1) one which lies above the ordinary level of the fluid, and (2) the other, which lies below the ordinary level of the fluid. The first is termed the "positive wave," the second the "negative wave." In the positive wave the particles of fluid are moving in the direction in which the wave is propagated, in the negative wave they are moving in the reverse direction.

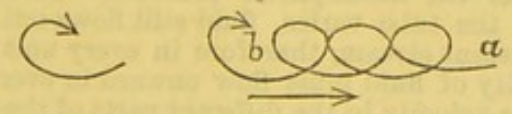
FIG. 3.



Positive wave. After E. H. Weber.

Owing to friction, the wave will be less in extent the further it is from the commencement of the tube, and will be so far deformed that the rise and fall will be less steep. Now, if an elastic tube is filled with water a wave may be set up in it either by suddenly injecting an addition of water at one end or by pinching the tube, in which case a positive wave will be caused, as in Fig. 3, or a wave may be caused by suddenly removing any compression from the other end. In the latter case, although the mass of fluid streams out of the tube in the same direction as it does in the first instance, a wave is propagated in the reverse direction towards the central end. This is a negative wave and can be better understood by reference to another figure. If a stream is set

FIG. 4.



Negative wave. After E. H. Weber.

flowing through an elastic tube open at its peripheral end, and the stream is then suddenly interrupted, a negative wave is set up which is carried onward to the periphery. If, again, water is thrown rhythmically into the tube from the central end, at every impulse a positive wave is

[15] Archiv für die Gesammte Physiologie, 1888.

propagated in the direction of the periphery, and at every cessation a negative wave is propagated, likewise in the direction of the periphery. Both these are called "primary waves," though the first is accompanied by a rise and the second by a fall of pressure. If the elastic tube is not long enough for the wave to be wholly extinguished by friction, and the peripheral end is closed, other waves, in a reverse direction, will be reflected from the closed end. For the sake of avoiding all confusion these reflected waves are not called "negative waves"—indeed, they are often "positive" in character—but "centripetal waves." A centripetal wave, in its turn, may reach the central end and be reflected from the central end again towards the periphery in the same direction as the primary wave. These are called "secondary centrifugal waves." If a reflected wave is accompanied by a rise of pressure it is a positive wave, if by a fall it is a negative wave.

From the considerations already mentioned as to the state of the capillary circulation, it is conceivable that such reflected waves may readily occur in the arterial system from the impact of a primary wave. Possible reflection may also be imagined from the points of division of the artery. It is likewise conceivable that a wave reflected back from the periphery along one artery may not only course to the heart, but may take a centrifugal direction along some other artery at a convenient point of junction. So many possibilities of interference are there, owing to the many branches of arterial division and the varying states of the circulation in the different organs of the body supplied by them, that it is difficult to imagine any but the most clearly dominating form of wave surviving the confusion. One can assume that the primary and main secondary waves are propagated without much change of character through all the arterial branches. The pressure movements caused by these waves are accurately recorded by means of Marey's well-known sphygmograph, and although it is a form of sphygmograph not much used at the present day it furnishes a typical pulse curve, or sphygmogram, which will serve as a basis for description. It represents the course of pressure in the artery, but not the amount of pressure. This is perhaps only to be obtained by means of manometrical observations in the physiological laboratory. Even should the whole series of curves under observation show a rise to a higher level, this is no evidence of a rise of intra-arterial pressure, since simple turgor of the skin may raise the lever. The curve, however, shows the course of pressure in the artery and presents peculiarities as it is taken from different arteries, but it has certain definite points peculiar to all pulse curves. First, there is a steep rise, corresponding to a primary positive wave, from the streaming of blood into the aorta. The slowness with which the rise takes place is some measure of the obstruction to the inflow of blood. After the summit of the primary wave is reached there is a drop to the dicrotic notch, following which there is a rise corresponding to the dicrotic wave. Between the summit of the first wave and the dicrotic notch there is an upheaval, which is usually on the falling line between the first summit and the dicrotic notch ; this upheaval may, however, in some cases reach a higher level than the initial rise. In this case the curve is called "anacrotic," in the first place it is called "katacrotic." According to Roy and Adami, the upheaval corresponds to the continuance of outflow from the ventricle, and it is by them termed the "outflow remainder wave" and by others the "tidal wave." The dicrotic notch is caused by a fall of pressure following

on the first distension of the vessel, and the subsequent dicrotic upheaval is caused by a centrifugal positive wave, according to some, arising from the recoil of blood at the root of the aorta against the closed semilunar valves, but according to v. Kries and v. Frey, and others also, by a positive centrifugal, but secondary wave, arising from a centripetal wave reflected from the periphery, which is again reflected from the semilunar valves. According to some writers (Grashey and Hoorweg) the dicrotic wave is propagated onwards in all arteries with the same velocity as the primary wave. Hürthle finds also that it occurs at almost the same period after the beginning of the primary wave ; on the other hand, Landois, v. Kries, and Edgren find that the dicrotic wave occurs somewhat later in the lower extremity. Whether the dicrotic wave owes its origin to central causes alone—that is to say, to a recoil of blood at the root of the aorta impacting against and reflected from the semilunar valves—or whether, on the other hand, it arises from a peripheral wave reflected back, and a second time reflected, has been much discussed. Bernstein and Hoorweg deny altogether that it is possible for a wave reflection to occur from the periphery with sufficient intensity to cause the dicrotic wave, since, owing to interference, only a small portion of the reflected waves can reach the heart ; on the other hand, v. Frey, Krehl, and v. Kries have proved that a reflection from the periphery does, in fact, take place. If in a lately killed animal the heart and lungs are removed and manometers are fixed in the subclavian and cœliac arteries, a wave driven into the arteries and suddenly broken shows by the manometer that reflection does occur ; these reflex waves can therefore reach the semilunar valve and be reflected from them again as positive waves. We must at least grant the possibility that if the dicrotic upheaval is caused by a wave of recoil at the root of the aorta a reflected wave from the periphery may accentuate and strengthen it. V. Frey and v. Kries, however, have opposed altogether the theory that the dicrotic wave can be of central origin ; while Grashey, Edgren, and Hoorweg, and also Hürthle, incline to the central origin of the wave and deny that it is conditioned by the central reflection of a centripetal wave from the periphery. To prove this point Hoorweg devised an ingenious experiment. Through an elastic tube provided with valves he sent the contents of a suddenly compressed ball, which was as suddenly relaxed. The sudden closure of the valves which followed also closed an electric circuit, and a spark was obtained from a Ruhmkorff's coil which bored a sharp hole from the point of the writing lever through the smoked paper of the recording cylinder. This perforation corresponded with the point of dicrotic upheaval. When the valves were taken away no upheaval occurred. Hürthle has made like and confirmatory experiments. With regard to these experiments and the deductions therefrom v. Kries objects that they presuppose a recoil at the commencement of the tubes and that it is a question whether a recoil of sufficient amplitude to cause the dicrotic wave can in fact arise at the root of the aorta. If we assume that the recoil occupies a very small fraction of time it would give rise to nearly nominal pressure movements, unless the quantity of blood thrown back against the valves were very great. In the latter case the rise of the dicrotic wave would be very sharp and steep, whereas the dicrotic wave is extended and broad-topped. Moreover, in the typical dicrotic pulses of nitrite of amyl and of fever we should have to assume a long period of time occupied in recoil, with an

attainment of great velocity of secondary wave, equal, as I have said, to the velocity of the direct primary wave, which can scarcely be the case. Tigerstedt points out that there is little true likeness between schematic experiments and the actual behaviour of the blood in relation to the aortic valve. In actual life, when the heart muscle relaxes, the muscular bundles which support the semilunar valves relax together with it; this relaxation permits the valves to be thrust back into the ventricle to a certain extent, and, owing to the great aortic pressure, more room is thus given, and, the semilunar valves being suddenly rendered tense, a new centrifugal positive wave ensues, which is shown as the dicrotic upheaval. Tigerstedt thinks the following view to be the most in accordance with known facts. Through the sudden influx of blood from the heart into the aorta a first positive wave arises, which corresponds to the first upheaval in the pulse curve. When the blood ceases to flow out of the heart a negative wave is propagated from the root of the aorta in a centrifugal direction through the arterial system. Another negative wave follows immediately on this, when the heart muscle relaxes, and the semilunar valves are no longer supported by their underlying muscular bundles and by the blood remaining in the ventricle. These two negative waves are commingled and are propagated in a centrifugal direction through the arterial system. At the same time with the second centrifugal negative wave a portion of the aortic blood streams backwards towards the heart, strikes against the closed semilunar valves, and sets up a second centrifugal positive wave which occasions the dicrotic upheaval. The slight upheaval on the falling limb of the curve of the cardiogram and that of the curve also of intra-cardial pressure are explained in the same way. The sudden shock of blood against the semilunar valves drives the valves to a certain extent into the heart and raises the intra-ventricular pressure, and the whole heart suffers a certain change of position. Tigerstedt believes in the possibility, under favourable circumstances, of a secondary upheaval caused by wave reflection from the periphery. If the reflection occurs as a positive wave it will add to the height of the dicrotic wave. Whether, as a rule, the reflected wave occurs without change of sign is difficult to say.

The matter is so interesting, and is so important for the understanding of the sphygmogram, that I must enter at a little more length into the views of v. Frey and Hürthle. V. Frey as well as v. Kries commence the discussion concerning the form of the original primary wave with the changes in velocity at the root of the aorta. Owing to friction, the changes of pressure observable in the pulse tracing do not correspond to the velocity; for instance, when the pressure falls the velocity continues. It is desirable to separate the wave into a systolic and a diastolic portion. In the systolic portion the change of pressure is very sudden; it is this portion of the wave which, according to v. Frey, gives rise to reflection from the periphery, and it is this portion, therefore, which has now to be considered. The systolic wave spreads into the arterial system with a velocity of several metres per second; the beginning of the rise of pressure, therefore, reaches the place of reflection before the heart is emptied of blood. If we assume a mean wave velocity of 23 feet per second, and the duration of systole to the point of closure of the semilunar valves to be 0·1″, 0·2″, or 0·3″, the head of the wave will have reached a distance respectively of 34, 68, or 102 inches from the aortic valve at their moment of closure. These numbers represent the

length of wave for the corresponding times of systole. "One must get rid of the idea that the pulse wave is a short wave which is propagated from part to part along the artery, and clearly understand that the pulse wave is of such a length that, under normal circumstances, one whole pulse wave has not room to extend from the root of the aorta to the artery of the great toe." The first part, or head, of the primary wave, therefore, reaches the periphery during the cardiac systole before the remainder of the wave, or tail, has left the root of the aorta, and there is ample time for the wave to be reflected from the periphery and to interfere with the oncoming primary wave. The interference wave (which is a centripetal wave of the first order) will, the nearer the sphygmographic tracing is taken towards the periphery, unite itself to the primary pressure rise, while diminishing the velocity. The nearer the tracing is taken towards the centre the further will the interference wave be from uniting itself with the primary rise, whose summit will, through division into its two component parts, become anacrotic or katacrotic, as the case may be. This first centripetal, or interference, wave reaches the heart and is reflected from the now closed aortic valve, becoming a so-called centrifugal wave of the second order. It is this which, according to v. Frey, when united to a second centripetal wave set up by it, is the cause in the peripheral arteries of the dicrotic upheaval. He thinks it likely that, in arteries near the centre, the dicrotic upheaval belongs to the type of secondary interference waves.

We have, then, the dicrotic wave, which is an expression of a positive centrifugal wave. This may originate either at the root of the aorta, owing to a recoil against the valves following on the first expansion of the aorta, or as a wave reflected from the periphery, and again reflected from the semilunar valves in a centrifugal direction, forming, so to speak, the tail of the pulse tracing, of which the first primary wave forms the head. As reflex waves we have also to recognise both the first centripetal wave, which is reflected from the periphery, and the waves which may be assumed to be reflected from the periphery at first in a centripetal direction, but which subsequently take a centrifugal direction along some branch or branches without journeying to the heart at all. It is clear that the first of these waves must cause a diminution of velocity, though possibly raising the pressure within the vessel. V. Kries places this wave as the first slight upheaval on the falling limb of the radial curve and says that pressure and velocity there show the most marked difference. The second slight upheaval on the falling limb before the dicrotic notch he considers to be a centrifugal wave, but one which, in its origin centripetal, has been reflected along some branch of junction. These conditions, if present, must necessarily give rise to considerable variations in the pulse tracings from different arteries. Tigerstedt points out that in the femoral tracing the dicrotic upheaval is much more spread out than in the radial tracing, and he thinks that the cause of this is not to be sought in the varying distance of the two points from the root of the aorta, since the distance of the two points from the root of the aorta is much the same in both cases, but that reflected waves from the abdominal vessels must considerably modify the femoral tracing. In order to make this clear I take from Tigerstedt, after v. Kries, a diagram of the radial curve with the arm pendant and raised.

V. Kries, who has studied these differences by means of the tachogram and plethysmogram, explains them thus. The

first positive reflexion from periphery to valves is proportionately less when the arm is pendant than when it is raised. In the pendant arm the vessels are distended with the column of blood in a greater degree than when the arm is raised; the positive reflexion of the centripetal wave from the valves in a centrifugal direction is therefore more marked in the raised arm and appears in the anacrotic summit. The amount of the heart's activity cannot be gauged by the amplitude of the pulse curve, as this amplitude is but the expression of wide variations in the internal pressure. The tone of the arterial wall and the amount of peripheral obstruction do to some extent influence the pulse tracing. A pulse, of course, shows more "dicrotism" the higher the dicrotic wave and the lower the preceding fall of pressure, being to some extent the expression of pressure varying largely at the instant, and usually goes therefore with low blood pressure, though in fever and in other conditions, where the tone of the vessel walls is diminished, dicrotism may arise even with an increase of blood-pressure, as has been specially noted by Roy and Adami [16]

As I have said, estimation of the mean velocity of blood has been made by Ludwig's "Stromuhr," which apparatus it is not my purpose to describe. The velocity of flow was shown to be about 0·3 to 0 5 metre per second. Of greater interest in connexion with sphygmography would be any successful effort to measure the velocity of the various parts of the pulse wave which are delineated in the sphygmogram. Vierordt attempted to do this in 1858 by applying the principle of inserting a pendulum into the blood stream and estimating the variations of velocity by the varying deviations of the pendulum. Chauveau and Lortet employed the same device, conveying, however, the movements of the pendulum by transmission to a Marey's tambour. By his "hæmodromograph" Chauveau made the following measurements on the carotid of a horse: Velocity during systole, 520 m.m. per second; velocity during diastole, 150 m.m. per second; and velocity of dicrotic wave, 220 m.m. per second. Of great interest, partly on account of their extreme ingenuity, are the methods employed by Fick and others. By inserting the forearm in a Mosso's "plethysmograph" variations in the volume of the arm during each heart-beat can be registered. The curve obtained has a general likeness to a pressure curve. That it represents a different series of events is apparent from the following considerations. The outflow of the blood from the forearm through the veins is constant and uniform, its quantity or rapidity being uninfluenced during the time occupied by a heart-beat, owing to the interposition of the capillaries. Though the mere increase in the volume of the forearm is due to increased quantity of blood injected into it through the arteries, the velocity of blood in the limb may diminish as the size of the arm increases. Any variations in the steepness of the volume curve can, however, only be due to increased or diminished velocity of blood entering the limb in a given time; for the blood only flows out of the limb at one steady and uniform rate. From the changes in direction of the volume curve, therefore—or, in other words, from the different angles of inclination of the various parts of a volume curve one to another in juxtaposition—can be deduced the velocity of the various parts of a pressure tracing taken synchronously from the pulse by means of a sphygmograph. The pulse tracing, it must be reiterated here, obtained by applying pressure to the side of an artery merely gives indications of the alterations of pressure within it, it being

conceivable that a return wave from the periphery along the artery in the direction of the heart may increase the pressure within the artery while diminishing the velocity. Any external pressure applied to the arteries of the forearm will consequently slightly vitiate the volume curve obtained by Mosso's apparatus, and it is necessary to eliminate this source of error, for the air within Mosso's cylinder exerts a pressure on the arteries of the forearm when the volume of the contained limb is increased, the elastic membranes which close the cylinder contributing to convert the apparatus somewhat into a pressure instrument. The whole difficulty has been most ingeniously avoided by v. Kries, who has almost succeeded in obtaining a pure direct velocity curve. In his "gas tachograph" he has connected the interior of Mosso's cylinder with the external air by leading a tube from it into a wide gas jet. Any steady

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[16] Practitioner, 1888, p. 423, et seq.

increase in the volume of the forearm would lead to no movement in the flame of the jet, but alteration in the steadiness would lead to unsteadiness in the flame—in other words, the flame would rise in correspondence with an increase of velocity and sink with a diminution of the velocity. By photographing the movements of the flame he therefore claims to have obtained a direct tracing of the velocity of blood flow during the various parts of the pulse wave. For the criticism of this method I must refer to an article by Hoorweg,[17] who thinks that there is much delay in transmission and in response, with difficulties owing to interference with the approach of gas to the flame, so as to destroy the value of the tracing for purposes of exact measurement. If disappointing in their result, the conception of these experiments by v. Kries cannot but excite our admiration.

The Goulstonian Lectures

ON

THE PHYSICS OF THE CIRCULATION.

Delivered before the Royal College of Physicians at the Examination Hall, Victoria Embankment, on March 6th, 1894,

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PHYSICIAN TO THE HEREFORD GENERAL INFIRMARY.

LECTURE III.

SINCE the sphygmograph only gives the course of pressure movements in the vessel and no absolute measurements of the amount of pressure, we are in great part indebted to the beautiful and accurate manometrical observations of Hürthle [1] for what proof there may be of the sequence of events in the arteries and the modifications introduced into the pulse tracing by different conditions. The changes of pulse, especially, which accompany blood pressure variations can, by the use of his manometer, be measured in conjunction with the pulse wave. The blood pressure was raised by irritation of the sympathetic nerve in the neck of the rabbit and lowered by irritation of the depressor nerve of the vagus. Hürthle follows Marey in distinguishing between an upward and downward limb of the pulse curve as well as between a single or double summit. He divides the curve into a systolic portion from the commencement of the rise to the dicrotic notch, and a diastolic portion from this point onwards. With regard to the double summit, if the second summit is higher than the first, so that the first appears upon the rising limb, the pulse is termed "anakrotic"; if the second summit appears on the falling limb, between the first summit and the dicrotic notch, the pulse is termed "katacrotic." He discards all terms of signification in naming the secondary waves, such as "outflow remainder wave" or "tidal wave," since the terms have not met with universal recognition, and he simply names them in their succession—first secondary wave, second secondary wave, and so on. The side pressure in the aorta was always obtained by inserting the glass tube of his manometer into the carotid.[2] The animals were always narcotised by morphia. The pressure movements varied during pulsation between 100 mm. and 130 mm. Hg., so that the pulsatorial increase of pressure amounted to not quite one-third of the minimum pressure. He found that the curve reached its first summit on an average in 0·025″ and in about the same time sank to a quarter or half the whole pressure movement, to rise again to the height of the first summit. This second summit is the first secondary wave and appears 0·05″ after the commencement of the primary wave. The curve sinks again from the second summit to a point either above or under half the height of the pulse, where a further upheaval occurs, which, however, does not attain to the same height as the foregoing. The second secondary wave occurs 0·12″ after the commencement of the curve. Since the descent of the second secondary wave is more rapid than the rest of the descending side of the curve, we can distinguish yet a third secondary

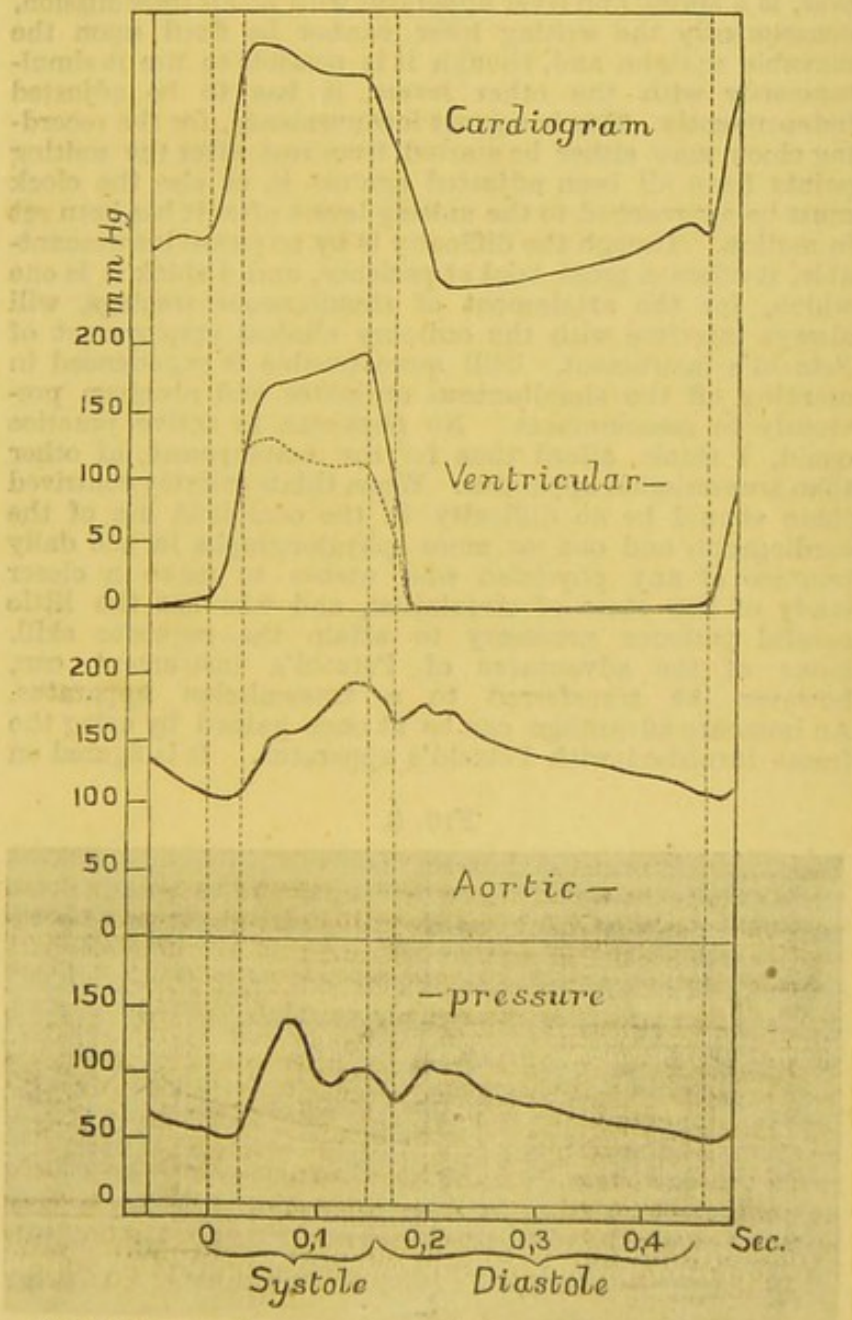
[1] Pflüger's Archiv, vol. xliii., 1888; vol. xlvii., 1890; vol. xlix., 1891.
[2] A full description of his manometer will be found in Pflüger's Archiv, 1888, vol. xliii.

wave, which often comes out as a distinct rise. Finally, before the commencement of the next pulse a fourth secondary rising is noticeable, which is quite short and low, but clearly distinguishable.

In comparing several consecutive pulses we can see that they are distinguished from each other by the second summit now rising above the first, now sinking below it. If the respiratory phases are registered synchronously with the pressure curve, then it becomes apparent that the second summit takes the highest place either at the end of expiration or beginning of inspiration, in order to sink again during inspiration above or below the level of the first summit.

A characteristic appearance of the pressure curve is furnished by different conditions of the vascular system. If the cervical sympathetic nerve is irritated the second summit appears to raise itself, some seconds after the commencement of irritation, more and more above the first, and so changes the appearance of the pulse curve that what was the first

FIG. 5.



Synchronous cardiogram and ventricular and high and low aortic pressure tracings. After Hürthle.

summit is only recognisable as a bend in the upward limb of

the curve; by this means the limb becomes longer and ascends less rapidly, and the angle at the curve summit becomes more obtuse. When the depressor nerve is irritated there is a different change: the curve attains its summit very quickly, although the rise is higher than normal; thereupon follows, just as suddenly, a sinking to beneath the level of the pulse height, so that the angle of the summit is very sharp. The first secondary wave now occurs 0·05″ to 0·01″ later than normal; the other secondary waves do not change their place in so constant a manner on irritation of the sympathetic and depressor nerves.

These experiments clearly prove that changes of the lumen of the peripheric vessels cause certain changes in the pulse form. The estimation of blood pressure in the various forms of pulse scarcely allows a doubt that the change of the tone of the vessels can only change the pulse form by means of the blood pressure. A change of pulse form is never to be seen without simultaneous rising or falling of the blood pressure. The tension of arterial blood alters with the tone of the vessels through increase or diminution of the obstacles which oppose themselves to the blood stream. Thus in irritation of the sympathetic nerve the minimum pressure is raised about 10 mm. Hg; the pulsatorial excursion is in comparison somewhat smaller. In irritation of the depressor nerve the minimum decreases from 10 to 50 mm. Hg, and the pulsatorial excursion becomes greater. By compressing the other carotid the rise of pressure brought about the same kind of result as that which was obtained by irritating the cervical sympathetic nerve, as also by compressing the abdominal aorta. By drawing away about 15 c.c. of blood from the second carotid the reverse effect was obtained. Hürthle found that the height to which the primary curve reached in a given time under a low pressure could amount to more than four times as much as the curve rises to under a high pressure. It has been already said that with high blood pressure the ascending limb rises more slowly. These time variations are suggestive and enable us to form some idea as to the varied form of the primary wave, as seen in the accompanying diagram from Hürthle. If A, B, C is the primary wave under normal blood pressure, then, on rise of the blood pressure, it is converted into the less excursive and more slowly rising form *a*, *b*, *c*, whilst under low pressure the rapidly rising and more excursive form α, β, γ is produced. The descending limb of the pulse curve falls more quickly under low pressure than under high. By accurate time measurements Hürthle found that the first

FIG. 6.

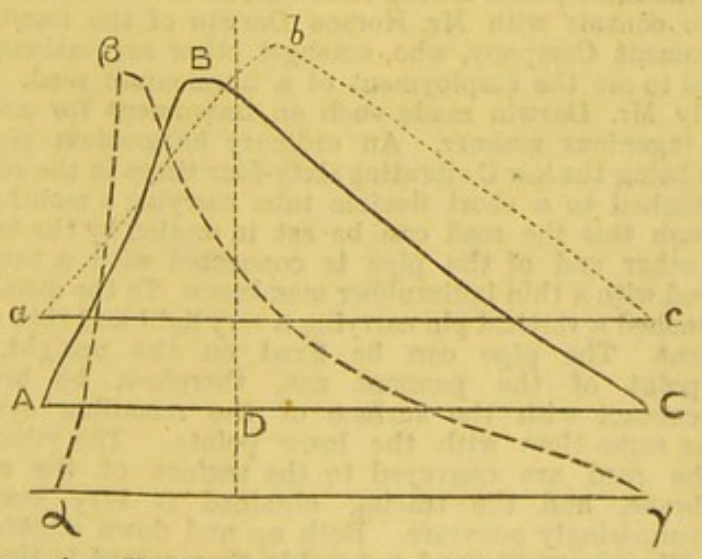


Diagram from Hürthle showing pulse variation with high and low pressure.

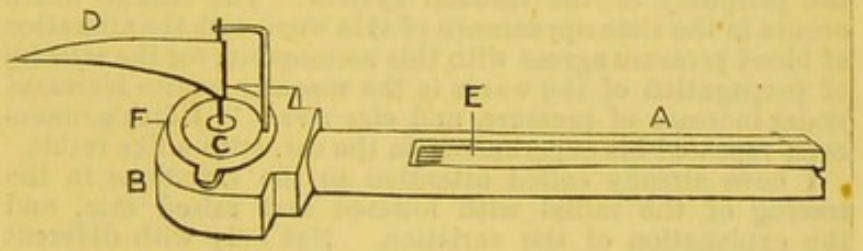
secondary wave occurred somewhat sooner than the primary under high pressure than under low pressure. If the first secondary wave occurs at the time-point D under normal blood pressure, then the double-pointed form of the pulse form *a*, *b*, *c* becomes anacrotic; on the other hand, if the wave appears under lower pressure, it occurs later, low in the descending limb of the pulse form α, β, γ. With regard to the nature of the first secondary wave Hürthle says: "If one can apply the results of the experiments made by Fick and v. Kries on human beings to the pressure pulse of the rabbit, then the first secondary rising which follows the summit must be regarded as a positive wave reflected from the periphery of the vascular system. The change which occurs in the time appearance of this wave with the alteration of blood pressure agrees with this assumption, for the velocity of propagation of the waves in the vascular system increases under increase of pressure, and *vice versâ*." Hürthle, moreover, repeated his experiments on the cat, with a like result.

I have already called attention to the variations in the tracing of the radial with lowered and raised arm, and the explanation of the variation. Not only with different conditions of the same pulse, but with different arteries, does the pulse form vary. Of these variations good examples are seen in the text-books.

To come to the possibility of the clinical application of all these facts. The pressure movements caused by the intra-arterial waves are accurately recorded by means of Marey's well-known sphygmograph. The pulse of high tension and the pulse of low tension there show their own distinct peculiarities. It has fallen into disuse for various reasons. To begin with, it does not admit of obtaining a record of a long series of pulsations, or of pulsations of sufficient amplitude for the accurate measurement of their component parts, nor is it provided with a time recorder for this purpose; but its chief defect for clinical use is that it does not admit of obtaining simultaneous records from different parts of one artery, or from two different arteries, or from the heart's apex and one or more arteries. For these purposes it is necessary to use an ordinary recording cylinder with a large surface, and in the highest degree convenient to employ the principle of air or water transmission through flexible tubes. With suitable apparatus of the latter kind the pulsations of the artery can be conveyed to an ordinary tambour, one or more of which can be fixed on the same upright; and their levers, by a slight rotation of the upright, can be brought all at once into the same uniformly light contact with the surface of the recording cylinder. When the cylinder is stopped the points of all the levers can be moved through any part of the tracings of which it is desirable to obtain a synchronous measurement, and the distances between the various points can be measured at leisure. The cylinder should revolve at a known speed, but for the purpose of measurement of the synchronous tracings so obtained it is not sufficient to trust to the uniformity of speed obtainable by clock work. The upright should, if possible, itself carry a time recorder. For this purpose an electric interrupter is the most accurate; but for ordinary clinical purposes it is too cumbersome and too expensive, and is not sufficiently portable. A vibrating tuning fork of sufficient size is too heavy and difficult to manage. The necessity of obtaining a light and accurate time recorder which would fit on to the upright and thus permit adjustment to the surface of the cylinder together with the lever points during the rotation of the cylinder led me to consult with Mr. Horace Darwin of the Cambridge

Instrument Company, who, amongst other suggestions, suggested to me the employment of a harmonium reed. Ultimately Mr. Darwin made such an instrument for me in a very ingenious manner. An ordinary harmonium pipe, its pitch being the low C vibrating sixty-four times in the second, is attached to a short flexible tube carrying a mouthpiece. Through this the reed can be set in motion by the breath. The other end of the pipe is connected with a tambour covered with a thin indiarubber membrane. To the membrane is attached a vertical pin carrying a very light and thin metal pennant. The pipe can be fixed on the upright, and the point of the pennant can, therefore, be brought into contact with the surface of the recording cylinder at the same time with the lever points. The vibrations of the reed are conveyed to the surface of the elastic membrane, and the tracing obtained is very beautiful and surprisingly accurate. Both up and down strokes can be easily measured, and a graphic time record is thus obtained which gives measurements to $\frac{1}{128}''$ or, in decimals, 0·0078125″. The extreme convenience of such an arrangement is obvious. It is alike inexpensive, easily worked, and lasting, and can be easily repaired. The experiment can be arranged, the lever points and time recorder adjusted in contact with the cylinder against a check, and then removed as one piece from the cylinder by a slight rotation of the upright. The cylinder is then set in motion, and at a favourable moment a current of air is blown by the mouth into the reed, and the upright is rotated against the check, a simultaneous record being at once obtained which can be accurately measured at leisure.

FIG. 7.



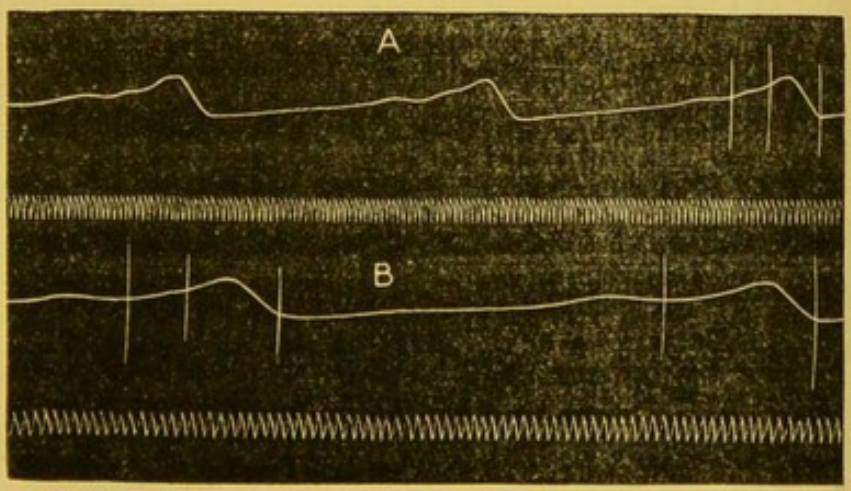
Harmonium reed adapted as a chronograph.

A sphygmograph *à transmission* was also devised for me at Cambridge. It consists of a wooden frame which rests on the front of the wrist, and which is kept in position by bands of inelastic material fastened behind it. Attached to the frame is a flat steel spring carrying a button at its free end, which is applied against the artery. The upper surface of the free end of the spring is jointed to a small vertical prop, which transmits the pulse movement to a tambour containing air. This tambour is made like a kettledrum, in that by turning some screws arranged round the circumference the tension of the membrane can be varied. The interior of the tambour is connected by means of a thick-walled elastic tube of small calibre to an ordinary Marey's tambour of the Cambridge pattern fixed on the upright. The steel spring can be depressed below the frame upon the artery by means of a screw; since this section also depresses the vertical prop, and with it the membrane of the tambour, the tambour itself is made to rise or fall on a vertical steel rod. The tension of the membrane and of the contained air can thus be modified. By introducing a T-piece into the elastic tube connecting the

two tambours further modifications of tension can be obtained. The amplitude obtained by this instrument is not great, but it appears to be almost entirely free from movements of inertia. The response to stimuli is exactly as quick as that of a Sanderson's cardiograph.

For ordinary purposes of demonstration the most suitable sphygmograph is that made by Petzold of Leipzig ; this, however, is a spring and lever apparatus with no air transmission, consequently the writing lever cannot be fixed upon the movable upright, and, though it is possible to use it simultaneously with the other levers, it has to be adjusted independently. This is a great inconvenience, for the recording clock must either be started from rest after the uniting points have all been adjusted against it, or else the clock must be approached to the uniting levers after it has been set in motion. Though the difficulty is by no means insurmountable, it offers a great trial of patience, and I think it is one which, for the attainment of simultaneous tracings, will always interfere with the ordinary clinical employment of Petzold's instrument. Still more trouble is experienced in marking off the simultaneous ordinates and abscissæ previously to measurement. No physician in active practice could, I think, afford time for the employment of other than transmission apparatus. When this is suitably contrived there should be no difficulty in the combined use of the cardiograph and one or more sphygmographs in the daily practice of any physician who wishes to make a closer study of the state of circulation, and who has the little careful patience necessary to attain the requisite skill. Some of the advantages of Petzold's instrument can, however, be transferred to a transmission apparatus. An immense advantage can be at once gained by using the frame furnished with Petzold's apparatus. It is figured on

FIG. 8.



A, Radial sphygmogram and chronogram, slow rate. B, The same at an increased rate.

page 23 of v. Frey's "Die Untersuchung des Pulses." The runners which lie on the wrist do not slip, as they lie in adaptation to almost any curve and are covered with velvet. The metal spring is of just the right flexibility and conveys the pulsations still more accurately and with greater amplitude than the spring of the instrument made for me at Cambridge. I have therefore obtained from Petzold two transmission sphygmographs, which combine all these advantages and which are beautiful examples of mechanical skill. As in his direct lever sphygmograph, there is an arrange-

ment on the frame of these which permits of considerable latitude of horizontal adjustment after the frame is fixed by the bands which keep it in position. By this means the most favourable position over the artery is readily obtained, and the instrument will not then slip. The value of the combined method is strangely neglected, the results being most valuable for the purpose of diagnosis, as I hope to make clear before the conclusion of these lectures.

The velocity of propagation of the pulse wave and the length of the pulse wave may be taken together. The velocity of propagation of wave is to be clearly distinguished from velocity of blood flow. The two things are quite independent of each other. For instance, a high blood pressure is accompanied with diminished blood flow in Nos. 1, 5, and 6 of Waller's table, but a high blood pressure conditions a greater velocity of pulse wave. As already stated, the mean velocity of blood-flow in the large arteries may be taken as 0 3 metre, or a little over one foot per second. The velocity of blood wave was first measured in 1827 by Weber, who found the delay between two points 1·32 metres apart to be about $\frac{1}{8}''$ to $\frac{1}{7}''$, indicating a velocity of about from 7 92 to 9·24 metres per second, or about 28½ ft. Since then observations on pulse wave velocity have been made by many observers, notably by Keyt of Cincinnati, who was perhaps the first to employ the graphic method as a physician in daily practice. He employed water transmission. A full description of his "compound sphygmograph," which is a beautiful, convenient, and ingenious instrument, is to be found in his suggestive work on "Sphygmography and Cardiography,"[3] edited after Keyt's death by Dr. Isham. It is claimed by these writers that the inertia of the contained water is in Keyt's instrument reduced to a negligeable quantity, while they acknowledge that the time lost in the transmission of a movement is greater by water than by air. They claim that water transmits movements with much greater power than air, and that this fact gives their instrument a wider range of application, embracing feeble movements that air is incapable of inscribing. A full discussion of these points would occupy more time than can be given to it in these lectures. I myself cannot help feeling that a water wave appears in many of their tracings, and that the inertia of the water is a thing which it cannot but be desirable to eliminate. Nevertheless, Keyt's work, together with that of Isham, is, on the whole, extremely valuable and must always excite admiration. Keyt gives "the following results regarding the pulse transmissions along different routes: carotid femoral time 0·0797″, which, with a distance of 18 in., gives a pulse velocity of 226 in. per second; carotid-radial time 0 0797″, which, with 23 in. distance, gives a pulse velocity of 288 in. per second; and femoral-posterior tibial time 0 0606″, which, with 33 in. distance, gives a pulse velocity of 544 in. per second." Other things being equal, it must be remembered that wave velocity is inversely proportional to the size and elasticity of the tube, and that these variations in velocity may be partly explained by the larger size of the aorta compared with the peripheral vessels. Since the less the elasticity of the tube the greater is the velocity of the pulse wave, it might be expected *a priori* that the velocity of wave is greater in old people than in young. Keyt found the pulse wave velocity in a child aged four and a half years to be 216 in. per second, in a young man aged twenty-five years 306 in., and in a man aged fifty years 416 in. per second. Again, taking the carotid and posterior-tibial interval in each case, he found the velocity in a child aged five to be

196 in., in a boy aged nine 247 in., in a man aged twenty-one 350 in., and in a man aged fifty-five 510 in. per second. He attributes these variations to the increased rigidity of the arterial walls with the progress of age. In the very rigid arteries of calcareous or atheromatous degeneration the velocity is largely increased; according to Keyt, increased or diminished intra-arterial blood pressure, on the other hand, does not appear to modify the pulse wave velocity to any considerable extent, nor is that velocity sensibly modified by variations of pulse frequency. The factors which chiefly influence pulse wave velocity are the size of the artery traversed and the amount of rigidity of the arterial wall. The carotid radial interval has been thus given by various observers :—

Time difference.	Velocity of propagation of pulse wave in metres per sec.	Writer.
0·094″	6·70	Czermak, 1864.
0·076″	8 29	Grummach, 1879.
0·080″	7·37	Keyt (died), 1885.
0·079″	7 63	Edgren, 1889.

My own measurements almost exactly correspond with those of Keyt and Edgren, being: time difference 0·078″ and velocity of propagation 7·9 metres (27¾ ft.). Dr. Waller[4] has much the same results. Grunmach[5] found that when the arm was immersed in water at a temperature of 104° F. the delay of the carotid radial interval was increased from 0·07″ to 0·096″,

[3] New York, 1887. [4] Journal of Physiology, vol. iii., No 1.
[5] Archiv für Anatomie und Physiologie, 1829.

owing to the local relaxation of the bloodvessels. Both Grunmach and Edgren have noticed a greatly diminished interval in arterial sclerosis and in chronic Bright's disease. Grunmach also found that the interval between the apex beat and the radial pulse increased by 0·162″ or by as much as 0·194″ when the blood pressure was lowered by expiratory compression of the thorax with closed glottis. According to Martius[6] the velocity is less during inspiration than during expiration—in the one case 6 m., in the other 8 m. per second. The length of the wave is calculated by Tigerstedt in the following way. Assuming that a ventricular systole lasts $\frac{1}{5}$″, and that the velocity of propagation of the pulse wave is 8 m. per second, the length of wave set up by each systole would reach to a distance of $\frac{8}{5}$ (or $8 \div 5$) = 1·6 m. (5 ft., "It follows, therefore, given this presupposition, that only the longest arterial paths are long enough to receive the whole length of a pulse wave and that the end of the pulse wave is still in the aorta when the first part of it has already reached to the periphery." From this point of view the formation of the dicrotic wave by reflection has been already discussed in these lectures. It must be remembered that, where there is lessened rigidity of the vascular wall, as in parts affected with vaso-motor paralysis, or where there is somewhere an increased width of the arterial path, as in parts affected with aneurysmal dilatation, the time of appearance of the distal pulse is delayed. Supposing that we now take simultaneous tracings from the heart's apex and from the radial artery, the cardio-radial interval will be found to be about 0·17″. Of this time we will assume 0·08″ to be occupied in the transmission of the pulse wave from the carotid to the radial. We may further deduct 0·02″ for the time occupied in the transmission of the wave from the semilunar valves to the point of the carotid whence our tracing is taken. This will leave a period of 0·07″, which we must take to be the time occupied between the commencement of ventricular systole and the

opening of the semilunar valves—in other words, this is the time, the *Anspannungszeit*, occupied in raising the intra-ventricular pressure sufficiently high to overcome the aortic pressure. This period of time is appropriately named by Keyt the "præsphygmic interval." He, however, considers the time occupied in the transmission of the wave along the path from the semilunar valves to the carotid to be so brief compared with the whole cardio-carotid interval that it may practically be neglected, and he, therefore, in speaking of the præsphygmic interval, must be understood to mean the interval between the commencement of the ventricular systole and the carotid pulse. For a pulse frequency of 70 Keyt's præsphygmic interval would be about 0·08″. In his very interesting paper,[7] "Notes of Observations on the Rate of Propagation of the Arterial Pulse Wave," Dr. Waller estimates this interval to be between 0·06″ and 0·09″ and remarks: "This considerable delay affords a reason for the distinct post-systolic character frequently recognisable as belonging to murmurs of aortic obstruction. It also explains why a first sound heard undivided at the heart's apex is often, as it were, split into two portions if listened to above the clavicle, the first portion being the transmitted first sound, the second the arterial rush beneath the stethoscope." Keyt states that the præsphygmic or cardio-carotid interval is, normally, about one-tenth the duration of the pertaining pulsation. "Thus a pulse of 60, 1″ long, will give for the interval $\frac{1}{10}$″; a pulse of 72, $\frac{5}{6}$″ long, would give $\frac{1}{12}$th, and so on." To everyone who has taken many tracings cases in which the præsphygmic interval is greatly diminished or even absent must afford matter for thought. My own attention was first called to it by the case of a man aged fifty-five suffering from emphysematous asthma and bronchitis. Wheezing and sibilus, with loud rhonchi, entirely obscured all sounds at the base of the heart; the apex-beat in a certain recumbent position was, however, plainly to be felt. The arteries were hard, tortuous, and atheromatous. A heart-radial tracing was easily obtained. The duration of cardiac systole was 0·2578″, the pulse-rate being 75 per minute and this duration being 0·03″ shorter than the shortest variations of normal systole for this pulse-rate. The heart-radial delay was 0·1328″ to 0·1406″, as compared with a normal interval of about 0·17″. A simultaneous carotid-radial tracing was then obtained, the carotid-radial delay being 0·10157″. Subtracting this from the heart-radial interval, the heart-carotid or præsphygmic interval was shown to be greatly diminished—viz., 0·039″ to 0·031″ as compared with a normal præsphygmic interval of 0·080″. Reflecting much over this case, it occurred to me that since the præsphygmic interval represented the

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[6] Ibid. 1891 Journal of Physiology, vol. iii., No. 1.

time occupied in raising the intra-ventricular pressure to an excess over the aortic pressure, and that a pulse-wave could not appear in the arteries until the semilunar valves were opened, there must in this case be some condition present whereby intra-ventricular pressure was equal to the aortic pressure at the commencement of systole. Such a condition would exist under the supposition that the semilunar valves were incomplete, whereby the aorta and ventricle formed one cavity. During diastole blood would freely regurgitate into the ventricle. At the end of diastole the intra-ventricular pressure would therefore be equal to that in the aorta, and at the commencement of systole a wave would straightway appear in the aorta without the intervention of a

præsphygmic interval. A simultaneous tracing was then taken from the heart and subclavian, when this interval was found to be practically *nil*. At the time I had met with no mention of this possibility among German writers, and I referred with much interest to Keyt, whom I found to have clearly recognised this abnormal shortening of the præsphygmic interval in aortic insufficiency. The fact was first demonstrated to the Société de Biologie as long ago as March, 1878, by François Franck. Keyt's tracings furnish a further proof, which seems to me to be conclusive. In my own case there was a "to and fro" rough double murmur, only to be heard at the apex owing to the wheezing and râles, which obscured all sounds at the base. I could not satisfy myself by auscultation that the murmur was mitral. The tracing, therefore, was most valuable as a means of diagnosis and satisfied me that my patient was suffering from aortic incompetency with ossification at the base of the heart. The case is specially interesting, since I do not know of any other method than the graphic one by which a correct diagnosis could have been made.

In his preliminary chapter on the "Variations of the Præsphygmic Interval in Disease" Keyt gives as his first proposition that "the præsphygmic interval is abnormally shortened in free aortic insufficiency," and I think that this proposition, confirmed as it is from independent sources, is one that should be admitted. He, with a positiveness which almost calls up antagonism, says: "The testimony is ample and complete and establishes the fact beyond question." If not quite so positive, we must allow, in full agreement with him, that the acquisition of the fact "is one of the triumphs of the graphic method." Keyt's positiveness often leads him to a logical fallacy. Having accepted the proposition that "the præsphygmic interval is always shortened in free aortic insufficiency," we are not bound to the proposition that the præsphygmic interval cannot be, even markedly, shortened in some other way, nor to reverse his proposition by saying that whenever the presphygmic interval is shortened we can diagnose aortic regurgitation. By the graphic method we are merely put into possession of an excellent means of more closely approaching to a correct diagnosis. Keyt himself, in another place, admits diminution of the interval in certain febrile conditions with rapid pulse; and it seems to me that there may be other conditions, as when there is congestion of the pulmonary veins and left auricle with low aortic pressure, tending to bring about an equilibrium of pressure on both sides of the semilunar valves towards the end of diastole, which would cause the same diminution of cardio-carotid interval.

If we accept, as I am disposed to do, that marked aortic insufficiency is always accompanied by great diminution of the cardio-carotid interval, it follows that in cases where such insufficiency is known to exist, and there is either no alteration from the normal delay or, still more, when there is an increase in the delay, we must assume the presence of some other retarding influence, of which the most obvious is aneurysm. François Franck[8] in discussing the well-known and accepted delay of the pulse due to an aneurysmal dilatation somewhere in its path, says that the retarding effect of the aneurysm may be counterbalanced by the inverse influence of aortic insufficiency, the combined results of the two opposing effects being the conservation of the normal delay. Keyt considers that in such a case the presence of aneurysm would be a positive certainty. I have very great admiration for Keyt's laborious and ingenious work, and for

his suggestiveness in connexion with this part of his subject; but I feel that he is inclined to rush precipitately to dogmatic statements from not wholly established premisses in connexion with difficult matters on which our present knowledge is necessarily imperfect. It can, however, be understood that when heart and radial delay is normal in a case of undoubted

[8] Journal de l'Anatomie et de la Physiologie, vol. xv., March to April, 1879.

aortic regurgitation, and still more clearly, if the heart and carotid delay is normal, we have something for which it is necessary to account, and that the most likely explanation would be the presence of an aneurysmal dilatation somewhere in the course of the vessel. Suppose now that simultaneous tracings are taken from both radials. If the radial pulses are synchronously delayed we may assume that the unseen and unsymptomed aneurysmal dilatation involves the ascending or transverse part of the aorta. Should the right radial show delay there is a probability in favour of the dilatation affecting the innominate and the vessels arising from it; should the delay be on the left side only the aneurysm is located further along the arch. In fact, delay affecting any artery may be an important step towards the localisation of a presumable aneurysm in a part beyond the reach of direct observation. I think, for those who require some immediate proof of practical use from any study, this should give the combined use of the sphygmograph and cardiograph a distinct claim on their attention.

Keyt, by means of experiments with a schema and from observations of cases of mitral regurgitation, was led to formulate another proposition—viz., that "the præsphygmic interval is abnormally lengthened in mitral insufficiency." He laments that his announcement attracted but little attention and that his observations have neither been confirmed nor disproved by others. I think it is as well to discard all experiments with a schema if by them we think to imitate with any accuracy the behaviour of our own extremely complicated organism. The assumption to which such experiments led Keyt is that the intra-ventricular blood pressure will not rise to an excess over the aortic pressure so soon as normally if some of the ventricular blood can escape back into the auricle owing to incompetent mitral valves. No one can deny the possibility of his proposition in some instances. That it can be accepted as a general proposition applicable to all cases I cannot think; it would, even theoretically, need innumerable qualifications. It would be necessary always to exactly know the amount of blood pressure in the aorta and the relation of aortic pressure to the length of the præsphygmic interval. To say the least, this has not yet been worked out. The varying amount of blood flow from the auricle and the varying pressure within it at the end of diastole, the state of the auricular muscular walls and the time of auricular relaxation, as well as the changes in the pulmonary circulation owing to various causes, would all have to be considered. When Keyt goes further and contrasts the value of the graphic method with the auscultatory, to the disadvantage of the latter as regards the diagnosis of mitral incompetency, we begin to ask for distinct clinical proof of his general statement. We then find that he divides cases of mitral regurgitation into two classes, one of which he distinguishes as consisting of cases of "pure, harmful mitral regurgitation," and the other of cases of "harmless regurgitation." The latter are cases presenting a bruit, but where the heart is unembarrassed and regular, and where the

compensation is permanently satisfactory, these cases, according to Keyt, present no lengthened præsphygmic interval. This arrangement may be convenient for his argument, but I do not think it will appear to be particularly helpful to us. He has, with the best intentions, begged the question in his eagerness to prove his point. Are we to assume that we obtain abnormal lengthening of the præsphygmic interval only in cases of uncompensated mitral disease? Then surely the deranged innervation, the irregularity, and the feebleness of contraction may also play some part in producing the pulse delay? Keyt, however, distinctly asserts that by the aid of this sign a positive diagnosis may be made at once, in any stage of the case, and without waiting for the development of sequences and symptoms; and that by the amount of retardation we may measure the amount of the regurgitation. One can but look wistfully at such a possibility. I do not think Keyt's tracings in this particular are very convincing.[9] Moreover, I have not myself obtained definite results in either direction, having in some cases met with cardio-carotid delay, but in others, where the mitral incompetency was very considerable indeed, I have failed to obtain any variation from the normal.

I have spoken of Dr. Waller's description of the post-systolic character given to a murmur of aortic obstruction owing to the intervention of the præsphygmic interval, the murmur following immediately after, though almost with, the first sound. The assumption here—and I think very probably a correct one—is that the first sound concurs with

[9] Loc. cit., p. 193.

the commencement of contraction, being a muscular rather than a valvular sound. Is it possible that the murmur of aortic obstruction, when there is also aortic insufficiency, may, the præsphygmic interval being absent, antedate the first sound? Keyt thinks it may, but we should have to assume in this case (1) that the first sound was purely due to valvular closure, and (2) that the closure occurred some time after the commencement of ventricular contraction—both of which assumptions are contradicted by physiological experiments.

An interesting tracing, which I obtained in a case of renal disease, well illustrates a point which I mentioned in an early part of these lectures, and gives an explanation of some cases of what, to the ear, appears to be an independent action on the part of the two ventricles.

FIG. 9.

Usually one pulse is felt to each pair of contractions. The heart seems to "tumble" in its action, and two first sounds may be distinctly heard in close sequence, followed by one second sound. The descending limb of the first of each pair shows no distinct angle of drop, and the descent is slow, as if during relaxation of the ventricle the heart was still distended with blood. There is, however, a distinct auricular systole preceding the first rise. The auricular systole is

The second effort of the heart is futile, action of an empty ventricle.

absent from the second of the pair, [redacted] s. It will always be a matter of regret to me that at that time I had not sufficient knowledge of methods to obtain a simultaneous pulse tracing, which would have placed the explanation beyond the possibility of doubt. It is, however, quite clear that the heart makes two distinct contractions and that the appearances on the tracing are not due to independent action of the two ventricles; it is an effort at compensation of a curious and hitherto unnoticed kind.

Table for facilitating Measurements by means of a Harmonium Reed vibrating 64 complete Vibrations in a Second.

Half vibrations.	Fractions of second in decimals.	Half vibrations.	Fractions of second in decimals.	Half vibrations.	Fractions of second in decimals.	Half vibrations.	Fractions of second in decimals.
1	0·0078125	33	0·2578125	65	0·5078125	97	0·7578125
2	0·015625	34	0·265625	66	0·515625	98	0·765625
3	0·0234375	35	0 2734375	67	0·5234375	99	0·7734375
4	0·03125	36	0·28125	68	0·53125	100	0·78125
5	0·0390625	37	0·2890625	69	0·5390625	101	0·7890625
6	0·046875	38	0·296875	70	0·546875	102	0·796875
7	0·0546875	39	0·3046875	71	0·5546875	103	0·8046875
8	0·0625	40	0·3125	72	0·5625	104	0·8125
9	0·0703125	41	0·3203125	73	0·5703125	105	0·8203125
10	0·078125	42	0·328125	74	0·578125	106	0·828125
11	0·0859375	43	0·3359375	75	0·5859375	107	0·8359375
12	0·09375	44	0·34375	76	0 59375	108	0·84375
13	0·1015625	45	0·3515625	77	0·6015625	109	0·8515625
14	0·109375	46	0·359375	78	0·609375	110	0·859375
15	0·1171875	47	0·3671875	79	0·6171875	111	0·8671875
16	0·125	48	0·375	80	0·625	112	0·875
17	0·1328125	49	0·3828125	81	0 6328125	113	0·8828125
18	0·140625	50	0·390625	82	0·640625	114	0·890625
19	0·1484375	51	0·3984375	83	0·6484375	115	0·8984375
20	0·15625	52	0·40625	84	0·65625	116	0·90625
21	0·1640625	53	0·4140625	85	0·6640625	117	0·9140625
22	0·171875	54	0·421875	86	0·671875	118	0·921875
23	0·1796875	55	0·4296875	87	0 6796875	119	0·9296875
24	0·1875	56	0·4375	88	0·6875	120	0·9375
25	0·1953125	57	0·4453125	89	0·6953125	121	0·9453125
26	0·203125	58	0·453125	90	0·703125	122	0·953125
27	0·2109375	59	0·4609375	91	0·7109375	123	0·9609375
28	0·21875	60	0·46875	92	0·71875	124	0·96875
29	0·2265625	61	0·4765625	93	0·7265625	125	0·9765625
30	0·234375	62	0·484375	94	0·734375	126	0·984375
31	0·2421875	63	0·4921875	95	0 7421875	127	0·9921875
32	0·25	64	0·5	96	0·75	128	1·0

These lectures must now come to a close. I should like publicly to recognise the enlightened spirit of the board of management of the Hereford General Infirmary, who granted me £25 for the improvement and purchase of apparatus, and to return my thanks to the Scientific Grants Committee of the British Medical Association for the sum of £5 for minor expenses. Finally, I must thank the College for entrusting these lectures to me. It has been my effort to be clear rather than literary, but the difficulty of my subject is great, and I fear that I can scarcely have succeeded in either attempt. The honour of giving them has, however, been something to me, and the feeling that they might be of use has made me think the task light indeed. I can only hope that you, in hearing them, as I, in writing them, have gained some clearer vision of things still lying in much obscurity.

THE LANCET.

MARCH 3, 1894.

GOULSTONIAN LECTURES (the) on the Physics of the Circulation. Delivered before the Royal College of Physicians at the Examination Hall, Victoria Embankment, on Feb. 27th, 1894, by PAUL M. CHAPMAN, M.D., F.R.C.P. Lond., Physician to the Hereford General Infirmary.—Lecture I.—(*Illustrated*) ....

MARCH 10, 1894.

GOULSTONIAN LECTURES (the) on the Physics of the Circulation. Delivered before the Royal College of Physicians at the Examination Hall, Victoria Embankment, on March 1st, 1894, by PAUL M. CHAPMAN, M.D., F.R.C.P. Lond., Physician to the Hereford General Infirmary.—Lecture II.—(*Illustrated*) ....

MARCH 17, 1894.

GOULSTONIAN LECTURES (the) on the Physics of the Circulation. Delivered before the Royal College of Physicians at the Examination Hall, Victoria Embankment, on March 6th, 1894, by PAUL M. CHAPMAN, M.D., F.R.C.P. Lond., Physician to the Hereford General Infirmary.—Lecture III.—(*Illustrated*) ..

normal

Pulse Rate 75 per min.
H. R. interval ".17"
Precordial " .080"